Integrating the Life Sciences from Molecule to Organism

The Physiologist

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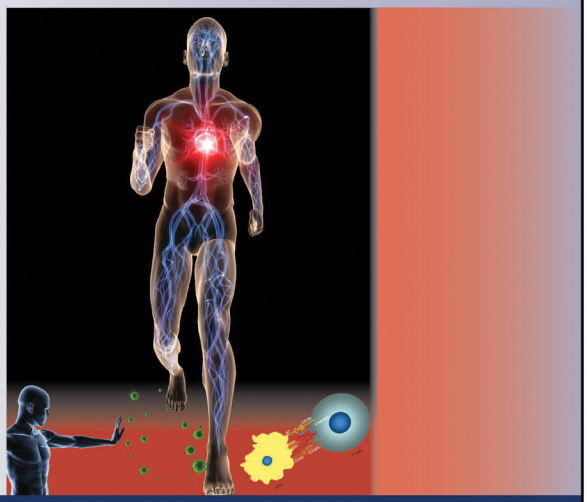
2010 APS Intersociety Meeting: Global Change & Global Science: Comparative Physiology in a Changing World

(August 2010, Westminster, Colorado)

2010 APS Conference: Inflammation, Immunity and Cardiovascular Disease

(August 2010, Westminster, Colorado)



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Does Open Access Lead to Increased Readership and Citations?

A Randomized Controlled Trial of Articles Published in APS Journals

Philip M. Davis, Cornell University

Introduction

Citations are a measure of scholarly impact in the research community and a basis for scientific reward. Prior studies have suggested that free (or open) access to scientific publications leads to increased citations. Several studies have reported very large citation effects associated with free access (e.g., between two and seven times) (2).

These studies, however, are based on unobtrusive, observational analysis—many without statistical controls. As a result, it has been difficult to determine whether this relationship between access and citations is causal, the direction of causality, or whether the relationship is an artifact of other explanatory variables. Secondly, while readership is implied as an intermediary cause between access and citation, no prior study has investigated the effect of access on article downloads.

In order to isolate the effect of access on readership and citations, we conducted a randomized controlled trial of open access publishing on articles published electronically in 11 APS journals. This report details the findings three years after the commencement of the experiment. Earlier findings of this study may be found in Davis, et al (4).

Methods

Scientific articles, 1,619 from 11 APS journals published between January and April, 2007, formed the study group. Upon electronic publication, articles were randomly selected into either the treatment group (n=247) or the control group (n=1372). Treatment articles received immediate free access; control articles followed their normal publication trajectory (subscription-access for the first 12 months, followed by free access).

A stratified random sample was used to ensure equal representation of articles from each section of APS journals. Only research articles and reviews were included in the study. Details of the sample dataset are provided in Table 1.

The sample size was designed to provide enough statistical power to detect a 25% difference in citations between groups. Since previous studies have reported differences on an order of 200-700%, we should have sufficient statistical power to detect a difference, if one exists.

Usage statistics were gathered on a monthly basis directly from the publisher via HighWire Press. Known robot activity (software robots downloading all free material for indexing purposes, e.g., Google) was removed from the dataset prior to analysis. Citation figures were

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From the President's Desk

In this issue, I thought to tell you about our wonderful staff at "APS Central" in Bethesda, MD. While the member-based leadership (Council, Committees, Sections, and Editors) variously have the final say on what programs are conducted, approve the annual budget, do the scientific editing of the journals, program the annual meeting, and provide expert scientific commentary, we (the membership) would be spinning our wheels (or treading water, or whatever metaphor you prefer) without the support and actions of our staff who faithfully implement our decisions and programs.

We have 73 staff divided among nine departments. Those departments are:

Publications (Rita Scheman with 40 staff), Membership & Meetings (Linda Allen with four staff); Science Policy (Alice Ra'anan with two staff); Communications (Donna Krupa); Education (Marsha Matyas with seven staff); Marketing (Sue Sabur with one staff); Information Technology/ Computer Support (James Chapman with two staff); Business/Circulation (Bob Price with six staff); and finally Executive (Marty Frank with two staff). You can find the complete listing on the APS website (www.the-aps.org/about/staff.htm).

Publications is the backbone of the Society, however you measure it--by effort, by budget (at least 80% of our $18 million annual Societal budget), by staff size, and by tradition. APS puts out 14 journals (seven in the *American Journal of Physiology* series, plus *Physiological Genomics*, *Journal of Applied Physiology*, *Journal of Neurophysiology*, *Physiological Reviews*, *Physiology*, *Advances in Physiology Education* and *The Physiologist*). This requires a small army of experts in business, publishing, peer review, art, copy editing and the care and feeding of editors. While every one of the 44 staff members carries out essential functions, I have come to know and respect Rita as a very even-keeled, realistic, publications director with great perspective and forward thinking abilities. We are very lucky to have the whole pubs crew on board. Without them, the APS would not be the unique Society it is today, and the financial returns realized each year by our publications would not be there to support many of our other programs.

Membership and Meetings keeps track of y'all, and supports the development and implementation of our meetings and conferences. Linda Allen, APS' longest term (not oldest!!) employee runs this department efficiently and expertly. Just imagine what it takes to control, year after year, a committee of members charged with programming Experimental Biology and coming up with a game plan. When she has something to say, Council listens.

Science Policy is a very important department in APS. We need to be in a position to influence decisions, made by non-scientists on Capitol Hill and elsewhere, on issues that affect science in general and the discipline of Physiology in particular. Alice Ra'anan and her staff provide the absolutely essential support and expertise to guide our members in developing position papers on many politically charged topics, and connect us with the relevant politicians or other dignitaries we need to engage in person. Advocating for Federal research funding and for the humane use of animals in research are two areas of major focus, and in the latter APS has become the "go to" organization.

Communications is an area in which we (that is, Donna Krupa) work out ways to establish the importance of Physiology to the health and welfare of people and animals everywhere. Donna spends much of her time identifying research advances, reported in our journals and at our meetings, which will be of widespread public interest. She then connects the media to those advances, making sure the APS is recognized. Most of us don't see the product of her efforts, but a quick visit to the APS website communications department will tell you how active she is.

Education is an APS crown jewel. I cannot do justice to Marsha Matyas and her staff here, so please visit the website to learn about our amazing array of educational outreach programs. Simply put, APS has become a model to many other organizations in the domain of education. Marsha has infiltrated essentially all educational levels from grade school to graduate school. In this age of molecular biology, we need to make it very clear to our youth that Physiology is the essential link between molecular discovery and clinical care, and Marsha and her crew have done an outstanding job in this regard. Just look at the number of pre-doctoral trainees who come to Experimental Biology to present their physiological research. That wouldn't happen without this department.

Marketing is a department that quietly supports all of our ventures—meetings, education, publications, etc - with great creativity. Sue and Kathleen never seem to be "visible" from a member's perspective (many of the other departments participate in Council meetings for example), but rest assured their work is critical, and will become more so as we go forward.

Information Technology/Computer Support is obviously essential to the functioning of the APS at all levels. James Chapman and his team - also "invisible" to most of us - support the website, the hardware and the management software without which APS could not function. We are fortunate to have them.

Business is a supporting department that manages our budget day by day and year by year, and coordinates a very well-managed investment plan for our reserves. Run by Bob Price, it is highly efficient and accurate. Speaking as immediate past chair of the finance committee, I can attest to a long string of clean audits, and an equally long string of balanced budgets that at the end of the year have actually turned in (modestly) positive margins that keep the Society strong and its myriad programs running. I know other organizations that would love to have such a lean, mean, accounting machine as we do.

Executive is, in essence, Linda Dresser and Esther Samuel supported by Marty Frank. All jokes aside, it is appropriate to end this discussion simply by saying that Marty, whose tenure at APS has just reached 25 years, has run the Society superbly. As an officer, it is obvious to me how much the membership relies on Marty and assumes (with full justification) that he will always come through. He does not just manage the office - he has passions (other than family and golf) that include in particular making APS a happy place to work, protecting our publishing enterprise, and reaching out to our trainees (and pre-trainees) to encourage them to consider physiology as a career. But he could not do this without Linda and Esther.

So yes, we have a large staff and a corresponding payroll. But our staff is a collection of dedicated and expert people who over many years have shown, and continue to show, unconditional support for us, the members of APS, and for the discipline itself. Without them, APS would be nothing. ❖

Table 1. Description of the American Physiological Society journal dataset.

Journals	Open Access (Total Articles)	% Open Access
AJP: Cell Physiology	36 (155)	23
AJP: Endocrinology and Metabolism	21 (147)	14
AJP: Gastrointestinal and Liver Physiology	22 (134)	16
AJP: Heart and Circulatory Physiology	32 (233)	14
AJP: Lung Cellular and Molecular Physiology	14 (109)	13
AJP: Regulatory, Integrative and Comparative Physiology	34 (195)	17
AJP: Renal Physiology	18 (140)	13
Journal of Applied Physiology	27 (201)	13
Journal of Neurophysiology	39 (278)	14
Physiology	2 (11)	18
Physiological Reviews	2 (16)	13
Total	**247 (1,619)**	15
Categorical Properties (totals)		
Research Articles	228 (1,519)	15
Review Articles	19 (100)	19
Methods Articles	7 (29)	24
Cover article	2 (11)	18
Press release	1 (5)	20
Total	**247 (1,619)**	15

gathered on a monthly basis from ISI's Web of Science.

The researcher was solely responsible for the randomization, data gathering, analysis and reporting of this study.

Results

Open access treatment articles received significantly more article downloads and reached a broader audience, yet were cited no more frequently, nor earlier, than subscription-access control articles.

Article downloads

During the first year of publication, open access articles received more than double the number of full-text downloads (119%, 95% C.I. 100% - 140%) and 61% more PDF downloads (95% C.I. 48% - 74%) from a third more unique visitors (32%, 95% C.I. 24% - 41%). Abstract views were reduced by nearly a third (-29%, 95% C.I. -34% – -24%) signaling a reader preference for the full article when available.

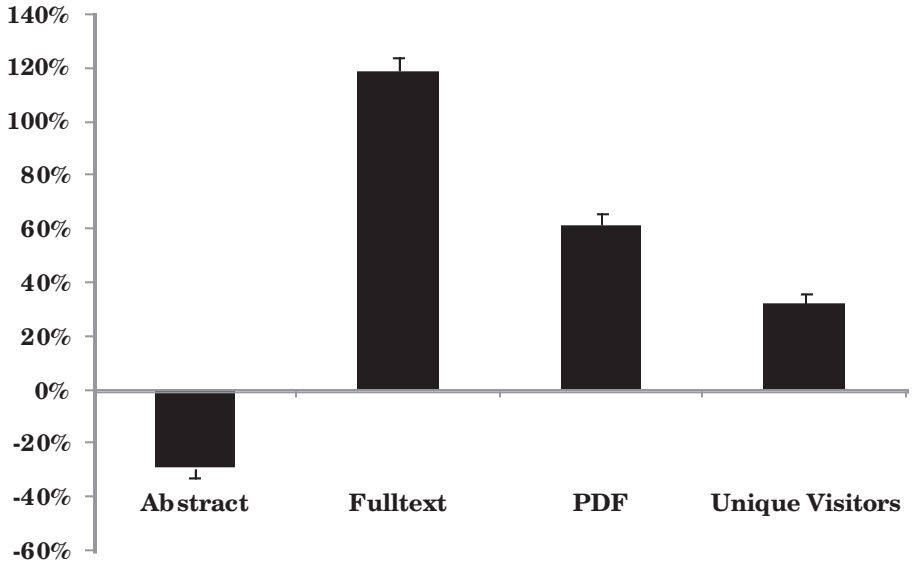

Figure 1. Percent increase (+ S.E.) in article downloads and unique visits to open access treatment articles compared to subscription-access articles published in 11 journals by the American Physiological Society, January-April 2007. Analysis controls for individual journal effects.

Citations

Open access treatment articles were no more likely to be cited in their first year than subscription-access control articles. A total of 71% (175 of 247) of open access articles were cited within their first year compared to 74% (1019 of 1372) of control articles.

Thirty-six months after publication, open access treatment articles were cited no more frequently than articles in the control group (Figure 2). Open access articles received, on average, 10.6 citations (95% C.I. 9.2 - 12.0) compared to 10.7 (95% C.I. 9.6 - 11.8) for the control group. No significant citation differences were detected at 12, 18, 24 and 30 months after publication.

Discussion

The results of this experiment suggest that providing free access to the scientific literature may increase readership (as measured by article downloads) and reach a larger potential audience (as measured by unique visitors), but have no effect on article

citations. These results are consistent with an earlier report of the APS study after one year (4) and the results of other scientific journals after two years (3).

The fact that we observe an increase in readership and visitors for Open Access articles but no citation advantage suggests that scientific authors are adequately served by the current APS model of information dissemination, and second, that the additional readership is taking place outside this core research community (4).

The increase in full text downloads for Open Access articles during their first year after publication (Figure 1) suggests that the primary benefit to the non-subscriber community is in browsing, as opposed to printing or saving, which would have been indicated by a commensurate increase in PDF downloads.

In sum, the real beneficiaries of Open Access may not be the scientific author community, who traditionally have excellent access to the research literature, but communities of practice that consume, but rarely contribute to, the corpus of literature. These individuals may include students, educators, physicians, patients, and researchers employed by private industry who depend on the publication of scientific literature. Further research is required to identify these groups and their use of the scientific literature.

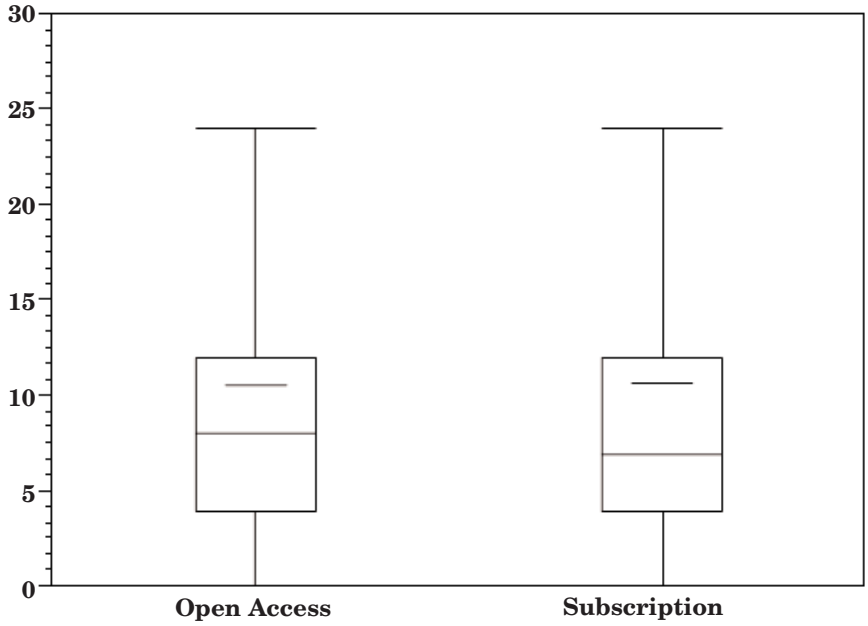

Figure 2. Variability plot of citations to APS articles 36 months after publication. Boxes represent the interquartile range (25th to 75th percentile) and contain the median value (horizontal line) and average value (horizontal dash). "Whiskers" extend 1.5x the length of the interquartile range.

Study Limitations

1. Access is not a necessary precondition to citation. An author may cite from the abstract of an article or simply copy the reference from another paper. The result of this behavior may attenuate any access-citation effect.

2. Readers of scientific articles without access to a journal from the publisher's website may find other avenues of access, such as through colleagues located at other institutions or by contacting the author directly for a copy (6). Authors may self-archive their article by placing it on the public Internet or in an institutional repository. In our study, we were only able to identify 18 instances of self-archiving of APS articles—too few for statistical analysis.

3. ISI's Web of Science was the source of citations in our study. While WoS does not index the entire corpus of research literature, it does provide a reliable and comparative sample of citations to other citation counting services such as Scopus (1, 5).

Acknowledgements

This experiment would not be possible without the help and support from Marty Frank, Rita Scheman, Margaret Reich, Mark Goodwin and Mike Gentry. This study was conducted as part of doctoral research in the Department of Communication at Cornell University. Funding was provided by a grant by the Andrew W. Mellon Foundation under the Scholarly Communications and Technology Program.

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APS Leadership Visits Brazil

Members of the APS leadership, (Peter D. Wagner, Gary C. Sieck, and Martin Frank) visited Brazil at the invitation of the Brazilian Society of Physiology (BSP) and to attend the annual FeSBE meeting, August 25-29, 2010, in Aguas De Lindoia. FeSBE is a multi-society meeting similar to the FASEB/Experimental Biology meeting. The seeds for the visit were planted at the 2009 IUPS Congress, Kyoto during conversations between Benedito Machado (Council member of the BSP), Sieck and Frank as we explored opportunities for collaboration between the American and Brazilian Physiological Societies. These discussions were continued during Experimental Biology 2010, Anaheim when the APS leadership met with Machado and members of the BSP to explore further collaboration, including the possibility of a Pan-American Physiological Congress. As a result of these discussions, the APS leadership was invited to Brazil in August to further those discussions and to learn about the research programs in Brazil.

The leadership visited three institutions: the Department of Physiology, Univ. of Sao Paulo, Ribeirão Preto (USP, RP); Department of Physiology, Institute of Biomedical Sciences, Univ. of Sao Paulo, Sao Paulo; and Division of Hypertension, Heart Institute, Univ. of Sao Paulo. The faculty members at these institutions all expressed enthusiasm for collaboration with the APS and its members, and specifically said they would welcome opportunities to host visits by US scientists, taking advantage of potential Brazilian government funding.

Peter Wagner, Gary Sieck, Benedito Machado and Martin Frank at the USP, Ribeirão Preto.

During our visit to USP, RP, Machado and his colleagues informed the leadership about their program. The institution is a six-year medical school which accepts approximately 100 students per year. During the first two years, the students receive instruction in the basic sciences with physiology taught as a block. USP, RP also has an undergraduate campus with approximately 7,500 students and a graduate program of approximately 2,500 students. The graduate program in physiology was initiated in 1970. It is comprised of 23 faculty, 16 within physiology, and seven with joint appointments from other departments.

Support for graduate education within Brazil is quite good with several State and Federal agencies providing support. There are several agencies that provide fellowship support for Masters and Doctoral students including the Coordination of Improvement of Higher Education Personnel (CAPES) http://www.capes.gov.br/, the National Council for Scientific and Technological Development (CNPq) http://www.cnpq.br/english/cnpq/index.htm, and the Foundation for Research Support of Sao Paulo (FAPESP) http://www.fapesp.br/. These fellowships provide funding for supplies and for some faculty support. In general, the Masters program lasts 30 months and the student would have the fellowship for two years. The PhD fellowship is for four years which is the anticipated duration of the PhD program. The fellowship funds can be used for the candidate to work outside of Brazil for short periods. Fellowships are also available for the faculties to bring foreign postdoctoral fellows to Brazil.

Under the leadership of Jose Antunes-Rodrigues and the Brazilian Society of Physiology, a multi-center graduate program in physiology was initiated in March 2009. The objectives of the program were to establish a new model for graduate programs and to encourage institutional/interdepartmental collaboration. Presently there are 11 programs in physiology in Brazil under the national coordination of CAPES. However, there are regional disparities in terms of Graduate Programs in Brazil, including all areas of knowledge, including physiology. In order to solve this problem, 30% of the federal budget for science and technology is currently devoted to institutions/programs in the northeast, central/west, and north. However, 73% of the graduate programs are in southeast and southern and Brazil. Overall, between 1970 and 2008, 1,704 MS degrees and 1,136 PhD degrees were awarded in physiology.

The goal of the new program is to help some of the smaller institutions in their efforts to have doctoral programs in physiology. The program is coordinated by the Brazilian Society of Physiology and is designed to establish an association of productive researchers in isolated public institutions, where implementation of an independent program is not yet possible, with researchers from large productive consolidated graduate programs. Students are able to spend part of their research program at an institution with a stronger research program, yet still receive their degree from the home institution. At present there are 71 students in the program spanning the seven associate institutions, with 62 students working on MS degrees and nine working on PhD degrees.

Programs designed to benefit both Brazilian and US physiologists were reviewed during our meetings and included the APS Latin American Initiative, Early Career International Travel award program, as well as student membership at a rate lower than for student membership in BSP. It was noted that there are opportunities for US and other international students, as well as postdoctoral and junior faculty, to come to Brazil on fellowships supported by the Brazilian agencies supporting research at the state and federal levels. Interested individuals were encouraged to contact individual laboratories to explore the possibility. Through CAPES and Brazilian universities, individuals can come for six months for teaching or research opportunities. Similarly, doctoral students from Brazil can go abroad with support from Brazilian funding agencies. All in all, the APS leadership was very impressed by the level of government

investment in research, especially in trainees, and the potential support for foreign scientists at many levels to come to Brazil for short periods.

During the course of our meetings, we also discussed funding for scientific meetings and research. It was noted that in general scientific meetings receive national support. When the BSP held its joint meeting with The Physiological Society in Ribeirão Preto in 2006, 90% of the support was derived from Federal and state sources. Similarly the FeSBE meeting was well supported by the Federal and state governments. Grant funding is also well funded by the Federal and state governments with approximately 50% of proposals receiving support. However, salaries are provided by the institution and not funded by the grants. The Federal government also sets aside funding for institutions in the less developed Brazilian states, for example institutions in the northeast and within the Amazon area as a means of stimulating academic and economic opportunities.

Prior to the start of the FeSBE meeting in Aguas de Lindoia, Wagner, Sieck, and Frank met with the leadership of the BSP including Ubiratan Fabres Machado (President), Aldo B. Lucion (President-elect), Celso Rodrigues Franci, Maria Tereza Nunes, Candido Celso Coimbra, Vagner Roberto Antunes, Lisete C. Michelini, Thiago S. Moreira, Eliana C. Pinheiro, and Benedito Machado. At the meeting, participants reviewed a draft letter of invitation (to participate in a Pan-American Congress proposed for 2014) to be circulated to the Presidents of all physiological societies in the Americas. It was agreed that Rodrigo Iturriaga, President, Association of Latin American Physiological Societies (ALACF) should be invited to be a co-signer of the letter.

In addition to visiting institutions in Brazil, Wagner, Sieck, and Frank participated in the FeSBE Meeting, a multi-society, multi-disciplinary meeting that attracted approximately 2,300 scientists and students. The APS leadership was greatly impressed by the number and enthusiasm of trainees attending the meeting, as well as the quality of their poster presentations. The majority of attendees were, in fact, trainees, and they included undergraduates, graduate students, and postdoctoral fellows. During the Opening Session, Wagner was invited to sit on stage at the head table along with the Presidents of the FeSBE member societies, offering comments at the behest of the FeSBE President Luis Eugenio Araujo de Moraes Mello. By invitation from Ubiratan Machado, BSP President, Wagner also made similar comments at the business meeting of the BSP. Frank presented a very well-attended session on the topic of "Publishing 101 - The Do's and Don'ts of Publishing" with Sieck and Wagner as discussants. Wagner and Sieck also presented research talks in a session titled "Challenges to the Respiratory Function."

In summary, this was a very productive visit. The APS leadership learned that the Brazilian government is heavily invested in supporting trainees in science, including physiology, and has programs offering financial support to foreigners wishing to come to Brazil for collaboration. It was very clear that the BSP is very keen to develop collaborative ventures both society-to-society and at the level of individual members, and it was agreed that our members would be made aware of these opportunities. There is enthusiasm and approval in concept of a Pan-American Physiology Congress for 2014, and together, BSP and APS will begin the process of organization. The first step will be to invite all physiological societies in North, Central and South America to take part in the Congress planning. ❖

APS Leadership Meets with Physiology Faculty at USP, RP. First Row: Wamberto A. Varanda, Benedito Machado, José Antunes Rodrigues, and Lucila K. Kagohara Elias. Second Row: Norberto Garcia-Cairasco, Martin Frank, Gary Sieck, Hélio C. Salgado, Peter Wagner, Luiz Carlos C. Navegantes, Eliane Comoli, and Rubens Fazan, Jr.

APS and TPS Leadership Meet in Manchester, UK

Members of the APS leadership (Peter Wagner and Gary Sieck) attended the annual meeting of The Physiological Society (TPS) in Manchester at the end of June 2010. Their participation represented a reaffirmation of the collaboration that had developed over the years between APS and TPS. For many years, the *Journal of Physiology,* a TPS journal, has been sponsoring a symposium at the Experimental Biology meeting. In 2009, the APS and TPS signed an agreement to organize a symposium at the other's annual meeting. The APS and TPS also collaborated, along with the Canadian Physiological Society, the Australian Physiological Society, and the Chinese Association for Physiological Sciences to organize the 2008 Beijing Physiology Meeting. The good relations that APS and TPS have enjoyed for some time provided an opportunity for the leadership of both societies to explore ways to strengthen our relationship and to identify ways to benefit physiology internationally.

The agenda for the leadership meeting was full and included a review of existing collaborations, as well as many new opportunities. A primary focus of the discussion was the International Union of Physiological Sciences (IUPS) and the 2013 IUPS Congress which will be hosted by The Physiological Society in Birmingham.

APS and TPS had each previously voiced similar concerns about IUPS, specifically the need for IUPS to express a clear mission that is distinct from that of the supporting societies; the need to create a governance structure that is transparent to and inclusive of the societies that directly or indirectly provide the financial support to IUPS; and a financial plan that is also transparent, up to date, and fair to all societies that are involved in IUPS support. TPS informed the APS leadership that the Royal Society, which pays half the TPS dues for IUPS membership, is likely to stop paying in the near future. It was noted that NSF is likely to do the same for APS and the five other members of the US National Committee of the IUPS. It was agreed that this would likely force the IUPS and the APS and TPS to address the finance issue in the near future.

Prior to meeting with the TPS leadership, Wagner and Sieck had met with Denis Noble, IUPS President, to discuss the future of IUPS. Wagner and Sieck updated the TPS on their discussions with Denis Noble, describing concerns about each of the key areas – mission, governance, and finances. During their discussions, Noble had indicated that he agreed that a concrete governance proposal and mission statement should be the next step. He also expressed a willingness to meet with the APS leadership in Brazil during the FeSBE meeting (due to illness, Noble was unable to attend). The Brazil discussions were viewed as an excellent opportunity for a follow-up discussion focused on the proposals being developed by the IUPS leadership. TPS was interested in participating in the meeting in Brazil if they could identify an appropriate participant. It was agreed that if leadership from any of the other IUPS-supporting societies were in Brazil, they should also be invited to the meeting with Noble. During their meeting in Manchester, the APS and TPS leadership learned that the (small) Dutch Physiological Society has decided to stop paying dues to IUPS. While the Scandinavian and Spanish societies still support IUPS, they appreciated that APS and TPS were having discussions with IUPS about its future.

The discussions re-affirmed the joint agreement to provide a symposium exchange at each of our respective meetings. However, it was also agreed that relations between the APS and TPS could be further enhanced by allowing members of our societies to register as members at the meeting of the other society. TPS was planning to take advantage of this opportunity by requesting guest society status at the Experimental Biology meeting starting in 2011 in Washington, DC, a request that was ultimately accepted. APS members will be able to register as members at the 2011 Physiological Society meeting in Oxford, as well. While the APS and IUPS jointly publish *Physiology,* most physiologists do not receive the journal. In order to extend the reach of the journal beyond APS, an effort is being made to offer *Physiology* to members of the TPS at a reduced price. The TPS leadership was quite interested in this possibility and efforts are being made to implement the option of access to the e-journal for TPS members. Once this option is successfully implemented with TPS, an effort will be made to extend the offer to other IUPS society members.

The leadership also discussed the possibility of collaborating on a new journal designed to build upon the success of *"Physiology in Medicine,"* a series of articles that have most recently appeared in the *Annals of Internal Medicine*. The series is being discontinued with *Annals*, and the APS is looking for alternative ways of continuing what is believed to be a valuable offering. TPS expressed an interest in collaborating with APS on this project, and several models could be imagined from simply identifying and flagging appropriate papers submitted randomly to APS/TPS journals to a full-fledged stand alone journal. A group consisting of staff and members from each society is engaged in ongoing discussions.

As a result of the increasing collaboration between the APS and TPS, it was suggested that the Societies hold an annual leadership meeting. The proposal was made for APS and TPS to host these in alternate years at, and in conjunction with, their annual meetings. There was enthusiasm for this, and agreement in principle on a once a year basis, for a one-day meeting, alternating on British and US soil. The model of embedding before, during or after the annual meeting was preferred to a stand-alone meeting. It was pointed out that next year's TPS meeting at Oxford would have the whole IUPS Executive Committee present, and would be a great opportunity to initiate this retreat and also to continue joint discussions among APS, TPS and IUPS. It was suggested that key committee chairs from both societies be added to the leadership meeting to facilitate discussions of major issues currently before the two societies.

A final item for discussion concerned how the two societies could assist those smaller societies seeking assistance with meetings and other educational activities. As two of the wealthiest physiological societies, both APS and TPS receive numerous requests for assistance. It was agreed that it would be desirable for APS and TPS to work together in order to avoid duplication and to develop stronger, joint programs. It was agreed to share our respective guidelines for handling international societal requests and that when requests were received they would be communicated with each other to determine if collaboration was warranted. The goal was to identify ways in which to make the involvement of the two societies more constructive and beneficial to the host society. ❖

Nebraska Physiological Society Hold 13th Annual Meeting

George J. Rozanski, NPS President

The 13th annual meeting of the Nebraska Physiological Society (NPS) was held on Saturday, September 11, at the Univ. of Nebraska Medical Center's (UNMC) Storz Pavillion in Omaha, NE. Attendance at the meeting totaled 85 registered individuals, including undergraduate and graduate students, postdoctoral fellows, faculty members and high school teachers. Thirty-five research posters from six research institutions were presented. The meeting began at 8:30 a.m. with welcome and introductory remarks from George J. Rozanski, NPS President and Professor, Dept. of Cellular and Integrative Physiology, UNMC. Rozanski thanked this year's sponsors for their support, including the American Physiological Society and the Dept. of Cellular and Integrative Physiology, UNMC. Corporate sponsors were Data Sciences International, North Central Instruments, and VisualSonics, Inc. Finally, Rozanski especially thanked the staff of the Dept. of Cellular and Integrative Physiology UNMC for their help and support during his presidency: Cindy Norton, Pearl Sorensen, Janine Wilson, and Debra Davis.

Brian Derby - Lee Zucker undergraduate presentation.

The scientific portion of the meeting began with the research keynote address by Hannah V. Carey, Univ. of Wisconsin School of Veterinary Medicine. The title of Carey's presentation was "Unraveling Mysteries of Hibernation: From Basic Science to Biomedical Application."

Carey's address was followed by alternate sessions, the Lee Zucker Featured Student Presentations and the Teacher's Breakout Session. The Lee Zucker Featured Student Presentations were presented by two undergraduate students, Brian Derby, Univ. of Nebraska-Lincoln and Sumit Kar, Creighton Univ., and two graduate students from UNMC, Ryan Cornelius and Jing Yang. The speakers were selected to present their research projects based on the quality of their submitted abstracts. Each student was allowed a 15-minute session followed by a short question/answer period.

Thomas A. Pressley, Professor, Texas Tech Univ. Health Sciences Center and David Holtzclaw, Instructor, UNMC, facilitated the Teacher's Breakout session. This year, the teacher's breakout session was a discussion on inquiry-based learning and how to implement it into the K-12 classroom.

NPS members interacting at the poster presentation.

Keynote Speaker Thomas Pressley, with teacher breakout session.

Lunch Table Topic Discussions.

2010 High School Students at NPS.

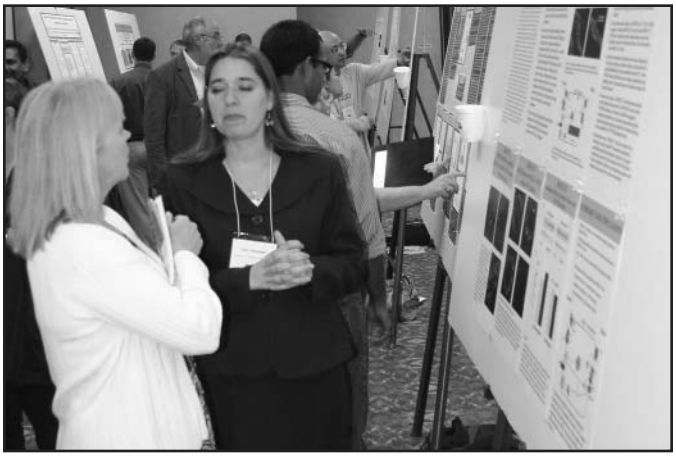
Following the alternate sessions, Pressley presented the educational keynote address entitled, "Teaching and Learning Physiology with the 'I-Phone' Generation."

Pressley's address was followed by a luncheon and Table Topic Talks. This element during the luncheon was new to the NPS meeting. A total of nine topics were designated this year. Students and postdoctoral fellows signed up for their topic of interest during registration. Irving Zucker and Matthew Zimmerman chaired the topic, "Pipes and Pumps: What's New in Cardiovascular Physiology." Pressley chaired, "What I Should Expect from My PhD/What Others Expect from Me Being a PhD." Pamela Carmines and Yulong Li chaired, "What Do Kidneys and Lungs Have in Common." Hannah Carey chaired, "How Can Physiologists Contribute to Global Change Research." Keshore Bidasee chaired, "Life After Getting a PhD."

Graduate students Erin Rosenbaugh and Tamra Llewellyn chaired the topic, "So You Are Thinking About Grad School." High school teachers Ed & Lee Brogie chaired, "Engaging K-12 Students in Science Research." William Mayhan chaired, "Getting Published," and Steven Sansom chaired the topic, "Strategies for Writing Successful Grants."

The afternoon portion of the meeting began with poster viewing and judging. Posters were judged in undergraduate and graduate categories from institutions that included: Univ. of Nebraska at Lincoln, UNMC, Creighton Univ., Univ. of Nebraska at Omaha, Univ. of South Dakota, and Wayne State College. Each poster was critiqued by three faculty members. The change to the poster judging this year was that the presenters were not ranked. They only received the comments from the judges.

Following the poster viewing and judging, Rozanski called the NPS business meeting. Carmines presented an update on the state of the American Physiological Society (APS). She highlighted current programs and strategic goals of the parent society. Holtzclaw highlighted the activities that took place during APS sponsored PhUn Week. Harold Schultz presented an update as NPS representative to the APS Chapter Advisory Committee. Hong Sun briefly presented the current financial status of NPS. Cindy R. Norton, Executive Director and Administrator, Dept. of Cellular and Integrative Physiology, UNMC, noted that two Certificates of Deposit would



NPS members interacting at the poster presentation.



2010 Undergraduate Students at NPS.


2010 Graduate Students at NPS.

be matured in November 2010. Norton suggested reinvesting these CDs for additional year. Rozanski called for a vote. Motion passed. Rozanski then presented certificates to all the teachers, certificates and gift cards to poster presenters in high school, undergraduate and graduate categories. Gift cards were $50 for the Lee Zucker Featured Student Presenters and $25 for the rest of the presenters. APS donated $500.00 and the NPS donated $250.00 for the awards.

Rozanski presented a plaque to NPS Past-President, Kaushik P. Patel, Dept. of Cellular and Integrative Physiology, UNMC, in recognition of his innovative efforts in planning the 2009 NPS Meeting.

The NPS business meeting reconvened and Rozanski declared the election results for NPS 2011-2012 President and NPS 2010-2011 Councilors. Rozanski then introduced G. Patrick Lambert, Dept. of Exercise Science, Creighton Univ. as the incoming 2010-2011 NPS President. Rozanski again thanked this year's sponsors for their support and thanked all individuals who helped make the meeting a success.

The meeting attracted students from institutions throughout the state and promoted interdisciplinary contacts among individuals interested in the physiological sciences at the local level. It also highlighted goals and teaching activities on education for science teachers.

2010-2011 Officers:
President: G. Patrick Lambert
President-Elect: Barbara Engebretsen
Past President: George J. Rozanski
Secretary/Treasurer: Hong Sun
Councilor: J. David Holtzclaw
Co-Councilor: Yifan Li
Co-Councilor: Carol Fassbinder-Orth
Councilor: Keshore R. Bidasee
Student Councilor: Anindit Mikherjee

The meeting concluded at 4:30 PM. ❖

Hong Sun
NPS Secretary / Treasurer


G. Patrick Lambert, 2011 NPS President.


2010 Postdoctoral Research Fellows at NPS.

15th Annual Meeting of the Iowa Physiological Society

The 15th Annual Meeting of the Iowa Physiological Society (IPS) was held on October 9, in the Olsen Medical Education Center on the campus of Des Moines Univ. in Des Moines, IA. Attendance at the meeting totaled 71 individuals from institutions within Iowa and Minnesota. A total of 29 posters from nine different institutions, five of which were primarily undergraduate institutions, were presented.

The meeting began with brief welcome and introductory remarks from the current IPS President, Julia Moffitt, Department of Physiology & Pharmacology, Des Moines Univ. Moffitt noted the record attendance and thanked the attendees for submitting abstracts and coming to the meeting. The meeting's theme this year was "Something for Everyone," and, thus, the meeting included a morning session specifically devoted to teaching physiology and an afternoon session specifically devoted to research in physiology.

The first speaker was the APS Keynote Lecturer, Stephen DiCarlo who presented "Too much content not enough thinking and too little FUN!" DiCarlo gave an engaging, energetic and thought provoking lecture in which the main message was that our current teaching methodology emphasizes memorization and does little to stimulate thought and critical thinking. This talk was followed by two shorter presentations by local physiologists, Jackie Brittingham, Simpson College, and Jeannine Matz, Mercy College of Health Sciences. These two talks were also oriented toward teaching methodology. The teaching sessions generated a great deal of discussion and were very well attended.

Following a break, during which poster viewing and judging for poster competition commenced, Irving Zucker delivered the DSI-sponsored Keynote lecture "The Regulation of Central Angiotensin Receptor Expression in Heart Failure." Lunch immediately followed and the poster competition and viewing continued.

Kristen Sprock presents her poster while graduate division judges, Ron Torry, Past-President IPS, Kim Tran and David Kline listen.

In the afternoon session, David Kline, Univ. of Missouri, delivered the IPS Keynote Lecture in Research in Physiology titled "Opening the gates to reflex control of the cardiorespiratory system: Importance of the nucleus tractus solitarius and its plasticity." This talk was followed by two shorter talks given by local physiologists, Quang-Kim Tran, Des Moines Univ. and Joshua Selsby, Iowa State Univ.

The afternoon break was dedicated to completing poster viewing and judging for the poster competition and compiling scores from the judges. The break was followed by a special topic session on current trends in issues related to animal research in which Kevin Kregel, Univ. of Iowa and current chair of the Animals in Research and Education subcommittee for FASEB, presented "Current scientific, regulatory and legislative issues related to animals in research and education."

The meeting concluded with a raffle drawing and poster competition award presentations. The winner of the graduate division received a $100 travel award and a framed certificate while runners up received a framed certificate. The graduate division award winners were as follows:

- 1st Place: Kathryn C. Welliver from the Des Moines Univ. for her presentation of "Hindlimb unloading results in an increased predisposition to cardiac arrhythmias and changes in

Mike Lyons, IPS President-Elect, presented the awards for the graduate division for the poster completion while Julia Moffitt, IPS President, looks on. Left: Kathryn Welliver, Des Moines Univ., Caleb Masterson, Des Moines Univ. ,and Kristen Sprock, Univ. of Iowa.



Mike Lyons, IPS President-Elect, presented the awards for the undergraduate division for the poster completion while Julia Moffitt, IPS President, looks on. From left, Zeng Su, Grinnell College; Tom Nordstrom, Univ. of Iowa; Kathryn Walder, Grinnell College.

left ventricular connexin43 expression." K.C. Welliver, A.J. Jepson, L.D. Nawrocki, E.R. Garnett, M.K. Henry and J.A. Moffitt

• 2nd Place (tie): Caleb Masterson from the Des Moines Univ. for his presentation of "DHE repression of ATP-mediate sensitization of trigeminal ganglion nociceptive neurons involves activation of alpha2-adrenergic receptors." G.G. Masterson and P.L Durham, Missouri State Univ.

• 2nd Place (tie): Kristen Sprock from the Univ. of Iowa and Veterans Affairs Medical Center for her presentation of "Sex differences in reactive oxygen species generation in sympathetic and sensory neurons: implications for control of sympathetic nerve activity." K. Sprock, S. Flanagan, C.A. Whiteis, F.M. Abboud, and M.W. Chapleau.

The winner of the undergraduate division was awarded for a travel award of $1,000 used to defray costs of attending the Experimental Biology conference in Washington, DC and a framed certificate. This award was sponsored by ADInstruments and the American Physiological Society. Runners up received a framed certificate.

• 1st Place: Zeng Su, Grinnell College, for his presentation of: "PGE2-glycerol, a metabolite of the endocannabinoid 2-arachidonyl glycerol, enhances neurotransmitter release at the vertebrate neuromuscular junction via activation of the TRPV1 receptor." Z. Su and C. Lindgren.

• 2nd Place: Tom Nordstrom, Univ. of Iowa, for his presentation of: "Identification of neurons in the midbrain raphe nuclei that are activated by salt appetite, contain serotonin, and project to the lateral parabrachial nucleus." T. Nordstrom, A.K. Johnson, R. Johnson and T. Beltz.

• 3rd Place: Kathryn Walder, Grinnell College, for her presentation of: "Optimization of complement mediated perisynaptic Schwann cell ablation at the lizard neuromuscular junction." K.K. Walder, Z. Feng, C.P. Ko, and C.A. Lindgren.

Concluding remarks were given by IPS President, Moffitt which included thanking the attendees, speakers, judges, assistants and the IPS board members for their outstanding efforts at helping to make the meeting such a

IPS Secretary-Treasurer, Harald Stauss discusses the meeting with Joshua Selsby, Iowa State Univ.

Pat Finnerty and Mike Lyons, undergraduate poster judges discuss a poster with a student from Simpson College.

American Physiological Society Keynote Speaker Dr. Stephen DiCarlo presents his address: "Too much content, not enough thinking and too little FUN!" to the IPS crowd.

success. Moffitt also thanked our sponsors, DSI, ADInstruments and the APS for their generous assistance allows the IPS to offer the meeting at a low cost to attendees.

The IPS business meeting was held immediately following the conclusion of the conference. Michael Lyons, Kirkwood Community College, was elected President-Elect while Moffitt was elected to be retained as IPS President for the 2010-2011 year. Initial plans were discussed for a joint meeting with the Nebraska Physiological Society with the current NPS president, Pat Lambert, Creighton Univ., who was in attendance. ❖

Julia A. Moffitt
IPS President

DSI-Sponsored Keynote speaker Irving Zucker.

David Kline, IPS Keynote speaker and Kevin Kregel, Special topics speaker, deliver their lectures.

Early Career Professional Service Award

To serve or not to serve? Should that be a question?

Catharine G. Clark

Our world is evolving at an exponential rate thanks to the rapid advancements of science. From nano-scale medical interventions to the introduction of a robot humanoid, improbable has turned possible. However, the price of such an evolution in the scientific field has fallen heavily upon the shoulders of its members. The quest to be a productive researcher requires meeting rigorous demands with schedules intertwining publishing, teaching, advising and the need to lead a balanced life outside of the work place. To many, the thought of adding one more responsibility, such as performing scientific outreach, to an already overwhelming list is a seemingly arduous task. So when posing a question such as "to serve or not to serve?" to a community of scientists, understandably, the majority choose the latter.

One question that, therefore, needs to be addressed is why members of the scientific community should consider participating in outreach. With the constant development of new scientific advancements, there is an alarming disconnect between the scientific field, America's youth and the public alike. It should come as no surprise that the future of America's strength in science, and its continued legacy in scientific contributions, depends on the proficiency of our nation's youth in math and science. However, the gravity of the situation regarding our educational system is at an unprecedented high. Only 29% of American fourth grade students, a third of eighth grade students, and barely 18% of 12th grade students perform at or above a proficient level in science (4). Additionally two-thirds of high school students enrolled in science have teachers who did not major in the subject in college or are not certified to teach it (6). Thankfully, troubling statistics like these have not gone unnoticed. In fact, at a recent National Academy of Sciences press conference, President Obama announced new initiatives for science research and education and challenged scientists to use their knowledge to think about creative ways to engage young people in science and engineering to improve student achievement in these fields (5).

Catharine G. Clark

It is in this regard that scientists, beginner and established, should take heed. It is imperative that we recognize the unique and influential position we are in to impart knowledge where it is needed most. This does not imply that scientists should assume the responsibility of educating America's youth themselves. Nor does it imply that scientists need to shoulder the burden to reform science education. Rather, combining forces with educators, powerful partnerships can begin to form (1). While most researchers may not be equipped with all of the tools or skill sets that trained educators are, they do possess a wealth of knowledge that may be lacking on the educators' behalf. Any effort in outreach participation by scientists may begin to close the apparent gap between the two professions. Taking advantage of nationally organized events that offer boundless resources and provide information on schools willing to participate in such collaborations (e.g. Brain Awareness Week and PhUn Week*), are excellent examples of how to begin participating in K-12 education outreach.

In addition to aiding the youth, the true value of outreach is that it can transcend age groups and reach all corners of society. In this regard, similar to the attention the nation's deficit in science education has drawn, much attention has been placed on the public's lack of scientific literacy. Alarming statistics have revealed that less than 7% of American adults are scientifically literate (3). Rooted in the understanding of general science principles, scientific literacy is an imperative skill for the public to possess, especially when evaluating information relayed by the media. One needs to merely glance through local and national headlines to realize the impact scientific discovery has on shaping our world. Therefore, to fully participate in modern-society, the public needs to make informed choices based on, at times, advanced scientific concepts without falling prey to common scientific misconceptions (2). Forming partnerships with local community centers such as the public library or retirement homes for example, can create an opportunity for scientists to interact with the public and provide open discussion forums on important current topics. Here scientists can aid the public's understanding of critical scientific concepts and in the process, translate a sometimes difficult and jargon-filled vocabulary.

In order to successfully inform the public and youth alike however, it is important to realize that scientists need to receive the necessary training to teach and communicate effectively with their targeted audiences. These words echo similar sentiments conveyed in the article written by Dr. Stephen DiCarlo, Arthur C. Guyton Educator of the Year (1). Understanding one's audience and developing the critical skills to be able to disseminate information in an engaging way is essential. Much like teaching, while scientists may have the necessary scientific knowledge to share, if not conveyed in the appropriate manner, the message will simply land on deaf ears. The task of making something ordinary in a scientist's life—extraordinary in the eyes of students and the public—is no small feat, but one that will undoubtedly be rewarding in itself. Taking advantage of science education workshops and classes or attending teaching related conferences may be outside of a researcher's realm, but are examples of how scientists can better prepare themselves to face the

challenge of captivating and educating public and student audiences.

To those who chose to enter this profession, we are of a privileged community—one in which members receive extensive training and, hence, a wealth of scientific knowledge. However, by keeping this knowledge confined to ourselves and within our field, science becomes self-indulgent. Rather, we need to shift our focus to serve society as a whole. When this occurs, science becomes self-rewarding. Performing outreach will afford you the experience in which you realize you have contributed to something on a much greater scale—one in which the rewards transcend simply donating a few hours of valued time and one where you will never have to ask whether performing service should ever be a question. ❖

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4. National Center for Education Statistics: http://nces.ed.gov/pressrelease/reform/
5. Obama press conference: "Educate To Innovate" Campaign for excellence in science, technology, engineering and math (STEM) education
6. "Rising Above the Gathering Storm" report by the National Academies

*Brain Awareness Week: http://www.sfn.org/index.aspx?pagename=baw_home
PhUN Week: http://www.phunweek.org/.

New Regular Members

***Transferred from Student Membership (5)**

Richard Andersen
California Inst. of Tech.
Sharon L. Ashworth
Univ. of Maine
Tanja Babic
Penn State Univ., Hershey
Naresh Chandra Bal
Ohio State Univ.
Denise D. Belsham
Univ. of Toronto, ON, Canada
Karen Francoise Bernard
Emory Univ., GA
Marie Sinthiya Bhanugopan
Charles Sturt Univ., Australia
Derek Boerboom
Univ. of Montreal, QC, Canada
Sihem Boudina
Univ. of Utah
Birger Brodin
Univ. of Copenhagen, Denmark
Tepmanas Bupha-Intr
Mahidol Univ., Bangkok, Thailand
Raymond Franklin Burk
Vanderbilt Univ., TN
Michael Todd Butcher
Youngstown State Univ., OH
Chandrasekar Bysani
Tulane Univ., LA
Carl-Johan Carlhall
Linkoping Univ., Sweden
Geoff Chandler
Aurora Scientific Inc., ON, Canada
Gang Chen
M.D. Anderson Cancer Ctr., Houston, TX
Qamrul Ghani Choudhury
Rocky Vista Univ., CO
Man-Kyo Chung
Univ. of Maryland Dental Sch.
Mirela Delibegovic
Univ. of Aberdeen, UK
Joseph F.X. Desouza
York Univ., ON, Canada
Allison Elizabeth Devan*
Univ. of Colorado, Boulder
Mauricio Di Fulvio
Wright State Univ., OH
Peter Francis Drain
Univ. of Pittsburgh, PA
Rachel Claire Drew
Pennsylvania State Univ., Hershey
Daniela A.M. Eichenberger
Univ. of São Paulo, Brazil
Mohamed Hassan Elsayed
Ain Shams Univ., Cairo, Egypt
Liang Fang
Univ. of Maryland, Baltimore
Marcel Filoche
Ecole Polytechnique, France
Philip Pierre Foster
Univ. of Texas Medical Branch
Andre H. Freiria-Oliveira
Paulista State Univ., Brazil
Seyed Ali Gaskari
St. Louis Univ., MO
Evgenia V. Gerasimovskaya
Univ. of Colorado
Todd Gillis
Univ. of Guelph, Canada
Tommaso Gori
Univ. Med. Ctr., Mainz, Germany
Nanami Gotoh
Yale Univ., CT
Zoran Grubic
Univ. of Ljubljana, Slovenia
Kenneth Harris
Imperial College, London, UK
Christopher M. Hearon
Texas A&M Univ.
Takahiro Hemoto
Nippon Med. Sch., Tokyo, Japan
Jennifer Herman
Rocky Vista Univ., Parker, CO
Rob H. Hilgers
Univ. of Alabama, Birmingham
Sandrine Horman
Univ. of Louvain, Brussels, Belgium
Ryan Thomas Hurt
Univ. of Louisville, KY
Rajiv Janardhanan
Mayo Clinic, MN
Thomas L. Jetton
Univ. of Vermont
Amit Arvind Joharapurkar
ZYDUS Res. Ctr., Ahmedabad, India
Keven Robert Johnson
Univ. of Illinois, Chicago
Anthony D. Kay
Univ. of Northampton, UK
Annette R. Khaled
Univ. of Central Florida
Jae-Sung Kim
Univ. of Florida, Gainesville
Severine Kirchner
Univ. of California, Irvine
Peter Kirkwood
UCL Inst. of Neurology, London, UK
Angela Kohler
Univ. of Pretoria, South Africa
David Kump
Winston-Salem State Univ., NC
Stan C. Kunigelis
Lincoln Memorial Univ., TN
Ngai Chin Lai
Univ. of California, San Diego
Matthew J. Laye*
Rigshospitalet, Copenhagen, Denmark
Claude J. Le Saux
Univ. of Texas HSC, San Antonio
Guillermo L. Lehmann*
Inst. Fisiol. Exper., Santa Fe, Argentina
Robert C. Liu
Emory Univ., GA,
Elizabeth L. Loreaux*
FDA, Cincinnati, OH
Jonathan Wayne Lowery*
Harvard Univ., MA
David Christian Lyon
Univ. of California, Irvine
Anna P. Malykhina
Univ. of Pennsylvania Sch. Med.
Andrew Edward McKechnie
Univ. of Pretoria, South Africa
Gerhard Meissner
Univ. of North Carolina, Chapel Hill
Jenna A. Monroy
Northern Arizona Univ.
Jean-Luc Morel
CNRS, Paris, France
Suresh D. Muthukumaraswamy
Cardiff Univ., Cardiff, UK
Adam K. Myers
Georgetown Univ., Washington, DC
Shizuko Nagao
Fujita Health Univ., Aichi, Japan
David W. Nauen
Johns Hopkins Univ., MD
Kyuichi Niizeki
Yamagata Univ., Yonezawa, Japan
Sheriff Lekan Ojulari
Univ. of Ilorin, Nigeria
James Michael Olcese
Florida State Univ.
Dervla O'Malley
Univ. College Cork, Ireland
Albert Ong
Univ. of Sheffield, UK
Brad Palmer
Univ. of Vermont, Burlington
Roberta L. Pohlman
Wright State Univ., OH
Feng Qiu
Univ. of Miami, FL
Jayalakshmi Ramachandran
Univ. of Med. and Dent. of NJ
Maria Isabel Ramirez
Boston Univ., MA
Raymond Reynolds
Univ. of Birmingham, UK
Rachael Rigby
Lancaster Univ., UK
Peter Stanley Rowe
Univ. of Kansas Med. Ctr.
Yogesh Saini
Univ. of North Carolina, Carrboro
Jennifer Sherwood
Holy Names Univ., Oakland, CA
Maurice A. Smith
Harvard Univ., MA
Dariusz Tomasz Stepniak
La Jolla Inst. of Allergy and Immun., CA

Sylvia Ottilie Suadicani
Albert Einstein Coll. of Med., Bronx, NY
Elizabeth Barbara Torres
Rutgers Univ., NJ
Lloyd A. Trueblood
La Sierra Univ., CA
Hui Yin Tu
Univ. of Nebraska Med. Ctr.
Mikko Vähäsöyrinki
Univ. of Oulu, Finland
Timothy Dennis Verstynen
Univ. of Pittsburgh, PA
David Waddell
Univ. of North Florida
Jieru Wang
National Jewish Health, Denver, CO
James N. Warnock
Mississippi State Univ.
Stanley John White
Ross Univ. Med. Sch., Dominica
Susan L. Whittemore
Keene State College, NH
Michael Eric Widlansky
Med. College of Wisconsin
Qun Wu
National Jewish Health, Denver, CO
John P. Wysocki
Common Med. College, Scranton, PA
Mitsuhiko Yamada
Shinshu Univ. Sch. of Med., Japan
Alexey V. Zaitsev
Univ. of Utah
Shao-Ling Zhang
Ctr. Hosp. L'Univ. Montréal-Hotel Dieu, Canada
Mei J. Zhu
Univ. of Wyoming, Laramie
Karmin Zouaoui-Boudjeltia
Univ. of Libra De Bruxelles, Belgium

New Graduate Student Members

Johannes Aartun
Univ. of South Carolina
Yaria Arroyo
Univ. of Puerto Rico
Sarah Elizabeth Baker
Univ. of Arizona
Bryan Becker
Univ. of Nebraska Med. Ctr.
Jennifer Ann Benton
Univ. of Nebraska
Jessica A. Bernard
Univ. of Michigan
Sayak Bhattacharya
Virginia Commonwealth Univ.
Cosme Franklim Buzzachera
Univ. of Rome, Italy
Ryan James Cornelius
Univ. of Nebraska Med. Ctr.
Georgina K. Cox
Univ. of British Columbia, Canada
Mads Damkjaer
Inst. of Molecular Med., Denmark
Robert Patrick Davis
Michigan State Univ.
Travis Matthew Doggett
Lousiana State Univ., HSC
SimIat O. Elias
Lagos St. Univ., Nigeria
Amy Eengevik
Univ. of Cincinnati, OH
Reza Farajian
New York Univ., School of Medicine
Di Feng
Medical College of Wisconsin
Monica Gochioco
Univ . of Toledo, OH
Jody L. Greancy
Univ. of Delaware
Adrienne Grzeda
Mayo Clinic, MN
Tracie Lynn Haines
Appalachian State Univ., NC
Thiago Gomes Heck
Univ. Fedl Do Rio Grande Do Sul, Alegre
Michael Ryan Hicks
Arizona State Univ.
David W. Howell
Texas A&M HSC
Chun-Chun Hsu
Univ. of Kentucky
Anthony C. Johnson
Univ. of Oklahoma HSC
Jeremy Michael Johnson
Univ. of Mississippi
Hui Yin Ler
Univ. of Sydney, Australia
Graham Ripley McGinnis
Auburn Univ., AL
Thuy Nguyen
Vanderbilt Univ., TN
Jessica Priestley
Michigan State Univ.
Laura Lucia Prieto Godino
Univ. of Cambridge, UK
Bill Pryor
Univ. of Georgia
Brett Rabeneck
Georgia Southern Univ.
Lauren Reynolds
Univ. of Nevada, Las Vegas
Markus M. Rinschen
Münster Med. Sch., Germany
Loredana Serafini
California Poly Tech. Univ.
Wei Shi
NEOUCOM, OH
Zoya Tahergorabi
Isfahan Univ. of Med Sci., Iran
Rui Tang
Univ. of Georgia
Ashlee J.Tipton
Medical College of Georgia
Magdalene Trzcionka
Univ. of Wollongong, Australia
Serge Amani Yao Afeli
Univ. of South Carolina

New Undergraduate Student Members

Joyonna Gamble-George
Nashville, TN
Arshavir Samuel Ghahramanyan
Yerevan State Med. Univ., Armenia
Zachary Ian Grunewald
Florida State Univ.
Joseph Elmer Jacobson
Univ. of Wisconsin, Madison
Mariana R. Lauar
Univ. Estadual Paulista, Brazil

New Affiliate Members

Sharon Holthaus
Madison College, WI
James Timothy Miller
Elite Personal Trainer Sport & Hlth., Wash., DC
Amity Peterson
Midwestern Univ., AZ

2010 APS Conference: Inflammation, Immunity and Cardiovascular Disease
Westminster, CO, August 25-28, 2010

The 2010 APS Conference: Inflammation, Immunity and Cardiovascular Disease, was held in suburban Westminster, CO. Attendees were not only treated to the spectacular views of the Rocky Mountains, but also to ample trails, shopping, dining and the close proximity of the cities of Denver and Boulder. The conference took place over four days at the Westin Westminster Hotel. The Organizing Committee was chaired by David Harrison, Emory Univ. School of Medicine and Co-Chair, David Pollock, Medical College of Georgia. In addition, Jorg Goronzy, Emory Univ. School of Medicine; Tomasz Guzik, Jagiellonian Univ. School of Medicine, Poland; Ernesto L. Schiffrin, McGill Univ., Canada; and Cornelia Weyland, Stanford Univ. School of Medicine made up the rest of the organizing committee and were instrumental in helping set up a successful conference program. The committee organized a program that would include symposia, oral presentations for students and postdoctoral fellows, interactive poster sessions, and social networking opportunities to make this conference a valuable experience for those who attended.

The conference was attended by 143 total registrants: 23% of registrants were represented by trainees, including 14 postdoctoral and 18 students. Forty-eight (33%) attendees identified themselves as APS members, and 37 (26%) registered as non-members; invited chairs and speakers made up twenty-three (16%) attendees, while the remaining 2% was made up of exhibitors for the event. Table 1 shows the breakdown of the different registration types. This conference also attracted a large group of registrants from outside the United States. Of the 143 registrants, 24 (17%) represented countries from Australia, Austria, Chile, China, Germany, Japan, Nigeria, Poland, South Korea, The Netherlands, Sweden, and the United Kingdom.

Conference attendees interacting during a poster session.

The conference program consisted of one key note lecture and five symposia on a wide variety of topics related to inflammation, immunity, and cardiovascular disease. The audience was encouraged to share their ideas and thoughts with the speakers at the end of their talks which often prompted a very animated discussion period. During the conference there were three oral presentation sessions which allowed a selected number of registrants the opportunity to present their abstract in an oral presentation format. In addition to the symposia and oral presentation sessions, there were two well-attended poster sessions and a Career Workshop designed to engage and encourage students and postdoctoral fellows in writing and data skills. The conference also had several social activities, including a Welcome and Opening Reception, which was designed to give attendees a chance to meet with long-time colleagues, create new friendships, and enjoy some hot and cold hor d'oeuves and beverages

Table 1. Registration Statistics

Registrant Type	**Number of Attendees** (%)
APS Member	48 (33%)
Nonmember	37 (26%)
Postdoctoral	14 (10%)
Student	18 (13%)
Invited Chairs/Speaker	23 (16%)
Exhibitors	3 (2%)
Total	**143**

Conference attendees enjoying the Welcome and Opening Reception.

while enjoying the beautiful weather and scenery. Furthermore, the conference schedule allowed for some free time during the afternoon so that registrants could get together and explore the surrounding area.

A total of 89 abstracts were submitted for the conference. Of these abstracts, 72 were programmed as poster presentations. Out of the 72 volunteered abstracts that were programmed as posters, 14 abstracts were selected for an oral presentation. The remaining 17 abstracts submitted for the conference were by invited speakers. Of the abstracts submitted for the conference, 16 (18%) were submitted by a female first author; 20 (22%) were submitted from institutions outside of the United States, including a total of nine abstracts from China, Japan and South Korea, five abstracts from the European nations of Austria, Germany, Poland and Sweden, two from South America, including abstracts from Chile and Venezuela. The remaining abstracts came from Australia and Canada.

Before the last evening session began on Friday, Harrison and Pollock presented travel awards to four postdoctoral fellows and students who were recognized as the recipients of the Research Recognition Award for Outstanding Abstract by a Graduate Student or Postdoctoral Fellow. The following individuals were presented with a certificate and cash prize: Kyle Diehl, Univ. of Colorado, Boulder; Antony Vinh, Emory Univ.; Kedra Wallace, Univ. of Mississippi Medical Center; and Brian Weil, Univ. of Colorado; Boulder.

In addition Elinette Albino, Ponce School of Medicine; Ashlee Tipton, Medical College of Georgia; Nekeithia Wade, Vanderbilt Univ.; and Justin Wilson, Howard Univ., were the recipients of the Porter Physiology Development Committee's Minority Travel Fellowship Award, which is provided to encourage participation of under-represented minority students in the physiological sciences. With support from the National Institutes of Diabetes and Digestive and Kidney Diseases (NIDDK), the fellowship provides reimbursement of all expenses associated with travel and participation in the conference. The recipients of the award were matched with APS members: Paula Pullen, Morehouse School of Medicine; Tracy Baynard, Univ. of Illinois, Urbana-Champaign; Michael Ryan, Univ. of Mississippi Medical Center; and Eric Belin de Chantemele, Medical College of Georgia, who offered guidance and made introductions to the other scientists.

The American Physiological Society and the Organizing Committee gratefully acknowledge the financial support provided though generous educational grants from NIH, National Institutes of Diabetes and Digestive and Kidney Diseases, Genentech, Inc., DMT-USA, Inc. and Boehringer Ingelheim Pharmaceuticals, Inc. ❖

Conference Organizers congratulate the winners of the 2010 APS Abstract Travel Award. L-R: David Harrison, Emory Univ., Brian Weil, Univ. of Colorado, Boulder, Kyle Diehl, Univ. of Colorado, Boulder, Kedra Wallace, Univ. of Mississippi Med. Ctr., Antony Vinh, Emory Univ., and David Pollock, Med. College of Georgia.

2010 APS Intersociety Meeting

Global Change & Global Science: Comparative Physiology in a Changing World

August 4-7, 2010, Westminster, CO

The 2010 APS Intersociety Meeting "Global Change & Global Science: Comparative Physiology in a Changing World" was held in the city of Westminster, CO, which is approximately 16 miles northwest of Denver. The meeting took place over four days at the Westin Westminster hotel located close to the majestic Rocky Mountains, the historic city of Boulder and numerous trails, shopping and dining amenities. The Organizing Committee, chaired by Jon Harrison, Arizona State Univ., included Siribhinya Benyajati, Univ. of Oklahoma Health Science Center, Andrew Biewener, Harvard Univ., David Goldstein, Wright State Univ., Carlos Martinez del Rio, Univ. of Wyoming, Don Mykles, Colorado State Univ., Hans-O Pörtner, Alfred Wegener Institute, Germany and Patricia Schultz, Univ. of British Columbia, Canada. The organizers worked together to select the wide array of different symposia, plenary lectures, speakers, topics, social networking opportunities and award competitions in order to make this meeting exciting and productive for the attendees.

The conference was attended by 254 total registrants, of whom, 39% were represented by young scientists, including 29 postdoctoral and 71 students. Thirty-eight attendees were APS members (15%), and 32 attendees were non-members (13%), 78 (31%) were invited chairs and speakers, and exhibitors made up the remaining 6 registrants (33%). Table 1 shows the breakdown of the different registration types. The meeting program also attracted a large group of registrants from outside of the United States. Out of the 254 registrants, 67 (26%) represented countries from Australia, Bahrain, Brazil, Canada, Denmark, Egypt, France, Germany, Israel, Japan, Monaco, Norway, South Africa, Sweden, The Netherlands and the United Kingdom.

The meeting program consisted of a series of concurrent symposia each morning on a wide variety of topics. The audience was encouraged to share ideas and thoughts with the speakers at the end of their talks. Each afternoon the attendees were invited to participate in the workshops and attend the oral presentations of their peers and support those who were competing in the prestigious Scholander Award oral presentation completion. The meeting had two plenary lectures scheduled, including the opening plenary lecture presented by George Somero, Stanford Univ. and Elizabeth Brainerd who presented during the Closing Banquet and Awards event. The meeting also had several social activities including a Welcome and Opening Reception, which gave the attendees a chance to meet with long time colleagues and enjoy some hors d' oeuvres and wine. There were also three poster sessions where scientists presented their work, were able to network with colleagues, and peruse the booths in the exhibit area.

A total of 226 abstracts were submitted for the meeting. Of these, 120 were programmed as poster presentations. Twenty-six abstracts were programmed in oral presentation sessions. The remaining 80 abstracts were submitted by invited speakers. Of the abstracts submitted for the meeting, 72 (32%) had a female first author; 63 (28%) were submitted from institutions outside of the United States, including 28 from Canada, 14 from Europe, 8 from Brazil, 6 from Israel, as well as abstracts from Australia, Japan and South Africa.

On Saturday evening, Harrison hosted the Banquet and Awards Presentation dinner, which also included an evening dinner lecture presented by Elizabeth Brainerd. Attendees gathered at the Westin Westminster ballroom for evening dinner, wine and conversation with new and old colleagues. After much anticipation, the winner of the highly competitive Scholander Competition was announced. The first place winner was Caroline Williams from the Univ. of Western Ontario, Canada. Williams received a certificate, waived registration, and a cash

2011 Scholander Awardee, sponsored by CEPS, GlaxoSmithKline, and supplemented by PBZ; Jon Harrison, Caroline Williams, Patricia Schulte-UBC representing PBZ, and David Goldstein.

Best Poster Awardee-sponsored by Sable Systems: Sable Systems Representative, Barbara Joos, Rudolf Schilder, and Gretchen Hofmann, Chair Poster Judging Committee.

Outstanding Oral presentations, sponsored by John Wiley: Jon Harrison, awardees Catalina Reyes, Jessica Meir, Patricia Schulte, and David Goldstein.

prize. Two runner-ups were also recognized for their work presented in the competition, including Jessica Meir and Catalina Reyes, both from the Univ. of British Columbia, Canada. Each runner-up received a cash prize and a certificate.

In addition to the Scholander Competition the students and postdoctoral fellows competed in a Best Poster Competition. Applicants presented their poster in their assigned session and then 10 finalists were selected to present again on the last day of the meeting. The winner of the Best Poster Competition went to Rudolf Schilder of Pennsylvania State Univ. College of Medicine who received a certificate and a cash prize. Special thanks to Gretchen Hoffmann, Univ. of California, Santa Barbara and the Best Poster Competition Committee for their hard work in judging all of the posters during the meeting.

There were 29 recipients of the Research Recognition Award for Outstanding Abstract by a Graduate Student or Postdoctoral Fellow presented during the award ceremony. The following awardees were presented with a certificate and cash prize: Inbal Brickner-Braun, Ben Gurion Univ. of the Negev, Israel; Cindy Canale, CNRS-MNHN-CF, Brunoy, France; Hayley Carter, San Francisco State Univ.; Lina Ceballos, San Francisco State Univ.; Xi Chen, San Francisco State Univ.; Tyler Evans, Pacific Biological Station, British Columbia, Canada; Alexander Gerson, Univ. of Western Ontario, Canada; Erica Heinrich, Arizona State Univ.; Martin Jastroch, Buck Institute for Age Research, Navato, CA; Amanda Kelley, Portland State Univ.; Gigi Lau, Univ. of British Columbia, Canada; Katie Marshall, Univ. of Western Ontario, Canada; Marshall McCue, Jacob Blaustein Institute for Desert Research, The Negev, Israel; Haydee Medina-Ruiloba, San Francisco State Univ.; Nathan Miller, San Francisco State Univ.; Linnea Pearson, Univ. of Alaska, Anchorage; Subhash Rajpurohit, Univ. of Nevada, Las Vegas; Lauren Reynolds, Univ. of Nevada, Las Vegas; Julie Reynolds, Ohio State Univ., Pascale Rossignol, San Francisco State Univ.; Koji Sato, Ritsumeikan Univ., Kusatsu, Japan; Rudolf Schilder, Pennsylvania State Univ. College of Medicine; Loredana Serafini, Cal Poly San Luis Obispo; Jonathan Stecyk, Univ. of Oslo, Norway; Jennifer Sunday, Simon Fraser Univ., Canada; Martin Tresguerres, Weill Cornell Medical College; John VandenBrooks, Arizona State Univ.; James Waters, Arizona State Univ.; and Jonathan Whiteman, Univ. of Wyoming.

Abstract Travel Awardees (Group 1) pose for a photo after receiving their award certificates.

Table 1. Registration Statistics

Registrant Type	Number of Attendees (%)
APS Member	38 (15%)
Nonmember	32 (13%)
Postdoctoral	29 (11%)
Student	71 (28%)
Invited Chairs/Speaker	78 (31%)
Exhibitors	6 (2%)
Total	**254**

Finally, Jinae Bartlett, California State Univ., Elyse Munoz, Arizona State Univ., Jose Pablo Vazquez-Medina, Univ. of California, Merced and Jose Viscarra, Univ. of California, Merced were the recipients of the Porter Physiology Development Committee's Minority Travel Fellowship Award, which is provided to encourage participation of under-represented minority students. With support from the National Institutes of Diabetes and Digestive and Kidney Diseases (NIDDK), the fellowship provides reimbursement of all expenses

associated with travel and participation in the conference. The recipients were matched with the following APS members: Paul Schaeffer, Miami Univ.; Melanie Frazier, U.S. Environmental Protection Agency; Michael Dillon, Univ. of Wyoming; and Gregory Florant, Colorado State Univ., who were attending the conference, offered guidance and made introductions to the other scientists.

The American Physiological Society and the Organizing Committee gratefully acknowledges the financial support provided through generous educational grants from NIH-NIDDK, National Science Foundation, The Society for Integrative and Comparative Biology, the European Society for Comparative Physiology and Biochemistry, Sable Systems International, Wiley-Blackwell, Australian & New Zealand Society of Comparative Physiology and Biochemistry, Journal of Comparative Physiology, Journal of Experimental Biology, Physiological and Biochemical Zoology Journal and the American Journal of Physiology: Regulatory, Integrative and Comparative Physiology. ❖

Abstract Travel Awardees (Group 2) pose for a photo after receiving their award certificates.

APS Minority Travel Fellows Attend the 2010 APS Conferences

"Global Change and Global Science: Comparative Physiology in a Changing World" and "Inflammation, Immunity and Cardiovascular Disease"

The APS regularly awards Travel Fellowships for underrepresented minority scientists and students to attend APS scientific meetings with funds provided by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). These Fellowships provide funds for registration, transportation, meals, and lodging. Four travel fellows received funding to attend the 2010 APS Intersociety Meeting, "Global Change and Global Science: Comparative Physiology in a Changing World," from August 4-7, 2010 in Westminster, CO. Four travel fellows received funding to attend the APS Conference, "Inflammation, Immunity and Cardiovascular Disease," from August 25-28, 2010 also in Westminster, CO. The application reviews were led by Committee Member, Johana Vallejo-Elias.

The travel awards are open to graduate students, postdoctoral students, and advanced undergraduate students from minority groups underrepresented in science (i.e., African Americans, Hispanics, Native Americans, and Pacific Islanders). The specific intent of this award is to increase participation of pre- and postdoctoral minority students in the physiological sciences.

Fellows in the NIDDK Minority Travel program not only received financial support to attend this meet-

Travel Fellows and Meeting Mentors at the 2010 Inflammation Meeting.

Fellow	Meeting Mentor
Elinette M. Albino Ponce School of Medicine Dept. of Physiology/Biochemistry	Paula R. Pullen Morehouse School of Medicine Dept. of Physiology
Ashlee Tipton Medical College of Georgia Dept. of Vascular Biology	Tracy Baynard Univ. of Illinois, Urbana-Champaign Dept. of Kinesiology & Community Health
Nekeithia S. Wade Vanderbilt University Dept. of Pathology	Michael J. Ryan Dept. of Physiology & Biophysics Univ. of Mississippi Med. Center
Justin L. Wilson Howard University Dept. of Physiology & Biophysics	Eric Beline de Chantemele Vascular Biology Center Medical College of Georgia

ing, but were also provided professional guidance through pairings with APS members who served as mentors to the Fellows for the duration of the meeting. Thanks to the time and expertise offered by mentor volunteers, Fellows were able to maximize their time and more fully experience the many aspects of this meeting.

For more information, contact Brooke Bruthers, Minority Programs Coordinator, in the APS Education Office at 301-634-7132 or bbruthers@the-aps.org , or visit http://www.the-aps.org/education/minority_prog/index.htm on the APS website. ❖

Travel Fellows and Meeting Mentors at the 2010 Comparative Meeting.

Fellow	Meeting Mentor
Jinae N. Bartlett California State Univ., Long Beach Biological Sciences	Paul Schaeffer Miami Univ. Dept. of Zoology
Elyse Munoz Arizona State Univ.	Melanie Frazier U. S. Environmental Protection Agency; Coastal Ecology Branch
Jose Pablo Vazquez-Medina Univ. of California, Merced School of Natural Sciences	Michael E. Dillon Univ. of Wyoming Dept. of Zoology and Physiology
Jose Viscarra Univ. of California, Merced School of Natural Sciences	Gregory L. Florant Colorado State Univ. Dept. of Biology

Jose Viscarra, Elyse Munoz, and Jose Pablo Vazquez-Medina at the Comparative Meeting.

From Left: D. Harrison (co-chair), A. Tipton, J. Wilson, N. Wade, and D. Pollock (co-chair) at the Inflammation Conference.

APS Science Policy Committee Meets with NIH Chief of Staff

On Wednesday, October 6, 2010, members of the APS Science Policy Committee met with NIH Chief of Staff Dr. Kathy Hudson to discuss NIH efforts to foster translational research and how physiology is well-positioned to help relate findings at the molecular level to functioning at the level of the cell, organ and whole organism level.

Hudson spoke to the committee about the need to bridge the gap between basic and clinical research and outlined some of NIH's plans to address the problem. The NIH is currently developing programs which include the Molecular Libraries Initiative, a Common Fund project that gives researchers access to high-throughput screening capacity to identify small molecules that act on genes, cells and biochemical pathways of interest. The NIH is also engaged in a partnership with the Food and Drug Administration to improve translational research and foster regulatory science to develop new tools to evaluate experimental preventatives, diagnostics and therapies. And as part of the federal health care reform effort, the Cures Acceleration Network (CAN) was created to accelerate the development of cures and treatments for diseases by reducing barriers between laboratory discoveries and clinical trials. CAN will provide funds for research grants, partnership awards (requiring matching dollars) and a flexible research award mechanism that will be similar to the Defense Advanced Research Projects Agency (DARPA) program.

In conjunction with the APS Science Policy Committee meeting on October 7, Committee Chair John Chatham and Loren Wold met with Dana Halvorson, Legislative Assistant to Senator Kent Conrad (D-ND) to discuss the importance of NIH funding..

In conjunction with the APS Science Policy Committee meeting on October 7, Committee Chair John Chatham and Loren Wold met with Graham Smith, Legislative Assistant to Senator Richard Shelby (R-AL), to discuss the importance of NIH funding.

Members of the Science Policy Committee emphasized that physiology has an important role to play in translational research, particularly with our membership's expertise in integrative physiology and whole animal research.

Discussion centered on identifying and focusing resources on those areas that are most in need of investment on the continuum between basic research and clinical application. Committee members also raised concerns about how to train the next generation of researchers to carry effectively out this mission, and the need to ensure that peer review accurately identifies and assesses proposals that focus on translational research.

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Vol. 53, No. 6, 2010

Research, Teaching, Service, and Life: How to Survive and Prosper

Chris Minson

I didn't grow up thinking I was going to be a university professor. In fact, the idea of becoming a professor didn't even occur to me until I had almost completed my Master's degree. Even at that point, I didn't get a sense of the challenges of this career choice until my first appointment as an Assistant Professor. I was aware of the Three Pillars of Professorship…Research, Teaching, and Service, and I knew that each of these areas would compete for my time and energy, particularly at a research institution with a large undergraduate emphasis. How could I enjoy my job, with so many expectations placed upon me? The goal is to find enjoyment in each area and to maintain a reasonable level of balance such that you don't get too consumed in one aspect to the detriment of others. This is not easy to accomplish, but, if you do so, you will ultimately be more successful and happier in all aspects of your life. I have a few suggestions you may find useful as you navigate the challenges of being a tenure-track professor.

Don't waste time worrying about tenure. Plan for it, yes… you know what the expectations are: publish, write grants, do a good job in the classroom, mentor students, perform service. If your motivation for doing a task is to help you make tenure, then you are doing it for the wrong reason or, at least, with the wrong mindset. It is important to know the expectations for tenure at your institution, but your motivation should come from your desire to be good researcher, teacher, and colleague. Your Department Head should be guiding you and providing honest evaluations of your progress and balance in each of the areas. However, it's ultimately your responsibility, so make it an enjoyable experience by doing what interests you.

Surround yourself by people smarter or more talented than you. For me, this has not been difficult! I learned early in my career that by observing and interacting with others, you can identify their skills and reap great benefits from their knowledge or abilities. You will find that collaborations can be very successful, enjoyable, and rewarding. An important aspect to this suggestion is #3.

Chris Minson

Don't get caught-up in being competitive. Often in our training, competitiveness amongst graduate students or post-docs (and even faculty!) is encouraged and sometimes fostered. You have more to lose in being competitive with those around you than you have to gain. No one is successful in a vacuum, so by sharing ideas and working together, rather than competing, you will be more productive and ultimately more successful.

Treat trainees fairly. In fact, more than fair. Be their champion at every opportunity. Support them in every way you can, and balance this with challenging them to improve themselves in all aspects of their life. It's been interesting for me to discover how well I have come to know those I have advised. At different times I served as their teacher, friend, advocate, disciplinarian, confidant. But at the end of the day, their successes are yours, and your successes are theirs.

Always take advantage of an opportunity to learn. This is one of the main reasons why I love being at a university. There are so many exciting things happening and so many opportunities to be engaged in the process of learning. Take advantage of courses, workshops, and seminars. Offer to have a beer or coffee with someone doing work you find interesting, even if it is far from your discipline. You will often find areas of potential collaboration for research or in the classroom.

Become a better instructor. This is an interesting one, because too often we feel that as long as we know the content we are presenting and have a dynamic PowerPoint presentation, we are doing a good job. I am blessed with being in a Department where quality instruction is highly valued, and I am surrounded by truly excellent, caring teachers. Although beyond the scope of this article, there are many relatively easy things you can implement to help students be more engaged in the process of learning (rather than only being focused on copying and memorizing content). The hidden secret about this suggestion is that rather than taking more of your time, this will save you time, you will be a better instructor, and you will have more fun.

Support of family and friends. Being a professor is demanding, and you are constantly being pulled in many different directions. There are

Christopher Minson's research is focused on two areas of cardiovascular physiology in humans. He investigates how natural and synthetic forms of estrogen and progesterone impact cardiovascular health and blood pressure regulation in women, with funding from the National Institutes of Health. He also investigates the neural and vascular interactions and adaptations during thermoregulatory challenges. His teaching emphasis is in the areas of cardiovascular and environmental physiology.

Minson received a BS in Psychology (Univ. of Arizona), a Master's degree in Exercise Science (San Diego State Univ.), and a PhD in Exercise Science (Pennsylvania State Univ.). He trained as a postdoc at Mayo Clinic in the Dept. of Anesthesiology. Minson joined the Dept. of Human Physiology at Univ. of Oregon in 2000. Minson is co-director of the Exercise and Environmental Physiology Labs and serves on the Editorial Boards of the Journal of Applied Physiology *and* Medicine and Science in Sports and Exercise. ❖

times that you will have to work long hours for days, even weeks on end. Having partners and friends who understand your challenges is not only important, it's necessary. But you can't expect them to understand this unless you talk with them. Without their understanding and support, the additional stress you will carry will be debilitating on all fronts. HOWEVER, you must also realize that in your life, family and friends come first. This is non-negotiable. One great aspect of being a professor is that the highly busy times are balanced by slower times. In the slower times, cut your hours back, take days off, give yourself fully to your family and friends. The pathway to success on this one is communication.

"Service" means being a good colleague. In other words, be involved in your department, university, and professional societies (especially APS!) because you care about your colleagues and your profession. What I mean here, other than the obvious of being supportive of others and playing nice, is that to be a good colleague you will be engaged in activities that have an impact on you and your colleague's professional lives. You can shape decisions that have important implications on people's careers and job satisfaction. If you take this approach to service, you will be taking care of your obligation without it being onerous.

Learn to say "no." As you move through your professional career, you will be asked to do more, and more, and more. The challenge is finding the balance between helping others, taking advantage of opportunities, and having time to focus on what is most important. Some questions to ask yourself when posed with a request: 1) Who or what will be impacted if I decide to take this on or not? 2) What will I have to shift in my current responsibilities if I take this on? 3) Is there someone who could do this that would be better suited than me? Letting go of hubris can be difficult, but ultimately it can be self-serving for your career. That's all I can say about saying "no"…. I struggle with this, so I am open to suggestions!

Exercise and rest. These are much too easy to forget. Your health and sanity absolutely depend on them, and your career depends on your health and sanity.

I was recently asked whether I would have chosen this career path, knowing what I now know about the life of a university professor. Having just finished an exhausting school term, I really thought about the question. But I quickly came to the answer…"Absolutely!" Despite the demands and challenges, this is one of the most rewarding of careers. So what have I learned to help me meet my professional obligations, enjoy my job, and be successful at Research, Teaching, and Service? Put people first, and be mindful of balance.

To comment on this article, go to: http://www.the-aps.org/careers/careers1/mentor/Researchinstitution.htm. ❖

Arthur C. Guyton Educator of the Year Award

Arthur C. Guyton Educator of the Year Award, supported by Elsevier, ($1,000 cash prize, complimentary registration to Experimental Biology 2010, a framed, inscribed certificate, and up to $750 in travel reimbursement to the Experimental Biology meeting) recognizes a full-time faculty member of an accredited college or university and member of the APS who has independent evidence of: (1) excellence in classroom teaching over a number of years at the undergraduate, graduate, or professional levels; (2) commitment to the improvement of physiology teaching within the candidate's own institution; and (3) contributions to physiology education at the local community, national or international levels. The awardee is requested to write an essay on his/her philosophy of education for publication in *The Physiologist*.

The typical nominee will have shown excellence in teaching and have made significant contributions in student advisement, graduate education, and/or curriculum design and reform at their institution. The activities that distinguish a candidate in the rankings include outreach activities at the state, national, or international level; contributions to education through APS activities; peer-reviewed educational journal articles; and widely disseminated publications such as commercially produced textbooks, lab manuals, or software.

Nominations Process: Each nominee must be nominated by a member of APS. The nominator must upload a letter of support outlining the qualifications of the nominee no later than January 8, 2011. To upload documents, please visit the APS Award Module at https://www.the-aps.org/awardapps/login/index.cfm. Finalists will be contacted and asked to provide further information.

Meetings & Conferences

of the American Physiological Society

Experimental Biology 2011

April 9-13, 2011 • Washington, D.C.

2011 APS Conference:

7th International Symposium on Aldosterone and the ENaC/Degenerin Family of Ion Channels: Molecular Mechanisms and Pathophysiology

September 18-22, 2011 • Asilomar Conference Grounds, Pacific Grove, California

2011 APS Conference:

Physiology of Cardiovascular Disease: Gender Disparities

October 12-14, 2011 • University of Mississippi Medical Center, Jackson, Mississippi

Experimental Biology 2012

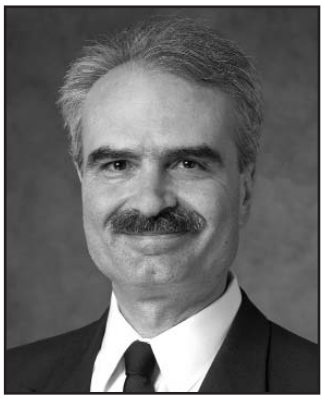
April 21-25, 2012 • San Diego, California

2012 APS Intersociety Meeting:

The Integrative Biology of Exercise VI

October 10-13, 2012 • Westin Westminster, Colorado

The American Physiological Society, Meetings Department
Phone: 301.634.7967, Fax: 301.634.7264, E-mail: meetings@the-aps.org



Physiology in Perspective: The Walter B. Cannon Award Lecture (Supported by the Grass Foundation)

Roberto Bolli
Univ. of Louisville

"The nitric oxide-carbon monoxide module: A fundamental mechanism of cellular resistance to stress"

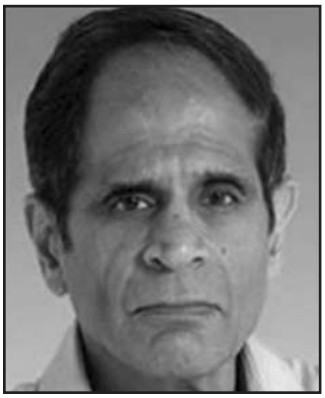
Saturday, April 9, 5:45 pm

Henry Pickering Bowditch Award Lecture

Larissa Akimi Shimoda
Johns Hopkins Univ.
School of Medicine

"Effects of chronic hypoxia on the pulmonary circulation: role of HIF-1"

Sunday, April 10, 5:45 pm



Claude Bernard Distinguished Lectureship of the Teaching of Physiology Section

Patangi Rangachari
McMaster Univ., Canada

"Steps To Pluripotent Learning: Unstrained, Undisciplined Teaching"

Sunday, April 10, 10:30 am

Hugh Davson Distinguished Lectureship of the Cell and Molecular Physiology Section

Dennis Brown
Massachusetts Gen. Hosp.

"Trafficking of proton pumps and aquaporins in urogenital epithelia: a tale of two CTs (cell types)"

Sunday, April 10, 2:00 pm

Ernest H. Starling Distinguished Lectureship of the Water and Electrolyte Homeostasis Section

Curt Sigmund
Univ. of Iowa

"Divergent Mechanisms Regulating Fluid Intake and Metabolism by the Brain Renin-Angiotensin System: A Story of Drinking and Donuts"

Sunday, April 10, 2:00 pm

Carl Ludwig Distinguished Lectureship of the Neural Control and Autonomic Regulation Section

Allyn L. Mark
Univ. of Iowa

"The Neurobiologic Regulation of Blood Pressure and Locomotor Activity in Obesity: Insights from Leptin"

Monday, April 11, 8:00 am

Solomon A. Berson Distinguished Lectureship of the Endocrinology and Metabolism Section

Christos Mantzoros
Harvard Medical School

"Leptin deficiency as a new hormone deficiency syndrome in humans: from fiction to fa(c)ts using translational research"

Monday, April 10, 10:30 am

Robert M. Berne Distinguished Lectureship of the Cardiovascular Section

Stephen Vatner
UMDNJ

"A Physiologist's Evolution: From Coronary Circulation to Adenylyl Cyclase Type 5 Inhibition"

Monday, April 11, 2:00 pm

Carl W. Gottschalk Distinguished Lectureship of the Renal Section

Jeffrey Garvin
Henry Ford Hospital

"Radical Interactions in the Kidney: A Tale of Dominance and Subversion"

Monday, April 11, 3:15 pm

Joseph Erlanger Distinguished Lectureship of the Central Nervous System Section

Larry Swanson
Univ. of Southern California

"Organization of neural systems underlying motivation and emotion"

Monday, April 11, 3:15 pm

August Krogh Distinguished Lectureship of the Comparative & Evolutionary Physiology Section (Supported by Novo Nordisk Foundation)

Gerald L. Kooyman
Univ. of California, San Diego

"Life in the Wild: Applying the Krogh Principle to Marine Birds and Mammals"

Tuesday, April 12, 8:00 am

Julius H. Comroe, Jr. Distinguished Lectureship of the Respiration Section

Jahar Bhattacharya
Columbia Univ.

"The Lung as I See it—Lessons from Live Lung Microscopy"

Tuesday, April 12, 10:30 am

Edward F. Adolph Distinguished Lectureship of the Environmental and Exercise Physiology Section

Michael Joyner
Mayo Clinic

"Giant Sucking Sound: Can Physiology Fill the Intellectual Void Left by the Reductionists?"

Tuesday, April 12, 2:00 pm

Horace W. Davenport Distinguished Lectureship of the Gastrointestinal & Liver Section

James Madara
Univ. of Chicago

"Pathobiology of Intestinal Epithelia: A Means to Mentor"

Tuesday, April 12, 3:15 pm

Walter C. Randall Lecturer in Biomedical Ethics

Gerald Koocher
Simmons College

"Colleagues as a Defense Against Bad Science"

Tuesday, April 12, 2:00 pm

The Physiologist
Vol. 53, No. 6, 2010

Dale J. Benos (1950-2010)

79th APS President

Dale J. Benos, 79th President of the American Physiological Society and Chair, Department of Physiology and Biophysics, University of Alabama, Birmingham, died suddenly on October 7. He is survived by his wife, Kim, and daughters Kaitie and Emilee.

Benos received a BA degree in Biology from Case Western Reserve Univ. and his PhD in Physiology and Pharmacology at Duke. He was an Andrew W. Mellon Scholar in the Laboratory of Human Reproduction and Reproductive Biology at Harvard Medical School, where he was both Assistant and Associate Professor in the Department of Physiology and Biophysics. After moving to the Univ. of Alabama, Birmingham in 1985, he was appointed Senior Research Scientist in the Gregory Fleming James Cystic Fibrosis Research Center, Senior Scientist in the Nephrology Research and Training Center, and then in 1987 was appointed full Professor in the Department of Physiology and Biophysics. Benos became Chair of the department in 1996. He subsequently obtained Senior Scientist positions in the UAB Center for AIDS Research, the Comprehensive Cancer Center, the Arthritis and Musculoskeletal Center, and the Center for Computational and Structural Biology. Benos also held secondary faculty status in the Departments of Cell Biology and Neurobiology. In 2005 he was named UAB's first holder of the Endowed Professorship in Physiology. In 2006 he received the UAB President's Award for Excellence in Teaching. In 2008, he was named UAB's first holder of the University of Alabama Health Services Foundation Endowed Chair in Biomedical Research. In 2007 Benos received UAB School of Medicine's highest faculty honor, the Distinguished Faculty Lecturer. It was a reflection of his contributions to UAB and the community.

Benos became a member of the APS in 1982. He held many positions in the Society including Councillor, Chair of the Awards Committee, Chair of the Joint Managing Board (IUPS and APS), and Chair of the Publications Committee. He was editor-in-chief of the *American Journal of Physiology: Cell Physiology* and served on its editorial board, and he was editor of APS's "Physiology in Medicine" series that is published in *Annals of Internal Medicine*. He was President of the Society from 2006-2007.

Dale J. Benos

Because of his experience as Chair of the APS Publications Committee, he developed several courses at UAB addressing ethics in publications. He also helped the Society refine its procedures for handling ethical cases arising through the Society's publications. He was a faculty member in the Society's Professional Skills Training Program on "Writing and Reviewing for Scientific Journals." His influence on students is exemplified by the comments received from one of the participants in the program. *"Always in every school, we have a teacher or person who is humble, dedicated to the student or people, makes us laugh, makes the most difficult subject easy to understand, finally a role model to follow....That person was Dr. Dale Benos."*

Continuously funded by the National Institutes of Health since 1976 and principal investigator for 19 individual research grants, Benos' research focused on understanding the movement of sodium ions present in the membranes of surface and nerve cells. That process is relative to the progression of certain diseases such as cystic fibrosis and hypertension. His laboratory also investigated the role of ion channels and transporters in human brain tumors. During his career, he trained more than 15 graduate students and 34 postdoctoral fellows. Benos authored more than 200 original articles and 80 invited reviews and edited four books.

Dale's passion for teaching and the students he interacted with is exemplified by the recognition he received in 2006 when he was presented with the UAB President's Award for Excellence in Teaching. However, it was exemplified even more clearly by the comments posted to the blog (Love is Free - http://bit.ly/aLE96e) by one of UAB's current medical students. In it she writes that *"He is a living declaration of passion. He takes an interest in us - not only in our education in the strictest sense of the word, but really in our ability to learn and reason. He converts abstract concepts into tangible objects. He is patient. He is kind. He is freely giving of himself. He is overflowing with life and love and a passion for both. He tells you about his family. He is a REAL person. He is a breath of fresh air and a glimmer of hope. His smile is warm, and his impact is lasting."* She continues *"Some people blaze through this life shining so brightly, that we can't help but be warmed by their light. Dr. Benos was one of these rare individuals. I was one student in a room of 175 who had the pleasure of being warmed by Dr. Benos's light for eleven hours over the course of two weeks. Can you imagine the impact that this man's life had? The number of smiles he created? Of hearts he warmed? The lessons he taught and never realized?"*

Dale was a close friend, golfing buddy, and colleague. He was an inspirational leader for the UAB Department of Physiology and Biophysics. He was a tireless advocate for APS and for the whole community of Physiologists. Donations in Dale's memory can be made to the Dale Benos Memorial Fund (http://bit.ly/adGPo5), c/o The American Physiological Society, 9650 Rockville Pike, Bethesda, MD 20814 or to the Dale Benos Research Fund, c/o UAB Gift Records, 1530 3rd Ave. S., AB1230, Birmingham, AL 35294. ❖

The Physiologist
Vol. 53, No. 6, 2010

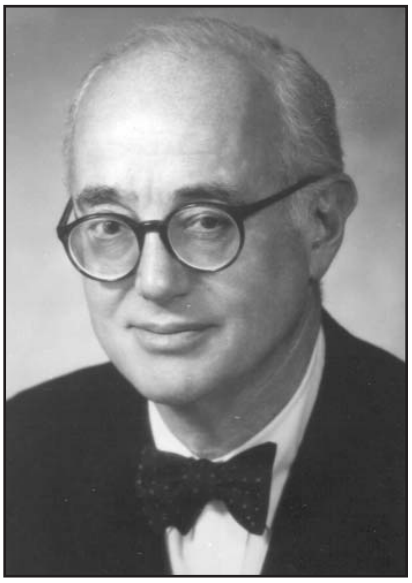
Alfred P. Fishman (1908-2010)

56th APS President

Alfred P. Fishman, a giant of modern medical science and former APS President, died at 92 years of age on Wednesday, October 6, 2010. He is survived by his wife, Linda, his three children, Mark, Jay, and Hannah, and his four grandchildren, Aaron, Brian, Eric, and Sarah. With a background in physiology, pathology, and medicine, he was an internationally recognized expert in diseases of the lung. He was the editor of the authoritative textbook of lung medicine, now in its fourth edition, and trained many of the leaders of contemporary medical science.

Alfred Fishman was the son of immigrants from Lithuania and grew up in Brooklyn during the Great Depression. At the age of 15, he started college at the Univ. of Michigan, where he received both a BA and MPH degree. He then went to the Univ. of Louisville Medical School for his MD degree. After discharge from service in the Army during World War II, Fishman began his investigative career as part of the team that built the first hemodialysis machines in the United States. He then worked with the team of Andre Cournand and Dickinson Richards on the project that described cardiac catheterization and led to the Nobel Prize in Physiology or Medicine. After working at Columbia-Presbyterian Medical Center and the Michael Reese Hospital in Chicago, Fishman came to the Univ. of Pennsylvania. During his tenure as chief of the Division of Cardio-Pulmonary Medicine, his group made many contributions in the field, including discovering why patients with severe deformations of the spine (kyphoscoliosis) suffer lung disease and how to improve their care. Fishman's central role in the characterization of pulmonary hypertension was a prelude to the care of this important syndrome and to advances in lung transplantation. Fishman was the William Maul Measey Professor of Medicine at the Univ. of Pennsylvania School of Medicine. He also served at various times as the Univ. Senior Associate Dean for Program Development, Chair of the Department of Rehabilitation Medicine, Chair of the Health Promotion and Disease Prevention Council, and Director of the Office of Complementary Medicine.

In 1961 Alfred Fishman began a more than 25-year association with the journals and publications of APS when he joined the Editorial Board of *Physiological Reviews*. He came with experience. From 1958-1963 he had been on the board of the *Journal of Clinical Investigation*, and in 1960 he was appointed to the boards of both *Circulation* and *Circulation Research*. By the end of his six years with *Physiological Reviews* (1961-67) he was serving as editor of *Physiology for Physicians* (1966-69) and of the series "Physiology in Medicine" (1969-79) in the *New England Journal of Medicine*, both sponsored by APS. He next became chairman of the Editorial Board of the *Handbook of Physiology*, where he served for five years (1967-72) and from which he moved to the Publications Committee of the Society. From 1975-1981 he was chairman of this committee and in 1979 he became editor of the Handbook volumes on respiratory physiology. In 1981 he was appointed editor of the *Journal of Applied Physiology*. From this rather considerable range of activities, Fishman will be known and remembered mainly for having sponsored and guided the reorganization of the Society's journals. His contributions to APS, however, have not been limited to its publications. He was chairman of the Program Committee (1965-68) and the Task Force on Programming (1976), and he served on the Task Force on Clinical Physiology (1974-75), the Long-Range Planning Task Force (1980-84), and the Centennial Celebration Committee, which he had chaired since 1985. He also served as chairman of the committee to find a successor to Orr Reynolds, hiring Martin Frank. Fishman received the Ray G. Daggs Award in 2004 in recognition of his contributions to the Society and discipline of physiology.



Alfred P. Fishman

While President of the Society, Fishman encouraged the US National Committee of the IUPS to assume its proper role in international science. The APS Council offered to collaborate with the IUPS to establish an international physiological journal, which was the first step toward the creation of *News in Physiological Sciences* (which was renamed *Physiology*). Fishman later served on the editorial board of this journal.

Fishman was engaged nationally and internationally in developing innovative medical programs. Over the years, he had been a consultant to NASA for the Mercury space program; a consultant to the executive office of the President of the United States; a member of the National Heart, Lung, and Blood Institute; and chairman of the Health Sciences Policy Board of the Institute of Medicine. He was a member of the American Society for Clinical Investigation, the Association of American Physicians, the Royal Society of Medicine (London), and the American Academy of Arts and Sciences. He was a former president of the College of Physicians of Philadelphia. He edited nine books and published more than 250 scientific articles.

Fishman was honored with numerous awards and distinguished lectureships. In 1980 he was elected to membership in the Institute of Medicine of the National Academy of Arts and Sciences. He was an honorary fellow of the American College of Cardiology and the American College of Chest Physicians and he received the Jacobi Medallion from the Mt. Sinai Medical Center, the Distinguished Achievement Award of AHA, and the Distinguished Alumnus Award from the Univ. of Louisville. He was honored by numerous named lectureships, including two in honor of Louis N. Katz. In 2001, Fishman was the recipient of the prestigious Edward Livingston Trudeau Medal, the highest award offered by the American Thoracic Society, a branch of the American Lung Association. The award recognized Fishman's excellence in original research on the lungs, the control of breathing in health and disease, and on normal and abnormal pulmonary circulation. In 2003, Fishman was designated a Fellow of the American Heart Association.

Alfred Fishman's amazing accomplishments—both professionally and personally—will be a lasting inspiration to all who knew him. ❖

APS Members Elected to the Institute of Medicine

Two APS members were among the group of new members announced by the Institute of Medicine (IOM) in conjunction with its 40th annual meeting. A total list announced by the IOM included the names of 65 new members and five foreign associates. Election to the IOM is considered one of the highest honors in the fields of health and medicine and recognizes individuals who have demonstrated outstanding professional achievement and commitment to service.

The APS members elected to the IOM include: Charles A. Czeisler, Baldino Professor of Sleep Medicine and Director, Division of Sleep Medicine, Harvard Medical School; and Chief, Division of Sleep Medicine, Brigham and Women's Hospital, Boston, MA; and Roger A. Johns, Professor, Department of Anesthesiology and Critical Care, Johns Hopkins Univ. School of Medicine, Baltimore, MD.

Magdalena Alonso-Galicia has moved to the Harborside Financial Center at Forest Research Institute, Jersey City, NJ. Prior to this position, Alonso-Galicia was in the Department of CardioMetabolic Diseases at Boehringer-Ingelheim Pharmaceuticals, Inc., Ridgefield, CT.

David Busija has been named chair of the Department of Pharmacology at Tulane Univ. School of Medicine, effective January 1, 2011. He is currently a professor of Physiology and Pharmacology at Wake Forest University (WFU) Health Sciences Center and a Professor of the WFU Institute for Regenerative Medicine. Busija will be filling the chair occupied by the late Krishna C. Agrawal, who passed away in December 2009.

William W. Chin is Executive Dean for Research and Professor of Medicine in the HMS-Medicine-BWH at Harvard Univ. Medical School, Boston, MA. Prior to this position, Chin was VP of Discovery Research and Clinical Investigation at E.I. Lilly and Co., Indianapolis, IN.

Demetra Demetriou Christou is Assistant Professor in the Department of Applied Physiology and Kinesiology at Univ. of Florida, Gainesville. Prior to this move, Christou was a Postdoctoral Fellow in the Department of Health and Kinesiology, Texas A&M Univ., College Station.

Albert Gjedde is now Professor and Chair of the Department of Neuroscience and Pharmacology, Univ. of Copenhagen, Copenhagen, Denmark. Prior to this move, Gjedde was Professor of Positron Emission Tomgraphy Center, Aarhus Univ. Hospital, Aarhus, Denmark.

Paul R. Grimm is Adjunct Professor of Department of Physiology, Univ. of Maryland, Baltimore. Previously, Grimm was in the Department of Cellular and Integrative Physiology at the Univ. of Nebraska Medical Center, Omaha.

Aaron J. Gruber has moved and is Assistant Professor in the Department of Neuroscience, Univ. of Lethbridge, Alberta, Canada. Prior to this move, Gruber was Postdoc in the Department of Anatomy and Neurobiology at the Univ. of Maryland, Baltimore.

Thomas J. Hawke is Associate Professor in Department of Pathology & Molecular Medicine, McMaster Univ., Hamilton, Canada. Prior to this move, Hawke was an Assistant Professor of Department of Kinesiology & Health Sciences, York Univ., North York, Canada.

Jay Robert Hoffman is presently at the Univ. of Central Florida, Department Sports & Fitness, Orlando. Prior to this position, Hoffman was at College of New Jersey, Department Health & Exercise Science, Ewing, NJ.

Ayako Makino is now an Assistant Professor of the Department of Medicine, Univ. of Illinois at Chicago. Prior to this more, Makino was an Assistant Professor of the Department of Medicine, Univ. of California, San Diego.

James E. Melvin is Clinical Director, NIDCR/NIH, Bethesda, MD. Prior to this position, Melvin was a Professor of the Center for Oral Biology, Univ. of Rochester, NY.

Shmuel Muallem is Chief, Epithelial Signaling and Transport Section, NIDCR, Bethesda, MD. Prior to this position, Muallem was a Professor of Department of Physiology, Univ. of Texas Southwestern Medical Center, Dallas.

Johnny Porter is now a Professor of Physiology in the Department of Biomedical Sciences, William Carey School of Osteopathic Medicine, Hattiesburg, MS. Prior to this move, Porter was a Professor of Physiology, Medicine, Pharmacology and Neuroscience in the Department of Physiology, Louisiana State Univ. Health Sciences Center, New Orleans.

Michael Anton Tevald, is currently an Assistant Professor in the Division of Physical Therapy at the Univ. of Toledo, OH. Previously, Tevald was a Postdoctoral Fellowship in the Department of Kinesiology at the Univ. of Massachusetts, Amherst.

James S. Wiley is a Professor at the Florey Neurosciences Institute, Univ. of Melbourne, Sarlton South, Australia. Prior to this move, Wiley was a Professor in the Department of Medicine, Nepean Hospital, Penrith, Australia.

Davie W. Wray is a Research Assistant Professor of Internal Medicine & Division of Geriatrics, Univ. of Utah, Salt Lake City. Prior to this move, Wray was Postdoctoral Fellow in the Department of Medicine, Univ. of California, San Diego. ❖

Recently Deceased Members

James F. Alexander
Houston, TX
Dale J. Benos
Birmingham, AL
Alfred P. Fishman
Center City, PA
Robert Galambos
La Jolla, CA
Carl S. Hammen
St. Petersburg, FL
Robert C. Little
Evans, GA
Allen F. Reid
Ontario, NY
Sheldon Rosenfeld
Los Angeles, CA
Robert A. Schnieder
Minneapolis, MN
John I. White
Sarasota, FL
William B. Wood
Memphis, TN

Postdoctoral Positions

Postdoctoral Positions at UC Davis School of Medicine: Postdoctoral positions are available at Univ. of CA Davis Medical Center Department of Surgery Division of Research to investigate the signaling and molecular mechanisms of cell-cell and cell-matrix interactions in regulating vascular barrier function in health and disease. Current research approaches include various animal and cell models of disease and injury, intravital microscopy, isolated microvessels, fluorescent microscopic imaging, protein biochemistry, and state-of-the-art molecular biology techniques. Require doctoral degree in related areas. Experience in physiology, cell biology, molecular biology, protein chemistry, or microscopic imaging is preferred. Send CV and contact information of three references to Dr. Sarah Yuan at: sarahyuan@ucdavis.edu.

Postdoctoral position: A postdoctoral position for an individual with a PhD degree is available immediately in an epithelial transport research laboratory at the Univ. of Florida, Gainesville. Current investigations in our laboratory are focused on several anion exchangers in the SLC26A gene family that are involved in epithelial oxalate transport. Using a variety of transport physiology and molecular biology approaches and studies that include whole animal (mouse knockout models) and tissue (intestine and kidney) in addition to cell culture studies, we are evaluating the contribution of specific transport proteins to epithelial movements of oxalate. The project is focused on the physiological interaction between the gut-resident Oxalobacter formigenes and the enterocyte which results in alterations of intestinal oxalate transport and enteric elimination of oxalate leading to reduced renal excretion of this kidney stone-forming compound. The position requires expertise in a variety of molecular biology approaches, microbiology, and willingness to conduct transport studies across mouse intestine following a training period. The candidate should have a strong background in implementing and conducting experiments as well as in the statistical analyses of experimental data. A proven record of verbal and written communication is necessary. Salary will be based upon experience. Please send a detailed CV as soon as possible to Dr. Marguerite Hatch, hatchma@ufl.edu. Please include contact information for three individuals who can provide letters of recommendations. The Univ. of Florida is an equal opportunity employer.

Postdoctoral Fellow in Exercise/Cardiovascular Physiology: A Postdoctoral Fellow position is available (NIH-funded) at the Univ. of Missouri, Columbia in the Department of Biomedical Science (http://www.dbms.missouri.edu) to study the impact of exercise on myocardial and coronary vascular remodeling in heart failure. A variety of experimental approaches will be utilized in these studies, including ultrasound, in vivo hemodynamic methods (LV pressure-volume, coronary blood flow), and in vitro techniques including cell electrophysiology, large and resistance vessel function, calcium imaging, cell culture, protein and molecular biochemistry. A background in exercise, cardiovascular or electrophysiology is required. Previous experience in cardiac ultrasound, isolated heart perfusion, and/or cardiac myocyte isolation a strength. Salary is commensurate with experience and based on current NIH guidelines. Applicants must hold a PhD or equivalent degree and send a cover letter (addressing research interests and goals), CV, and the names and contact information for three references to: Craig Emter, PhD, Department of Biomedical Sciences, E117 Veterinary Medicine, Univ. of Missouri, Columbia, Missouri 65211 or email electronic application in PDF format to EmterC@missouri.edu. The Univ. of Missouri is an Affirmative Action/Equal Opportunity Employer. Applications from women and minorities are strongly encouraged. To request ADA accommodations, please contact our ADA Coordinator at http://ada.missouri.edu/index.html.

Postdoctoral Research Associate: Available immediately in the Department of Cell & Molecular Physiology at Loyola Univ. Medical Center to participate in a muscle physiology research project. The study involves the role of heat shock protein in the protection of muscle structure-function during frostbite injury. The successful applicant will be expected to master such techniques as immunohistochemistry and muscle contraction measurements. Prior experience with whole muscle contraction studies is desirable. Although outstanding candidates in all areas of muscle physiology will be considered, special consideration will be given to individuals who will complement our existing strengths in the study of muscle physiology. Qualified applicants should submit (preferably in PDF format): a letter describing research interests, a current curriculum vitae and the names, phone numbers and email addresses of three referees. Application should be sent by Email to: Ruben Mestril, PhD., Professor of Physiology & Medicine, Assistant Dean for Basic Science Research & Postdoctoral Affairs, Stritch School of Medicine, Loyola Univ. Chicago, 2160 South First Avenue, Maywood, IL 60153. Email: rmestri@lumc.edu.

Postdoctoral Research Associate: Available immediately in the Department of Medicine at McMaster Univ. (Hamilton, Ontario, Canada) to participate in cardiovascular research. Ultimately, the focus of this project is to better understand the molecular and cellular mechanisms underlying arterial graft failure following coronary artery bypass surgery. A variety of experimental techniques are available to be applied to this problem. An animal model of post-surgical post-operative vasospasm and/or intimal hyperplasia will be developed, and in vivo interventions (both surgical and pharmacological) can be tested. Videorecordings of vasoconstrictor/vasodilator responses in excised arterial vessels (cannulated, pressurized, perfused) will be obtained under various conditions and in the presence/absence of various pharmacological tools. Cultured smooth muscle and/or endothelial cells may also be used to evaluate those interventions. Molecular biological approaches (Westerns; RT-PCR; kinase/phosphatase assays) will provide additional insight towards the signalling pathways involved. Although outstanding candidates in all areas of physiology will be considered, special consideration will be given to individuals who

complement our existing strengths in smooth muscle physiology and have previous experience in one or more of the research approaches described herein (see our web-site at http://univmail.cis.mcmaster.ca/~janssenl/index.htm). Qualified applicants should submit a single PDF file consisting of a letter describing research interests, current curriculum vitae and the names, phone numbers and email addresses of three referees to Dr. L. J. Janssen at janssenl@mcmaster.ca.

Postdoctoral research fellowship (Cardiovascular Molecular Imaging): Four Postdoctoral research fellow positions available, in cardiovascular imaging program at Yale Univ. School of Medicine. Looking for candidate to work under NIH funded training grant titled: Multi-modality Molecular and Translational Cardiovascular Imaging. There are three primary research focuses: *1*) cardiovascular molecular imaging; *2*) cardiovascular imaging technologies and analyses; and *3*) translational cardiovascular imaging NIH funded projects involve cardiovascular imaging with multiple modalities, including; 3D echocardiography, SPECT, PET, CT, angiography, and MRI. Applicant should hold PhD and/or MD degree, with a background in bioengineering, cardiovascular physiology, or imaging. Applicants must be a citizen or a noncitizen national of the United States or have been lawfully admitted for permanent residence at the time of appointment. Yale is an Equal Opportunity/Affirmative Action Employer. Applications from women and members of minority groups are encouraged. If interested, please send a statement of research interests, curriculum vitae, and the names of three references to Albert J. Sinusas, MD, Professor of Medicine and Diagnostic Radiology, Director of Yale Translational Research Imaging Center, Section of Cardiovascular Medicine, Yale Univ. School of Medicine, P.O. Box 208017, New Haven, CT 06520-8017. Email:albert.sinusas@yale.edu.

Faculty Positions

Physiology Faculty Position: Ross Univ. School of Medicine, located on the beautiful Caribbean island of Dominica in the West Indies, invites applications for a faculty post at any rank in Physiology. We invite applicants from any area of expertise, but are currently very interested in an individual who can teach Endocrine and Reproductive Physiology. Our mission is to prepare highly dedicated students to become effective, successful physicians in the United States. Basic science coursework is taught in Dominica and students then complete their clinical studies in the United States. After passing all prerequisite examinations, Ross graduates are licensed to practice medicine in all 50 states of the US. Ross Univ. School of Medicine is a division of DeVry, Inc. (NYSE:DV). Education is the primary focus of the faculty. The academic year is divided into three semesters with a new class of students admitted each semester. Lectures and other educational responsibilities continue throughout the year. Effective teachers are sought, particularly individuals who are interested in improving medical education and who work well on a team. Research opportunities exist, primarily in the area of medical education. **Essential Duties and Responsibilities:** *1*) the preparation of course material (handouts etc.); *2*) the delivery of effective lectures; *3*) the preparation, administration, marking and reporting of examinations; *4*) undergo training to qualify as a facilitator in the problem-based learning program; *5*) supervise educational activities of students under actual or simulated situations; *6*) prepare instructional plans and career analyses to reflect current changes in the field; *7*) advise individuals or groups of students in academic matters and exercise professional judgment in referring students to appropriate personnel; *8*) develop new instructional materials and teaching techniques with participation in on-going reviews and revision of curriculum planning; *9*) actively participate in relevant professional activities in order to improve teaching and subject matter competence; *10*) serve on faculty committees as appointed or elected, and confer with advisory groups in order to modify course content; *11*) prepare, administer and evaluate examinations to assess the development of student accomplishments; *12*) participate in other activities as assigned by the department chair or executive dean. **Qualifications:** *1*) content expertise in endocrine or reproductive physiology; *2*) ability to relate physiology to clinical scenarios; *3*) experience in computer-assisted delivery of course content; *4*) excellent communication skills in English; *5*) strong teaching skills and experience or evidence of potential; *6*) interest in medical education; *7*) desire for self improvement; *8*) flexibility and ability to work well on a team. **Education, Experience, Knowledge and Skills:** *1*) PhD, MD or MD/PhD degree in physiology; *2*) enthusiastic teacher with previous teaching experience at a North American or United Kingdom medical school. Ross Univ. offers a competitive potentially tax-free annual salary, relocation assistance to and from the island, a deferred pension program, tuition assistance benefit, scholarship program for dependents, 100% medical benefits paid for the employee, travel benefits, a living allowance, 25 days of paid annual leave is provided along with opportunities for professional development, which includes a conference and book allowance. To apply, please visit our website http://www.rossu.edu; select Careers and complete our online application process. [EOE]

Assistant/Associate Professor (tenure track) Position as Physiologist/Pharmacologist: The Department of Pharmacodynamics in the Univ. of Florida College of Pharmacy invites applications from scientists with a strong background in physiology or pharmacology for a 12-month tenure track position at the Assistant or Associate Professor rank. The College of Pharmacy is located within the Health Sciences Center at the Univ. of Florida, offering opportunities for collaborations with active research groups in The Hypertension Center, The Powell Gene Therapy Center, The McKnight Brain Institute, The Institute on Aging, The Genetics Institute and The Cancer Center. Current department faculty have broad research interests in physiology and pharmacology, including cellular and behavioral neuroscience, cardiovascular and endocrine physiology, food intake, and addiction. Candidates are expected to hold a PhD in physiology, pharmacology or a related discipline and have postdoctoral experience. While junior faculty candidates are encouraged to apply, preference will be

given to candidates with an established research program and a strong teaching background. A successful candidate would be expected to maintain an extramurally funded research program in physiology or pharmacology and, to contribute to education of graduate students and entry level PharmD students. Teaching assignments for this position will include lectures in physiology and pathophysiology (including renal and/or respiratory physiology) for professional students and teaching in the graduate curriculum. The search committee will begin to review applications on November 1 and will continue to review applications until the position is filled. Applicants should submit their curriculum vitae, a description of their research program, and a statement describing the applicant's teaching experience to: Search Committee, Department of Pharmacodynamics, Box 100487, 1600 SW Archer Rd, P1-20, Univ. of Florida, Gainesville, FL 32610 (Email: pdsearch@cop.ufl.edu). Three letters of reference should be separately sent to the search committee. If an accommodation due to a disability is needed to apply for this position, please call 352-392-2477 or the Florida Relay System at 800-955-8771 (TDD). [EOE]

Assistant, Associate, or Full Professor of Exercise Physiology/ Science: The School of Kinesiology (Kin) in the College of Education and Human Development (CEHD), in partnership with the Division of Epidemiology and Community Health (EpiCH) in the School of Public Health at the Univ. of Minnesota, seeks applicants for a joint appointment at the rank of Assistant, Associate, or Full Professor of Exercise Physiology/ Science. Located in the culturally diverse cities of Minneapolis and Saint Paul, the Univ. of Minnesota offers unique opportunities for research, teaching and public engagement. The Univ. is a hub for creativity, research, artistic expression, critical thinking, and debate about the world's most pressing issues such as climate change, poverty, and health care. Further information about the Univ. of Minnesota can be found at www.umn.edu/twincities/about.php.The successful candidate will be expected to lead grant-funded research efforts in areas related to exercise science and public health, and contribute to teaching and service in graduate and undergraduate programs. We seek an individual with a proven history in securing federal NIH R01 or similar external grant funding. This individual should have an educational background and demonstrated professional experience in exercise physiology, clinical physiology, kinesiology, public health, or a related field. In addition, they will have publications in top-tier professional journals and presentations in national and international professional conferences. The School of Kinesiology at the Univ. of Minnesota has a rich tradition of exceptional scholarship and academic excellence. Our research supports the interdisciplinary study of the physiological, biological, developmental, social, and behavioral bases of human movement. Our faculty is committed to excellence in research, teaching, and service as they strive to understand human physical activity across the lifespan and its relationship and impact on health and society. Further information about the School of Kinesiology's programs can be found on the web at http://cehd.umn.edu/kin and information on the Division of Epidemiology and Community Health in the School of Public Health can be found at http://www.sph.umn.edu/epi. Specific responsibilities include, but are not limited to: Research: serve as director or co-director of the Laboratory of Physiological Hygiene & Exercise Science (LPHES) and its programs with a focus on integrating basic and applied research in exercise science within a broad public health context such as health/wellness and disease prevention and management; maintain a strong record of basic and applied scholarship pertinent to exercise physiology and public health while sustaining a commitment to securing external funds to support scholarly inquiry; engage in interdisciplinary research collaborations with other scholars in Kinesiology, the College of Education and Human Development, Epidemiology and Community Health, the School of Public Health, and/or the Medical School, and within the community as part of a multidisciplinary research approach. Teaching and Advising: work cooperatively with other exercise physiology/science faculty in program development and leadership to strengthen existing programs for kinesiology majors, and develop and enhance graduate level courses; advise and mentor MS and PhD students in kinesiology graduate degree programs with an emphasis in exercise physiology; some teaching, mentoring and advising may also be possible within EpiCH/SPH. Service: participate in School, College, and Univ. service activities such as internal committees or task forces; evidence activity in professional organizations and forums; demonstrate outreach and civic engagement with the larger community through research and educational efforts consistent with one's expertise. Desired background and experience: Minimum qualifications: *1*) earned doctorate or other terminal degree in exercise physiology, clinical physiology, kinesiology, epidemiology, public health, or other closely associated field; *2*) current Associate or Full Professor, or Assistant Professor with a minimum of four years in a current tenure-track faculty position; *3*) relevant related college-level experience in graduate level teaching and academic program development; *4*) proven record of success in securing external research funding (e.g., NIH R01grants) in areas related to exercise physiology, clinical physiology, and public health; *5*) demonstrated record of excellence in research productivity in the form of peer-reviewed publications and conference publications related to exercise physiology and public health. Preferred qualifications: *1*) leadership role in basic and applied research laboratory setting including supervision and coordination of multiple research projects; *2*) a minor or other degree in fields related to exercise physiology such as nutrition, epidemiology or public health; *3*)working knowledge of the uses of technology in research and teaching; *4*) scholarship record focusing on physical activity epidemiology and population-based interventions to increase physical activity, particularly those contributing to research design, grant writing, and manuscript writing; *5*) established research collaboration with Univ., community, and professional organizations involved in sport and exercise science; *6*) postdoctoral experience in a related field. A demonstrated record of excellence in research scholarship and publications, teaching and academic program development at the graduate level, and service/outreach activities that would qualify the candidate for tenure at the University of Minnesota is required for appointment

as a tenured Associate or Full Professor. Appointment details: the individual hired will be appointed to a full-time faculty position as an Assistant, Associate, or Full Professor, dependent upon qualifications. Salary is competitive and commensurate with experience. The start date is the beginning of the 2011-2012 academic. Fall semester begins on Monday, August 28, 2011. Application process: application materials will be reviewed as they are received with the position open until filled. To receive the fullest consideration, applications should be submitted no later than Friday, November 19, 2010. Completed applications will consist of a letter addressing qualifications as they relate to the position; a curriculum vitae; an academic transcript; and the names, addresses, phone numbers, and email addresses of at least three references. An online application is required for this position and is available via the Univ. of Minnesota's Employment System at http://employment.umn.edu. Candidates should search for Requisition Number: 168576. Supporting materials such as the cover letter and curriculum vitae may be attached electronically to the online application or mailed to: Diane Wiese-Bjornstal, Chair, Exercise Physiology Search Committee, School of Kinesiology, Univ. of Minnesota, 111 Cooke Hall, 1900 University Avenue SE, Minneapolis, MN 55455. For additional information, please contact Jonathan Sweet at sweet006@umn.edu. The Univ. of Minnesota is one of the nation's premier research universities, located in a diverse, vibrant metropolitan area that offers exciting opportunities for research with diverse populations. The Univ. of Minnesota is committed to the policy that all persons shall have equal access to its programs, facilities, and employment without regard to race, color, creed, religion, national origin, sex, age, marital status, disability, public assistance status, veteran status, or sexual orientation.

Associate Dean, Academic Affairs (Biological Sciences): The Associate Dean of the Biological Sciences Department at Central Campus serves as the "department chair." This is an academic leadership and management position reporting directly to the Dean of Academic Affairs. Generally, an academic administrator in this job classification is responsible for overall departmental operations in accordance with federal/state regulations, college policies, procedures, contractual agreements, and college/campus strategic planning goals and objectives. Specifically, Central Campus' Associate Dean of the Biological Sciences Department is responsible for the comprehensive administration and coordination of all operational matters related to student learning, lecture and lab instruction, curriculum development and revision of all Biology, Botany, Microbiology, Physiology, and Zoology programs and related lecture/lab courses, course scheduling, program advising, transfer articulation agreements, resolving student behavioral matters, addressing and resolving personnel (faculty and staff) needs and issues, using student learning outcomes data to promote continuous quality improvement, and managing all budgetary and financial assets related to the academic department. Equally important, this associate dean will be expected to provide college/campus leadership and policy advice on matters related to all teaching and academic support services which include all departmental lecture/lab courses within the associate in arts, associate in science, and associate in applied science degree programs. Performs other duties as assigned. Serve in a management and leadership capacity reporting to the Academic Dean as the individual responsible for the overall Biological Sciences Department operations in accordance with College policies, procedures, and contractual agreements, and in support of the overall plan of the College. Coordinate and administer all functions relating to personnel, students, scheduling, curriculum implementation, external agencies, instruction, and financial considerations relating to the Biological Sciences Department, and accomplish such in accordance with the College's mission and College-wide goals and initiatives. **Minimum Education:** Master's degree in Biology, Botany, Microbiology, Physiology, and/or Zoology or related discipline. Doctorate in Biology, Botany, Microbiology, Physiology, and/or Zoology preferred. **Minimum Experience/ Training:** Requires three years teaching experience at the post-secondary level, with a history of demonstrated leadership roles in educational settings. Excellent oral and written communication, financial management and budget design skills. Some positions may require a minimum of 18 additional credit hours in a specified discipline. Some positions require several years of teaching experience at a post secondary or higher education institution. Demonstrated excellence in teaching and scholarly/creative research in higher education. In-depth knowledge of the academic discipline(s). An equivalent combination of education and experience may be considered for some positions. Close Date: **January 4, 2011**. **How to Apply for Broward College Positions:** Apply on-line through https://jobs.broward.edu. A complete online employment application is required and if the position requests it, additional documents may need to be attached: *1*) Cover Letter, *2*) CV/Resume, *3*)Transcripts. Upon selection, official transcripts will be required. Employment is contingent upon Florida Department of Law Enforcement background verification, to include fingerprinting of candidates. Positions are contingent upon budget approval by the Board of Trustees. Call 800-682-3646 or 954-201-7338 for more information Broward Colleges is an affirmative action, equal opportunity employer and encourages applications from underrepresented groups, including minorities, women, and person with disabilities.

Assistant, Associate, or Full Professor of Exercise Physiology/ Science: The School of Kinesiology (Kin) in the College of Education and Human Development (CEHD), in partnership with the Division of Epidemiology and Community Health (EpiCH) in the School of Public Health at the Univ. of Minnesota, seeks applicants for a joint appointment at the rank of Assistant, Associate, or Full Professor of Exercise Physiology/ Science. Located in the culturally diverse cities of Minneapolis and Saint Paul, the Univ. of Minnesota offers unique opportunities for research, teaching and public engagement. The Univ. is a hub for creativity, research, artistic expression, critical thinking, and debate about the world's most pressing issues such as climate change, poverty, and health care. Further information about the Univ. of Minnesota can be found at http://www.umn.edu/twincities/about.php.The suc-

cessful candidate will be expected to lead grant-funded research efforts in areas related to exercise science and public health, and contribute to teaching and service in graduate and undergraduate programs. We seek an individual with a proven history in securing federal NIH R01 or similar external grant funding. This individual should have an educational background and demonstrated professional experience in exercise physiology, clinical physiology, kinesiology, public health, or a related field. In addition, they will have publications in top-tier professional journals and presentations in national and international professional conferences. The School of Kinesiology at the Univ. of Minnesota has a rich tradition of exceptional scholarship and academic excellence. Our research supports the interdisciplinary study of the physiological, biological, developmental, social, and behavioral bases of human movement. Our faculty is committed to excellence in research, teaching, and service as they strive to understand human physical activity across the lifespan and its relationship and impact on health and society. Further information about the School of Kinesiology's programs can be found on the web at http://cehd.umn.edu/kin and information on the Division of Epidemiology and Community Health in the School of Public Health can be found at http://www.sph.umn.edu/epi. Specific responsibilities include, but are not limited to: Research: serve as director or co-director of the Laboratory of Physiological Hygiene & Exercise Science (LPHES) and its programs with a focus on integrating basic and applied research in exercise science within a broad public health context such as health/wellness and disease prevention and management; maintain a strong record of basic and applied scholarship pertinent to exercise physiology and public health while sustaining a commitment to securing external funds to support scholarly inquiry; engage in interdisciplinary research collaborations with other scholars in Kinesiology, the College of Education and Human Development, Epidemiology and Community Health, the School of Public Health, and/or the Medical School, and within the community as part of a multidisciplinary research approach. **Teaching and Advising:** work cooperatively with other exercise physiology/science faculty in program development and leadership to strengthen existing programs for kinesiology majors, and develop and enhance graduate level courses; advise and mentor MS and PhD students in kinesiology graduate degree programs with an emphasis in exercise physiology; some teaching, mentoring and advising may also be possible within EpiCH/SPH. Service: participate in School, College, and Univ. service activities such as internal committees or task forces; evidence activity in professional organizations and forums; demonstrate outreach and civic engagement with the larger community through research and educational efforts consistent with one's expertise. **Desired background and experience:** Minimum qualifications: *1*) earned doctorate or other terminal degree in exercise physiology, clinical physiology, kinesiology, epidemiology, public health, or other closely associated field; *2*) current Associate or Full Professor, or Assistant Professor with a minimum of four years in a current tenure-track faculty position; *3*) relevant related college-level experience in graduate level teaching and academic program development; *4*) proven record of success in securing external research funding (e.g., NIH R01grants) in areas related to exercise physiology, clinical physiology, and public health; *5*) demonstrated record of excellence in research productivity in the form of peer-reviewed publications and conference publications related to exercise physiology and public health. **Preferred qualifications:** *1*) leadership role in basic and applied research laboratory setting including supervision and coordination of multiple research projects; *2*) a minor or other degree in fields related to exercise physiology such as nutrition, epidemiology or public health; *3*) working knowledge of the uses of technology in research and teaching; *4*) scholarship record focusing on physical activity epidemiology and population-based interventions to increase physical activity, particularly those contributing to research design, grant writing, and manuscript writing; *5*) established research collaboration with Univ., community, and professional organizations involved in sport and exercise science; *6*) postdoctoral experience in a related field. A demonstrated record of excellence in research scholarship and publications, teaching and academic program development at the graduate level, and service/outreach activities that would qualify the candidate for tenure at the Univ. of Minnesota is required for appointment as a tenured Associate or Full Professor. **Appointment details:** the individual hired will be appointed to a full-time faculty position as an Assistant, Associate, or Full Professor, dependent upon qualifications. Salary is competitive and commensurate with experience. The start date is the beginning of the 2011-2012 academic. Fall semester begins on Monday, August 28, 2011. Application process: application materials will be reviewed as they are received with the position open until filled. To receive the fullest consideration, applications should be submitted no later than Friday, November 19, 2010. Completed applications will consist of a letter addressing qualifications as they relate to the position; a curriculum vitae; an academic transcript; and the names, addresses, phone numbers, and email addresses of at least three references. An online application is required for this position and is available via the Univ. of Minnesota's Employment System at employment.umn.edu. Candidates should search for Requisition Number: 168576. Supporting materials such as the cover letter and curriculum vitae may be attached electronically to the online application or mailed to: Diane Wiese-Bjornstal, Chair, Exercise Physiology Search Committee, School of Kinesiology, Univ. of Minnesota, 111 Cooke Hall, 1900 University Avenue SE, Minneapolis, MN 55455. For additional information, please contact Jonathan Sweet at sweet006@umn.edu. The Univ. of Minnesota is one of the nation's premier research universities, located in a diverse, vibrant metropolitan area that offers exciting opportunities for research with diverse populations. The Univ. of Minnesota is committed to the policy that all persons shall have equal access to its programs, facilities, and employment without regard to race, color, creed, religion, national origin, sex, age, marital status, disability, public assistance status, veteran status, or sexual orientation.

Assistant Professor: The Department of Health and Human Physiology at the Univ. of Iowa invites applications from candidates with outstanding

research records in any area of physiology for a tenure track faculty position at the rank of assistant professor to begin in August 2011. Individuals who would contribute to research and teaching in aging and/or age-related diseases are particularly encouraged to apply. Start-up support will be highly competitive and ample laboratory space is available. The Department and the College of Liberal Arts and Sciences are strongly committed to gender and ethnic diversity; the strategic plans of the Univ. and College reflect this commitment. Women and minorities are especially encouraged to apply. The Univ. of Iowa is an affirmative action/equal opportunity employer. See http://jobs.uiowa.edu/faculty/view/58455 to apply for this position.

Tenure Track Faculty Position: The Department of Kinesiology at the Univ. of Toledo is searching for an exercise science professional with a strong research agenda/program who can augment research, teaching, and service activities within the department and Univ. This individual will maintain a nationally visible research program, compete for extramural grants, mentor students in research, teach undergraduate and/or graduate (MS and PhD) courses, and serve on committees. We are particularly interested in candidates whose research focuses on the role of physical activity/exercise in aging, obesity, diabetes, and/or cardiopulmonary diseases. Excellent opportunities exist for interdisciplinary research with faculty members within the Dept. of Kinesiology, Dept. of Physiology & Pharmacology, and Dept. of Biological Sciences, as well as a number of clinical departments in the university's medical school and hospital. Rank/Salary: the appointment is anticipated to be at the Assistant or Associate Professor level. Rank and salary will be commensurate with applicant's experience and qualifications. **Qualifications:** the position requires a PhD in Kinesiology or related field, a record of scholarship, and a commitment to excellence in teaching. Postdoctoral research experience is preferred. The Department is one of six departments in the Univ.'s College of Health Science and Human Service. Approximately 600 students are actively pursuing majors in the department at the BS, MS, and PhD levels. Undergraduate students may choose to major in Athletic Training, Exercise Science, or Respiratory Care. Specializations include Biomechanics, Human Performance and Health Promotion, Exercise Physiology, and Pre-PT/OT/PA. The Department also provides instruction in microbiology, pathophysiology, and anatomy and physiology to a variety of students in non-kinesiology majors. Areas of emphasis at the MS level include Athletic Training, Biomechanics, and Exercise Physiology. At the doctoral level students develop an individualized program that reflects a combination of their professional goals and the research specialization of their advisor. The department is located in the Univ.'s Health and Human Services building, and includes comprehensive research laboratories, as well as dedicated and fully mediated classroom and teaching laboratory space. Additional information on the department can be found at http://www.utoledo.edu/hshs/kinesiology/index.html. The anticipated starting date for the position is August 2011. Review of the applications will begin on December 15, 2010 and the search will remain open until the position is filled. Application Procedures: interested applicants should submit a letter of application, curriculum vitae, a statement of research program that includes a three-year plan, three letters of recommendation, reprints of three recent publications, three letters of recommendation, and graduate transcripts to: Frank X. Pizza, PhD, Dept. of Kinesiology Mailstop # 119, College of Health Science and Human Services, The Univ. of Toledo, Toledo, OH 43606, Email: Francis.Pizza@utoledo.edu; Phone: 419-530-4178; Fax: 419-530-2477. The University of Toledo is an Equal Access, Equal Opportunity, Affirmative Action, Title IX Employer, committed to excellence through diversity.

Faculty Position: The Department of Physiology and Developmental Biology at Brigham Young Univ. announces the availability of a continuing faculty status track faculty position. Review of applications will begin December 3, 2010 and continue until the position is filled. Applicants should have a doctorate degree and postdoctoral experience, with expertise and teaching capability in physiology. Research interest could include: cell biology, developmental biology, physiology, biophysics, or neuroscience. Candidates must demonstrate a high potential for establishment of an externally funded research program. Interested scientists are invited to complete a faculty application at https://yjobs.byu.edu and attach a letter of application, curriculum vitae, and one-page statement of research interests and goals. For further information contact Dr. Allan Judd, Chair Search Committee, Department of Physiology and Developmental Biology, Brigham Young Univ., Provo, UT 84602. (Tel.: 801-422-3179, Fax: 801-422-0700, Email: allan_judd@byu.edu). BYU is an equal employment opportunity employer. Preference is given to qualified candidates who are members in good standing of the affiliated church, The Church of Jesus Christ of Latter-day Saints.

Faculty Position: The Department of Neurobiology and Anatomy at Wake Forest Univ. School of Medicine (WFUSM) invites applications for a faculty position (rank open) from investigators with outstanding, multi-disciplinary research programs utilizing molecular, cellular and functional approaches including behavioral assessments to study mechanisms of neuronal injury and repair or neurodegeneration in any suitable experimental model system. The individual appointed for this position will join an active faculty whose research investigates synaptic plasticity, regulation of Ca2+ channels, neuronal activity and cellular and molecular changes associated with CNS development, aging, multisensory integration, epilepsy and motor neuron diseases. WFUSM has a large and vibrant neuroscience community with strong tradition of collegiality and collaboration, and the successful candidate will be expected to participate in translational research by pro-

moting collaborations with clinicians with shared interests and expertise. The position will be available July 1, 2011. Candidates should send a CV, statement of specific research interest and three letters of recommendation to: Search Committee, Department of Neurobiology and Anatomy; Wake Forest Univ. School of Medicine, Winston-Salem, NC 27157-1010. For more information on the Department and areas of research emphasis, visit our website at http://www.wfubmc.edu/nba Wake Forest Univ. Baptist Medical, and its component institutions, is an affirmative action and equal opportunity employer with a strong commitment to achieving diversity among its faculty and staff.

Research Positions

Molecular Physiologist: The Department of Biological Sciences at the Univ. of Wisconsin-Milwaukee seeks applicants for a tenure-track position in molecular physiology at the rank of Assistant Professor. We seek candidates with research interests in the cellular and molecular mechanisms by which signaling pathways in the endocrine or nervous systems are disrupted by environmental perturbations. Candidate qualifications include: minimum qualifications: Doctorate degree in Molecular Biology, Physiology, Toxicology or related field; Postdoctoral research and teaching experience in the areas of physiology/toxicology and/or eukaryotic molecular biology. Preferred qualifications: demonstrated ability to establish an independent, extramurally funded research program involving MS and PhD students. To apply, please go to http://www.jobs.uwm.edu/applicants/Central?quickFind=5075. A completed application should include: cover letter, curriculum vita, statement of research goals, statement of teaching interests, and letters of professional reference. Applicants should arrange to have three letters of reference sent as pdf attachments to the departmental chair (daads@uwm.edu) or mailed to: Molecular Physiologist Search, Attn: Dr. Saffarini-Chair, Department of Biological Sciences, Univ. of Wisconsin-Milwaukee, PO Box 413, Milwaukee, WI 53201. Screening of candidates will begin November 15, 2010 and continue until the position is filled. Appointment begins August 2011. [AA/EOE]

Senior Scientist, Product Pipeline Scientific Support: Boehringer Ingelheim is currently seeking a full-time talented and innovative Senior Scientist, Product Pipeline Scientific Support to join our CardioMetabolic Disease Research department located at our US headquarters in Ridgefield, CT. As an employee of Boehringer Ingelheim, you will actively contribute to the discovery, development and delivery of our products to our patients and customers. Our global presence provides opportunity for all employees to collaborate internationally, offering visibility and opportunity to directly contribute to the companies' success. We realize that our strength and competitive advantage lie with our people. We support our employees in a number of ways to foster a healthy working environment, meaningful work, diversity and inclusion, mobility, networking and work-life balance. Our competitive compensation and benefit programs reflect Boehringer Ingelheim's high regard for our employees. Responsibilities: [In coordination with the Global PPSS Pradaxa scientist (in Biberach, Germany)]: initiate and perform in vitro preclinical studies in the field of thrombosis; design, run, and interpret experiments when needed; initiate and manage external collaborations with international academic groups in the area of thrombosis, hemostasis and cardiovascular research; plan, evaluate, prioritize, modify and coordinate internal and external research activities focusing on inhibitors of thrombosis, including budgeting, planning resources and coordinating with legal/patent departments; in close collaboration with the patent department, ensure scientific management and support of all Intellectual Property (IP) interests; coordinate necessary and reasonable research patent activities for antithrombotics and anticoagulants, particularly combination or use patents; manage key opinion leaders through coordination of routine reports, oral presentations and on site visits; become an integral part of product life cycle management, including involvement in: scientific platform development, publication strategy development, including preparation of publications (abstracts) for congresses and writing/organizing reviews and primary scientific articles, planning, evaluation and modification of phase IV clinical trials based on preclinical perspectives, representing product at national and/or international congresses. Qualifications: research experience in the area of thrombosis and hemostasis, emphasis in coagulation and its inhibition is preferred; doctoral degree is required with experience in designing, running and interpreting in vitro and in vivo experiments pertinent to thrombosis /cardiovascular research; job experience in the pharmaceutical industry and understanding of drug development is helpful but not required; candidate must have demonstrated excellence in working in teams. The qualifying candidate is expected to work closely with corporate functions. Excellent communication skills, both written and oral and fluency in English are critical. Travel flexibility is required. As a member of the PPSS CardioMetabolic team, reporting into the local lead in PPSS, this person should interact with team members in Ridgefield, CT and also be tightly connected/aligned with PPSS team members CardioMetabolic in Biberach, Germany. Boehringer Ingelheim is firmly committed to ensuring a safe, healthy, productive and efficient work environment for our employees, partners and customers. As part of that commitment, Boehringer Ingelheim conducts pre-employment background investigations and drug screenings. Boehringer Ingelheim is an equal opportunity employer. M/F/D/V How to apply: please copy and paste the following link into your browser address bar: http://appclix.postmasterlx.com/track.html?pid=ff8080812b1a37c0012b4fd23a532ea9&source=asppost. ❖

Letter to Bill Dantzler

Gordon Ross writes: "Thanks for the birthday greeting. After retiring in 1994, I gave up research but continued to teach medical students in courses offered by the departments of Medicine and Physiology at UCLA. It was a pleasant surprise to be asked to do this again this year. I urge young colleagues to continually hone their teaching skills. They may seem not to be important for career development just now, but research bubbles tend to burst quite rapidly, whereas ideas imparted by a teacher may influence a student for a lifetime and give immense satisfaction to both."

Letter to Margaret Anderson

Vincent F. Castellucci writes : "I just came back from abroad and received your letter of July 15th asking me about my activities now.
"It is true I was born July 26th 1940. After being in New York for 20 years as a colleague of Dr. Eric Kandel, I was recruited at Université de Montréal in 1988. I must tell you that I was born in Montreal. In 1993 I became chairman of the Physiology Department at the Faculty of medicine. My mandate was from 1993 to 2001.
"In 2001 I became vice-dean research at the Faculty. I am still active as associate vice-dean research (since 2003).
"I closed my laboratory last year and I continue to teach at the undergraduate and graduate levels.
"By the way, I think that this initiative is excellent." ❖

Book Review

Explaining Research - How to Reach Key Audiences to Advance Your Work
Dennis Meredith

New York, USA: Oxford University Press, 2010, 357 pp., index, $35.00
ISBN: 978-0-19-973205-0 (pbk)

"Leadership tomorrow depends on how we educate our students today-especially in science, technology, engineering and math." Pres. Obama, September 16, 2010. Both boys and girls are losing interest in science and math forever by the fifth grade. The number of students enrolling in life science majors is falling in colleges across the country. The public appreciates the benefits of science, but is willfully ignorant about it. We are dismally failing to inspire interest in the sciences. In Explaining Research - How to Reach Key Audiences to Advance Your Work author Dennis Meredith identifies the critical knowledge gap between the scientific community and the lay public. He challenges scientists to explain their work by engaging and educating the public because it will "benefit your field, your institution and your own research career."

Research Explainer Meredith provides more than an expression of opinion regarding the urgent need to reach the public and bridge the gap in science. He provides data and information on the trends that will be quite surprising to most scientists in his "Introduction: Explaining Your Research Is a Professional Necessity." The remainder of the book is a well thought out How-to guideline for scientists who wish to create a communication strategy that is effective in today's world.

Explaining Research is divided into four parts and includes an index. References and resources cited in the book are listed online at ExplainingResearch.com.

"Part I-Learning a New Communications Paradigm" addresses the need to understand your audiences and to plan your research communication strategy accordingly. Show your audience why they need to know about science. Strive to get across one or two points. Pop your ego bubble so you can be a storyteller, not an authority. A scientist who only gets across a positive attitude can have a positive effect and benefit science.

"Part II-Effectively Reaching Your Peers" by methodologies to give compelling talks. Meredith emphasizes informative visuals and clear research explanations. "Develop a strategy of synergy" to develop content intended for multiple audiences. Reaching out to lay audiences does not need to be a completely separate effort or drain on your valuable time. Recognize your research website is your face to the world so make it a quality website.

"Part III-Engaging Lay Audiences" by first developing a communication strategy that will tell your research story. Understand the components of the essential news release, target releases to key audiences, include e-Newsletters, Wiki, Blogs, Podcasts, Social Networks and Webinars. Meredith explores the pros and cons of these media modalities. As an example, Meredith provides a good discussion of the question To Blog or Not to Blog. Why not consider writing popular articles, Op-Eds, essays or even a popular book? Meredith provides pointers about how each of these elements might be using your communication strategy. By becoming a public educator, scientists develop skills to persuade administrators, donors and legislators. Such skills can only be of benefit to research endeavors.

"Part IV-Explaining Your Research through the Media" is the final part of the book. Many other related books begin and end with what is commonly called "media training." Meredith's approach provides an effective overview of current media training. Meredith's background as a public information officer and experience working with scientists, engineers, journalists and fellow public information officers shows through in the guidance provided in this part of the book. Especially useful is the section outlining communications traps and strategies to avoid being caught in them.

Meredith's appreciation for science and its heroes comes through in this text. He creates a compelling case to motivate scientists into action and he provides an authoritative guide to show how it can be done. Any scientist in today's culture of media should have "Explaining Research" on hand. ❖

Diane McClure
Western Univ. of Health Sciences

The Physiologist
Vol. 53, No. 6, 2010

Physiology and Pathology of Chloride Transporters and Channels in the Nervous System: From Molecules to Diseases

Edited by Francisco Javier Alvarez-Lefmans and Eric Delpire. New York, USA: Academic Press, 2009, 617 pp, illus., index $150.00
ISBN: 978-0-12-374373-2

Chloride (Cl^-) is the most abundant extracellular anion in metazoans. Cl^- diffusion through membrane anion channels enables several life-supporting processes, such as electrical excitability, cell volume regulation, trans-epithelial salt transport, fluid secretion, and acidification of internal and external compartments. Unfortunately, back until the 1980s, the standard idea was that Cl^- is in equilibrium across most cell membranes, preventing it from doing some work of physiological relevance. Consequently, Cl^- channels and transporters, already known to exist at that time, lived under unfortunate circumstances and were mainly unrecognized, just like Cinderella in the fairy tale. It is now clear that the above idea was a faulty impression, because active Cl^- transport is present in most cells, putting Cl^- out of electrochemical equilibrium and making it capable of doing work and signaling.

The conception changed considerably in the 1980s when it became clear that the internal Cl^- concentration differs in various cell types, depending on the activity of expressed Cl^- transporters. This finding has placed Cl^- in a special position among biological ions. Due to the active Cl^- transport, electrochemical Cl^- gradients can be more negative ($E_{Cl} < V_{rest}$) or more positive (ECl > Vrest) than the resting membrane potential, thus, leading to outward and inward flow, respectively, upon opening Cl^- channels.

A first book on the issue, a classic and, in a sense, the forerunner of the present book, was published in 1990 ("Chloride channels and carriers in nerve, muscle and glial cells"; edited by Francisco Javier Alvarez-Leefmans and John M. Russell). With the rapid growth, particularly in molecular biology and physiology since then, a new treatise was long overdue. Let's take a look at numbers: for the year 1990, a Pubmed search (chloride channel* or cl channel*) resulted in 257 publications, whereas this number increased to 479 in 2009. The respective numbers for the search (chloride transporter* or cl transporter* or chloride cotransporter* or cl cotransporter*) are 118 and 178. Between 1991 and August 2010, a total of ca. 9,000 and 2,900 papers have appeared for the Cl^- channel and transporter group, respectively, illustrating the progress in these fields.

The present book, to which the leading peers have contributed, now summarizes the progress in an elegant way and restricts its focus on the nervous system, not only the brain, but also peripheral sensory systems (auditory, vestibular, visual, olfactory). The book is composed of five main parts, divided into 30 chapters and written by 55 authors, in total covering slightly more than 600 pages. The figures were predominantly taken from original papers and reprinted with permission, but the authors are to be praised for having prepared several color figures specifically for this book. I find particularly these figures very felicitous and of high didactic quality. Several chapters cover the issue from a historical perspective: Part I gives an overview of Cl^- transporters and channels, with Chapter 1 being entitled "Chloride Channels: A Historical Perspective." Part II covers the Methods for studying Cl^- regulation, Part III focuses on Cloning and structure-function analyses, and Part IV on Cation-Cl^- cotransporters in neural function, including dysfunction. Finally, Part V provides some relatively short insights into the role of Cl^- cotransporters as ion and water pumps in the choroid plexus and the blood-brain barrier. In contrast to what the cover picture might suggest, emphasis is not on anatomy and histology. Rather, the chapters cover virtually all areas of interest, bridging the broad gap from molecules to diseases. The book also critically embarks on weaknesses: for example, our understanding of how Cl^- channels work still lags behind our understanding of cation channels. One reason is that Cl^- channel blockers are notorious for their low affinity and specificity.

Overall, the authors and editors have done a marvelous job. I strongly recommend their book to those already in the Cl^- field and those who need an introduction to it, because the topics are appealing for a specialized as well as a general audience. The editors have been very successful in getting an up-to-date review from many of the major players in the field and in covering the key topics (literature is cited until 2008). Although the pace of discoveries in the field is brisk, at present this book provides an excellent overview. For a 2nd edition update, which would be very welcomed in the future, it would be an improvement if the selected references were not listed after each chapter, yet altogether at the end of the book, with appropriate links to all pages on which the reference is cited. This kind of organization, like the elaborate and very helpful Index, would serve at least four purposes: it would provide a central pool of references, make reverse searching easy, demonstrate which work is of particular importance, and emphasize the impact that a given paper has on the field. But even without this useful annex, the book edited by Alvarez-Leefmans and Delpire is outstanding and of great value to physiologists, pharmacologists, cell biologists, and others. Research on Cl^- channels and transporters started with the drawback of being overlooked, like Cinderella. However, just as Cinderella finally moved into the limelight and received unexpected, yet well-deserved recognition, the areas covered in the present book have blossomed and boomed. There is a bright future ahead for research on Cl^- transporters and channels, and a glorious time for chloride. ❖

Eckhard Friauf
Univ. of Kaiserslautern, Germany

The Physiologist
Vol. 53, No. 6, 2010

The Wine Wizard

Peter Wagner

Hi all – Well, I shouldn't have bragged about the weather in San Diego last month, it has been dreadful this month. You would think we were in Seattle with all the clouds, drizzle and 60 degree temperatures. Good wine drinking weather though.

Whites

2010 Oyster Bay Sauvignon Blanc, Marlborough, New Zealand $9. Here we go again. A very typical NZSB once more. Very clean, lots of ripe gooseberry/passionfruit on the nose and palate; lemon as well on the palate. Very crisp acidity, no question, but not mouth-puckering. Good length, very tasty, especially at this price. And yes, 2010 already.

2009 Nessa Albarino, Rias Baixas, Spain $11.50. I have waxed poetic (OK; just waxed) about the Albarino grape more than once. Done well, it makes for very tasty and interesting wine. The nose has citrus, stone fruit, with slight pear and tropical characters. Yes, all of those. The palate is similar, clean, balanced, with a nice very dry finish to balance the ripe fruit.

2008 Trevor Jones Chardonnay, Barossa, Australia $13. I offer this one as an interesting example of a chardonnay that has never exposed itself to oak. So you just get the fruit. The nose has some herbal tropical fruit, a hint of passionfruit and yeast. The palate is clean, obviously oakless, with citrus and passionfruit flavors.

2008 Girard Chardonnay, Russian River Valley, California $17. This wine has oak, but it is in the background. The nose has ripe, herbal fruit and a hint of vanilla. The palate is rich and quite viscous. It is apply in flavor, with some citrus and tropical characters as well. Oak is low; acidity is medium. It is viscous, clean, long and balanced.

Peter Wagner

Reds

2007 Sixth Sense Syrah, Lodi, California $12. This is a rich, opulent, 15% alcohol effort that is way too easy to drink. Lots of ripe dark berry fruit in the nose and palate, with some chocolate and coffee elements for interest. There is American oak (dill), but not too much. The fruit is quite ripe, which, with the high alcohol gives a sense of sweetness. But the wine is dry (no discernible residual sugar). The tannins are soft and the acid medium.

2005 Silverstone "Keeper", San Luis Obispo, California $9. This wine is 75% Syrah, 25% Mourvedre, thus qualifying as a Rhone blend. This is spicy and jammy in the nose and palate, with some chocolate as well. There is spice, even cinnamon, and while it comes across as mature, it is not over the hill at all.

2007 Leal Cabernet Sauvignon, San Benito, California $21. This was generally a very good year for California cabernet, and Leal's effort fits. The nose has lots of dark berry fruit, along with some American oak (dill) and a touch of charred oak (which some will call sulfur). The palate has excellent dark fruit intensity, with a fair amount of dill. It comes over as very nicely integrated/balanced, with good length, and enough tannins and acidity for structure, but not too much. While the price is a bit higher than I like, it is at the low end of good California cabernet.

2006 Bishop's Peak Syrah, Edna Valley, California $10. This wine has a classical syrah nose with dark plums and black pepper. The palate is similar, with some nice vanilla to sweeten the spiciness. Tannins are medium to soft, acidity is firm, and the length is reasonable. There is a touch of sulfur that was diminishing over time. ❖

Books Received

Handbook of Brain Microcircuits
Edited by Gordon M. Shepherd and Sten Grillner
New York, USA: Oxford Press, 2010, 536 pp, illus, index, $125
ISBN: 978-0-19-538988-3

Fundamentals of Medical Physiology
Edited by Joel Michael
Pennsylvania, USA: Thieme, 2010, 648 pp, index, $79.95
ISBN: 978-1-60-406274-8

MEMBERSHIP APPLICATION FORM
The American Physiological Society

1. Check membership category you are applying for: ❑ Regular ❑ Affiliate ❑ Graduate Student ❑ Undergraduate Student

2. Name of Applicant: _____ / _____ / _____
Last Name or Family Name *First Name* *Middle Name*

3. Date of Birth _____ / _____ / _____ Optional: Male ❑ Female ❑
Month *Day* *Year*

4. Institution Name _____ Department _____
(Please do not abbreviate Institution Name)

5. Institution Street Address _____

6. City/State/Zip/Country _____

7. Home Address ***(Students Only)*** _____

8. Work Phone _____ Home Phone _____

9. Fax _____ E-mail _____

10. **Educational Status:** ▶ **IMPORTANT for STUDENTS: ** If you are enrolled as a graduate student for an advanced degree, or as an undergraduate student, please include the month and year you expect to receive your degree.**

Dates**	Degree	Institution	Major Field	Advisor

11. **WHAT IS YOUR SECTION AFFILIATION?** Please identify your primary sectional affiliation with a "1" and check (✓) up to two additional sections with which you would like to affiliate. **There can be only one "Primary" affiliation.**

___Cardiovascular
___Cell & Molecular Physiology
___Central Nervous System
___Comparative & Evolutionary Physiology
___Endocrinology & Metabolism
___Environmental & Exercise Physiology
___Gastrointestinal & Liver Physiology
___Neural Control & Autonomic Regulation
___Renal Physiology
___Respiration Physiology
___Teaching of Physiology
___Water & Electrolyte Homeostasis

12. **DO YOU WORK IN INDUSTRY?** ❑ YES ❑ NO

13. **SPONSORS** (Sponsors must be Regular APS Members. If you are unable to find sponsors, check the box below, and we will locate them for you.) *Undergraduate Students do not require sponsors but must supply proof of enrollment such as transcripts or letter from your advisor.*

CHECK THIS BOX IF APPLICABLE: ❑ Please locate sponsors on my behalf.

#1 Sponsor Name _____
Mailing Address _____
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Phone _____
Fax _____
E-mail _____
Sponsor Signature* _____

#2 Sponsor Name _____
Mailing Address _____
_____
_____
Phone _____
Fax _____
E-mail _____
Sponsor Signature* _____

**signature indicates that sponsor attests applicant is qualified for membership.*

▶ **Please turn over for more questions...and mailing instructions.**

Membership Application (Continued...)

Applicant Last Name (please print) _____

14. OCCUPATIONAL HISTORY [Check if student ❑]

Current Position:

Dates	Title	Institution	Department	Supervisor

Prior Positions:

Dates	Title	Institution	Department	Supervisor

15. LIST YOUR MOST SIGNIFICANT PUBLICATIONS, WITH EMPHASIS ON THE PAST 5 YEARS

(Publications should consist of manuscripts in peer-reviewed journals. List them in the same style as sample below.)

Sample: MacLeod RJ and Hamilton JR. Volume Regulation initiated by Na^+ -nutrient contransport in isolated mammalian villus enterocytes. Am J Physiol Gastrointest Liver Physiol 280: G26-G33, 1991.

16. DOCTORAL DISSERTATION TITLE (if applicable):

_____

_____

17. POSTDOCTORAL RESEARCH TOPIC (if applicable):

_____

_____

18. WHICH FACTOR INFLUENCED YOU TO FILL OUT OUR MEMBERSHIP APPLICATION?

❑Mailer ❑Meeting (Which meeting? _____) ❑Colleague ❑Other _____

Mail your application to: Membership Services Department, The American Physiological Society
9650 Rockville Pike, Bethesda, Maryland 20814-3991 (U.S.A.)
(or fax to 301-634-7264) (or submit online at: www.the-aps.org/membership/application.htm)

Send no money now—you will receive a dues statement upon approval of membership.

Approval Deadlines: Membership applications are considered for approval on a monthly basis.

Questions? Call: 301-634-7171 • Fax: 301-634-7264 • E-mail: members@the-aps.org • Web: www.the-aps.org

2011

January 27-28

The Leukocyte in Cardiovascular Disease, Geneva, Switzerland. *Information:* Lucy Purser, Events and Marketing Coordinator, Abcam plc, 330 Cambridge Science Park, Milton Road, Cambridge, CB4 0FL, United Kingdom. Tel.: +44 (0) 1223 696000; Fax: +44 (0) 1223 771600; Email: events@abcam.com; Internet: http://www.abcam.com/geneva.

February 1-4

The Ramanbhai Foundation 5-th International Symposium: Current Treads in Pharmaceutical Sciences- Advances in Translation Research & Medicine, Ahmedabad, Gujarat, India. *Information:* Dr. Mukul Jain. Email: mukul.jain@zyduscadila.com; Internet: http://www.rbfsymposium.net.

February 12-17

SPIE Medical Imaging, Lake Buena Vista, Florida. *Information:* Internet: http://spie.org/medical-imaging.xml?WT.mc_id=RCALLACE.

February 24-27

The International Conference on Prehypertension and Cardio Metabolic Syndrome, Vienna, Austria. *Information:* Ravit Levy. Tel.: +41 (0) 22 533 0948; Email: secretariat@prehypertension.org; Internet: http://www.prehypertension.org.

March 10-11

Immunochemotherapy: Correcting Immune Escape in Cancer, Philadelphia, PA. *Information:* Lucy Purser, Events and Marketing Coordinator, Abcam plc, 330 Cambridge Science Park, Milton Road, Cambridge, CB4 0FL, United Kingdom. Tel.: +44 (0) 1223 696000; Fax: +44 (0) 1223 771600; Email: events@abcam.com; Internet: http://www.abcam.com/philadelphia.

March 21-23

AAAI Spring Symposia 2011: Computational Physiology, Stanford, CA. *Information:* Internet: http://sites.google.com/site/aaaicomputationalphysiology/home.

March 31-April 3

2nd International Course on Pain Medicine (ICPM 2011), Porto, Portugal. *Information:* Email: icpm@icpm.net; Internet: http://www.icpm.net/.

April 7-8

Injury and Repair Mechanisms in Chronic Airway Disease, London, United Kingdom. *Information:* Lucy Purser, Events and Marketing Coordinator, Abcam plc, 330 Cambridge Science Park, Milton Road, Cambridge, CB4 0FL, United Kingdom. Tel.: +44 (0) 1223 696000; Fax: +44 (0) 1223 771600; Email: events@abcam.com; Internet: http://www.abcam.com/londonimmunology.

April 29-May 1

Southern Biomedical Engineering Conference, Arlington, TX. *Information:* Internet: http://www.uta.edu/bioengineering/sbec2011/.

May 18-21

Nicotinic Acetylcholine Receptors 2011, Cambridge, United Kingdom. *Information:* Jemma Beard. Tel.: +44 (0) 1223 495120; Email: jbeard@hinxton.wellcome.ac.uk.

June 2-4

Organization for the Study of Sex Differences (OSSD) Fifth Annual Meeting, Oklahoma City, OK. *Information:* Internet: http://www.ossdweb.org.

June 27-29

9th International Conference on Modeling in Medicine and Biology (BIOMED 2011), Riga, Latvia. *Information:* Irene Moreno, Conference Coordinator. Email: imoreno@wessex.ac.uk; Internet: http://www.wessex.ac.uk/11-conferences/biomed-2011.html.

August 1-5

12th International Congress on Amino Acids, Peptides and Proteins, Beijing, China. *Information:* Professof Gert Lubec, FRSC (UK), c/o Medical University of Vienna, Währinger Gürte 18, A-1090 Vienna, Austria. Fax: +43.1.40400 6065; Email: gert.lubec@medunivwien.ac.at.

September 9-11

Oskar Kellner Symposium 2011: Metabolic Flexibility in Animal and Human Nutrition, Warnemunde, Germany. *Information:* Email: oks-info@fbn-dummerstorf.de; Internet: http://oks.fbn-dummerstorf.de/.

2012

September 1-6

AAPS 2012 Congress, Alexandria, Egypt. *Information:* African Association of Physiological Sciences, Office of the Secretariat, 82 Bulwer Road, Durban 4001, South Africa. Tel.: +27 31 2011392; Fax: +27 31 2013950; Internet: http://www.aapsnet.org/conferences.htm.

2013

July 21-26

37th Congress of the International Union of Physiological Sciences (IUPS 2013), Birmingham, United Kingdom. *Information:* Internet: http://www.iups2013.org/. ❖

2010 American Physiological Society Intersociety Meeting

Global Change & Global Science: Comparative Physiology in a Changing World

Westminster, Colorado
August 4-7, 2010

MEETING PROGRAM & ABSTRACTS

The American Physiological Society

SEB Society for Experimental Biology

Society for Integrative and Comparative Biology

Canadian Society of Zoologists

The Australian and New Zealand Society for Comparative Physiology and Biochemistry

South American Society of Comparative Physiology and Biochemistry

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2010 APS Intersociety Meeting
Global Change & Global Science:
Comparative Physiology in a Changing World

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Jon F. Harrison
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Siribhinya Benyajati
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Andrew Biewener
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David Goldstein
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Colorado State Univ.

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Alfred Wegener Inst., Germany

Patricia Schulte
Univ. of British Columbia, Canada

Acknowledgements

The Conference Organizers and The American Physiological Society gratefully recognize the generous financial support provided through unrestricted educational grants from:

National Science Foundation
NIH, National Institute of Diabetes and Digestive and Kidney Diseases
American Journal of Physiology: Regulatory, Integrative and Comparative Physiology

2010 APS Intersociety Meeting

Global Change & Global Science: Comparative Physiology in a Changing World

August 4—7, 2010, Westminster, Colorado

Wednesday, August 4	Thursday, August 5	Friday, August 6	Saturday, August 7
2:00 PM **Registration**	7:00 AM **Registration** 8:00—12:00 Noon **Concurrent Symposia** The Role of Physiology in Organismal Responses to Global Warming **R. Huey, M. Frazier and W. Porter** Off the Beaten Path: Integrative Aspects of Muscle Function During Diverse Locomotor Behaviors **T. Higham and A. Biewener** Hydration, Desiccation, Regulation: The Comparative Physiology of Water Balance **D. Goldstein and J. Williams**	7:30 AM **Registration** 8:00—12:00 Noon **Concurrent Symposia** Physiological Effects of Ocean Acidification on Marine Animals in Times of Ocean Warming: Ecosystem Implications **H-O. Pörtner** Evaluating the Effects of Global Climate Change to the Fauna of South American Biomes: Consequences of Diversity from a Physiological Perspective **C. Navas** **J. Carvalho** Environmental Adaptations of Cardio-respiratory Systems **P. Frappell and A. Woods**	7:30 AM **Registration** 8:00—12:00 Noon **Concurrent Symposia** Genomics and Proteomics Approaches to Understanding Environmental Stress **D. Kültz, and P. Schulte** Putting Comparative Physiology in the Field: Stable Isotopes as Tracers of Ecological Processes **C. Martinez del Rio** The Life History Physiology Nexus **J. Williams** The Physiology of Trace Metal Homeostasis and Toxicity **M. Grosell** Global Change and the Emerging Threat of Dead Zones: Can Individuals, Populations, Species, and Communities Respond to Increasing Hypoxic Oceans? **A. Altieri**
	1:30—3:30 PM **Concurrent Afternoon Activities** Abstract Oral Presentations **B. Wolf** **D. Hahn** **Workshop:** Opportunities for Comparative Physiologists: Building Links with Agencies, Institutions and Foundations **J. Harrison**	1:30—3:30 PM **Afternoon Activities** Scholander Oral Presentations **J. Harrison**	1:30—3:30 PM **Concurrent Afternoon Activities** Abstract Oral Presentations **G. McClelland** **S. Place** **Workshop:** Career Paths/Options for a Comparative Physiologist **S. Benyajati and K. L. Sweazea**
	3:30—6:30 PM **Poster Session** Best Poster Competition	3:30—6:30 PM **Poster Session** Best Poster Competition	3:30—6:30 PM **Poster Session**
6:00—8:00 PM **Opening Reception** 8:00—9:00 PM **Plenary Lecture:** Participants: **G. Somero**	**Evening Free**	**Evening Free**	7:30—11:00 PM **Awards Banquet** 9:30—10:30 PM **Plenary Lecture:** Participants: **E. Brainerd**

GENERAL INFORMATION

Location:

The 2010 APS Intersociety Meeting, Global Change & Global Science: Comparative Physiology in a Changing World, will be held August 4—7, 2010, at the Westin Westminster Hotel, 10600 Westminster Blvd., Westminster, CO 80020, telephone (303) 410-5000, FAX: (303) 410-5005.

On-site Registration Hours:

Wednesday, August 4…….2:00—9:00PM
Thursday, August 5 ……...….7:00 AM—6:30 PM
Friday, August 6……..7:30 AM—6:30 PM
Saturday, August 7 ….....7:30 AM—5:00 PM

On-Site Registration Fees (in US Dollars):

APS Member..... $450
Retired Member $215
Nonmember..... $500
Postdoctoral..... $350
Student $300

The registration fee includes entry into all scientific sessions, opening reception and closing banquet.

Payment Information:

Registrants may pay by institutional or personal check, traveler's check, MasterCard, VISA or American Express. Checks must be payable to *"The American Physiological Society"* and drawn on a United States bank payable in US dollars.

Student Registration:

Any student member or regularly matriculated student working toward a degree in one of the biomedical sciences is eligible to register at the student fee. Nonmember postdoctoral fellows, hospital residents and interns, and laboratory technicians do not qualify as students. Nonmember students who register on-site must provide a valid university student ID card. APS student members should present their current APS membership card indicating their student category status.

Postdoctoral Registration:

Any person who has received a Ph.D. degree in physiology or related field, within five years of this conference, as attested to by the department head is eligible to register at the postdoctoral fee. **A statement signed by the department head must accompany the registration form and remittance when registering.**

Press:

Press badges will be issued at the APS registration desk, only to members of the working press and freelance writers bearing a letter of assignment from an editor. Representatives of allied fields (public relations, public affairs, etc.) must register as nonmembers.

Program Objective:

The comparative approach to physiology seeks to use the diversity of organisms as a framework for studying physiological processes. Ranging from molecular mechanisms of osmoregulation or nutrient transport, to study of the evolution of physicological traits, comparative physiology explicitly recognizes that unifying principles can be derived from studies of animals from diverse environments and evolutionary histories.

This conference aims to exemplify this breadth of approaches and applications. Some sessions will highlight technical advances while others will emphasize areas of biomedicine that could receive important insights from comparative physiology. The program will highlight recent accomplishments and will provide a forum to showcase new directions and approaches.

Target Audience:

This meeting is intended for all professionals involved in teaching, research and clinical fields related to comparative physiology.

This meeting has been made possible through the generous support from:

National Science Foundation

Society for Experimental Biology

Company of Biologists/Journal of Experimental Biologists

AJP-Regulatory, Integrative and Comparative Physiology

NIH, National Institute of Diabetes and Digestive and Kidney Diseases

Australian & New Zealand Society for Comparative Physiology & Biochemistry

Sable Systems International

Springer-Verlag GMbH

Univ. of Chicago Press/PBZ

John Wiley & Sons/ Journal of Experimental Zoology Part A

WEDNESDAY, AUGUST 4, 2010

Plenary Lecture

1.0 **PLENARY LECTURE**
Wednes., 8:00-9:00 PM, Standley Ballroom.

Sponsored by *The Company of Biologists.*

8:00 PM **1.1** Comparative Physiology: A "Crystal Ball" for Predicting Consequences of Global Change. **George Somero.** *Stanford Univ.*

THURSDAY, AUGUST 5, 2010

Symposia

2.0 **THE ROLE OF PHYSIOLOGY IN ORGANISMAL RESPONSES TO GLOBAL WARMING**
Thurs., 8:00 AM-12:00 Noon, Standley Ballroom.

Co-Chairs: **Ray Huey,** *Univ. of Washington.*
Melanie Frazier, *U.S. Environmental Protection Agency.*
Warren Porter, *Univ. of Wisconsin.*

8:00 AM **2.1** Introduction. **Ray Huey.** *Univ. of Washington.*

8:05 AM **2.2** Integrating Physiology and Genomics in the Study of Geographic Ranges and Geographic Responses to Climate Change. **Jessica J. Hellmann.** *Univ. of Notre Dame.*

8:30 AM **2.3** Models of Climate Change. What Aspects Will Change and How Fast? **Curtis Deutsch.** *UCLA.*

8:55 AM **2.4** Effects of Climate Change in Bioenergetic Costs of Migration in Chinook Salmon, and Consequences for Life-history Diversity in the Columbia River Basin. **Lisa Crozier.** *Natl. Marine Fisheries.*

9:20 AM **2.5** Animals are not Glaciers: Targets of Selection During Rapid Climate Change. **Bill Bradshaw and Chris Holzapfel.** *Univ. of Oregon.*

9:45 AM Break.

10:10 AM **2.6** Using Biogeographic Distributions and Natural History to Predict Marine/estuarine Species at Risk to Climate Change. **Henry Lee, II and Deborah A. Reusser.** *U.S. Environmental Protection Agency.*

10:35 AM **2.7** Integrating Momentum, Heat and Mass Balances in Leatherback Sea Turtles: Body Size and Oceanic Distribution Consequences Under Climate Change. **Warren Porter.** *Univ. of Wisconsin.*

11:00 AM **2.8** Integrating Biophysical, Population Dynamic and Evolutionary Models in the Context of Climate Change. **Michael Kearney.** *Melbourne Univ., Australia.*

11:25 AM **2.9** Physiology and Genomics of Animal Dispersal in a Metapopulation: Present Function and Relevance for Future Range Shifts. **James Marden.** *Pennsylvania State Univ.*

Symposia

3.0 **OFF THE BEATEN PATH: INTEGRATIVE ASPECTS OF MUSCLE FUNCTION DURING DIVERSE LOCOMOTOR BEHAVIORS**
Thurs., 8:00 AM-12:00 Noon, Westminster Ballroom I.

Co-Chairs: **Tim Higham,** *Clemson Univ.*
Andy Biewener, *Harvard Univ.*

8:00 AM **3.1** Introduction. **Tim Higham.** *Clemson Univ.*

8:05 AM **3.2** Muscles, Morphology and Movement: Dynamics of Running in Vertebrates. **Tim Higham.** *Clemson Univ.*

8:30 AM **3.3** Muscular Control of Avian Flight. **Andy Biewener.** *Harvard. Univ.*

8:55 AM **3.4** Neuromuscular Strategies for the Transitions Between Level and Hill Surfaces. **Jinger Gottschall.** *Pennsylvania State Univ.*

9:20 AM **3.5** Red Muscle Function in Stiff-bodied Swimmers: There and Almost Back Again. **Doug Syme.** *Univ. of Calgary, Canada.* **Robert Shadwick.** *Univ. of British Columbia, Canada.*

9:45 AM Break.

10:10 AM **3.6** Neuromechanical Deterinates of Recruitment within and Between Muscles During Cyclic Locomotion. **James Wakeling.** *Simon Fraser Univ., Canada.*

10:35 AM **3.7** Control of Stable Bipedal Running: Integration of Experimental and Computational Approaches for New Insights. **Monica Daley.** *Royal Vet. Coll., UK.*

11:00 AM **3.8** The Weak Link: Do Muscle Properties Determine Locomotor Performance in Frogs? **Tom Roberts.** *Brown Univ.*

11:25 AM **3.9** Achieving Control: Interpreting Neural Feedback Through Changes in Muscle Function for Stability and Maneuvering in Locomoting Insects. **Simon Sponberg.** *Univ. of Washington.*

Don't forget…
Pick up your Banquet Tickets by 10:00 AM on Thursday at the APS Registration Desk

The banquet tickets are free but you MUST have a ticket for entry

Symposia

4.0 **HYDRATION, DESICCATION, REGULATION: THE COMPARATIVE PHYSIOLOGY OF WATER BALANCE**
Thurs., 8:00 AM-12:00 Noon, Westminster Ballroom II.

Chairs: **David Goldstein,** *Wright State Univ.*
Joe B. Williams, *Ohio State Univ.*

8:00 AM **4.1** Introduction. **David Goldstein.** *Wright State Univ.*

8:05 AM **4.2** Energy and Water Regulation in Avian Nectarivores: Insights from the Geometric Framework. **Angela Köhler.** *Univ. of Pretoria, South Africa.*

8:30 AM **4.3** Renal Adaptations to Dehydration in Desert Mammals. **Thomas Pannabecker.** *Univ. of Arizona.*

8:55 AM **4.4** Evolution of Ion-motive ATPase Transporters During Transitions from Saline to Freshwater Environments: Evidence from Natural Invasions and Laboratory Selection. **Carol E. Lee.** *Univ. of Wisconsin.*

9:20 AM **4.5** Adjustment of Cutaneous Water Loss to Changing Environments. **Joe B. Williams.** *Ohio State Univ.*

9:45 AM Break.

10:10 AM **4.6** Vertebrate Freeze-tolorance: An Osmoregulatory Perspective. **David Goldstein.** *Wright State Univ.*

10:35 AM **4.7** Dehydration and Related Adaptations of Insects to Subzero Temperature. **Jack G. Duman.** *Univ. of Notre Dame.*

11:00 AM **4.8** Water Homeostasis in Nectar-feeding Vertebrates: Out of the Lake and Into the Ocean. **Bradley H. Bakken.** *Kansas State Univ.*

11:25 AM **4.9** Hot and Dry: Molecular Regulation of Estivation. **Ken Storey.** *Carleton Univ., Canada.*

Oral Presentations

5.0 SELECTED ORAL PRESENTATIONS I

Thurs., 1:30-3:30 PM, Westminster Ballroom I.

Co-Chairs: **Blair Wolf,** *Univ. of New Mexico.*
Dan Hahn, *Univ. of Florida.*

1:30 PM **5.1** Cardiac Function in Tunas: The Role of Excitation Contraction Coupling in Building a Thermal Tolerant Fish Heart. **Barbara Block.** *Stanford Univ.*

1:45 PM **5.2** Interplay Between Intestinal Performance and Microbiota Community in an Intermittent Feeder. **Stephen. Secor.** *Univ. of Alabama.*

2:00 PM **5.3** Climate Change Increases the Likelihood of Catastrophic Avian Mortality Events During Extreme Heat Waves and Droughts. **Blair Wolf.** *Univ. of New Mexico.*

2:15 PM **5.4** Light at Night and Comparative Physiology in a Changing World. **Abraham Haim.** *Univ. of Haifa, Israel.*

2:30 PM **5.5** Will Winter Provide a Limit for an Invasive Species? A Bottom-up Approach for the Emerald Ash Borer. **Brent Sinclair.** *Univ. of Western Ontario, Canada.*

2:45 PM **5.6** From Global to Microclimate Changes: Biophysics Reveals Buffering Mechanisms. **Sylvain Pincebourde.** *CNRS, Tours, France.*

3:00 PM **5.7** Metabolic Mechanisms Mediate the Miserable Months: Seasonal Diapause Promotes Speciation in the Apple Maggot. **Daniel Hahn.** *Univ. of Florida, Gainesville.*

3:15 PM **5.8** Effects of Temperature on Performance and Reproduction of a Sierra Willow Beetle: Implications for Population Persistence in Face of Climate Change. **Elizabeth Dahlhoff.** *Santa Clara Univ.*

Workshop

6.0 OPPORTUNITIES FOR COMPARATIVE PHYSIOLOGISTS: BUILDING LINKS WITH AGENCIES, INSTITUTIONS AND FOUNDATIONS

Thurs., 1:30-3:30 PM, Westminster Ballroom II.

Chair: **Jon Harrison,** *Arizona State Univ.*

1:30 PM **6.1** Introduction. **Jon Harrison.** *Arizona State Univ.*

1:35 PM **6.2** Building a Diversified Research Portfolio in Physiological Ecology. **William Karasov.** *Univ. of Wisconsin.*

2:00 PM **6.3** Opportunities for Comparative Physiologists: Building Bridges with Zoo and Aquarium Communities for Research and Species Conservation. **Laura Yeates.** *Natl. Marine Mammal Fdn.*

2:25 PM **6.4** Scientific Contribution to Endangered Species Management: Devils Hole, Desert Fishes and Climate Change. **Stanley Hillyard.** *Univ. of Nevada, Las Vegas.*

2:50 PM **6.5** Climate Change and Physiology: New Exciting Opportunities for Sponsored Research at NSF. **Richard Zimmer.** *Inte-grated Organismal Sys. and Natl. Sci. Fdn.*

Poster Session

7.0 POSTERS IN RESPIRATORY PHYSIOLOGY, THERMAL PHYSIOLOGICAL ECOLOGY, MUSCLE/LOCOMOTORY PHYSIOLOGY AND EVOLUTIONARY PHYSIOLOGY

Thurs., 3:30-6:30 PM, Westminster Ballroom III.

Board #

1 **7.1** Atmospheric Hypoxia Increases Bone Robusticity in the American Alligator. **T. Owerkowicz, F. Andrade, R. Elsey, K. Middleton and J. Hicks.** *Univ. of California, Irvine, Louisiana Dept. of Wildlife & Fisheries and California State Univ.*

2 **7.2** Chronic Exercise Does Not Alter Limb Bone Morphology or Microstructure in the American Alligator. **T. Owerkowicz, H. Tsai, L. Sanchez, K. Felbinger, F. Andrade, J. Blank, J. Eme, J. Gwalthney and J. Hicks.** *Univ. of California, Irvine and Cal. Poly. State Univ.*

3 **7.3** The Metabolic Changes Associated with Gravidity in an *Oviparous squamate.* **A. Jackson and J. Hicks.** *Univ. of California, Irvine.*

4 **7.4** Respiratory Vasculature of the Mudskipper, *Periophthalmodon schlosseri* (Gobiidae: Oxudercinae). **T. Gonzales, M. Katoh, M. A. Ghaffar and A. Ishimatsu.** *Nagasaki Univ., Seikai Natl. Fisheries Res. Inst., Japan and Univ. of Kebangsaan, Malaysia.*

5 **7.5** Effect of Temperature on the Cardiorespiratory System in Grass Shrimp *Palaemonetes pugio.* **T. Mika and C. Reiber.** *Univ. of Nevada, Las Vegas.*

6 **7.6** Seasononal Variation of Baroreflex Activity in the Black and White Tegu Lizard. **K. C. Bicego, V. Dantonio, A. G. Toledo, L. H. Gargaglioni, A. S. Abe and D. O. Andrade.** *São Paulo State Univ. and Natl. Inst. of Sci. & Tech. in Comparative Physiology, Brazil.*

7 **7.7** Relationship Between Serum Iron Levels and Mass and Hb in Vertebrates. **G. Birchard.** *George Mason Univ.*

Board #

8 **7.8** Measuring the Oxidative Cost of Breathing: A Comparison of Methods Using Red-eared Sliders *Trachemys scripta elegans*. **S. Lee and W. K. Milsom.** *Univ. of British Columbia, Canada.*

9 **7.9** Do Semifossorial Rodents Pay an Energetic Cost for Their Ability to Tolerate High Burrow PCO_2? **I. Brickner-Braun and B. Pinshow.** *Ben Gurion Univ. of the Negev., Israel.*

10 **7.10** The Mechanisms of Oxygen Effects on the Fruit Fly *Drosophila melanogaster*. **E. Heinrich and J. Harrison.** *Arizona State Univ.*

11 **7.11** The Effect of Paleozoic Oxygen Levels on the Development of the Tracheal System in the Extant *Blatella germanica*, the German Cockroach. **E. Munoz, M. Weed and J. VandenBrooks.** *Arizona State Univ.*

12 **7.12** A Proposed National Facility for the Study of Global Climate Change: The Variable Atmosphere Laboratory. **J. VandenBrooks and J. Harrison.** *Arizona State Univ.*

13 **7.13** Vulnerable Life Stages Under Siege of Climate Change: Ontogenesis of Eurythermy in Carcinus spp. **F. Giomi, F. Bartolini, A. Barausse and H-O. Pörtner.** *Alfred-Wegener Inst. for Polar & Marine Res., Bremerhaven, Germany, Univ. of Firenze and Univ. of Padova, Italy.*

14 **7.14** Thermal Tolerances in Andean Frogs: Are High-elevation Species More Vulnerable to Climate Warming? **A. Catenazzi and E. Ledr.** *Univ. of California, Berkeley and Illinois Wesleyan Univ.*

15 **7.15** Physiological and Ecological Basis of Recruitment Success in Populations of a Stream-breeding Frog. **A. Catenazzi and S. Kupferberg.** *Univ. of California, Berkeley and Questa Eng., Richmond, CA.*

16 **7.16** Physiological Predictors of Coral Growth After Thermal Stress. **A. Parrin, S. Netherton, L. Bross and N. Blackstone.** *Northern Illinois Univ.*

17 **7.17** Upper Thermal Limits of Growth in Brook Trout (*Salvelinus fontinalis*) and their Relation to Stress Physiology. **J. Chadwick, Jr. and S. D. McCormick.** *Univ. of Massachusetts, Amherst and Conte Anadromous Fish Res. Ctr., Turner Falls, MA.*

18 **7.18** Hemoglobin Expression Influences Thermal Tolerance of Antarctic Notothenioid Fishes. **J. Beers and B. Sidell.** *Univ. of Maine.*

19 **7.19** Physiological Responses to Thermal Ramping in Three Life Stages of a Tenebrionid Beetle. **A. Vorhees.** *Univ. of California, Irvine.*

20 **7.20** Global Analysis of Thermal Tolerance and Latitudinal Range Size in Ectotherms. **J. M. Sunday, A. E. Bates, and N. K. Dulvy.** *Simon Fraser Univ., Canada and Portobello Marine Lab., Dunedin, New Zealand.*

21 **7.21** Intraspecific Variation in Thermotolerance of the Invasive European Green Crab, *Carcinus maenas*, on the West Coast of North America. **A. Kelley and B. Buckley.** *Portland State Univ.*

Board #

22 **7.22** The "Weakest Link" in Thermal Tolerance: A Comparative Study of Neuronal and Cardiac Thermal Tolerance Limits in Porcelain Crabs, *genus Petrolisthes*. **N. Miller and J. Stillman.** *San Francisco State Univ.*

23 **7.23** Can Walking Hibernation Help Polar Bears (*Ursus maritimus*) Cope with Climate Change? **J. Whiteman, H. Harlow and M. Ben-David.** *Univ. of Wyoming.*

24 **7.24** A Physiological Based Model of Landscape Use in Elephants: Interactions Between Thermal Physiology, Water Use, and Energy Demand. **R. Dunkin, M. T. Tinker and T. M. Williams.** *Univ. of California, Santa Cruz and U.S. Geological Survey.*

25 **7.25** Withdrawn.

26 **7.26** Withdrawn.

27 **7.27** The Role of Muscle Plasticity During Cold Acclimation in a Brown Adipose Tissue Deficient Mouse. **P. Mineo and P. Schaeffer.** *Miami Univ.*

28 **7.28** Physiological Differentiation Among Nine-spined Stickleback Populations. **W. Waser.** *Univ. of Turku, Finland.*

29 **7.29** Acclimation, Adaptation or Extinction During Global Warming? Can Comparative Studies of Drosophila Thermo-tolerance Provide an Answer? **J. Overgaard, T. N. Kristensen, V. Kellermann, V. Loeschcke, M. R. Kearney, K. Mitchell and A. A. Hoffmann.** *Aarhus Univ., Denmark and the Univ. of Melbourne, Australia.*

30 **7.30** Physiological and Life History Consequences of Starvation Selection in Drosophila. **L. Reynolds and A. Gibbs.** *Univ. of Nevada, Las Vegas.*

31 **7.31** Transcriptome Analysis of Desiccation in Desert Adapted *Drosophila mojavensis*. **S. Rajpurohit, C. Cardoso de Oliveira, W. J. Etges and A. Gibbs.** *Univ. of Nevada, Las Vegas and Univ. of Arkansas.*

32 **7.32** Heat Acclimation Plasticity in Evolutionary Adapted Desert Rodents: A Lesson From the Heat Shock Response. **A. Abbas, N. Kronfeld-Schor and M. Horowitz.** *The Hebrew Univ. and Tel Aviv Univ., Israel.*

33 **7.33** HSP 70 Expression in the Western Fence Lizard, *Sceloporus occidentalis*: Geographic Variation in Stress and its Role in Sprint Speed Selection. **D. McMillan, B. Rees and D. Irschick.** *Univ. of Massachusetts, Amherst and Univ. of New Orleans.*

34 **7.34** Atlantic Forest Fragmentation and Exercise Physiology in Sub-tropical Anurans. **L. Otani, J. E. Carvalho and C. A. Navas.** *Univ. of São Paulo and Univ. Fed. de São Paulo, Brazil.*

35 **7.35** Unraveling the Causes of Immune Defense Variation Between Life-history Ecotypes of the Garter Snake *Thamnophis elegans*. **M. Palacios, A. Sparkman and A. Bronikowski.** *Iowa State Univ.*

Board #	
36	**7.36** Thermal Safety Margins and Fossoriality During Evolutionary Radiations into Open Habitats: The Case of Gymnophthalmini Lizards at the Brazilian Caatingas. **A. Camacho, A. C. Fonseca-Pinto, C. A. Navas and M. Rodrigues.** *Univ. of São Paulo, City Univ. de São Paulo and Sch. of Vet Med. & Animal Science, São Paulo, Brazil.*
37	**7.37** Effect of Increased Seasonal Temperature on the Reproductive Success of Desert Toads Using Short-term Ephemeral Pools. **J. Kovatch and K. Griffis-Kyle.** *Marshall Univ. and Texas Tech Univ.*
38	**7.38** Changing Sexes in a Complex Environment-Shifts in Skeletal Myosin Isoforms in Coastal Populations of California Sheephead Fish. **J. Bartlett, K. Loke, K. Young, C. Lowe, and B. Rourke.** *California State Univ., Long Beach.*
39	**7.39** A New Tool for Insect Flight Metabolic Rate Studies: Electro-stimulation of Flight in Goliath Beetles. **J. C. Klok, A. Kaiser, J. F. Harrison, H. Sato and M. M. Maharbiz.** *Arizona State Univ., Midwestern Univ. and Univ. of California, Berkeley.*
40	**7.40** Body Weight-sensitive Regulation of Skeletal Muscle Composition. **R. Schilder, S. Kimball, J. Marden and L. Jefferson.** *Pennsylvania State Univ.*
41	**7.41** Force-length Properties of the Lateral Gastrocnemius in the Goat. **M. de Boef Miara, S. Lee, J. Wakeling and A. Biewener.** *Harvard Univ. and Simon Fraser Univ., Burnaby, Canada.*
42	**7.42** Scaling of Metabolism, Growth, and Network Organization in Colonies of the Seed Harvester Ant, *Pogonomyrmex californicus*. **J. Waters and J. F. Harrison.** *Arizona State Univ.*
43	**7.43** Sleep and Diving-Associated Apneas do not Cause Systemic Oxidative Damage in Northern Elephant Seal Pups. **J. P. Vazquez-Medina, T. Zenteno-Savin, H. Forman, D. Crocker and R. Ortiz.** *Univ. of California, Merced, Ctr. de Investigaciones Biologicas del Noroeste, La Paz, Mexico and Sonoma State Univ.*
44	**7.44** Prolonged Fasting Induces Insulin Resistance in the Northern Elephant Seal (*Mirounga angustirostris*). **J. Viscarra, C. Champagne, J. Vazquez-Medina, J. Minas, D. Crocker and R. Ortiz.** *Univ. of California, Merced, Univ. of California, Santa Cruz and Sonoma State Univ.*
45	**7.45** Brown Adipose Tissue and Non-shivering Thermogenesis Aid Harp Seals (*Pagophilus groenlandicus*) but not Hooded Seals (*Cystophora cristata*) at Birth. **L. Pearson, M. Hammill, and J. Burnes.** *Univ. of Alaska, Anchorage and Maurice Lamontagne Inst., Mont-Joli, Canada.*
46	**7.46** The Sublethal Effects of Multiple Freeze-thaw Cycles in the Woolly Bear Caterpillar *Pyrrharctia isabella*. **K. Marshall and B. Sinclair.** *Univ. of Western Ontario, Canada.*
47	**7.47** Comparative Proteomics: Analyzing the Response of the Ascidian Congeners *Ciona intestinalis* and *C. savignyi* to Acute Heat Stress. **L. Serafini and L. Tomanek.** *Cal Poly, San Luis Obispo.*

FRIDAY, AUGUST 6, 2010

Symposia

8.0 **PHYSIOLOGICAL EFFECTS OF OCEAN ACIDIFICATION ON MARINE ANIMALS IN TIMES OF OCEAN WARMING: ECOSYSTEM IMPLICATIONS**

Fri., 8:00 AM-12:00 Noon, Standley Ballroom.

Chair: **Hans-O. Pörtner,** *Alfred-Wegener Inst. for Polar & Marine Res., Bremerhaven, Germany.*

8:00 AM **8.1** Introduction. **Hans-O. Pörtner.** *Alfred-Wegener Inst. for Polar & Marine Res., Bremerhaven, Germany.*

8:05 AM **8.2** Carbonate Formation in Fish Guts: Roles and Consequences. **Rod Wilson.** *Exeter Univ., UK.*

8:30 AM **8.3** Impact of Elevated Temperature and CO_2 on Coral Reef Fish. **Göran Nilsson.** *Univ. of Oslo, Norway.*

8:55 AM **8.4** Patterns of Acid-base Regulation in Crustaceans from Various Habitats. **Jim Barry.** *Monterey Bay Aquarium Res. Inst.*

9:20 AM **8.5** pH Regulation in Reef Corals: Understanding Fundamental Physiology in an Era of Global Change. **Alex A. Venn.** *Monaco Scientific Ctr.*

9:45 AM Break

10:10 AM **8.6** Predicting the Consequences of Ocean Acidification-How Environmental Genomics can Contribute. **Gretchen Hofmann.** *Univ. of California, Santa Barbara.*

10:35 AM **8.7** Performance Capacity Affected by Ocean Warming and Acidification: The Link to Ecosystem Change. **Hans-O. Pörtner.** *Alfred-Wegener Inst. for Polar & Marine Res., Bremerhaven, Germany.*

11:00 AM **8.8** Evolutionary Rules in the Brave New Ocean. **Samuel Dupont.** *Kristineberg Marine Lab, Sweden.*

11:25 AM **8.9** What Can the Study of Physiological Response Tell Us About the Future of Marine Biodiversity and Ecosystem Function in a High CO_2 World? **Stephen Widdicombe.** *Plymouth Marine Lab & Univ., UK.* **John I. Spicer.** *Plymouth Marine Lab & Univ., UK.*

Join us for the

APS Intersociety Meeting Opening Reception

North Courtyard at 6:00 PM (Weather permitting)

Symposia

9.0 **EVALUATING THE EFFECTS OF GLOBAL CLIMATE TO THE FAUNA OF SOUTH AMERICAN BIOMES: CONSEQUENCES TO DIVERSITY FROM A PHYSIOLOGICAL PERSPECTIVE**

Fri., 8:00 AM-12:00 Noon, Westminster Ballroom I.

Co-Chairs: **Carlos A. Navas,** *Univ. of São Paulo, Brazil.*
José E. Carvalho, *Univ. of São Paulo, Brazil.*

8:00 AM **9.1** Introduction. **Carlos A. Navas.** *Univ. of São Paulo, Brazil.*

8:05 AM **9.2** Amphibian Chytridiomycosis and Climate Change: The Need for Integrative Studies in the Context of Anuran Conservation. **Luis Felipe de Toledo.** *Univ. of Campinas, Brazil.*

8:30 AM **9.3** Impact of Thermal Variation on the Life-history Traits of Patagonian Herpetofauna. **Carla Piantoni.** *Univ. of São Paulo, Brazil.*

8:55 AM **9.4** Thermal and Hydric Consequences for the Physiology and Natural History of Select Frogs and Snakes of Brazil. **Glenn Tattersall.** *Brock Univ., Canada.*

9:20 AM **9.5** Anurans in South American Extreme Environments: Convergence, Divergence, and Implications for Global Warming. **José E. Carvalho.** *Univ. of São Paulo, Brazil.*

9:45 AM **9.6** Dry and Getting Dryer: Physiological and Ecological Aspects of Anuran Aestivation in the Brazilian Semi-arid. **José E. Carvalho.** *Univ. of São Paulo, Brazil.*

10:10 AM Break

10:35 AM **9.7** Studies of Fish Reproductive Physiology as a Tool to Understand the Consequences of Fragmentation in Aquatic Habitats. **Renata Moreira.** *Univ. of São Paulo, Brazil.*

11:00 AM **9.8** Global Metabolic Impacts of Recent Climate Warming. **Michael E. Dillon.** *Univ. of Wyoming.*

Symposia

10.0 **ENVIRONMENTAL ADAPTATIONS OF CARDIO-RESPIRATORY SYSTEMS**

Fri., 8:00 AM-12:00 Noon, Westminster Ballroom II.

Co-Chairs: **Peter Frappell,** *Univ. of Tasmania, Australia.*
Art Woods, *Univ. of Montana.*

8:00 AM **10.1** Introduction. **Art Woods.** *Univ. of Montana.*

8:05 AM **10.2** Influence on Oxygen, Temperature and Water on Insect Physiological Plasticity: Life in Leaf Boundary Layers. **Art Woods.** *Univ. of Montana.*

8:30 AM **10.3** Effects of Environmental Oxygen on Cardiopulmonary Development and Function in the American Alligator: A Case Study in Experimental Paleophysiology. **Tomasz Owerkowski.** *Univ. of California, Irvine.*

8:55 AM **10.4** Developmental and Evolutionary Responses of Insects to Environmental Stress. **Jon F. Harrison.** *Arizona State Univ.*

9:20 AM Break

9:45 AM **10.5** Cardio-respiratory Environmental Acclimation in Developing Animals. **Warren Burggren.** *Univ. of North Texas.*

10:10 AM **10.6** Adaptations to High Elevations in Hummingbirds. **Doug Altshuler.** *Univ. of California, Riverside.*

10:35 AM **10.7** Mechanisms and Evolution of Hypoxia Tolorance. **Jeff Richards.** *Univ. of British Columbia.*

11:00 AM **10.8** Physiological Mechanisms of High-altitude Flight in Bar-headed Geese. **Graham Scott.** *St. Andrew's Univ., UK.*

11:25 AM **10.9** Individual and Population Differences in Behavioral Physiology and Oxygen Delivery in Daphnia. **Peter Frappell.** *Univ. of Tasmania, Australia.*

Scholander Oral Presentations

11.0 **SCHOLANDER ORAL PRESENTATIONS**

Fri., 1:30 - 3:30 PM, Standley Ballroom.

Chair: **Jon F. Harrison.** *Arizona State Univ.*

1:30 PM **11.1** Introduction. **Jon F. Harrison.** *Arizona State Univ.*

1:35 PM **11.2** Environmental and Handling Impacts on Cortisol in the Northern Elephant Seal. **Cory Champagne.** *Univ. of California, Santa Cruz.*

1:45 PM **11.3** Constraining the Thermal Limits of Metazoan Life: Comparing Thermally Induced Shifts in Global Protein Expression in Vent Worms *Paralvinella sulfincola* and *Paralvinella palmiformis*. **Geoffrey Dilly.** *Harvard Univ.*

1:55 PM **11.4** Thermal Stress, Oxygen Limitation, and Protein Damage in Intertidal Mussels. **Wes Dowd.** *Stanford Univ.*

2:05 PM **11.5** Using Mitochondrial Bioenergetics to Determine if Good Fish Live in Bad Neighborhoods. **Martin Jastroch.** *Buck Inst. for Age Res., Navato, CA.*

2:15 PM **11.6** Effects of Temperature Acclimation on Cardiac Performance of Longjaw Mudsuckers *Gillichthys mirabilis*. **Nishad Jayasundara.** *Stanford Univ.*

2:25 PM **11.7** Birds vs. Mammals: Blood Oxygen Management Strategies in Divers. **Jessica Meir.** *Univ. of British Columbia, Canada.*

2:35 PM **11.8** Peripheral Arterial Chemoreceptors in the South American Rattlesnake (*Crotalus durissus*) and their Role in Cardio-respiratory Control. **Catalina Reyes.** *Univ. of British Columbia, Canada.*

2:45 PM **11.9** Can Deep-sea Fishes Keep Pace with Ocean Acidification? CO_2 Tolerance in Deep and Shallow-water Rockfishes from Monterey Bay, CA. **Josi Taylor.** *Monterey Bay Aquarium Res. Inst.*

2:55 PM **11.10** Energetic Costs of Increasing Thermal Variability: An Exploration of the Mitigating Effects of Plasticity in Lepidoptera. **Caroline Williams.** *Univ. of Western Ontario, Canada.*

3:05 PM **11.11** Locomotory Cost and Myoglobin Desaturation in Diving Emperor Penguins. **Cassondra Williams.** *Univ. of California, San Diego.*

Poster Session

12.0 POSTERS IN GENOMICS AND PROTEOMICS, RESPONSES TO HYPOXIC, THERMAL AND OSMOTIC STRESS, NUTRITIONAL PHYSIOLOGY AND OCEAN ACIDIFICATION

Fri., 3:30 - 6:30 PM, Westminster Ballroom III.

Sponsored by *AJP-Regulatory, Integrative and Comparative Physiology*

Board #

1 **12.1** Functional Characterization of Hypoxia-inducible Factors from the Killifish, *Fundulus heteroclitus*. **B. Rees and I. Townley.** *Univ. of New Orleans.*

2 **12.2** Cardiorespiratory Function and Hypoxia Tolerance in Elasmobranchs. **B. Speers-Roesch, C. J. Brauner, A. P. Farrell, A. J. R. Hickey, G. M. C. Renshaw, Y. Wang and J. G. Richards.** *Univ. of British Columbia, Canada, Univ. of Auckland, New Zealand, Giffith Univ., Southport, Australia and Queen's Univ., Kingston, Canada.*

3 **12.3** Lactate Kinetics in Rainbow Trout During Acute Hypoxia. **T. Omlin and J-M. Weber.** *Univ. of Ottawa, Canada.*

4 **12.4** Mechanisms Underlying the Crucian Carp's Remarkable Feat to Maintain Cardiac Pumping During Anoxia. **J. Stecyk, K-O. Stensløkken, L. Hanson, B. Larsen, A. Farrell and G. Nilsson.** *Univ. of Oslo, Norway and Univ. of British Columbia, Canada.*

5 **12.5** Cardiorespiratory and Metabolic Response to Acute Hypoxia in Atlantic Salmon Alevins Acclimated to 4°C and 8°C. **E. Polymeropoulos, N. Elliott and P. Frappell.** *Univ. of Tasmania and Natl. Food Futures Flagship, Salamanca, Australia.*

6 **12.6** The Role of AMP-activated Protein Kinase in Coordinating Metabolic Rate Suppression in Hepatocytes from the Common Goldfish, *Carassius auratus*. **G. Lau and J. Richards.** *Univ. of British Columbia, Canada.*

7 **12.7** Seasonal plasticity of GABAergic synaptic transmission in *Lymnaea stagnalis*. **L. T. Buck and A. Malik.** *Univ. of Toronto, Canada.*

8 **12.8** Transcriptomic Responses of Porcelain Crabs to Thermal Acclimation. **P. Rossignol, C. T. Miranda and J. H. Stillman.** *San Francisco State Univ.*

9 **12.9** Temperature Induced Changes in the Expression of the Enzymes Involved in Membrane Restructuring. **S. Kunciw and E. Williams.** *Salisbury Univ.*

10 **12.10** Development and Use of an *in vitro* Tool for Assessing Mechanisms Underlying the Osmotic Stress Response in a *Euryhaline teleost*. **A. Gardell, R. Sacchi and D. Kültz.** *Univ. of California, Davis.*

Board #

11 **12.11** Effects of Acute Temperature and Salinity Stresses on the Transcriptomes of Invasive and Native Mussel Species (*Genus mytilus*). **B. Lockwood, J. G. Sanders and G. N. Somero.** *Stanford Univ. and Harvard Univ.*

12 **12.12** Gas Exchange Variation in the Alfalfa Leafcutter Bee, *Megachile rotundata*, in Response to Fluctuating Thermal Regimes. **K. J. Greenlee, J. P. Rinehart and G. D. Yocum.** *North Dakota State Univ. and USDA.*

13 **12.13** Transcriptomics of Environmental Adaptation in Wild Migrating Adult Pacific Sockeye Salmon, *Oncorhynchus nerka*. **T. Evans, E. Hammill, K. Kaukinen, A. Schulze, D. Patterson and K. Miller.** *Pacific Biological Stn., British Columbia and Simon Fraser Univ., Burnaby, Canada.*

14 **12.14** Heat Induction of C/EBP-delta in Two Fishes: New Insights into the Cellular Stress Response. **I. Sleadd and B. Buckley.** *Portland State Univ.*

15 **12.15** Qualitative Characterization of a Novel NHE2c Protein in the Marine Longhorn Sculpin (*Myoxecephalus octodecem spinosus*) After Exposure to Adverse Environmental Conditions Emerging in Ocean Waters. **D. Rabeneck, A. Diamanduros and J. Claiborne.** *Georgia Southern Univ.*

16 **12.16** Stress Resistance and Resilience in the Coral *Acropora hyacinthus*: Gene Expression Analysis Based on High-throughput Sequencing. **D. Barshis, T. Oliver and S. Palumbi.** *Stanford Univ.*

17 **12.17** The Impact of Temperature Stress on Hemocytes of *Mytilus californianus*: DNA Damage and Signaling Pathways. **C. Yao and G. N. Somero.** *Stanford Univ.*

18 **12.18** Increased Antioxidant Activity in the Caribbean Fruit Fly, *Anastrepha suspensa*, Serves a Protective Role During Irradiation. **G. Lopez-Martinez and D. A. Hahn.** *Univ. of Florida, Gainesville.*

19 **12.19** Apoptosis-related Genes Respond Uniquely to UV Radiation and Cold Stress in the Fly *Sarcophaga crassipalpis*. **J. Reynolds and D. Denlinger.** *Ohio State Univ.*

20 **12.20** Using Stable Isotopes to Track Exogenous Nutrient Allocation and Routing in Postprandial House Sparrows. **M. McCue, B. Pinshow and S. McWilliams.** *Jacob Blaustein Inst. for Desert Res., The Negev, Israel and Univ. of Rhode Island.*

21 **12.21** Ontogeny and Nutritional Status Influence Oxidative Kinetics of Exogenous Nutrients and Whole-animal Bioenergetics in Zebra Finches. **M. McCue, S. McWilliams and B. Pinshow.** *Jacob Blaustein Inst. for Desert Res., The Negev, Israel and Univ. of Rhode Island.*

22 **12.22** Isotope Dilution and Body Composition: Updating a Tool for Nutritional Ecology. **R. Eisert, O.**

Board #

Oftedal, W. Hood and A. Mitchell. *Smithsonian Inst., Auburn Univ. and USDA.*

23 **12.23** The Effect of Rice Bran Arabinoxylan Compound on Glucose Metabolism in Skeletal Muscle. **K. Sato, F. Kimura, K. Shimizu, S. Ikemune, I. Kono and R. Ajisaka.** *Ritsumeikan Univ., Kusatsu and Univ. of Tsukuba, Japan.*

24 **12.24** Torpor Flexibility in Response to Environmental Constraints: Food Shortages and Immune Challenge. **C. Canale and P-Y. Henry.** *UMR 7179 CNRS-MNHN-CF, Brunoy, France.*

25 **12.25** Patterns of Gut Size and Digestive Enzyme Activity with Chronic Food Restriction in Nestling House Sparrows. **T. Killpack and W. Karasov.** *Univ. of Wisconsin, Madison.*

26 **12.26** Dietary Polyunsaturated Fatty Acids, Endurance Flight, and Mitochondrial Function in European Starlings (*Sturnus vulgaris*). **A. Gerson, S. R. McWilliams, U. Bauchinger, J. F. Staples, M. L. Boyles, L. A. Langlois, E. R. Price, B. Pierce and C. G. Guglielmo.** *Univ. of Western Ontario, Canada, Univ. of Rhode Island and Sacred Heart Univ., Fairfield, CT.*

27 **12.27** Increased Life Span From Ovariectomy May be Due to Dietary Restriction in Grasshoppers. **J. Hatle, M. Drewry and J. M. Williams.** *Univ. of North Florida, Jacksonville.*

28 **12.28** Maximal Metabolic Rate in Birds Tracks Daily Energy Expenditure. **P. Schaeffer, C. Sgueo, D. Wagner, M. Wikelski, P. Mineo and D. Russell.** *Miami Univ. and Princeton Univ.*

29 **12.29** Estimating the Potential for Adaptation to Ocean Acidification in Two Coastal Species. **J. M. Sunday, R. Crim, C. D. G. Harley and M. W. Hart.** *Simon Fraser Univ., Burnaby, and Univ. of British Columbia, Canada.*

30 **12.30** Effects of Ocean Acidification on De-velopment and Growth of Porcelain Crab Larvae. **L. Ceballos, H. Carter, J-C. Breach, N. Miller and J. Stillman.** *San Francisco State Univ.*

31 **12.31** Effect of Ocean Acidification on the Metabolism of Porcelain Crab Larvae. **H. Carter, L. Ceballos, J-C. Breach, N. Miller and J. Stillman.** *San Francisco State Univ.*

32 **12.32** Physiological Effects of Elevated Temperature and Ocean Acidification on the Purple Sea Urchin, *Strongylocentrotus purpuratus*. **N. Fangueand G. Hofmann.** *Univ. of California, Davis and Univ. of California, Santa Barbara.*

33 **12.33** CO_2/HCO_3^-/pH Sensing via Soluble Adenylyl Cyclase. **M. Tresguerres, J. Buck and L. Levin.** *Weill Cornell Med. Coll.*

34 **12.34** Species Differences in the Effects of Exercise on the Stability of the Glycolytic Enzyme LDH in Porcelain Crabs. **H. Medina-Ruiloba and J. Stillman.** *San Francisco State Univ.*

35 **12.35** Metabolic Responses to Environmental Salinity in the Invasive Clam *Corbula amurensis*. **A. Paganini, J. Stillman and W. Kimmerer.** *San Francisco State Univ.*

36 **12.36** Is There a Synergistic Effect of Thermal and Osmotic Stress on Metabolic Performance in Freshwater Zooplankton? **X. Chen and J. Stillman.** *San Francisco State Univ.*

37 **12.37** Proteomic Analysis of Acute Salinity Stress in the Two Ascidian Species *Ciona savignyi* and *C. intestinalis*. **J. Koman and L. Tomanek.** *Cal Poly, San Luis Obispo*

38 **12.38** The Role of Ion and Water Homeostasis in Chill-coma and Chilling Injury of the Fall Field Cricket. **H. MacMillan and B. Sinclair.** *Univ. of Western Ontario, Canada.*

39 **12.39** Phylogenetic Differences of Mammalian Basal Metabolic Rate are not Explained by Lower Mitochondrial Basal Proton Leak. **E. Polymeropoulos, G. Heldmaier, P. Frappell, B. McAllen, K. Withers and M. Jastroch.** *Univ. of Tasmania, Australia, Philipps Univ., Marburg, Germany, Univ. of Sydney, Univ. of Southern Queensland, Toowoomba, Australia, and Buck Inst. of Aging, Novato, CA.*

40 **12.40** Biophysical Modeling of Foraging Behavior: How Climate Change May In-crease Foraging Constraints. **O. Levy, T. Dayan, W. P. Porter and N. Kronfeld-Schor.** *Tel Aviv Univ., Israel and Univ. of Wisconsin.*

41 **12.41** Withdrawn.

42 **12.42** Post-molt Condition of Weddell Seals and Links with Overwinter Diving Behavior. **M. Shero, L. Pearson, K. Goetz, P. Robinson, D. Costa and J. Burns.** *St. Mary's Coll. of Maryland, Univ. of Alaska, Anchorage and Univ. of California, Santa Cruz.*

43 **12.43** Metabolic Effects of Stress in Rural and Urban Male Abert's Towhees. **N. Rodriguez, S. Davies, P. Deviche and K. Sweazea.** *Arizona State Univ.*

SATURDAY, AUGUST 7, 2010

Symposia

13.0 **GENOMICS AND PROTEOMICS APPROACHES TO UNDERSTANDING ENVIRONMENTAL STRESS**

Sat., 8:00 AM - 12:00 Noon, Standley Ballroom.

Co-Chairs: **Dietmar Kültz,** *Univ. of California, Davis.*
Patricia Schulte, *Univ. of British Columbia, Canada.*

8:00 AM **13.1** Introduction. **Dietmar Kültz.** *Univ. of California, Davis.*

8:05 AM **13.2** The Role of Oxidative Stress in Setting Thermal Tolerance Limits: A Proteomics Approach. **Lars Tomanek.** *Cal Poly, San Luis Obispo.*

8:30 AM **13.3** Metabolomic, Transcriptomic, and Physiologic Approaches to Understanding Anoxia Tolerance in Embryos of the Annual Killifish *Austrofundulus limnaeus*. **Jason Podrabsky.** *Portland State Univ.*

8:55 AM **13.4** Transcriptomic Profiling Reveals the Capacity to Respond to Heat Stress in Temperate and

Polar Marine Fishes. **Brad Buckley.** *Portland State Univ.*

9:20 AM **13.5** Genomic Responses to Thermal Stress in Fishes. **Patricia Schulte.** *Univ. of British Columbia, Canada.*

9:45 AM Break

10:10 AM **13.6** Proteomics of Salinity Stress Signaling in Tilapia. **Dietmar Kültz.** *Univ. of California, Davis.*

10:35 AM **13.7** Transcriptomics of Environmental Adaptation in Wild Migrating Adult Pacific Sockeye Salmon, *Oncorhynchus nerka*. **Tyler Evans.** *Pacific Biological Stn., British Columbia, Canada.*

11:00 AM **13.8** Using Transcriptomics to Understand the Impacts of Ocean Acidification on Larval Sea Urchins. **Anne Todgham.** *San Francisco State Univ.*

11:25 AM **13.9** Acclimation and Acclimatization of Porcelain Crab Cardiac Transcriptome Responses to Thermal Stress Across Environmental Gradients in Thermal Variability **Jonathon Stillman.** *San Francisco State Univ.*

Symposia

14.0 **PUTTING COMPARATIVE PHYSIOLOGY TO WORK IN THE FIELD: STABLE ISOTOPES AS TRACER OF ECOLOGICAL PROCESSES**

Sat., 8:00 AM - 10:00 AM, Westminster Ballroom I.

Chair: **Carlos Martinez del Rio,** *Univ. of Wyoming.*

8:00 AM **14.1** Introduction. **Carlos Martinez del Rio.** *Univ. of Wyoming.*

8:05 AM **14.2** Stable Isotopes and Animal Biology: Mechanisms, Processes, and Patterns. **Carlos Martinez del Rio.** *Univ. of Wyoming.*

8:30 AM **14.3** Back to Basics: How Understanding the Dynamics of Isotope Incorporation into Tissues Reveals Much About the Ecology of Migratory Birds. **Scott R. McWilliams.** *Univ. of Rhode Island.*

8:55 AM **14.4** Compound Specific Stable Isotope Analyses: The Power of a New Technology for Ecological Physiology. **Seth Newsome.** *Univ. of Wyoming.*

9:20 AM **14.5** What Can Measuring Isotopes in Breath Tell Us About an Animal's Biology? **Christian Voigt.** *Leibniz Inst. for Zoo and Wildlife Res., Berlin, Germany.*

Don't Forget to Visit the Exhibits Daily during the Poster Sessions

Symposia

15.0 **THE LIFE HISTORY PHYSIOLOGY NEXUS**

Sat., 10:00 AM - 12:00 Noon, Westminster Ballroom I.

Chair: **Joe B. Williams,** *Ohio State Univ.*

10:00 AM **15.1** Introduction. **Joe B. Williams.** *Ohio State Univ.*

10:05 AM **15.2** Linking Physiological Evolution and Life-history Divergence in Natural Populations of Snakes. **Anne Bronikowski.** *Iowa State Univ.*

10:30 AM **15.3** Interaction of Temperature and Embryo Metabolism on Embryonic Periods and Immune Function in Tropical and Temperate Birds. **Tom Martin.** *Univ. of Montana.*

10:55 AM **15.4** Functional Linkages Between Physiology and Life-History in Birds. **Joe B. Williams.** *Ohio State Univ.*

11:20 AM **15.5** Can Camouflage Keep up with Climate Change? Creating a Working Model of Seasonal Color Change in Snow-shoe Hares. **Creagh Breuner.** *Univ. of Montana.*

Symposia

16.0 **THE PHYSIOLOGY OF TRACE METAL HOMEOSTASIS AND TOXICITY**

Sat., 8:00 AM - 10:00 AM, Westminster Ballroom II.

Chair: **Martin Grosell,** *Univ. of Miami.*

8:00 AM **16.1** Introduction. **Martin Grosell.** *Univ. of Miami.*

8:05 AM **16.2** Effect of Ocean Acidification on the Speciation of Metals in Seawater. **Frank Millero.** *Univ. of Miami.*

8:30 AM **16.3** Cadmium Accumulation and Toxicity in Aquatic Insects. **David Buchwalter.** *North Carolina State Univ.*

8:55 AM **16.4** When is Chemical Contamination an Important Factor in Considering Ecological Implications of Climate Change? **Samuel N. Luoma.** *U.S. Geological Survey.*

9:20 AM **16.5** Accounting for Metal Sensitivity Differences Spanning Order of Magnitude. **Martin Grosell.** *Univ. of Miami.*

Symposia

17.0 **GLOBAL CHANGE AND THE EMERGING THREAT OF DEAD ZONES: CAN INDIVIDUALS, POPULATIONS, SPECIES, AND COMMUNITIES RESPOND TO INCREASINGLY HYPOXIC OCEANS?**

Sat., 10:00 AM - 12:00 Noon, Westminster Ballroom II.

Chair: **Andrew Altieri,** *Brown Univ.*

10:00 AM **17.1** Introduction. **Andrew Altieri.** *Brown Univ.*

10:05 AM **17.2** Using Physiological Perspectives to Predict Ecological Responses to Hypoxia: Integrating Individuals to Ecosystems. **Andrew Altieri.** *Brown Univ.*

10:30 AM **17.3** Building a Mechanistic Understanding of Hypoxia's Impact on Coastal Ecosystems: Can Physiological Responses to Hypoxia Predict Population Consequences in a Changing World? **Geoffrey W. Bell.** *North Carolina State Univ.*

10:55 AM **17.4** Hypoxia Does Not Always Generate Dead Zones: Adaptations of Fish to Low-oxygen Habitats. **Göran E. Nilsson.** *Univ. of Oslo, Norway.*

11:20 AM **17.5** Metabolic Suppression in Vertically Migrating Pelagic Animals in Oxygen Minimum Zones. **Brad A. Seibel.** *Univ. of Rhode Island.*

Oral Presentations

18.0 **SELECTED ORAL PRESENTATIONS II**

Sat., 1:30-3:30 PM, Westminster Ballroom I.

Co-Chairs: **Grant McClelland,** *McMaster Univ., Canada.*
Sean Place, *Univ. of California, Santa Barbara.*

1:30 PM **18.1** Phenology of Hibernation and Reproduction in Free-living Populations of Arctic Ground Squirrels in Alaska. **Brian Barnes.** *Univ. of Alaska, Fairbanks.*

1:45 PM **18.2** Fuel Metabolism in Cardiac and Skeletal Muscles of Harp and Hooded Seals. **Jennifer Burns.** *Univ. of Alaska, Anchorage.*

2:00 PM **18.3** Atmospheric Oxygen Level and the Evolution of Insect Body Size. **John M. Vanden-Brooks.** *Arizona State Univ.*

2:15 PM **18.4** Changes in Peak Blood Lactate and Muscle Pyruvate Dehydrogenase Following an Acute to Chronic Hypoxia Transition in CD-1 Mice. **Grant McClelland.** *McMaster Univ., Canada.*

2:30 PM **18.5** A Comparison of Proteomic Responses to Heat, Hyposalinity and Emersion in the Gills of the Ribbed Marsh Mussel *Geukensia demissa*. **Peter Fields.** *Franklin and Marshall Coll., Lancaster, PA.*

2:45 PM **18.6** Effects of Elevated Atmospheric Carbon Dioxide Levels on Shell Formation and Metabolism in Oysters *Crassostrea virginica* (Gmelin). **Inna Sokolova.** *Univ. of North Carolina, Charlotte.*

3:00 PM **18.7** Ocean Acidification Impacts on Cell-cycle Control in *Strongylocentrotus purpuratus* Embryos. **Sean Place.** *Univ. of South Carolina.*

3:15 PM **18.8** Ocean Acidifcation and Hypoxia: A Double Threat to Immune Defense. **Louis Burnett.** *Coll. of Charleston, South Carolina.*

Workshop

19.0 **CAREER PATHS/OPTIONS FOR A COMPARATIVE PHYSIOLOGIST**

Sat., 1:30-3:30 PM, Westminster Ballroom II.

Co-Chairs: **Siribhinya Benyajati,** *Univ. of Oklahoma Hlth. Sci. Ctr.*
Karen L. Sweazea. *Arizona State Univ.*

1:30 PM **19.1** Introduction. **Siribhinya Benyajati.** *Univ. of Oklahoma Hlth. Sci. Ctr.*

1:35 PM **19.2** Taking the Road Most Traveled: Academic Positions. **Karen Sweazea.** *Arizona State Univ.*

2:00 PM **19.3** Comparative Physiology in Government Labs. **Kyle Hanson.** *U.S. Fish and Wildlife Service.*

2:25 PM **19.4** Career Paths Through the U.S. EPA. **Melanie Frazier.** *U.S. Environmental Protection Agency.*

2:50 PM **19.5** Taking the Road Less Traveled: Alternate Career Paths. **Regina Eisert.** *Smithsonian Inst.*

Poster Session

20.0 **POSTERS IN HIBERNATION/TORPOR, AGING/CONDITION, IMMUNE FUNCTION, SENSORY PHYSIOLOGY, FUNCTIONAL MORPHOLOGY, AND RENAL AND OSMOTIC PHYSIOLOGY**

Sat., 3:30 - 6:30 PM, Westminster Ballroom III.

Board #

1 **20.1** Mechanisms of Urea Nitrogen Salvage During Protein Scarcity in a Fast-adapted Hindgut Fermenter, the Wyoming Ground Squirrel *Spermophilus elegans*. **K. Greller and H. Harlow.** *Univ. of Wyoming.*

2 **20.2** Coping with Rapid Changes in Renal Cortico-papillary Gradients During Arousal from Torpor. **C. Cotton and H. Harlow.** *Univ. of Wyoming.*

3 **20.3** Urea-induced Hypometabolism in the Hibernating Wood Frog (*Rana sylvatica*) is not Reflected in Isolated Mitochondria. **T. Muir, J. Costanzo and R. Lee.** *Augustana Coll., Rock Island, IL.*

4 **20.4** Fast In, Slow Out: Kinetics of Mitochondrial Metabolic Suppression During Hibernation Entrance and Arousal. **J. Staples, D. Chung and C. Armstrong.** *Univ. of Western Ontario, Canada.*

5 **20.5** Latitudinal Differences in the Hibernation Characteristics of Woodchucks (*Marmota monax*). **G. Florant, C. Maher and S. Zervanos.** *Colorado State Univ.,Univ. of Southern Maine and Penn State Univ.*

6 **20.6** 'Til Death Do Us Part: Quantifying Predation on Endangered Juvenile Steller Sea Lions in the Gulf of Alaska. **J. Mellish and M. Horning.** *Univ. of Alaska, Fairbank and Oregon State Univ.*

7 **20.7** Health and Condition in the Adult Weddell Seal. **J. Mellish, A. Hindle and M. Horning.** *Univ. of Alaska, Fairbanks, Univ. of Colorado, Denver and Oregon State Univ.*

8 **20.8** Apoptosis but not Oxidative Stress Underlies Skeletal Muscle Senescence in a Free-living Diver the Weddell Seal. **A. Hindle, M. Horning, J. Mellish and J. Lawler.** *Univ. of Colorado, Denver, Oregon State Univ., Univ. of Alaska, Fairbanks and Texas A&M Univ.*

9 **20.9** Energetic Consequences of Maternal Antibody Transfer in Tree Swallows. **G. Burness, D. Moher and R. Kelly.** *Trent Univ., Peterborough, Canada.*

10 **20.10** COX-2 and Inflammatory Gene Expression in Response to *Aspergillus fumigatus* Metabolites in Macrophage Cell Line Raw 64.7 and Mouse Bone Marrow Derived Macrophages. **A. Azim, A. Stalworth and D. Osei-Bonsu.** *Chicago State Univ.*

11 **20.11** Effects of Stress on Indices of Innate Immunity During Development in a Sexually-sized Dimorphic Bird (*Larus delawarensis*). **E. Chin, J. Quinn and G. Burness.** *Trent Univ., Peterborough and McMaster Univ., Canada.*

12 **20.12** Withdrawn.

Board #

13 **20.13** High Altitude Conservation Physiology: Developing Physiological Tools to Measure Environmental Stress in the Sierra Madre Mountains. **I. Hunt von Herbing, M. Garduno, O. Gallegos, F. Mendez and F. Pan.** *Univ. of North Texas and* Univ. Autonoma de Estada de Mexico.

14 **20.14** Vision in Antarctic Euphausiids *Thysanoessa macrura* and *Euphausia superba*: Spectral Sensitivity and Temporal Resolution. **J. Cohen.** *Eckerd Coll., St. Petersburg, FL.*

15 **20.15** Sensory Mechanisms Driving Community Ecological Interactions. **R. Zimmer, G. Ferrier, S. Kim, and C. A. Zimmer.** *Natl. Sc. Fdn. and UCLA.*

16 **20.16** Chemical Communication, Fluid Dynamics, and Mechanisms Driving Sperm-egg Interactions. **R. Zimmer and J. Riffell.** *Natl. Sc. Fdn. and Univ. of Arizona.*

17 **20.17** Functional Morphology, Construction and the Hatching Mechanisms of the 'Sand Collars' of *Euspira lewisii* (Gastropoda: Naticidae). **M. Newel and G. Bourne.** *Univ. of Calgary and Bamfield Marine Sci. Ctr., Bamfield, Canada.*

18 **20.18** Effects of Cadmium on Metabolic Responses to Environmental Hypoxia in Eastern Oysters *Crassostrea virginica* Gmelin. **A. Ivanina, E. Sokolov and I. Sokolova.** *Univ. of North Carolina, Charlotte and Carolinas Med. Ctr., Charlotte.*

19 **20.19** Effects of cadmium on Anaerobic Energy Metabolism and Gene Expression During Air Exposure and Recovery of an Intertidal Mollusk *Crassostrea virginica.* **A. Ivanina, E. Sokolov and I. Sokolova.** *Univ. of North Carolina, Charlotte and Carolinas Med. Ctr., Charlotte.*

20 **20.20** Sites of Cadmium Action in Molluscan Mitochondria and the Role of Oxidative Stress in Cadmium-induced Mitochondrial Dysfunction. **I. Kurochkin, M. Etzkorn, D. Buchwalter, L. Leamy and I. Sokolova.** *Univ. of North Carolina, Charlotte and North Carolina State Univ., Ra-leigh.*

21 **20.21** 3H-L-leucine Influx is Stimulated by Protons and Zn in Lobster Antennal Gland BBMV. **J. Bedford and G. Ahearn.** *Univ. of North Florida, Jacksonville.*

22 **20.22** Ammonium Uptake Inhibits Calcification in a Widespread Marine Coccolithophore. **S. Lefebvre, I. Benner, M. Drake, P. Rossignol, K. Okimura. T. Komada, J. Stillman and E. Carpenter.** *San Francisco State Univ.*

23 **20.23** Variation in Behavioral Aversion to Road Deicers in Sympatric Temperate Zone Amphibian Species. **L. Hazard, K. Kwasek and S. Gerges.** *Montclair State Univ., Montclair, NJ.*

24 **20.24** Drought Tolerance of Pelagic Sea Snakes in Costa Rica. **H. Lillywhite, C. Sheehy, III, J. Pfaller and F. Brischoux.** *Univ. of Florida, Gainesville and Univ. of Texas, Arlington.*

25 **20.25** Importance of a Putative Urea Transporter during Physiological Stress in the Wood Frog, *Rana sylvatica.* **A. Rosendale, J. P. Costanzo and R. E. Lee, Jr.** *Miami Univ.*

Board #

26 **20.26** The Regulatory Role of Insulin in Avian Kidneys. **K. Sweazea, R. Sparr and E. Braun.** *Arizona State Univ. and Univ. of Arizona.*

27 **20.27** Relationships Among Diverse Immune Defense Measures in Free-Living Tree Swallows. **M. G. Palacios, J. Cunnick, D. Winkler and C. Vleck.** *Iowa State Univ. and Cornell Univ.*

28 **20.28** Oxidative Status of the Mice Tissues Following Exposure to deca BDE. **N. Kubeldis and Mary Vagula.** *Gannon Univ., Erie, PA.*

29 **20.29** Thermal Stress During Pre-incubation Induces Subsequent Development Plasticity in Northern Bobwhites. **K. S. Reyna and W. G. Burggren.** *Univ. of North Texas.*

30 **20.30** Withdrawn.

31 **20.31** Evolved Responses to Heat Stress in Snake Life-history Ecotypes. **T. Schwartz and A. Bronikowski.** *Iowa State Univ.*

32 **20.32** Genetic Variation in Nuclear and Mitochondrial Genes Important for Energy Metabolism Vary Along a Climatic Gradient in Montane Populations of a Leaf Beetle. **N. E. Rank, S. Heidl, J. T. Smiley, P. Mardulyn and E. P. Dahlhoff.** *Sonoma State Univ., White Mtn. Res. Stn., Bishop, CA, Free Univ. of Brussels, Belgium and Santa Clara Univ.*

33 **20.33** Evolution of Thermal Sensitivity of a Tropical rainforest Lizard: Are Tropical Species Particulary Vulnerable to Warming? **R. Damasceno and A. C. Carnaval.** *Univ. of California, Berkeley and City Univ. of New York.*

34 **20.34** The Effects of Environmental Pollutants on Innate Immune Function of Amphibians: Nonmonotonic Innate Immune Supression of Adult Red-spotted Newts (*Notophthalmus viridescens*) Exposed to Atrazine. **I. Vatnick, R. Howanski, E. Shank, L. Stashefski and M. Brodkin.** *Widener Univ., Chester, PA.*

35 **20.35** Energetic Status Modulates Vulnerability of Neural Circuits to Environmental Stress. **R. M. Robertson, C. I. Rodgers, G. A. B. Armstrong and T. G. A. Money.** *Queen's Univ., Kingston, Canada.*

Plenary Lecture

21.0 **PLENARY LECTURE**
Sat., 9:00 - 10:00 PM, Westminster Ballroom I/II.

9:00 PM **21.1** Visualizing Change. **Elizabeth Brainerd.** *Brown Univ.*

2010 APS Intersociety Meeting
Global Change and Global Science: Comparative Physiology in a Changing World

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1.0: PLENARY LECTURE

1.1

COMPARATIVE PHYSIOLOGY: A "CRYSTAL BALL" FOR PREDICTING CONSEQUENCES OF GLOBAL CHANGE

George Somero[1]
[1]Hopkins Marine Station, Stanford Univ., 120 Oceanview Blvd, Pacific Grove, CA, 93950.

Comparative physiological approaches offer enormous potential for predicting how global change, including shifts in temperature, dissolved oxygen, ocean acidity and rainfall patterns, will influence the functions and distributions of organisms. Comparative analyses, especially studies of congeneric species with different biogeographic or vertical distributions, can identify the mechanisms underlying differences in environmental optima and tolerance limits and reveal how closely some species approach their tolerance limits in nature. Lessons learned to date from such analyses show that (i) the most warm-adapted congeners of a lineage may be most threatened by further global warming, (ii) cardiac function is apt to be a 'weak link' in the physiological chain, (iii) thermal and osmotic stress interact to govern overall responses to acute environmental change and (iv) adaptive modification of protein stability and function is common in differently adapted congeners, often requiring only one or two amino acid substitutions. Comparative studies of genomes is revealing how long-term evolution in stable thermal conditions, such as those of the Southern Ocean, has led to loss of genetic capacities that may prove critical for tolerating rising temperatures and changing oxygen concentrations. The power of the comparative, mechanistic approach needs to be more fully appreciated and exploited by scientists developing models for predicting consequences of global change on the biosphere. NSF IOS-0718734. Somero, G.N. (2010) Physiology of climate change. J Exp Biol 213, 912-920.

2.0: THE ROLE OF PHYSIOLOGY IN ORGANISMAL RESPONSES TO GLOBAL WARMING

2.2

INTEGRATING PHYSIOLOGY AND GENOMICS IN THE STUDY OF GEOGRAPHIC RANGES AND GEOGRAPHIC RESPONSES TO CLIMATE CHANGE

Jessica Hellmann[1], Jason Dzurisin[1], Shannon Pelini[1], Caroline Williams[2], Shawn O'Neil[3], Scott Emrich[3], Brent Sinclair[2]
[1]Biological Sciences, Univ. of Notre Dame, 100 Galvin Life Science Ctr., Notre Dame, IN, 46556, [2]Biology, Univ. of Western Ontario, Biological and Geological Sciences Bldg., London, ON, N6A 5B7, Canada, [3]Computer Science and Engineering, Univ. of Notre Dame, 384 Fitzpatrick Hall, Notre Dame, IN, 46556.

Integrative biology offers an opportunity to reveal the future dynamics of species under climate change. We demonstrate this by combining field and laboratory experiments, ecological and physiological fitness measures, and genomic biology to reveal factors that affect organismal responses to climate change. In particular, we demonstrate local adaptation to climate and predict factors that limit geographic range expansion under climate change in two butterfly species. These species were selected because they share a northern range boundary, occupy the same habitat across a large portion of their ranges, differ in host plant specialization, and differ in extent of population differentiation. Experiments show local adaptation during the winter months in the more specialized species that also has higher population differentiation. This local adaptation undermined fitness gains due to warming in the growing season. In the more generalized species, neither central nor peripheral populations benefited from warming, and no local adaptation was detected. Microarray experiments, however, reveal local adaptation in both species and identify genes that confer differential tolerance to climatic conditions in different locations within a species' range. These genes include metabolic and stress genes that are differentially regulated by different source populations under different climatic scenarios. A comparison of gene regulation in the two species also will be presented.

2.3

MODELS OF CLIMATE CHANGE: WHAT ASPECTS WILL CHANGE AND HOW FAST?

Curtis Deutsch[1]
[1]Atmospheric and Oceanic Sciences, UCLA, 405 Hilgard Ave, 7125 Math Science Bldg., Los Angeles, CA, 90095-1565.

Models of Earth's climate system are crucial tools for understanding the complex phenomena that arise from the interaction of the atmosphere, oceans, and land surface on time scales from days to millennia. The scientific scope of climate models is rapidly expanding, and now extends well beyond solving the physical equations for energy, mass and momentum conservation, to include ever more processes and to resolve ever-smaller scales of environmental variation. In their evolution toward greater complexity, some climate model predictions have been remarkably robust, while others remain highly dependent on the assumptions that are made and the processes that are represented. In this talk, I will briefly describe the basic construction of climate models and how they are evolving toward more comprehensive models of the earth system, highlighting the range of uncertainty associated with different types of simulated climate patterns of particular relevance to ecosystem change. I will discuss applications and limitations of climate predictions for understanding physiological responses of organisms to anthropogenic climate warming.

2.4

EFFECTS OF CLIMATE CHANGE ON BIOENERGETIC COSTS OF MIGRATION IN CHINOOK SALMON, AND CONSEQUENCES FOR LIFE-HISTORY DIVERSITY IN THE COLUMBIA RIVER BASIN

Lisa Crozier[1], Brian Burke[1], Matt Keefer[2], Chris Caudill[2]
[1]Northwest Fisheries Science Ctr., NOAA-Fisheries, 2725 Montlake Blvd E., Seattle, WA, 98112, [2]Idaho Cooperative Fish and Wildlife Res. Unit, Univ. of Idaho, Univ. of Idaho, Moscow, Idaho, 83844.

Life-history diversity contributes substantially to resilience in the face of environmental variability. In salmon, variation in migration and spawn timing play a crucial role in regional biocomplexity. Salmon migrate up the Columbia River to spawning grounds every month of the year. Individual populations, however, have very restricted migration timing, reflecting local adaptation to diverse constraints associated with specific spawning grounds. I here explore how the bioenergetic cost of migration plus holding near the spawning grounds varies with migration date for particular populations. Detailed records of individual migration times and energy usage through dams and reservoirs provide a very rich picture of these costs. I test the hypothesis that current migration timing reflects the optimal timing predicted by bioenergetic constraints and thermal tolerances. I assess how changing hydrological conditions with global warming will shift the optimal phenology, based on a hybrid-delta downscaling method and the VIC hydrology model. By incorporating potential evolutionary and plastic responses to this shift in optimal phenology into population-specific life cycle models, I assess the impact of climate change on the diversity of life histories currently exhibited in the Columbia River Basin.

2.5

ANIMALS ARE NOT GLACIERS: TARGETS OF SELECTION DURING RAPID CLIMATE CHANGE

William Bradshaw[1], Christina M. Holzapfel[1]
[1]Biology, Univ. of Oregon, CEEB, 5289 Univ. of Oregon, Eugene, OR, 97403-5289.

Examination of temperate and polar regions of Earth shows that the nonbiological world is exquisitely sensitive to the direct effects of temperature, whereas the biological world is largely organized by light. Herein, we discuss the use of day length by animals at physiological and genetic levels, beginning with a comparative experimental study that shows the preeminent role of light in determining fitness in seasonal environments. Typically, at seasonally appropriate times, light initiates a cascade of physiological events mediating the input and interpretation of day length to the output of specific hormones that ultimately determine whether animals prepare to develop, reproduce, hibernate, enter dormancy, or migrate. The mechanisms that form the basis of seasonal time keeping and their adjustment during climate change are reviewed at the physiological and genetic levels. Future avenues for research are proposed that span basic questions from how animals transition from dependency on tropical cues to temperate cues during range expansions, to more applied questions of species survival and conservation biology during periods of climatic stress. (NSF IOS-0839998 & NSF DEB-0917827). Bradshaw, W.E. & Holzapfel C.M. (2010). Light, time and the physiology of biotic response to rapid climate change. Annu. Rev. Physiol. 72:147-166.

2.6

USING BIOGEOGRAPHIC DISTRIBUTIONS AND NATURAL HISTORY TO PREDICT MARINE/ESTUARINE SPECIES AT RISK TO CLIMATE CHANGE

Henry Lee II[1], Deborah A. Reusser[2]
[1]U.S. EPA, Western Ecology Div., Pacific Coastal Ecology Branch, Newport, OR 97365.[2]US Geological Survey, Western Fisheries Res. Ctr., Newport, OR 97365.

Effects of climate change on marine and estuarine species will vary with attributes of the species and the spatial patterns of environmental change at the habitat and global scales. To better predict which species are at greatest risk, we are developing a knowledge base of species' biogeographic distributions, habitat requirements, natural history, and physiological tolerances. Presently, the database contains information on >17,000 marine/estuarine invertebrates, fishes, and macroalgae. This biotic information will be coupled with predictions of environmental changes and rule sets to predict effects on species, with different rule sets for specific types of biotic attributes and stressors. A biogeographic-based rule is that species endemic to a single biogeographic ecoregion are at higher risk than wide-spread species. A habitat-based rule is that intertidal species are more vulnerable to changes in air and ocean temperatures than deep species, while another is that estuarine species are more vulnerable to sea level rise due to habitat alterations and changes in salinity regimes. Multiple types of rules will be needed to predict the effects of the "other CO_2 problem", ocean acidification, including those based on the species' depth range and whether it has a calcium carbonate shell, in particular an aragonite shell. An example of the approach to predicting vulnerability to multiple climatic alterations is given for chitons in the Northeast Pacific and Arctic. Invited Presentation at: 2010 American Physiological Society Intersociety Meeting: Global Change and Global Science: Comparative Physiology in a Changing World; Aug. 4-7, 2010; Westminister, Colorado, USA.

2.7

INTEGRATING MOMENTUM, HEAT AND MASS BALANCES IN LEATHERBACK SEA TURTLES: BODY SIZE AND OCEANIC DISTRIBUTION CONSEQUENCES UNDER CLIMATE CHANGE.

Warren Porter[1], Riccardo Bonazza[2], David Michaels[3], T. Todd Jones[4]
[1]Zoology, U. of Wisconsin, 250 N. Mills St., Madison, WI, 53706, [2]Engineering Physics, U. of Wisconsin, 1500 Engineering Dr., Madison, WI, 53706, [3]Zoology, U. of Wisconsin, 250 N. Mills St., Madison, WI, 53706, [4]Joint Inst. for Marine and Atmospheric Res., Univ. of Hawaii at Manoa, 1125B Ala Moana Blvd, Honolulu, HI, 98614.

Landscape scale energetics and behavior associated with fundamental niches has traditionally been largely confined to terrestrial or near shore environments where heat and mass balances are the primary conceptual paradigms. Static models of animals are also typical. This paper addresses momentum, heat and mass balances of leatherback sea turtles ranging in size from hatchlings to full size adults. We present the first phase in the development of connections between momentum balances (drag and thrust), heat balances (distributed heat generation in ellipsoid bodies, body temperature gradients, water temperature requirements) and mass balances (jellyfish consumption requirements) and oceanic distribution requirements for the full range of leatherback sea turtle body sizes for present and future oceanic climates.

2.8

INTEGRATING BIOPHYSICAL, POPULATION DYNAMIC AND EVOLUTIONARY MODELS IN THE CONTEXT OF CLIMATE CHANGE

Michael Kearney[1], Brendan Wintle[2], Tracey Regan[3], Ary Hoffmann[4]
[1]Dept. of Zoology, The Univ. of Melbourne, Melbourne, 3010, Australia, [2]School of Botany/Applied Environmental Decision Analysis, The Univ. of Melbourne, Melbourne, 3010, Australia, [3]School of Botany, The Univ. of Melbourne, Melbourne, 3010, Australia, [4]Dept. of Zoology/Genetics, The Univ. of Melbourne, Melbourne, 3010, Australia.

Evolutionary responses have rarely been considered in the context of species distribution modelling, yet they may be critical in the context of environmental change. The emergence of mechanistic models of species' distributions provides the opportunity to include evolution explicitly by allowing the underlying functional traits to be mutable. We present a general model of the evolutionary response to selection in a quantitative trait. This model is driven by fitness estimates from a mechanistic species distribution model coupled to a population dynamics model. We use the approach to explore climatically imposed selection on body size in folivorous possums across the Australian landscape under different rates of climate change. The approach is general and can be applied to consider evolutionary shifts in the functional traits of endotherms and ectotherms along environmental gradients. Such analyses provide insight into geographic range limits and the potential role of evolutionary responses in buffering range shifts under climate change.

2.9

PHYSIOLOGY AND GENOMICS OF ANIMAL DISPERSAL IN A METAPOPULATION; PRESENT FUNCTION AND RELEVANCE FOR FUTURE RANGE SHIFTS

Jim Marden[1]
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Species living in fragmented landscapes need to disperse to new habitat in order to offset stochastic local extinctions. Using the well-characterized metapopulation of the Glanville fritillary butterfly in the Aland Islands of Finland, we tested the hypothesis that dispersal and establishment of new populations systematically selects for particular physiological, life history, and gene expression traits. We compared common-garden reared virgin females from geographically interspersed newly-established versus old local populations. New-population

females had higher expression of genes involved in egg provisioning and maintenance of flight muscle proteins. Physiological studies confirmed that new-population butterflies had accelerated egg maturation and enhanced flight metabolism. Expression of genes involved in oogenesis and central metabolism varied between allelic forms of two metabolic genes (Pgi and Sdhd), which themselves were associated with differences in flight metabolic rate, population age and population growth rate. These results suggest that extinction-colonization dynamics select for particular alleles, gene expression phenotypes and life history traits, and hence the metapopulation processes appear to maintain genetic variation with large fitness effects. Research integrating across genomics, physiology, and ecology can provide insight regarding mechanisms underlying processes that will be important for species following geographic shifts in the location of suitable habitat. (NSF EF-0412651, NSF IOS 0950416).

3.0: OFF THE BEATEN PATH: INTEGRATIVE ASPECTS OF MUSCLE FUNCTION DURING DIVERSE LOCOMOTOR BEHAVIORS

3.2

MUSCLES, MORPHOLOGY AND MOVEMENT: DYNAMICS OF RUNNING IN VERTEBRATES

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Running fast is vital for many animals that need to capture mobile prey, escape predators, and perform many other behaviors. The fiber composition and architecture of muscles likely imparts, to a large extent, limits on how fast an animal can run. However, the *in vivo* patterns of muscle recruitment and strain can vary through time or can depend on the demands placed on the locomotor system, resulting in variable force, work and power output. With everything else held constant, which attributes of muscles will facilitate maximum running velocity and/or acceleration? Can relatively small variations in dynamic *in vivo* muscle activation patterns explain intra-individual differences in kinematics, strain, and/or force generation? The objectives of this study are to address these two questions and discuss possible avenues for future research. Experimental data were obtained from the helmeted guinea fowl (*Numida meleagris*) and the Florida scrub lizard (*Sceloporus woodi*), both of which have been the focus of several studies related to locomotion. Techniques to measure *in vivo* muscle function included sonomicrometry, electromyography, and tendon force buckles. To assess architecture and fiber composition within a muscle, standard histochemistry was employed. All procedures were approved by IACUC. Funding was provided by NIH and start-up funds from Clemson University.

3.3

MUSCULAR CONTROL OF AVIAN FLIGHT

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Avian flight is powered by two primary muscles: the pectoralis (~8-11% body mass) powers downstroke and wing pronation, and the supracoracoideus, SC (~2% body mass) powers upstroke and wing supination. Smaller intrinsic wing muscles control wing shape. Direct in vivo measurements of muscle force, strain and activation using strain gauges, sonomicrometry and electromyography reveal the the pectoralis develops force rapidly late in upstroke enabling it to shorten over 35% strain of its fascicles during downstroke, producing the majority of mechanical power required for flight. The supracoracoideus shortens with similarly large fascicle strains, producing force at the end of downstroke when the wing is rapidly supinated and accelerated into upstroke. The activation timing of these muscles reduces their antagonistic work, allowing them to produce aerodynamic power during downstroke and overcome wing inertia to initiate the rapid reversal of wing motion. Elastic energy stored in the SC tendon helps facilitate this at the start of upstroke. Based on wind-tunnel recordings, the mechanical power produced by the pectoralis yields a U-shaped power curve over a range of flight speeds in cockatiels and doves, but a flatter curve in magpies at faster speeds, consistent with aerodynamic power requirements and differences in wing shape and flight style. Recent experiments show that remarkably similar patterns of activation and contraction of intrinsic wing and power muscles control take-off and landing flight in pigeons. (NSF IBN 0090265 & IOS-0744056) Toblaske, BW, TL Hedrick, KP Dial & AA Biewener. 2003. Comparative power curves in bird flight. Nature 421, 363-366. Tobalske, BW & AA Biewener. 2008. Contractile properties of the pigeon supracoracoideus during different modes of flight. J. Exp. Biol. 211, 170-179.

3.4

NEUROMUSCULAR STRATEGIES FOR THE TRANSITIONS BETWEEN LEVEL AND HILL SURFACES

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Walking humans continually modulate their muscle activity patterns in order to seamlessly transition between level and hill surfaces in the natural environment. Because it is mandatory to continually adjust kinematic, kinetic, and electromyography patterns between the initial and final surfaces, transitions have the potential to decrease both anterior-posterior and medial-lateral stability. Thus, our objective was to evaluate the neuromuscular strategies that humans employ during the transitions between level and hill surfaces. We hypothesized that lower limb muscle activity during both the strides preceding a surface transition (anticipation) and the strides subsequent to a transition (aftereffect) would be significantly different than either level walking or hill walking independently. Healthy, young adults completed a series of randomly assigned level and hill trials on an over-ground walkway while we recorded kinematic, kinetic, and electromyography data. Our results demonstrated that in anticipation of a transition to a hill, the participants modified their head position 4-6 steps prior to the change in surface angle, which was followed by alterations in muscle activity patterns that resembled the approaching hill. We also evaluated the aftereffect of a transition from a hill and the muscle activity of our participants did not mimic level walking until 2-4 steps after the return to the level surface. In summary, we accept our hypothesis that the neuromuscular strategies during the anticipation and aftereffect transition strides are functionally distinct from either level or hill walking independently.

3.5

RED MUSCLE FUNCTION IN STIFF-BODIED SWIMMERS: THERE AND ALMOST BACK AGAIN.

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The apparent convergence of body form, thunniform swimming, and red muscle thermal physiology in the lamind sharks and tunas is perhaps reflective of fishes with internalized red muscle. In both groups, anteriorly and medially placed red muscle is linked via discreet tendons to the caudal region, focusing lateral body motion to the tail. Unlike other fish with superficial red muscle, in tunas and lamnids the shortening of internal red muscle substantially lags behind changes in local midline curvature, being in phase with midline curvature at locations up to 20% of body length more posterior. Relatively loose connections between these red muscles and adjacent white fibres allow shearing so red and white muscles shorten out of phase with each other. The recent discovery of internalized and regionally endothermic red muscle in members of the thresher sharks (Alopiidae) provides a strategic opportunity to test the universality of this apparent convergence, particularly as common thresher sharks appear to swim with much larger lateral undulations than the tunas and lamnids. New data suggest strain patterns in red and white muscles of common threshers are also out of phase, but novel to the threshers is that the polarity of the phase shift can vary between fish, possibly related to their undulatory swimming. Thermal sensitivity of red muscle appears influenced by the degree of endothermy and fish size. Perhaps internalized red muscle does not dictate a tuna/lamnid-like phenotype.

3.6

NEUROMECHANICAL DETERINATES OF RECRUITMENT WITHIN AND BETWEEN MUSCLES DURING CYCLIC LOCOMOTION

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Efficient and powerful muscle contractions require an appropriate gearing of the muscle fibres relative to the limb motion, and activation patterns that coordinate the optimal muscles and motor units for each mechanical task. Both these phenomena can be tested on a cycle ergometer where the mechanical demands of the motion (force, velocity and power output) can be experimentally manipulated. Considerable gearing of the muscle fibres occurs within the muscle belly, and is only possible due to fibre rotations and bulging of the muscle. Variable gearing occurs between the muscle belly and tendon and may be due to altered activation patterns of the muscle. Velocity-dependent recruitment between the different types of motor units matches their contractile properties to the task dynamics within the muscles of the gastrocnemii. Task-specific coordination matches the balance of muscle activities to the mechanical demands of the motion. The muscle coordination patterns are a major determinate of the whole limb power and efficiency. Muscle structure and neural activation thus both interact to generate smooth and effective motion of the body. REFERENCES: Wakeling JM, Uehli K & Rozitis AI (2006). Muscle fibre recruitment can respond to the mechanics of the muscle contraction. J Roy Soc Interface 3, 533-544. Wakeling JM & Horn T (2009). Neuromechanics of muscle synergies during cycling. J Neurophysiol 101, 843-854. Wakeling JM, Blake OM & Chan HK (2010). Muscle coordination is key to the power output and mechanical efficiency of limb movements. J Exp Biol 213, 487-492.

3.7

CONTROL OF STABLE BIPEDAL RUNNING: INTEGRATION OF EXPERIMENTAL AND COMPUTATIONAL APPROACHES FOR NEW INSIGHTS

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Simple models are remarkably good at predicting whole-body dynamics of steady legged locomotion. Nonetheless, even simple whole-body dynamics involve a complex interplay of neuromuscular control, leg mechanics and terrain interaction. Here, I explore these interactions to understand how animals maintain stability and avoid injury in varied terrain. Leg dynamics and in vivo muscle function are compared during running over level ground, steady inclines and uneven terrain. I test the ability of a simple neural model to predict variation muscle activity (EMG) and cycle timing. The neural model consists of a single oscillator coupled to linear feedback from two distal muscles. Similar control mechanisms are used in both level and uneven terrain. The neural model predicts 78% of the variation in stance timing; but is a poorer predictor of swing timing and EMG amplitude. Swing timing depends on ground interaction: obstacles result in earlier ground contact and, likewise, terrain drops in later contact. There is little evidence for feed-forward adjustment of swing dynamics in varied terrain. Prediction of EMG amplitude may require a more complex model with proximal muscle feedback. Nonetheless, fascicle length and velocity effects explain 70-90% of variation in distal muscle work; which correlates strongly with leg work. The results suggest a simple model for stable running in varied terrain, consisting of feed-forward leg recirculation and load-dependent distal muscle actuation.

3.8

THE WEAK LINK: DO MUSCLE PROPERTIES DETERMINE LOCOMOTOR PERFORMANCE IN FROGS?

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Muscles power movement, yet the conceptual link between muscle performance and locomotor performance is poorly developed. Frog jumping provides an ideal system to probe the relationship between muscle capcity and locomotor performance, because a jump is a single discreet event and jump distance is directly proportional to mechanical power developed. We tested the hypothesis that interspecific variation in jump performance could be explained by variability in available muscle power. We used force plate ergometry to measure power produced during jumping in Cuban tree frogs (Osteopilus septentrionalis), leopard frogs (Rana pipiens), and cane toads (Bufo marinus). We also measured peak isotonic power output in isolated plantaris muscles for each species. As expected, jump performance varied widely. Tree frogs developed peak power outputs of 198.8 ± 20.1 W/kg body mass, about 6 times that of cane toads (36.0±19.7 W/kg). Values for leopard frogs were intermediate (134.3±18.1 W/kg). These differences in jump power were not matched by differences in available muscle power, which were 47.7 W/kg, 78.7 W/kg and 34.7 W/kg body mass for tree frogs, leopard frogs and cane toads respectively. The lack of correlation between available muscle power and jump power suggests that non-muscular mechanisms (e.g., elastic energy storage) can obscure the link between muscle mechanical performance and locomotor performance. Supported by NSF grant IOS064248 to TJR and NIH grant F32AR054246 to EA.

3.9

ACHIEVING CONTROL: INTERPRETING NEURAL FEEDBACK THROUGH CHANGES IN MUSCLE FUNCTION FOR STABILITY AND MANEUVERING IN LOCOMOTING INSECTS

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Muscles integrate neural and mechanical factors to generate controlled locomotion. To test mechanical conditioning of motor control, I spliced motor activation patterns into a control muscle of a locomoting cockroach (*Blaberus discoidalis*). Inserting the same known pattern of neural feedback* under different mechanical contexts revealed three distinct control potentials: graded actuation, a non-linearly recruited vertical force, or a turning response. To explore the mechanisms behind this diversity of control, I developed an intact-joint workloop preparation

that preserves natural oxygen delivery, maintains passive joint mechanics, and enables proper muscle actuation. Activation and strain parameters for the workloops were set to sequentially capture the strides encompassing modified motor activation. A positive mechanical feedback loop between stress and strain coupled with graded neural feedback transformed the muscle from an exclusive brake into a non-linearly recruitable motor. Modulation of activation phase shifted positive work within the locomotor cycle leading to the turning response. Changes in motor activation had functional consequences over multiple strides and a significant portion of positive work arose from muscle prestressing. Neuromechanical coupling within this muscle generated control effects not predicted by biomechanics or neural feedback alone.*Reference: Sponberg, S., Full, R.J. (2008) "Neuromechanical response of musculo-skeletal structures in cockroaches during rapid running on rough terrain." *J. Exp. Bio.*, 211: 433-446.

4.0: HYDRATION, DESICCATION, REGULATION: THE COMPARATIVE PHYSIOLOGY OF WATER BALANCE

4.2

ENERGY AND WATER REGULATION IN AVIAN NECTARIVORES: INSIGHTS FROM THE GEOMETRIC FRAMEWORK

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The geometric framework investigates how animals balance nutrient needs in a multidimensional and dynamic nutritional environment. As an animal ingests food, it moves along multiple axes each representing a nutrient, aiming to ingest the optimal amount of all nutrients, called the intake target. We apply this conceptual framework to whitebellied sunbirds Cinnyris talatala, focusing on carbohydrate and water intake. When given a choice between two dilute or two concentrated sucrose diets, sunbirds converge on the same sugar intake. They maintain this sugar intake over a wide range of concentrations, thus defending a sugar intake target at the expense of processing variable water loads. On very dilute diets, when birds lose body mass, the addition of salts to the diet helps to achieve the carbohydrate intake target. However, sunbirds not only encounter variations in nectar quality, but also in metabolic needs. When exposed to decreases in ambient temperature, they increase their sugar intake on dilute and concentrated diets. When simultaneously experiencing low temperature and dilute nectar, birds do not meet their sugar intake target, suggesting digestive and osmoregulatory constraints. The geometric framework approach, derived initially from work on insects, provides a powerful tool for integrating data on energy and water balance in avian nectarivores. NRF 44207. Raubenheimer D & Simpson SJ. 1997. Integrative models of nutrient balancing: application from insects to vertebrates. Nutr Res Rev 10:151–179.

4.3

RENAL ADAPTATIONS TO DEHYDRATION IN DESERT MAMMALS

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Desert rodents conserve water, in part, by excreting highly concentrated urine. Structure-function features that may relate to high urine osmolalities include papilla length, relative medullary thickness, relative abundance of short and long loops of Henle, vascular organization, and variation in loop of Henle fluid and solute permeabilities. Although a correlation exists between some of these features and the ability of different species to form a high urine concentration, the relationships are often unpredictable and mechanisms are poorly understood. In *Dipodomys merriami*, a desert rodent that concentrates its urine to ~6000 mOsm/Kg water, collecting duct (CD) clusters form the central organizing motif in the renal inner medulla. CDs, ascending thin limbs (ATLs), and ascending vasa recta (AVR) form transverse arrays of discrete interstitial compartments along the inner medullary axis. Fluid and solute reabsorbed from CDs and ATLs likely diffuse preferentially from these compartments into AVR. Moderately water-permeable descending thin limbs (DTLs) and descending vasa recta (DVR) associate with highly water-permeable AVR in a second interstitial compartment lying apart from CDs. The interstitial, tubular, and vascular compartments define multiple countercurrent systems that may underlie renal adaptation to water conservation in desert mammals. (Support: NSF IOS-0952885) REFERENCE: Bankir L and De Rouffignac C. Urinary concentrating ability: insights from comparative anatomy. Am J Physiol 249: R643-R666, 1985. A review of medullary architecture and physiology associated with species variation in urine concentration.

4.4

EVOLUTION OF ION-MOTIVE ATPASE (V-TYPE H+ ATPASE, NA+,K+-ATPASE) ACTIVITY AND EXPRESSION DURING INDEPENDENT INVASIONS INTO FRESHWATER HABITATS

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Marine to freshwater transitions constitute formidable physiological barriers for most animal taxa. However, the estuarine copepod Eurytemora affinis has invaded freshwater habitats multiple times independently within the past century. We examined activity and gene expression of ion transport enzymes V-type H+ ATPase and Na+,K+ ATPase for saline and fresh population pairs under common-garden salinities (0, 5, 15 PSU). We found parallel evolutionary shifts across multiple independent invasions. Relative to saline populations, freshwater populations showed an evolutionary increase in V-ATPase activity in fresh water (0 PSU) and a decline at higher salinity (15 PSU). In contrast, freshwater populations displayed a decline in Na,K-ATPase activity across salinities, relative to saline populations. Results were consistent with expression differences observed in underlying genes. Most notably, ancestral saline populations selected for freshwater tolerance in the lab (12 generations) exhibited adaptive shifts in V-ATPase activity observed in the natural populations. Thus, modifying a single variable in the lab (salinity) recapitulated the evolutionary shifts observed in the wild. Developmental acclimation could not reproduce the patterns of enzyme activity and expression found in freshwater populations or freshwater-selected lines. V-ATPase localization and activity have been hypothesized to be critical for freshwater adaptation and the invasion of land, but evolution of this enzyme function during a habitat transition had not been demonstrated previously.

4.5

ADJUSTMENT OF CUTANEOUS WATER LOSS TO CHANGING ENVIRONMENTS

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For terrestrial organisms, prevention of excessive water loss through their integument is essential to maintain normal cellular hydration. As climates change, we need to understand the ability, or lack thereof of, of animals to adjust their cutaneous water loss (CWL). Here I review what we know about adjustment of CWL to different environmental circumstances. I show that adult birds reduce their CWL when exposed to low relative humidity and higher air temperatures, and that lower CWL is associated with major changes in the intercellular lipids of the stratum corneum. In adult birds, lipids covalently bound to the protein envelope of corneocytes of the stratum corneum remain relatively constant regardless of environment. CWL of nestling birds and lipids of their skin are relatively unstudied. Nestlings from Saudi Arabia and from Ohio were raised in a humid environment and dry environment. Nestlings from Saudi Arabia had higher CWL than did those from Ohio. In both habitats, CWL of nestlings was always higher than that of adults. Nestlings from deserts rely more on CWL for thermoregulation than do nestlings from temperate environments. Intercellular and covalently bound lipids differed in nestlings from Saudi Arabia and Ohio. However, when acclimated to a moist or dry environment, both groups of nestlings changed the intercellular lipids of their stratum corneum, but covalently bound lipids remain unchanged.

4.6

VERTEBRATE FREEZE-TOLORANCE: AN OSMOREGULATORY PERSPECTIVE

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In freeze-tolerant vertebrates, a substantial component of meeting the challenges from freezing entails management of body water and solutes. In gray treefrogs, Hyla chrysoscelis, that is accomplished in part by accumulation of cryoprotective glycerol during cold acclimation. Thus, during cold, freezing, and thawing, water and glycerol must cross cell membranes as the solute is distributed and conserved, and water shifts between fluid compartments. In H. chrysoscelis, expression of aquaporins (proteins that facilitate transport of water and glycerol) changes during cold acclimation, and we hypothesize that aquaporin function is critical to freeze tolerance. We now address two aspects of these changes in expression. First, what regulatory mechanisms are involved? We explore both stimulatory factors (e.g., expression of the glyceroporin HC-3 is enhanced in erythrocytes and hepatocytes cultured in glycerol-containing media) and cellular mechanisms (e.g., post-translational glycosylation is altered in association with varying aquaporin expression in the plasma membrane). Second, what are the functional consequences of changing aquaporin expression? Permeabilities to water and glycerol differ in cells from warm- and cold-acclimated animals. How do these relate to the ability to survive freezing and thawing? (NSF IOB-0517301 to DLG and CMK). Reference: Zimmerman SL, et al. 2007. Excretion and conservation of glycerol, and expression of aquaporins and glyceroporins, during cold acclimation in Cope's gray tree frog. Am J Phys 292: R544-555.

4.7

THE ROLE OF DEHYDRATION IN INSECT SUBZERO TEMPERATURE TOLERANCE

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Insects survive subzero temperatures by becoming either freeze tolerant (able to survive freezing of extracellular body fluids) or freeze avoiding. There are multiple adaptations associated with each, but dehydration can be important in both. In freeze tolerance, extracellular ice induces osmotic out-flux of water from cells that decreases the cytoplasm freezing point, inhibiting lethal intracellular ice formation. However, as temperature decreases and more ice forms, cell dehydration may become lethal. Multiple freeze/thaw cycles associated with climate change can be lethal. In freeze avoiding species dehydration can also be positive or negative. Environmental ice lowers vapor pressure relative to the unfrozen insect, and can lead to deleterious desiccation. However, dehydration can be an adaptation that results in concentration of antifreezes and less freezable water. The beetle Cucujus clavipes in Alaska often undergoes cryoprotective dehydration to less than 30% body water during especially cold periods. During this time they cannot be frozen, even at -150C, but body water vitrifies at ~-65C. Since this critical dehydration requires days, more frequent freeze/thaw cycles resulting from climate change may be troublesome. Also, climate change can disrupt the critical timing of the gain and loss of various seasonal changes required for both freeze tolerance and avoidance. C. clavipes in Alaska produce antifreeze proteins much earlier than is now necessary, while short photoperiods alone no longer cue antifreeze protein production in Dendroides canadensis beetles in Indiana. (NSF IOB-18342).

4.8

WATER HOMEOSTASIS IN NECTAR-FEEDING VERTEBRATES: OUT OF THE LAKE AND INTO THE OCEAN

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To maintain water balance, nectar-feeding vertebrates oscillate between meeting two contrasting extremes: avoiding overhydration when feeding and preventing dehydration during fasts. To understand how they meet these challenges, I examined the influence that water intake rates and time of day had on water-handling processes in two groups of nectarivores: hummingbirds (Trochilidae) and leaf-nosed bats (Phyllostomidae). The technique I devised allowed me to construct complete water budgets for freely-feeding animals under laboratory conditions. To avoid overhydration, both bats and hummingbirds increased rates of glomerular filtration (GFR) and decreased renal water reabsorption. Although nectarivores could also avoid overhydration by having an unusually fast GFR and/or by modulating water absorption in the gut, my findings indicate that neither group does. To alleviate dehydration, both hummingbirds and bats limited water losses by reducing GFR. In each group, these GFR reductions were dramatic: hummingbirds ceased GFR during the night and halved it during a short, midday fast; bats reduced GFR by ~90% during their daytime fast. Interestingly, in spite of having markedly different osmoregulatory systems, these distant relatives converged evolutionarily on the traits they use to maintain water balance. This work was funded by the American Ornithologists' Union, the Company of Biologists, the National Institutes of Health, the Society for Integrative and Comparative Biology, and the University of Wyoming.

4.9

HOT AND DRY: MOLECULAR REGULATION OF ESTIVATION

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When challenged with drought conditions, a survival option for many species is Physiological Aspects (Navas CA, Carvalho JE, eds), Springer, Heidelberg, 49:25-45. estivation; animals orchestrate a coordinated suppression of cell functions and enter a dormant state with metabolic rate typically reduced by at least 70% compared with normal resting rates. The pulmonate land snail, Otala lactea, is a well-studied model estivator and recent research in my lab has focused on key issues of biochemical adaptation: differential control of signal transduction cascades, targeted suppression of cell functions by reversible protein phosphorylation, global suppression of transcription and translation, selective gene upregulation. Selected topics for discussion will include roles of AMPK and Akt (protein kinases mediating low energy vs anabolic signals), respectively, FOXO transcription factors that mediate cytoprotective actions (e.g. cell cycle arrest, antioxidant defense), translational suppression by control of ribosomal initiation and

elongation factors, and inhibition of pathways (e.g. pentose phosphate cycle, glycolysis) and ion motive ATPases (NaK-ATPase, Ca-ATPase) by covalent modification of key enzymes. Such mechanisms are proving to be widely expressed in phenomena including diapause, hibernation, and anaerobiosis and illustrate conserved principles of hypometabolism and life extension across the animal kingdom. For more information visit: www.carleton.ca/~kbstorey. Funded by NSERC Canada. REFERENCE: Storey KB, Storey JM. 2010. Metabolic regulation and gene expression during aestivation. in: Aestivation: Molecular and Physiological Aspects (Navas CA, Carvalho JE, eds), Springer, Heidelberg, 49:25-45.

5.0: SELECTED ORAL PRESENTATIONS I

5.1

CARDIAC FUNCTION IN TUNAS: THE ROLE OF EXCITATION CONTRACTION COUPLING IN BUILDING A THERMAL TOLERANT FISH HEART

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Bluefin tunas have unique attributes that allow occupation of a wide thermal niche including endothermy and a robust cardiac physiology that is reliant on excitation-contraction coupling. In bluefin tunas, elevated metabolic rates are coupled with heat exchangers that enable heat conservation in muscle, viscera, eye and brain tissues. However, the hearts of all tunas operate at ambient temperatures. This unusual physiological arrangement is unique amongst vertebrates and can result in a cold heart supplying the metabolic demands of warm tissues. To better understand tuna physiology we examined the thermal niche of two closely related species, the bluefin and yellowfin tunas and the cardiac physiology of both species in the lab. Mean daily surface temperatures from 377 individuals revealed a thermal preference for Pacific bluefin of 17.36°C ± 0.05 s.e. and for yellowfin of 21.55°C ± 0.16. Results from molecular, structural and physiological data indicate a significant role of Ca^{2+} induced- Ca^{2+} release in the cardiac myocytes of tunas in a similar form to mammals. We maintain that increasing reliance on SR function is crucial to the performance of the cold tolerant bluefin heart and that the SR Ca^{2+} ATPase is the malleable unit of cellular Ca^{2+} flux, vital for increasing the capacity to operate at both low and high temperatures. These findings have implications beyond endothermic fish and may help to delineate the key steps required to protect vertebrate cardiac function.

5.2

INTERPLAY BETWEEN INTESTINAL PERFORMANCE AND MICROBIOTA COMMUNITY IN AN INTERMITTENT FEEDER

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We used the intermittent-feeding Burmese python (*Python molurus*) to investigate the relationships between extreme variation in intestinal nutrient processing and its microbial community (microbiota). We quantified intestinal mass and function for 30-day fasted pythons and at 8 time points postfeeding, and sequenced bacterial 16S rRNA genes to survey gut community ecology. With feeding, the python small intestine doubles in mass and rapidly upregulates hydrolase activity and nutrient uptake, responses that are reversed following cessation of digestion. The large intestine rapidly fills with dietary material, though only modestly changes function. Twelve bacterial phyla were detected in intestinal contents with the majority of sequences identified as Firmicutes or Bacteroidetes. Bacterial diversity of the cecum and large intestine was twice that of the small intestine and increased after feeding, nearly tripling. There was an equal proportion of Firmicutes and Bacteroidetes in the fasted small intestine: this ratio diverged with feeding as Firmicutes increased to 90% of bacterial 16S rRNA sequences. These results establish the python as an attractive model for examining how upregulation of host intestinal functions and alterations in the configuration of the gut microbiota are coordinated to enhance harvest of available nutrients and to adapt to prolonged periods of nutrient deprivation.

5.3

CLIMATE CHANGE INCREASES THE LIKELIHOOD OF CATASTROPHIC AVIAN MORTALITY EVENTS DURING EXTREME HEAT WAVES AND DROUGHTS

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For the avifaunas of hot subtropical deserts, among the most important, but understudied, direct effects of climate change may involve catastrophic mortality events associated with extreme heat waves and droughts. A number of large-scale die-offs during extremely hot weather have been documented in the past, and general circulation models predict increases in the intensity, frequency and duration of heat waves. Here, we identify the physiological mechanisms underlying avian mortality associated with heat stress and the lack of water, and develop a model that predicts rates of evaporative water loss and survival times during very hot weather as functions of body mass and dehydration tolerance. Applying our model to current and projected maximum air temperatures for two localities in hot subtropical deserts, we find that the increase in maximum air temperatures predicted for the 2080s will increase rates of evaporative water loss by more than 50-80% in very small birds, reducing survival times by 30-40%. For birds weighing < 100g (more than 80% of species in most desert bird communities), rates of evaporative water loss will increase by 30-50% and survival times will be reduced by at least 25%. The existing literature suggests that many species will simply be unable to up-regulate EWL to maintain body temperatures below critical lethal limits given the predicted increases in heat stress. Current and historical accounts already document catastrophic mortality caused by hyperthermia or through dehydration. Increasing global temperatures, combined with more intense and frequent heat waves, will result in more frequent die-offs among desert birds, potentially depopulating regional communities.

5.4

LIGHT AT NIGHT AND COMPARATIVE PHYSIOLOGY IN A CHANGING WORLD

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The most dramatic environmental global change that took place in the last hundred years and even more intensively in the last decays is the phenomena of light at night (LAN) or light pollution. Our environment contains temporal variable such as time of activity during the 24h cycles and seasonality. We studied for many years seasonal acclimatization of thermoregulatory mechanisms in relation to photoperiod exposures and showed that acclimation to long days (LD) is a signal for summer acclimatization while continuous exposure to short days (SD) is a signal for winter acclimatization. Therefore, a question to be asked is: Dose LAN affects physiological responses of animals? Results of studies from the recent years show that SD-acclimated voles (Microtus socialis) (8L:16D) exposed to LAN and light interference (LI) affect the physiological systems via changes in two different endocrine systems. On the one hand, decrease in pineal melatonin production and secretion while on the other hand increase in cortisol and adrenaline as a response to stress. Furthermore, results of several studies revealed that the daily rhythm response can be species specific even when comparing between sibling species or species of the same family with a different temporal organization.

5.5

WILL WINTER PROVIDE A LIMIT FOR AN INVASIVE SPECIES? A BOTTOM-UP APPROACH FOR THE EMERALD ASH BORER

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Bioclimatic envelope models use information about the current (or native) range of a species to predict its future distribution under climate change or its potential range as an invasive species. The Emerald Ash Borer is an invasive buprestid beetle moving rapidly from its initial establishment sites in Michigan and Ontario into the USA and Canada. Because its native range in Asia is not well-known, bioclimatic envelope models are difficult to apply. We are using a bottom-up approach based on the hypothesis that low winter temperatures may limit the potential distribution of this species in Canada. To this end, we have determined the cold tolerance strategy, explored seasonal and short-term plasticity in cold tolerance, and the impacts of repeated cold exposures in the overwintering stage (prepupae). We couple this with a large network of microclimate measurements and are now developing a potential distribution model to determine risk for uninvaded sites in Canada.

5.6

FROM GLOBAL TO MICROCLIMATE CHANGES: BIOPHYSICS REVEALS BUFFERING MECHANISMS

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GCM models of climate change improve our understanding of the global mechanisms but very little is known on the effects of global warming on the microclimate of species. Here, we quantify the microclimate change following an increase in temperature during a heat wave event in a leafmining moth. We developed a nested model downscaling framework to spatially and functionally interconnect regional and microhabitat (mine) scales. This mechanistic biophysical approach integrates heat budgets of organisms, physiology of the plant and the insect, behavior (thermoregulation), and physics of radiative transfer within canopy architectures. The model predicts mine and body temperatures for a larva at a given position within a tree canopy from regional climatic variables. The comparison of predicted temperature increase during a moderately warm day and during the heat wave event shows that the amplitude of the mine microclimate warming does not equal that of the global temperature increase. Instead, the biophysical functioning of a mine partially buffers against overheating, which ameliorates survival of the leafminer. By confronting our results to the few other published works on climate change effects on microclimate and body temperature of species, we suggest that the buffering nature of species microclimate might be widespread if not the rule. This study was supported by CNRS and the University François Rabelais (Tours, France).

5.7

METABOLIC MECHANISMS MEDIATE THE MISERABLE MONTHS: SEASONAL DIAPAUSE PROMOTES SPECIATION IN THE APPLE MAGGOT.

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The apple maggot, *Rhagoletis pomonella*, a text-book model for sympatric speciation and adaptive genetic divergence has recently (~200ya) expanded its host range from native hawthorns to domesticated apples. Apples flower and fruit earlier in the summer than hawthorns and to synchronize themselves with the novel plant apple flies undergo a longer, more metabolically demanding diapause. To compensate, we expected apple pupae would: 1) tank up with more fuel reserves (greater lipid storage), or 2) use their fuel reserves more judiciously (decreased metabolic rates).We have shown that field-collected apple race individuals have significantly greater lipid reserves than hawthorn race individuals. But, are apple race individuals more efficient? First, we use microarrays to characterize the tanscriptome of diapausing and non-diapausing individuals, indentifying several candidates for adaptive shifts in diapause in metabolic, endocrine, and stress resistance pathways. Second, we assay variation in diapause metabolic rates in a large group of individuals, clearly identifying classes of individuals with greater or lesser metabolic depression. Climate change will affect the phenology of plants and their insect herbivores, potentially yielding both rapid adaptive change in life history timing and novel interactions; this work bridges the gap between physiological and genetic mechanisms underlying the evolution of diapause timing, host plant shifts, and diversification.

5.8

EFFECTS OF TEMPERATURE ON PERFORMANCE AND REPRODUCTION OF A SIERRA WILLOW BEETLE- IMPLICATIONS FOR POPULATION PERSISTENCE IN FACE OF CLIMATE CHANGE

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The willow beetle Chrysomela aeneicollis lives in the Eastern Sierra Nevada mountains of California. Beetles experience elevated air temperatures during summer, which causes physiological stress that affects fecundity, mating frequency, and larval survival. Prior studies have shown that the glycolytic enzyme locus phosphoglucose isomerase (PGI) is under temperature selection in these populations. The magnitude of PGI allele fluctuation with climate appears to be related to air temperature, as frequency of PGI-4 increases in the hottest part of summer. To identify mechanisms causing shifts in PGI allele frequency, we measured metabolic rate, fecundity, and male mating frequency for beetles in the laboratory and in nature. We found that metabolic rate at high temperature was higher for PGI 4-4 genotypes than for PGI 1-4 or PGI 1-1 individuals. In contrast, laboratory fecundity was greatest for 1-1 and 1-4 females. Hsp70 expression was positively related to fecundity, suggesting minimal reproductive cost of upregulation of Hsps. In nature, fecundity was greatest for PGI genotypes that were most common in each area. For males, running speed after mating was correlated with mating

frequency. Exposure to elevated temperature reduced mating frequency of PGI 1-1 and 1-4 males, but increased that of 4-4 males. These data suggest that hot, dry summers in the Sierras may result in an increase in frequency of the PGI-4 allele in response to climate change.

6.0: OPPORTUNITIES FOR COMPARATIVE PHYSIOLOGISTS: BUILDING LINKS WITH AGENCIES, INSTITUTIONS AND FOUNDATIONS

6.1

OPPORTUNITIES FOR COMPARATIVE PHYSIOLOGISTS: BUILDING LINKS WITH AGENCIES, INSTITUTIONS AND FOUNDATIONS

Jon F. Harrison
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This workshop will address mechanisms to link comparative physiologists with agencies, institutions and foundations interested in conservation and effects of climate change. Oral presentations will be followed by a breakout session in which attendees can meet with the individual speakers to ask questions and discuss.

6.2

BUILDING A DIVERSIFIED RESEARCH PORTFOLIO IN PHYSIOLOGICAL ECOLOGY

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Research in physiological animal ecology can advance fundamental knowledge important to physiologists and ecologists and can also have applications important to society. I describe steps taken to develop a research portfolio that generated about $175,000/year in support over 25 years, of which about a third was oriented to fundamental research supported by the U.S. National Science Foundation and 2/3 oriented to other agencies and foundations that typically have management objectives. An early strategic choice was made among possible ways to apply research, based on both inherent research interests and academic culture. Then good matches were developed in several ways with potential funding agencies (federal and state) and foundations (nongovernmental and industry). Key aspects of these relationships were a willingness to extend oneself scientifically, to create partnerships, and to be a conscientious partner.

6.3

OPPORTUNITIES FOR COMPARATIVE PHYSIOLOGISTS: BUILDING BRIDGES WITH ZOOLOGICAL COMMUNITIES FOR RESEARCH AND SPECIES CONSERVATION

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Large size, slow reproductive rates and comparatively high demands on environmental resources make big animals especially vulnerable to global-climate events. Statistics regarding the status of mammal populations reflect this sensitivity with >25% of large mammals listed as threatened or endangered by the International Union for Conservation of Nature (IUCN). Despite this, fewer scientists are engaging in the study of the most endangered animals, in part due to a lack of opportunity for research involving large organisms. Here we discuss how to solve this problem by developing greater partnerships with the zoological and aquarium industry. An important aspect of such partnerships is recognition of the financial, intellectual and time commitments made by both the researcher and zoo. Broadening funding bases beyond traditional NSF and NIH sources is critical for success. These may include foundations with zoological parks, wildlife groups and individuals as well as in other governmental agencies. Creative use of these resources provides an underutilized tool for conducting the type of science that will enable *prediction* rather than *reaction* to rapid environmental change that is impacting large animals. (NSF OPP 0739163 & ONR N00014-0510808.) REFERENCE: Williams, T.M. and Yeates, L. 2004. The energetics of foraging in large mammals: a comparison of marine and terrestrial predators. Int. Congress Series 1275: 351-358.

6.4

SCIENTIFIC CONTRIBUTION TO ENDANGERED SPECIES MANAGEMENT; DEVILS HOLE, DESERT FISHES AND CLIMATE CHANGE

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The Devils Hole pupfish, *Cyprinodon diabolis*, occurs only in Devils Hole which is administered by the US National Park Service (NPS), within Ash Meadows National Wildlife Refuge. The population fluctuates seasonally. In the winter, sunlight is not available and primary production is limited. In summer, primary production increases albeit high temperatures influence population dynamics. After a landmark Supreme Court decision allowed the water level to recover from agricultural pumping the population size increased to approximately 500 maximum and 200-300 minimum. Beginning in 1996 the population began to decline, reaching a minimum of 38 in spring 2006. At this point supplemental feeding was initiated and the population size began to recover with a minimal count between 120-140 in spring 2010. An analysis of primary productivity in 1999-2001 indicated annual primary production to be about 5000 kJ/yr. We measured oxygen consumption of adult fish and conservatively estimate minimal energy requirement to be 17.5 kJ/yr for adult fish. Assuming a conversion efficiency of 10% a maximum population, pre-feed, can be estimated as 500/17.5 or 28 fish indicating the system is energy limited. Our results have contributed to the management and assessment of the feeding schedule and the recovery of the population.

6.5

CLIMATE CHANGE AND PHYSIOLOGY: NEW EXCITING OPPORTUNITIES FOR SPONSORED RESEARCH AT NSF

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NSF is poised to increase significantly investments in climate change research across a broad range of disciplines and, importantly, in relevant multidisciplinary research. Forecasting climate change effects on biological systems, a long-term goal, requires intimate knowledge of basic physiological principles. NSF/BIO is encouraging submission of proposals that address mechanisms of adaptation and biological feed backs to climate using molecular, cellular, and organismal perspectives and approaches. For example, one new program targets research on ocean acidification and is aimed at support of cross-disciplinary projects that will yield novel insights about climate change and biological systems. The estimated $12 million/year program is anticipated to run for five years, with the first competition in spring, 2010. Proposals relating broadly to neural systems, development, animal behavior, and metabolism, as well as other topics in physiology, would be welcome in this competition. In addition, NSF core programs strongly encourage innovative research on basic principles governing interactions between organisms and their environments. Through causal links that relate environmental stress to biological function, physiologists are challenged to explore creatively major unsolved problems, towards predicting organism performance in a changing world. Through basic sponsored research, NSF continually provides meaningful opportunities for finding seminal answers to critical research questions.

7.0: POSTERS IN RESPIRATORY PHYSIOLOGY, THERMAL PHYSIOLOGICAL ECOLOGY, MUSCLE/LOCOMOTORY PHYSIOLOGY AND EVOLUTIONARY PHYSIOLOGY

7.1

ATMOSPHERIC HYPOXIA INCREASES BONE ROBUSTICITY IN THE AMERICAN ALLIGATOR

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During vertebrate evolution, atmospheric oxygen (O_2) level may have varied from as low as 12% to as high as 30%, but no studies to date have considered the effect of ambient O_2 on skeletal plasticity. We incubated eggs and subsequently grew alligator hatchlings under chronic hypoxia ($12\%O_2$), normoxia ($21\%O_2$) and hyperoxia ($30\%O_2$). Animals received monthly injections of fluochrome dyes to determine mineral apposition rates. After three months, animals were sacrificed and their femora either sectioned at mid-diaphysis, or ashed. We found femora of hypoxic alligators to have significantly greater mass-specific cross-sectional area (+15%), second moment of area (+20%) and polar moment of inertia (+23%) than those of either normoxic or hyperoxic hatchlings. Mineral content was also significantly higher (+6%) in bones of hypoxic animals. This suggests that exposure to chronic hypoxia, but not hyperoxia, resulted in increased resistance to compressive, bending and torsional stresses on the skeleton. Furthermore, the relationship between body mass growth and periosteal deposition rate was different between treatments groups, with hypoxic animals accruing more bone per unit body mass. We suggest that prevalent atmospheric O_2 level need be considered when reconstructing size and growth curves of extinct vertebrates. Funded by the NSF IOB 04445680 and IOS 0922756 to JWH.

7.2

CHRONIC EXERCISE DOES NOT ALTER LIMB BONE MORPHOLOGY OR MICROSTRUCTURE IN THE AMERICAN ALLIGATOR

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In contrast to mammals and birds, effects of exercise on bone microstructure in reptiles have received scant attention. We investigated the effects of long-term exercise on a treadmill or in a flume on limb bones of the American alligator. Juvenile female alligators were run or swum to exhaustion every other day for 17 months. Animals were measured every other week, and received injections of fluorochrome dyes (calcein and alizarin) to determine mineral apposition rates in the skeleton. We found no significant differences in whole bone morphology and cortical bone deposition rates in the alligator humeral midshaft, regardless of exercise regimen. Similarly, we found no effects of either exercise regimen on cancellous bone architecture (bone volume fraction, bone surface density, bone specific surface, as well as trabecular number, thickness and separation) of the distal femur. In addition, bone mineral content was similar across exercise groups. Altogether, this suggests that long-term locomotor exercise has no discernible effect on bone microstructure in alligators. These results stand in contrast to studies on mammals and birds. The disparity could be due to metabolic differences between ectothermic and endothermic vertebrates. Alternatively, alligator limb bones are subjected to insufficient strain levels or load cycles during short exercise bouts, which may account for lack of skeletal plasticity in response to exercise. Funded by the NSF IOB 04445680 to JWH.

7.3

THE METABOLIC CHANGES ASSOCIATED WITH GRAVIDITY IN AN *OVIPAROUS SQUAMATE.*

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The objective of this study is to ascertain whether or not significant metabolic changes accompany gravidity in Lamprophis fuliginosus, an oviparous squamate prevalent throughout sub-Saharan Africa. Here we use flow through respirometry to obtain exhaled gas concentrations in 6 or more gravid adult females, and 6 or more non-gravid adult control females, and then calculate standard metabolic rate throughout gestation for each animal. Measurements continue 2-7 times per week, until the gravid females lays a full clutch of eggs. Our results indicate an increased metabolic rate associated with gravidity in L. fuliginosus. We conclude that gravidity induces physiological changes in the oxygen transport cascade in L. fuliginosus.

7.4

RESPIRATORY VASCULATURE OF THE MUDSKIPPER, *PERIOPHTHALMODON SCHLOSSERI* (GOBIIDAE: OXUDERCINAE)

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Previous evidence illustrates that the gills of the mudskipper, Periophthalmodon schlosseri, are not structurally optimized for gas exchange. We investigated the respiratory vasculatures of the branchial and bucco-opercular cavities of this species using corrosion casts, and quantified the degree of capillarization in the mouth linings by histological analysis. The vascular system of this species retains the typical fish configuration with a serial connection of the gills and the systemic circuits. The gills appear to play a limited role in gas exchange. They bear a relatively small numbers of short filaments, and the microvasculature of the lamellae consists of parallel channels that are larger than red blood cell size. In contrast, the epithelia of the bucco-opercular chamber, branchial arches, and leading edge of the filaments are extensively laden with

capillaries having a short (<10 □m) diffusion distance, thus strongly demonstrating their principal respiratory function. These respiratory capillaries are supplied by efferent blood from the gills and drained by the systemic venous pathway. Quantification of capillary density in the bucco-opercular epithelia of 4 related species of intertidal gobies showed that P. schlosseri has a significantly higher capillary density in the palate, tongue, and operculum than in the non-air-breathing Acanthogobius hasta, facultative air-breathing Odontamblyopus lacepedii and amphibious air-breathing Periophthalmus modestus (except operculum).

7.5

EFFECT OF TEMPERATURE ON THE CARDIO-RESPIRATORY SYSTEM IN GRASS SHRIMP (*PALAEMONETES PUGIO*)

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Oxygen delivery rates must keep pace with the increased metabolic rate observed in poikilothermic animals exposed to increased temperature if aerobic metabolism is to be maintained. Physical failure of the oxygen delivery system to meet this increased demand may be a mechanism of cardiac failure in crustaceans at temperature extremes. Analyses were performed in both normoxic and hyperoxic environments to determine which component of the cardio-respiratory system is most limiting over a temperature range (5° – 40° C) in grass shrimp (Palaemonetes pugio) acclimated to 20° C. Ventilatory parameters (rate and an index of flow) and cardiac parameters (heart rate, stroke volume, cardiac output, ejection fraction) were measured using high-speed video analysis to identify when and how the cardio-respiratory system failed. Finally lactate levels and oxygen consumption were measured. Animals placed in a hyperoxic environment had a higher CT max than animals in a normoxic environment, suggesting a physical limitation in oxygen delivery. Stroke volume appears to be temperature insensitive in normoxic animals, leaving cardiac output dependent on heart rate. As heart rate increases, diastolic filling time decreases to a point where stroke volume can no longer be maintained, leading to a decline in cardiac output and decreased oxygen delivery.

7.6

SEOSONAL VARIATION OF BAROREFLEX ACTIVITY IN THE BLACK AND WHITE TEGU LIZARD

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There is evidence of baroreflex control of blood pressure in some species of reptiles, but no conclusive data exists regarding tegu lizards. These animals present a clear annual cycle of metabolic rate, which includes periods of activity during the rainy and hot seasons (spring and summer) and metabolic reduction during the dry and cold winter. Thus, the present study aimed to verify the presence of a functional baroreflex in the tegu lizard, Tupinambis merianae, during autumn (April-May), winter (June-August), spring (October-December) and summer (January-February). Arterial blood pressure (ABP) and heart rate (HR) were determined after systemic injections of sodium nitroprusside (SNP; 2.5, 5, 10, 25 □g/kg) and phenylephrine (PE; 5, 10, 25, 50 □g/kg). Experiments were conducted at 24-27oC in all seasons to abolish temperature effect on ABP and HR. Basal ABP (mmHg) and HR (bpm) were, respectively: 38.1 ± 4.0 and 14.0 ± 0.8 (autumn); 35.5 ± 1.3 and 10 ± 1.5 (winter); 34.7 ± 1.4 and 13.3 ± 0.7 (spring); 35.0 ± 1.7 and 12.1 ± 0.9 (summer). No seasonal variation of the basal values was observed. All doses of SNP caused significant decreases in ABP with respective increases in HR in all seasons, responses that were less prominent during winter. PE induced a dose dependent increase in ABP during autumn, spring and summer, but not during winter. In this latter, the hypertensive response was less evident than in the other seasons. PE induced a weak baroreflex response of HR regardless season. The results indicate the presence of a hypotensive baroreflex in T. merianae during all seasons. The lack of reflex response of HR to the PE injections remains to be clarified. Supported by FAPESP and CNPq.

7.7

RELATIONSHIP BETWEEN SERUM IRON LEVELS AND MASS AND HB IN VERTEBRATES

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Iron (Fe) is required in vertebrates because of its role in oxygen transport and biochemical oxidative metabolism but free Fe can catalyze free radicals from oxygen which can result in significant negative effects on biological systems. Because of the central role Fe plays in supporting the rate of oxygen consumption it was hypothesized that blood Fe levels might scale with body mass in a manner consistent with that for basal metabolic rate. In addition, it was thought probable that due to the large amount of Fe necessary for erythropoiesis, vertebrates exhibiting adaptively important higher blood hemoglobin concentrations (Hb) may exhibit greater serum iron levels (SI). SI concentrations were extracted from papers published in the primary literature for all major vertebrate lineages. From these publications Hb and hematocrit (Hct) were also recorded if given. SI levels were found for 135 mammals, 19 birds, 6 reptiles, 2 amphibians and 7 fish. Only the data for mammals was deemed of sufficient quantity and quality to justify significant statistical analysis. Regression analysis of SI showed that body mass did not account for a significant amount of the variability observed. The mean SI for mammals was 29 mmol/L, the 95 % confidence interval was 26.6-31.4 mmol/L. Examination of scatter plots indicated that generally avian SI was similar to those of mammals and those for all other groups were somewhat less than those for mammals. Scatter plots and regression analysis of those species for which there were matched SI and Hb or Hct values demonstrated that SI increased with increasing amounts of Hb. This increase was nonlinear.Increased measurement of SI and related Fe parameters is clearly needed to more fully explore the role of Fe in physiology.

7.8

MEASURING THE OXIDATIVE COST OF BREATHING: A COMPARISON OF METHODS USING RED-EARED SLIDERS TRACHEMYS SCRIPTA ELEGANS

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There is metabolic cost associated with the work required to overcome the elastic and resistive forces associated with breathing. These forces vary widely as a function of the anatomy of the lungs and body wall in different species. In turtles, the lungs are relatively compliant but the body wall with the carapace is very stiff. Studies designed to measure the cost of overcoming these forces in turtles using different methods have produced very different results. In an attempt to resolve this controversy, the present study obtained data using three different methods in a single group of red-eared sliders; a data regression method, unidirectional ventilation, and a hybrid method combining data regression and unidirectional ventilation. All three methods produced highly variable results (individual variability, differences between use of hypoxia and hypercapnia, evidence of hypoxia and hypercapnia-induced metabolic suppression). Based on data plotted for individual animals versus groups of animals and using all data points versus mean values for different levels of inspired gases, oxidative costs were obtained ranging from 0.4 to 20 mL O2/L air ventilated using the regression method, 80 mL O2/L air for the UDV method and 23-24 mL O2/L air for the hybrid method. All values are high compared to the estimate of 0.3 mL O2/L air obtained based on measurements of mechanical work of breathing and an assumed respiratory muscle efficiency of 10%. Supported by the NSERC of Canada.

7.9

DO SEMIFOSSORIAL RODENTS PAY AN ENERGETIC COST FOR THEIR ABILITY TO TOLERATE HIGH BURROW PCO2?

Inbal Brickner-Braun[1], Berry Pinshow[2]

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Semi-fossorial rodents spend daytime inside their burrows and forage on the surface at night, moving freely between normal atmospheric conditions on the surface and the hypercapnic environment of burrows, clearly having the capacity to tolerate hypercapnia and short-term changes in F_{CO_2}. For example, when allowed to choose between atmospheric air and air containing 5% CO_2 in a shuttle box, Prange et al.* found that *Acomys cahirinus* and *Gerbillus allenbyi* showed no preference and spent equal time in each. To assess whether this ability has an associated energetic cost, we measured resting VO_2 of 21 female Sundevall's jirds (*Meriones crassus*) at 25°C, for 2 h under atmospheric conditions, then for 2 h under hypercapnic conditions (7%CO_2, 19.5%O_2) and finally for 1 h in atmospheric conditions again. Initial results indicate that there is no difference between resting VO_2 under atmospheric conditions and hypercapnic conditions. Our results suggest that semi-fossorial rodents do not pay an energetic price in order to enjoy benefits of burrowing such as protection from predators and adverse environmental conditions, raising young in safety and hoarding food. *Prange, H., Reilly, W., Pinshow, B. *FASEB J*. 12(4):A419, 1998.

7.10

THE MECHANISMS OF OXYGEN EFFECTS ON THE FRUIT FLY *DROSOPHILA MELANOGASTER*

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Low oxygen conditions decrease adult body size in a variety of organisms, including humans, but the underlying mechanisms have yet to be determined. We examined this question in the fruit fly, *Drosophila melanogaster*, by comparing growth in normoxia (21% oxygen) and hypoxia (10% oxygen). To test for a specific developmental stage of hypoxia-sensitivity, we used 24 h or stage-specific hypoxia exposure. Our results suggest that the late larval and early pupal stages are most sensitive to hypoxic exposure. We also compared cell size and number in wings of flies reared constantly in 21%, constantly in 10%, or with exposure to 10% only in the egg, larval or pupal stage (with the remainder of life spent in 21% oxygen). Flies reared constantly in 21% oxygen had larger wings and more and larger wing cells than flies reared constantly in 10% oxygen. Exposure to hypoxia during the pupal phase reduced wing cell size but not number. In contrast, exposure to hypoxia during the larval phase decreased both cell size and number. These data suggest hypoxia effects on size during the larval phase are mediated by effects on feeding behavior, and that oxygen level affects the growth of imaginal cells during the pupal phase. This research was partially supported by NSF EAR 0746352 to JFH, the Howard Hughes Medical Institute through the Undergraduate Science Education Program, the ASU School of Life Sciences, and the Arnold and Mabel Beckman Foundation.

7.11

THE EFFECT OF PALEOZOIC OXYGEN LEVELS ON THE DEVELOPMENT OF THE TRACHEAL SYSTEM IN THE EXTANT BLATELLA GERMANICA, THE GERMAN COCKROACH

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The causes for insect gigantism during the Paleozoic Era are still poorly understood. However, recent oxygen models indicate the evolution of giant insects is coincident with an episode of hyperoxia, leading to the hypothesis that an increase in available oxygen allowed the evolution of giant insects. As shown in studies on beetles, as insect body size increases the volume of tracheae increases at a faster rate, eventually placing a spatial constraint on maximal body size. We hypothesized that diameter of the tracheae may decrease under hyperoxic rearing conditions, ultimately relieving the spatial constraint. In order to test this hypothesis, 500 Blattella germanica were reared in seven different oxygen levels ranging from hypoxia (~12%) to hyperoxia (~40%). A subset of the cockroaches was transported to Argonne National Labs where they were imaged by x-ray synchrotron phase contrast imaging. The resulting images were analyzed using ImageJ, measuring the diameter of the tracheae at several different points including the legs, lateral abdominal cavity, and upper thoracic region. The results show a negative correlation between oxygen and tracheal diameter with the strongest correlation in the legs (ANOVA, $p<.003$). This decrease in tracheal volume due to increased oxygen supports a possible mechanism for insect gigantism, as hyperoxia could relieve the spatial constraints on size imposed by the tracheal system. This research was partially supported by NSF EAR 07463522.

7.12

A PROPOSED NATIONAL FACILITY FOR THE STUDY OF GLOBAL CLIMATE CHANGE - THE VARIABLE ATMOSPHERE LABORATORY (VAL)

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The Earth's climate and atmosphere are changing rapidly in response to the rise of a global industrialized society. Scientists have suggested that the resilience of many ecosystems will be compromised by climate change over the coming century; leading to decreased grain productivity, increased water stress, and widespread human health effects. However, key uncertainties remain in understanding the effects of global change on biological and geological processes. Current methodologies to study these effects are limited in their ability to distinguish cause and effect, primarily due to a lack of the technology required to conduct large, replicated experimental manipulations of multiple variables. We are proposing to develop and build the premiere climate change facility in the U.S. – the Variable Atmosphere Laboratory (VAL). Two NSF-sponsored workshops have supported a national VAL composed of miniworlds that: 1) allow regulation of multiple atmospheric gases and environmental conditions, 2) are sufficiently large to house small ecological communities and animals, 3) allow measurement of net fluxes of molecules between communities and the environment, and 4) are sufficiently replicated to allow statistical evaluation of hypotheses concerning climate change. VAL would provide a national

interdisciplinary facility for generating critical information necessary for rational planning of a sustainable Earth. Supported by NSF IOS-0748882 and NSF IOS-0929344.

7.13
VULNERABLE LIFE STAGES UNDER SIEGE OF CLIMATE CHANGE: ONTOGENESIS OF EURYTHERMY IN CARCINUS SPP.
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The research has been addressed on the physiological tolerance and the adaptative flexibility of marine model organisms in order to forecast and test the animal resilience to climate change. The hypothesis considers that the species tolerance range could be narrower in particular ontogenetic stages which represent the vulnerable phase with respect of marked environmental fluctuation. We have investigated thermal tolerance in the model eurytherm crabs Carcinus spp. and compared the physiological mechanism of resistance between adult and embryos. The results from ventilation rate and oxygen consumption measurements highlight the capability of adult to maintain the oxygen extraction efficiency over a wide range of temperature (10-25°C). Thus, the eurythermy of Carcinus spp. is achieved trough an integrated series of metabolic adjustment which overcome the usual oxygen limitation, well-known for many marine ectotherms. Conversely embryos display an acute thermal sensitivity, particularly high at level of blastula and gastrula while an initial tolerance is recorded for the following stages of development. This result has been integrated with data on 65 years of climatic observation and population abundance to cast the result obtained in vivo with the current trend of climate change. In conclusion, this study provides evidence that climate change has an impact on highly tolerant species by reducing the fitness of early stages during the ontogenesis of eurythermy.

7.14
THERMAL TOLERANCES IN ANDEAN FROGS: ARE HIGH-ELEVATION SPECIES MORE VULNERABLE TO CLIMATE WARMING?
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Amphibians are declining worldwide, but declines are particularly dramatic in tropical mountains where high endemism and vulnerability to introduced disease is leading to catastrophic extinctions. One the most important aspects of climate change in tropical mountains is the increase in temperature, which in the tropical Andes is accelerating deglaciation and is projected to reach 2–7°C by 2100. Models based on organismal physiology predict that tropical ectotherms will be severely impacted by climate warming. In order to better understand the potential effects of climate warming in montane frogs, we measured the tolerance to heat stress in 15 species along an elevational transect between 1200 and 3700 m in the eastern slopes of the Andes (Manu National Park, Peru). We used the loss of righting response to measure the critical thermal maximum of frogs in the species-rich family Strabomantidae. We found that critical thermal maxima decreased with elevation across strabomantid frogs and within species, but not as steeply as ambient temperatures. Consequently, thermal tolerance (the difference between the critical thermal maximum and the maximum ambient temperature at the corresponding elevation) increased with elevation, indicating that frogs at lower elevations are more vulnerable to thermal stress under a global warming scenario than frogs at higher elevations. This finding is not consistent with the fact that most threatened frogs are found at higher elevations. Our results suggest that climate warming is not the main driver of amphibian declines, which disproportionally affect high-elevation taxa.

7.15
PHYSIOLOGICAL AND ECOLOGICAL BASIS OF RECRUITMENT SUCCESS IN POPULATIONS OF A STREAM-BREEDING FROG
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Predicting species' vulnerability to environmental change requires integrating physiological and behavioral factors with population and community level factors. We focus on predicting the response of a stream-breeding frog (*Rana boylii*) to thermal stressors within the context of spatial variation in aquatic primary productivity. We evaluate whether tadpole thermoregulatory behavior can be used to predict survival to metamorphosis and distribution of populations across the landscape. In a thermal gradient, tadpoles selected temperatures (T_{set}) between 16.5–22.2°C. We calculated an index of habitat thermal quality, d_e from the difference between T_{set} and water temperatures. We reared tadpoles in four streams where temperatures are colder, warmer, or close to T_{set}. Temperature effects were mediated through the quantity and quality of algae available for tadpoles. Mortality increased with d_e if tadpoles ate low-quality periphyton, but not if they consumed nitrogen-rich diatoms. We tested whether temperatures associated with larval mortality were consistent with spatial patterns of recruitment by relating an index of breeding activity to d_e. When controlling for watershed area, there was an inverse relationship between breeding activity and d_e. These results show that an integration of thermoregulatory behavior and ecological context is needed to forecast recruitment success in response to thermal regime change. This work was supported by a grant from the California Energy Commission (Award # 500-08-031).

7.16
PHYSIOLOGICAL PREDICTORS OF CORAL GROWTH AFTER THERMAL STRESS
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Coral bleaching is currently a major ecological problem. In this process, the coral loses its symbiotic dinoflagellates, which are photosynthetic. Since the symbionts provide substantial nutrition, bleached colonies typically regress or even die. Insight into this process can be gained by laboratory studies. Using Cervera octocorals, colonies were thermally perturbed by placing them in incubators at 32°C for 12 hours. Several stolons of each colony were then filmed for 50 sec and the number of symbionts in the tissue and in the gastrovascular flow was measured. As well, velocities were taken of symbionts in the gastrovascular flow for each stolon. Colonies were then returned to normal culture conditions at 27°C. After 36 hours, colony growth or regression was measured. Five experiments with 5-6 colonies each were done. Colonies with large number of detached symbionts in the stolons generally recovered poorly. Remarkably, within-experiment variation in colony growth was predicted by gastrovascular velocities during thermal stress (paired-comparison t-test, $t = 7.8$ $p = 0.002$). Colonies with slower velocities showed faster growth. Slower velocities during thermal stress may allow detached symbionts to reattach, resulting in faster growth. These data suggest a mechanism (gastrovascular velocity) by which coral colonies can regulate their symbiont population. Such a mechanism may be relevant to patterns of bleaching observed in the field.

7.17
UPPER THEMAL LIMITS OF GROWTH IN BROOK TROUT (*SALVELINUS FONTINALIS*) AND THEIR RELATION TO STRESS PHYSIOLOGY
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Global warming presents many challenges for the conservation and management of wildlife, yet the mechanisms by which temperature affects populations are often unknown. Although somatic growth is a key aspect of population persistence, our understanding of the means by which temperature impacts growth and stress physiology is limited. In the present study, brook trout (Salvelinus fontinalis) exposed to constant temperatures (16, 18, 20, 22, and 24 °C) were monitored for growth and tissue samples were collected at 8 and 24 days for physiological analysis. Through 24 days, specific growth rate was highest at 16 °C (3.2 g•d-1) and decreased with temperature to a low at 24 °C (-0.9 g•d-1). The upper limit for growth was 23.4 °C. Plasma cortisol levels were lowest at 16 °C (1.3 ng•ml-1) and increased with temperature to a peak of 23.4 ng•ml-1 at 24 °C. Abundance of the inducible isoform of heat shock protein (Hsp)-70 in gill tissue increased with temperature and was 10.7- and 56.0-fold higher at 22 and 24 °C than at 16 °C. Thus, both plasma cortisol and Hsp-70 were elevated at temperatures where growth was reduced but had not become negative. Additionally, a reduction in gill N+/K+-ATPase abundance (80%) and activity (50%) may indicate impaired osmoregulatory capacity at stressfully elevated temperatures. These data suggest that sublethal yet stressfully elevated temperatures limit growth in brook trout and may provide a mechanism by which this species is ecologically limited.

7.18
HEMOGLOBIN EXPRESSION INFLUENCES THERMAL TOLERANCE OF ANTARCTIC NOTOTHENIOID FISHES
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The Antarctic Peninsula region is experiencing among the most rapid elevations in temperature of any marine environment. We assessed thermal tolerance of white- and red-blooded Antarctic notothenioid fishes using a modified CTmax (Critical Thermal Maximum) design. Temperature was elevated acutely from ambient at a constant rate of +3.6°C per hour and CTmax was defined as the temperature where animals lost righting response. CTmax temperatures of white-blooded icefishes Chionodraco rastrospinosus (13.3±0.2°C) and Chaenocephalus aceratus (13.9±0.4°C) were significantly lower than red-blooded Gobionotothen gibberifrons (15.5±0.2°C) and Notothenia coriiceps (17.1±0.2°C). Lepidonotothen kempi, a red-blooded species with low hematocrit, exhibited a CTmax (14.2±0.4°C) that was significantly lower than the other red-blooded animals and similar to icefishes. A strong relationship between CTmax and hematocrit (R2= 0.82) suggests that oxygen-carrying capacity may partially dictate acute lethal temperature. However, we detected no change in mRNA abundance of genes known to be upregulated under hypoxic conditions (HIF-1α; PHD2). One week of acclimation to +4°C had no effect on CTmax of N. coriiceps, indicating an inability to adapt to rising temperature under these experimental conditions. Our results suggest that icefishes are particularly sensitive to temperature rise due to lack of hemoglobin and may be sentinel species for climate change. Supported by NSF grant ANT 07-39637.

7.19
PHYSIOLOGICAL RESPONSES TO THERMAL RAMPING IN THREE LIFE STAGES OF A TENEBRIONID BEETLE
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Understanding the physiological limitations of temperature tolerance is an important step in characterizing how organisms will respond to significant changes in their thermal environments. Upper thermal limits are typically assessed in animals by ramping temperature at a set rate until a marked decline in physiological performance occurs. Despite the widespread use of this technique for assessing thermal tolerance, the physiological responses to thermal ramping are not well understood. We assessed thermal tolerance in three life stages (larvae, pupae, and adults) of Tenebrionid beetles (Tenebrio molitor) by measuring CO2 release during thermal ramping (thermolimit respirometry). We recorded three independent estimates of the critical thermal maximum (CTmax) for each animal: 1) temperature at peak CO2 release, 2) temperature at the last breath, and 3) temperature before a final CO2 burst. We compared these CTmax estimates among the three life stages, each of which produced a unique pattern of CO2 release. All three measures of CTmax were statistically identical among the three life stages. These results demonstrate that CTmax does not vary significantly among different life stages of this species, despite widely varying environments and behavior. Additionally, our results suggest that each of the three CTmax estimates we used may be reliable measures of thermal tolerance.

7.20
GLOBAL ANALYSIS OF THERMAL TOLERANCE AND LATITUDINAL RANGE SIZE IN ECTOTHERMS
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The extent to which species' geographic distributions reflect their limits of physiological tolerance provides the foundation for predicting relative vulnerabilities and range shifts under climate change. However, the degree to which physiological limits relate to species range boundaries remains a largely open question. We tested for relationships between temperature limits of survival and latitudinal range boundaries in marine and terrestrial biomes using a global synthesis of ectotherm data. Among terrestrial species, latitudinal range size does not increase with species' breadths of thermal tolerance, mainly due to the decoupling of the lower (equatorial) latitudinal extents of their ranges and physiological heat tolerance. By contrast, marine species with greater thermal tolerance breadths have larger latitudinal ranges, and both lower and upper latitudinal range boundaries extend further in species with greater heat and cold tolerance, respectively. Our findings indicate that the equatorial range boundaries of terrestrial species are not limited by heat tolerance, and that marine species distributions are more likely to track climate change.

7.21
INTRASPECIFIC VARIATION IN THERMOTOLERANCE OF THE INVASIVE EUROPEAN GREEN CRAB, CARCINUS MAENAS, ON THE WEST COAST OF NORTH AMERICA

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With the imminent threat of global climate change, in particular the predicted increase of oceanic temperatures of 2-4.5°C ±1.8°C by the year 2100, there has never been a greater need to understand the mechanisms that allow both native and invasive marine species to mitigate these changes. A comparative laboratory analysis of Carcinus maenas, the European green crab sampled from the northern (British Columbia-BC) and southern (California-CA) sub-populations of its invasive range on the west coast of North America has yielded intraspecific differences in both organismal and cellular thermotolerance. After laboratory acclimation at 6 and 23°C, heat ramping of 4°C/ hour was conducted to assess CTmaxima for both sub-populations. At the 6°C acclimation, the CA group had a CTmax of 34.72°C whereas the CTmax of the BC group was 31.65°C, a difference of 3.07°C. Moreover, after 23°C acclimation, the CA group displayed a CTmax of 36.25°C which was significantly different than the CTmax of the BC group which was 35.52°C. Also, the inducible Hsp70 protein produced during this heat exposure was quantified and revealed that at both acclimation temperatures, the CA group had a greater relative Hsp70 expression than the BC group. Coupled with the traditional approaches that define thermotolerance, novel tools must be investigated. C/EBP d has been shown to be induced by heat stress in C. maenas, and may provide yet another metric of sub-lethal heat stress in marine species. Funding sources: National Science Foundation Graduate Research Fellowship Program grant number 220005 to A. L. K. and Portland State University Department of Biology.

7.22
THE "WEAKEST LINK" IN THERMAL TOLERANCE: A COMPARATIVE STUDY OF NEURONAL AND CARDIAC THERMAL TOLERANCE LIMITS IN PORCELAIN CRABS, *GENUS PETROLISTHES*
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Aerobic thermal tolerance limits are hypothesized to depend upon the ability of the cardiorespiratory system to effectively deliver oxygen to tissues. Thus, what makes one organism more eurythermal than another may depend upon the thermal performance of the "weakest-link" in the cardiorespiratory system. We hypothesize that in decapod crustaceans thermal limits of neurogenic hearts may be set by neuronal pacemaker cells. To test this hypothesis, we examined cardiac and neuronal thermal performance in five species of porcelain crabs, genus Petrolisthes. Crabs were acclimated to constant or fluctuating temperatures and performance of afferent neurons determined by extracellular recording of spontaneous action potentials. Arrhenius Break Temperatures were used to calculate critical temperatures for neuronal performance and found to correlate with habitat temperature. Neuronal performance near cardiac CT_{max} was assessed in one species acclimated to different temperatures. Thermal acclimation influenced performance half-life with 8°C acclimated nerves losing all activity within 20 min, whereas 25°C acclimated nerves lost almost none. Thermal performance limits of neuronal and cardiac function were correlated, though cardiac limits were more extreme. Our data suggest nerve function is more thermally sensitive than heart beat rate and raise the possibility that nerve failure may underlie heart failure, ultimately setting organismal thermal limits. Funded by NSF 0113184 and 0533920.

7.23
CAN WALKING HIBERNATION HELP POLAR BEARS (*URSUS MARITIMUS*) COPE WITH CLIMATE CHANGE?
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Hibernation gives animals flexibility in adjusting to changes in resource availability. Polar bears (Ursus maritimus) may enter a state of adaptive fasting or "walking hibernation" when access to their preferred prey, seals, is limited. During summer in the southern Beaufort Sea, some bears follow the retreating sea ice north while others remain land-bound. As a result of climate change, ice retreat is extended, forcing bears to remain on shore longer and carrying ice-bound bears into areas where seal density may be low, likely inducing walking hibernation in both groups. In 2008 and 2009, 38 adult bears were captured in early summer. Tissue samples were collected and each individual was implanted with a temperature (Tb) logger and fitted with a radio transmitter; 18 bears were recaptured in late summer, re-sampled and instruments recovered. Preliminary analyses of δ13C of exhaled CO2, respiratory quotients (RQs), serum urea:creatinine ratios, Tb, and activity suggest that ice-bound bears exhibit high activity rates, slightly reduced metabolic rates, and may exhaust their lipid stores by fasting. Bears on shore had low activity rates, variable metabolic rates, and apparently did not exhaust lipid stores. Pending analyses of δ13C of fat tissue, δ15N of muscle tissue, muscle morphology, serum non-esterified fatty acids, and other blood parameters will verify the use and benefits of walking hibernation in polar bears. Principal funding from National Science Foundation (0732713).

7.24
A PHYSIOLOGICAL BASED MODEL OF LANDSCAPE USE IN ELEPHANTS: INTERACTIONS BETWEEN THERMAL PHYSIOLOGY, WATER USE, AND ENERGY DEMAND
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Elephants, the largest land mammal, are highly dependent on surface water for efficient heat dissipation. As a result, they must shuttle between two critical resources, water holes to satisfy thermoregulatory demands and quality foraging areas to meet nutritional needs. This lifestyle has physiological consequences for the elephant and substantial landscape-level impacts associated with elephant grazing. Here we examine how climate interacts with relative food abundance, thermal physiology and water use patterns of elephants. Relationships between ambient temperature (T_a), water flux, and heat transfer of adult elephants (mean body mass = 3780kg, total n = 12) were empirically measured and used to construct a physiology–climate based stochastic dynamic state variable model (SDSV). This model was used to explore how limitations in surface water, ambient temperature, and food abundance/quality together influence spatial-use patterns and habitat impacts by elephants. Elephants relied on evaporative cooling to dissipate more than 100% of resting heat production at $T_a \geq 22.7$ºC. This temperature-water relationship resulted in predicted mean ranging distances from water that were more than 4 fold greater in habitats with mild climate ($T_{amax} = 21.2$ºC) relative to warmer climates ($T_{amax} = 32.9$ºC). Ultimately we show that physiology-based SDSV models provide a powerful process driven tool for evaluating habitat needs and impacts of large mammals. Funding by Wings World Quest and SICB.

7.25 Withdrawn

7.26 Withdrawn

7.27
THE ROLE OF MUSCLE PLASTICITY DURING COLD ACCLIMATION IN A BROWN ADIPOSE TISSUE DEFICIENT MOUSE.
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Shivering thermogenesis is the primary mode of adaptive heat production in mammals lacking brown adipose tissue (BAT), and sustained shivering is a powerful stimulus to drive an increase in muscle oxidative capacity in these animals. We are interested in the plasticity of the skeletal muscle metabolic phenotype during cold acclimation in the UCP-dta mouse. UCP-dta mice, a transgenic line lacking brown adipose tissue (BAT), do not appear to be cold intolerant and can survive a prolonged cold challenge at 4C. When presented with an acute cold challenge, summit VO2 in UCP-dta mice is significantly lower than in wild type controls. Cold acclimation significantly increases summit VO2 in UCP-dta mice, although it remains significantly lower than in cold acclimated wild type mice. Cold acclimation increases muscle oxidative capacity nearly equivalently in wild type and UCP-dta mice, but does not affect the respiratory function of isolated mitochondria. These results suggest that the lack of functional BAT in UCP-dta mice compromises their ability to produce heat during a cold challenge. While cold acclimation increased muscle oxidative capacity, summit VO2 is still compromised in UCP-dta mice and therefore the increase in the thermogenic capacity of muscle is not sufficient to fully compensate for the BAT deficit.

7.28
PHYSIOLOGICAL DIFFERENTIATION AMONG NINE-SPINED STICKLEBACK POPULATIONS
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Sticklebacks exhibit a wide geographical range and are increasingly used in ecological and evolutionary research. While pronounced morphological and behavioural differentiation among local populations has been shown, physiological differentiation, especially with respect to stress responses, has not been investigated.Metabolic rates (Mo_2) of nine-spined sticklebacks from five populations (including two populations of 'giant' fish) residing in similar latitude across Fennoscandia were compared under control conditions and under copper (Cu) exposure. Mo_2 were significantly different among populations (Baltic: 89.6 ± 18.3 to Russian: 56.5 ± 10.3 nmol O_2 min^{-1} g^{-1}, n = 12). Allometric Mo_2 – weight analysis revealed a metabolic scaling exponent of 0.97, significantly higher than a general exponent for fish (0.88), suggesting an elevated Mo_2 for the 'giant' stickleback populations. Cu exposure caused an increase in metabolic rate of 3.1 nmol O_2 min^{-1} g^{-1} / 1 mol l^{-1} increase in Cu. However, the Cu-induced changes in Mo_2 differed significantly among populations. Survival of fish following Cu exposure differed significantly among populations, with mean time to death from 32 days for the Baltic, and 68 days for a Russian population. The results demonstrate significant physiological differences in Mo_2 rate and stress response among local populations, and suggest that caution has to be exercised when different wild populations of a single species are used for ecotoxicological monitoring.

7.29
ACCLIMATION, ADAPTATION OR EXTINCTION DURING GLOBAL WARMING? CAN COMPARATIVE STUDIES OF DROSOPHILA THERMO-TOLERANCE PROVIDE AN ANSWER?
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Many species are believed to be threatened under a future global warming scenario. It remains, however, difficult to predict how such changes will affect populations as it is hard to identify the climatic and physiological parameters that restrict population range and as populations respond to climate change by acclimation and adaptation. Using a comparative approach we measured thermal tolerance in multiple species of drosophila and found tight correlations between functional measures of thermal resistance and species distributions. These correlations only applied if assessments of thermal tolerance were measured in acclimated animals. Thus, both developmental and adult acclimation should be considered if true thermal limitations are to be identified. Contrary to our expectations we found no differences in levels of phenotypic plasticity between tropical and temperate species. All species showed a larger degree of phenotypic plasticity for cold tolerance and a limited degree of heat acclimation suggesting that heat acclimation responses is unlikely to buffer the impact of global warming in any significant way. To complement these experiments we are testing thermal tolerance of 100 drosophila species which we have also found to be restricted by thermal tolerance. We are analyzing these data within a phylogenetic framework to examine whether there are evolutionary constraints which could potentially influence distributional responses in a future climate change scenario. Funded by The Danish and Australian Research Council.

7.30
PHYSIOLOGICAL AND LIFE HISTORY CONSEQUENCES OF STARVATION SELECTION IN DROSOPHILA
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In nature, animals may endure periods of famine to complete their life cycles. At the organismal level, these periods can be survived by increasing energy storage or reducing metabolism. We selected for starvation resistance in replicated populations of Drosophila melanogaster. After 45+ generations of starvation selection, populations survive starvation significantly longer (>10 days) than control populations (~3 days), with females surviving longer than males. Selected populations contain much higher amounts of lipid and carbohydrate than controls. These resources are acquired during an extended larval feeding period, so that newly eclosed adults immediately contain greater energy stores. Thus, selection for starvation resistance in adult D. melanogaster has significant effects on larval physiology. Metabolic rates of adults are lower in starvation-selected populations, despite their larger body size. In addition to decreased metabolism, activity levels of adults appear lower in selected populations. Increased starvation resistance has come at a life history cost; selected populations eclose ~24 hr later, have reduced egg-to-adult survival, and have lower fecundity. Supported by NSF award IOS-0719591.

7.31
TRANSCRIPTOME ANALYSIS OF DESICCATION IN DESERT ADAPTED *DROSOPHILA MOJAVENSIS*
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The recently sequenced genome of cactophilic Drosophila mojavensis provides a unique opportunity to obtain an integrated understanding of resistance to arid conditions in desert

animals. We used whole transcriptome microarrays to assess gene expression in four recently collected natural populations that breed and feed on different host cacti. Over 200 genes differed in expression among populations, while over 500 were differentially expressed as a result of desiccation stress. Host cactus affected the expression of only 18 genes, and the magnitude of these differences was always less than 50%. However, interactions between host cactus, population, and desiccation affected nearly 1/3 of the transcriptome. Thus, host plants affected how different populations responded to desiccation. Functional annotation clustering resulted in 49 clusters, the majority of them involved in metabolism, olfaction, phototransduction, cuticle, and behavior. Overall, 280 differentially expressed genes were found to belong to a total of 20 different KEGG pathways. Time series analysis of desiccation stress revealed sets of genes whose expression increased or decreased as desiccation progressed. Thus coordinated expression of metabolism, behavior, and cuticle genes helps this fly to survive harsh desert conditions, and suggests that climate change will have varying effects on local populations feeding on different host plants. Supported by NSF award EF-0723930.

7.32
HEAT ACCLIMATION PLASTICITY IN EVOLUTIONARY ADAPTED DESERT RODENTS: A LESSOM FROM THE HEAT SHOCK RESPONSE.
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Previous studies substantiated the axiom that in poikilotherms, "adaptive specialization" to specific extreme environments, limits adaptive plasticity and abrogates coping with acute exposure to extreme waves of similar environmental stress. Here, using the evolutionarily conserved HSP70 system and the heat shock response (HSR), we characterized differences between homeotherms and poikilotherms with this respect. The experimental model HSR/HSP70, prior to and after heat acclimation (HA, 34ºC, 1mo; that recapitulates evolutionary adaptation to heat) was used. We demonstrated that rodents evolutionarily adapted desert dwelling, have a stress recognition response similar to poikilotherms inhabiting extreme environmental conditions, with an attenuated HSR but with makedly elevated HSP72 reserves following HA. Proteomic analyses revealed that the diurnal desert rodents (Acomys russatus, Psammomys obesus) have less HSP70 isoforms than nocturnal congeners (A. cahirinus, Merioness crassus) thereby diminishing diurnals cytoprotective flexibility. Furthermore, comparing hsp72 mRNA and the HSR in these species, show that diurnal desert rodents respond to stress similarly to the HA nocturnals (with attenuated transcriptional flexibility vs. controls). Thus, improved thermotolerance seen following heat acclimation in homeotherms desert dwellers, stems from efficient homeostatic mechanisms to compensate for non-flexible molecular systems operating during acute heat stress.

7.33
HSP 70 EXPRESSION IN THE WESTERN FENCE LIZARD, *SCELOPORUS OCCIDENTALIS*: GEOGRAPHIC VARIATION IN STRESS AND ITS ROLE IN SPRINT SPEED SELECTION.
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The paradigm of local adaptation to temperature has been extensively studied in the laboratory as well as in the field. However, many studies have exclusively examined invertebrate species which are often restricted to thermally isolated microhabitats. In this study, we examined the potential for temperature adaptation in the western fence lizard, Sceloporus occidentalis, which occurs in the western United States across thermally heterogeneous habitats. We collected lizards from sites paired in altitude and latitude (four sites total) and measured sprint speed of lizards before and after exposure to an experimental heat stress (or control treatment). In addition, we measured abundance of HSP70, a molecular chaperone protein produced in response to thermal stress, at the end of sprinting trials. We found that all lizards exposed to a heat treatment suffered reduced sprint speeds relative to control animals but the magnitude of the decrease depended upon the original site of capture. Also, across all sites, increased expression of Hsp70 was correlated with reduced sprint speeds. These results indicate the potential for temperature adaptation to act within these populations and may lead to locally adapted populations of S. occidentalis.

7.34
ATLANTIC FOREST FRAGMENTATION AND EXERCISE PHYSIOLOGY IN SUB-TROPICAL ANURANS
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Fragmented areas display simpler communities relative to continuous areas, especially on Brazilian Atlantic Forest. Anuran survival in fragmented areas seems linked to the ability to establish metapopulations across a fragment matrix, and it may be a partial function of body size and exercise physiology. We test the hypothesis that "species or populations present only in continuous habitat have lower physiological capacity associated to locomotion than those present in fragmented habitats". We studied three anuran species in the Altantic forest of Ribeirão Grande, São Paulo using a comparative approach. First, we compared Physalaemus cuvieri and P. olfersii, as the former is a generalist and the latter is restricted to continuous forest. Second, we compared P. cuvieri and Proceratophrys boiei, from both continuous and fragmented habitats. P. cuvieri displayed higher resting and after-exercise metabolic rates, higher metabolic scope and better jumping stamina than P. olfersii. Regarding interpopulation comparisons, higher time to reach exhaustion for P. boiei and higher jumping stamina for P.cuvieri were detected on continuous habitas. Our results indicate that different environmental factors may affect each species differently with apparently no correlation with habitat structure. Further studies will contribute to understanding present and future's biodiversity patterns. Research in agreement of Declaration of Helsinki and APS. Supported by FAPESP (Process #2007/55053-0).

7.35
UNRAVELING THE CAUSES OF IMMUNE DEFENSE VARIATION BETWEEN LIFE-HISTORY ECOTYPES OF THE GARTER SNAKE *THAMNOPHIS ELEGANS.*
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Ecoimmunology theory predicts a link between life-history and immune defense strategies such that fast-living organisms should rely more on constitutive innate defenses compared to slow-living ones. This prediction is supported in two life-history ecotypes of the garter snake, Thamnophis elegans. Fast-living snakes that live in lakeshore habitats show higher levels of natural antibodies, complement-mediated lysis, and bactericidal competence than slow-living snakes inhabiting meadows around the lake. We performed a common-garden experiment to test whether these differences observed in the field are fixed or are mainly plastic responses to environmental differences between lakeshore and meadow habitats. Snakes born and raised in common-garden conditions reflected the immune phenotype of their native habitats when initially sampled at 4 months of age (i.e., lakeshore snakes showed higher levels of innate immunity than meadow snakes), but no longer showed differences a year later. These results suggest that differences in innate immunity between the two ecotypes have a strong environmental influence, although they do rule out a genetic contribution. Early genetic and/or prenatal maternal effects could explain the initial differences in innate immunity observed in the common garden. We discuss approaches to differentiate between these explanations and the implications of our results for ecoimmunology theory. The National Science Foundation provided funding for this research.

7.36
THERMAL SAFETY MARGINS AND FOSSORIALITY DURING EVOLUTIONARY RADIATIONS INTO OPEN HABITATS: THE CASE OF GYMNOPHTHALMINI LIZARDS AT THE BRAZILIAN CAATINGAS
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Exhibiting very low thermal safety margins, tropical forest lizards have arisen as the most vulnerable lizards worldwide. During dry climate periods, tropical forests gradually fade away into more open habitats with hotter microenvironments. As tropical forest lizard lineages have radiated extensively into these habitats, an important fraction of tropical lizard diversity might be at the extreme of their physiological resistance. Therefore, they might be unable to resist additional anthropogenic environmental warming. Alternatively, evolution at these regions may have armed them with abilities to cope with that problem. For example, the Gymnophthalmini (Fam. Gymnophthalmidae) is one the most diverse groups of lizards from Brazilian semiarid Caatingas and includes fossorial lifestyles and snake-like morphotypes. Here we assess thermal safety margins for four Gymnophthalmini species, two of them with fossorial behavior. Using X-ray imaging, we also analyze the impact of their burying abilities on their vulnerability to environmental warming. We show that, under the leaf litter, their thermal safety margins place them among the most vulnerable species registered to date (0,7-3,6 Cº). However, burying in the sand may increase their thermal safety margins more than three degrees. The advantage of snake-like morphotypes for burying is discussed. Financial support: CAPES and FAPESP.

7.37
EFFECT OF INCREASED SEASONAL TEMPERATURE ON THE REPRODUCTIVE SUCCESS OF DESERT TOADS USING SHORT-TERM EPHEMERAL POOLS
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Desert anurans use short-term ephemeral pools to breed and are faced with the challenge of completing metamorphosis before pools desiccate. Most anurans can reach metamorphosis in less than a month, and some, like Scaphiopus couchii, can develop and leave the pools in less than two weeks. Others have demonstrated that S. couchii is adapted to using smaller pools in arid regions in which other species (Anaxyrus sp.) cannot complete metamorphosis. Density dependant effects on growth and development in larger ephemeral pools can prevent the smaller S. couchii from ever reaching metamorphosis. Hence, through natural selection, S. couchii have presumably adapted to using the pools with the shortest hydroperiods by metamorphosing faster and at smaller sizes than other desert anurans. Developmental rates for anurans are likely influenced by pool temperature, food availability and larval density. Increased seasonal temperatures due to global climate change may reduce the advantage of S. couchii in short-term pools due to increased pool desiccation rates. Short-term ephemeral pools in the Sonoran Desert are monitored for surface areas, volumes, pool and ambient temperatures, and relative humidity. Evaporation rates for variable pool sizes and ambient temperature scenarios are modeled to evaluate time lost for metamorphosis and potential effects on larval fitness. This information is compared to developmental rates and pool sizes used by S. couchii and other desert anurans.

7.38
CHANGING SEXES IN A COMPLEX ENVIRONMENT - SHIFTS IN SKELETAL MYOSIN ISOFORMS IN COASTAL POPULATIONS OF CALIFORNIA SHEEPHEAD FISH
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Myosin heavy chain (MyHC) isoform expression was investigated in California sheephead, Semicossyphus pulcher, a protogynous hermaphroditic fish found in coastal Southern California and Northern Mexico. This species was collected from nine capture sites near the Channel Islands where both overall size, and size of individuals at the transitional sexual stage, varies considerably. We therefore cloned MyHC isoforms and analyzed proteins by SDS-PAGE to determine if gender, size, water temperature, or geographic location influenced contractile protein expression. The relative percentage of the fast MyHC isoform decreased as sheephead fish transitioned from female to male. The highest expression of slow MyHC isoform was observed in male sheephead. The lowest expression of slow MyHC was seen in Northern and off-shore female populations, when compared to Southern and near-shore female populations. Relative percentage of slow MyHC isoform positively correlated with standard length. Thus, in this interesting system where muscle mass undergoes dramatic growth accompanying gender transitions, MyHC isoforms are likely influenced by both sexual development and environment.

7.39
A NEW TOOL FOR INSECT FLIGHT METABOLIC RATE STUDIES: ELECTRO-STIMULATION OF FLIGHT IN GOLIATH BEETLES
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Flight is a key adaptation for insect ecological and evolutionary success. Flight metabolism and performance is preferably tested with insects in free flight because tethered flight has often been shown to yield low metabolic rates and performance. While optical methods have been developed for elicitation of high flight performance in flies, these have not yet been applied to

other insects. We have recently developed electro-stimulation techniques for beetles that reliably induce strong flight, turning, and variation in wing kinematics. These approaches allow simultaneous assessment of metabolic rate, gas exchange kinematics, wing kinematics and lift in large flying goliath beetles. Supported by DOD 3000654843 to M. Maharbiz.

7.40

BODY WEIGHT-SENSITIVE REGULATION OF SKELETAL MUSCLE COMPOSITION

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The molecular and biochemical bases of body weight-associated effects on muscle physiology are poorly understood. Here we show how expression of rat skeletal muscle troponin T, a key regulator of muscle activation and force output, responds precisely to natural and experimental variation in body weight. In obese rats, the troponin T response to weight gain was impaired, as heavier obese animals displayed troponin T expression profiles characteristic of much lighter lean animals and therefore a mismatch between body weight and muscle molecular composition. We propose that in obesity, body weight-inappropriate skeletal muscle troponin T expression may promote muscle weakness, reduced ability to participate in exercise, and a reduction in the rate of energy consumption. More broadly, we conclude that regulation of troponin T expression to accommodate variability in body weight is an evolutionarily conserved feature of skeletal muscle, as we recently demonstrated that an identical response occurs in insect flight muscles. This mechanism appears to be part of an active role of skeletal muscle in body weight and energy homeostasis, and provides a quantitative molecular marker to track how animal tissues sense and respond to body weight. Animal protocols were approved by the Institutional Animal Care and Use Committee at The Pennsylvania State University College of Medicine. This work was supported by NIH grant DK15658.

7.41

FORCE-LENGTH PROPERTIES OF THE LATERAL GASTROCNEMIUS IN THE GOAT

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Muscles are able to actively produce the greatest amount of force when operating at moderate lengths along their force-length (F-L) curve. In vivo recordings of operating lengths of the goat lateral gastrocnemius (LG) have previously been made, but it is unknown how these lengths correspond to the muscle's F-L properties. Consequently, in situ measures of LG muscle fascicle length and muscle force were obtained during fixed–end contractions following in vivo treadmill and jumping trials. LG fascicle length was obtained using sonomicrometry crystals and muscle force via a tendon buckle, with the muscle stimulated by a nerve cuff. Fascicle lengths were taken at proximal and distal locations in the muscle. For the two muscle locations examined, both were found to have optimal (maximum isometric force) operating lengths that were 40-50% greater than lengths measured during quiet stance. Measures of muscle force and length were also taken in vivo during various locomotor behaviours. It was found that during level locomotion the LG often operated at lengths that spanned the measured optimal length (105%-80% optimal length). This did not vary with speed. During locomotion on an incline the muscle tended to operate at even longer lengths (115%-85% optimal length), a trend that was exaggerated as locomotor speed increased. This indicates that during incline locomotion the goat LG begins to contract at longer lengths and relies more heavily on passive tension from stretched elastic elements in this system. In contrast, during jumping the LG operated at lengths shorter (92%-77% optimal length) than either level or incline locomotion. (Supported by NIH AR055648).

7.42

SCALING OF METABOLISM, GROWTH, AND NETWORK ORGANIZATION IN COLONIES OF THE SEED HARVESTER ANT, *POGONOMYRMEX CALIFORNICUS*

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The hypometric scaling of metabolic rate with body size is among the most general patterns in biology. We investigated whether this pattern extends to physically independent eusocial systems by measuring the standard metabolic rates of whole functioning colonies of Pogonomyrmex californicus. Contrary to the predictions of an additive model, colony metabolic rate allometry resembled the pattern observed for individual organisms, scaling hypometrically with the 3/4-power of whole colony mass. This pattern could not be explained by scaling of individual ant size across colonies or by the effect of density on metabolic rate. While growth rates typically decrease in larger individuals, larger ant colonies exhibited higher growth rates and growth efficiencies. Isolated worker groups exhibited isometric metabolic rate scaling, suggesting that the social environment of the colony was critical to regulating individual patterns of metabolism and work output. The distribution of individual walking speeds became less uniform as colony size increased, suggesting that disparities in effort among individuals increased with colony size. The fraction of relatively inactive ants may explain a major component of metabolic hypometry. This work was supported by NSF IBN 0419704, NSF EAR 0746352, NSF 0446415, a NSF GRFP, Sigma Xi GIAR, and ASU GPSA.

7.43

SLEEP- AND DIVING-ASSOCIATED APNEAS DO NOT CAUSE SYSTEMIC OXIDATIVE DAMAGE IN NORTHERN ELEPHANT SEAL PUPS

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Seals are routinely exposed to breath hold (apnea) bouts while diving and sleeping. Apnea in seals is characterized by decreases in cardiac output, bradycardia, and peripheral vasoconstriction, resulting in blood flow redistribution towards oxygen-sensitive tissues and in hypoxemia. Following a bout of apnea, an increased cardiac output restores blood flow to tissues and blood oxygen content. These cyclic bouts of apnea-induced hypoxemia and ischemia/reperfusion present seals with the potential for reactive oxygen species production and, thus, oxidative stress. Because apnea is a natural component of seal's behavior, we hypothesize that does not increase oxidative damage. To test our hypothesis, sleep apnea and voluntary submersion experiments were conducted in 6 northern elephant seal pups. Seals were catheterized and allowed to sleep or dive in a shallow tank. Blood samples were collected during catheterization and at several time points during submersions and sleep apneas (1, 3, 5, 7, 10 min), as well as after the bouts (1, 3 min). Plasma levels of four oxidative damage markers (4-hidroxynonenal, nitrotyrosine, protein carbonyls, and 8-isoprostanes) were measured by ELISA. Apneas did not elicit changes in any of the markers of oxidative damage suggesting that seals are adapted to cope with apnea-derived hypoxemia and ischemia/reperfusion. These data emphasize the protective effects of the seal's antioxidant system, contributing to their natural adaptability to tolerate these potentially pathological conditions.

7.44

PROLONGED FASTING INDUCES INSULIN RESISTANCE IN THE NORTHERN ELEPHANT SEAL (*MIROUNGA ANGUSTIROSTRIS*)

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During its prolonged fast, the northern elephant seal exhibits hyperglycemia, hypoinsulinemia, and hypercortisolemia, conditions commonly associated with insulin resistance. Because seals do not exhibit any apparent detriment, they provide an ideal model for the study of adaptive mechanisms to the tolerance of insulin resistance. We measured the response of early- (2-3 weeks postweaning; n=5) and late-fasted (over 6 weeks postweaning; n=8) pups to glucose tolerance tests. Blood glucose was measured using a commercial glucometer. Plasma insulin and cortisol were measured by RIA. The area under the curve (AUC) was calculated for all parameters. Mean $AUC_{glucose}$ increased 74% between early and late fasting (35 ± 1.8 vs 61 ± 5.4 %/min) while mean $AUC_{insulin}$ decreased nearly 2-fold (4.5 ± 0.3 vs 2.3 ± 0.2 %/min) suggesting that glucose clearance and glucose-dependent insulin secretion are impaired. Mean plasma cortisol increased 2-fold with fasting (124 ± 6 vs 257 ± 30 nM) and the glucose challenge suppressed $AUC_{cortisol}$ similarly at early and late fasting (-39.4 ± 4.0 vs -41.9 ± 5.9 %/min) suggesting that adrenal responsiveness to glucose was not altered. The cellular mechanisms evolved by these animals to tolerate chronic insulin resistance without harmful effects suggests that insulin is not critical for the regulation of glucose metabolism in fasting seals, and thus, may be an ideal model for the study of type 2 diabetes mellitus. Funded by NIH HL091767 and UC Start-up funds.

7.45

BROWN ADIPOSE TISSUE AND NON-SHIVERING THERMOGENESIS AID HARP SEALS (*PAGOPHILUS GROENLANDICUS*) BUT NOT HOODED SEALS (*CYSTOPHORA CRISTATA*) AT BIRTH

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Both harp and hooded seal neonates face harsh environmental conditions in the North Atlantic. But, because harp seals are born with a thinner blubber layer than hooded seals (1.2±0.2 vs 2.0±0.2cm, p=0.01) and have a wetable lanugo pelage, they may use different tactics to maintain euthermia. To test whether neonates rely on non-shivering thermogenesis (NST) to generate additional heat, brown adipose-like tissue (BAT-lt) was collected from the neck region of neonatal harp (n=5) and hooded seals (n= 6) and examined histologically and biochemically. BAT-lt from both species consisted of multilocular cells filled with lipid droplets. BAT-lt from harp seals contained more lipid droplets/100um2 (p=0.02), however, these lipid droplets were only slightly smaller than those in hooded seals (65.8um^2vs 91.1um^2, p=0.16). Uncoupling protein 1 was only detected BAT-lt from neonatal harp seals. Activities of citrate synthase, cytochrome c oxidase, and β-hydroxyacyl CoA dehydrogenase were all significantly higher in harp seals. Together, these findings suggest lipids in BAT-lt tissue of harp seals are oxidized to generate heat via NST prior to the establishment of a thick blubber layer, while the neonatal hooded seals' thicker blubber layer and dense pelage are sufficient for maintaining euthermia without NST. This project is funded with support from Alaska EPSCoR (NSF EPS-0346770), Department of Fisheries and Oceans Canada and a UAF Center for Global Change Student Research Award.

7.46

THE SUBLETHAL EFFECTS OF MULTIPLE FREEZE-THAW CYCLES IN THE WOOLLY BEAR CATERPILLAR *PYRRHARCTIA ISABELLA*.

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One of the most significant abiotic challenges for insects that overwinter in temperate environments is the threat of freezing. However, for insects that survive freezing, this stress can occur more than once. Freeze-thaw cycles are common in temperate regions and are changing in frequency much more rapidly than changes in mean temperature. And while biochemical correlates of a single freeze exposure are relatively well-understood, little work has been completed on the effects of more than one freeze-thaw cycle. In this study we investigated the sublethal effects of these cycles in the freeze tolerant final instar Arctiid caterpillar Pyrrharctia isabella. We compared groups frozen 5 times for 7 hours with groups frozen once for 35 hours, and quantified sublethal effects with immune function, changes in metabolic reserves, metabolic rate, tissue damage, and changes in freezing point. A single freezing event was associated with a 1M increase in glycerol content, and a concomitant approximately 1 degree Celsius decline in freezing point. We found that while caterpillars that experienced multiple freeze thaw cycles had enhanced immune function (mortality after fungal infection was similar to controls at 11.1%), they also had higher mortality (varied between 22.2 and 40.5%) and greater gut damage than controls or long freeze caterpillars. This, along with no changes found in metabolic rate or reserves indicates a likely lack of ability to repair freeze-induced damage.

7.47

COMPARATIVE PROTEOMICS: ANALYZING THE RESPONSE OF THE ASCIDIAN CONGENERS *CIONA INTESTINALIS* AND *C. SAVIGNYI* TO ACUTE HEAT STRESS

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The sea squirts Ciona intestinalis and C. savignyi have disparate distribution patterns, which may result from differences in their thermal tolerance limits. Because C. intestinalis, an almost cosmopolitan species, has a more widespread distribution, it is thought that they are better adapted to endure a wide range of temperatures. In order to compare the heat stress response between these two congeners, we studied global changes in protein expression using a proteomics approach. To induce heat stress, animals of both species were exposed to temperatures including 18˚C, 20˚C, 22˚C, 23˚C, 25˚C, or 28˚C for 6 h, and then were left to recover at a control temperature (13˚C) for 16 h. A quantitative analysis, using 2D gel electrophoresis and gel-image analysis, showed that 15% and 20% of the all protein spots detected demonstrated changes in expression in C. intestinalis and C. savignyi, respectively. Using matrix-assisted laser desorption ionization (MALDI) tandem time-of-flight mass spectrometry, we were able to identify proteins with a 90% success rate. Our results indicate that

proteins involved in cytoskeletal organization, calcium-regulation and apoptosis differ between the species in response to heat stress. These cellular processes may suggest possible sites of adaptive variation that could lead to differences in thermal tolerance limits.

8.0: PHYSIOLOGICAL EFFECTS OF OCEAN ACIDIFICATION ON MARINE ANIMALS IN TIMES OF OCEAN WARMING: ECOSYSTEM IMPLICATIONS

8.2

CARBONATE FORMATION IN FISH GUTS: ROLES AND CONSEQUENCES

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The majority of oceanic production of calcium carbonate (i.e. the inorganic half of carbon cycle) is conventionally attributed to marine plankton (coccolithophores and foraminifera in particular). However, marine teleosts also produce precipitated carbonate crystals within their guts and excrete these at remarkably high rates. Carbonate formation is a by-product of drinking Ca2+-rich seawater and high rates of intestinal bicarbonate secretion, and plays important roles in physiological processes such as osmotic and ionic regulation, acid-base balance and respiratory gas exchange. When individual CaCO3 excretion rates are combined with estimates of global fish biomass it conservatively suggests they contribute 3-15% of total oceanic production, with more liberal assumptions providing values 3 times higher than this (1). The unusually high magnesium content of fish carbonates is predicted to speed up their dissolution with depth which would play an important role in replenishing the alkalinity of the surface ocean and enhancing the absorption of atmospheric CO2. Fish carbonate production increases considerably more with rising temperature (Q10 ~ 2.3 to 2.7) and decreasing size (doubling for every 10-fold drop in body mass) than is explained by metabolism alone. Increasing ambient PCO2 to 4,500 ppm also doubles carbonate excretion rate. So fish are likely to become an increasingly important component of the inorganic carbon cycle. Funded by BBSRC & NERC. 1) Wilson. R.W. et al. Contribution of Fish to the Marine Inorganic Carbon Cycle. Science 323, (2009) 359-362.

8.4

COMPARING THE RESPONSE OF SHALLOW-WATER AND DEEP-SEA DECAPOD CRABS TO OCEAN ACIDIFICATION

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Decapod crabs play a key role in most benthic marine food webs, often serving as both predator and prey, and thus are important in the function of marine ecosystems. Future changes in ocean conditions due to ocean acidification, warming, and hypoxia could affect the survival, performance, and productivity of decapods, with indirect impacts on ecosystem performance. We compared the physiological performance of deep-sea (Chionoecetes tanneri) and shallow-living (Cancer magister) decapod crabs under normocapnia and acute hypercapnia (1% CO2), in combination with oxygen levels typically found in their habitats (hypoxia for C. tanneri; normoxia for C. magister). Acid-base regulatory capacity in the deep-sea crab was far weaker than demonstrated in the shallow water crab. Cancer magister was able to recover to normal hemolymph pH in 24 h, while in the deep-sea crab, extracellular pH was completely uncompensated. Gill enzyme activities (ATPases and carbonic anhydrase) were 2 to 13.2-fold lower in the deep-sea crab, and routine metabolic rate was 4.5 times lower. Reduced ion-regulatory capacity, coupled with lower metabolism is likely to render deep-sea crabs more susceptible to ocean acidification and other climate-related stresses than shallow water crabs. This implies that the function of deep-sea ecosystems may be more sensitive to stress from ocean acidification than shallow water systems. (MBARI Proj. 900703).

8.5

PH REGULATION IN REEF CORALS: UNDERSTANDING FUNDAMENTAL PHYSIOLOGY IN AN ERA OF GLOBAL CHANGE.

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The maintenance of pH is a fundamental part of homeostasis in all living organisms, modulating many aspects of cell physiology. In corals, pH regulation is involved in processes linked to the productivity and growth of coral reef ecosystems, namely symbiosis with photosynthetic algae and coral calcification. These physiological systems are disrupted by changes in seawater pH and carbonate chemistry predicted to occur under ocean acidification. A research programme at CSM concerns the regulation of pH in extracellular and intracellular compartments of corals and model symbiotic cnidarians. Recent work using confocal microscopy and fluorescent probes has achieved the first measurements of coral intracellular pH in cells with and without symbiotic algae. Light exposure of these cells induces an increase in intracellular pH in algae-containing cells only, suggesting a link between changes in acid-base balance and photosynthesis. Current studies also focus on links between pH regulation and calcification, and aim to provide greater mechanistic understanding to how ocean acidification causes declines in coral calcification. Using intact corals, we are investigating pH in the calicoblastic (skeleton producing) cells and in the medium at the tissue –skeleton interface. Regulation of pH in these compartments is critical to the calcification process and the maintenance of coral reefs under global change. SUPPORT: Government of the Principality of Monaco. REFERENCES: Venn AA., Tambutte E, Lotto S, Zoccola D, Allemand D, Tambutte S. (2009). Imaging intracellular pH in a reef coral and symbiotic anemone. Proceedings of the National Academy of Sciences of the USA. 106 (39) 16574-16579. Weis V., Allemand D. (2009) What determines coral health? Science. 324 : 1153-1155.

8.6

PREDICTING THE CONSEQUENCES OF OCEAN CHANGE – HOW ECOLOGICAL PHYSIOLOGY AND GENOMICS CAN CONTRIBUTE

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Ocean acidification (OA), the reduction of ocean pH via the absorption of anthropogenic atmospheric CO2, is expected to impact marine ecosystems through its affects on marine calcifying organisms. These impacts are not well understood at the community and ecosystem level, although the consequences are likely to be substantial, involving range shifts and population declines. A current focus in OA research community is to understand the resilience that organisms possess to withstand such changes, and furthermore to extend these investigations beyond calcification, addressing impact on other vulnerable physiological processes. Using standard morphometric methods and gene expression profiling with a DNA microarray, my lab group and I have investigated the impacts of high CO2 conditions on the development of purple sea urchin larvae, a pelagic stage that forms a calcium carbonate endoskeleton during development. One of our larger goals is to see if we can identify thresholds in the physiology of the larvae in response to high CO2 conditions. In our studies, the larvae were raised from fertilization to pluteus stage in seawater with elevated CO2 concentrations. In this presentation, I will discuss the morphometric data and I will link these organismal-level observations with gene expression profiles. Gene expression patterns suggest that there is indeed a threshold for the larvae that is around 500 ppm CO2. These data will be considered in the light of the evolutionary history and biogeography of the organism, i.e., what levels of pCO2 do purple sea urchin larvae encounter in nature, and how might this influence our conclusions about their response to OA conditions as simulated in the lab? Supported by NSF awards OCE-0425107 & ANT-0944201 to GEH.

8.7

PERFORMANCE CAPACITY AFFECTED BY OCEAN WARMING AND ACIDIFICATION: THE LINK TO ECOSYSTEM CHANGE

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Warming of the world's oceans causes shifts in the composition of marine ecosystems; CO_2 induced ocean acidification likely interferes with these patterns. The paper addresses the physiological and genomic mechanisms shaping performance capacity. In molluscs, crustaceans and fishes from various latitudes we tested hypotheses proposing physiological principles, not only of temperature but also CO2 effects. Thermal windows of optimized performance were considered as a basic character defining species fitness and shaping species interactions. Here, ocean acidification may disadvantage those species characterized by a low capacity of extracellular ion and acid-base regulation. Available data suggest that one key consequence of these features is a narrowing of thermal tolerance windows, as well as a reduced scope for performance at ecosystem level. These changes in bioenvelopes may have major implications for the ranges of geographical distribution of these organisms and in species interactions. The respective understanding of cause and effect is required beyond empirical observations, for a more accurate projection of ecosystem effects and for quantitative scenarios. Support: EPOCA, BIOACID, European and German projects on ocean acidification. REFERENCES: Pörtner H.O.(2010) Oxygen and capacity limitation of thermal tolerance: a matrix for integrating climate related stressors in marine ecosystems. J. Exp. Biol. 213, 881-893. Pörtner H.O., Farrell A.P. (2008) Physiology and climate change. Science 322, 690-692.

8.8

EVOLUTIONARY RULES IN THE BRAVE NEW OCEAN

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As a consequence of anthropogenic CO2 emissions and climate change, oceans are becoming warmer (global warming) and more acidic (Ocean Acidification, OA). Rates of change are increasingly fast and we can only guess at the kinds of organisms that will suffer ("losers") or benefit ("winners") from this mayhem that is radically altering ecosystem structure. OA research is still in its infancy and the increasing amount of data available highlight the complexity of this ecological question. The impact of OA appears to be extremely species- and even population-specific and depends on life-history stages and the processes studied. The current paradigms (e.g. OA will negatively impact calcifiers) are now being revisited making any large scale prediction impossible or over-simplistic. However, sufficient data are now available to move on to the next generation of OA research and start testing hypotheses and predictions. To allow large scale predictions of the impact of climate change on marine ecosystems it is then needed to understand how OA will modify the evolutionary rules shaping marine ecosystems. This presentation will review the literature on the impact of OA on physiology and energetic balance to identify the new evolutionary rules in this changing ocean using echinoderms as models.

8.9

WHAT CAN THE STUDY OF PHYSIOLOGICAL RESPONSE TELL US ABOUT THE FUTURE OF MARINE BIODIVERSITY AND ECOSYSTEM FUNCTION IN A HIGH CO_2 WORLD?

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For the past 200 years, the oceans have been absorbing carbon dioxide at an unprecedented rate. It is now evident that this ongoing process has already significantly altered seawater carbon chemistry at a global scale and will continue to do so for hundreds of years to come; a phenomenon termed "ocean acidification". The challenge currently facing both scientists and environmental managers is to predict the long term implications of ocean acidification for the diversity of marine organisms and for the ecosystem functions this diversity currently supports. This challenge is all the more difficult considering that empirical data which specifically address the impact of ocean acidification on marine biodiversity are scarce. In the face of growing political and public pressure to provide answers, what predictions can be made and how reliable are the assumptions on which those predictions depend? In contrast to the study of community level impacts, the effects of elevated CO2 on the physiology of marine organisms are better understood. Here we examine the extent to which existing data, and understanding gained from previous physiological studies, can be scaled up community level impacts and thereby assist ecologists in predicting the likely impact of ocean acidification on marine biodiversity and ecosystem function.

9.0: EVALUATING THE EFFECTS OF GLOBAL CLIMATE TO THE FAUNA OF SOUTH AMERICAN BIOMES: CONSEQUENCES TO DIVERSITY FROM A PHYSIOLOGICAL PERSPECTIVE

9.2

AMPHIBIAN CHYTRIDIOMYCOSIS AND CLIMATE CHANGE: THE NEED FOR INTEGRATIVE STUDIES

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Amphibian chytridiomycosis, a disease caused by the fungus Batrachochytrium dendrobatidis (Bd), is currently an important subject for amphibian conservation. This disease recently emerged and is spreading over the globe affecting many amphibian (Anura and Caudata) populations, causing declines and extinctions. This fungus infects keratinized skin; how it kills is not well understood, but, near death, normal epidermal functioning is disrupted, leading to osmotic imbalance through loss of electrolytes. Has Bd affected all susceptible populations or will it continue to kill amphibians? Global climate change can affect these ectothermic animals

from the molecular level to species distribution. The amphibian immune system is also temperature sensitive and its relationships with diseases are still barely studied. Climate change also can be predicted to alter the distribution and virulence of Bd, which has a maximum growth temperature (~25o C) within the range at which amphibians are active. Predicting how global climate change will affect relationships between amphibian populations and Bd is an important and interesting field of study in which ecologists and physiologists must act together. Piotrowski et al. 2004. Physiology of Batrachochytrium dendrobatidis, a chytrid pathogen of amphibians. Mycol. 96(1):9–15; Voyles et al. 2007. Electrolyte depletion and osmotic imbalance in amphibians with chytridiomycosis. Dis. Aqu. Org. 77:113–118; Rödder et al. 2010. The potential of the emerging amphibian chytrid fungus under anthropogenic future climate change. Dis. Aqu. Org., in press. Funding: FAPESP.

9.3
IMPACT OF THERMAL VARIATION ON THE LIFE-HISTORY TRAITS OF PATAGONIAN HERPETOFAUNA
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Thermal constraints on activity may be the main source of geographical variation in reproduction and growth in lizards and amphibians inhabiting the Andes and Patagonian steppe. We used skeletochronology to estimate the thermal environment impacts on growth rates, age at sexual maturity and longevity. Shorter activity periods associated with high altitudes and latitudes result in lower growth rates, extended life-span, delayed maturity, and prolonged reproductive cycles, a combination that results in low fecundity. Accordingly, populations of *Liolaemus multicolor* and *L. irregularis* from the Andean region of Salta and the Patagonian *Phymaturus tenebrosus* reach maturity between the ages of 7 and 11 and life spans from 10 to 19 years. Intra-specific studies have shown that *H. darwini* achieves maturity between the ages of 3-4 but longevity increases with latitude, resulting in a maximum of 7 hatchlings against 5 at lower latitudes. A different pattern is seen in *L. pictus argentinus*: high altitude populations attain maturity one year earlier and have a shorter life-span, resulting in the same number of hatchlings than those at low altitudes. Strategies of Andean amphibians differ: life span of *Pleurodema thaul* is 5 years in all populations but severe environmental conditions are associated with maturity delay, higher annual growth rates and larger body size. Our preliminary results provide a framework for analyzing the eco-physiological responses of Patagonian herpetofauna to environmental changes.

9.4
THERMAL AND HYDRIC CONSEQUENCES FOR THE PHYSIOLOGY AND NATURAL HISTORY OF SELECT FROGS AND SNAKES OF BRAZIL
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Habitat selection and sensory detection are primary means by which ecology and physiological functions are linked. Detection and reaction to changes in environmental temperatures and humidity over appropriate time scales (e.g. daily, monthly, yearly) are essential elements in an ectotherm's toolkit to coping with or exploiting thermal and hydric fluctuations. Over the past 5 years, we have been examining the role of selective evaporative cooling in the physiology and behavior of basking frogs (Bokermannohyla alvarengai) and South American rattlesnakes (Crotalus durissus). In the former, evaporative water loss is kept to a minimum, primarily via behavioral means, rather than through specific physiological adaptations. Basking frogs are capable of maintaining body temperatures very similar to the environment, minimizing evaporative water and loss through postural adjustments. In the case of the rattlesnakes, we have witnessed the importance of respiratory cooling of the entire rostrum contributing to influencing thermal detection of prey and prey recapture behaviors in rattlesnakes, and propose that rostral cooling is influenced by environmental humidities. Establishing the capacity for ventilatory changes to specifically modify rostral temperature under natural conditions remains a challenge for future research. These results highlight the potential for humidity and temperature to interact and influence the interplay between physiology and behavior in reptiles and amphibians. Understanding these functions may help us to comprehend the importance of changes in these environmental variables in influencing habitat selection under natural conditions. (NSERC DG2620872009).

9.5
ANURANS IN SOUTH AMERICAN EXTREME ENVIRONMENTS: CONVERGENCE, DIVERGENCE, AND IMPLICATIONS FOR GLOBAL WARMING
Carlos Navas[1] and Jose E. Carvalho.
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Several South American extreme habitats are prone of modification due to global warming, with uncertain impact on the anuran fauna. Some such habitats exhibit remarkable lineage diversity, and lineages within a habitat may display common adaptive patterns, signaling evolutionary convergence. In this case, changes induced by global warming may have common effects to lineages, and generalizations regarding anurans may be habitat-specific. On the other hand, alternative patterns of adaptation may characterize lineages within a habitat. In this case, expected effects of global warming may contrast among lineages within a habitat, and generalizations need to be lineage-specific. I address this problem focusing on anuran thermal physiology in three South American extreme habitats: elevations above 3500 m in the tropical Andes, the semi-arid Brazilian Caatinga, and the Patagonian Andes, all of them characterized by significant anuran species richness and lineage diversity. Evolutionary convergence is evident in high elevation tropical anurans, even if lineages may differ in ability to tolerate freezing. Patagonian species exhibit two alternative paths of development, with our without tadpole overwintering, a differences that has implications in the context here discussed. Caatinga frogs evidence contrasting patterns of adjustment that range from behaviorally- to physiologically-based strategies. This synthesis suggests that the impact of global warming on anurans shall be better understood with lineage-specific approaches. Supported by State of São Paulo Science Foundation.

9.6
DRY AND GETTING DRYER: PHYSIOLOGICAL AND ECOLOGICAL ASPECTS OF ANURAN AESTIVATION IN THE BRAZILIAN SEMI-ARID
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The Brazilian semi-arid bioma called the Caatinga is characterized by the low rainy regime, higher temperatures and by the unpredictability of the rain fall. Despite of the apparent harsh conditions, several species of anuran amphibians have colonized the Caatinga. Given the length of the dry season, physiological adjustments to survive under hydric and dietary restrictions seem essential. We focus on the specific case of Pleurodema diplolistris (Anura, Leiuperidae), a species in which physiological strategies during dry season involve apparent underground activity, preservation of muscle structure and function, and only moderate aerobic metabolic depression. The pattern of aestivation observed in P. diplistris seems favored by a normoxic substrate and high water potential at increasing depths during the drought. These frogs do not feed during the dry season and spare stores of lipids and carbohydrates from muscle and liver, as indicated by the seasonal patterns of change in such reserves, and by the activities of selected metabolic enzymes (as PK, LDH, HOAD and CS). Future directions include studies with other anuran species that also aestivate in the Caatinga, hoping to understand common and specific underlying mechanisms across anuran lineages in this peculiar habitat. Financial support: FAPESP and CNPq.

9.7
STUDIES OF FISH REPRODUCTIVE PHYSIOLOGY AS A TOOL TO UNDERSTAND THE CONSEQUENCES OF FRAGMENTATION IN AQUATIC HABITATS
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Potamodrous fish, those living exclusively in rivers, usually migrate upriver for reproduction. When migration is not possible, females do not ovulate and consequently do not spawn. However, non-migrating females undergo final oocyte maturation (FOM) and ovulate after hormonal manipulation to the hypothalamus-pituitary-gonads (HPG). Why migration is necessary for the endocrine system to elicit FOM remains as an elusive, yet consequential question. Potamodrous fish often have reproductive behavior affected by dams, and barriers to migration seem the main cause or reproductive disruptions. The impact of barriers depends on climatic patterns because tributary rivers may be used as alternative migration routes. The problem of migration and reproduction in potamodrous fish apparently roots at the gonad level, and involves also gamete maturation and egg laying. Research with both captive and wild Salminus hilarii shows that disrupted migration blocks FOM and ovulation through shifts in the expression profile of gonadotropins and changes in gonad progestogens. Overall, migration seems necessary for the correct functioning of the endocrine system, particularly the HPG axis. References: Honji RM, Narcizo AM, Borella MI, Romagosa E, Moreira RG. 2009. Patterns of oocyte development in natural habitat and captive Salminus hilarii (Teleostei: Characidae). Fish Physiol. Biochem. 35, 109-123. Amaral JS, Melo RG, Honji RM, Moreira RG. 2007. Effects of migration impediment of Salminus hilarii on the pituitary-gonad axis. Comp. Biochem. Physiol. 148A, S44.

9.8
GLOBAL METABOLIC IMPACTS OF RECENT CLIMATE WARMING
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Documented shifts in geographic ranges, seasonal phenology, community interactions, genetics, and extinctions have been attributed to recent global warming. Many such biotic shifts have been detected at mid- to high latitudes in the northern hemisphere—a latitudinal pattern that is expected because warming is fastest in these regions. In contrast, shifts in tropical regions are expected to be less marked because warming is less pronounced there. However, biotic impacts of warming are mediated through physiology; and metabolic rate, which is a fundamental measure of physiological activity and ecological impact, increases exponentially not linearly with temperature. Therefore, tropical organisms (with warm baseline temperatures) should experience larger absolute shifts in metabolic rate than the magnitude of tropical temperature change itself would suggest, but the metabolic impact of climate warming on metabolic rate has never been quantified on a global scale. Here we show that estimated changes in metabolic rates in the tropics are large, are equivalent in magnitude to those in the northern temperate-zone regions, and are in fact far greater than those in the Arctic, even though tropical temperature change has been relatively small. Because of temperature's non-linear effects on metabolism, tropical organisms, which constitute much of earth's biodiversity, should be profoundly affected by recent and projected climate warming.

10.0: ENVIRONMENTAL ADAPTATIONS OF CARDIO-RESPIRATORY SYSTEMS

10.2
INFLUENCE OF OXYGEN, TEMPERATURE AND WATER ON INSECT PHYSIOLOGICAL PLASTICITY: LIFE IN LEAF BOUNDARY LAYERS
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As a group, herbivorous insects are the most successful and diverse animals in the world. Much of the work on them has focused mainly on the chemistry of how they interact with their host plants. By contrast, little work has examined an equally important issue: how plants structure insect-relevant microclimates. For small insects, including those still inside their eggs, plant-structured microclimates can differ markedly from nearby macroclimates. We show, in the context of a desert system (the sphingid moth Manduca sexta on the desert host plants Datura wrightii and Proboscidea parviflora), that microenvironments around eggs and neonate larvae are substantially more hospitable than are conditions even a few centimeters away. The degree of hospitality depends on a suite of complex but understandable leaf and environmental factors. Second, we show that the development of the embryonic respiratory system (both the eggshell and the tracheal system) reflects characteristics of the leaf microenvironments in which eggs are laid. A key problem for developing embryos is to balance the tradeoff between exchanging metabolic gases (oxygen and carbon dioxide) and conserving water. Embryos manage this tradeoff by using short-term plasticity to control permeability of the eggshell and by activating the tracheal system well before hatching. Leaf microclimates thus play an important but underappreciated role in defining the physical universe in which egg plasticity has evolved.

10.3
EFFECTS OF ENVIRONMENTAL OXYGEN ON CARDIOPULMONARY DEVELOPMENT AND FUNCTION IN THE AMERICAN ALLIGATOR: A CASE STUDY IN EXPERIMENTAL PALEOPHYSIOLOGY
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Recent models of Earth's atmospheric composition across the Phanerozoic Eon suggest O2 levels as high as 30-35% in the Permian, and as low as 12% in the Late Triassic and Early Jurassic. Thus, for some 550 million years, animals have evolved in an environment with a

variable source of atmospheric O2. Tobetter understand how global hypoxia and hyperoxia might haveaffected the growth and physiology of contemporary vertebrates, we created chambers serving as "atmospheric time machines". Since crocodilians survived theseglobal changes with distinctly conservative morphology, we incubated eggs and raised hatchlings of the American alligator (Alligator mississipiensis) at 30°C under chronic hypoxia (12% O2), normoxia (21% O2)or hyperoxia (30% O2). At hatching, hypoxic animals were significantlysmaller than their normoxic and hyperoxic siblings. Over thecourse of 3 months, post-hatching growth was fastest under hyperoxiaand slowest under hypoxia. Hypoxia, but not hyperoxia, causeddistinct scaling of major visceral organs–reduction ofliver mass, enlargement of the heart and accelerated growthof lungs. Despite compensatorycardiopulmonary remodeling, growth of hypoxic alligators isconstrained by hypoxia, which may limittheir food utilization capacity. This suggests that growth and metabolicpatterns of extinct vertebrates would have been significantly affectedby changes in the atmospheric oxygen level. Funded by the NSF IOB 04445680 and IOS 0922756 to JWH. REFERENCES: Owerkowicz, T; Elsey, RE and Hicks, JW (2009) Atmospheric oxygen affects growth trajectory, cardiopulmonary allometry and metabolic rate in the American alligator (Alligator mississipiensis). J Exp. Biol; 212; 1237-1247.

10.4
DEVELOPMENTAL AND EVOLUTIONARY RESPONSES OF INSECTS TO ENVIRONMENTAL STRESS
Jon Harrison[1], Erica Heinrich[1], Manoush Farzin[1], Katrina Ramsey[1], Saundra Dohrenwend[1], C. Jaco Klok[1]
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Coping with variation in oxygen availability is likely one of the most ubiquitous physiological challenges for animals. In fruit flies, Drosophila melanogaster, (outbred Oregon R or mixed lines to enhance genetic diversity) acute PO2 reduction down to 4 kPa has no effect on organismal metabolic rates, arguing against direct limitation of ATP production by moderate hypoxia. However, across this same range of PO2, survival, growth rate and adult size are progressively reduced, and development time extended. Moderate hypoxia affects both larval (reduced feeding rates, growth and cell number, extended development) and pupal phases (extended development, reduced cell number and adult size). The generality of these effects suggests signaling systems that suppress growth and adult size, perhaps to enable successful maturation under stressful conditions. Consistent with this hypothesis, knockdown of HIF signaling using RNA interference of tango expression reduced the effect of hypoxia on adult size. However, suppression of HIF signaling did not have the expected negative effect on survival during hypoxia. Exposure to multiple generations of flies to hypoxia did not result in evolution of a smaller size. Other studies have shown that D. melanogaster can evolve an improved capacity to survive and reproduce in hypoxia. However, these data suggest that plastic hypoxic responses that suppress development do not benefit fitness. Supported by NSF EAR: 0746352 and IBN 0419704 to JFH.

10.5
CARDIO-RESPIRATORY ENVIRONMENTAL ACCLIMATION IN DEVELOPING ANIMALS
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Future ecosystems will variously be warmer, colder, and/or more unstable as climate change accelerates. Knowing how development is affected by long-term changes in abiotic factors (e.g. temperature, oxygen, pH) must then be an essential component of future developmental studies. Environmental change is likely to especially affect the linked development of the cardiovascular and respiratory systems. While the most obvious disruptive effects to development are those leading to failure to thrive or mortality through ineffective gas exchange and convective transport, many cardio-respiratory responses are both subtle and adaptive, reflecting the ability of the embryo/larvae/fetus to mitigate the effects on development of environmental degradation (especially temperature change and hypoxia). Adaptive responses often involve modified morphological development coupled with emergent physiological phenotypes better able to cope with environmental changes. Mechanisms of interest include developmental plasticity, self-repair, and epigenetics, to name but a few. Developmental physiologists are also expanding cardio-respiratory studies across the life cycle to explore "fetal programming", in which developmental changes in early ontogeny that may be adaptive to environmental change may actually emerge later in the adult as phenotypic "time bombs" that reduce fitness. Because selection acts on a species' entire ontogenetic profile - not just the adult - understanding how the environment affects development will be crucial in understanding the overall impact of global climate change.

10.6
ADAPTATIONS TO HIGH ELEVATIONS IN HUMMINGBIRDS
Doug Altshuler[1]
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The hovering flight of hummingbirds is one of the most derived and metabolically costly locomotor behaviors of any animal. Hummingbirds exhibit the highest mass-specific metabolic rates among vertebrates, a feat accomplished by small muscle fiber size, increased mitochondrial density, and higher capillary volume density relative to other skeletal muscle. These enhancements for oxygen delivery allow hummingbirds to hover in hypoxic gas mixtures at experimental conditions well above their equivalent maximum elevations of occurrence. When experimentally supplied with supplemental oxygen in low density gas mixtures, hummingbirds can hover longer indicating that increased oxygen availability leads to increases in flight performance. However, the ultimate limits to hovering performance are set by aerodynamic requirements for flight. Many of the most evolutionarily relevant flight behaviors such as mating displays, escape from predators, and competitive interactions are characterized by brief, ballistic flights that are likely to be anaerobic. Difference in burst performance ability has been demonstrated to influence territorial behaviors and competitive abilities, especially at high elevations where aerodynamic power is limited through low air density. Support: NSF Grant IOS-0923849.

10.7
MECHANISMS AND EVOLUTION OF HYPOXIA TOLERANCE
Jeffrey Richards[1], Milica Mandic[1]
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Environmental hypoxia is a common occurrence in many aquatic environments and fish that inhabit hypoxic waters have evolved a remarkable suite of adaptive strategies to survive low oxygen including modifications to behaviour, morphology, physiology, biochemistry, and gene expression patterns. In order to study these adaptive strategies, we use a phylogenetically corrected analysis of 12 species of fish from the family cottidate (commonly called sculpins). These fish are distributed along the marine intertidal zone and differentially experience variation in the frequency and severity of hypoxia exposure. We have quantified the metabolic and behavioural responses of these fish to hypoxia exposure and determined that there is direct relationship between hypoxia tolerance and species distribution. Variation in hypoxia tolerance is primarily related to traits that affect oxygen uptake. Hypoxia tolerant sculpins have large gill surface areas, low routine metabolic rates, and high whole-cell hemoglobin-oxygen binding affinity. Variation in the red cell hemoglobin-oxygen binding affinity is an intrinsic property of the hemoglobin protein, but hypoxia tolerant sculpins have higher levels of allosteric modulators in their red cells than intolerant sculpins. (Supported by NSERC) Reference: Mandic, M., Todgham, A. & Richards, J. 2009. Mechanisms and evolution of hypoxia tolerance in fish. Proc. Roy Soc. 276:735-744.

10.8
PHYSIOLOGICAL MECHANISMS OF HIGH-ALTITUDE FLIGHT IN BAR-HEADED GEESE
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Bar-headed geese reach altitudes of up to 9000m during their biannual migration over the Himalayas. The ability of this species to maintain O_2 supply in the severe hypoxia at these elevations is impressive when considering the high metabolic costs of flight. The basis for this feat appears to involve evolutionary changes at multiple steps in the O_2 transport pathway. Bar-headed geese can breathe much more than low-altitude birds during hypoxia, largely due to alterations in the pattern and chemosensitivity of breathing. This species also has larger lungs and a higher haemoglobin-O_2 affinity. As a result of these characteristics, pulmonary O_2 uptake during hypoxia is markedly improved. Evolutionary changes in the heart, such as a higher capillarity, also improve circulatory O_2 delivery. Oxygen diffusion capacity in the flight muscle is improved by an enhanced capillarity and a redistribution of mitochondria towards the cell membrane. The relationship of these unique characteristics to flight altitude is independent of phylogeny and exists without prior exercise or hypoxia exposure. However, mitochondria isolated from bar-headed goose muscle are equally sensitive to reductions in O_2 tension to those from other geese. High-altitude adaptations that enhance O_2 supply, and sustain O_2 tensions at the mitochondria, therefore appear to be essential for the exceptional ability of bar-headed geese to fly high. Supported by NSERC of Canada.

10.9
INDIVIDUAL AND POPULATION DIFFERENCES IN BEHAVIORAL PHYSIOLOGY AND OXYGEN DELIVERY IN DAPHNIA
Peter Frappell[1]
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Daphnia reproduce parthenogenetically under favourable environmental conditions and the species complexes are cosmopolitan in distribution. They inhabit water bodies ranging from small ponds to large lakes, many of which undergo seasonal or daily fluctuations in oxygen. This talk explores the effects of altering temperature and oxygen tension on behavioural and physiological responses in Daphnia carinata</I>. The distribution of daphnia within a water column depends on the oxygen tension of the water, levels of hemoglobin, thermal preference and aggregation behavior. In particular, we show the degree of phenotypic variability in regulatory responses, primarily heart rate, and thermal preference is as marked between individuals within a clonal brood as it is in a population. The individual variation found within broods of this parthenogenetic species is likely to ensure survival of a clone line within fluctuating environments and may have the potential to increase population fitness.

11.0: SCHOLANDER ORAL PRESENTATIONS

11.2
ENVIRONMENTAL AND HANDLING IMPACTS ON CORTISOL IN THE NORTHERN ELEPHANT SEAL
Cory Champagne[1], Dan Crocker[2], Melinda Fowler[1], Dan Costa[1]
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Conservation physiologists are increasingly using glucocorticoid hormones as indicators of general stress since cortisol levels may indicate environmental and anthropogenic effects on animals. Glucocorticoid levels, however, may change in response to several features including capture and handling stress, body condition and fasting duration, complicating the interpretation of stress hormone variability. To understand changes in glucocorticoid levels over time and in response to handling in a free-ranging pinniped, we measured serum cortisol levels between foraging trips in adult northern elephant seals (Mirounga angustirostris). Additionally, we evaluated changes in cortisol concentration during capture procedures in juveniles. The type of handling significantly affected the magnitude of the stress response: the total cortisol release was greater under physical restraint than chemical immobilization while spatial confinement showed the least response ($n = 4$ & 5; paired t=-3.6 & 3.3; $p<0.05$). There was an inverse relationship between cortisol level and foraging success in adult elephant seals, measured by the rate of energy gain during foraging trips ($r2=0.29$, $p<0.01$). Previous research has demonstrated a link between conditions in the Pacific Ocean, foraging success and the allocation of resources toward reproduction (i.e. pup mass) in this species. These data suggest that elevated cortisol levels on return from sea may be associated with suppression of annual breeding.

11.3
CONSTRAINING THE THERMAL LIMITS OF METAZOAN LIFE: COMPARING THERMALLY INDUCED SHIFTS IN GLOBAL PROTEIN EXPRESSION IN VENT WORMS *PARALVINELLA SULFINCOLA* AND *PARALVINELLA PALMIFORMIS*
Geoffrey Dilly[1], C. Robert Young[2], William Lane[3], Jasmyn Pangilinan[4], Peter Girguis[1]
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Eukaryotic thermotolerance is pushed to its limits at one of the most extreme ecosystems on the planet, deep-sea hydrothermal vents. Temperatures span 2°C to >300°C. This range has given rise to some of the most thermally tolerant organisms on earth including the annelid Paralvinella sulfincola (P.s.) (5 to >50°C). P.s. and Paralvinella palmiformis (P.p.) (a congener with a reduced thermal range – 2 to 40°C) live at vents on the Juan de Fuca Ridge. We present data on global, quantitative changes in protein expression P.s. and P.p. across a range of environmentally relevant temps (P.s. 10, 30, 45°C; P.p. 12, 21, 38°C) to determine which factor(s) play a role in limiting the upper thermal bounds for each species. Live worms were maintained in flow-through chambers at in situ pressure to replicate vent conditions. Global protein profiles were generated using label-free MS/MS and referenced against a de novo P.s. EST library. As temps increase, both species exhibit an increase in enzymes involved in the creation/regulation of glutathione, an antioxidant. P.p. shows an expected increase in Heat Shock Proteins (HSPs); however, P.s. exhibit high levels in all treatments. We suggest that continual production of HSPs, as well as increased synthesis of antioxidants and downward control of aerobic metabolism allow P.s. to

survive high thermal stress. These data provide first insights into the biochemical factors that enable an organism to exist at the edge of eukaryotic thermotolerance.

11.4
THERMAL STRESS, OXYGEN LIMITATION, AND PROTEIN DAMAGE IN INTERTIDAL MUSSELS
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Intertidal mussels experience dramatic variations in temperature and oxygen availability (low during emersion when shells are closed). Such dynamic conditions, alone or in concert, may damage mussel proteins via several mechanisms: thermal denaturation, oxidative damage due to increased production of reactive oxygen species at warm temperatures, or reoxygenation injury. Using a combination of semiquantitative (dot blots) and qualitative (2D Western blots) immunoblotting, mass spectrometry, and biochemical methods, we are examining the amount, the specific targets, and the consequences of oxidative protein lesions following simulated tidal cycles in three Californian Mytilus congeners: the cold-adapted native blue mussel (M. trossulus), the warm-adapted invasive blue mussel (M. galloprovincialis), and the eurythermal ribbed mussel (M. californianus). These congeners differ in their constitutive levels of oxidative protein damage. Behavioral studies and measurements of mantle oxygen concentration demonstrate that periodic shell valve movements create repeated reoxygenation events, particularly in the blue mussels. These mussels also differ in their susceptibility to further oxidative damage during/following heat stress. M. californianus suffers prolonged periods of elevated protein carbonylation following simulated intertidal stress, while the two blue mussels reestablish baseline levels by 24 h after stressful episodes. Metabolic enzyme activity appears to decrease following these reoxygenation events. Overall, our data demonstrate the ecological relevance of oxidative protein damage in the intertidal. Funded by NSF grant IOS-0718734 and PISCO.

11.5
USING MITOCHONDRIAL BIOENERGETICS TO DETERMINE IF GOOD FISH LIVE IN BAD NEIGHBORHOODS
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We presume organisms are adapted to their environment, but what happens when conditions in the niche fluctuate due to global climate change? We contend that desert pupfish were stranded in warm springs (>33-34° C) and are chronically subject to physiological stress due to temperature driven effects on metabolic reactions and low oxygen availability. A lack of the requisite genetic diversity may have prevented natural selection to allow these fish to thrive or develop safety factors. Therefore, these fish are particularly susceptible to the effects of the modest increases in temperatures that will result from global climate change. By rearing fish at ecologically relevant temperatures and altering assay temperatures of hepatocytes and isolated mitochondria, we show that temperatures of the fish influence the efficiency of energy metabolism and the production of mitochondrial reactive oxygen species (ROS). We propose a model wherein fish limit their energetic status to allow for avoidance of presumably detrimental ROS but that this avoidance carries a cost. By monitoring the responses of mitochondria over a range of temperatures, we may predict biochemical/biophysical borders of mitochondrial adaptations in these fish. Our data may inform on how fish exploit new niches and concepts of evolution, conservation, and population dynamics.

11.6
EFFECTS OF TEMPERATURE ACCLIMATION ON CARDIAC PERFORMANCE OF LONGJAW MUDSUCKERS *GILLICHTHYS MIRABILIS*
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The oxygen limitation hypothesis states that an insufficient supply of O2 under thermal stress may be a critical factor in setting thermal limits in marine organisms. As the power supply of the circulatory system, heart plays a key role in establishing these limits. We used a highly eurythermal goby fish, the longjaw mudsucker Gillichthys mirabilis, to investigate temperature acclimation effects on cardiac performance. We measured heart rate, metabolic enzyme activity and hemoglobin content in fish acclimated to 9oC, 19oC and 26oC, as a function of acclimation time. Heart rate measurements were recorded under an acute heat ramp until the fish reached cardiac arrhythmia temperature (T_A). Fish acclimated to 9ºC for one month had a lower heart rate than 19ºC and 26ºC acclimated fish at their respective acclimation temperatures. 19ºC acclimated fish were able to reach the highest maximum heart rate of 120bpm (±5) whereas the 9ºC fish only reached 80bpm (±8). Hemoglobin (Hb) content of blood rose significantly with acclimation temperature, from 35 g/L in 9ºC fish to 60-80 g/L in 19ºC and 26ºC fish. Fish acclimated to higher temperatures were able to extend their T_A by 7ºC; however, there was no significant difference between 19ºC and 26ºC acclimated fish. These results illustrate the phenotypic plasticity of *G. mirabilis* and suggest that this eurythermal species maximizes its ability to circulate O_2 at 19ºC, a temperature shown by behavioral studies to be close to the species' preferred temperature.

11.7
BIRDS VS. MAMMALS: BLOOD OXYGEN MANAGEMENT STRATEGIES IN DIVERS
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Oxygen-hemoglobin dissociation curves were characterized for emperor penguins and elephant seals and applied to blood P_{O2} profiles obtained with backpack recorders during diving, revealing differing blood O_2 store management strategies between these two consummate divers. In emperor penguins, arterial O_2 saturation (S_{O2}) remained near 100% for much of the dive, preserving high O_2 content in the arterial system for critical organs such as the brain. Arterial S_{O2} did not decrease significantly until the final ascent of the dive, consistent with the decline in ambient pressure and decrease in air sac and arterial P_{O2} during ascent. These profiles demonstrate the significance of the respiratory O_2 store and the biochemical adaptation of the emperor penguin's high affinity hemoglobin documented in this work. In contrast, although there is a transient rise and peak near 100% arterial S_{O2} in the initial dive phase of elephant seals, these values decreased rapidly after this point, reaching levels as low as 8% and demonstrating exceptional hypoxemic tolerance for this species. Venous S_{O2} profiles revealed highly efficient and near complete utilization of the venous blood O_2 store during dives of both species. These findings reflect differences in the magnitude of the respiratory O_2 store and maintenance of gas exchange during diving between this bird and mammal, and reveal attributes that undoubtedly contribute to the extraordinary dives of these animals.

11.8
PERIPHERAL ARTERIAL CHEMORECEPTORS IN THE SOUTH AMERICAN RATTLESNAKE (*CROTALUS DURISSUS*) AND THEIR ROLE IN CARDIORESPIRATORY CONTROL
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Peripheral chemoreceptors monitor the levels of arterial blood gases and adjust ventilation and perfusion to meet metabolic demands. Chemoreceptors are present in all vertebrates studied to date but have not been described in non-chelonian reptiles. The goals of this study were to identify functional chemosensory areas in the South American rattlesnake (Crotalus durissus) and the role they play in ventilatory and cardiovascular control. Rattlesnakes were instrumented with transonic flow probes, arterial catheters and subcutaneous needle electrodes to measure shunt fraction, heart rate, blood pressure and ventilation, respectively. Catheters were placed at three putative chemosensory sites; aortic arch, pulmonary artery and carotid bifurcation for site specific activation of the chemoreceptors with sodium cyanide (NaCN, 0.5mg/0.1ml). All three putative chemoreceptor sites appeared to be chemosensory and stimulating each led to both respiratory and cardiovascular (shunt fraction) adjustments. Activation of aortic and pulmonary chemoreceptors, however, primarily increased pulmonary blood flow and reduced systemic flow, reducing shunt, while chemoreceptors located at the carotid bifurcation were primarily involved in ventilatory control. Multiple chemosensory sites, each with distinct reflex roles, may allow animals with incomplete separation of systemic and pulmonary circulations to regulate blood gases more efficiently. Funded by NSERC Canada.

11.9
CAN DEEP-SEA FISHES KEEP PACE WITH OCEAN ACIDIFICATION? CO_2 TOLERANCE IN DEEP AND SHALLOW-WATER ROCKFISHES FROM MONTEREY BAY, CA
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Commercially important deep-sea fishes have received increasing attention with respect to fisheries sustainability, but effects of ocean acidification on these species are unclear. Deep-sea thornyhead rockfish (Sebastolobus sp.) are the deepest dwelling scorpaenids in the eastern Pacific Ocean, inhabiting oxygen minimum zones between 300 and 1000 m. In addition to low temperatures characteristic of the deep sea, these zones have O2 tensions as low as 10 ?M and pH near 7.6, resulting in low metabolic rates of animals living therein. Conversely, black rockfish, Sebastes melanops, account for the majority of nearshore rockfish landings in the eastern Pacific, and must have the metabolic scope to allow survival during seasonal and even tidal shifts in temperature, O2, and pH. In both groups, we are investigating the effects of acute and chronic exposure to seawater CO2 concentrations representing current atmospheric levels of 385 ppm (pH 8.0) and predicted future levels from 750 ppm (pH 7.7) to extremes of 2000 (pH 7.3) and nearly 5000 ppm (pH 7.0) intended to highlight the physiological processes most susceptible to disturbance by climate change. Key measurements include O2 consumption, simultaneous whole body acid-base flux, and blood chemistry. Preliminary results suggest deep-sea rockfish compensate for acute CO2 exposure by increasing net acid efflux, in part via ammonia excretion, and have a non-linear metabolic response to seawater acidification.

11.10
ENERGETIC COSTS OF INCREASING THERMAL VARIABILITY: AN EXPLORATION OF THE MITIGATING EFFECTS OF PLASTICITY IN LEPIDOPTERA
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Thermal variability impacts organismal physiology due to the non-linear nature of response curves, and plasticity may mitigate these effects. For holometabolous insects, overwintering energy reserves must fuel metamorphosis and influence adult size and fitness. A reduction in temperature sensitivity, and thus metabolic rate, will reduce consumption of reserves, and is expected in populations exposed to energy-demanding (e.g. highly variable) environments. To test this hypothesis we used Erynnis propertius (Lepidoptera: Hesperiidae) from a stable (Vancouver Island [VI]) or variable thermal environment (Oregon [OR]), raised at VI or OR temperatures. We measured metabolic rate-temperature relationships and modeled overwinter energy use in both environments for the past 50 years using historical climate data. E. propertius show metabolic down-regulation in response to variable environments, whether experienced by themselves or their mothers. OR winters impose increased energy costs, but the metabolic down-regulation in OR groups compensates for the increased cost in average, but not highly variable, winters. This work provides an example of the capacity of some organisms to compensate for environmental variability and illustrates how physiological data can inform ecophysiological models. These models can help predict species responses to climate change, particularly where traits are plastic. Funded by a Discovery Grant from NSERC to BJS and a US Dept of Energy Grant to JJH.

11.11
LOCOMOTORY COST AND MYOGLOBIN DESATURATION IN DIVING EMPEROR PENGUINS
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Stroke frequency and pectoral muscle myoglobin (Mb) desaturation were measured with a near-infrared backpack spectrophotometer in emperor penguins diving under sea ice to investigate the locomotory costs of diving and the role of muscle O_2 depletion in the aerobic dive limit (ADL, dive duration associated with the onset of post-dive blood lactate accumulation). Mb desaturation profiles revealed two patterns: a monotonic decline (Type A) consistent with complete muscle ischemia, and a biphasic decline (Type B) consistent with blood supplementation of the muscle O_2 store early in the dive. Mb desaturation was nearly complete in Type A dives at 5.6-min duration, the emperor penguin ADL. Mb desaturation rates in these aerobic Type A dives were 13.3 ± 1.3 % min^{-1}, resulting in a muscle O_2 depletion rate of 7.63 ± 0.59 ml O_2 kg^{-1} muscle min^{-1} and an energetic cost of 1.02 ± 0.09 ml O_2 $stroke^{-1}$. Mean muscle O_2 depletion rates in Type B dives were slower due to presumed blood-to-muscle O_2 transfer early in the dive and resulted in near-complete muscle O_2 depletion over a range of dive durations (7 to 12 min). These results demonstrate the low cost of locomotion in diving emperor penguins, and support the concept that the ADL is triggered by depletion of the muscle O_2 store. Supported by NSF grant OPP 0538594, a NIH Marine Biotechnology Fellowship and a UC Regents Fellowship.

12.0: POSTERS IN GENOMICS AND PROTEOMICS, RESPONSES TO HYPOXIC, THERMAL AND OSMOTIC STRESS, NUTRITIONAL PHYSIOLOGY AND OCEAN ACIDIFICATION

12.1

FUNCTIONAL CHARACTERIZATION OF HYPOXIA-INDUCIBLE FACTORS FROM THE KILLIFISH, *FUNDULUS HETEROCLITUS*

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The hypoxia inducible factor (HIF) family of transcription factors is implicated in the regulation of genes whose expression underlies cellular, tissue, and organismal responses to low oxygen. HIF functions as a dimer composed of an oxygen-dependent alpha subunit and a constitutively expressed beta subunit (also known as the Aryl Hydrocarbon Receptor Nuclear Translocator, ARNT). In vertebrates, there are three forms of the alpha subunit, HIF1α, HIF2α, and HIF3α, which differ in their tissue distribution and target genes. In the present study, the capacity of HIF1α, HIF2α, and HIF3α of the killifish, Fundulus heteroclitus, to bind DNA and regulate transcription was evaluated in vitro and in cell culture. Each alpha subunit associates with ARNT to bind the concensus HIF-binding sequence in electrophoretic mobility shift assays. HIF1α and HIF3α, but not HIF2α, also bind a non-canonical HIF-binding sequence from the F heteroclitus lactate dehydrogenase B promoter. Similarly, over-expression of alpha subunits in mammalian Cos-7 cells drives reporter gene expression in a sequence specific fashion. These results suggest that in fish, as in mammals, target genes of HIF regulation differ for the different forms of HIF alpha subunits. Funded by the UNO Office of Research and Sponsored Programs.

12.2

CARDIORESPIRATORY FUNCTION AND HYPOXIA TOLERANCE IN ELASMOBRANCHS

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We investigated cardiorespiratory function during graded hypoxia exposure in two elasmobranchs with differing hypoxia tolerance to better understand the physiological correlates of low oxygen survival. The hypoxia-tolerant epaulette shark, Hemiscyllium ocellatum, had a lower critical oxygen tension (Pcrit) than the less tolerant shovelnose ray, Aptychotrema rostrata (25.0±1.8% vs. 35.4±2.0% air sat). The shovelnose ray did not survive at PO2 lower than 10% air sat whereas the epaulette shark tolerated near-anoxia. In both species heart rate (HR), cardiac output (Q), and estimated cardiac power output decreased in parallel with the decline in MO_2 that occurred below their respective P_{crit}. Stroke volume increased and dorsal aortic pressure (P_{DA}) decreased in both species and while these changes were not necessarily correlated with P_{crit}, they occurred at lower PO_2 in the shark. In both species HR, Q, and P_{DA} were similar above P_{crit}, but higher in the shark below P_{crit}. Maintenance of routine cardiac function to lower PO_2 appears to be a component of hypoxia tolerance in elasmobranchs. This may be explained in part by our finding of enhanced oxygen transport in the shark compared with the ray. (Funding: NSERC, CoB, CSZ).

12.3

LACTATE KINETICS IN RAINBOW TROUT DURING ACUTE HYPOXIA

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Aquatic hypoxia has become more widespread due to human impact on the environment. During hypoxia, fish must resort to anaerobic glycolysis to produce ATP, causing lactate to accumulate in blood and tissues. The rate of lactate appearance (R_a) and disposal (R_d) of rainbow trout (*Oncorhynchus mykiss*) were measured *in vivo* by continuous infusion of [U-^{14}C] lactate. We aimed at determining how R_a and R_d account for the increase in blood lactate during acute hypoxia, and what were the main tissues responsible for lactate production. The fish exposed to hypoxia (25% O_2, 90 min, 13ºC) showed a progressive increase in blood lactate from 1.0 to 8.9 mM, R_a lactate was strongly stimulated from a baseline value of 18.4 to 36.5 µmol kg^{-1} min^{-1}. Surprisingly, R_d lactate was also increased from 19.9 to 30.3 µmol kg^{-1} min^{-1}. In contrast, the control group (92% O_2, 90 min, 13ºC) maintained baseline blood lactate concentration of 0.8 mM. Its R_a lactate went from 18.2 to 13.1 µmol kg^{-1} min^{-1}, and R_d lactate from 19.0 to 12.8 µmol kg^{-1} min^{-1}. Hypoxia caused the highest lactate accumulation in white muscle from 3.6 to 19.2 µmol g^{-1}. This study shows that the hypoxia-induced increase in blood lactate is due to a stronger stimulation of R_a than R_d lactate. Blood lactate accumulation would actually be two-fold higher if R_d lactate was maintained at baseline levels rather than stimulated by hypoxia as demonstrated in these experiments. All procedures were approved by the Animal Care Committee of the University of Ottawa. Supported by – an NSERC discovery grant.

12.4

MECHANISMS UNDERLYING THE CRUCIAN CARP'S REMARKABLE FEAT TO MAINTAIN CARDIAC PUMPING DURING ANOXIA

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The crucian carp (Carassius carassius) seems unique among vertebrates in its remarkable ability to maintain cardiac performance during prolonged anoxia. We have shown that cardiac output (Q) remains unchanged for 5 days of anoxia. Measurement of ethanol excretion rate in anoxic carp in response to a manipulated Q, direct measurement of cardiac power output of normoxic and anoxic in situ perfused carp hearts, determination of carp blood plasma pH during anoxia, and examination of the combined effects of anoxia and graded acidosis on spontaneously contracting carp heart preparations suggests the remarkable feat is possible because of interlinked ultimate and proximate determinants. The ultimate basis is the evolution of two anoxia-survival strategies: the ability to avoid self-poisoning by lactate and H+ during anoxia by fermenting lactate to ethanol (the excretion of which likely necessitates a sustained Q); and a low routine level of cardiac work that can be sustained by anaerobic metabolism. At the proximate level, ethanol production avoids an extracellular acidosis that would otherwise impair cardiac performance. Thus, the maintained cardiac performance of anoxic crucian carp is permitted by a low routine cardiac ATP demand and the absence of severe extracellular acidosis, and is seemingly necessary for management of ethanol excretion.

12.5

CARDIORESPIRATORY AND METABOLIC RESPONSE TO ACUTE HYPOXIA IN ATLANTIC SALMON ALEVINS ACCLIMATED TO 4°C AND 8°C

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Cardiorespiratory and metabolic adaptation in fish to environmental hypoxia includes reflex-bradycardia, hyperventilation and metabolic suppression. An increase in ambient water temperature usually leads to higher hypoxia sensitivity. In Salmo salar alevins at hatching, O2 uptake is almost entirely cutaneous (Wells & Pinder, 1996. J. Exp Biol 199: 2725-2736) and it is assumed that O2 delivery to tissues is independent of cardiovascular convection because direct diffusion to metabolically active tissues suffices to supply the animal with O2. We investigated the cardio-respiratory and metabolic response by measuring heart rate (Fh), ventilatory frequency (Fb) and rate of oxygen consumption (VO2) to a stepwise challenge of hypoxia (21→15→10 kPa) using optometrical respirometry in newly hatched alevins incubated at 4°C and 8°C. Fish incubated and measured at 4°C displayed a reduced Fh (Q10=2.5) and Fb (Q10=6.5) compared to fish raised and measured at 8°C. In both groups, Fh and Fb remained constant at all levels of O2 whereas VO2 decreased with hypoxia, the decrease being greater the more severe the hypoxia. The temperature dependent reduction in VO2 (Q10=4.3) was proportionally the same at each level of O2. These results indicate that Fh and Fb are not coupled to VO2 at this stage of development and therefore cardiovascular and ventilatory convection may not be critical for oxygen supply as indicated by non sensitivity to hypoxia. (ARC DP0987626; CSIRO ICT Schol. to EP).

12.6

THE ROLE OF AMP-ACTIVATED PROTEIN KINASE IN COORDINATING METABOLIC RATE SUPPRESSION IN HEPATOCYTES FROM THE COMMON GOLDFISH, *CARASSIUS AURATUS*

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Hypoxia-induced metabolic rate suppression (MRS) is thought to be an important adaptation enhancing survival in low oxygen environments. During hypoxia exposure, hypoxia-tolerant cells reduce energy expenditure by reversibly down-regulating ATP-consuming pathways to match the decreased energy production of oxygen-independent pathways. AMP-activated protein kinase (AMPK) is activated by a decrease in cellular energy status and our lab has previously shown that activation of AMPK is associated with phosphorylation of its downstream target eukaryotic elongation factor-2 (eEF-2) and a reduction in protein synthesis in liver of goldfish exposed to hypoxia. To test the hypothesis that AMPK initiates MRS, we pharmacologically manipulated AMPK activity in goldfish hepatocytes and examined the subsequent effects on eEF-2, protein synthesis, and cellular metabolic rate (measured as MO_2). Pharmacological activation of AMPK (with AICAR and A769662) under normoxia caused an increase in AMPK activity concomitant with a decrease in MO_2. This effect is enhanced under physiologically relevant O_2 levels. This reduction in MO_2 can be attributed to a decrease in protein synthesis caused by an increase in phosphorylated eEF-2. Overall, our study provides evidence that AMPK plays an important role in coordinating metabolic suppression in goldfish hepatocytes. (Funding: NSERC).

12.7

SEASONAL PLASTICITY OF GABAERGIC SYNAPTIC TRANSMISSION IN *LYMNAEA STAGNALIS*

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of γ-aminobutyric acid (GABA) on neuronal activity, we conducted intracellular recordings from isolated central ring ganglia of the hypoxia- tolerant pond snail *Lymnaea stagnalis*. GABA decreased neuronal activity by hyperpolarizing the resting membrane potential (V_m) and inhibiting electrical firing, while bicuculline methiodide, an antagonist of $GABA_A$ receptors, blocked the GABA-induced inhibition and excited "silent" cells. To determine the contributions of the K/Cl co-transporter KCC2 to setting E_{GABA} below action potential (AP) threshold and rendering GABA inhibitory, we used a pharmacological inhibitor of KCC2, furosemide. Similar to bicuculline perfusion, this manipulation blocked the GABA-induced inhibition. Contrary to the inhibitory response observed in "summer" animals, in the majority of neurons obtained during the winter months (Dec-Mar) GABA excited "silent" cells and increased neuronal activity by depolarizing V_m and increasing AP frequency, while bicuculline blocked this response. $GABA_A$ receptor-mediated excitation failed to occur when intracellular Cl^- homeostasis was perturbed with the Na/K/2Cl (NKCC1) antagonist bumetanide. These data indicate that environmental stimuli regulate the activity of transmembrane co-transporters, leading to changes in anionic gradients that determine the polarity of GABAergic transmission.

12.8

TRANSCRIPTOMIC RESPONSES OF PORCELAIN CRABS TO THERMAL ACCLIMATION

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Our lab is interested in understanding how porcelain crabs from the genus Petrolisthes respond to variation in their thermal microhabitat. Within species, porcelain crabs live in specific vertical zones from subtidal to uppertidal zone habitats1 and are thus experiencing a wide range of temperature extremes as well as temperature fluctuations yet these species are well adapted to their environment2 making it a species of choice for ecophysiology studies. Our lab has developed EST libraries and a Porcelain Crab Array Database for Petrolisthes cinctipes3 that provide us with means to undertake functional genomics and ask how the crabs transcriptome reponds to temperature changes. In this experiment, crabs were acclimated to 14°C for 28 days followed by acclimation to control, low (6°C) and high temperatures (22°C), reflecting the range of average temperatures these crabs experience in their natural habitat4. To assess how crabs physiology is altered in responses to these changes, RNA from heart tissue samples taken at several points during the experiment were used as template for cDNA synthesis, labeling and used for microarray hybridization and analysis. This experiment will help us to understand how cardiac tissues are remodeled to perform optimally at different temperatures. NSF 0533920. 1Mol. Phylogenetics & Evo. 19: 236-245. 2Int. Congress Series 1275: 267-274. 3PLoS ONE 5: e9327. 4Mol. Ecology 18: 4206-4226.

12.9

TEMPERATURE INDUCED CHANGES IN THE EXPRESSION OF THE ENZYMES INVOLVED IN MEMBRANE RESTRUCTURING.

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With the current uncertainty over climate change, it is important to understand how individuals respond to changing temperature. Cell membranes are crucial contributors to an individual's ability to acclimate to a new temperature. Many animals routinely survive changing environmental temperatures because they adjust the physical characteristics of their membranes. The phospholipids of their membranes are restructured through specific enzymatic reactions. Our goal is to use molecular biology techniques to examine the temporal patterns of the expression of the genes that code for these phospholipid restructuring enzymes. Biochemical evidence suggests there may be a distinct temporal pattern in these expressions. Some appear to be switched on early, while others are activated later in the acclimation process to augment the earlier changes. We have designed primers for many of these enzymes and have tested them using cDNA synthesized from mRNA extracted from Coho salmon (Oncorhynchus kisutch) embryo cells. In a series of experiments, cells were maintained at 22°C then transferred to 10°C for periods from 2 hours to 27 days. We have found that messages for some enzymes of membrane restructuring are constitutively produced, while others are activated after different times of cold exposure. There are also differences in the expression of enzymes of the de novo synthetic pathway (Kennedy pathway) and the phospholipid in situ restructuring pathways (Land's cycle).

12.10
DEVELOPMENT AND USE OF AN *IN VITRO* TOOL FOR ASSESSING MECHANISMS UNDERLYING THE OSMOTIC STRESS RESPONSE IN A *EURYHALINE TELEOST*
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The osmotic stress response in fish is driven by a suite of complex physiological and molecular mechanisms. Myo-inositol phosphate synthase (MIPS) has recently been identified as a key player involved in the osmotic stress response of euryhaline tilapia (Oreochromis mossambicus). MIPS is an enzyme responsible for the synthesis of myo-inositol, a compatible osmolyte. In order to conduct in-depth mechanistic studies on MIPS hyperosmotic regulation and function, a tilapia endothelial cell line derived from bulbus arteriosus was obtained in addition to a recently immortalized tilapia fibroblast cell line we successfully derived from brain by subculturing tissue explants. Surprisingly, we found that the acute hyperosmotic tolerance of the endothelial cell line is markedly higher than that of any other vertebrate cell lines. To validate this in vitro system, MIPS mRNA expression was quantified using quantitative real-time PCR following a two-day acute hyperosmotic exposure at 23ppt (700mOsm). An in vivo study was also conducted by an acute two-day acclimation of tilapia from 0ppt to 35ppt (1050mOsm) saltwater resulting in a ~30 fold increase in MIPS mRNA expression detected in gill tissue. A comparable expressional response in vitro will validate the use of this cell line system as a valuable tool for further characterization and manipulation of MIPS in addition to other molecular mechanisms involved in the osmotic stress response. Funded by NSF grant IOS-0542755.

12.11
EFFECTS OF ACUTE TEMPERATURE AND SALINITY STRESSES ON THE TRANSCRIPTOMES OF INVASIVE AND NATIVE MUSSEL SPECIES (*GENUS MYTILUS*)
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Invasive species are prevalent in marine ecosystems, and the future success of invasives is likely to be influenced by global climate change as it will cause the expansion or contraction of available habitat and alter the relative competitive abilities of invasive and native species. Using an oligonucleotide microarray, we consider the effects of two environmental stresses, temperature and salinity, on the gene expression (i.e. transcriptome) of an invasive mussel species, the warm-adapted Mediterranean mussel Mytilus galloprovincialis, and a noninvasive congener, the more cold-adapted Mytilus trossulus, under common-garden conditions. We find that the transcriptomic responses to heat-stress are remarkably similar in these two species and that the differential transcriptional control of relatively few genes seems to underlie their well-documented differences in thermotolerance. Transcriptional responses to salinity-stress are also discussed.

12.12
GAS EXCHANGE VARIATION IN THE ALFALFA LEAFCUTTER BEE, *MEGACHILE ROTUNDATA*, IN RESPONSE TO FLUCTUATING THERMAL REGIMES
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Survival of developing, alfalfa leafcutter bees exposed to low temperatures during the interruption of spring incubation can be increased by exposing pupae to short, high temperature pulses. We hypothesized that these fluctuating thermal regimes (FTR) also affect metabolism and gas exchange in the pupae. To test the hypothesis, we used flow-through respirometry to measure CO_2 emission during the exposure to FTR, as an index of metabolism. All pupae were exposed to beginning and ending temperatures of 6°C, while high temperatures and exposure time varied (15°C for 2 h, 20°C for 1 h, 20°C for 2 h, and 25°C for 2 h). Forty-five percent of pupae in 20°C for 2 h and 25% of pupae in 25°C for 2 h exhibited cyclic gas exchange during the elevated temperature. Total number of CO_2 peaks and peak frequency were higher in 25°C pupae compared to 20°C pupae. The number of days the pupae spent in the FTR had no significant effect on CO_2 emission. CO_2 emission for animals exposed to 25°C for 2 h was significantly higher than those from all other FTR. In addition, CO_2 emission from pupae in 20°C for 2 h was significantly higher than that of pupae exposed to 15 °C. Ending CO_2 emission at 6°C for pupae in all FTRs was higher than beginning baseline CO_2 emission at 6°C. While these data do not explain the increased survival of pupae in FTR, the possible relationship between the two parameters is intriguing.

12.13
TRANSCRIPTOMICS OF ENVIRONMENTAL ADAPTATION IN WILD MIGRATING ADULT PACIFIC SOCKEYE SALMON (*ONCORHYNCHUS NERKA*)
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Successful migration of adult salmon from ocean feeding grounds to natal freshwater streams requires adapting to a suite of new environmental conditions. Global climate change is thought to intensify environmental factors and is an often cited cause of recent increases in salmon migration mortality rates. In this study, a salmonid microarray was used to characterize environmentally-regulated shifts in gene expression in gill and liver tissues of wild migrating adult Pacific sockeye salmon (Oncorhynchus nerka) between ocean and river habitats. Expression profiles indicate the transcriptome of wild migrating salmon is strongly influenced by new environmental conditions encountered within the river, including genes associated with thermal stress (molecular chaperones), osmotic stress (hormones and ion-transporters), oxygen flux (hemoglobin), detoxification (cytochrome p450s), and pathogen infection (immunoglobulins). Biopsy sampling of gill tissue and microarray-based profiling was combined with radiotelemetry and genetic stock identification to quantify the influence of gene expression on survival. 183 features were significantly differentially expressed between fish that reached spawning grounds and in-river mortalities. Only one of the five genes most strongly correlated to this signature significantly effected survival, CRSP1, part of a complex involved in regulating the transcription of nearly all RNA polymerase II-dependent genes. Funded by NSERC Canada and Genome BC.

12.14
HEAT INDUCTION OF C/EBP-DELTA IN TWO FISHES: NEW INSIGHTS INTO THE CELLULAR STRESS RESPONSE
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The cellular stress response (CSR) is an important mechanism by which organisms respond to environmental perturbation. It involves the coordinated regulation of hundreds of genes, many of which are highly conserved across taxa. Anthropogenic climate change is stimulating researchers to identify these changes in gene expression, in order to better predict the physiological capacity of organisms in the coming centuries. To this end, recent cDNA microarray studies have done much to elucidate new facets of the CSR. Aquatic ectotherms differentially regulate the expression of certain CCAAT/enhancer-binding proteins (C/EBPs) in response to sublethal heat and osmotic stress. Because C/EBPs are implicated in inflammation, cell cycle arrest, apoptosis, and cellular differentiation, the fact that they are stress responsive offers important clues about cellular behavior during exposure to environmental stress. Here we demonstrate the tissue-specific heat induction of C/EBP-delta in two fishes: the extremely cold-adapted Antarctic Notothenioid Trematomus bernacchii and the zebrafish Danio rerio.

12.15
QUALITATIVE CHARACTERIZATION OF A NOVEL NHE2C PROTEIN IN THE MARINE LONGHORN SCULPIN (*MYOXECEPHALUS OCTODECEMSPINOSUS*) AFTER EXPOSURE TO ADVERSE ENVIRONMENTAL CONDITIONS EMERGING IN OCEAN WATERS
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Interest in maintaining marine biodiversity creates awareness of ocean acidity and its impact on fish physiology. We characterized the pH and associated water quality parameters, including temperature, salinity, dissolved inorganic carbon (DIC), and total alkalinity (TA), of a 1.25 mile transect across Frenchman's Bay, ME. The coordinates stretched from the Mount Dessert Island Biological Laboratory (N44°26'02.3'', W068°17'25.5"), through open pelagic waters (N44°26'21.8", W068°17'21.8") to Lamoine Beach (N44°27'00.4", W068°17'10.2"). DIC, TA, and pH were measured in replicates (3 or 4). A Mann-Whitney-U test showed a significant difference between pH at depth and at surface ($p < 0.0001$), with a mean pH of 7.75 ± 0.08 and 7.91 ± 0.41, respectively. This difference in pH coincided with a significant difference in TA at depth, 2086 ± 32.5 µmol/kg, and at surface, 2035 ± 79.3 µmol/kg ($p < 0.0001$). Simulated bay values will be linked to the physiological expression of a novel gill Na+/H+ transporter (NHE2c) which we have recently cloned from sculpin gill tissue. Sculpin specific antibodies against NHE2c have been developed in order to determine the cellular location of this Na+/H+ ortholog and characterize the subcellular distribution in comparison to NHE2b and NHE3. We hypothesize that NHE2c plays a role in acid-base adjustments as the animal encounters decreasing ambient pH and internal acidosis.

12.16
STRESS RESISTANCE AND RESILIENCE IN THE CORAL ACROPORA HYACINTHUS: GENE EXPRESSION ANALYSIS BASED ON HIGH-THROUGHPUT SEQUENCING
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Anthropogenic disturbances and the increased isolation of coral reef habitats in the last century have seriously impacted reef ecosystems, weakening their resilience and increasing their susceptibility to global climate change. However, when large-scale coral bleaching and mortality occur during warming events there is some survival of scattered colonies and localized communities. This resilience in the face of high temperatures may relate to extrinsic factors that reduce the severity of the thermal stresses or to the intrinsic ability of corals to acclimatize or adapt to the stressful conditions. We investigated gene expression profiles from Acropora hyacinthus colonies found in a dynamically fluctuating back reef on Ofu Island in American Samoa. Previous research has identified elevated stress tolerances in corals from the section of the back reef with extreme (up to 6 °C) daily temperature fluctuations versus corals from the more moderate back reef areas (2 to 3 °C). A. hyacinthus from the more dynamic areas also exclusively harbor clade D Symbiodinium, a symbiont type known to confer greater thermotolerance. This study aims to investigate the differential contributions of environmental conditioning and symbiont genotype to the overall stress gene expression of A. hyacinthus. These results will be used to identify novel candidate biomarkers for assessment of coral stress resistance, tolerance, and resilience to environmental variability.

12.17
THE IMPACT OF TEMPERATURE STRESS ON HEMOCYTES OF MYTILUS CALIFORNIANUS: DNA DAMAGE AND SIGNALING PATHWAYS
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We investigated the effects of acute heat- and cold stress on double- and single-stand DNA breakage, lysosome membrane stability, caspase-3 activity and signaling mechanisms involved in the protection and apoptosis of Mytilus californianus hemocytes. Both hyperthermia (28 ºC, 32 ºC) and hypothermia (2 ºC, 6 ºC) induced significant double- and single-strand breaks in DNA. The types and extent of DNA damage and levels of caspase-3 activity were temperature- and time-dependent. Numbers of lytic and necrotic hemocytes significantly increased and lysosome membrane stability of hemocytes decreased significantly under heat stress. Furthermore, western blot analyses of hemocyte extracts with antibodies for proteins associated with cell signaling and stress responses [including members of the phospho-specific Mitogen Activated Protein Kinase (MAPK) family (c-JUN NH(2)-terminal kinase (JNK), Stress-activated protein kinase (SAPK) and p38 MAPK), tumor suppressor factors (p53 and p21), DNA base excision repair enzyme (Apurinic/apyrimidinic (AP) endonuclease), and apoptosis executor caspase-3] indicated that high- and low temperature stress induced a time dependent activation of different classes of stress-related kinases, proteins induced by DNA damage and effectors of apoptosis. Our results indicate that DNA damage, stress signal transduction and apoptosis play critical roles in responses to temperature stress by mussel hemocytes.

12.18

INCREASED ANTIOXIDANT ACTIVITY IN THE CARIBBEAN FRUIT FLY, *ANASTREPHA SUSPENSA*, SERVES A PROTECTIVE ROLE DURING IRRADIATION.

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The Caribbean fruit fly, Anastrepha suspensa, is subjected to low doses of gamma radiation as part of area-wide pest control by the sterile insect technique (SIT). Even though SIT can be very effective, sterilized males suffer performance costs compared to non-irradiated counterparts. We hypothesized that the use of low-oxygen pre-treatments on these flies might trigger their antioxidant defenses in the anticipation of normal oxygen reperfusion; thus allowing the flies to better cope with protein, membrane and DNA/RNA damage due to oxygen radical attack from the irradiation. We assayed total antioxidant capacity of the flies, as well as the activities of several antioxidants enzymes (superoxide dismutase-SOD, catalase, and glutathione peroxidase-GPx). We also monitored protein, membrane and DNA/RNA damage in relation to organism performance parameters such as adult emergence, flight ability and mating. A 1hr anoxia treatment before irradiation led to a higher total antioxidant capacity that can be explained by an increased in SOD and GPx activities. Anoxia also led to increased organismal performance (higher emergence, flight ability and mating rates). We present strong evidence that the damage the flies sustain during radiation can be reduced by placing pupae in a low to no oxygen environment an hour prior to radiation treatment.

12.19

APOPTOSIS-RELATED GENES RESPOND UNIQUELY TO UV RADIATION AND COLD STRESS IN THE FLY *SARCOPHAGA CRASSIPALPIS*

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Apoptosis, a specific type of programmed cell death, maintains tissue homeostasis by removing sub-lethally damaged cells and occurs in response to stressors such as ultraviolet radiation (UV) and cold shock (CS). mRNA expression of p53, buffy, BOK, decay, eiger, wengen, HtrA2, hsp70, and 14-3-3, genes which encode proteins with known roles in stress-induced apoptosis pathways, was measured in Sarcophaga crassipalpis pharate adults 2 and 4 h post exposure to UV (10 mJ/cm2 or CS (-8° C). In addition, transcript abundance of these genes was measured in flies pre-exposed to 0°C for 2 h prior to exposing them to -8°C (RCH+CS) and allowed to recover for 2 and 4h. Pre-exposure to 0°C, also known as rapid cold hardening (RCH), can improve cold tolerance and protect against cold-shock injury. Quantitative PCR (qPCR) shows the mRNA expression level response of apoptosis genes depends on the type of stress. Notably, expression of p53, bok, and buffy significantly change in response to UV but are unaffected by either CS or RCH+CS. Conversely, htra2 is reduced by 70% 4 h after CS but is unaffected by UV. Surprisingly, hsp70 expression is reduced by 30% in UV exposed flies, but shows a 5-fold increase 4h after exposure to RCH+CS and a 50-fold increase 2h after CS alone. Clearly specific types of stress invoke unique transcript-level responses in apoptosis related genes. NSF IOS-0840772.

12.20

USING STABLE ISOTOPES TO TRACK EXOGENOUS NUTRIENT ALLOCATION AND ROUTING IN POSTPRANDIAL HOUSE SPARROWS

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Since animals are heterotrophic, their diets must provide all of the metabolic fuel required to carry out essential life processes. Most animals do not eat continuously and must therefore be able to store recently ingested nutrients in a way that they can be mobilized to meet future energy demands. We examined the economics of nutrient allocation in small birds since, in addition to fuel storage limitations imposed by flapping flight, they have very high mass specific metabolic requirements. We enriched the diets of house sparrows, Passer domesticus, with one of seven 13C-labeled metabolic tracers representing the three major classes of macronutrients; carbohydrates, amino acids and fatty acids. We hypothesized that nutrients are not homogenously allocated amongst tissues; our goal being to identify instances of preferential nutrient allocation to specific tissues. While dosed birds became postabsorptive, we quantified the isotopic enrichment of the lean and lipid fractions of organs and tissues, and compared the δ13C of the different tissue fractions with null models that we developed to describe nutrient distribution. We found that different molecules belonging to the same class of macronutrients exhibit systematic patterns of allocation. The approach developed here for identifying preferential nutrient allocation can be applied to virtually any animal to test specific predictions about how physiological and environmental factors influence its life history.

12.21

ONTOGENY AND NUTRITIONAL STATUS INFLUENCE OXIDATIVE KINETICS OF EXOGENOUS NUTRIENTS AND WHOLE-ANIMAL BIOENERGETICS IN ZEBRA FINCHES

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Rapidly growing animals or those that are recovering from nutritional stress may use exogenous nutrients differently than well fed adults. To explore this concept, we compared body mass (m_b), organ mass, core body temperature (T_b), metabolic rate (MR), and rates of exogenous nutrient oxidation among fledgling, fasted adult, and refed adult zebra finches. We found that fasted birds had lower T_b, relative liver and intestine masses, MR, and respiratory exchange ratios (RER) than fed adults. Moreover, breath testing revealed that adults recovering from nutritional stress had much lower rates of exogenous amino acid, carbohydrate, and fatty acid oxidation than fed birds, suggesting that previously fasted birds were using recently assimilated nutrients to replenish exhausted fuel stores. The m_b and T_b of rapidly growing fledglings was lower than fed adults, but their relative liver and intestine masses were the same as their fed adult counterparts; yet they were able to oxidize exogenous nutrients as fast as adults. We suggest that the high rates of exogenous nutrient oxidation in growing fledglings are required to compensate for the relatively low conversion efficiency to feathers and other lean growing tissue in young birds. In general these results confirm that the patterns of exogenous nutrient use by growing fledglings and adult birds recovering from nutritional stress are quite different, thereby underscoring how differences in nutrient use can be obscured when mass alone is used to predict animal nutrition and bioenergetics.

12.22

ISOTOPE DILUTION AND BODY COMPOSITION: UPDATING A TOOL FOR NUTRITIONAL ECOLOGY

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The assessment of body composition in living animals is a key to many physiological and ecophysiological questions, and dynamic change of body energy can reveal temporal shifts in environmental quality. Isotope dilution (ID) with labeled water has been used extensively in wild mammal populations, but the validity of inferences depends critically on (a) analytical methodology and (b) choice of model for conversion of ID data into estimates of body composition. We examined isotopic data (^2H_2O) for pups of Weddell seal, harp seal and hooded seal in relation to body composition by direct carcass analysis. Several factors contributed to a discrepancy between ID space and total body water content: Gut water accounted for 1.0-1.8% and 0.2-1.0% of body mass in suckling and weaned pups, respectively. Losses of 2-5% of body water occurred during dissection, and drying procedure affected measured carcass water by 1-2%. If these factors are controlled, isotopic exchange between water and organic molecules caused no more than 2-3% overestimation of body water. We evaluated different models for the prediction of body composition from ID space in seals and other mammals. We conclude that these models produce divergent results, particularly when applied to young seals or animals undergoing rapid changes in body composition, and that any model so applied should take into account the effect of developmental stage on body composition. This work was supported by NSF grant 0538592.

12.23

THE EFFECT OF RICE BRAN ARABINOXYLAN COMPOUND ON GLUCOSE METABOLISM IN SKELETAL MUSCLE

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Purpose: Obesity and type 2 diabetes patients impaired activation of glucose metabolism-related signaling pathway in muscles. Recently, rice Bran Arabinoxylan Compound (RBAC; Daiwa Pharmaceutical Co., Ltd.) is beneficial for the treatment of hyperglycemia patients, RBAC is an enzymatically modified arabinoxylan extracted from rice bran, has a potential to act as a biological response modifier. However, it is still unclear that the effect of RBAC on the glucose metabolism-related signaling pathway in skeletal muscle. Methods: Male Wistar rats (8 weeks of age) were assigned to one of the following treatment groups: (1) RBACEx, a RBAC ingested exercised group (n=7); (2) RBAC-Sed, a RBAC ingested sedentary group (n=6); (3) DW-Ex, adistilled water ingested exercised group (n=7); (4) DW-Sed, a distilled water ingested sedentary group (n=6). Rats received RBAC (150 mg/kg body weight) or distilled water orally for two weeks. Their gastrocnemius muscles were taken for the measurement of Akt and PKC ζ/λ phosphorylations, and glucose transporter-4 (GLUT-4) translocation. Results: Akt and PKC ζ/λ phosphorylations and GLUT-4 translocation was significantly increased in DW-Ex and RBACEx, also these were significantly increased in RBAC-Sed group. Conclusion: RBAC induced significant increase of muscle glucose metabolism in normal rats. It might be beneficial treatment for impaired muscle glucose metabolism patients such as obesity and type 2 diabetes.

12.24

TORPOR FLEXIBILITY IN RESPONSE TO ENVIRONMENTAL CONSTRAINTS: FOOD SHORTAGES AND IMMUNE CHALLENGE

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As ecosystems undergo global changes, physiological flexibility is likely to be an important adaptive response to increased climate instability. Extreme weather fluctuations impose new energetical constraints such as food shortage and disease outbreaks. We tested with a food restriction experiment, how captive Grey Mouse Lemurs (Microcebus murinus) modify torpor-based energy savings in response to contrasted regimes of food availability and to an immune challenge. Twelve individuals were fed ad libitum and 12 were exposed to a 40% calorie restriction during 11 weeks. Afterwards, six of each group were exposed to a 2-week 80% calorie restriction. Two weeks later, six individuals per group received a challenge that activates both innate and acquired immunity. Daily torpor was increased by calorie restriction, with an acclimation effect. All immune-challenged individuals underwent fever and skipped diurnal torpor. On the following day, normothermia was maintained by individuals fed ad libitum, whereas food-restricted individuals returned to deep torpor. During 'bad' years and extreme events, individuals could increase the amount of energy savings by increasing the length and the depth of torpor. When exposed to a pathogen, the acute response of undernourished individuals would be energetically constrained. The flexible control of energy expenditure conferred by heterothermy is likely to facilitate the plastic response of heterothermic species to more frequent climatic hazards.

12.25

PATTERNS OF GUT SIZE AND DIGESTIVE ENZYME ACTIVITY WITH CHRONIC FOOD RESTRICTION IN NESTLING HOUSE SPARROWS.

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Food restriction can occur during chick development, though response in altricial birds has been little studied. Food-restricted adults typically have reduced intestine and liver masses and total enzymatic capacity, though mass-specific enzyme activities may not be altered. We hypothesized that developing nestling birds maintain masses and biochemical capacities of assimilation organs constant despite food restriction, to support demands of growth and development. House sparrow (Passer domesticus) nestlings were raised in the laboratory from day 3 to 25 post-hatch and fed age-specific meal sizes (control group) or meals 75% of age-specific size (restricted group). In 2009, we took body and organ masses and stored intestines for digestive enzyme assays at day 25. In 2010 we collected data at various time-points prior to day 25 to assess patterns throughout chronic restriction. Intestine and liver masses were significantly lower in restricted birds (by 17% and 18%) at day 25 compared to controls. Summed maltase activity of whole intestine of 25-day-old restricted birds was significantly reduced compared to controls, though there was little difference between groups in mass-specific activity. Thus, young fledged birds that completed growth under food restriction showed the responses of adult food-restricted birds, but earlier time

points during the growth phase will be evaluated for differences from that pattern. Approval: UW ACUC. Support: NSF IOS0615678 to WHK, NSF GRFP to TLK.

12.26
DIETARY POLYUNSATURATED FATTY ACIDS, ENDURANCE FLIGHT, AND MITOCHONDRIAL FUNCTION IN EUROPEAN STARLINGS (*STURNUS VULGARIS*)
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Dietary polyunsaturated fatty acids (PUFA) affect both peak metabolic rate and endurance flight efficiency in songbirds. Yet, few studies have investigated mechanisms by which dietary PUFA may alter metabolism during exercise. Dietary PUFA have been shown to alter various aspects of mitochondrial function that could be related to exercise performance. Therefore, this study investigated the effects of exercise and dietary fats on mitochondrial function in European starlings. Birds were fed isocaloric diets varying in levels of particular unsaturated fatty acids and vitamin E for a minimum of 6 weeks and were then flown in a wind tunnel. Mitochondrial substrate preferences, proton leak, phosphorylation kinetics, and reactive oxygen species (ROS) production rates were measured in control birds, after 15 days of training (~12h total flight time), and upon immediate completion of an endurance flight that followed this training. State 3 succinate oxidation under rotenone was significantly elevated as a result of training, and high PUFA diet. Mitochondrial oxidation rates were generally depressed immediately after flight. Proton leak, phosphorylation kinetics and ROS production rates will also be presented. These results are consistent with the hypothesis that dietary PUFA affects key aspects of mitochondrial function that in turn affects flight performance of birds. Funded by NSERC, NSF, CFI, AMNH Chapman award, UWO Locke award.

12.27
INCREASED LIFE SPAN FROM OVARIECTOMY MAY BE DUE TO DIETARY RESTRICTION IN GRASSHOPPERS
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Reduced diet can produce longevity, but usually results in reduced fecundity. Directly reducing reproduction can also extend life span. Whether reduced reproduction secondarily decreases feeding rate is unclear, as ingestion is typically not measured in these studies. We used a fully factorial design to test effects of dietary restriction (DR, 70% of ad libitum feeding) and ovariectomy (ovx) on life span, and measured ingestion rate and hemolymph proteins. Treatments were: sham-FD (full diet); sham-DR; ovx-FD; and ovx-DR. To date, 71% have died. Pair-wise comparisons of Kaplan-Meier survival estimates were significant for all logical comparisons (i.e., sham-FD<sham-DR; sham-FD<ovx-FD; ovx-FD<ovx-DR). A more conservative comparison by two-way ANOVA showed that life span has been increased 13% by DR ($P=0.02$) but only 4% by ovx ($P=0.41$). Feeding was significantly reduced by ovx; from 100 to 225 days ovx-FD females consumed ~50% less than sham-FD females. A preliminary correlation implies that feeding rate explains 85% of the variation in life span. The survivorship data are consistent with the notion that reduced reproduction extends life span mostly by causing DR. At the same time, early in adulthood ovx increased levels of the egg yolk-precursor protein (vitellogenin) 10-fold ($P < 0.01$) but did not alter total anti-oxidant activity of the hemolymph ($P > 0.25$). These hemolymph data hint that increased vitellogenin may not function to increase anti-oxidant activity.

12.28
MAXIMAL METABOLIC RATE IN BIRDS TRACKS DAILY ENERGY EXPENDITURE.
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To investigate the role of phenotypic plasticity in metabolic physiology of birds we measured maximal and daily mean metabolic rates across two environmental gradients. First, we compared temperate and tropic species of robins (American and Clay-colored) as recent life history theory proposes that birds that are resident in the tropics have a slower “pace of life” as compared to temperate residents, reflected in lower daily activity patterns and lower capacity to use energy than temperate birds. Second, we compared seasonal challenges to metabolic demand in birds that are resident at higher latitudes (Northern cardinal) while over-wintering or during breeding season. We found that while American robins have higher metabolic capacity than their tropical relative, this is matched by a similarly higher daily energy expenditure. Similarly, while the metabolic capacity of Northern cardinals is higher in winter, there is a similar increase in daily energy expenditure. Thus it appears that daily energy use rates may be a compelling determinant of metabolic capacity in wild birds.

12.29
ESTIMATING THE POTENTIAL FOR ADAPTATION TO OCEAN ACIDIFICATION IN TWO COASTAL SPECIES
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Ocean acidification has been identified as one of the most wide-spread and biologically significant of anthropogenic effects on life in the oceans, yet few data exist on the ability of species to adapt to this environmental change. The rate of evolutionary response to elevated oceanic pCO_2 depends on both the amount of phenotypic variation on which selection can act, and the proportion of that phenotypic variation that is genetically heritable. Using sibling comparisons, we estimated the heritability of growth responses to ocean acidification in two ecologically important species in the Northeast Pacific, the sea urchin *Strongylocentrotus franciscanus* and the mussel *Mytilus trossulus*. In both species, we found the heritability of early larval growth rates at elevated CO_2 (1000 ppm) to be low ($h^2<0.1$), but maternal effects to be high ($m^2=0.4$-0.9). However, the amount of phenotypic variation relative to the effect of CO_2 was 1-2 orders of magnitude greater in *S. franciscanus* than it was in *M. trossulus*. We used these estimates and the observed effects of CO_2 on larval growth to simulate responses to selection under future ocean acidification. Larval growth rates are predicted to adapt more rapidly in *S. franciscanus*, and could rebound to growth rates under present day pCO_2 conditions within 3 to 418 simulated years, while those of *M. trossulus* are predicted to require 35 to 10000 years. These results suggest that, provided that heritability is non-zero, the scale of phenotypic variation relative to the strength of selection is a key parameter needed for estimating species’ evolutionary potentials under climate change.

12.30
EFFECTS OF OCEAN ACIDIFICATION ON DEVELOPMENT AND GROWTH OF PORCELAIN CRAB LARVAE
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The absorption of atmospheric CO2 into the ocean causes a decrease in pH. The energetic cost of responding to reduced pH during an already costly period such as larval development may be manifested in delayed growth. Very little is known about the impacts of ocean acidification in decapod crustaceans, so we have investigated the effects of increased pCO2 on porcelain crab larval growth. Newly hatched larvae of Petrolisthes cinctipes and Petrolisthes manimaculis were raised in two different conditions of pCO2; ambient pCO2 (~400ppm) and elevated pCO2 (~800ppm). The carbonate chemistry of artificial seawater was manipulated by the addition of NaHCO3 and HCl to create the high pCO2 condition based on calculations from the CO2Sys program. Alkalinity, DIC, pH, salinity and temperature were monitored regularly during the experiment and water was changed every 2 days. Carapace length was measured in specimens preserved at 5, 11, and 12 days post-hatch. Preliminary results show that larvae of P.cinctipes raised under elevated pCO2 had reduced carapace length compared to larvae raised in ambient conditions. Under elevated CO2 P. manimaculis showed no growth, yet there was a trend of increasing carapace length under ambient conditions. Our results suggest that elevated pCO2 suppresses growth of porcelain crab larvae and therefore they may spend more time as planktonic organisms leaving them more susceptible to predation and potentially reducing settlement. NSF IOS0920050.

12.31
EFFECT OF OCEAN ACIDIFICATION ON THE METABOLISM OF PORCELAIN CRAB LARVAE
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An increase in anthropogenic emission of CO2 into the atmosphere since the industrial revolution is predicted to decrease surface ocean pH by up to 0.4 units by 2100. Very little is known regarding the physiological effects of ocean acidification on crustaceans, including that of the porcelain crab, Petrolisthes cinctipes. As adult benthic intertidal organisms, they experience diurnal and seasonal fluctuations in pH. However, larval stages live in relatively stable open ocean waters and may be particularly vulnerable to decreases in pH. Here we examine the effect of pH on larval metabolic rates at two developmental stages. Newly hatched P. cinctipes were divided into a current pH condition (~8.2) and a low pH condition (~7.9). The carbonate chemistry and acidity of artificial SW was adjusted by the addition of NaHCO3 and HCl, respectively, calculated using the program CO2Sys. Parameters of the water chemistry, including alkalinity, DIC, temperature and pH, were monitored throughout the experiment. Rates of oxygen consumption were measured after 5 and 12 days using an optode 96-well microplate approach. Results indicate that larvae raised in lower pH tend to have lower metabolic rates. Reduced O2 perfusion to tissues may negatively impact survival by lowering ATP production and minimizing energy available for growth, recruitment and reproduction. NSF grant IOS-0920050.

12.32
PHYSIOLOGICAL EFFECTS OF ELEVATED TEMPERATURE AND OCEAN ACIDIFICATION ON THE PURPLE SEA URCHIN, *STRONGYLOCENTROTUS PURPURATUS*
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Global climate change (GCC) has profound implications for coastal marine ecosystems. Current predictions include elevated sea-surface temperatures and ocean acidification due to increased atmospheric and dissolved carbon dioxide (CO2). Sea urchins may be particularly sensitive because they are marine calcifiers, reliant on high carbonate concentrations for skeletal formation, and have larval phases that are heavily influenced by factors such as temperature. We raised purple sea urchin larvae at 11, 15, and 19 C and under three CO2 levels (380, 540 and 970 ppm) chosen to approximate current and predicted levels of atmospheric CO2 by the year 2100. Blastula, gastrula and 4-arm larval stages were exposed to 1-hour thermal challenges and mortality was assessed to determine whether there are interacting effects of temperature and ocean acidification on larval performance. While elevated CO2 did not result in significant effects on thermal tolerance, both developmental stage and rearing temperature were positively correlated with upper thermal tolerance limits. Skeletal morphometric analysis revealed a significant effect of elevated CO2 on 4-arm larvae with all three temperature groups showing a reduced skeleton size. We are currently using a DNA oligonucleotide microarray to identify changes in gene expression patterns associated with exposure to multiple stressors. These results underscore the importance of considering interactive stressors when assessing the effects of GCC. Funded by the National Science Foundation: International Postdoctoral Fellowship (NAF) & Biological Oceanography (GEH).

12.33
CO_2/HCO_3^-/PH SENSING VIA SOLUBLE ADENYLYL CYCLASE (SAC)
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Carbon dioxide (CO2), bicarbonate ions (HCO3-) and protons (H+) are central to biology. Enzymes and chemical reactions are sensitive to pH, and in addition to buffering physiological fluids, CO2/HCO3- are essential substrates or end products of biological calcification, respiration and photosynthesis. Therefore, cell and organismal homeostasis depends on sensing and tightly regulating CO2/HCO3-/pH levels. Soluble adenylyl cyclase (sAC) is a most recently identified source of cyclic AMP (cAMP), the ubiquitous intracellular second messenger in animals. sAC was originally cloned and characterized from mammalian sperm, where it is essential for fertilization. sAC activity is directly modulated by HCO3-, and over the past few years, additional functions of sAC have been identified in diverse mammalian organs including kidney, epididymis, pancreas and brain. Using molecular, cellular and whole animal approaches, we have identified sAC as a regulator of blood pH in shark gills and of salt absorption in the intestine of marine bony fishes. These functions identify sAC as an evolutionarily conserved CO2/HCO3-/pH chemosensor. Database searches reveal as yet uncharacterized sAC orthologs in most animal groups, including cnidarians, mollusks and echinoderms, as well as in phytoplanktonic organisms such as diatoms and dinoflagellates. We are currently testing the hypothesis that metabolic and environmental CO2/HCO3-/pH chemosensing via sAC is conserved in these marine organisms.

12.34
SPECIES DIFFERENCES IN THE EFFECTS OF EXERCISE ON THE STABILITY OF THE GLYCOLYTIC ENZYME LDH IN PORCELAIN CRABS
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Glycolytic activity in anaerobic tissues is maintained by the redox enzyme lactate dehydrogenase (LDH). In addition to generation of ATP, LDH activity allows glycolysis to continue during physiological hypoxia by the recycling of NAD+ and production of lactic acid. Because enzymatic proteins are only marginally stable, acidification can destabilize enzymes and reduce their functionality. LDH stability in 22 species of porcelain crabs (genus Petrolisthes) varies by over several orders of magnitude (1), and is due to both intrinsic differences in the LDH molecule and, in species with high-stability LDHs, LDH-specific extrinsic protein stabilizers. We hypothesize physiological hypoxia induces the protein stabilizers. Crabs were exercised to exhaustion on three consecutive days or held as controls. Four species of porcelain crab were studied, including 3 that have evidence for extrinsic protein stabilizers (P. donadio, P. galathinus and P.armatus) and one that does not (P. cinctipes). Aliquots of claw muscle homogenate supernatant were heated at 70°C for intervals between 0 and 60min. Residual LDH enzymatic activity assessed spectrophotometrically and time to 50% activity loss (half-life) calculated. In P. donadio, LDH stability was significantly increased by exercise; mean half-life ranged from 137 min in control to 795 min in exercised specimens. In contrast in P. cinctipes LDH stability was not significantly affected by exercise; mean half-life was 44 min in control and 60 min in exercised specimens. Differences in LDH activity were observed with exercise as well as among species. NIH SCORE GM52588. Reference: (1) JH Stillman and GN Somero. 2001. J Exp Biol 204: 767-*776*

12.35
METABOLIC RESPONSES TO ENVIRONMENTAL SALINITY IN THE INVASIVE CLAM *CORBULA AMURENSIS*
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The clam Corbula amurensis is believed to have caused a large shift in the pelagic food web in the northern reach of the San Francisco Estuary since its introduction in the 1980s. This shift has been attributed to the clam's high density, high suspension-feeding rates, and ability to thrive in a wide range of salinities. We investigated the metabolic responses of C. amurensis following acclimation to constant low, constant high and fluctuating salinities. We measured growth rate, feeding rate, respiration rate, activity of the metabolic enzyme malate dehydrogenase (MDH), and osmoregulatory performance. On average, clams did not grow during a three-month period at either high or low salinity. Clams fed more rapidly following acclimation to high salinity than low. Activity of MDH was higher at high salinity in both foot and mantle tissues. In comparison to other clam and fish species, C. amurensis had substantially higher MDH activities. C. amurensis had higher metabolic rates in both high and low salinity than in fluctuating salinities. Osmotic pressure of C. amurensis tissues was always lower than that in the acclimation water, but clams hyporegulated to a greater extent in high-salinity conditions. Overall, our results suggest that clams experiencing higher salinities have a higher metabolic demand to support osmoregulation and compensate by increasing their filter-feeding rate. This work was supported by a State of California Interagency Ecological Program.

12.36
IS THERE A SYNERGISTIC EFFECT OF THERMAL AND OSMOTIC STRESS ON METABOLIC PERFORMANCE IN FRESHWATER ZOOPLANKTON?
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The Sacramento -San Joaquin Delta is a highly altered estuarine ecosystem. People regulate the storage and diversion of freshwater from Sacramento River and San Joaquin River to serve human consumption and agriculture. Reduced freshwater flow during dry seasons, warm temperatures and increasing tidal inundation result in seasonally obvious variations of temperature and salinity. To investigate the synergistic effect of thermal and osmotic stress on metabolic performance in freshwater zooplankton, Daphnia pulex were exposed to 9 different combinations of daily fluctuations in temperature (15, 15-25, 15-30 degree Celsius) and salinity (0, 0-2, 0-5ppt). Metabolic rates and growth rates of 5 replicate isofemale lines of Daphnia pulex cultured in each condition were measured and recorded at the 1st and 5th generations. Microplate optode respirometry was performed at 15 degree Celsius and 0ppt. Daphnia exposed to the treatments of 15-30 degree Celsius 0ppt, and 15-30 degree Celsius 0-5ppt showed the lowest metabolic rates at both 1st and 5th generations. However, no obvious lower metabolic rates were observed in other treatments, indicating that high temperature is the dominant factor for the synergistic effect of temperature and salinity on metabolism. Daphnia developed from newborn to adult in 2-3 fewer days in the 15-25 degree Celsius treatments than at other temperatures. Exposure to lower temperature may slow growth due to Q10 effects, whereas exposure to higher temperatures may slow growth due to energetic tradeoffs with stress responses.

12.37
PROTEOMIC ANALYSIS OF ACUTE SALINITY STRESS IN THE TWO ASCIDIAN SPECIES *CIONA SAVIGNYI* AND *C. INTESTINALIS*
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Heavy winter rains cause hyposaline conditions near shore and in estruaries resulting in die-offs of populations of the ascidian species Ciona savignyi and C. intestinalis along the West coast of North America. C. savignyi populations rebound faster following such conditions, suggesting they may be more tolerant to osmotic change than C. intestinalis. To investigate the physiological response and interspecific differences to osmotic stress between these two congeners we analyzed changes in the proteome of whole organisms after exposure to decreasing salinities: 100%, 85% and 70% full-strength seawater for 6 h with a recovery at 100% seawater for 4 h. A quantitative analysis using 2D gel electrophoresis showed that 5 and 19% of the proteins changed abundance in C. savignyi and C. intestinalis, respectively, in response to hyposaline conditions. Analysis of these proteins with matrix-assisted laser desorption ionization (MALDI) tandem time-of-flight mass spectrometry identified numerous proteins, including those involved in the cytoskeleton, oxidative stress, fatty acid metabolism, and calcium flux, implicating cellular pathways activated in response to hyposaline treatment. Patterns of protein expression suggest that C. savignyi may be more tolerant to acute salinity stress than C. intestinalis, based on the onset of changes in protein levels and the cellular pathways present. Funding: Office of Naval Research, NSF.

12.38
THE ROLE OF ION AND WATER HOMEOSTASIS IN CHILL-COMA AND CHILLING INJURY OF THE FALL FIELD CRICKET.
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At their critical thermal minimum (CTmin), insects enter chill-coma, where movement ceases entirely. Prolonged exposure to temperatures below the CTmin result in the time and temperature dependent accumulation of whole animal injury known as chilling-injury and eventual death. Chilling injury typcially manifests as a persistent inability to coordinate movements or respond to external stimuli. Onset of chill-coma and progression of chilling injury have been shown to correlate with a loss of ion homeostasis leading to decreased cell membrane potentials in the neuromuscular system. We quantified the concentration of individual ion species and volume of hemolymph and muscle tissue during exposures to cold that induce chill-coma and chilling injury to discern the effect of chilling on gradients of individual ion species and the effects of disrupted ion homeostasis on water partitioning. Surprisingly, effects of chilling on membrane potentials appear to have little to do with muscle tissue itself, but much to do with the hemolymph and its relationship with the gut.

12.39
PHYLOGENETIC DIFFERENCES OF MAMMALIAN BASAL METABOLIC RATE ARE NOT EXPLAINED BY LOWER MITOCHONDRIAL BASAL PROTON LEAK
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The molecular nature of mammalian basal metabolic rate (BMR) is not understood but mitochondrial basal proton leak in liver and skeletal muscle is considered to contribute significantly to BMR. Comparative studies among a diversity of species showed that BMR correlates with body mass and presumably has increased during evolution of endothermy. In previous studies, marsupial mammals were not included and the proton leak was assumed to be smaller than in eutherian mammals, due to lower metabolic rates. In this study, we kept similar-sized individuals of a marsupial (Monodelphis domestica) and a eutherian (Mesocricetus auratus) species under identical conditions and compared BMR and basal proton conductance. We observed a ~40% higher mass specific BMR of the eutherian but unexpectedly the basal proton conductance was higher (~43%) in isolated liver mitochondria of the marsupial. Further measurements in marsupial species revealed an allometry of BMR and basal proton leak as found in eutherians, however the higher mitochondrial leak persisted in marsupials. Although we demonstrate that an increase of BMR during mammalian evolution is not explained by the mitochondrial leak, the general difference of proton leak between endothermic groups may assist to elucidate distinct metabolic and habitat requirements that have evolved during mammalian divergence. (CSIRO ICT Scholarship to EP).

12.40
BIOPHYSICAL MODELING OF FORAGING BEHAVIOR: HOW CLIMATE CHANGE MAY INCREASE FORAGING CONSTRAINTS
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An almost unstudied area is the effect of climate change on activity patterns of animals and thus on the structure of ecological communities. We studied field foraging behavior of diurnal rocky desert golden spiny mice (Acomys russatus), and developed a two-stage statistical model to describe how biotic and abiotic conditions affect this behavior. We found that energy expenditure and evaporative water loss, as predicted by a biophysical model, constrain spiny mouse foraging behavior. During summer ambient temperatures (Ta) increased to a range where mice needed to evaporate water in order to prevent hyperthermia; foraging behavior declined sharply when the necessary evaporation rate increased to 0.2 [mg H2O/sec*gram] when Ta was higher than 41°C during summer in the non-shaded, between-boulder (BB), microhabitat. Under RegCM future climate scenarios for 2100, maximum Ta in the study area will increase by 5.2°C and 3.6°C in A2 and B2 scenarios respectively; our biophysical model shows a reduction in the number of hours available for foraging in the BB microhabitat from 10 in current climate to 7 and 9 in A2 and B2 scenarios respectively. Consequently, mice may be forced to concentrate their summer foraging in more sheltered microhabitats, under boulders, where summer active vipers are a serious predation risk. Moreover, mice may shift their activity towards morning and evening. This reduction in spatial and temporal niche may increase intraspecific competition. The funding source: Fulbright Doctoral Dissertation Research Fellow. A grant to doctoral candidates, who wish to carry out short working visits at American research institutions.

12.41 Withdrawn.

12.42
POST-MOLT CONDITION OF WEDDELL SEALS AND LINKS WITH OVERWINTER DIVING BEHAVIOR
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Despite the extensive amount of research that has been conducted on Weddell seals, there has been remarkably little work on overwinter diving behavior or foraging success. In the McMurdo Sound region, adult Weddell seals molt in late summer (Jan/Feb), and begin their winter foraging activities at the end of a long period of relatively limited aquatic activity. As their physiological condition at this time may influence their behavioral options, we measured the blood and muscle oxygen stores and body composition of 22 adult Weddell seals (18F:4M) prior to attaching satellite-linked dive recorders which gathered behavioral data (dive depth, duration) over the winter. As expected at the end of the breeding season, both males and females were relatively thin (blubber depth 2.7 ± 0.2cm), and lean (23.5 ± 1.1% blubber). Blood oxygen stores will be estimated from measured hematocrit (63 ± 1%), hemoglobin, and plasma volume values, and muscle oxygen stores determined from collected muscle biopsies. The resulting oxygen stores and calculated aerobic dive limit will be compared to that determined in seals handled during spring (Oct-Nov) to determine if inactivity induces changes in dive physiology. Finally, the proportion of anaerobic dives will be calculated for each seal for each week following tag

deployment; a decrease in proportion could indicate a training effect. Funding for this work was provided by NSF (ANT-0838892).

12.43
METABOLIC EFFECTS OF STRESS IN RURAL AND URBAN MALE ABERT'S TOWHEES
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In mammals, stress elevates plasma glucose (PGlu) for energy. In contrast, birds have naturally high PGlu levels in non-stressed states. To determine metabolic responses to acute stress in birds, PGlu and uric acid (PUA) were assayed using wild-caught male Abert's Towhees (Pipilo aberti), from urban and rural populations. We hypothesized that rural birds would mobilize more energy in response to stress due to unpredictable food and water availability. Blood was collected immediately upon capture and after 60-minutes of restraint stress. Pre-stress levels of PGlu and PUA were similar between populations. Acute stress significantly increased PGlu by 12-18% whereas PUA significantly decreased by 34-52%, suggesting decreased protein utilization in both populations. Birds were brought into captivity and allowed to acclimate prior to receiving either corticosterone (CORT) to induce chronic stress or control treatment for 5 weeks. Blood was collected weekly and liver and skeletal muscle samples were collected at the end of the study to measure glycogen content. No changes in PGlu or PUA were observed with chronic stress. In contrast to skeletal muscle glycogen, which showed no significant change, liver glycogen in rural birds was significantly less ($p<0.05$) compared to urban birds following CORT treatment. In conclusion, differences between the two populations of birds persist in captivity with rural birds either storing less glycogen or increasing glycogenolysis to maintain their high PGlu.

13.0: GENOMICS AND PROTEOMICS APPROACHES TO UNDERSTANDING ENVIRONMENTAL STRESS

13.2
THE ROLE OF OXIDATIVE STRESS IN SETTING THERMAL TOLERANCE LIMITS
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Increasing temperatures accelerate the flux through the electron transport chain and thereby increase the production of reactive oxygen species (ROS). We tested the role of temperature in causing oxidative stress in mussel species (genus Mytilus) that differ in thermal tolerance by comparing their proteomic response to acute heat stress. A quantitative proteomic analysis, using 2D gel electrophoresis and mass spectrometry, showed that the two congeners, the warm-adapted M. galloprovincialis and the cold-adapted M. trossulus, show broad similarities in their response to acute exposure to 24°C, 28°C and 32°C and a 24 h recovery at 13°C (control). However, changes in protein levels suggest that the highest exposure temperature causes a switch from ROS-generating NADH-producing pathways to NADPH-producing pathways that can scavenge ROS in the cold- but not the warm-adapted species. Reduced levels of oxidative stress proteins in the cold-adapted M. trossulus accompany these changes, suggesting that a limited response to ROS production may cause these changes. Lower levels of a NAD-dependent deacetylase (sirtuin-2), an indicator of cellular lifespan, in M. trossulus suggest that these changes may in part be caused by modifications in the acetylation status of proteins. Additional interspecific differences in levels of proteins involved in molecular chaperoning, proteolysis and cytoskeleton suggest several new hypotheses on cellular processes limiting thermal tolerance.

13.3
METABOLOMIC, TRANSCRIPTOMIC, AND PHYSIOLOGIC APPROACHES TO UNDERSTANDING ANOXIA TOLERANCE IN EMBRYOS OF THE ANNUAL KILLIFISH *AUSTROFUNDULUS LIMNAEUS*
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The embryo of A. limnaeus is perhaps the most anoxia tolerant vertebrate. Tolerance of anoxia is inducible in this species and is gained and then subsequently lost during embryological development. Metabolomic approaches indicate that anoxic metabolism is primarily supported by lactate, but alanine and succinate also accumulate to a lesser degree. In addition, large amounts of gamma amino butyrate accumulate during exposure to anoxia, but only in embryos that exhibit substantial tolerance of anoxia. The metabolite profile described above requires mitochondrial metabolic pathways be active during anoxia. Physiologic evidence suggests that mitochondrial physiology is distinct in anoxia tolerance embryos, and this may help to explain the extreme tolerance of this species. Transcriptomic analysis of gene expression during entry into and recovery from anoxia suggest that induction of phosphoenolpyruvate carboxykinase (PEPCK) may play an important role in supporting anoxic metabolism in this species. The use of broad, survey-level approaches has lead to the development of a model for the restructuring of metabolism in embryos of A. limnaeus exposed to anoxia that can be tested with standard physiological approaches.

13.4
TRANSCRIPTOMIC PROFILING REVEALS THE CAPACITY TO RESPOND TO HEAT STRESS IN TEMPERATE AND POLAR MARINE FISHES
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Understanding how evolutionary history and genotype are linked to modern phenotypic responses to the environment is a central goal of comparative physiology. In the post-genomic era, a greater understanding of this basic concept is aided by the increasing applicability of genomic resources such as cDNA microarrays to the investigation of environmental regulation of gene expression. It becomes increasingly clear that within related phylogenetic groups, adequate sequence identity exists for many genes to allow for a genomic platform developed for one species in the group to be applied to its other members. We provide an example of this approach in a comparison of the effect of heat stress on several species of fishes, including temperate eurytherms and cold-adapted polar species. We profiled their respective responses to heat stress using a 9200-feature cDNA microarray generated for the estuarine goby, Gillichthys mirabilis.
The fishes endemic to the near-freezing waters of Antarctica, whose ability to tolerate the presence of ice has allowed them to flourish for 14 million years in sub-zero temperatures have previously been shown to have lost the ability to induce the expression of heat shock proteins in response to thermal stress. Here we demonstrate that their ability to modulate the transcriptome during exposure to elevated temperature is broader than originally hypothesized, with heat affecting the expression of hundreds of genes from numerous functional classes. Furthermore, combining multi-species datasets allowed for the identification of a novel, heat-inducible pathway linked to cell cycle arrest and apoptosis in mammals, but with undescribed function in ectotherms.

13.5
GENOMIC RESPONSES TO THERMAL STRESS IN FISHES
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Changes in temperature can represent a profound stressor for ectotherms such as fish. We have investigated the effects of acute temperature exposure on gene expression using heterologous cDNA microarrays and homologous oligonucleotide microarrays in Atlantic killifish, Fundulus heteroclitus, and threespine stickleback, Gasterosteus aculeatus. Populations of Atlantic killifish are distributed through a steep thermal gradient along the Atlantic coast of North America. Consistent with the differences in their thermal habitats, northern populations of Atlantic killifish are more tolerant of cold temperatures, and less tolerant of high temperatures, than are their southern counterparts. Similarly freshwater resident forms of stickleback, which experience low winter temperatures in their native habitat are more tolerant of cold temperatures than are anadromous forms, which experience more moderate winter temperatures in marine environments. The differences in whole-organism thermal tolerance in both species are associated with substantial differences in the responses of the transcriptome to acute thermal stress at both high and low temperatures in the laboratory. In addition, there are profound differences in gene expression between ecomorphs within a species independent of thermal stress that may reflect differences in a variety of whole-organism traits including swimming performance and thermal sensitivity.

13.6
PROTEOMICS OF SALINITY STRESS SIGNALING IN TILAPIA
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Salinity is one of the most pervasive environmental factors for fish and other aquatic organisms. It is also one of the environmental factors that is predicted to change most dramatically as a result of anthropogenically accelerated global climate change. Thus, a better understanding of how salinity stress affects fish and other aquatic organisms is needed. Salinity effects on the proteome of euryhaline tilapia are presented and discussed in the context of their adaptive significance. Such changes occur at multiple levels including protein abundance, protein posttranslational modification (PTM) state, and protein compartmentation. Analysis of such proteome changes in response to salinity stress in tilapia provide insight into the mechanistic basis of salinity acclimation of this euryhaline fish. This research was supported by the National Science Foundation, grant # IOS-0542755.

13.7
TRANSCRIPTOMICS OF ENVIRONMENTAL ADAPTATION IN WILD MIGRATING ADULT PACIFIC SOCKEYE SALMON (*ONCORHYNCHUS NERKA*)
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Successful migration of adult salmon from ocean feeding grounds to natal freshwater streams requires adapting to a suite of new environmental conditions. Global climate change is thought to intensify environmental factors and is an often cited cause of recent increases in salmon migration mortality rates. In this study, a salmonid microarray was used to characterize environmentally-regulated shifts in gene expression in gill and liver tissues of wild migrating adult Pacific sockeye salmon (Oncorhynchus nerka) between ocean and river habitats. Expression profiles indicate the transcriptome of wild migrating salmon is strongly influenced by new environmental conditions encountered within the river, including genes associated with thermal stress (molecular chaperones), osmotic stress (hormones and ion-transporters), oxygen flux (hemoglobin), detoxification (cytochrome p450s), and pathogen infection (immunoglobulins). Biopsy sampling of gill tissue and microarray-based profiling was combined with radiotelemetry and genetic stock identification to quantify the influence of gene expression on survival. 183 features were significantly differentially expressed between fish that reached spawning grounds and in-river mortalities. Only one of the five genes most strongly correlated to this signature significantly affected survival, CRSP1, part of a complex involved in regulating the transcription of nearly all RNA polymerase II-dependent genes. Funded by NSERC Canada and Genome BC.

13.8
USING TRANSCRIPTOMICS TO UNDERSTAND THE IMPACTS OF OCEAN ACIDIFICATION ON LARVAL SEA URCHINS
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Ocean acidification from the uptake of anthropogenic CO2 is expected to have deleterious consequences for many calcifying marine animals. Forecasting the vulnerability of these marine organisms to climate change is linked to an understanding of whether species possess the physiological capacity to compensate for the potentially adverse effects of CO2-acidified seawater. We have leveraged genomics techniques to examine the response of developing sea urchin larvae in high CO2 experiments. Here, we have used a custom designed DNA oligonucleotide microarray to target a suite of genes that are known to be involved in calcification, acid-base balance, energy metabolism, the cellular stress response and development. These studies underscore that physiological processes beyond calcification are impacted, and suggest that consideration of the overall physiological capacity and not just a singular focus on biomineralization processes is essential for forecasting the impact of future CO2 conditions on marine organisms. Conducted on targeted and vulnerable species, genomics-based studies have the potential to identify potential 'weak links' in physiological function that may ultimately determine an organism's sensitivity to future ocean conditions. Support: NSF OCE-0425107 to GEH. Reference: Todgham AE and Hofmann GE. 2009. Transcriptomic response of sea urchin larvae Strongylocentrotus purpuratus to CO2-driven seawater acidification. J. Exp. Biol. 212:2579-2594.

13.9
ACCLIMATION AND ACCLIMATIZATION OF PORCELAIN CRAB CARDIAC TRANSCRIPTOME RESPONSES TO THERMAL STRESS ACROSS ENVIRONMENTAL GRADIENTS IN THERMAL VARIABILITY
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Porcelain crabs, genus Petrolisthes, inhabit a wide-range of thermal microhabitats, from thermally-variable warm intertidal zone environments to cool subtidal habitats with little thermal

variability, and possess large interspecific diversity in thermal tolerance limits, plasticity of thermal tolerance limits, and thermal safety margin. Using a custom 26K unigene cDNA microarray for one porcelain crab species, Petrolisthes cinctipes, we have analyzed the responses to thermal stress in specimens that were acclimated to a range of constant temperatures where crabs experienced constant temperature for one month, acclimated to a range of fluctuating temperatures where crabs had a heat spike to intermediate (non-stressful) or high (stressful) temperatures each day for one month, or acclimatized across latitudinal and seasonal gradients in temperature. Here I will highlight variation in responses of two clusters of genes across these three sets of experiments: 1) the canonical heat-shock protein induction profiles, where heat stress always induces expression, but to varying levels depending on thermal history, and 2) a cluster of genes that have opposite responses to heat stress depending on thermal history. The first cluster of genes encodes heat shock proteins (hsps), ribosomal proteins and structural proteins, whereas the second cluster encodes other hsps, proteases, and chromatin assembly proteins. NSF 0533920. REFERENCES: Tagmount A, et al. (2010) The porcelain crab transcriptome and PCAD, the porcelain crab microarray and sequence database. PLoS ONE 5: e9327; Stillman JH, Tagmount A (2009) Seasonal and latitudinal acclimatization of cardiac transcriptome responses to thermal stress in porcelain crabs, Petrolisthes cinctipes. Molecular Ecology 18: 4206-4226.

14.0: PUTTING COMPARATIVE PHYSIOLOGY TO WORK IN THE FIELD: STABLE ISOTOPES AS TRACER OF ECOLOGICAL PROCESSES

14.2

STABLE ISOTOPES AND ANIMAL BIOLOGY: MECHANISMS, PROCESSES, AND PATTERNS

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Several biologically important elements, including H, C, O, N, and S have more than one stable isotope. Depending on the biosynthetic processes that form them, the relative abundances of each of these isotopes vary among biological materials. Thus, stable isotope analyses (SIAs) of tissues reveal much about an animal's biology. SIAs in animal biology have a multiplicity of uses which include: diagnosing trophic levels, finding out an animal's trophic position, estimating patterns of resource allocation, and tracking the movement of migratory animals. The number of papers in animal biology that rely on SIAs roughly doubles every two years. This remarkable rate of incorporation of the method into the fabric of our discipline is the result of both technological and conceptual advances. The isotopic analysis of biological samples is becoming more accurate, faster and cheaper. Isotopic biologists devise increasingly sophisticated and more realistic methods to analyze isotopic data. Here I will review the multiplicity of applications of stable isotopes in animal biology. I will highlight with example how animal physiologists have contributed to the development of these applications by providing a mechanistic foundation to the patterns that ecologists have discovered. I will contend that progress in animal isotopic ecology hinges on the insight that physiologists can give on the dynamics and faithfulness of isotopic incorporation in animal tissues. (NSF IBN-0114016. Biol. Rev. Martínez del Rio, C., Wolf, H. Carleton, S. A. & Martinez del Rio, C. 2009. Isotopic ecology ten years after a call for more laboratory experiments. 84: 91-111).

14.3

BACK TO THE BASICS: HOW UNDERSTANDING THE DYNAMICS OF ISOTOPE INCORPORATION INTO TISSUES REVEALS MUCH ABOUT THE ECOLOGY OF MIGRATORY BIRDS

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Determining whether certain natural foods satisfy nutritional requirements of animals necessitates understanding how dietary nutrients are metabolically routed to tissues in relation to nutritional demand. In general, metabolic routing of dietary protein and lipids into warbler tissue protein and fat stores decreased with that available in their diet. We also found that increases in metabolism of zebra finches associated with cold-exposure but not exercise produced measurable increases in carbon turnover rate for four organs even though daily energy intake was similar for these birds. This evidence does not support the hypothesis that carbon turnover increases with energy expenditure, and we invoke processes related to protein metabolism to explain these effects. We describe how these results can be used along with estimates of stable isotope values of dietary macronutrients to determine which nutritional resource(s) are most important for songbirds during migration. (NSF IBN-9984920, IOS-0748349). Podlesak, D & S McWilliams. 2006 & 2007. Metabolic routing of dietary nutrients in birds. PBZ 79:534-549 & Auk 124:916-925. Bauchinger, U & S McWilliams. 2009. Carbon turnover in tissues of a passerine bird: allometry, isotopic clocks, and phenotypic flexibility in organ size. PBZ 82:787-797. Bauchinger, U, J Keil, R McKinney, JM Starck, & S McWilliams. 2010. Exposure to cold but not exercise increases carbon turnover rates in specific tissues of a passerine. JEB 213:526-534.

14.4

COMPOUND SPECIFIC STABLE ISOTOPE ANALYSES: THE POWER OF A NEW TECHNOLOGY FOR ECOLOGICAL PHYSIOLOGY

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Further advancement in the use of stable isotope analysis in ecology and physiology requires a better understanding of how organisms incorporate the macromolecular elements they consume into tissue synthesis. Compound specific isotope analysis (CSIA) of amino acids (AAs) offers an informative method for tracing the elemental origins of biomolecules used to build tissues. Here we present results of two experiments that utilize CSIA to trace the synthesis and/or routing of AAs from diet. The first experiment measured the hydrogen isotope (□D) composition of AAs synthesized by bacteria (Escherichia coli) grown on the same nutrient medium but in waters of varying □D. Our results show that the contribution of water to the hydrogen of AAs depends on both the content of available AAs in the growth medium, and on the number of steps in the AA biosynthetic pathway. Our second experiment showed that routing of dietary protein into muscle tissue occurs in an omnivorous fish (Oreochromis niloticus). Surprisingly, for tilapia fed protein-deficient diets, routing of dietary protein was minimal, even for indispensable amino acids. Tilapia may be able to acquire their indispensable AA requirements directly from gastrointestinal microbiota. Overall, these results show that CSIA is a powerful tool for exploring questions that lie at the intersection of microbiology, physiology, and ecology.

14.5

WHAT CAN MEASURING ISOTOPES IN BREATH TELL US ABOUT AN ANIMAL'S BIOLOGY?

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Exhaled CO2 carries the stable isotopic signature of the oxidized substrate. We can use this intrinsic link to understand the foraging behaviour, the energetics of locomotion, and the dynamics of substrate oxidation in captive and free-ranging animals13CO2/12CO2 in exhaled breath a method with yet overseen potential and impact. (DFG Vo890/13). Voigt, C.C., Baier, L., Speakman, J.R. & Siemers, B.M. 2008. Stable carbon isotopes in exhaled breath as tracers for dietary information in birds and mammals. JEB 211,2233-2238. Voigt, C.C., Sörgel, K. & Dechmann, D.K.N. 2010. Refuelling while flying: Foraging bats combust food rapidly and directly to fuel flight. Ecology in press. Voigt, C.C., Schuller, B.M., Greif, S. & Siemers, B.M. 2010. Perch-hunting in insectivorous Rhinolophus bats is related to the high energy costs of manoeuvring in flight. JCPB in press. Here, I show that natural enrichment in $^{13}CO_2/^{12}CO_2$ indicates whether animals oxidize food directly as exogenous substrates and/or indirectly as endogenous substrates (e.g. lipids). We can also deduce from the stable carbon isotope ratios in exhaled breath whether animals belong to C3 or C4 food webs. Applying ^{13}C-labeled Na-bicarbonate warrants the quantification of energy costs of locomotion such as flight. And feeding ^{13}C-labeled substrates to animals enables us to shed light on the dynamics of substrate oxidation during in both resting and active animals. Novel portable laser isotope analysers make the analysis of $^{13}CO_2/^{12}CO_2$ in exhaled breath a method with yet overseen potential and impact. (DFG Vo890/13). Voigt, C.C., Baier, L., Speakman, J.R. & Siemers, B.M. 2008. Stable carbon isotopes in exhaled breath as tracers for dietary information in birds and mammals. *JEB* 211,2233-2238. Voigt, C.C., Sörgel, K. & Dechmann, D.K.N. 2010. Refuelling while flying: Foraging bats combust food rapidly and directly to fuel flight. *Ecology* in press. Voigt, C.C., Schuller, B.M., Greif, S. & Siemers, B.M. 2010. Perch-hunting in insectivorous *Rhinolophus* bats is related to the high energy costs of manoeuvring in flight. *JCPB* in press.

15.0: THE LIFE HISTORY PHYSIOLOGY NEXUS

15.2

LINKING PHYSIOLOGICAL EVOLUTION AND LIFE-HISTORY DIVERGENCE IN NATURAL POPULATIONS OF SNAKES

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Reptiles are an underutilized model for studying the biology of life-history variation and links to physiological adaptation. Furthermore, the existence of negligible senescence in many reptile species suggests that their study could provide intriguing insights into the biology of "not aging." Long-term studies of natural populations of garter snakes combined with laboratory manipulative experiments will be discussed. Specifically, populations of garter snakes (Thamnophis elegans) in the Sierran Nevada range are differentiated along a slow/fast pace-of-life continuum. Individuals either: grow slow, reproduce late, and live many years; or they grow fast, reproduce early, and have short lifespan. I will provide detailed results of physiological experiments in the laboratory that reveal that the long-lived phenotype has evolved better resilience to oxidative and other stresses. (NSF IOS 0922528 & NSF DEB 0710158) REFERENCES: K Robert & AM Bronikowski. 2010. Evolution of senescence: Physiological evolution in natural populations of the garter snake with divergent life history ecotypes. The American Naturalist 175: 147-159. AM Sparkman, C Vleck, & AM Bronikowski. 2009. Evolutionary ecology of endocrine-mediated life history variation in the garter snake, Thamnophis elegans. Ecology 90: 720–728.

15.3

INTERACTION OF TEMPERATURE AND EMBRYO METABOLISM ON EMBRYONIC PERIODS AND IMMUNE FUNCTION IN TROPICAL AND TEMPERATE BIRDS

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Long embryonic periods are thought to reflect a physiological trade-off between slow intrinsic development and enhanced offspring quality, with enhanced immune function thought to be one critical component of high quality offspring. Yet, previous tests have not found the expected positive relationship between embryonic periods and immune function in avian offspring. Studies here of tropical (21 spp) and north temperate (11 spp) songbirds also did not yield positive relationships between embryonic periods and the innate and acquired components of offspring immune function. However, tests have not accounted for extrinsic temperature, which varies due to parental behavior and influences length of embryonic periods to mask intrinsic rates of development. Intrinsic rates of development should be related to evolved rates of embryo metabolism. Variation in embryonic periods of 15 tropical species, once corrected for extrinsic temperature, indeed reflected intrinsic differences in mass-specific embryo metabolism and its sensitivity to extrinsic temperatures. Embryonic periods corrected for extrinsic temperatures to estimate intrinsic development rates then explained offspring immune function as predicted by theory. Thus, variation among species in offspring immune function can reflect a trade-off with evolved intrinsic rates of development, but the mediating effect of extrinsic temperature from parental behavior is a critical evolutionary and ecological influence. (NSF DEB-0543178, DEB-0841764). Martin, T.E. & Schwabl, H. 2008. Variation in maternal effects and embryonic development rates among passerine species. Phil. Trans. Roy. Soc. B 363, 1663-1674.

15.4

FUNCTIONAL LINKAGES BETWEEN PHYSIOLOGY AND LIFE-HISTORY IN BIRDS

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Life-history theory postulates that many physiological traits of organisms have evolved in relation to age-specific schedules of reproduction and mortality. Our understanding of how an organism's physiology relates to and is connected with its life-history remains inadequate. Research has shown that tropical birds have low rates of metabolism, small clutch size, slow growth as nestlings, and relatively low rates of adult mortality, whereas temperate birds have the opposite. We attempt to relate the life history of tropical birds to attributes of their cells. We found that dermal fibroblasts from tropical species grow slower than do fibroblasts from temperate species. Cells from tropical birds with low overall metabolism may have attributes that resist stress agents, especially reactive oxygen species, better than do temperate birds. To test this idea, we compared the LD_{50} of dermal fibroblasts from tropical and temperate birds to various forms of chemical injury. Preliminary results indicate that dermal fibroblasts derived from tropical birds have higher resistance to cadmium, paraquat, MMS, and thapsigargin, than do fibroblasts from temperate species. Experiments are underway to evaluate the lipid

composition of cell membranes from these two groups in an attempt to relate cell membrane composition with resistance to chemical stress, cell growth rate, and metabolism. Support: NIH grants AG024824 and AG023122, and NSF grant IBN 0212587.

15.5
CAN CAMOUFLAGE KEEP UP WITH CLIMATE CHANGE? CREATING A WORKING MODEL OF SEASONAL COLOR CHANGE IN SNOW-SHOE HARES
Creagh Breuner[1], Scott Mills[2], Doug Emlen[1], Jeff Good[1], Steve Running[3]
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Although climate change is widely accepted among scientists and the public, no global picture has emerged for how changing environmental variables are met by successful or failed adaptive responses in wild populations. Over the last 40 years snow-cover duration has declined by up to 1 month in temperate North America. However, seasonal pelt change is driven by photoperiod. This dichotomy could result in significant phenotypic mismatch in seasonally color changing animals. Camouflage is thought to be the single most important evolutionary force explaining coloration in mammals, and cases of mismatch in camouflage are among the most powerful examples of selection in the wild. The team of biologists from the University of Montana is collaborating to elucidate the selective force of the phenotypic mismatch, the mechanisms underlying coat color change and variation in sensitivity of those mechanisms, and the genetic diversity present in wild populations of hares. I have created a working model for the regulation of coat color change, incorporating melatonin, prolactin, alpha-MSH, melatonin and prolactin receptor expression, Mc1r, and agouti. Here I will discuss the model, presenting different physiological mechanisms through which a change in sensitivity to daylength may occur.

16.0: THE PHYSIOLOGY OF TRACE METAL HOMEOSTASIS AND TOXICITY

16.2
EFFECT OF OCEAN ACIDIFICATION ON THE SPECIATION OF METALS IN SEAWATER
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The continued production of CO_2 from the burning of fossil fuel is estimated to increase the pCO_2 in the atmosphere to 2000 µatm by the year ~2300. The equilibration of the CO_2 in the atmosphere with surface ocean waters will decrease the pH from the present value 8.1 to 7.4. Although a number of workers have examined how this decrease in pH can affect the production and dissolution of calcareous organisms in ocean waters, studies on the effect this has on the speciation of metals have not been examined. This decrease in the pH of seawater will decrease the concentrations of OH^- and CO_3^{2-} in the surface ocean, respectively by as much as 82 and 77%. Since these anions form strong complexes with divalent and trivalent metals, their decrease will increase the concentration of the free or uncomplexed metals in seawater. This will also increase the thermodynamic and kinetic activity of these ions. Since the ionic form of Cu^{2+} is toxic to some organisms, the lower pH may affect the productivity of phytoplankton in the oceans. A decrease in the concentrations of OH^- and CO_3^{2-} will also slow down the rate of oxidation of Fe(II) with O_2 and H_2O_2 and increase the solubility of Fe(III). These two effects will make iron more available to phytoplankton. Recent studies however have shown the decrease in pH makes it more difficult to take up iron. The reasons are not clear why this is the case. Some possibilities for the causes will be discussed. The author acknowledges the Oceanographic Section of the National Science Foundation and the National Oceanic and Atmospheric Administration for supporting this work.

16.3
CADMIUM ACCUMULATION AND TOXICITY IN AQUATIC INSECTS
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A major disconnection has come to light, where trace metals rarely elicit toxic responses in aquatic insects in standard toxicity assays at environmentally relevant concentrations, yet in nature, insect communities are profoundly impacted by trace metal contamination. Using 109-Cd as a tracer we have been examining rates of Cd influx and efflux, considering both dissolved and dietary routes of exposure. We have coupled whole organism Cd trafficking studies with subcellular compartmentalization and studies of insect antioxidant responses and provide several explanations for the disconnection between lab and field responses of insects to metal pollution. First, dissolved uptake and depuration kinetics suggest that most insect species tested require a relatively long time (40-60 days) to reach tissue steady state concentrations, with kinetic parameters following phylogeny rather closely. Second, diet appears to be a more important exposure pathway than dissolved Cd among taxa, regardless of whether species are herbivores, omnivores and predators. Third, both subcellular compartmentalization and antioxidant studies suggest that diet derived Cd is more physiologically challenging than aqueous derived Cd. In the mayfly Centroptilum triangulifer, catalase, superoxide dismutase, and reduced glutathione were all depressed after dietary exposure, whereas animals receiving comparable body burdens of Cd from water showed no differences in these endpoints. Experiments are ongoing to examine how thermal challenge alters both trafficking and antioxidant responses in aquatic insects. (NSF 0919614).

16.4
WHEN IS CHEMICAL CONTAMINATION AN IMPORTANT FACTOR WHEN CONSIDERING ECOLOGICAL CONSEQUENCES OF CLIMATE CHANGE?
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Since passage of the Clean Water Act, metal contamination from many sources has declined. But as we have learned with other water quality problems (e.g. eutrophication) such controls require constant vigilance. Climate change is one of the factors that could change the situation. Biodynamic models are a tool that provides a unified view of the influences of environment and species-specific physiology on metal exposure in different food webs. Here we use a biodynamic model to quantitatively forecast how changes projected for the future in climate and climate-driven water management in San Francisco Bay could affect the implications of Se discharges on the Bay food web. For example, model forecasts quantitatively illustrate how a shift toward a drier climate, if manifested as the number of days in the year with river inflows less than 3000 cms could exacerbate effects of the refinery discharges of Se on the Bay food web. Similar effects could occur with a shift in the balance between Sacramento River and San Joaquin River inflows to the Bay during the drier seasons. A loss of migratory salmonids and sturgeon, due to higher temperatures in upstream habitats (as projected in some models) could reduce implications of the discharges to the Bay, especially if sturgeon were replaced by species like striped bass. The latter changes are linked back to physiological differences among invertebrate prey species, that drive Se bioaccumulation. In a world where nothing is static, realistic integrated hydrological -ecological forecasts are an essential part of planning for the future. Such models must include quantitative, realistic forecasts of implications for contamination that are based upon understanding of species-specific physiological drivers of metal exposure and toxicity. Funding: UC Davis.

16.5
ACCOUNTING FOR METAL SENSITITIVY DIFFERENCES SPANNING ORDERS OF MAGNITUDE
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Sensitivity differences among aquatic organisms are common for metals and span orders of magnitude presenting a challenge to environmental regulators when setting water quality criteria. For copper (Cu) and lead (Pb) a significant part of the sensitivity variation can be accounted for by differences in physiology. For Pb, a recognized Ca2+ antagonist, the most sensitive organisms tested so far are freshwater pulmonate snails. The hypersensitivity of these snails to Pb correlates with very high Ca2+ requirements for shell formation but does not appear to be due to direct inhibition of apical Ca2+ uptake. For Cu, a recognized Na+ antagonist, the majority of variation in sensitivity among freshwater species is related to Na+ turnover rates; the higher the requirements, the higher the sensitivity. However, as is the case for Pb, pulmonate snails are the most Cu-sensitive organisms tested to date despite only modest Na+ uptake rates. The mechanisms of metal hypersensitivity in pulmonate snails will be discussed. Ambient salinity affects sensitivity to copper in euryhaline fish in a non-linear manner with the highest tolerance seen at intermediate salinities and greatest sensitivity in freshwater and full strength seawater. Measurements of transepithelial potential across the gill revealed the electrochemical driving force for Na+ transport across as a strong predictor of Cu2+ sensitivity. Predicted elevations of aquatic CO2 levels and associated pH reductions will alter metal speciation and the physiology of aquatic organisms potentially rendering them more susceptible to elevated metal concentrations.

17.0: GLOBAL CHANGE AND THE EMERGING THREAT OF DEAD ZONES: CAN INDIVIDUALS, POPULATIONS, SPECIES, AND COMMUNITIES RESPOND TO INCREASINGLY HYPOXIC OCEANS?

17.2
USING PHYSIOLOGICAL PERSPECTIVES TO PREDICT ECOLOGICAL RESPONSES TO HYPOXIA: INTEGRATING INDIVIDUALS TO ECOSYSTEMS
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Oxygen depletion in the world's oceans has recently emerged as one of the greatest anthropogenic threats to marine life due to increased rates of eutrophication and climate change. Ecologists have recently documented that severe aquatic hypoxia can cause mass mortality events and catastrophic disruption of community dynamics – leading oxygen depleted areas to be labeled as "dead zones". Physiologists, on the other hand, have long explored variation among species in traits that allow some organisms to cope with severe hypoxic conditions. Combining ecological and physiological perspectives allows us to understand how populations and communities of organisms change predictably in response to hypoxia, with important implications for ecosystem resilience and the provision of ecosystem services. This interdisciplinary approach explains why some "dead zones" are in fact full of life and can support valuable fisheries, how behavior is often coupled with physiological responses to hypoxia, and how low-oxygen will act synergistically with other environmental stressors linked to human activity. REFERENCES: Altieri AH (2008) Dead zones enhance key fisheries species by providing predation refuge. Ecology 89:2808-2818. Altieri AH and Witman JD (2006) Local extinction of a foundation species in a hypoxic estuary: integrating individuals to ecosystem. Ecology 87:717-730. Altieri AH (2006) Inducible variation in hypoxia tolerance across the intertidal-subtidal distribution of the blue mussel Mytilus edulis. Marine Ecology Progress Series 325:295-300.

17.3
BUILDING A MECHANISTIC UNDERSTANDING OF HYPOXIA'S IMPACT ON COASTAL ECOSYSTEMS: CAN PHYSIOLOGICAL RESPONSES TO HYPOXIA PREDICT POPULATION CONSEQUENCES IN A CHANGING WORLD?
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Hypoxia in coastal ecosystems impacts mobile animals when they die from exposure to low dissolved oxygen (DO) and when they avoid hypoxia and aggregate in areas along the edges of hypoxic water. Assessing hypoxia's impact on animal populations is difficult because hypoxic event hydrodynamics vary in space and time. Moreover, individuals acclimatize to hypoxia, which may affect their behavioral responses to and survival of subsequent hypoxic exposures. We used laboratory and modeling experiments, coupled with molecular techniques, to examine survival and behavioral responses of blue crabs (Callinectes sapidus) to hypoxia, test if differences in physiological state (structure of the oxygen-binding protein, hemocyanin – Hcy) alter these responses, and predict hypoxia's impact on blue crab populations. Blue crabs were tolerant of severe hypoxia and increased their movement rates during drops in DO. Crabs with hypoxia-tolerant Hcy structures, survived longer, and were less active during exposures to hypoxia than conspecifics with hypoxia-sensitive Hcy structures. Our individual-based population simulation model predicted that mortality from hypoxic events is not substantial but the degree of hypoxia acclimatization in a population influences mortality rates during hypoxic events. Our findings highlight the importance of quantifying the relationships between physiology, behavior, and survival for developing mechanistic models that predict how spatiotemporally variable disturbances impact animal populations.

17.4
HYPOXIA DOES NOT ALWAYS GENERATE DEAD ZONES: ADAPTATIONS OF FISH TO LOW-OXYGEN HABITATS
Goran Nilsson[1]
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Even without any anthropogenic influence hypoxia has long been common in many aquatic habitats due to the relatively low content and slow diffusion of O2 in water. Thus, there are several fish species that are extremely well adapted to limited and variable oxygen levels. I will discuss hypoxia adaptations in two fishes: the crucian carp (Carassius carassius) – the only fish surviving wintertime anoxia under the ice in small anoxic ponds in Northern Europe, and the

bearded goby (Sufflogobius bibarbatus) – the only fish surviving in the anoxic and sulfuric bottom mud at the Benguela upwelling along the Namibian coast. Both fishes appear to be extremely well adapted to their habitats, showing several distinct physiological adaptations, and since anoxia tolerance makes them able to avoid predation, they reach very high population densities. The crucian carp is arguably the most anoxia-tolerant fish species there is, and its capacity to withstand long-term anoxia is aided by the exotic ability to convert lactate to ethanol. The bearded goby lacks this ability but instead relies on unique behavioral traits to utilize anoxic mud as a shelter and food resource. Funded by the Research Council of Norway. REFERENCE: Vornanen, M., Stecyk, J. A. W. & Nilsson, G. E. 2009. The anoxia-tolerant crucian carp (Carassius carassius L.). Fish Physiology 27: Hypoxia (eds. J. G. Richards, A. P. Farrell & C. J. Brauner). pp. 397-441.

17.5
METABOLIC SUPPRESSION IN VERTICALLY MIGRATING PELAGIC ANIMALS IN OXYGEN MINIMUM ZONES
Brad Seibel[1], Jillian Schneider[1]
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The deep, pelagic biosphere is the largest living space on the planet and its animal inhabitants play important roles in oceanic biogeochemical cycles. Yet, the basic biology and ecology of resident animals is poorly constrained in relevant models. For example, a large fraction of oceanic animals migrate vertically each day, across large gradients in temperature, carbon dioxide and oxygen, between the surface and intermediate depths. Despite exceptional adaptations in oxygen transport capacities in many species, the extreme hypoxia and low temperature at depth in some regions suppress total metabolism, as anaerobic ATP synthesis is insufficient to compensate for reduced oxygen availability. Expanding oxygen minimum zones, warming surface waters and ocean acidification, resulting from human emissions of carbon dioxide, may act synergistically to vertically-compress the habitable night-time depth range of many oceanic species. I'll disucss critical levels of environmental variables with special emphasis on oceanic oxygen minimum zones as they constrain metabolic rates and dependent biogeochemical cycles. Rosa, R., and Seibel, B. A. 2008. Synergistic effect of climate-related variables suggests future physiological impairment in a top oceanic predator. Proceedings National Academy Sciences. 52. 20776-20780. Rosa, R., and Seibel, B. A. 2010. Respiratory and metabolic physiology in a top pelagic predator, Dosidicus gigas: implications for vertical migration in a pronounced oxygen minimum zone. Progress in Oceanography. In press

18.0: SELECTED ORAL PRESENTATIONS II

18.1
PHENOLOGY OF HIBERNATION AND REPRODUCTION IN FREE-LIVING POPULATIONS OF ARCTIC GROUND SQUIRRELS IN ALASKA
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Since 1996, the timing of annual events has been determined in natural populations of arctic ground squirrels, Spermophilus parryii, living near theToolik Field Station in arctic Alaska (Lat. 68o38â€™N). Precision in dates of immergence, first torpor, end of heterothermy, emergence, and birth of young are available from use of temperature-sensitive data loggers implanted into juvenile and adult animals of both sexes. Across the past 14 years, overall timing of hibernation and breeding has not changed significantly. Within-year dates of immergence and emergence, however, differ consistently among sex and age cohorts. Two populations of ground squirrels living 20 km apart differed consistently in length of active season, and they do not overlap in timing of reproduction which shows substantial flexibility in response to local conditions in timing of the annual cycle. Although northern Alaska is recognized as a zone of current rapid warming, most measures of climate in the Toolik Lake region have not shown consistent trends since 1996. With sustained warming, increases in the length of growing seasons here will likely be asymmetrical, with warm, but snowy springs and warm, dry falls. With this future scenario, we expect that arctic ground squirrels will delay their emergence from hibernation and delay reproduction, resulting in more favorable conditions for adults in spring while still affording juveniles adequate time to prepare for hibernation during an extended fall.

18.2
FUEL METABOLISM IN CARDIAC AND SKELETAL MUSCLES OF HARP AND HOODED SEALS
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In diving animals, skeletal muscle adaptations to extend underwater time despite vasoconstriction include elevated myoglobin (Mb) concentrations, high acid buffering ability (B), and high aerobic and anaerobic enzyme activities. However, because cardiac muscle is perfused during dives, it may rely less heavily on Mb, B, and anaerobic pathways to support contractile activity. In addition, because cardiac tissue must sustain contractile activity even before birth, it may reach physiological maturity faster than skeletal muscles. To test theses hypotheses, we measured Mb levels, B, and the activities of citrate synthase (CS), B-hydroxyacyl-CoA dehydrogenase (HOAD), and lactate dehydrogenase (LDH, measured in both directions), in cardiac and skeletal muscle samples from 65 harp and hooded seals ranging in age from fetuses to adults. As expected, cardiac muscles had lower Mb (14.7%), B (55.5%), and LDH activity (36.2%), but higher CS and HOAD (CS: 459.6%, HOAD: 371.3%, all $p < 0.05$) than skeletal muscle; it also expressed significantly more H-LDH isozyme, and was better able to oxidize lactate. In addition, heart tissue was significantly more mature at birth and weaning than skeletal muscle. These patterns are similar to those in terrestrial species, suggesting that seal hearts do not exhibit unique adaptations to the challenges of an aquatic existence. Financed by contributions from the Alaska Heart Institute and the Department of Fisheries and Oceans, Canada.

18.3
ATMOSPHERIC OXYGEN LEVEL AND THE EVOLUTION OF INSECT BODY SIZE
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While not all models agree, most estimate that over the last 500 million years atmospheric oxygen has varied from 12% to 31%. The giant insects of the late Paleozoic occurred when atmospheric PO_2 (aPO_2) was hyperoxic, supporting a role for oxygen in the evolution of insect body size. However, the paucity of the insect fossil record and the complex interactions between oxygen levels, organisms and communities makes it impossible to definitively accept or reject the historical oxygen-size link. Yet, the results of a variety of our recent rearing studies support a link between oxygen and insect size, including: 1) most insects develop smaller body sizes in hypoxia, and some develop and evolve larger sizes in hyperoxia; 2) insects developmentally and evolutionarily reduce their proportional investment in the tracheal system when living in higher aPO_2; and 3) larger insects invest more of their body in the tracheal system, potentially leading to greater effects of aPO_2 on large insects. These provide plausible mechanisms by which tracheal oxygen delivery may be involved in the small size of insects and hyperoxia-enabled Paleozoic gigantism. When we begin to examine the fossil record closely, we see that certain groups have responded more strongly to oxygen variation. While taxa such as *Protodonata* and *Paleodictyoptera* exhibited gigantism, *Blattodea* show little variation in maximum size, but do show shifts in average size. Here we examine the role of atmospheric oxygen in the evolution of insect body size. This research was supported by NSF EAR 0746352 and DOD 3000654843 to JFH.

18.4
CHANGES IN PEAK BLOOD LACTATE AND MUSCLE PYRUVATE DEHYDROGENASE FOLLOWING AN ACUTE TO CHRONIC HYPOXIA TRANSITION IN CD-1 MICE
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For nearly 8 decades researchers have observed a decline in peak and submaximal exercise blood lactate $[La]_b$, independent of O_2 delivery, in humans with the transition from acute to chronic hypoxia. Underlying mechanisms have been difficult to elucidate. Missing are animal model experiments to uncover muscle-specific explanations for what is an apparent paradox. We have developed CD-1 mice as a suitable animal model since they display lower peak and submaximal (26m/min) $[La]_b$ even after only 1W (week) of acclimation to simulated altitude (~4300m). Since lactate formation is, in part, determined by the balance between pyruvate production and oxidation we examined the possibility that pyruvate dehydrogenase complex (PDC) phosphorylation may be involved. Specifically, we examined the pathway from HIF to PD kinase (PDK)1 to PDC activity. Acute hypoxia increased muscle HIF and PDK1 protein and decreased PDC activity. 1W acclimation showed a drop in peak lactate. Also after 8min at 26m/min HIF, PDK1 protein and PDC activity returned to normoxic levels. Phosphoryation of the $pSer^{232}$ site on PDC by PDK1 followed the same pattern. These data suggest a plausible mechanism to help explain a decades old paradox.

18.5
A COMPARISON OF PROTEOMIC RESPONSES TO HEAT, HYPOSALINITY AND EMERSION IN THE GILLS OF THE RIBBED MARSH MUSSEL *GEUKENSIA DEMISSA*
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Geukensia demissa is an important member of the salt marsh community of the east coast of North America. In this variable habitat, Geukensia experiences multiple abiotic stresses including heat, hyposalinity and hypoxia. We compared changes in the gill proteome of Geukensia in response to independent exposure to each of these conditions. Mussels were immersed at 15C for 3 weeks (control), and groups of 6 were exposed to heat shock (HS; emersion at 40C for 1h), hyposalinity (OS; 14 ppt salinity (40% seawater) for 6h), or emersion (EM; emersion for 12h), followed by 24h recovery immersed at 15C. Gill proteins were extracted and separated using 2D gel electrophoresis. We used hierarchical clustering and principal components analysis (PCA) to examine similarities in response among the three treatments.PCA indicates that HS clusters with control and OS clusters with EM on the 1st component (14.23% of variance), while HS clusters with OS and EM clusters with control on the 2nd component (12.94%). On the 3rd component (8.81%) HS and EM form one cluster while OS and control form another. The similar levels of variance and the highly discrete clustering of treatments indicate that the proteomic responses to each of the stresses are relatively distinct. Proteins most important in separating treatments along each component have been detected by the magnitude of their component loadings, and will be identified using ESI-MS/MS. This work is supported by NSF grant IOS-0920103 to PAF.

18.6
EFFECTS OF ELEVATED ATMOSPHERIC CARBON DIOXIDE LEVELS ON SHELL FORMATION AND METABOLISM IN OYSTERS *CRASSOSTREA VIRGINICA* (GMELIN)
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Ocean acidification due to global rise in CO2 can afflict marine organisms negatively impacting ecosystem health. Calcium carbonate-producing marine species can be especially vulnerable to such changes, since elevated CO2 concentration and lower pH lead to a decrease of the degree of saturation of calcium carbonate potentially affecting biomineralization. We determined the effects of elevated CO2 levels on biomineralization and metabolic physiology of an intertidal mollusk, eastern oyster Crassostrea virginica. For hypercapnic exposures, we used CO2 enriched air (5000 ppm CO2).We showed that the increase in CO2 partial pressure in sea water has negative effects on physiology, rates of shell deposition and mechanical properties of the shells of Crassostrea virginica. High CO2 levels cause significant increases in the juvenile mortality rates and inhibit both shell and soft body growth. The increase in CO2 concentration results in elevated standard metabolic rates in oyster juveniles due to the higher energy cost of homeostasis and also lead to changes in the ultrastructure and mechanical properties of shells, and an upregulation of carbonic anhydrase gene expression in mantle tissue of oysters indicating that elevated CO2 levels have negative effects on the biomineralization process. Our data strongly suggest that the rise in CO2 can impact the physiology and biomineralization in marine calcifiers such as oysters threatening the survival of this species and leading to profound ecological and economic impacts. Supported by NSF,North Carolina Sea Grant and UNC Charlotte.

18.7
OCEAN ACIDIFICATION IMPACTS ON CELL-CYCLE CONTROL IN STRONGYLOCENTROTUS PURPURATUS EMBRYOS
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Anthropogenicaly driven decreases in ocean pH have been shown to impact both fertilization and development of many marine species. While researchers are rapidly gaining new insight into the impacts ocean acidification will have on calcification and developmental morphology of marine calcifiers, we still have very little understanding of the cellular mechanisms involved in these altered developmental outcomes. The aim of this project is to assess the underlying mechanism involved in the developmental delays reported for the sea urchin, Strongylocentrotus purpuratus, when cultured in CO2-acidified seawater. The effect of decreased seawater pH on early cell cleavage events was examined by tracking key regulatory checkpoints surrounding cell-cycle progression. We utilized the incorporation of a chemically modified nucleotide, BrdU, and a fluorescently tagged antibody to determine if the reduction in cleavage is a result of a blockage in DNA synthesis. In addition, the temporal changes in S-phase and mitotic cyclins were tracked using custom antibodies to determine when cell-cycle arrest is occurring. Preliminary results confirm previous reports of a significant decrease in observed cleavage rates of S. purpuratus

embryos in low pH seawater. However, cell-cycle arrest appears to be independent of standard checkpoint responses and may involve early induction of apoptotic pathways.

18.8
OCEAN ACIDIFCATION AND HYPOXIA: A DOUBLE THREAT TO IMMUNE DEFENSE
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Marine organisms have many mechanisms to respond to hypoxia and hypercapnia (elevated CO_2). However, the ability of many organisms (fish, oysters, shrimp, and crabs) to defend themselves against bacterial challenges is greatly diminished in moderate levels of environmental hypoxia. These defenses are even further reduced if hypoxia is accompanied by hypercapnia. Hypoxia in many, if not most, coastal areas has greatly increased in recent decades due to human activity. In many coastal ecosystems hypoxia is accompanied by hypercapnia and an associated acidosis, when respiration exceeds photosynthesis. We have used a variety of approaches to understand how immune defenses are impacted by hypoxia alone and in combination with hypercapnia. For example, the production of reactive oxygen species by immune cells, i.e. hemocytes or macrophages, and their ability to "kill" bacteria in co-culture, were assessed under the oxygen and carbon dioxide partial pressures known to occur within the circulatory systems of test organisms under hypercapnic hypoxic conditions. Whole organism studies measuring lethality of the bacterium Vibrio campbellii, tissue inactivation and elimination of sublethal bacterial doses and transcriptional profiles using microarrays are consistent with the notion that hypoxia impairs immune defense in marine organisms, an effect which is exacerbated by hypercapnia. Immune defense may be the Achille's heal of hypercapnic hypoxia. Supported by NSF IOS-0725245.

19.0: CAREER PATHS/OPTIONS FOR A COMPARATIVE PHYSIOLOGIST

19.1
CAREER PATHS/OPTIONS FOR A COMPARATIVE PHYSIOLOGIST
Siribhinya Benyajati
With increasing competition for traditional career paths in academia, it is of utmost importance for comparative physiologists to explore the variety of available career paths to their unique field. According to the most recent Trainee Advisory Committee Needs Assessment Survey, many graduate students and postdoctoral fellows are willing to consider career paths outside of academia. This workshop will offer attendees the unique opportunity to not only explore these career options, but to also explore specific career paths available to comparative physiologists. Therefore, this workshop is designed to examine a variety of career paths that young investigators in the field of comparative physiology have chosen, including the traditional academic route, industry, government and wildlife laboratories. Each speaker will focus on the particular skills necessary to obtain a position similar to their own. The speakers and topics will be relevant to all graduate students, postdocs and investigators interested in pursuing a job in any of these fields. This session will consist of four oral presentations followed by a breakout session in which attendees can meet with the individual speakers to ask specific questions.

19.2
TAKING THE ROAD MOST TRAVELED: ACADEMIC POSITIONS
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You are thinking about, or have chosen, to take the career road most travelled after graduate studies in Comparative Physiology. You are in luck, as many who have gone before you have left behind advice and helpful suggestions on how to navigate to your final destination, a successful career in academia. Finding a job in academia that is both a good match for the applicant as well as the university is of the utmost importance to ensure success in your career as a Comparative Physiologist. In this seminar, tools needed to qualify for and find that job as well as the pros and cons of a career in academia will be discussed. Once you have landed a position, it is important to remember that there are three aspects that the university will use to evaluate your performance in a traditional academic career track: research, teaching and service. The weight of each of these aspects can vary dramatically between departments and universities so it is important to find out the expectations of your position as early as possible. Similarities and differences between these responsibilities for a Comparative Physiologist versus a regular university faculty position will be discussed. Finally, the seminar will conclude with an overall picture of the relative job satisfaction of established Comparative Physiologists in academia.

19.3
COMPARATIVE PHYSIOLOGY IN GOVERNMENT LABS
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Many job opportunities exist for comparative physiologists with the various departments of the federal government. Specifically, the Department of the Interior consists of eight agencies with varying scientific capabilities. Within the U.S. Fish and Wildlife Service (USFWS), research programs in each region of the country support the overall mission statement of the agency. Research projects are often associated with applied management concerns affecting resources that are managed by the USFWS including endangered species of wildlife, fish, and plants and the habitats inhabited by these species. Funding for research programs is typically a combination of base funding from the federal budget and contracts with public or private entities. In the USFWS, Fisheries and Habitat Conservation Program, physiologists may be employed at a number of facilities including Fish Technology Centers, Fish Health Centers, and Fisheries Program Offices. Physiologists employed by the USFWS have varied roles including serving as primary investigators on research projects, program heads managing physiological research within a region, or science and management advisors. For students who wish to learn more about opportunities for comparative physiologists within the federal government, job postings are centralized at the USAjobs website (www.usajobs.gov) which lists available jobs and their specific duties, required qualifications for applicants, starting salary and benefits, and information needed for submitting an application.

19.4
CAREER PATHS THROUGH THE U.S. EPA
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In a recent survey of employee satisfaction, the U.S. EPA was ranked 6th of the 30 large federal agencies that were surveyed (http://data.bestplacestowork.org). I have been working as a post doc at the EPA since receiving my Ph.D. in ecological physiology from the University of Washington in 2007. There are many great opportunities at EPA for researchers, but this career path may not be for everyone. To help determine whether the EPA is right for you, I will describe: my work environment and research; the organizational structure of EPA; benefits and salaries; and my impression of how EPA compares to academics as well as some of the pros and cons of a career at EPA. I will also discuss how to find post doctoral positions at the EPA. There are three primary types of post doctoral opportunities at EPA: 1) The National Research Council (NRC) coordinates post doc positions for several federal agencies, including the EPA (http://sites.nationalacademies.org/PGA/RAP). 2) Student Services Contract Positions are becoming increasingly popular within the EPA (http://epa.gov/ord/orma/ssa-jobs.htm -> OAM RTP -> "Student Services for…"). These positions are paid hourly and have no benefits, but provide a great introduction to the EPA. 3) Post-docs are also available through the EPA's Office of Research and Development (ORD; www.epa.gov/ord/htm/jobs_ord.htm). ORD post doctoral positions have all the benefits available to other federal employees. This is a proposed presentation and does not necessarily reflect EPA policy.

19.5
TAKING THE ROAD LESS TRAVELED: ALTERNATE CAREER PATHS
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The Smithsonian Institution (SI) is more likely to be a stepping stone in a scientific career than a final destination: most scientific positions are federal and rarely become available. However, there are many opportunities to work with SI scientists as a postdoctoral research fellow or research associate. Research projects are typically developed in close collaboration with staff scientists, and it may be necessary to obtain outside grants. The Smithsonian Institution consists of 29 different bureaus, including 10 quasi-independent research centers that cover a huge range of scientific disciplines from conservation of artwork to conservation of species. Whatever the research interest, question, or analytical requirement, there is likely to be someone at the Smithsonian who shares the interest, has the right instrument, or knows someone who does. In biology, research at SI emphasizes animal conservation, ecology, and environmental science, as well as anthropology, paleobiology, evolutionary studies and systematic biology. While there are no specific programs devoted to comparative physiology, it is relevant to many existing projects. Because SI incorporates such a large spectrum of scientists and research disciplines, comparative and interdisciplinary work can flourish. Comparative physiology is currently pursued in the study of nutritional ecology of sea otters, reproductive investment in Antarctic seals, and comparative studies of lactation. For young scientists, the Smithsonian can be an exciting stage of their careers: it provides unique opportunities to acquire a broad range of skills, access to world-class facilities and expertise, and a professional network second to none.

20.0: POSTERS IN HIBERNATION/TORPOR, AGING/CONDITION, IMMUNE FUNCTION, SENSORY PHYSIOLOGY, FUNCTIONAL MORPHOLOGY, AND RENAL/OSMOTIC PHYSIOLOGY

20.1
MECHANISMS OF UREA NITROGEN SALVAGE DURING PROTEIN SCARCITY IN A FAST-ADAPTED HINDGUT FERMENTER, THE WYOMING GROUND SQUIRREL (*SPERMOPHILUS ELEGANS*)
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Urea nitrogen salvage (UNS) conserves potential waste nitrogen by recycling urea nitrogen back into body protein. We investigated UNS in a hindgut fermenting mammal, the Wyoming ground squirrel (Spermophilus elegans), which experiences seasonal nitrogen deprivation associated with winter hibernation. With approval of all methods by the University of Wyoming Institutional Animal Care and Use Committee, groups of ground squirrels were subject to diets differing in protein content (5%, 20%, and 40%), a two week euthermic fast, and a prolonged period of hibernation. We identified the presence of urea transporters in bladder and intestinal tissues in Wyoming ground squirrels, indicating a facilitated pathway for UNS. Through intraperitoneal administration of 15N labeled urea, we found that squirrels fed low protein diets and fasted squirrels had higher enrichments of labeled 15N in skeletal muscle, smooth muscle, and liver tissue than natural abundances indicating UNS. Hibernating squirrels showed greater enrichments of 15N from labeled urea in all tissue types than other groups including the fasting ground squirrels, suggesting UNS is greatly enhanced during hibernation. Through in vitro urease and urea assays we found that although ureolytic activity does not increase in hibernators, there is greater available urea within the gut allowing for a possible enhanced efficiency of urea hydrolysis during hibernation. NASA WY Space Grant Consortium, EPSCoR PiE, UWINBRE.

20.2
COPING WITH RAPID CHANGES IN RENAL CORTICO-PAPILLARY GRADIENTS DURING AROUSAL FROM TORPOR
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Kidney function is almost nonexistent during torpor, resulting in the loss of osmotic gradients required for concentrating urine. However, previous studies indicate that these gradients are rapidly restored during arousal. Our primary objective was to characterize the relationship between kidney perfusion and the regeneration of cortico-papillary salt, urea, and protective organic osmolyte gradients during arousal from torpor. We predicted that the onset of kidney perfusion would coincide with increases in osmotic gradients, albeit with delayed organic osmolyte sequestration. Renal plasma flow (RPF) and glomerular filtration rate (GFR) were measured in 6 animals arousing from torpor. Twenty-five additional animals were euthanized at various stages of arousal and renal osmotic gradients, including the protective organic osmolytes, were measured. Despite the rapid onset of blood pressure, RPF and GFR were delayed until body temperatures reached 17.6°C and 26.5°C respectively, coinciding with the re-establishment of cortico-papillary salt and urea gradients. Initial results show that during torpor most organic osmolytes were retained to a greater extent than predicted by existing salt and urea gradients. This may aid in down regulating kidney function during torpor bouts. All studies were approved by the UW IACUC committee and complied with APS guidelines. Funding was provided in part by UW NASA Space Grant Consortium and UW Neuroscience COBRE.

20.3
UREA-INDUCED HYPOMETABOLISM IN THE HIBERNATING WOOD FROG (*RANA SYLVATICA*) IS NOT REFLECTED IN ISOLATED MITOCHONDRIA
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It has long been speculated that urea accumulated during seasonal dormancy contributes to metabolic depression in many ureogenic ectotherms. Recent work suggests urea can indeed act as a metabolic depressant during dormancy in a number of ectotherms. The mechanisms by which urea exerts its hypometabolic effect are unknown, but potentially stem from inhibition of mitochondrial respiration. We isolated mitochondria from Rana sylvatica skeletal muscle, an organ that is metabolically responsive to urea, and measured respiration rates in the absence or presence of 80 mM urea. Because the effect of urea may be influenced by the intracellular milieu, we varied substrate (pyruvate or palmitoylcarnitine), temperature (4, 10, or 15°C), and pH (6.8 or 7.4). Respiration of control and urea-treated mitochondria was affected by all of these factors. However, neither state 3 nor state 4 respiration was reduced by urea treatment under any of the substrate, thermal, or pH conditions. Our results suggest that urea-induced hypometabolism observed at higher levels of organization in hibernating R. sylvatica does not result from direct inhibition of mitochondrial metabolism, and may instead be due to inhibition of energy-utilizing processes elsewhere in the cell. Investigation into urea's effects on non-mitochondrial metabolic pathways is necessary to uncover the mechanisms by which urea depresses metabolic rate. Supported by National Science Foundation Grant IOB0416750 to JPC.

20.4

FAST IN, SLOW OUT: KINETICS OF MITOCHONDRIAL METABOLIC SUPPRESSION DURING HIBERNATION ENTRANCE AND AROUSAL

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1Biology, Univ. of Western Ontario, UWO, London, ON, N6A5B8, Canada, During entrance into a torpor bout the whole-animal metabolic rate of mammalian hibernators can decrease by 100-fold within a matter of hours. This metabolic suppression corresponds with a rapid decrease in succinate-fuelled state 3 respiration rates of liver mitochondria, measured at 37oC. When isolated from 13-lined ground squirrel (Ictodomys tridecemlineatus) in early entrance (Tb=30oC), respiration decreased by 61% compared with interbout euthermia. Respiration did not decline further in late entrance (Tb=15oC) or steady-state torpor (Tb=5oC). In contrast, during early arousal (Tb=15oC), despite rapid increases in metabolic rate, state 3 respiration did not increase significantly from torpor levels. By the time Tb reached 30oC in late arousal, however, respiration was 3-fold higher than torpor and continued to increase gradually, so that in interbout euthermia it was 6-fold higher than torpor and not significantly different from the summer active state. Preincubation with 2mM isocitrate (removing oxaloacetate inhibition of succinate dehydrogenase) increased respiration only in torpor and early arousal. The "fast" initiation of mitochondrial metabolic suppression during entrance and its "slow" reversal during arousal suggests a mechanism that requires high fairly high body temperatures. The strong correlation of respiration with the mitochondrial phospholipid fatty acid 16:1 during arousal suggests that membrane remodelling may be one such mechanism. Funding: NSERC Canada.

20.5

LATITUDINAL DIFFERENCES IN THE HIBERNATION CHARACTERISTICS OF WOODCHUCKS (*MARMOTA MONAX*)

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The phenotypic flexability of hibernation and torpor characteristics within a species has received little attention. Thus, we investigated differences in hibernation and torpor characteristics of three free-ranging populations of woodchucks (Marmota monax) distributed along a latitudinal gradient from Maine to South Carolina. Data from free-ranging animals exhibited a direct relationship between latitude and length of the hibernation season. As expected, woodchucks in the northern latitudes exhibited torpor longer than those in the southern latitudes, and the arousal bout frequency decreased in a northern direction, whereas the length of torpor bouts and the number of arousals increased. Based on the latitudinal climate differences experienced by each population, phenotypic plasticity in both hibernation timing and torpor bout characteristics were observed. Further analysis revealed a direct relationship between latitude and total time spent in torpor. Maine animals spent 68 % more time in torpor than South Carolina animals. However, total time spent euthermic during the hibernation season did not differ among the three populations. We suggest that hibernators may avoid the physiological stress of torpor by staying euthermic as much as possible. Thus, woodchucks in each population maximized the time spent euthermic, utilizing torpor only at the level needed to survive winter hibernation and to commence reproduction in the spring.

20.6

'TIL DEATH DO US PART: QUANTIFYING PREDATION ON ENDANGERED JUVENILE STELLER SEA LIONS IN THE GULF OF ALASKA

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Despite decades of study, the endangered western stock of Steller sea lions (Eumetopias jubatus) has not recovered for unknown reasons. Explicit evidence of mortality and underlying causes are essential yet difficult to obtain. We present the first direct measure of predation on western Steller sea lions from post-mortem satellite-linked transmissions by specially designed implanted monitors. Twenty-seven juvenile sea lions from Prince William Sound (PWS) have been implanted with dual Life History Transmitters (LHX tags) since 2005 and released in Kenai Fjords (KF). Within the limits of our small sample size, survival rate estimates derived from 8 detected mortality events in >14,000 monitoring days are comparable to estimates based on traditional hot-branding resight methods. Control studies incurred zero mortality indicating that no mortalities were undetected or caused by our approach. Temperature profiles received from 7 of 8 mortality events indicate acute death at sea by massive trauma, likely from predation. We estimate that cumulative survival in the first three years post-weaning is 0.5 (0.34-0.61), suggesting that more than half of the animals born in the region may be consumed by predators prior to the age of primiparity. Such high levels of post-weaning predation may be the primary impediment to recovery of the Steller sea lion population in the PWS/KF region.

20.7

HEALTH AND CONDITION IN THE ADULT WEDDELL SEAL

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Weddell seals (Leptonychotes weddellii) are large-bodied, long-lived mammals that live only in the circumpolar Antarctic. They are an ideal sentinel species for the impacts of environmental change on polar species. However, longitudinal studies are notoriously difficult due to their aquatic lifestyle, therefore many baseline indicators of health and condition are not well defined. Fifty adult seals (9-27 yrs, 24M, 26F) in McMurdo Sound were sampled for mass, total body fat, blubber depth and a suite of 22 blood parameters to assess hydration state, nutritional plane, organ function and immune status. We defined results based on variation with mass, sex and age as a continuum (years) or a threshold (old vs young). Body mass increased with age, but blubber depth was best described by mass and/or sex. Ten blood parameters showed a significant effect of one or more model variables, however the predominant effect (9 of 10) was by sex. Serum chemistry suggested females were better nourished and had lower indicators of acute stress compared to males. There were limited age-declines in several parameters that could indicate impaired organ function with advancing age. Overall, seals were within clinically normal values for large mammals even at the upper end of the age continuum. Future physiological studies are strongly encouraged to incorporate potential sex effects in experimental design and analysis.

20.8

APOPTOSIS BUT NOT OXIDATIVE STRESS UNDERLIES SKELETAL MUSCLE SENESCENCE IN A FREE-LIVING DIVER, THE WEDDELL SEAL

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Any reduction in locomotory muscle performance could impact survival in the wild. Aging is one such process; typically characterized by loss of muscle quality and quantity. In diving mammals, the exercise hypoxia routinely incurred during apneustic foraging may promote cellular aging via oxidative stress. We have previously described histological senescence in the swimming muscles of old (17+ year old) relative to young (9-16 year old) free-ranging Weddell seals, a consummate mammalian diver. Therefore we investigated the association of apoptosis and oxidative stress with this observed muscle senescence. Cell death ELISA revealed a 40% age-elevation of apoptotic index. Of the three glutathione antioxidant enzymes (Glutathione peroxidase, reductase and S-transferase), only peroxidase was age-elevated overall. Reductase increased in older females only (while a decline occurred in males). No age-effect was noted for S-transferase. By contrast, catalase activity declined with age. Taken alone, this lack of clear antioxidant age-increase could support elevated apoptosis in older adults. However, we did not note a significant age-increase in oxidative stress markers (lipid peroxidation, dihydroethidium oxidation). This suggests that although apoptosis is a significant pathway for muscle senescence in Weddell seals, it is not mediated by oxidative stress. Funded by National Science Foundation Polar Programs.

20.9

ENERGETIC CONSEQUENCES OF MATERNAL ANTIBODY TRANSFER IN TREE SWALLOWS.

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Across taxa, females transfer various non-genetic factors to their offspring. Of recent interest is the transfer of maternal antibodies (matAb), which in birds stimulates endogenous offspring antibody production, and is hypothesized to reduce reliance on costly innate immune system activation. To test the energetic consequences of matAb on offspring in the field, we challenged female tree swallows (Tachycineta bicolor) prior to egg-laying with an injection of either a non-replicating antigen (lipopolysaccharide, LPS) or saline (Control). Following hatching, one half of a female's nestlings were immunized with LPS and the other half injected with saline (Control), and for 4-hrs post injection nestling metabolic rate (MR) was measured using flow through respirometry. Nestlings from LPS-mothers had a lower metabolic rate following treatment with LPS than did nestlings from Control-mothers. However, immunization of offspring with LPS depressed offspring growth, irrespective of maternal treatment. Near fledging, we challenged all nestlings with phytohaemagglutinin (PHA) to test the impact of matAb when encountering a different "pathogen." LPS-nestlings from LPS-mothers had a stronger response to PHA than did LPS-nestlings from Control-mothers, and lost the least mass. Together, these data suggest that during the nestling phase matAb may offset the energetic costs of mounting an immune response, and aid in responding to subsequent novel challenges. Funding: NSERC (Canada).

20.10

COX-2 AND INFLAMMATORY GENE EXPRESSION IN RESPONSE TO ASPERGILLUS FUMIGATUS METABOLITES IN MACROPHAGE CELL LINE RAW 64.7 AND MOUSE BONE MARROW DERIVED MACROPHAGES.

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Toll-like receptors (TLRs) are transmembrane proteins found mainly on the surfaces of immune cells that detect conserved molecular motifs known as "microbe-associated molecular patterns" from a variety of organisms. TLRs interact with several adapter proteins to activate transcription factors, leading to the production of inflammatory cytokines and the activation of adaptive immunity. COX-2 is an enzyme that converts arachidonic acid into the prostaglandin H2. This product is the critical point of the synthetic pathway of numerous members of the prostaglandin family. COX exists as two major isoforms derived from two separate genes:COX-1 and COX2. COX-2 expression is inducible. Inducible cyclooxygenase (COX-2) and its metabolites have diverse and potent biological actions that are important for both physiological and disease states of lung, which is poorly understood. Our recent findings suggest that p47 null mice have reduced ROS production and aberrant clearance of Pseudomonas aeroginosa in NFkB dependent pathway. It is known that Aspergillus species activate innate immune cells through both toll-like receptor 2 (TLR2) and toll-like receptor 4 (TLR4). In this study we have investigated TLR4 /TLR2 mediated signaling and regulation of iNOS and COX-2 expression in murine macrophages in response to fungal metabolites from Aspergillus fumigatus. Our preliminary results suggest about three fold up-regulation in COX-2 gene expression and a 1.5 fold increase iNOS gene expression in response to conidia mediated toll-like receptor activation.

20.11

EFFECTS OF STRESS ON INDICES OF INNATE IMMUNITY DURING DEVELOPMENT IN A SEXUALLY-SIZED DIMORPHIC BIRD (*LARUS DELAWARENSIS*)

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In wild birds, there is increasing interest in the ontogeny of innate immune function, its possible trade-offs with growth, and interactions with stress. However, most studies to date have focused on passerine birds, in which neonates are altricial. Moreover, potential trade-offs between growth and immune function may be amplified in sexually-sized dimorphic species, where the larger sex may grow faster to reach a larger size. Using a colony of ring-billed gulls (Larus delawarensis), in which adult males are ~15% larger than females and chicks are semi-precocial, we examined

the effects of stress on the development of the innate immune system. We collected blood samples from chicks at hatch and on post-hatch days 10 and 20 (~70% of the nestling period). Baseline samples were collected in under 3 min and stress-induced samples at 30 min post-disturbance. Corticosterone was measured with a sensitive and specific radioimmunoassay and measures of innate immunity was assessed with hemolysis/hemagglutinin (HL/HA) assays. This study will shed light on the potential growth/immune function tradeoffs, and increase understanding of the effects of environmental stress during development.

20.12 Withdrawn.

20.13
HIGH ALTITUDE CONSERVATION PHYSIOLOGY: DEVELOPING PHYSIOLOGICAL TOOLS TO MEASURE ENVIRONMENTAL STRESS IN THE MEXICAN SIERRA MADRE MOUNTAINS
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Located nine thousand feet up in the Sierra Madre Mountains in Mexico is a unique and precious biome in Corral de Piedra watershed. Mexico has been designated as a megadiversity country, one of only five in the world. As climate change continues to inflict its inexorable changes upon the world, the alpine regions are one of the most affected. In the Corral de Piedra watershed in Mexico state, freshwater trout aquaculture compete for water resources with endemic fishes, amphibians and snakes,as well as the demands for drinking water for Mexico City. In an intensive physiological study of threatened endemic fishes, amphibians and reptiles and cultured fishes (rainbow trout), we explore the physiology of stress responses associated with anthropogenic changes. Several physiological variables are measured including cortisol, glucose and lactate levels, as well as other blood related variables such as red cell hemolysis rates, a measure of oxidative stress. The study explores the possible mechanisms that result in the relationship between physiological and environmental stress in both the field and in the laboratory. Stress is of central concern in conservation and cultured, as the various stressors, which accompany intensive fish husbandry and biosecurity issues result in compromised growth and health, and promote disease. Stress may also alter metabolic scope in fish and affect growth, partly as a result of either the catabolic or gluconeogenic effect of corticosteroids. Further, it may alter the metabolic cost of growth and bioenergetic budget in rapidly growing larval stages, compromising the amount of energy allocated to growth and survival.

20.14
VISION IN ANTARCTIC EUPHAUSIIDS *THYSANOESSA MACRURA* AND *EUPHAUSIA SUPERBA*: SPECTRAL SENSITIVITY AND TEMPORAL RESOLUTION
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Euphausiid crustaceans (krill) are a key component of the Antarctic ecosystem, where they consume phyto- and zooplankton, and are a key food source for higher trophic levels. Krill are highly visual, using visually-mediated schooling and bioluminescence to avoid predators. In an effort to better understand the role of vision in these interactions, and their sensitivity to rapid climate warming along the West Antarctic Peninsula, the present study employed electroretinography on specimens collected at Palmer Station, Antarctica, to characterize spectral sensitivity of bi-lobed eyes of Big-eye Krill Thysanoessa macrura, and to measure maximal critical flicker fusion frequency (CFF) of light- and dark-adapted eyes of T. macrura and Antarctic Krill Euphausia superba at 1 and 4°C. Extracellular recording from dark-adapted T. macrura eyes indicate rhodopsins at 464 and 492nm in dorsal and ventral lobes, respectively. Chromatic adaptation experiments (>590nm adaptation) support the presence of two visual pigments, which differs from deep-sea bi-lobed euaphausiids. Temporal resolution experiments suggest speed of vision in both T. macrura and E. superba are comparable to each other and deep-sea euphausiids, with maximal CFF of 18 – 38 Hz, depending on the temperature and light treatment. This research was funded in part by a collaboration with the Palmer LTER (NSF-OPP award #0823101 to H. Ducklow) and the Faculty Development Fund of Eckerd College.

20.15
SENSORY MECHANISMS DRIVING COMMUNITY ECOLOGICAL INTERACTIONS
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Sensory systems provide critical filters that enable organisms to detect and recognize valuable resources. Trophic cascades structure populations and communities and are established to a large degree by behavior-mediated interactions that rely on sensory inputs. Specific molecules serve as chemosensory stimuli and play keystone roles in determining outcomes of species interactions at multiple trophic levels. Here, we investigated the potential contributions of surface-associated proteins as signal molecules within wave-swept, rocky intertidal habitats. As indicated by experimental results, barnacles (Balanus glandula) were constrained to produce a high molecular weight (~276 kDa), insoluble, glycoprotein complex for cuticle/shell formation. A primary subunit (~98 kDa) of this complex evoked habitat selection and settlement of conspecific larvae, thereby operating as a seminal recruitment cue. The same subunit also triggered predation by whelks and seastars on barnacle juveniles and adults in the lab and field. Such proteins simultaneously influenced demographic processes that enhanced, or diminished, barnacle populations. As dominant competitors for space, the relative balance between barnacle recruitment and predation mortality would have strong, cascading direct and indirect effects on community dynamics. By stimulating chemosensory systems, contact protein cues play keystone roles and structure species assemblages within natural communities.

20.16
CHEMICAL COMMUNICATION, FLUID DYNAMICS, AND MECHANISMS DRIVING SPERM-EGG INTERACTIONS
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Gamete chemical communication is pervasive among taxa with divergent reproductive strategies. At the scale of gamete interactions (0.01-1 mm; Reynolds numbers << 1), sperm encounter eggs while being transported within a laminar (viscous) shear flow. The magnitude of fluid forces in a human reproductive tract is remarkably similar to those in many coastal ocean environments. Consequently, mechanisms driving sperm-egg interactions for external-fertilizing marine organisms may be indicative of processes operating in their internal-fertilizing terrestrial counterparts. For red abalone (Haliotis rufescens, a large marine snail), the relationship between chemical signaling and fluid motion largely determines fertilization success. Egg-derived attractant plumes are dynamic, changing their size and shape in response to unique combinations of physical and chemical environmental features. Attractant broadcasts promoting sexual reproduction are, however, limited to a precise set of hydrodynamic conditions. Performance-maximizing shears are those most closely simulating flows in native spawning habitats. Acting either to facilitate or suppress, the principal consequence of fluid motion is modulating the strength of chemically mediated gamete interactions. High shears constrain, whereas low shears conspire with, sperm chemoattraction to establish fertilization. Because egg signaling and sperm response are tuned to meet specific fluid-dynamic demands, shear acts ostensibly as a critical selective pressure that shapes gamete evolution and determines fitness.

20.17
FUNCTIONAL MORPHOLOGY, CONSTRUCTION AND THE HATCHING MECHANISMS OF THE 'SAND COLLARS' OF *EUSPIRA LEWISII* (GASTROPODA: NATICIDAE)
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Naticids, including Lewis' moon snail (Euspira lewisii), deposit egg masses as distinctive 'sand collars'. Collars are composites of encapsulated eggs sandwiched within a matrix of extruded mucus and sediment from local environments. Thus, we compared morphology, chemical composition and sediment profiles of collars deposited by snails maintained in various sediments in our lab, with field-collected collars from Barkley Sound, British Columbia. The matrix is a relatively homogeneous mixture of acid and neutral mucosubstances and faint traces of protein. However, comparison of the grain size distributions from collars and their respective depositional environments found that collars included sediment with a reduced median grain size and an increased sorting coefficient, indicating a 'selection preference' for the finest grain sediments available in the snails' environment for their collars. These details, along with observations of snail behavior and egg hatching, suggest mechanisms for both collar construction and hatching. Previous discussion on the role of sediment, this characteristic feature of naticid egg masses, focused on camouflage, predator deterrence, and structural support. Although those are important, we believe that sediments play another but underappreciated role as ballast, particularly as these collars, deposited on shallow sub-tidal sand flats, have no other anchor to the substrate.

20.18
EFFECTS OF CADMIUM ON METABOLIC RESPONSES TO ENVIRONMENTAL HYPOXIA IN EASTERN OYSTERS *CRASSOSTREA VIRGINICA* GMELIN
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Prolonged hypoxia and heavy metal contamination are important environmental stressors for estuarine inhabitants including bivalves such as eastern oysters Crassostrea virginica. We studied metabolic responses to prolonged (2 weeks) acclimation under normoxic (21% O2) or hypoxic (5% O2) conditions in control and Cd-exposed (30 days at 50 µg L-1 Cd) oysters. Hypoxia-acclimated oysters retained normal standard metabolic rates (SMR), in contrast to a decline of SMR observed during the acute hypoxic exposure at or below the critical oxygen tension (6% O2). Cd exposure changed the metabolic profile and response to prolonged hypoxia in oysters. Cd exposure had no effect on the rate of aerobic or anaerobic metabolism, but led to a significant increase in tissue glycogen stores and elevated activity of glycolytic enzymes (hexokinase and aldolase) indicating a greater dependence on carbohydrate catabolism for energy conservation. Activities of two key mitochondrial enzymes (citrate synthase and cytochrome c oxidase) were increased during prolonged hypoxia in control oysters but were suppressed in Cd-exposed ones. No depletion in ATP or glycogen was found during prolonged hypoxia in control or Cd-exposed oysters. Our data suggest that oysters can efficiently adjust their metabolism during prolonged moderate hypoxia maintaining aerobic scope and energy homeostasis; however the mechanisms of these adjustments appear to differ in control and Cd-exposed oysters. Supported by NSF.

20.19
EFFECTS OF CADMIUM ON ANAEROBIC ENERGY META-BOLISM AND GENE EXPRESSION DURING AIR EXPOSURE AND RECOVERY OF AN INTERTIDAL MOLLUSK *CRASSOSTREA VIRGINICA*
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Marine organisms are exposed to periodical oxygen deficiency and pollution stress in estuarine and coastal zones which may affect their performance and survival. We have studied the combined effects of exposure to cadmium (Cd), and intermittent anoxia on anaerobic metabolism, energy status and mRNA expression of 12 genes involved in oxygen sensing, aerobic and anaerobic metabolism in hepatopancreas of eastern oyster Crassostrea virginica. In control oysters, prolonged anoxia resulted in suppression of nitric oxide synthase (NOS) and upregulation of cytochrome c oxidase subunit IV (COX4) while the levels of other transcripts remained unchanged. During post-anoxic recovery, mRNA expression of hypoxia inducible factor-1a was elevated, phosphoenolpyruvate carboxykinase, NOS and LON protease suppressed, and expression of other studied genes not changed. Gene expression patterns during anoxia and reoxygenation were significantly altered by Cd exposure in a gene-specific manner. In control oysters, anaerobic ATP turnover rate (MATP) during anoxia was sustained by anaerobic glycolysis with negligible contributions from ATP and PLA breakdown. In Cd-exposed oysters ATP breakdown contributed significantly to anaerobic MATP resulting in ATP depletion. This energy deficiency combined with suppression of anaerobic metabolism may strongly affect performance and survival of oysters in polluted estuaries where Cd pollution may co-occur with "dead zones". Supported by NSF.

20.20
SITES OF CADMIUM ACTION IN MOLLUSCAN MITOCHONDRIA AND THE ROLE OF OXIDATIVE STRESS IN CADMIUM-INDUCED MITOCHONDRIAL DYSFUNCTION
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Cadmium (Cd) is a toxic metal and an important environmental pollutant that can strongly affect mitochondrial function in animals. We investigated the mechanisms of Cd action on mitochondrial function of Crassostrea virginica by performing a top-down control analysis of the three major mitochondrial subsystems (substrate oxidation, proton leak and phosphorylation). Our results showed that the substrate oxidation and proton leak subsystems are the main targets for Cd toxicity. Exposure to 12.5 µM Cd strongly inhibited the substrate oxidation subsystem and stimulated the proton conductance across the inner mitochondrial membrane. The effects of Cd on the proton conductance were reactive oxygen species (ROS)-dependent and fully reverted

by an antioxidant MitoVitE. Stimulation of the mitochondrial proton conductance by ROS suggests involvement of a redox-sensitive proton transport mechanism. Cd effects on the substrate oxidation subsystem were ROS-independent and likely due to the direct inhibition of the electron transport chain. Cd had no effects phosphorylation system. Cd exposure altered the patterns of control over mitochondrial respiration, increasing the degree of control conferred by the substrate oxidation subsystem especially in resting mitochondria. These data suggest that Cd-induced decrease of mitochondrial efficiency and ATP production are predominantly driven by the high sensitivity of substrate oxidation and proton leak subsystems to this metal. Supported by NSF.

20.21

3H-L-LEUCINE INFLUX IS STIMULATED BY PROTONS AND ZN IN LOBSTER ANTENNAL GLAND BBMV

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In the antennal glands of the American lobster, (Homarus americanus), renal functions of secretion, filtration, and re-absorption occur much as they do in the analogous mammalian organs, however, the details of these invertebrate processes are little understood. The purpose of the present study is to examine the interactions between ions and amino acids during their respective re-absorption by lobster antennal gland BBMV. 3H-L-leucine uptake by BBMV was stimulated by either a proton or divalent metal (zinc) gradient across the vesicle membranes. As the concentration of protons outside the vesicles decreased (from pHo = 4 to pHo = 8; pHi = 7), the influx of 3H-L-leucine also decreased. However, as external pH became more alkaline over this pH range, 20 µM zinc was more effective at stimulating amino acid transport (3H-L-leucine influx was 31% greater at pH 8 than at pH 4). These results suggest that zinc and protons may compete for this stimulatory process. At pHi = pHo = 7, an inwardly-directed 20 µM ZnCl2 gradient led to a 4-fold overshoot of 1 mM 3H-L-leucine influx. Addition of 10 mM glycylsarcosine (glysar), a dipeptide, to the external pH 7 medium containing zinc, significantly ($p < 0.05$) reduced the magnitude of the 1 mM 3H-L-leucine uptake overshoot by a factor of two. In mammalian kidneys, glysar is reabsorbed from urine to blood by the proton-dependent PEPT2 dipeptide membrane transport protein. Inhibition of 3H-L-leucine influx by glysar in the present study suggests that an analogous dipeptide transport protein may be one process by which 3H-L-leucine may be re-absorbed by lobster antennal glands, and that the metal, zinc, may be a significant stimulatory agent for this transporter under specific physiological conditions.

20.22

AMMONIUM UPTAKE INHIBITS CALCIFICATION IN A WIDESPREAD MARINE COCCOLITHOPHORE

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Future ammonium concentration in seawater is expected to rise from anthropogenic nitrogen deposition and as a secondary effect of oceanic acidification. Emiliania huxleyi is the most abundant marine coccolithophore and plays a major role in the global carbon cycle through photosynthesis and calcification. Here we show that in Emiliania huxleyi assimilation of nitrogen as ammonium depresses calcification at both low and elevated pCO2, alters coccolith morphology, while increasing primary production. Both the nitrogen source and pCO2 synergistically drive growth rates, cell size and the ratio of inorganic to organic carbon. The ongoing anthropogenic activity may extend the ammonium availability to more open waters and could reduce bio-mineralisation by calcifying organisms, while increasing primary production in these species, thus exerting feedback on climate. η

20.23

VARIATION IN BEHAVIORAL AVERSION TO ROAD DEICERS IN SYMPATRIC TEMPERATE ZONE AMPHIBIAN SPECIES

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Global climate change may lead to local changes in winter snowfall (and therefore application of road deicers), as well as increased seawater incursions into freshwater habitats. Salts may contaminate local watersheds to levels high enough to potentially impact amphibian populations. We tested adults of four sympatric amphibian species from the northeastern United States using choice trials to determine whether they showed aversion to road deicers (primarily NaCl, but also CaCl2 and other alternatives), and if so, at what threshold concentration. Individuals of two obligate vernal pool-breeding species, wood frogs (Lithobates sylvaticus) and spotted salamanders (Ambystoma maculatum), and two non-obligate vernal pool breeders, green frogs (Lithobates clamitans) and eastern newts (Notophthalmus viridescens), were presented with two shallow dishes containing aged tap water and solutions of either NaCl or CaCl2 (0 to 500 mM). Location and behaviors were recorded during a 10-minute trial. Animals showed greater aversion to CaCl2 than to NaCl, and species differences in threshold concentrations were observed. Species with higher behavioral thresholds may attempt to breed in habitats unsuitable for successful egg and larval development. These results will help predict future impacts of increased habitat salinity on amphibian communities. Funding: New Jersey Water Resources Research Institute/USGS and the College of Science and Mathematics, Montclair State University.

20.24

DROUGHT TOLERANCE OF PELAGIC SEA SNAKES IN COSTA RICA

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We tested the hypotheses that (1) pelagic sea snakes (Pelamis platurus) dehydrate during the dry season when precipitation is absent (Dec–May) off the Guanacaste coast of Costa Rica, and (2) these snakes utilize coastal rivers as sources of fresh water. We looked for sea snakes at coastal sites near Playas del Coco, counted the numbers of snakes, measured water salinity, and determined whether captured snakes would drink fresh water during a 20-h test period immediately following capture. Most snakes were observed on slicks or drift lines and were found in roughly equal abundance during early February and during mid–April, 2010. Contrary to expectation, snakes were less abundant near each of two rivers than at sites farther out from the coast. Water salinity varied from 32–34 PPT during February and from 34–35 PPT during April. Three of 25 snakes (12%) returned to the laboratory in February drank fresh water, and 5 of 49 snakes (10.2%) drank freshwater when captured in April. February snakes drank 3.7 ± 2.6 (SD) % body mass, whereas April snakes drank 7.1 ± 4.4 % body mass. Indices of body condition suggest these snakes were only moderately dehydrated. We conclude that some individuals dehydrate to unknown extent during the dry season, but the majority of snakes in the population survive the dry season without severe dehydration while at sea without access to estuarine sources of fresh water. Supported by NSF IOS–0926802 to HBL.

20.25

IMPORTANCE OF A PUTATIVE UREA TRANSPORTER DURING PHYSIOLOGICAL STRESS IN THE WOOD FROG, *RANA SYLVATICA*

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High levels of urea contribute to the survival of various physiological stresses in anurans. Recent evidence suggests urea transport proteins (UTs) are involved in urea accumulation; however, little is known about how they may contribute to the survival of anurans during osmotic stress. Two stresses the wood frog, Rana sylvatica, is able to tolerate due in part to increased urea levels are dehydration and freezing. To elucidate the importance of UTs, we examined the tissue distribution of a putative UT and its expression in response to organismal dehydration and freezing in R. sylvatica. The UT was cloned from the urinary bladder, and its expression was analyzed using real-time qPCR and immunoblot analysis. Initial immunoblot analysis detected the presence of a 60-kDa protein in liver, kidney, bladder, gut, and lung tissue, but not in skeletal muscle or skin. Abundance of this putative UT was variable among tissues, with bladder and kidney having the highest amounts of protein. Preliminary findings suggest that UT expression in the kidney and bladder increase as a result of dehydration, whereas there was no regulatory response to experimental freezing. These results suggest that the putative UT plays an important role in urea accumulation in R. sylvatica during dehydration, but that organismal freezing, a stress that does not result in urea accumulation, may not require a change in UT abundance. Supported in part by NSF grant IAB 0416750.

20.26

THE REGULATORY ROLE OF INSULIN IN AVIAN KIDNEYS

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Birds are known to have naturally high blood glucose concentrations. Moreover, they are resistant to the glucose lowering effects of physiological doses of insulin such that supraphysiological doses must be administered to observe significant decreases in blood glucose levels. In mammals insulin lowers blood glucose by increasing uptake into skeletal and cardiac muscle as well as adipose tissues. However, prior studies have shown that supraphysiological doses of insulin were ineffective at increasing glucose uptake into these tissues of mourning doves (MODO) despite significant decreases in blood glucose. Therefore, the purpose of the present study was to test the hypothesis that insulin lowers blood glucose in birds by augmenting renal glucose excretion. The results indicate that insulin (400µg/kg body mass) significantly decreased plasma glucose concentrations (22-30%), and increased glomerular filtration rate (126-1071%) as well as glucose excretion (97-124%) providing evidence for insulin-mediated glucose regulation in the avian kidneys. Further studies are required to determine whether the effect of insulin is on the glomerular filtration rate or the transport of glucose by the renal tubules.

20.27

RELATIONSHIPS AMONG DIVERSE IMMUNE DEFENSE MEASURES IN FREE-LIVING TREE SWALLOWS

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Knowledge on how immune defense indices commonly measured in wild animals are related to one another is important for the advancement of ecological immunology. Identifying immune indices that vary in unison and those that show trade-offs within individuals is relevant not only to the understanding of immune system function in free-living animals, but also to selecting the number and type of immune measures more likely to be useful in future studies. Here we explored the relationships among six indices of immune defense measured simultaneously in individual free-living tree swallows. A principal components analysis of the indices identified three main axes of variation, representing variation in T-cell function, B-cell function, and innate immune function respectively. Indices within each axis tended to be positively correlated, while indices in different axes were uncorrelated. Only when variation in age, body condition, and general quality among individuals was taken into account, a trade-off between T-cell and B-cell function became apparent. Our results suggest that despite the complexity of the immune system and the challenge of working with non-model, free-living animals, important insights into immune defense strategies in an ecological context can be gained by using diverse indices of immune defense commonly measured in ecoimmunology studies. This research was partially funded by the National Science Foundation and Iowa State University (William Clark Award).

20.28

OXIDATIVE STATUS OF THE MICE TISSUES FOLLOWING EXPOSURE TO DECA BDE (BDE -209)

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Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants that are added to many commercial and household products such as computers, television sets, foam mattresses, carpets etc., to inhibit combustion. Due to their lipophilic and persistent nature they have become widespread environmental contaminants and are found in humans, wildlife, and biota worldwide. About 97% of American adult population has detectable levels of PBDEs. These compounds are shown to affect the nervous, reproductive and endocrine systems upon entering the body. Although some research findings report on the toxicity of PBDEs, very little is known about the specific toxicity of BDE-209, a deca brominated flame retardant. This study investigates the impact of deca brominated diphenyl ether (BDE-209) on the oxidative status and antioxidative protective mechanisms in the mice tissues *viz.*, kidney, liver, brain, and intestine. The adult male mice were injected intraperitoneally with 0.25mg/kg body weight of BDE-209 for four consecutive days (a total of 1mg/kg body weight). On the fifth day mice tissues were separated for the determination of lipid hydroperoxides, superoxide dismutase and glutathione peroxidase. There was an overall change in the oxidant/antioxidant equilibrium of the exposed mice. There was a significant increase in the levels of lipid hydroperoxides in the mice tissues. Comparatively lipid hydroperoxide levels were higher in the liver than in other tissues. The activity of antioxidative enzymes, glutathione peroxidase and superoxide dismutase have shown tissue specific alterations in exposed animals.

20.29

THERMAL STRESS DURING PRE-INCUBATION INDUCES SUBSEQUENT DEVELOPMENTAL PLASTICITY IN NORTHERN BOBWHITES

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Northern bobwhite populations (*Colinus virginianus*) are declining concurrent with climate warming. We sought to describe the relationship between chronic and acute thermal stress (Ta)

experienced during pre-incubation and the ontogeny of northern bobwhites. For 12 days prior to incubation (pre-incubation), bobwhite eggs were exposed to 1 of 5 thermal treatments: low-fluctuating Ta (LF; 25–40°C), high-fluctuating Ta (HF; 30–45°C), or treatments of HF during pre-incubation days 1–4 (H1), 4–8 (H2), or 9–12 (H3) with LF during the remainder of pre-incubation. Normal incubation followed (23d @ 37.5°C). Morphologically, HF eggs developed more (stage 14) during the pre-incubation period than LF eggs (stage 2). Eggs from acute treatment groups (H1, H2, and H3) displayed differential development with equal heating degree-hours. LF hatchling mass (7g) was higher than hatchlings receiving thermal stress during pre-incubation (6g). Physiologically, HF embryos had a higher oxygen consumption rate than other groups through day 14 of incubation. HF and H1 eggs hatched earlier than other groups (2d and 1d respectively) and exhibited the lowest hatching success (6% and 21% respectively). These data indicate that the earliest stages of bobwhite development are more affected by thermal stress during pre-incubation, resulting in aberrant development and reduced hatch rates. Further, the reduced hatch rates experienced by groups receiving thermal stress suggest that the embryonic susceptibility of the bobwhite life-cycle may be a factor in population declines concurrent with climate warming.

20.30 Withdrawn.

20.31
EVOLVED RESPONSES TO HEAT STRESS IN SNAKE LIFE-HISTORY ECOTYPES
Tonia Schwartz and Anne Bronikowski
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For a species to persist in a changing environment, individuals need to respond appropriately to physiological/environmental stress, and the molecular networks underlying these responses need to evolve at the population level. Evidence from laboratory model systems indicate that molecular networks regulating stress response are entwined with networks underlying life-history trade-offs between longevity and growth/reproduction. To understand how these molecular networks are functioning in natural populations, we are comparing stress responses between naturally evolved, closely related populations, of garter snake ecotypes. These ecotypes lie on opposite ends of a pace-of-life-history continuum with the fast-living ecotype having faster growth, higher reproductive output, and shorter lifespan relative to the slow-living ecotype. In a common garden experiment, we use a heat stress as an activator of general stress and metabolic response to investigate if these molecular networks have diverged in these ecotypes. Preliminary results indicated that the ecotypes respond differently to heat stress in their 1) levels of circulating free radicals (superoxide and hydrogen peroxide), 2) production of hydrogen peroxide in the liver mitochondria, and 3) amount of DNA damage in blood cells. We are currently analyzing their response to stress at the level of the transcriptome RNA-seq. Thus far, these results support the hypothesis that these closely related populations of garter snakes have evolved different responses to physiological stress. This is particularly interesting in the context of their evolved life-history differences.

20.32
GENETIC VARIATION IN NUCLEAR AND MITOCHONDRIAL GENES IMPORTANT FOR ENERGY METABOLISM VARY ALONG A CLIMATIC GRADIENT IN MONTANE POPULATIONS OF A LEAF BEETLE
NE Rank[1,2], S Heidl[1], JT Smiley[2], P Mardulyn[3] and EP Dahlhoff[2,4].
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Many organisms in montane habitats live in fragmented populations that are especially vulnerable to climate change. The ability of small montane populations to persist depends partly on whether they possess genetic variation in their capacity to respond and adapt physiologically to altered environments. In the Sierra Nevada Mountains of California, the willow leaf beetle *Chrysomela aeneicollis* occurs at high elevations just below tree line (2400-3600 m). Variation at genetic marker loci [5 allozymes, 5 microsatellites, and a 550 bp region of mitochondrial cytochrome II oxidase (COII)] shows significant differentiation among montane drainages along a 75 km transect from the King's River in the southwestern Sierra to Rock Creek in the central Sierra. Geographic variation along this transect is much greater for the allozyme locus phosphoglucose isomerase (PGI) than for other nuclear marker loci. In prior studies, we described functional, physiological, and reproductive differences among PGI genotypes that correspond to differences in frequency over this latitudinal temperature transect. Here we show that PGI variation and environmental variability jointly affected persistence or disappearance of local populations over the past decade. In addition, we have found that latitudinal variation in frequencies of mitochondrial COII haplotypes is concordant to variation in PGI frequencies previously observed. Natural selection may act on COII as it does on PGI. Genetic variability at loci, such as COII and PGI, which are critical to energy metabolism, may contribute to success of population persistence in the face of anticipated rapid environmental change.

20.33
EVOLUTION OF THERMAL SENSITIVITY OF A TROPICAL RAINFOREST LIZARD: ARE TROPICAL SPECIES PARTICULARLY VULNERABLE TO WARMING?
Roberta Damasceno[1] and Ana Carolina Carnaval[1,2]
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We test hypotheses about the evolution and plasticity of whole organism responses to temperature in a widespread and phylogeographically structured tropical lizard (*Leposoma scincoides*, Gymnophthalmidae) from the critically endangered Brazilian Atlantic Forest. Theory and accumulating empirical evidence point to narrower physiological tolerances of tropical organisms (e.g. Tewksbury et al. 2008 *Science* 320, p.1296; Kearney et al. 2009 *PNAS* 106, p.3835), but how this evolves across elevational gradients is less explored. We present preliminary data on critical thermal maximum and minimum (CT max and CT min), and selected temperature (T sel) in a thermal gradient, of three high altitude (1,000 m) populations (sample sizes varying from 1 to 7 animals). CT max was similar among populations (40.4, 40.0, and 40.5 ºC) and one population (N=1) showed a lower CT min (CT min: 5.67, 12.3 and 13.0 ºC). Temperature tolerances (CT max – Ct min) were wide, exceeding temperatures experienced voluntarily in the thermal gradient. Behavior observations of *L. scincoides* in the gradients suggest that it may select preferred temperatures in the leaf litter. There is no consistent evidence of plasticity in thermal physiology after animals are subject to acclimation periods at 21 and 26 ºC. Further data (being collected) on thermal physiology of two lowland populations, as well as temperature availability in microhabitats will bring insights on possible faunal responses to future environmental change.

20.34
THE EFFECTS OF ENVIRONMENTAL POLLUTANTS ON INNATE IMMUNE FUNCTION OF AMPHIBIANS:NONMONOTONIC INNATE IMMUNE SUPPRESSION OF ADULT RED-SPOTTED NEWTS (*NOTOPHTHALMUS VIRIDESCENS*) EXPOSED TO ATRAZINE
Itzick Vatnick, Raymond Howanski, Elizabeth Shank, Laura Stashefski, and Marc Brodkin.
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The effects of environmental pollutants on amphibians have been well studied. Environmental pollutants affect amphibians in all stages of development and have a wide range of effects. For example, many of these toxicants alter endocrine function and therefore have been classified as endocrine disrupting chemicals (EDC). In the past decade we have been studying the effect of environmental acidification on the innate immune system of adult frogs and have demonstrated its detrimental effects. We became interested in examining atrazine's effects on the innate immune function of adult frogs after reading the work of Tyrone Hayes on *Rana pipien*s, the same species we have been working with. His work clearly demonstrated that atrazine functions as an EDC. We have expanded on that work and demonstrated that atrazine also acts as an immune disruptor in adult *Rana pipiens*. The work presented here demonstrates that the immune disruption extends to adult Red-spotted newts (*Notophthalmus viridescens*), a nonanuran amphibian. Most importantly, this disruption exhibits a nonmonotonic dose response (NMDR). The mounting evidence in the past decade of hormetic effects of environmental pollutants bring into question the risk assessment paradigms that underline the regulatory framework for conservation of rapidly declining amphibian populations.

20.35
ENERGETIC STATUS MODULATES VULNERABILITY OF NEURAL CIRCUITS TO ENVIRONMENTAL STRESS
Robertson, R.M., Rodgers, C.I., Armstrong, G.A.B. and Money, T.G.A.
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The sensitivity of neural circuits to ambient conditions can be tuned by exposure to stressors but the mechanisms underlying this are incompletely understood. We investigated the operation of the ventilatory central pattern generator (vCPG) in the locust, *Locusta migratoria*, in response to anoxic coma. We tested the role of the metabolic stress sensor, AMP-activated protein kinase (AMPK), in mediating the responses. We recorded vCPG activity extracellularly from a median nerve of the metathoracic ganglion. 10^{-3}M AICAR and 10^{-4}M Compound C were used to activate and inhibit AMPK. Intact locusts were suffocated for 30 minutes by submersion in water at 22°C and on return to air the times to recover ventilation were recorded. Azide-induced anoxia had a multi-phasic effect on the motor pattern (transient increased frequency; motor pattern arrest (spreading depression; SD); prolonged reduced frequency). AMPK activation was necessary and sufficient for the frequency modulation but not for SD. However the vulnerability to SD induced by treatment with 10^{-4}M ouabain was exacerbated by AICAR and inhibited by Compound C. Food-deprivation for 4 days increased the time for intact locusts to recover from suffocation. AMPK activation was necessary and sufficient for this effect. Anoxia-induced reductions in motor pattern frequency were associated with reductions in metabolic rate that could be eliminated by inhibiting AMPK. We propose that AMPK mediates changes to neuronal properties that tune the vCPG for operation in an energy-save mode after exposure to environmental stress.

21.0: PLENARY LECTURE

21.1
VISUALIZING CHANGE
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Advances in biological imaging are rapidly increasing our ability to observe living systems across vast and minute scales of space and time. Satellites collect images of biotic and abiotic processes on continental and oceanic scales, tracking data such as temperature, primary productivity, and large animal migrations. On the other end of the spatial scale, microscopy techniques have blossomed in recent decades, with 3D microscale imaging becoming common through modalities such as confocal microscopy, microCT, synchrotron tomography, and high-field MRI. Gene expression and physiological function can be visualized in the context of 3D micro and gross anatomical structures. At the organismal level, high-speed video at up to 100,000 frames per second acts as a microscope for time, slowing the beat of a fly's wing or snap of a trap jaw ant to a timescale compatible with human perception. Beyond issues of time and space, imaging techniques such as CT and MRI also allow us to see internal structures through optically opaque tissues. In recent years at Brown University we have been developing a dynamic, 3D skeletal imaging method called X-ray Reconstruction of Moving Morphology (XROMM; xromm.org). XROMM combines static 3D bone morphology from a bone scan with 3D skeletal motion data from biplanar videoradiography. With XROMM, rapid skeletal motion, such as frog jumping or bird flight, can be visualized at up to 1000 frames per second and with 0.1 mm precision. New challenges in biological imaging include automated image analysis, feature recognition, and image data and metadata curation. (W.M. Keck Foundation, NSF DBI-0552051, NSF IOS-0840950.)

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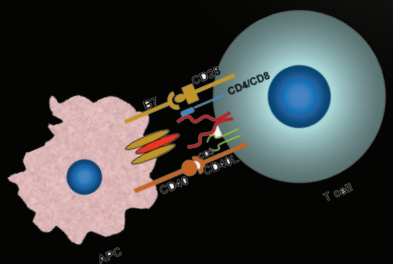
2010 American Physiological Society Conference

Inflammation, Immunity and Cardiovascular Disease

Westminster, Colorado

August 25-28, 2010

CONFERENCE PROGRAM & ABSTRACTS



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Inflammation, Immunity and Cardiovascular Disease

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Acknowledgements

The Conference Organizers and The American Physiological Society gratefully recognize the generous financial support provided through unrestricted educational grants from:

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2010 APS Conference
Inflammation, Immunity and Cardiovascular Disease
August 25—28, 2010, Westminster, Colorado

Wednesday, August 25	Thursday, August 26	Friday, August 27	Saturday, August 28
3:00 PM **Registration** 6:00—7:45 PM **Opening Reception** 7:45—8:00 PM **Opening Comments:** **David Harrison,** Emory Univ. Sch. of Med. 8:00—9:00 PM **Plenary Lecture:** Participants: **Goran K. Hansson,** Karolinska Univ. Hosp., Sweden	7:00 AM **Registration** 8:30 AM—12:30 PM Symposia I: **Basic Aspects of Immunology I** Participants: **Cornelia Weyland,** (Chair) Stanford Univ. Sch. of Med. **Ross Kedl,** Univ. of Colorado **Holden T. Maecker**, Stanford Univ. 10:00—10:30 AM **Break** **E. William. St. Clair,** Duke Univ. Med. Ctr. **Cornelia Weyland,** Stanford Univ. Sch. of Med. **David Mosser,** Univ. of Maryland 12:45—2:30 PM **Poster Session I** 2:30—6:00 PM Free time 7:00—8:30 PM Symposia II: **Basic Aspects of Immunology II** Participants: **Amy Major,** (Chair), Vanderbilt Univ. Sch. of Med. **Luc Van Kaer,** Vanderbilt Univ. Sch. of Med. **Alexander Niessner,** Med. Univ. of Vienna, Austria 8:45—10:00 PM Oral Presentations I: **T Cells and Cardiovascular Disease**	7:30 AM **Registration** 8:00—11:30 AM Symposia III: **Inflammation, the Vascular Wall and Lesion Formation** Participants: **Goran K. Hansson,** (Chair) Karolinska Univ. Hosp., Sweden **Eric Belin de Chantemele,** (Chair), Med. Coll. of Georgia **Jordan S. Pober,** Yale Univ. **Peter Libby,** Brigham & Women's Hosp. **Klaus Ley,** La Jolla Inst. For Allergy & Immunology **Amy S. Major,** Vanderbilt Univ. Sch. of Med. 10:00—10:30 AM **Break** **Andrew Lichtman,** Brigham & Women's Hosp. **Elena Galkina,** Eastern Virginia Med. Sch. 11:30 AM—12:30 PM Oral Presentations II: **Inflammation, Vascular Function and Atherosclerosis** 12:45—2:30 PM **Poster Session II** 2:30—5:30 PM Free time 5:30—6:30 PM Career Workshop **The *Ins* and *Outs* of Authorship** 7:00—9:30 PM Symposia IV: **Inflammation and the Metabolic Syndrome** Participants: **Tomasc Guzik,** (Chair), Jagiellonian Univ. Sch. of Med., Poland **Paul Marvar,** (Chair), Emory Univ. Sch. of Med. **Andrew Gewirtz,** Emory Univ. Sch. of Med. 8:15—10:00 PM Oral Presentations III: **Hypertension, Renal Disease and Inflammation**	7:30 AM **Registration** 8:30 AM—12:30 PM Symposia V **Inflammation, Immunity, Hypertension and Renal Disease** Participants: **David M. Pollock,** (Chair) Med. Coll. of Georgia **Babette LaMarca,** (Chair, Univ. of Mississippi Med. Ctr. **Bernando Rodriguez-Iturbe,** Hosp. Univ. of Maracaibo & Univ. of Zulia, Venezuela **Ernesto L. Schiffrin,** Jewish Genl. Hosp., Montreal **Meena Madhur,** Emory Univ. Sch. of Med. 10:00—10:30 AM **Break** **David Mattson,** Med. Coll. of Wisconsin **Jennifer Pollock,** Med. Coll. of Georgia **Oscar Carretero,** Henry Ford Univ., Detroit

Location:

The 2010 APS Conference: Inflammation, Immunity and Cardiovascular Disease, will be held August 25—28, 2010, at the Westin Westminster Hotel, 10600 Westminster Blvd., Westminster, CO 80020, telephone (303) 410-5000, FAX: (303) 410-5005.

On-site Registration Hours:

Wednesday, August 25……3:00—8:30PM
Thursday, August 26..….....7:00AM—2:30 PM
Thursday, August 26..….....6:00—8:00 PM
Friday, August 27……7:00 AM—2:30 PM
Friday, August 27……...5:30—8:00 PM
Saturday, August 28….....7:30—10:30 AM

On-Site Registration Fees (in US Dollars):

APS Member..... $450
Retired Member $265
Nonmember..... $500
Postdoctoral..... $400
Student $350

The registration fee includes entry into all scientific sessions and opening reception and lunches.

Payment Information:

Registrants may pay by institutional or personal check, traveler's check, MasterCard, VISA or American Express. Checks must be payable to *"The American Physiological Society"* and drawn on a United States bank payable in US dollars.

Student Registration:

Any student member or regularly matriculated student working toward a degree in one of the biomedical sciences is eligible to register at the student fee. Nonmember postdoctoral fellows, hospital residents and interns, and laboratory technicians do not qualify as students. Nonmember students who register on-site must provide a valid university student ID card. APS student members should present their current APS membership card indicating their student category status.

Postdoctoral Registration:

Any person who has received a Ph.D. degree in physiology or related field, within five years of this conference, as attested to by the department head is eligible to register at the postdoctoral fee. **A statement signed by the department head must accompany the registration form and remittance when registering.**

Press:

Press badges will be issued at the APS registration desk, only to members of the working press and freelance writers bearing a letter of assignment from an editor. Representatives of allied fields (public relations, public affairs, etc.) must register as nonmembers.

Ancillary Session:

APS Career Workshop: This special session entitled: "Writing Your First Paper: The *Ins* and *Outs* of Authorship" will be presented by Lacy A. Holowatz, member of the APS Career Opportunities in Physiology Committee. Discuss the criteria for authorship and various roles authors can play during the research process and preparation and publication of a manuscript. Through case studies, explore real-life scenarios and how best to deal with the various issues that can arise with authorship.

Program Objective:

The goals and objectives of this conference are to review the fundamentals of innate immunity and antigen presentation and how various cardiovascular diseases could affect these functions. In addition, the conference will review the fundamentals of adaptive immunity, and in particular discuss T-cell function and why many facets of this might be altered in diseases like atherosclerosis and hypertension. Function of various T cells subtypes, such as CD4+, CD8+, T regulatory cells and TH17 cells will be highlighted.

The conference will review methods for study of sophisticated study of the immune system in cardiovascular disease, including fluorescent cell sorting, appropriate measurement of cytokines, and use of transgenic mice, studies of antigen presentation and studies of T cell activation. Furthermore, there will be state-of-the-art presentations regarding the role of inflammation, and in particular innate and adaptive immunity in cardiovascular disease.

Target Audience:

This meeting is intended for all professionals involved in teaching, research and clinical fields related to inflammation, immunity and cardiovascular diseases.

This meeting has been made possible through the generous support from:

Genentech, Inc.

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WEDNESDAY, AUGUST 25, 2010

Plenary Lecture

1.0 PLENARY LECTURE

Wednes., 7:45-9:00 PM, Westminster Ballroom III.

7:45 PM **1.1** Opening Comments. **David Harrison,** *Emory Univ. Sch. of Med.* and *David* **Pollock,** *Med. Coll. of Georgia.*

8:00 PM **1.2** Atherosclerosis as an Inflammatory Disease: A Paradigm Shift in Cardiovascular Medicine. **Goran K. Hansson,** *Karolinska Univ. Inst., Sweden.*

THURSDAY, AUGUST 26, 2010

Symposia I

2.0 BASIC ASPECTS OF IMMUNOLOGY

Thurs., 8:30 AM-12:30 PM, Westminster Ballroom III.

Chair: **Cornelia Weyand,** *Stanford Univ. Sch. of Med.*

8:30 AM **2.1** Introduction. **Cornelia Weyand,** *Stanford Univ. Sch. of Med.*

8:35 AM **2.2** Basic Aspects of T Cell Function. **Ross Kedl,** *Univ. of Colorado.*

9:20 AM **2.3** How to Measure and Study Immune Responses. **Holden T. Maecker,** *Stanford Univ. Sch. of Med.*

10:05 AM Break.

10:30 AM **2.4** Cytokines and the Use of Cytokine Directed Therapy. **William E. St. Clair,** *Duke Univ. Med. Ctr.*

11:15 AM **2.5** Adaptive Immune Responses and Resident Vascular Cells. **Cornelia Weyand,** *Stanford Univ. Sch. of Med.*

12:00 Noon **2.6** The Identification and Characterization of Subpopulations of Moncoytes and Macrophages During Inflammation and Infection. **David Mosser,** *Univ. of Maryland.*

Poster Session I

3.0 CARDIOVASCULAR DISEASE AND IMMUNITY

Thurs., 12:45-2:30 PM, Westminster Ballroom IV.

Board #

1 **3.1** Withdrawn.

2 **3.2** Psychological Stress, Vascular Inflammatory & Remodeling Proteins: Evidence of "Outside-In" Inflammation? **M. Fleshner and R. Thompson.** *Univ. of Colorado, Boulder.*

3 **3.3** Activation of Protein Phosphatase Type 2A Facilitates its Binding to Tight Junction Proteins and Causes Endothelial Barrier Dysfunction. **F. Wu and J. X. Wilson.** *Univ. of Buffalo.*

Board #

4 **3.4** Soluble IL-6 Receptor Alpha is Negatively Associated with FMD in Women 20-50 years of Age. **M. Cortez-Cooper, E. Meaders, J. Stallings, S. Haddow, B. Kraj, G. Sloan, A. Mulloy, K. McCully and J. Cannon.** *Med. Coll. of Georgia and Univ. of Georgia.*

5 **3.5** Interleukin-1 Receptor Type I Expression is Related to Lumbar Bone Mineral Density and Intima-media Thickness in Adult women. **J. Cannon, M. Cortez-Cooper, E. Meaders, J. Stallings, S. Haddow, B. Kraj, G. Sloan, K. McCully and A. Mulloy.** *Med. Coll. of Georgia and Univ. of Georgia.*

6 **3.6** C-Reactive Protein and Endothelial Fibrinolytic Dysfunction. **B. Weil, K. Diehl, J. Greiner, B. Stauffer and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

7 **3.7** Impaired Endothelium-Dependent Vasodilation with HIV-1 Infection is Due to Oxidative Stress. **B. Weil, B. Stauffer, M. Mestek, J. Greiner. E. Connick and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

8 **3.8** Influence of HIV-1 Infection and HAART Therapy on Endothelial Progenitor Cell Number and Function. **O. MacEneaney, E. Kushner, J. Cech, R. Morgan, M. Mestek, B. Weil, J. Greiner, B. Stauffer, E. Connick and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

9 **3.9** HIV-1 Viral Proteins gp120 and Tat Impair Endothelial Progenitor Cell Function. **O. MacEneaney, E. Kushner, J. Stoner, B. Stauffer, E. Connick and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

10 **3.10** CD31+ T cells, Endothelial Function and Cardiovascular Disease Risk. **E. Kushner, R. Moragan, O. MacEneaney, B. Weil, B. Stauffer and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

11 **3.11** Deletion of Murine LDL Receptor Related Protein in Granulocytes and Monocytes Causes Decreased Activation of Invariant Natural Killer T Cells. **R. Covarrubias, N. Braun and A. Major.** *Vanderbilt Univ. Sch. of Med.*

12 **3.12** FcγRIIb Influences Cardiovascular Disease and Modulates the Inflammatory Response of Dendritic Cells. **Y. Mendez-Fernandez, B. Stevenson, N. Braun, N.**

Board #

Wade, R. Covarrubias and A. Major. *Vanderbilt Univ. Sch. of Med.*

13 **3.13** Higher White Blood Cell Count is Associated with Enhanced Endothelin-1 System Activity in Adult Humans. **K. Diehl, B. Weil, J. Greiner, B. Stauffer and C. DeSouza.** *Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

14 **3.14** Withdrawn.

15 **3.15** Disturbed Flow Induces Acute and Dynamic Inflammation Leading to Rapid Atherosclerosis in Mouse Carotid Artery. **N. Alberts-Grill, A. Rezvan and H. Jo.** *Emory Univ. Sch. of Med.*

16 **3.16** ApoE Reduces Atherosclerosis Independently of Lowering Plasma Cholesterol by Decreasing Inflammation in Circulating Leukocytes and Vascular Endothelium. **N. Gaudreault, J. Posada, N. Kumar, K. B. Stephens, M. Harms, N. S. Reyes de Mochel, D. Eberlé, R. Y. Kim, J. H. Rapp and R. L. Raffai.** *Univ. of California, San Francisco/NCIRE/ VA Med. Ctr.*

17 **3.17** The Lupus Susceptibility Locus *Sle1* both Singularly and in Concert with *Sle3* does not Contribute to the Acceleration of Atherosclerosis in LDLr$^{-/-}$.mice. **N. Wade, Y. Mendez-Fernandez and A. Major.** *Vanderbilt Univ. Sch. of Med.*

18 **3.18** Stimulation of GTP Cyclohydrolase I by Phosphorylation Upon T Cell Activation. **W. Chen, L. Li, T. Brod, O. Saeed, S. Dikalov and D. Harrison.** *Emory Univ. Sch. of Med.*

19 **3.19** Role of Nox2 in Mouse Hindlimb Collateral Growth. **J. Unthank, M. Distasi, C. Labarrere and S. Miller.** *Indiana Univ. and Clarian Hlth. Methodist Res. Inst., Indianapolis.*

20 **3.20** Targeting Integrin Signaling in Inflammation–Identification of Small Molecule Antagonists of Integrin Cytoplasmic Domain–Syk Interactions. **D. Woodside, A. Caivano, D. Bakthavatsalam, R. Biediger, P. Vanderslice, J. Willerson and R. Dixon.** *The Texas Heart Inst. at St. Luke's Episcopal Hosp.*

21 **3.21** Creation of a Suite of Immunocompromised Knockout Rat Models. **D. Ji, Y. Wu, L. Little, J. Books, E. Weinstein and X. Cui.** *Sigma-Aldrich Adv. Genetic Engineering Labs., St. Louis.*

Board #

22 **3.22** The Protective Role of TRIF in the Progression of Dilated Cardiomyopathy. **E. Abston, M. Coronado, J. A. Frisancho, A. Bucek, J. Shin, D. Bedja, K. Gabrielson, W. Mitzner and D. Fairweather.** *Johns Hopkins Univ.*

23 **3.23** Development of Spontaneous Anergy in Invariant NKT Cells in a MouseModel of Dyslipidemia. **N. Braun and A. Major.** *Vanderbilt Univ. Sch. of Med.*

24 **3.24** N-Acetyl-Seryl-Aspartyl-Lysysl-Proline (Ac-SDKP) Ameliorates the Severity of Experimental Autoimmune Myocarditis in Rats. **P. Nakagawa, Y-H. Liu, T-D. Liao, G. Gonzalez, X-P. Yang, N-E. Rhaleb and O. Carretero.** *Henry Ford Hosp. Detroit.*

25 **3.25** Caspase Inhibition Reduces Inflammatory Cell Apoptosis in the Heart and Ameliorates Cardiac Dysfunction with Chronic Pressure Overload Hypertrophy. **M. Park, S. Gao, C. Hong, R. Gelpi, S. J. Leibovich, R. N. Kitsis, S. F. Vatner and D. E. Vatner.** *Univ. of New Jersey Dental & Med. Sch. and Albert Einstein Coll. of Med.*

26 **3.26** Body Mass Index and Thrombogenic Factors in Newly Menopausal Women. **M. Jayachandran, R. Litwiller, W. Owen and V. Miller.** *Mayo Clinic.*

27 **3.27** Long-Term Vagus Nerve Stimulation Improves Left Ventricular Function and Normalizes Pro-Inflammatory Cytokines in Dogs with Chronic Heart Failure. **S. Ruble, J. Hamann, R. Gupta, M. wang, I. Ilsar and H. Sabbah.** *Boston Scientific Corp., St. Paul, MN, and Henry Ford Hosp.*

28 **3.28** Stress-induced Hypertension Promotes T Lymphocyte Activation and Vascular Inflammation. **P. Marvar, A. Vinh, S. Thabet, D. Geem, K. Ressler and D. Harrison.** *Emory Univ. Sch. of Med.*

29 **3.29** Adverse Myocardial Remodeling Involves Interactions Between Sensory Nerves and Inflammatory Cells. **S. Levick and G. Melendez.** *Univ. of South Carolina Sch. of Med.*

30 **3.30** Mechanisms of Leukocyte and Platelet Recruitment in the Microvasculature during Chronic Angiotensin II Administration. **A. Yildirim and D. N. Granger.** *Lousisana State Univ. Hlth. Sci. Ctr., Shreveport.*

Board #

31 **3.31** Oxidative Stress and Inflammation Plays a Role in Cigarette Smoke Induced Unfavorable Effects of Cardiac Stem Cell Signaling and Functions in Rat Cardiac Stem Cells. **W. Sumanasekera, D. Tran and H. Tran.** *Sullivan Univ., Louisville and Univ. of Louisville.*

32 **3.32** Effect of a CXCR2 Antagonist on CXCL1, CXCL2 and CXCR2 Brain Expression and Infarct Volume After Stroke in C57Bl6 Mice. **C. Sobey, V. Brait, J. Rivera and G. Drummond.** *Monash Univ., Australia.*

33 **3.33** Homocysteine Decreased Differentiation of Splenic Regulatory T cell in Mouse. **J. Feng, Z. Zhou and X. Wang.** *Peking Univ. Hlth. Sci. Ctr., Beijing, People's Rep. of China.*

34 **3.34** Costimulatory Signaling Pathway Mediated Hyperhomocysteinaemia-induced Imbalance Between Treg and Teff in Mice. **X. Wang, K. Ma and J. Feng.** *Peking Univ. Hlth. Sci. Ctr., Beijing, People's Rep. of China.*

35 **3.35** Regulatory T cells/T-helper cell 17 Functional Imbalance in Patients with End Stage Kidney Disease: A Pivotal Link Between Microinflammation and Adverse Cardiovascular Events. **J. Zhang,** *The First Affiliated Hosp. of ChongQing Med. Univ., People's Rep. of China.*

36 **3.36** Interleukin-4 Mediates Hypoxia-induced Mitogenic Factor (HIMF/FIZZ1/ RELMα) induced Lung In-flammation and Pulmonary Microvascular Endothelial Cell Activation. **K. Kegan, Q. Su, D. Angelini, A. Myers and R. Johns.** *Johns Hopkins Med. Inst.*

37 **3.37** Stimulation of Alpha7 Nicotinic Acetylcholine Receptor Attenuates Inflammatory Response in Macrophages and Improves Survival in Experimental Model of Sepsis Through Heme Oxygenase-1 Induction. **K. C. Chang and K. Tsoyi.** *Gyeongsang Natl. Univ., People's Rep. of Korea.*

38 **3.38** Characterization of T Cells Infiltrating Perivascular Coronary Adipose Tissue in Atherosclerosis. **D. Ludew, K. Urbanski, R. Nosalski, P. Rudzinski, R. Korbut and T. J. Guzik.** *Jagiellonian Univ. Sch. of Med., Cracow, Poland.*

39 **3.39** The Mammalian STE20 Orthologue Links TLR Signaling to MAPKs and Plays a Crucial Role in Atherogenesis. **X. Song.** *Tufts Med. Ctr.*

Board #

40 **3.40** Ac-SDKP and endogenous peptide with cardiovascular and renal anti-inflammatory and anti-fibrotic properties **O. Carretero.** *Henry Ford Hosp. Detroit.*

Join us for the
APS Conference Opening Reception
Plaza South Grove at 6:00 PM
(Weather permitting)

Symposia II

4.0 BASIC ASPECTS OF IMMUNOLOGY II

Thurs., 7:00-8:35 PM, Westminster Ballroom III.

Chair: **Amy S. Major,** *Vanderbilt Univ. Sch. of Med.*

7:00 PM **4.1** Introduction. **Amy S. Major.** *Vanderbilt Univ. Sch. of Med.*

7:05 PM **4.2** Natural Killer Cells and Natural Killer T Cells. **Luc Van Kaer.** *Vanderbilt Univ. Sch. of Med.*

7:50 PM **4.3** Cell Interactions and Antigen Presentation. **Alexander Niessner.** *Med. Univ. of Vienna., Austria.*

Oral Presentations I

5.0 T CELLS AND CARDIOVASCULAR DISEASE

Thurs., 8:45-10:00 PM, Westminster Ballroom III.

Chairs: **Ernesto Schiffrin,** *McGill Univ., Canada.* **Brett Mitchell,** *Texas A&M Univ.*

8:45 PM **5.1** Introduction. **Ernesto Schiffrin.** *McGill Univ., Canada.*

8:50 PM **5.2** Induction of Regulatory T Cells Reduces Endothelial Dysfunction in a Mouse Model of Hypertension with Elevated Th17 Cells. **Brett Mitchell,** *Texas A&M Univ.***(8.11).**

9:05 PM **5.3** Adaptive Transfer of T-Regulatory Lymphocytes Attenuates Angiotensin II-Induced Hypertension and Vascular Injury. **Pierre Paradis.** *McGill Univ., Canada.* **(8.13).**

9:20 PM **5.4** Withdrawn.

9:35 PM **5.5** Protein Tyrosine Phosphatase 1B Deletion Increases Blood Pressure in Th2 Responsive Mice Only by Increasing Pro-inflammatory Cytokines Secretion. **Eric Belin de Chantemele.** *Med. Coll. of Georgia.* **(8.25).**

9:50 PM **5.6** Assessment of Indolamine 2, 3-Dioxygenase (IDO) and FoxP3+ Regulatory T Cells (FoxP3+ Tregs) During Early Phase of Renal Ischemia Reperfusion Injury.

Babak Baban. *Med. Coll. of Georgia.* **(8.17).**

FRIDAY, AUGUST 27, 2010

Symposia III

6.0 INFLAMMATION, THE VASCULAR WALL AND LESION FORMATION

Fri., 8:00 AM-11:35 AM, Westminster Ballroom III.

Chairs: **Goran K. Hansson,** *Karolinska Univ. Inst., Sweden.*
Eric Belin de Chantemele, *Med. Coll. of Georgia.*

8:00 AM **6.1** Introduction. **Goran K. Hansson.** *Karolinska Univ. Inst., Sweden.*

8:05 AM **6.2** Immunobiology of the Endothelium. **Jordan S. Pober.** *Yale Univ.*

8:35 AM **6.3** Innate Immunity and Atherosclerotic Lesion Formation. **Peter Libby.** *Brigham & Women's Hosp.*

9:05 AM **6.4** The Adaptive Immune Response and Atherosclerosis. **Klaus Ley.** *La Jolla Inst. For Allergy & Immunology.*

9:35 AM **6.5** Atherosclerosis and Autoimmune Diseases. **Amy S. Major.** *Vanderbilt Univ. Sch. of Med.*

10:05 AM Break.

10:30 AM **6.6** Co-stimulation and Atherosclerosis. **Andrew Lichtman.** *Brigham & Women's Hosp.*

11:00 AM **6.7** Immune Cell Recruitment and Atherosclerosis. **Elena Galkina.** *Eastern Virginia Med. Sch.*

Oral Presentations II

7.0 INFLAMMATION, VASCULAR FUNCTION AND ATHEROSCLEROSIS

Fri., 11:40 AM-12:40 PM, Westminster Ballroom III.

Chairs: **Virginia Miller,** *Mayo Clinic.*
Rayna Gonzales, *Univ. of Arizona.*

11:30 AM **7.1** Introduction. **Virginia Miller.** *Mayo Clinic.*

11:35 AM **7.2** Body Mass Index and Thrombogenic Factors in Newly Menopausal Women. **Muthuvel Jayachandran.** *Mayo Clinic.* **(3.26).**

11:50 AM **7.3** Soluble IL-6 Receptor Alpha is Negatively Associated with FMD in Women 20-50 years of Age. **Mariam Cortez-Cooper.** *Med. Coll. of Georgia.* **(3.4).**

12:05 PM **7.4** Withdrawn.

12:20 PM **7.5** FcγRIIb Influences Cardiovascular Disease and Modulates the Inflammatory Response of Dendritic Cells. **Yanice Mendez-Fernandez.** *Vanderbilt Univ. Sch. of Med.* **(3.12).**

Poster Session II

8.0 INFLAMMATION, METABOLIC SYNDROME AND HYPERTENSION

Fri., 12:45-2:30 PM, Westminster Ballroom IV.

Board #

1 **8.1** Potential Role of Indolamine 2, 3-Dioxygenase and Inflammatory Cytokines in Type 2 Diabetic Nephropathy: Effect of Chromium Picolinate. **M. Mozaffari, J. Liu and B. Baban.** *Med. Coll. of Georgia.*

2 **8.2** Accelerated Insulin Resistance and Nephritis in a Mouse Model of Lupus. **C. Gabriel, Y. Mendez-Fernandez, N. Wade, B. Stevenson and A. Major.** *Vanderbilt Univ. Sch. of Med.*

3 **8.3** Lysophosphatidylcholines with Eicosapentaenoyl or Docosahexaenoyl Group, and their Oxygenation Products Decrease Levels of LTC4 and Cytokines Such as TNF-alpha or IL-6 in Zymosan A-induced Peritonitis of Mice. **M. R. Kim and M-J. Kim.** *Chungnam Natl. Univ., South Korea.*

4 **8.4** Oral Administration of 2-docosahexaenoyl Lysophosphatidylcholine Reduces Levels of Inflammatory Mediators Such as TNF-alpha, IL-6 and LTC4. **D-E. Sok and N. D. Hung.** *Chungnam Natl. Univ., South Korea.*

5 **8.5** Iron Deprivation Ameliorates Glucose Tolerance Through Reduction of Oxidative Stress and Inflammation in Diabetic KKAy Mice. **S. Tajima, Y. Ikeda, N. Yamano, K. Tsuchiya, Y. Kihira, K. Ishizawa, K. Kawazoe, S. Tomita, K. Minakuchi and T. Tamaki.** *Univ. of Tokushima Grad. Sch. of Hlth. Biosciences, Japan.*

6 **8.6** Double Negative T cells in Perivascular Inflammation in Angiotensin II Dependent Hypertension. **T. Mikolajczyk, R. Nosalski, D. Ludew, P. Matusik, R. Korbut and T. J. Guzik.** *Jagiellonian Univ. Sch. of Med., Cracow, Poland.*

7 **8.7** Role of NKT Cells in the Development of Hypertension. **A. Vinh, T. Jansen, L. Van Kaer and D. Harrison.** *Emory Univ. Sch. of Med., Karolinska Univ. Hosp., Stockholm, Sweden, and Vanderbilt Univ.*

Board #

8 **8.8** The Induction of Th17 in DOCA-salt Rats is Prevented by Spironolactone. **C. Amador, J. P. Peña, M. González and L. Michea.** *Univ. of Chile, Santiago, Chile.*

9 **8.9** Salt is a Key Player in Angiotensin II-induced Kidney Injury as a Trigger Puller of Inflammation. **W. Kawarazaki, M. Nagase, S. Yoshida, M. Takeuchi, K. Isizawa, N. Ayuzawa, H. Kawarazaki and T. Fijita.** *Univ. of Tokyo Grad. Sch. of Med., Japan.*

10 **8.10** Aldosterone Induces Cellular Senescence in the Kidney via Mineralocorticoid Receptor/p21-dependent Pathway. **D. Nakano, Y-Y. Fan, H. Hitomi and A. Nishiyama.** *Kagawa Univ., Japan.*

11 **8.11** Induction of Regulatory T Cells Reduces Endothelial Dysfunction in a Mouse Model of Hypertension with Elevated Th17 Cells. **M. Quinn, V. Chiasson, K. Young and B. Mitchell.** *Texas A&M Univ.*

12 **8.12** Inhibition of the FK506 Target FKBP12 Increases Th17 Cell-Mediated Inflammation and Hypertension. **V. Chiasson, M. Quinn, K. Young and B. Mitchell.** *Texas A&M Univ.*

13 **8.13** Adaptive Transfer of T-Regulatory Lymphocytes Attenuates Angiotensin II-Induced Hypertension and Vascular Injury. **P. Paradis, T. Barhoumi, D. Kasal, L. Shbat, P. Laurant, M. Neves and E. Schiffrin.** *McGill Univ., Canada, Univ. d' Avignon, France and Univ. do Estado do Rio de Janeiro, Brazil.*

14 **8.14** Interleukin-10 Attenuates the Acute Pressor Response to a Low Dose of Angiotensin II. **J. Wilson, R. Duan and D. Lee.** *Howard Univ.*

15 **8.15** IL-10 Deficiency Augments Inflammation, Endothelial Dysfunction, and Blood Pressure in a Mouse Model of Gestational Hypertension. **P. Chatterjee, M. Quinn, V. Chiasson, K. Young and B. Mitchell.** *Texas A&M Univ.*

16 **8.16** Hypertension in Response to Placental Ischemia in Pregnant Rats is Associated with Increased CD4+ T Helper Cells: A Link in the Pathophysiology of Preeclampsia. **K. Wallace, S. Richards, A. Weimer, P. Dhillion, J. Martin and B. LaMarca.** *Univ. of Mississippi Med. Ctr.*

17 **8.17** Assessment of Indolamine 2, 3-Dioxygenase (IDO) and FoxP3+ Regulatory T Cells (FoxP3+ Tregs) During Early Phase of Renal Ischemia Reperfusion Injury. **B. Baban, A. El-Awady and M. Mozaffari.** *Med. Coll. of Georgia.*

18 **8.18** Role of Inflammation in Hypertension in Male and Female Spontaneously Hypertensive Rats. **A. Tipton and J. Sullivan.** *Med. Coll. of Georgia.*

19 **8.19** Bone Marrow Macrophages (MΦ) Infiltrate Mesenteric Arteries in DOCA-salt Hypertensive Rats. **L. Thang, R. Crawford, N. Kaminski and J. Galligan.** *Michigan State Univ.*

20 **8.20** TNF-α Promotes Hypertension and Renal Injury in the Chronic Inflammatory Disorder Systemic Lupus Erythematosus. **M. Ryan.** *Univ. of Mississippi Med. Ctr.*

21 **8.21** Age-associated Inflammation and Oxidative Stress Alter Renal Angiotensin II AT1 And Dopamine D1 Receptor Function and Contribute To Hypertension In Aging. **M. Asghar, G. Chugh and M. Lokhandwala.** *Univ. of Houston.*

22 **8.22** Upregulation of CCR2 and its Ligands CCL7 and CCL8 in the Vascular Wall During Hypertension in Mice. **J. P. Moore, K. Budzyn, C. G. Sobey and G. R. Drummond.** *Monash Univ., Australia.*

23 **8.23** A Role for AT1 receptors on T lymphocytes in Angiotensin II-Dependent Hypertension. **S. Crowley, Y-S. Song, R. Griffiths, Mi. Yan, M. Sparks, J. Burchette, D. Howell, P. Ruiz, T. Le and T. Coffman.** *Duke Univ., Univ. of Miami and Univ. of Virginia, Charlottesville.*

24 **8.24** Effects of Ang II–induced Hypertension on the Heart in Mice with Th1/Th2 Profiles. **N-E. Rhaleb, X-P. Yang, H. Peng, J. Xu, Y-H. Liu, P. Nakagawa, P. Leung and O. Carretero.** *Henry Ford Hosp., Detroit.*

25 **8.25** Protein Tyrosine Phosphatase 1B Deletion Increases Blood Pressure in Th2 Responsive Mice only by Increasing Pro-inflammatory Cytokines Secretion. **E. Belin de Chantemèle, J. Mintz, M. Marrero, M. Tremblay and D. Stepp.** *Med. Coll. of Georgia and McGill Univ., Canada.*

26 **8.26** Differences in Pulmonary Hypertensive Conduit Artery Material Property Changes of the Human, Calf and Rat. **S. Lammers, P. Kao, L. Tian, K. Hunter, Z. VanRheen, C. Lanning, J. Albeitz, S. Hofmeister, H. J. Qu, S. Miyamoto, T. Kulik, R. Shandas and K. Stenmark.** *Univ. of*

Board #

Colorado, Denver, Univ. of Colorado, Boulder and The Children's Hosp., Boston.

27 **8.27** Impact of Residual Stretch on Collagen Engagement in the Main Pulmonary Arteries of Healthy and Pulmonary Hypertensive Calves at Physiological Pressures. **L. Tian, S. Lammers, P. Kao, J. Albietz, K. Stenmark, K. Hunter, H. J. Qu and R. Shandas.***Univ. of Colorado, Boulder and Univ. of Colorado, Denver.*

28 **8.28** Immune Responses in the Lung to Second Hand Cigarette Smoke. **A. Kratzer, J. Salys, L. Shapiro and L. Tarasevicience-Stewart.** *Univ. of Colorado, Denver.*

29 **8.29** Ac-SDKP Reduces Cardiac Cross-Linked Collagen, NFκB Expression, Inflammation and Lysyl Oxidase Expression in Angiotensin II Induced Hypertension. **G. Gonzalez, Y-H. Liu, P. Nakagawa, T-D. Liao, X-P. Yang, N-E. Rhaleb and O. Carretero.** *Henry Ford Hosp., Detroit.*

30 **8.30** ETA Receptor Activation Increases Renal T Cells and Cellular Proliferation During Chronic Ang II Infusion Independent of Hypertension. **D. Pollock, K. Krishnan, E. Boesen and J. Pollock.** *Med. Coll. of Georgia.*

31 **8.31** Suppression of Angiotensin II Induced Hypertension with Adoptive Transfer of Regulatory T-lymphocytes. **S. Tawinwung, R. Vazquez, N. Larmonier, E. Slack, A. Kupsco and D. F. Larson.** *Univ. of Arizona, Tucson.*

32 **8.32** Adoptive Transfer of Hypertension with $CD4^+$ Lymphocytes. **R. Vazquez, S. Tawinwung, A. Kupsco and D. F. Larson.** *Univ. of Arizona, Tucson.*

33 **8.33** Interferon-gamma Receptor Knock-out Mice Develop Less Cardiac and Tubular Damage Despite Albuminuria in An-giotensin II-infused Mice. **L. Marko, V. Fokuhl, H. Kvakan, J-K. Park, F. Qadry, F. C. Luft, R. Dechend and D. N. Muller.** *Max-Delbruck Ctr. & Clinical Res. Ctr., Helios Clinic, Berlin, Germany and Med. Sch. of Hannover, Germany.*

Career Workshop

9.0 THE INS AND OUTS OF AUTHORSHIP

Fri., 5:30-6:30 PM, Westminster Ballroom III.

Chair: **Lacy A. Holowatz,** *Pennsylvania State Univ.*

5:30 PM **9.1** Introduction. **Lacy A. Holowatz.** *Pennsylvania State Univ.*

Symposia IV

10.0 INFLAMMATION AND THE METABOLIC SYNDROME

Fri., 7:00-8:05 PM, Westminster Ballroom III.

Chairs: **Tomasz Guzik,** *Jagiellonian Univ. Sch. of Med., Cracow, Poland.*
Paul Marvar, *Emory Univ. Sch. of Med.*

7:00 PM **10.1** Introduction. **Tomasz Guzik.** *Jagiellonian Univ. Sch. of Med., Cracow, Poland.*

7:05 PM **10.2** Obesity and Inflammation. **Tomasz Guzik.** *Jagiellonian Univ. Sch. of Med., Cracow, Poland.*

7:35 PM **10.3** Inability to Manage Gut Microbiota can Result in Chronic Low-Grade Inflammation that Results in Metabolic Syndrome. **Andrew Gewirtz.** *Emory Univ. Sch. of Med.*

Oral Presentations III

11.0 HYPERTENSION, RENAL DISEASE AND INFLAMMATION

Fri., 8:15-10:00 PM, Westminster Ballroom III.

Chairs: **Steven Crowley,** *Duke Univ.*
Grant Drummond, *Monash Univ., Australia.*

8:15 PM **11.1** Introduction. **Steven Crowley.** *Duke Univ.*

8:20 PM **11.2** Inhibition of the FK506 Target FKBP12 Increases Th17 Cell-Mediated Inflammation and Hypertension. **Brett Mitchell.** *Texas A&M Univ.* **(8.12).**

8:35 PM **11.3** Stress-induced Hypertension Promotes T Lymphocyte Activation and Vascular Inflammation. **Paul Marvar.** *Emory Univ. Sch. of Med.* **(3.28).**

8:50 PM **11.4** Upregulation of CCR2 and its Ligands CCL7 and CCL8 in the Vascular Wall During Hypertension in Mice. **Jeffrey P. Moore.** *Monash Univ., Australia.* **(8.22).**

9:05 PM **11.5** Hypertension in Response to Placental Ischemia in Pregnant Rats is Associated with Increased CD4+ T Helper Cells: A Link in the Pathophysiology of Preeclampsia. **Kedra Wallace.** *Univ. of Mississippi Med. Ctr.* **(8.16).**

9:20 PM **11.6** A Role for AT1 receptors on T lymphocytes in Angiotensin II-Dependent Hypertension. **Steven Crowley.** *Emory Univ. Sch. of Med.* **(8.23).**

9:35 PM **11.7** TNF-α Promotes Hypertension and Renal Injury in the Chronic Inflammatory Disorder Systemic Lupus Erythematosus. **Michael Ryan.** *Univ. of Mississippi Med. Ctr.* **(8.20).**

SATURDAY, AUGUST 28, 2010

Symposia V

12.0 INFLAMMATION, IMMUNITY, HYPERTENSION AND RENAL DISEASE

Sat., 8:30 AM-12:00 Noon, Westminster Ballroom III.

Chairs: **David M. Pollock,** *Med. Coll. of Georgia.* **Babbette LaMarca,** *Univ. of Mississippi Med. Ctr.*

8:30 AM **12.1** Introduction. **David M. Pollock.** *Med. Coll. of Georgia.*

8:35 AM **12.2** Immune Mechanisms in Salt-sensitive Hypertension. **Bernardo Rodriguez-Iturbe.** *Hosp. Univ. of Maracaibo & Univ. of Zulia, Venezuela.*

9:05 AM **12.3** T Regulatory Cells, Renal Disease and Hypertension. **Ernesto L. Schiffrin.** *McGill Univ., Canada.*

9:35 AM **12.4** IL-17 and Hypertension. **Meena Madhur.** *Emory Univ. Sch. of Med.*

10:05 AM Break.

10:30 AM **12.5** Immune Cells Mediate Sodium-sensitive Hypertension and Renal Injury in Rats. **David Mattson.** *Med. Coll. of Wisconsin.*

11:00 AM **12.6** Renal Inflammation and Hypertension. **Jennifer Pollock.** *Med. Coll. of Georgia.*

11:30 AM **12.7** Ac-SDKP and Endogenous Peptide with Cardiovascular and Renal Anti-inflammatory and Anti-fibrotic Properties. **Oscar Carretero.** *Henry Ford Univ., Detroit.* **(3.40).**

NOTES

2010 APS Conference
Inflammation, Immunity and Cardiovascular Disease

Abstracts of Invited and Contributed Presentations

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3.0 Cardiovascular Disease and Immunity…….12

4.0 Basic Aspects of Immunology II……..19

6.0 Inflammation, The Vascular Wall and Lesion Formation……19

8.0 Inflammation, Metabolic Syndrome and Hypertension.……...20

10.0 Inflammation and the Metabolic Syndrome ……....…….26

12.0 Inflammation, Immunity, Hypertension and Renal Disease……..26

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1.0: PLENARY LECTURE

1.2

ATHEROSCLEROSIS AS AN INFLAMMATORY DISEASE: A PARADIGM SHIFT IN CARDIOVASCULAR MEDICINE

Goran Hansson[1]
[1]Center for Molecular Med., Karolinska Inst., CMM L8:03, Karolinska Univ. Hospital, Stockholm, SE-17176, Sweden.

Atherosclerosis develops as a response to accumulation of cholesterol in the artery wall. The properties of this response was debated for years, until immunohistochemical analysis identified a significant infiltrate of immune-inflammatory cells and a vascular – immune cell crosstalk mediated by cytokines and cell-surface molecules was discovered. All these findings led to the new paradigm that atherosclerosis is an inflammatory disease. Today, inflammatory markers are used for prediction and monitoring of coronary heart disease. Further insights into the immunopathogenesis of atherosclerosis may generate new approaches to therapy. Low-density lipoprotein (LDL) acts as an autoantigen in atherosclerosis, with CD4+ T cells recognizing components of the LDL particle. Our recent studies show that the T cell recognizes native peptide sequences in LDL protein (apoB100), rather than oxidatively modified ones. While mild oxidation of the LDL particle increases its uptake into antigen-presenting cells, more profound LDL oxidation destroys its immunoreactivity. A limited set of rearranged T cell receptors are employed for the recognition of apoB100 motifs; inhibition of this recognition by blocking antibodies to these receptors inhibit the development of atherosclerosis. These findings challenge the current view that oxidation breaks immunological tolerance.

2.0: BASIC ASPECTS OF IMMUNOLOGY

2.2

BASIC ASPECTS OF T CELL FUNCTION

Ross Kedl[1]
[1]Dept. of Immunology, NJMRC, Univ. of Colorado, 1400 Jackson St., Denver, CO, 80206

Though it was not known at the time, the importance of T cells and of T cell memory was probably first demonstrated experimentally in the 18th century by Edward Jenner and his successful, if somewhat ethically questionable, initial vaccination experiments against smallpox. Many years and experiments later, we have a good deal of information regarding the magnitude, potency, and duration of T cell responses and the formation of T cell memory in response to a variety of experimental and clinical entities. During normal primary T cell responses, naïve T cells are induced to proliferate and differentiate, giving rise first to an effector pool and then to a long lived memory population. A variety of T cell subsets exist, each with functional advantages in various infectious or vaccination settings. This talk will concentrate on an overview of T cells, their generation, their attributes, and their clinical importance.

2.3

HOW TO MEASURE AND STUDY IMMUNE RESPONSES

Holden T. Maecker, Ph.D.
Inst. for Immunity, Transplantation, and Infection. Stanford Univ., Stanford, CA 94305.

Immune assessment in clinical trials has become a focus of much recent attention, as new assays have shown promise for identifying biomarkers of disease diagnosis, prognosis, and therapeutic response. Many of these assays make use of flow cytometry, a platform that is still in need of some optimization and standardization for clinical use. In this presentation, basic principles of flow cytometry will be reviewed, including instrument setup, optical spillover, and compensation. Several flow cytometric assays of immune function, including intracellular cytokine staining, phosphoepitope flow cytometry, and CFSE assays, will be introduced, along with key variables to consider when implementing them as immune assessment tools. Stimulation conditions, sample processing, and data acquisition and analysis will be briefly described for each assay. Standardized implementation of these assays will be important to maximize the value of data obtained in clinical trials.

2.6

THE IDENTIFICATION AND CHARACTERIZATION OF SUBPOPULATIONS OF MONCOYTES AND MACROPHAGES DURING INFLAMMATION AND INFECTION

David Mosser[1]
1Cell Biology and Molecular Genetics, Univ. of Maryland, Rm. 3102 BRB, College Park, MD, 20742.

During inflammation, monocytes leave the blood and develop into mature tissue macrophages. We and others have identified a subset of monocytes that is particularly adept at exiting the blood in response to inflammatory signals. These monocytes appear to be important innate immune effector cells that produce high levels of superoxide and can kill a variety of pathogens. There remain questions about the importance of the other monocyte subsets, and whether specific monocyte subpopulations are predetermined to differentiate into specific macrophages or dendritic cells in tissue. Tissue macrophages can respond to a variety of endogenous and exogenous signals and change their physiology in response to locally-produced cues. The best-characterized of these macrophages is the classically activated macrophage, which has potent antimicrobial activity. These macrophages are prodigious secretory cells that can contribute to autoimmune pathology. Macrophages can also develop into cells that produce components of the extracellular matrix, and these cells are thought to be important in wound healing. Other macrophages can produce high levels of anti-inflammatory cytokines and these cells play an important role in down-regulating immune responses. Pathogens have developed ways to exploit these alternative stages of macrophage activation as safe havens for intracellular growth.

3.0: CARDIOVASCULAR DISEASE AND IMMUNITY

3.1 Withdrawn.

3.2

PSYCHOLOGICAL STRESS, VASCULAR INFLAMMATORY & REMODELING PROTEINS: EVIDENCE OF "OUTSIDE-IN" INFLAMMATION?

Monika Fleshner[1], Robert Thompson[1]
[1]Integrative Physiology, Univ. of Colorado, 1725 Pleasant St., Clare Small Bldg., Boulder, CO, 80309-0354.

Psychological stressors are associated with reductions in cardiovascular health. The mechanism(s) for such effects are unknown. Using contextual conditioned fear in male F344 rats, we investigated the impact of repeated activation of the stress response by a psychological stressor on vascular biology. Our results were that conditioned fear repeatedly activates the sympathetic nervous system to elevate blood pressure; and exposure to the conditioned fear environment modulates arterial vascular inflammatory and remodeling proteins in a regionally selective fashion. Specifically, 20 days of stress elevates inflammatory cytokines and chemokines in perivascular fat (PV fat) and adventitia but not in "naked" artery or the blood. Repeated conditioned fear stress also perturbs remodeling protein balance: decreasing tissue inhibitor of the metalloproteinase (TIMP1) and increasing activation of matrix metalloproteinase (MMP2) in arterial tissues. We hypothesize, therefore, that repeated exposure to a conditioned fear stressor elevates inflammatory proteins initially in the outer layers (PV fat & adventitia), but not in the inner layers (media & intima) of the vasculature or in the blood. Resultant inflammatory proteins then modulate remodeling proteins placing the vasculature in a pathological remodeling state. Such changes are demonstrative of an "outside-in" conceptualization of vascular inflammation and could mediate stress-associated increases in vascular disease risk. NIH-RO1AI-057797

3.3

ACTIVATION OF PROTEIN PHOSPHATASE TYPE 2A FACILITATES ITS BINDING TO TIGHT JUNCTION PROTEINS AND CAUSES ENDOTHELIAL BARRIER DYS-FUNCTION

Feng Wu[1], John X. Wilson[1]
[1]Exercise and Nutrition Sciences, Univ. at Buffalo, 3435 Main St., UB South Campus, Buffalo, NY, 14214.

Our previous studies showed that nitration of protein phosphatase type 2A (PP2A) catalytic subunit causes PP2A activation and endothelial barrier dysfunction. The present study elucidated the mechanism by which activation of PP2A disrupts the endothelial barrier in primary microvascular endothelial cell cultures. We observed that exposure of cell monolayers to lipopolysaccharide (LPS) and interferon-γ (IFNγ) for 24 h increased inducible nitric oxide synthase (NOS) expression, nitric oxide production, PP2A activity and paracellular permeability to albumin. The increases in PP2A activity and paracellular permeability were prevented by the NOS inhibitor L-NAME. The PP2A inhibitor okadaic acid had no effect on iNOS expression and NO production but nevertheless attenuated the permeability increase. Stimulation of cells with LPS+IFNγ did not affect total protein levels of the tight junction proteins ZO-1 and occludin. Western blot analysis of PP2A catalytic subunit immunoprecipitates revealed that LPS+IFNγ stimulated increased binding of the catalytic subunit to ZO-1 and occludin. Further, this stimulation of binding was prevented by L-NAME and okadaic acid. We conclude that NOS-dependent PP2A activation facilitates the binding of PP2A catalytic subunit to ZO-1 and occludin and, by dephosphorylating these tight junction proteins, increases paracellular permeability.

3.4

SOLUBLE IL-6 RECEPTOR ALPHA IS NEGATIVELY ASSOCIATED WITH FMD IN WOMEN 20-50 YEARS OF AGE

Miriam Cortez-Cooper[1], Eric Meaders[1], Judith Stallings[1], Sara Haddow[1], Barbara Kraj[1], Gloria Sloan[1], Anthony Mulloy[2], Kevin McCully[3], Joseph Cannon[1]
[1]School of Allied Health Sciences, Med. College of Georgia, 987 Sebastian Way, Augusta, GA, 30912, [2]School of Medicine, Med. College of Georgia, 1120 15th St., Augusta, GA, 30912, [3]Kinesiology, Univ. of Georgia, 115 Ramsey, Athens, GA, 30602.

The risk for coronary artery disease in women increases during perimenopause when follicle-stimulating hormone (FSH) concentration is rising. FSH influences proatherogenic cytokine secretion from mononuclear cells (MNC).We hypothesized that FSH-stimulated cytokine secretion and/or receptor expression

would correlate with endothelial dysfunction (ED). Healthy women, 20-50 years of age (n = 36) were tested in the follicular phase of their menstrual cycles. Circulating concentrations of IL-1β, TNFα, IL-6, their soluble receptors, sex hormones and traditional CVD risk factors were measured. MNC were isolated for in vitro analysis of basal and FSH-stimulated cytokine secretion, cytokine receptor expression and soluble receptor shedding. ED was assessed by brachial artery flow-mediated dilation (FMD). Plasma soluble interleukin-6 receptor alpha (sIL-6R□) concentrations correlated with sIL-6Rα secretion rates in vitro (R = 0.38, P = 0.045), and the secretion rates were inversely related to plasma HDL (b = -0.52) and positively related to oxidized LDL (b = 0.40, P = 0.01), but not FSH. The main finding was that plasma sIL-6Rα was negatively correlated with FMD (R = -0.41, P = 0.02) independent of age and other CVD risk factors. These data are consistent with the trans-signaling mechanism of sIL-6R□ that enables endothelial cells and vascular smooth muscle cells to respond to IL-6, an inhibitor of endothelium-dependent vascular relaxation. Funded by NIH grant AG027714.

3.5

INTERLEUKIN-1 RECEPTOR TYPE I EXPRESSION IS RELATED TO LUMBAR BONE MINERAL DENSITY AND INTIMA-MEDIA THICKNESS IN ADULT WOMEN

Joseph Cannon[1], Miriam Cortez-Cooper[1], Eric Meaders[1], Judith Stallings[1], Sara Haddow[1], Barbara Kraj[1], Gloria Sloan[1], Kevin McCully[2], Anthony Mulloy[3]
[1]Allied Health Sciences, Med. College of Georgia, 1120 15th St., Augusta, GA, 30912, [2]Kinesiology, Univ. of Georgia, 330 River Rd., Athens, GA, 30602, [3]Medicine, Med. College of Georgia, 1120 15th St., Augusta, GA, 30912.

Diminished bone mineral density (BMD) is often accompanied by increased carotid intima-media thickness (IMT) due to atherosclerosis and vascular calcification. Since cytokine-mediated mechanisms have been implicated in bone resorption and independently implicated in atherosclerosis, we sought to determine if any aspects of cytokine status were related to both BMD and IMT within the same subjects. Thirty six healthy women (20-50 years old) were assessed for BMD by dual-energy X-ray absorptiometry, IMT by ultrasonography, circulating IL-1□, TNF□, IL-6 and soluble receptor concentrations by immunoassay, and expression of cytokine receptors on monocytes by flow cytometry. BMD at the lumbar spine was inversely related to IMT (R = -0.54, P = 0.001). Monocyte Expression of interleukin-1 receptor type I (IL-1RI) was positively related to IMT (R = 0.41, P = 0.03) and negatively related to BMD (-0.42, P = 0.02). Thus high expression of IL-1RI may indicate a phenotype of monocyte that has deleterious effects at both the arterial wall and bone. Within the arterial wall, activated monocytes can promote neointimal hyperplasia and can transform into foam cells. Monocytes can also develop into bone-resorbing osteoclasts under the influence of M-CSF, RANK-ligand and osteoprotegerin, but the serum concentrations of these factors were not associated with expression of IL-1RI. Supported by NIH grant AG027714.

3.6

C-REACTIVE PROTEIN AND ENDOTHELIAL FIBRINOLYTIC DYSFUNCTION

Brian Weil1, Kyle Diehl1, Jared Greiner1, Brian Stauffer2, Christopher DeSouza1
[1]Integrative Physiology, Univ. of Colorado at Boulder, 354 UCB, Boulder, CO, 80309, [2]Dept. of Medicine, Univ. of Colorado Denver, 12700 E. 19th Avenue, Aurora, CO, 80045.

Objective: We tested the hypothesis that elevated plasma C-reactive protein (CRP) levels are associated with impaired vascular endothelial release of tissue-type plasminogen activator (t-PA), the primary activator of the endogenous fibrinolytic system. Methods: Fifty-four adults were stratified based on their CRP levels according to AHA/CDC guidelines: 24 with CRP < 1.0 mg/L (low CRP; 18M/6F; age: 55±2 yr), 18 with CRP 1.0-3.0 mg/L (moderate CRP; 8M/10F; age: 58±2 yr), and 12 with CRP > 3.0 mg/L (high CRP; 7M/5F; age: 56±2 yr). Net endothelial release of t-PA was determined, *in vivo*, in response to intrabrachial infusions of bradykinin (BK: 125-500 ng/min) and sodium nitroprusside (SNP: 2.0-8.0 μg/min). Results: Net endothelial t-PA release to BK was significantly lower (~30%) in the high CRP (from 0.32±0.5 to 38.9±6.0 ng/100 mL tissue/min) and moderate CRP (-0.05±0.4 to 39.3±5.2 ng/100 mL tissue/min) groups compared with the low CRP group (0.42±0.9 to 61.8±5.2 ng/100 mL tissue/min). Interestingly, the impairment in endothelial t-PA release was similar between the high and moderate CRP groups. Furthermore, there was an inverse relation between CRP and peak t-PA release to BK (r=-0.38, P<0.01). Conclusions: Elevated CRP levels are associated with impaired endothelial t-PA release, suggesting that inflammation is associated with endothelial fibrinolytic dysfunction. This impairment may underlie the increased atherothrombotic risk associated with elevated levels of CRP.

Don't forget to visit the posters and exhibits each day
Lunch will be served

3.7

IMPAIRED ENDOTHELIUM-DEPENDENT VASODILATION WITH HIV-1 INFECTION IS DUE TO OXIDATIVE STRESS

Brian Weil[1], Brian Stauffer[2], Michael Mestek[1], Jared Greiner[1], Elizabeth Connick[3], Christopher DeSouza[1]
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Objective: The aim of the present study was to determine if impairment in endothelium-dependent vasodilation in HIV-1-seropositive adults is due, at least in part, to oxidative stress. Methods: Ten HIV-1-seronegative (7M/3F; age: 35±3 yr) and 10 treatment naïve HIV-1-seropositive (7M/3F; age: 35±2 yr) adults were studied. Forearm blood flow (FBF) responses to intrabrachial infusions of acetylcholine (ACh: 8.0-32.0 μg/min) and sodium nitroprusside (SNP: 2.0-8.0 μg/min) were measured by venous occlusion plethysmography. FBF responses to ACh were repeated with a co-infusion of the antioxidant vitamin C (24 mg/min). Results: FBF responses to ACh were ~25% lower (P<0.05) in the HIV-1-seropositive (from 4.5±0.4 to 12.2±1.3 mL/100mL tissue/min) compared with HIV-1-seronegative (4.6±0.4 to 15.4±1.3 mL/100mL tissue/min) adults. FBF responses to SNP were not different between the groups. Co-infusion of vitamin C significantly increased (~40%) FBF in response to ACh in the HIV-1-seropositive (6.4±0.5 to 15.8±1.4 mL/100mL tissue/min) but not HIV-1-seronegative (6.8±0.9 to 16.3±1.6 mL/100mL tissue/min) adults. Interestingly, the difference in ACh-mediated vasodilation observed between groups was abolished by co-infusion of vitamin C. Conclusions: Oxidative stress contributes to endothelial vasodilator dysfunction in HIV-1-seropositive treatment naïve adults. Oxidative stress-induced endothelial dysfunction may underlie the increased cardiovascular risk observed in this population.

3.8

INFLUENCE OF HIV-1 INFECTION AND HAART THERAPY ON ENDOTHELIAL PROGENITOR CELL NUMBER AND FUNCTION

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Objective: The aim of the present study was to test the hypothesis that circulating endothelial progenitor cell (EPC) number and in vitro colony-forming capacity are reduced in treatment-naïve and HAART-treated HIV-1-seropositive men. Methods: 20 HIV-1-seronegative (age: 42±2 yr), 10 HIV-1-seropositive treatment-naïve (36±2 yr) and 10 HIV-1-seropositive men receiving HAART therapy (efavirenz, tenofovir, emtricitabine; 42±3 yr) were studied. Circulating EPC number was assessed by flow cytometric analysis of the percentage of peripheral blood mononuclear cells staining for CD45-, CD34, VEGFR-2 and CD133 antigens. EPC colony-forming capacity was measured in vitro using a colony-forming unit (CFU) assay. Results: EPC number was ~60% lower (P<0.05) in HIV-1-seropositive treatment-naïve (0.0005±0.0002%) and HIV-1-seropositive men on HAART (0.0005±0.0002%) compared with HIV-1-seronegative men (0.0014±0.0002%). EPC colony-forming capacity was significantly lower in cells from HIV-1-seropositive treatment-naïve (1±1 CFU) and HAART-treated men (3±3 CFU) compared with HIV-1-seronegative (14±3 CFU) men. There were no differences in EPC number or colony-forming capacity between the treatment naïve and HAART-treated HIV-1-seropositive adults. Conclusions: HIV-1 infection, regardless of treatment status, is associated with diminished EPC number and function. Numerical and functional impairment in circulating EPCs may contribute to HIV-1-related cardiovascular risk.

3.9

HIV-1 VIRAL PROTEINS GP120 AND TAT IMPAIR ENDOTHELIAL PROGENITOR CELL FUNCTION

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Objective: We tested the hypothesis that the HIV-1 viral proteins gp120 and Tat impair endothelial progenitor cell (EPC) function. Methods: EPCs were isolated from 12 healthy adults and treated with gp120Bal (R5; 100 ng/mL), gp120Lav (X4; 100 ng/mL) or Tat (500 ng/mL). EPC colony-forming capacity was determined by colony-forming unit (CFU) assay; migration by Boyden chamber; and VEGF and G-CSF release by ELISA. Results: CFU number was reduced (P<0.05) following treatment with R5 (28±7 CFU) and X4 (25±8 CFU) compared with untreated control (39±10 CFU). Tat marginally reduced CFU number (29±5 CFU) compared with control. Migration to SDF-1 was ~10% lower than control in EPCs treated with R5 (982±85 vs. 1132±112 AU; P<0.05), but not X4 (1062±114 AU). Tat produced a 15% reduction in EPC migration (1018±80 vs. 1190±102 AU;

P<0.05). R5 (35.1±6.2 pg/mL) and X4 (33.5±5.4 pg/mL) did not alter VEGF release compared with control (37.3±8.2 pg/mL), but caused a 15% reduction in the release of G-CSF (R5: 49.5±5.8 pg/mL; X4: 50.4±5.6 pg/mL; control: 58.7±7.3 pg/mL). Tat caused a 5-fold increase in VEGF release (240.2±67.0 vs. 49.7±18.2 pg/mL; P<0.05), but did not affect G-CSF release (128.4±28.5 vs. 94.9±16.4 pg/mL). Heat-inactivated gp120 and Tat did not affect EPC function. Conclusions: Overall, HIV-1 gp120 and Tat can have a negative effect on EPC function. This represents a novel mechanism that may underlie the increased cardiovascular risk observed in HIV-1 seropositive adults.

3.10

CD31+ T CELLS, ENDOTHELIAL FUNCTION AND CARDIO-VASCULAR DISEASE RISK

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Deficits in endothelial cell repair mechanisms are thought to contribute to the etiology of endothelial dysfunction and, subsequently, cardiovascular disease (CVD). CD31+ T cells or so-called "angiogenic T cells" are a newly defined T cell subset that exhibit favorable vascular qualities and show a strong negative relation with atherosclerotic disease severity. Despite growing evidence that CD31+ T cells are important for vascular homeostasis, it is currently unknown if CD31+ T cell number and function are related with endothelial function and CVD risk. To address this question, we studied 24 healthy adult men (ages: 21-70). Endothelial function was assessed by forearm blood flow (FBF) response to intra-arterial infusion of acetylcholine (ACh) and CVD risk was estimated by Framingham risk score (FRS). CD31+ T cell number was determined by fluorescence-activated cell sorting and magnetic-activated cell sorting was used to isolate CD31+ T cells for Boyden chamber migration. No relation was observed between CD31+ T cell number and FBF to ACh or FRS. However, CD31+ T cell migration to vascular endothelial growth factor and stromal cell-derived factor-1α had a significant positive correlation with FBF (r=0.37 and r=0.43) and an inverse relation with FRS (r=-0.50 and r=-0.52). These results extend previous studies by demonstrating that CD31+ T cell function, but not number, is associated with in vivo endothelial function and CVD risk in healthy adult men.

3.11

DELETION OF MURINE LDL RECEPTOR RELATED PROTEIN IN GRANULOCYTES AND MONOCYTES CAUSES DECREASED ACTIVATION OF INVARIANT NATURAL KILLER T CELLS

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Introduction: Activation of Invariant Natural Killer T Cells (iNKT) has been shown to be pro-atherogenic. It has also been found that apoE and its receptor (LDLr) plays a role in glycolipid uptake and iNKT activation. A different receptor, LDL receptor related protein (LRP), shares LDLr homology and ability to bind apoE. LRP is constitutively present in antigen presenting cells (APCs) such as macrophages (MΦs) and dendritic cells (DCs). Although lipoprotein uptake mediated by LRP has been characterized, the role LRP plays in antigen presentation, processing and activation of iNKT cells is unknown. Objective: to examine the effect decreased LRP expression has on APCs and activation of iNKT cells. Methods: Using mice with the LRP allele flanked by loxP sites ($LRP^{flox/flox}$) and expressed the cre recombinase under the control of the lysozyme promoter, which targets MΦs and DCs, we examined the activation of iNKT cells in response to glycolipid antigen. Results: We found decreased expression of LRP on APCs results in modification of iNKT cell activation by decreasing cytokine secretion. A similar cytokine profile is observed when purified LRP deficient $CD11c^{+}$DCs are used to stimulate iNKT cell hybridomas. Intracellular cytokine staining shows iNKT cells from $LRP^{flox/flox}$ mice have a two-fold reduction in IFNG production. Conclusion: We have found that LRP plays a role in activation of iNKT cells. Thus it is possible that given its ligand diversity, LRP represents a bridge between lipid metabolism and host defense. In addition, understanding how conventional members of lipoprotein uptake pathways modulate iNKT cell function is essential to determine how iNKT cell based therapies can be affected by dyslipidemia.

3.12

FCYRIIB INFLUENCES CARDIOVASCULAR DISEASE AND MODULATES THE INFLAMMATORY RESPONSE OF DENDRITIC CELLS

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Atherosclerosis is now commonly accepted as an inflammatory disease involving both innate and adaptive immunity. In particular, it has been shown that B cells and/or antibodies have a protective role against atherosclerosis. One way antibodies can influence inflammatory responses is by interacting with various Fcγ receptors on the surface of antigen presenting cells. To determine the importance of the low affinity inhibitory FcγRIIb in modulating the adaptive immune response to hyperlipidemia, we generated FcγRIIb-deficient mice on the apoE-deficient background. In this study, we report that immunological changes, rather than changes in lipid levels, result in exacerbated atherosclerosis in the absence of FcγRIIb. Specifically, we show that dendritic cells lacking FcγRIIb expression have an increased inflammatory response via TLR4 that results in increased levels of IL-23 and IL-1β, correlating with elevated levels of IL-17 and IL-23 in the aorta. Our findings provide evidence to support the hypothesis that antibodies against oxLDL partially protect against atherosclerosis by conveying inhibitory signals via the FcγRIIb that downregulate pro-inflammatory signaling. These data are the first to describe a significant in vivo effect for FcγRIIb in dendritic cell modulation and atherogenesis in apoE-/- mice. This work was partially supported by an Irvington Postdoctoral Fellowship of the Cancer Research Institute.

3.13

HIGHER WHITE BLOOD CELL COUNT IS ASSOCIATED WITH ENHANCED ENDOTHELIN-1 SYSTEM ACTIVITY IN ADULT HUMANS

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Objective: Elevated white blood cell (WBC) count is associated with impaired endothelium-dependent vasodilation and increased cardiovascular disease risk. Endothelin (ET)-1 is a potent vasoconstrictor peptide with proatherogenic effects. The influence of WBC count on ET-1 vasoconstrictor activity is currently unknown. We determined whether elevated WBC count is associated with enhanced ET-1 system activity. Methods: 34 healthy adults were studied: 17 with WBC count < 5.0x109cells/L (low WBC: 4.4±0.2; 9M/8F; age: 53±2yr) and 17 with WBC count > 5.0x109cells/L (higher WBC: 6.4±0.3; 10M/7F; age: 54±3yr). Forearm blood flow (FBF) responses to intrabrachial infusion of ET-1 (5 pmol/min; 20 min) and selective ETA receptor blockade (BQ-123; 100 nmol/min; 60 min) were measured by venous occlusion plethysmography. Results: The vasoconstrictor response to ET-1 was significantly blunted (~60%) in the higher compared with low WBC group. In response to BQ-123, resting FBF increased (~20%; p<0.05) in the higher WBC group; but was unchanged in the low WBC group. Furthermore, WBC count was inversely related to FBF responses to ET-1 (r=-0.43, p<0.05) and positively correlated with FBF responses to BQ-123 (r=0.41, p<0.05). Conclusions: WBC count > 5.0x109cells/L is associated with enhanced ET-1-mediated vasoconstrictor tone. ET-1 system activity may be a contributing mechanism linking WBC count with impaired endothelium-dependent vasodilation and increased cardiovascular disease risk.

3.14 Withdrawn.

3.15

DISTURBED FLOW INDUCES ACUTE AND DYNAMIC INFLAMMATION LEADING TO RAPID ATHEROSCLEROSIS IN MOUSE CAROTID ARTERY

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Recently, we have shown that disturbed flow caused by partial ligation of the carotid artery induces atherosclerosis within 2 weeks in ApoE-/- mice on high fat diet (HFD), demonstrating a direct causal relationship between flow and atherosclerosis. While inflammation is known to be critical in atherosclerosis, the precise kinetics of immune cell recruitment in response to disturbed flow is unknown. We hypothesize that disturbed flow induces dynamic infiltration of distinct subsets of innate and adaptive immune cells into the vessel wall. We developed an expanded 10 color 14 marker FACS panel to identify distinct subsets of leukocytes. ApoE-/- mice underwent partial left carotid artery (LCA) ligation and were fed HFD for 4, 7, 14, 21, and 28 days. LCA and control non-ligated right carotid arteries (RCA) were excised, the cells extracted and phenotyped using the expanded FACS panel. Leukocyte infiltration into the LCA was observed as early as day 4, and peaked at day 7 for all cell types except for B-cells, which showed a delayed peak at day 21. DC to macrophage ratio was 1.43 at day 4, but dramatically dropped to 0.46 by day 7. Unexpectedly, the majority of DCs had an anti-inflammatory CD8a- CD11b+ phenotype. Sham-ligated LCAs were comparable to RCAs, showing no significant leukocyte infiltration. Our data demonstrate disturbed flow induces dynamic inflammatory cell infiltration leading to atherosclerosis in this hypercholesterolemic mouse model. Funded by NIH grant to HJ.

3.16

APOE REDUCES ATHEROSCLEROSIS INDEPENDENTLY OF LOWERING PLASMA CHOLESTEROL BY DECREASING INFLAMMATION IN CIRCULATING LEUKOCYTES AND VASCULAR ENDOTHELIUM

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We investigated atheroprotective properties of apoE beyond its ability to lower plasma cholesterol. We developed and studied mice with spontaneous hypercholesterolemia in the presence and absence of apoE: Hypomorphic apoE mice deficient in low-density lipoprotein receptor expression (Apoeh/hLdlr–/–) were compared to Apoe–/–Ldlr–/– mice. Despite similar plasma cholesterol levels (597.5±24.3 mg/dl versus 662.4±26.4 mg/dl, respectively), Apoeh/hLdlr–/–mice developed ~4 fold less oil red O and ~3 fold less macrophage-positive aortic surface area than Apoe–/–Ldlr–/–mice by five months of age. HDL isolated from Apoeh/hLdlr–/– mice were ~3 fold more potent at promoting cellular cholesterol efflux than HDL isolated from Apoe–/–Ldlr–/– mice. In parallel, we found an up-regulation of LXRα and ABCG-1 in lesional macrophages of Apoeh/hLdlr–/– mice isolated by laser capture micro-dissection. Using flow cytometry, we revealed increased intracellular lipid in circulating monocytes of Apoe–/–Ldlr–/–. This increase in intracellular lipid was associated with an increased expression of PECAM-1, CD11b and L-selectin. Lastly, we identified elevated levels of PECAM-1 and JAM-A in preparations of enface aortic endothelium of Apoe–/–Ldlr–/– mice using confocal microscopy. Taken together, our results indicate that apoE reduces atherosclerosis in the setting of hypercholesterolemia by reducing cellular lipid accumulation and inflammation in circulating leukocytes and endothelial activation.

3.17

THE LUPUS SUSCEPTIBILITY LOCUS *SLE1* BOTH SINGULARLY AND IN CONCERT WITH *SLE3* DOES NOT CONTRIBUTE TO THE ACCELERATION OF ATHEROSCLEROSIS IN LDLR$^{-/-}$ MICE

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Persons with the autoimmune disease systemic lupus erythematosus (SLE) have an increased risk for cardiovascular disease (CVD). Studies in our laboratory have focused on determining the contribution of each lupus susceptibility locus to the development of accelerated CVD in our SLE susceptible LDLr.Sle1.2.3 bone marrow chimera model. In this study, we hypothesized that Sle1 and Sle3, which leads to a break in tolerance and T cell hyperactivity, respectively, could promote accelerated CVD progression when combined. To test this, we transferred control (B6) or experimental (B6.Sle1, B6.Sle3, or B6.Sle1.3) bone marrow into lethally irradiated low density lipoprotein deficient (LDLr-/-) mice and placed them on Western diet sixteen weeks after transplantation. All three experimental groups had decreased serum cholesterol and no difference in serum triglyceride compared to LDLr.B6 mice. Autoantibody titer assessment showed that LDLr.Sle1.3 had significantly increased anti-dsDNA titers compared to LDLr.B6 mice. Flow cytometric analyses of splenocyte populations revealed increased T cell activation (CD4+CD69+), increased Th17 cells and decreased regulatory T cells in the experimental groups, specifically in B6.Sle1 and B6.Sle1.3 mice. However, oil-red-O staining revealed decreased lesion area in the experimental groups compared to control mice. These data present an interesting finding in which the combination of Sle1 and Sle3 may have disparate and unlikely effects on the progression of SLE-mediated atherosclerosis.

3.18

STIMULATION OF GTP CYCLOHYDROLASE I BY PHOSPHORYLATION UPON T CELL ACTIVATION

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Recently, we found that phosphorylation of GTP cyclohydrolase-1 (GTPCH-1), the rate-limiting enzyme for BH4 biosynthesis, increases its activity and BH4 levels in endothelial cells. To determine if this occurs in non-endothelial cells, we stimulated T cells both in vitro and in vivo. Naïve T cells had nearly undetectable levels of BH4 and minimal expression of either endothelial or inducible NOS. Anti-CD3 stimulation robustly induced T cell eNOS and iNOS and increased biopterin from near undetectable levels to 5.4 pMol/mg protein. Western blots showed that this was associated with GTPCH-1 phosphorylation at S72. Inhibition of GTPCH-1 with diaminohdroxypyimidine (DAHP, 500 µm) prevented T cell BH4 accumulation and increased T cell superoxide produciton, which was dependent on uncoupling of both eNOS and iNOS. GTPCH-1 inhibition also promoted TH2 polarization in memory CD4 cells. OVA immunization in vivo confirmed a marked increase in T cell BH4. Thus, T cell activation is associated with GTPCH-1 phosphorylation, which increases GTPCH-1 activity and biopterin production. In inflammatory settings where BH4 oxidation is favored, this could modulate T cell function. This study was supported by NIH grants PO1 HL 58000 and RO1 HL 39006.

3.19

ROLE OF NOX2 IN MOUSE HINDLIMB COLLATERAL GROWTH

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A role for NADPH oxidase 2 (Nox2) is established in atherosclerotic plaque and neo-intimal formation, and hypoxia-induced progenitor cell mobilization and neovascularization. But its role in collateral artery enlargement, the primary compensation to arterial occlusion, is unknown. Nox2-/- mice were used with a novel method of identifying hindlimb collaterals to determine if Nox2 mediates collateral growth induced by femoral artery excision. Greater ischemic injury occurred in Nox2 -/- than WT mice as evidenced by necrotic and regenerated muscle fibers and lipid and collagen deposition. The diameters of primary collaterals identified with intravital microscopy during Microfil injection were enlarged to a similar extent in WT and Nox2 mice. Intimal cells in collateral cross sections were increased in number in both strains and were CD31 positive and CD45 negative. The data demonstrate that injury in response to the severe ischemia that results from femoral artery excision is greater in Nox2-/- mice but that collateral luminal expansion and intimal cell recruitment/proliferation are normal. Collateral growth is stimulated by shear stress rather than hypoxia and these data are consistent with observations of flow-mediated outward remodeling in carotid arteries of Nox2 -/- mice. Nox2 is important in vascular responses to injury and hypoxia, but it does not mediate compensatory flow-mediated outward remodeling and collateral growth in the hindlimb of young healthy WT mice.

3.20

TARGETING INTEGRIN SIGNALING IN INFLAMMATION – IDENTIFICATION OF SMALL MOLECULE ANTAGONISTS OF INTEGRIN CYTOPLASMIC DOMAIN–SYK INTER-ACTIONS

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Integrin cell adhesion molecules regulate leukocyte trafficking and activation, playing critical roles in inflammation and cardiovascular disease. Their cytoplasmic domains are essential for signal transduction yet they do not possess any intrinsic catalytic function, relying on direct interactions with intracellular effectors to transmit signals. The tyrosine kinase Syk is regulated by its direct interaction with integrin beta-chain cytoplasmic domains. Here we describe the development of a novel ALPHAscreen-based platform that can be used to identify antagonists of integrin cytoplasmic domain - Syk interactions. A pilot screen of a small molecule library (<10,000 compounds) was performed (Z' > 0.6) and specific antagonists of integrin cytoplasmic domain – Syk interactions were identified. Compound IC50s ranged from ~ 1.0 – 10 uM. The antagonists identified are independent of Syk's kinase domain. One compound was identified that could penetrate the membrane of the monocytic cell line THP-1, and inhibit integrin alpha4beta1 dependent activation of Syk. Interestingly, the compound did not inhibit alpha4beta1 dependent activation of Pyk-2. This demonstrates, for the first time, that small molecule drugs can be identified that selectively target the integrin : Syk signaling axis, while leaving other integrin signal transduction pathways intact. This has important implications in the development of safe and novel integrin signaling antagonists.

3.21

CREATION OF A SUITE OF IMMUNO-COMPROMISED KNOCKOUT RAT MODELS

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The laboratory rat is the preferred model system in many biological studies for its physiology and larger size. However, until recently, the lack of genetic manipulation tools for the rat genome made the mouse the predominant choice for researchers. Zinc-finger nuclease (ZFN) technology is now revolutionizing rat research. ZFN technology allows specific and efficient introduction of desired mutations to the gene of interest. ZFNs are fusion proteins of a zinc finger protein and the DNA endonuclease domain of a type II restriction enzyme, FokI. ZFNs are engineered to bind and cleave at a specific chromosomal locus to generate double strand breaks, leading to non-homologous end joining-mediated gene disruption.
We have successfully created Rag1 knockout rats and are in the process of generating Rag2, FoxN1, and DNAPK knockouts by ZFN microinjection into single-cell embryos that are transferred to pseudopregnant females. The immuno-compromised models will be useful in studies on cancer biology, infection, gene therapy, xenograft and more. Injection statistics, current status of the models, and available phenotype data will be discussed.

3.22

THE PROTECTIVE ROLE OF TRIF IN THE PROGRESSION OF DILATED CARDIOMYOPATHY

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Autoimmune myocarditis progresses to chronic Dilated Cardiomyopathy (DCM) in susceptible people and mice. TRIF is a signaling adapter required for TLR3 and TLR4/MyD88-independent signaling. TLR4 signaling has been shown to promote inflammation, leading to DCM. TLR3 responds to viral infection. TRIF may be situated along a signaling pathway that is critical to the innate immune response to DCM. To test the role of TRIF signaling in the progression of DCM, TRIF-/- males and C57BL/6 controls were infected with heart-passaged Coxsackie Virus B3 (CVB3) to induce autoimmune disease. Ten days later, heart function was assessed by echocardiography, and heart catheterization. Tissue was harvested to quantify inflammatory cell infiltrate, viral replication, and cytokine levels. Heart function assessment revealed left ventricular dilation and systolic dysfunction in TRIF-/- as characterized by: 30% increase in left ventricular end diastolic diameter, ejection fraction of 38%, a 42% decrease in contractility, a developed pressure of 68 mmHg. Myocardial inflammatory cell infiltration increased by 40% in TRIF-/- mice. Viral replication increased from 600 PFU/g in WT to 10,000 PFU/g in TRIF-/-. Cytokine analysis showed an increase in IL-33, and a decrease in IFN-b in TRIF-/- mice. TRIF-/- hearts show an accelerated progression to dilation and failure, increased viral replication, and greater inflammatory infiltration. TRIF regulates IFN-b signaling, and IL-33. NIH R01 HL087033.

3.23
DEVELOPMENT OF SPONTANEOUS ANERGY IN INVARIANT NKT CELLS IN A MOUSE MODEL OF DYSLIPIDEMIA

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Objective. We investigated whether dyslipidemia-associated perturbed invariant natural killer T (iNKT) cell function is due to intrinsic changes in iNKT cells or defects in the ability of antigen presenting cells to activate iNKT cells. Methods and Results. We compared iNKT cell expansion and cytokine production in C57Bl/6 (B6) and apolipoprotein E-deficient (apoE-/-) mice. In response to in vivo stimulation with □-galactosylceramide (□-GalCer), a prototypic iNKT cell glycolipid antigen, apoE-/- mice showed significantly decreased splenic iNKT cell expansion at 3 days post-injection, a profile associated with iNKT cell anergy due to chronic stimulation. This decrease in expansion was accompanied by a two-fold increase in percentage of iNKT cells expressing the inhibitory marker PD-1 in apoE-/- mice compared to controls. However, in vivo and in vitro blockade of PD-1 using monoclonal antibody was not able to restore function of iNKT cells from apoE-/- mice. iNKT cells from apoE-/- mice also had increased intracellular TCR expression; a phenotype associated with previous activation. Changes in iNKT cell functions were cell autonomous, as dendritic cells (DCs) from apoE-/- mice were able to activate B6 iNKT cells but iNKT cells from apoE-/- mice were not able to respond to B6 DCs. Conclusions. These data suggest chronic dyslipidemia induces an iNKT cell phenotype unresponsive to further simulation by exogenous glycolipid and that sustained unresponsiveness is iNKT cell intrinsic.

3.24
N-ACETYL-SERYL-ASPARTYL-LYSYSL-PROLINE (AC-SDKP) AMELIORATES THE SEVERITY OF EXPERIMENTAL AUTOIMMUNE MYOCARDITIS (EAM) IN RATS

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Ac-SDKP protects against cardiac inflammation and fibrosis. Hypothesis: Ac-SDKP reduces innate/adaptive immunity, preventing cardiac fibrosis and dysfunction in EAM. To induce EAM rats were immunized with an injection of porcine cardiomyosin and treated with vehicle or Ac-SDKP for 4 weeks. Non-immunized rats were used as control. Left ventricular (LV) ejection fraction (EF, index of systolic function) and trans-mitral flow velocity (E/A ratio, index of LV diastolic function) were measured by echocardiography. Macrophage and T helper infiltration (cells/mm2) and collagen deposition (%) in the heart were analyzed by immunohistochemistry and picrosirius red, respectively. EAM had significantly increased macrophage and T helper infiltration and collagen deposition with decreased LV systolic and diastolic function. Ac-SDKP ameliorated all these detrimental effects.

	Vehicle	SDKP	EAM Vehicle	EAM SDKP
Macroph	101±9	98±7	257±37*	138±9†
T helper	59±4	62±6	148±20*	97±17†
Collagen	15±2	12±1	20±2*	16±2†
EF (%)	82±1	83±1	67±3*	78±3†
E/A ratio	1.5±.1	1.5±.1	1.2±.1*	1.5±.1†

*p <.02 EAM *vs* Vehicle; †p <.04 EAM + Ac-SDKP *vs* EAM

Conclusion: In EAM, Ac-SDKP reduces innate/adaptive immunity, collagen deposition and improved LV systolic and diastolic function. Our study may provide an important therapeutic strategy for autoimmune-associated myocarditis. NIH Grant HL028982

3.25
CASPASE INHIBITION REDUCES INFLAMMATORY CELL APOPTOSIS IN THE HEART AND AMELIORATES CARDIAC DYSFUNCTION WITH CHRONIC PRESSURE OVERLOAD HYPERTROPHY

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Left ventricular (LV) hypertrophy, a major compensatory mechanism in response to pressure overload eventually fails, leading to LV decompensation. It is thought that a mechanism underlying the reduced LV function is myocyte apoptosis with resultant loss of contractile elements and LV mass. We examined the effects of 3 weeks transverse aortic constriction (TAC) in C57BL/6 mice with or without administration of caspase inhibition (CI) with Z-Asp-2,6-DCBMk. In mice with CI and TAC, LV ejection fraction was preserved (66±3%) compared with the vehicle group (54±3%). CI also prevented LV dilation after TAC, but did not affect LV mass; LV/tibia length increased from 4.9 to 7.2 in the vehicle group and similarly, from 4.7 to 6.6 with CI. Apoptosis (%) was determined by TUNEL, and predominated in non-myocytes at 3wk after TAC. CI significantly reduced apoptosis, but surprisingly more in non-myocytes (from 1.50 to 1.15 cells/mm2) than myocytes (0.11 to 0.08 cells/mm2), of which F4/80-positive macrophages were a major cell type of the rescued non-myocytes by CI. Thus, as expected, CI reduced apoptosis, protecting LV function following TAC. However, the overwhelming majority of apoptotic cells rescued by CI were non-myocytes. It is unlikely that the rescue of LV function was preservation of contractile elements, since LV mass was not affected; but more likely other cell types, particularly inflammatory cells, mediate the salutary action of CI.

3.26
BODY MASS INDEX AND THROMBOGENIC FACTORS IN NEWLY MENOPAUSAL WOMEN

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Although increase in body mass index (BMI) is a recognized risk factor for cardiovascular disease, the relationships of BMI to physiological processes associated with thrombotic risk are not known. Analyses were performed to evaluate the relationship of BMI to thrombogenicity of the blood in women who (n=118) were 42-58 years of age and within the 3 years of their last menses from the Kronos Early Estrogen Prevention Study. Data were analyzed by groups based on BMI classification as Low (BMI <25 kg/mm2, n=40), Moderate (BMI 25<30 kg/mm2, n=43), and High (BMI 30<35 kg/mm2, n=35). Although systolic and diastolic blood pressure, blood glucose, total cholesterol, LDL and triglycerides were considered in the normal range, these parameters were significantly greater in the moderate and high BMI groups compared to Group I ($P<0.05$). Number of platelets, high-sensitive CRP, and activated protein C were greater in women in the high compared to the low BMI group. Total and platelet-derived blood borne thrombogenic microvesicles (or microparticles) increased with increasing BMI. Therefore, platelet-derived, thrombogenic microvesicles may represent an additional marker for cardiovascular risk even in women in whom conventional risk parameters are within the normal range. This study is supported from Kronos Longevity Research Institute, NIH HL90639, 1UL1 RR024150 and AHA 08-30503Z.

3.27
LONG-TERM VAGUS NERVE STIMULATION IMPROVES LEFT VENTRICULAR FUNCTION AND NORMALIZES PRO-INFLAMMATORY CYTOKINES IN DOGS WITH CHRONIC HEART FAILURE

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Background: Vagus nerve stimulation (VS) can improve left ventricular (LV) function in heart failure (HF) by restoring autonomic balance. Pro-inflammatory cytokines are elevated in HF and contribute to worsening of the disease state. This study examined the effects of long-term VS on left ventricular systolic function and circulating levels of pro-inflammatory cytokines. Methods: Studies were performed in 15 dogs with HF (LV ejection fraction, EF 30%-40%) produced by coronary microembolizations. A bipolar cuff electrode was implanted around the right cervical vagus and connected to an implantable pulse generator. Dogs were randomized to no therapy (control, n=7) or to active VS therapy (n=8) maintained for 3 months. LV end-diastolic and end-systolic volumes and EF were measured

from ventriculograms taken at time of randomization and after 3 months of therapy. Plasma samples obtained at randomization and end of study to measure circulating levels of tumor necrosis factor-alpha, interlukin-6 and c-reactive protein. Results: Controls animals showed no improvement in any variable over 3 months. In VS-treated dogs, ESV decreased (43.0±1.5ml to 35.9±1.6ml) and EF increased significantly (31.7 ±1.1% to 40.1 ± 0.9%). VS therapy also significantly reduced the levels of all 3 pro-inflammatory cytokines (pg/ml; TNFα 6.1±0.5 to 3.7±0.3; IL-6 57 ±2 to 31±4 and CRP 297±14 to 200±7). Conclusions: Long-term VS therapy improves LV function, prevents progressive LV dilation and markedly attenuates pro-inflammatory cytokines in dogs with moderate HF.

3.28
STRESS-INDUCED HYPERTENSION PROMOTES T LYMPHOCYTE ACTIVATION AND VASCULAR INFLAMMATION

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The central nervous system (CNS) plays an essential role in the regulation of blood pressure. Recently our group has shown that the CNS is critical for the development of peripheral vascular inflammation and T cell activation during angiotensin II-induced hypertension. Chronic psychological stress has also been identified as a key contributing factor to the development and maintenance of hypertension. We therefore hypothesized that T lymphocytes are involved in chronic stress-induced hypertension. To test this hypothesis, C57Bl/6 mice were exposed to daily restraint stress combined with cage switch stress for one week. A mild increase in blood pressure was observed in the stressed group (125 ± 1.1 mmHg) compared to the non-stressed control (117 ± 1.3 mmHg) and this elevation in pressure was severely augmented with low-dose angiotensin II infusion (140 ng/kg/min). The total number of circulating CD4+ cells (Control: 13.7 ± 0.9 vs Stress:18.4 ± 1.2) as well as the T cell activation markers CD69 (Control:7.7 ± 1.4 vs Stress:12.7 ± 1.2) and CD44 (Control:17.1 ± 1.3 vs Stress:22.8 ± 1.1) in peripheral blood were also increased following stress. Moreover, there was a marked increase in aortic and kidney infiltration of total leukocytes (CD45+) and T cells (CD3+) in the stressed group. These data indicate that mild elevations in blood pressure due to chronic stress contribute to the activation of circulating T cells and the promotion of vascular and renal inflammation.

3.29
ADVERSE MYOCARDIAL REMODELING INVOLVES INTERACTIONS BETWEEN SENSORY NERVES AND INFLAMMATORY CELLS

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The cardiac mast cell is an inflammatory cell that mediates adverse myocardial remodeling induced by chronic volume overload, the initial phase of which includes collagen degradation via a mechanism involving TNF-α. While little is known about what activates these cells, mast cells often maintain a close spatial relationship with nerves, raising the possibility of a neuro-immune interaction initiating myocardial remodeling. We sought to test the hypothesis that neuropeptides contained in sensory nerves regulate cardiac mast cell mediated adverse myocardial remodeling. Isolated rat cardiac mast cells were incubated with increasing concentrations of substance P and neurokinin A and histamine release measured as a marker of activation. Only substance P induced a concentration-dependent degranulation of these cells that was via the neurokinin-1 (NK-1) receptor. Substance P also induced an increased release of TNF-α from a mixed population of inflammatory cells, including T cells, mast cells and macrophages, isolated from the heart. Blockade of substance P in vivo with the selective NK-1 receptor antagonist, L732138 (5 mg/kg/d), in the aortacaval fistula model of volume overload, prevented increases in cardiac mast cell density and myocardial TNF-α as well as preventing myocardial collagen degradation. Thus, a substance P-induced inflammatory cell response, via the NK-1 receptor, causes an elevation in myocardial TNF-α which in turn initiates adverse myocardial remodeling. This work was supported by American Heart Association fellowship 0825510E (to S.P.L.).

3.30
MECHANISMS OF LEUKOCYTE AND PLATELET RECRUITMENT IN THE MICROVASCULATURE DURING CHRONIC ANGIOTENSIN II ADMINISTRATION

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Angiotensin II (AngII) contributes to the pathogenesis of hypertension and other cardiovascular diseases. AngII is also known to induce a pro-oxidative, pro-inflammatory, and pro-thrombogenic phenotype in vascular endothelial cells. Although the peptide promotes the recruitment of leukocytes and platelets in the microvasculature, the mechanisms that underlie these responses remain poorly defined. In this study, we addressed the contributions of angiotensin II type-1 receptors (AT1r), RANTES, and interleukin-1b to the recruitment of leukocytes and platelets in skeletal muscle arterioles and venules during chronic (2 wks) infusion of AngII (1 ug/Kg/min) in wild type (WT) and mutant mice. Intravital microscopy was used to quantify the adhesion of leukocytes and platelets, and the emigration of leukocytes in the cremaster muscle microvasculature. In WT mice, AngII infusion induced the adhesion of both leukocytes and platelets in venules and only leukocytes in arterioles. These adhesion responses were not observed in AT1r-/- mice and in AT1r-/- bone marrow chimeras (blood cells deficient in AT1r). Although RANTES-/- mice responded similarly to WT mice, IL-1b-/- mice exhibited significant reductions in leukocyte adhesion and emigration in venules, and platelet adhesion was unaffected. These findings implicate blood cell-associated AT1r and IL-1b in the pro-inflammatory and prothrombogenic phenotype assumed by microvessels exposed to chronic AngII. (Supported by HL26441).

3.31
OXIDATIVE STRESS AND INFLAMMATION PLAYS A ROLE IN CIGARETTE SMOKE INDUCED UNFAVORABLE EFFECTS OF CARDIAC STEM CELL SIGNALING AND FUNCTIONS IN RAT CARDIAC STEM CELLS

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Cigarette smoke (CS) is a major cardiovascular risk factor. Because of the unique properties of stem cells (migration, differentiation at the site of injury etc.) they can be used to treat myocardial injuries and we used rat cardiac stem cells (rCSC) as our experimental model. Hypothesis: CS induced oxidative stress attenuates cardiac stem cell signaling and functions via recruiting inflammatory lipid mediators. Specific aims: 1. Investigate effects of CS on extra cellular signal regulated kinase (ERK) cell signaling pathway in rCSC. 2. Investigate effects of CS on rCSC functions, which are migration, proliferation, and apoptosis. 3. Investigate whether oxidative stress and inflammation plays a role in CS induced effects of rCSCs. Methods: Smoking was modeled in-vitro by preparing cigarette smoke extracts according to a published method. rCSCs were exposed to either control or medium with varying CS exposures for 24 hours and experimental assays were conducted. Results: CS significantly inhibited ERK activation. In rCSC, CS caused an increase in apoptosis and decrease in migration. Smoke induced apoptosis was prevented by pre-treatment of rat cardiac stem cells with vitamin C. The experiments to address recruitment of inflammatory lipid mediators (cytokines, prostaglandins) in response to CS exposure are currently in progress. Conclusions: cigarette smoke inhibited cardiac stem cell viability, migration, and ERK activation. Implications: CS induced impaired cardiac stem cell responses may contribute to the increase in smoke related cardiovascular risk.

3.32
EFFECT OF A CXCR2 ANTAGONIST ON CXCL1, CXCL2 AND CXCR2 BRAIN EXPRESSION AND INFARCT VOLUME AFTER STROKE IN C57BL6 MICE

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Stroke induces brain inflammation and attraction of leukocytes. CXCL1 and CXCL2 are chemokine ligands expressed by monocytes and neutrophils with strong neutrophil chemoattractant activity via the neutrophil CXCR2 receptor. We examined the expression profile of these molecules in the brain and the effects of a CXCR2 antagonist, SB225002 (SB), after stroke. A PCR array using Sybr Green revealed that at 4, 24 and 72 h after 30 min occlusion of the middle cerebral artery, CXCR2 was increased by 2-, 6- and 13-fold; CXCL1 was increased 1-, 37- and 14-fold; and CXCL2 was increased 3-, 303- and 8-fold, respectively. Similar findings were obtained by Taqman PCR. We then treated mice with vehicle (V; 1% DMSO) or SB (2 mg/kg/d ip) commencing at reperfusion. Expression levels of CXCR2, CXCL1 and CXCL2 after 24 h in SB-treated mice were reduced to 9%, 12% and 13%, respectively, of the levels in V-treated mice, possibly due to reduced neutrophil infiltration. However, while these findings indicate that SB effectively antagonized the interaction between CXCR2 and its chemokine ligands in the ischemic brain, we found that compared with V treatment (n=7), mice treated with SB (n=9) had similar motor impairment as assessed by hanging wire latency (V: 34±7 s; SB: 27±8 s; P=0.50) and similar infarct volume (V: 18±5mm3; SB: 23±8mm3; P=0.62) at 72 h. Thus, reduced neutrophil-related inflammation achieved by a CXCR2 antagonist administered after stroke does not appear to improve outcome.

3.33
HOMOCYSTEINE DECREASED DIFFERENTIATION OF SPLENIC REGULATORY T CELL IN MOUSE

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Hyperhomocysteinaemia (HHcy) has been implicated as an independent risk factor for atherosclerosis. We have found that decreased number of regulatory T cell (Treg) is responsible for HHcy–accelerated atherosclerosis. Here, we investigated

potential mechanism of Treg impairment by homocysteine (Hcy) in vitro. Firstly, we set up a system for Treg differentiation in vitro. In the system, Th0 was induced to differentiate into Treg which expressed high level CD4, CD25, GITR and Foxp3. We found that Treg differentiation was inhibited by 30 μM Hcy, which was indicated by decreased Foxp3 expression to 75%. Fenofibrate, a classical PPARα activator, has been discovered to affect the immune system. In our research, we found that fenofibrate (10-20μM) could promote Foxp3+ Treg differentiation in a dose-dependent manner. WY14643, another PPARα activator, showed a similar effect with fenofibrate. And HHcy-induced Treg impairment might be partly reversed by fenofibrate in vivo. The experimental procedures were approved by the Declaration of Helsinki and the APS "Guiding Principals in the care and Use of Animals." Decreased Treg differentiation by Hcy is reversed by PPARα activator in vitro. And fenofibrate might reverse HHcy–induced atherosclerosis through PPARα activation in vivo. This work was supported by the National Basic Research Program (No. 2006CB503802) and NSFC Foundation (No. 30730042 and 30821001) of P. R. China to XW.

3.34
COSTIMULATORY SIGNALING PATHWAY MEDIATED HYPERHOMOCYSTEINAEMIA-INDUCED IMBALANCE BETWEEN TREG AND TEFF IN MICE

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Atherosclerosis is known as a chronic inflammatory disease. Our previous work suggests that regulatory T cell (Treg) impairment is responsible for hyperhomocysteinaemia (HHcy)-accelerated atherosclerosis. Here, we investigated the modulation of HHcy to costimulatory molecules in murine T cell. HHcy was induced by supplemented with 1.8 g/L Hcy in drinking water for 2 weeks in C57 mice. Splenic T cells surface costimulators were measured by FACS. We found that the expression of inhibitory molecules GITR and CTLA4 were decreased similar by 10% and 5% in Treg and effector T cell (Teffs), while the ICOS expression was not changed. The activatory CD28 expression was decreased in Treg but was increased in Teff. And the protein expression may be due to change in gene expression. HHcy-induced inflammation might be improved by rescuing the imbalance between Treg and Teff through modulating costimulatory signaling with activator or blocker. The experimental procedures were approved by the Declaration of Helsinki and the APS "Guiding Principals in the care and Use of Animals." HHcy could induce imbalance between Treg and Teff by modulating costimulatory signaling pathway to promote inflammation in mice. This work was supported by the National Basic Research Program (No.2006CB503802) and NSFC Foundation (No. 30730042 and 30821001) of P. R. China to XW.

3.35
REGULATORY T CELLS/T-HELPER CELL 17 FUNCTIONAL IMBALANCE IN PATIENTS WITH END STAGE KIDNEY DISEASE: A PIVOTAL LINK BETWEEN MICROINFLAMMATION AND ADVERSE CARDIOVASCULAR EVENTS

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Aim:Adverse cardiovascular events resulting from accelerated atherosclerosis are the leading cause of mortality in patients with end stage kidney disease(ESKD). The aim of the present study was to assess regulatory T cells (Treg) /T helper (Th)17 cells pattern in patients with ESKD and to explore the significance of Treg/Th17 imbalance in the development and outcome of acute cardiovascular events(ACE). Methods: 22 patients with ACE and 20 patients without ACE were evaluated. 30 healthy volunteers were enrolled as control group. Treg cell frequency, Th17 cell frequency , Foxp3 mRNA expression, ROR*ƒ*×t mRNA expression, Serum cytokines and C-reactive protein were detected. Results: patients with ESKD exhibited an obvious imbalance of Treg/Th17 function when compared to the normal controls, displaying increased peripheral Th17 frequency, Th17-related cytokines (interleukin [IL]-17, IL-6 and IL-23) and ROR*ƒ*×t mRNA levels. These patients also displayed decreased Treg frequency, Treg-related cytokines (IL-10, transforming growth factor-*ƒ*Ò1) and Foxp3 mRNA levels. This imbalance was more pronounced in patients with acute cardiovascular events. It was also observed that the imbalance of Treg/Th17 was not only consistent with the cardiovascular disease but also correlated with a microinflammatory state. Conclusion: The Treg/Th17 balance was disturbed by uraemia, especially in patients with adverse cardiovascular events. This Th17/Treg imbalance might act synergistically with microinflammation on immune-mediated atherosclerosis and contribute to the high incidence of adverse cardiovascular events.

3.36
INTERLEUKIN-4 MEDIATES HYPOXIA-INDUCED MITOGENIC FACTOR (HIMF/FIZZ1/RELMA)-INDUCED LUNG INFLAMMATION AND PULMONARY MICROVASCULAR ENDOTHELIAL CELL ACTIVATION

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Hypoxia-induced mitogenic factor (HIMF), also known as found in inflammatory zone 1 (FIZZ1) and resistin-like molecule a (RELMα) has mitogenic and chemotactic properties during pulmonary hypertension (PH)-associated vascular remodeling. HIMF expression in the lung has been suggested to be regulated by Th2 cytokines in bleomycin-induced pulmonary fibrosis model. However, the mechanistic basis for this observation in hypoxia-induced PH model is unknown. In the present study, we have shown that hypoxia increased lung HIMF expression in both interleukin-4 (IL-4) knockout (KO) mice to the same degree as compared to wild type (WT) mice. We introduced HIMF protein via tail vein to each genotype and have shown that HIMF-induced proliferative activity, collagen synthesis and extracellular matrix (ECM) deposition in the pulmonary arteries were significantly decreased in IL-4 KO mice. In ex vivo lung organ culture model, HIMF-induced angiogenic growth factor production was significantly suppressed in IL-4 KO mice. Furthermore, our in vitro studies revealed that HIMF increased endothelial cell proliferation and migration in a manner dependent on the IL-4 receptor a. These findings suggest that IL-4 signaling may play a significant role in HIMF-induced lung inflammation during PH-associated vascular remodeling. This work was supported by NIH Grant RO139706 (R.A.J.) and NIH Specialized Centers of Clinically Oriented Research Grant P50084946 (R.A.J.)

3.37
STIMULATION OF ALPHA7 NICOTINIC ACETYLCHOLINE RECEPTOR ATTENUATES INFLAMMATORY RESPONSE IN MACROPHAGES AND IMPROVES SURVIVAL IN EXPERIMENTAL MODEL OF SEPSIS THROUGH HEME OXYGENASE-1 INDUCTION

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Several reports suggested that activation of nicotinic acetylcholine receptor alpha7 subunit (α7-nAChR) by nicotine leads to the improved survival during experimental model of sepsis by attenuation of inflammatory response. Thus, it is important to investigate the molecular mechanism by which nicotine provides anti-inflammatory action. We found that nicotine can stimulate heme oxygenase-1 (HO-1) expression in murine macrophages (RAW264.7 cells). HO-1-inducible effect by nicotine was mediated by following sequences; Ca2+ influx, classical protein kinase C activation, reactive oxygen species production, and phosphoinositol 3 kinase/Akt pathway activation. Further, to identify whether HO-1 induction is important for anti-inflammatory action of nicotine siRNA technique was utilized. In scramble RNA-transfected cells nicotine significantly inhibited tumor necrosis factor-alpha (TNF-α), inducible nitric oxide synthase (iNOS) and high mobility group box 1 (HMGB1) expressions induced by lipopolysaccharide (LPS), whereas, nicotine failed to inhibit the production of these mediaotrs in siHO-1 trasfected cells. Additionally, we demonstrated that nicotine can not improve survival in the presence of ZnPPIX (HO-1 inhibitor) during lethal endotoxemia and cecal ligation and puncture model. Thus, we conclude that activation of α7-nAChR by nicotine provides anti-inflammatory action through HO-1 upregulation.

3.38
CHARACTERIZATION OF T CELLS INFILTRATING PERIVASCULAR CORONARY ADIPOSE TISSUE IN ATHEROSCLEROSIS

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Atherosclerosis is associated with perivascular inflammation. While role of macrophages infiltrating pericardial fat is well characterized, the characteristics of T cells infiltrating peri-coronary adipose tissue is less known. Accordingly we aimed to study T cells infiltrating pericardial adipose tissue surrounding coronary arteries (CORO) in comparison with adipose tissue of internal mammary arteries (IMA), which do not develop atherosclerosis. T cell infiltration of adipose tissue was studied by flow cytometry of collagense / hyaluronidase digested tissues. Results: The content of total leukocytes was 4-fold higher in adipose tissue surrounding coronary arteries than in IMA (2.7±0.02 vs. 0.6±0.4%; $p<0.05$). CD3+ cells (T cells) constituted – 37±3.5% of leukocytes similarly in CORO and IMA. The absolute number of T cells was higher in CORO than IMA adipose tissue (23.3±13.1 vs. 14.2±18.9 cells/mg of tissue). Interestingly, a high content of T cells in CORO was neither CD4 nor CD8+ (11.1 vs. 4.2 cells/mg of tissue) and these cells were predominantly CCR5+ particularly in CORO (71% in IMA and 90%in CORO). The increase in CCR5+ cells was particularly prevalent in CD4 cells (4% vs. 49.1%). Conclusion: Development of atherosclerosis in coronary arteries is accompanied by double negative (CD4-/8-) T-cell infiltration, characterized by high expression of RANTES receptor CCR5. This work is funded by European Union Structural Grant for the Foundation for Polish Science/Welcome/2009/02.

3.39

THE MAMMALIAN STE20 ORTHOLOGUE LINKS TLR SIGNALING TO MAPKS AND PLAYS A CRUCIAL ROLE IN ATHEROGENESIS

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Evidence has linked toll like receptors (TLRs) and their immediate downstream adaptor proteins to atherosclerosis. However, the molecular mechanism linking these proximal events to established downstream effectors, notably MAPKs, and to atherosclerosis are unknown. Therefore, our objective in this study was to identify such key elements linking TLR complexes to MAPKs and atherogenesis. Germinal center kinase (GCK), a mammalian STE20 orthologue, directly activates MAP3Ks mixed lineage kinases (MLKs)-2 and -3, which in turn leads to the activation of JNKs and p38. GCK can be activated by multiple TLR ligands as well as oxLDL. To test GCK's possible role in atherosclerosis, we set up a mouse model to compare high fat diet-induced atherogenesis in atherosclerosis-prone apoe-/- and the apoe-/-; gck-/- double knockout mice. En-face analysis of the aortic arch and descending aorta, showed a drastic reduction of atheroscleric plaque formation in apoe-/-; gck-/- versus apoe-/- mice. Quantification of the lesion area in the aortic root using immunohistochemical analysis also demonstrated the reduced atherosclerosis in fat-fed apoe-/-; gck-/- mice. Therefore, our study suggests that GCK, a downstream effector of TLR signaling plays a critical role in atherosclerosis. This is the first evidence from an intact animal model linking TLR and MAPK activation to atherogenesis. Our results suggest that targeting the GCK pathway could prove effective in treating atherosclerosis. Funding: NIH Grant GM46577.

3.40

AC-SDKP AND ENDOGENOUS PEPTIDE WITH CARDIO-VASCULAR AND RENAL ANTI-INFLAMMATORY AND ANTI-FIBROTIC PROPERTIES

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Innate and adaptive immunity have been implicated in both development of hypertension and target organ damage. We reported that in various models of hypertension and post myocardial infarction Ac-SDKP; a naturally occurring tetrapeptide; decreased inflammation (macrophage infiltration) and cardiovascular fibrosis. In a model of 5/6 nephrectomy induced-hypertension, Ac-SDKP decreases inflammation, fibrosis and glomerulosclerosis and improves renal function. Also in Ang II-induced hypertension the effects of ACE inhibitor on inflammation and fibrosis are mediated by Ac-SDKP. Inhibition of Ac-SDKP release causes cardiac and renal perivascular fibrosis and glomerular nephrosclerosis. Also in Ang II induced-hypertension reduction of Ac-SDKP accelerates interstitial cardiac fibrosis attributable to an increase in cells that produce collagen. In a model of cardiac inflammation (pericardial infusion of Galectin-3), Ac-SDKP not only reduced cardiac inflammation, fibrosis and hypertrophy but improved systolic and diastolic cardiac function. Recently we studied the effect of Ac-SDKP on adaptive immunity in Ang II-induced hypertension and in autoimmune induced myocarditis. In these two models Ac-SDKP reduced adaptive immunity and cardiac fibrosis and improved function. All the effects of Ac-SDKP occurred with no changes in blood pressure. We concluded that reduction of innate and adaptive immunity contributes to the effects of Ac-SDKP in hypertension and also in myocarditis. NIH HL028982

4.0: BASIC ASPECTS OF IMMUNOLOGY II

4.2

NATURAL KILLER AND NATURAL KILLER T CELLS

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Natural killer (NK) cells are a subset of lymphocytes that belong to the innate arm of the immune system. These cells play important roles in immune defense against viruses and tumors. NK cells can directly lyse target cells and are capable of producing a variety of immunomodulatory cytokines. NK cells recognize susceptible targets based on interactions of a variety of activating and inhibitory receptors with their ligands on target cells. Natural killer T (NKT) cells bear several similarities with NK cells, but also express T cell receptors (TCRs), which are a hallmark of T lymphocytes of the adaptive immune system. Interestingly, however, the TCR of NKT cells is semi-invariant and is specific for glycolipid antigens presented by the major histocompatibility complex class I-related protein CD1d. NKT cells play a regulatory role in the immune system, contributing to immune responses against many infections, tumors and alloantigens. Numerous studies have further shown that both NK cells and NKT cells can impact autoimmune and inflammatory conditions, including cardiovascular disease. References: Shi FD, and Van Kaer L. 2006. Reciprocal regulation between natural killer cells and autoreactive T cells. Nat. Rev. Immunol. 6, 751-760. Major AS, Joyce S, and Van Kaer L. 2006. Lipid metabolism, atherogenesis and CD1-restricted antigen presentation. Trends Mol. Med. 12, 270-278. Wu L, Gabriel CL, Parekh VV, and Van Kaer L. 2009. Invariant natural killer T cells: innate-like T cells with potent immunomodulatory activities. Tissue Antigens 73, 535-545.

4.3

ANTIGEN PRESENTATION AND DENDRITIC CELL FUNCTION

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Dendritic cells have been identified among immune cells infiltrating rupture-prone areas of atherosclerotic plaques. While the role of monocytes and lymphocytes has been extensively investigated, little is known about the role of dendritic cells (DC) in the plaque. We have recently shown that classical myeloid and type-I-interferon producing plasmacytoid DC, the two major subtypes of DC with distinct functions, are present in the atherosclerotic plaque. These professional antigen-presenting cells are at the interface between innate and adaptive immune system. DC play a key role in immune activation by sensing "danger signals" via Toll-like receptors. Upon activation DC control the activation of other immune cells via antigen presentation, co-stimulatory signals and cytokine production. In particular, type I interferon upregulates expression of the cytotoxic molecule TRAIL on the surface of T lymphocytes which leads to apoptosis of plaque-resident cells. This presentation will elucidate DC function in atherogenesis and particularly in the development of myocardial infarction. REFERENCES: Niessner A, Weyand CM. Dendritic cells in atherosclerotic disease. Clin Immunol. 2010;134(1):25-32. Niessner A, Shin MS, Pryshchep O, Goronzy JJ, Chaikof EL, Weyand CM.Synergistic proinflammatory effects of the antiviral cytokine interferon-alpha and Toll-like receptor 4 ligands in the atherosclerotic plaque. Circulation. 2007;116(18):2043-52. Niessner A, Sato K, Chaikof EL, Colmegna I, Goronzy JJ, Weyand CM.Pathogen-sensing plasmacytoid dendritic cells stimulate cytotoxic T-cell function in the atherosclerotic plaque through interferon-alpha. Circulation. 2006;114(23):2482-9.

6.0: INFLAMMATION, THE VASCULAR WALL AND LESION FORMATION

6.2

IMMUNOBIOLOGY OF THE ENDOTHELIUM

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Interactions between endothelial cells (EC) and the immune system are dynamic and many are species-specific. Quiescent human EC lack signals (chemokines, adhesion molecules) needed to attract circulating leukocytes and may suppress inflammation by secreting anti-inflammatory mediators or by sequestering and/or degrading pro-inflammatory mediators. Microbial products or inflammatory cytokines, like TNF or IL-1, activate EC to promote inflammation by inducing adhesion molecules and chemokines and by potentiating pro-inflammatory actions of autacoids. Cytokines also cause paracellular leakiness, creating an extravascular provisional matrix to support inflammation. EC responses to cytokines vary with anatomic location and are modulated by shear stress, rendering arteries and capillaries less responsive than venules, and by tissue injury. EC may activate adaptive immunity by presenting antigens (peptide-MHC complexes) to circulating effector memory T cells, inducing this T cell subset to extravasate through molecular interactions not used in recruitment by chemokines. Perivascular cells (smooth muscle or pericytes) may modulate T cell responses to EC. Cells and molecules of the innate and adaptive immune systems may also injure EC, leading to dysfunction or death, responses that can be modulated positively or negatively by cytokines such as IFN-γ or IL-11, respectively. Injury of EC provides additional signals to the immune system, such as cytokines like IL-1α or IL-6, that intensify responses of effector memory T cells to antigens. Ref: Al-Lamki RS, Bradley JR, Pober JS. Endothelial cells in allograft rejection. Transplantation. 2008; 86:1340-1348.

6.3

INNATE IMMUNITY AND ATHEROSCLEROTIC LESION FORMATION

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Multiple lines of evidence now implicate inflammation in all stages of atherosclerosis. Innate immune responses provide a central transducer of risk factors and altered artery wall biology. Chemoattractant cytokines promote migration of monocytes into the arterial intima, which mature into macrophages when stimulated by other cytokines. These phagocytic cells drive many aspects of atherosclerotic progression, and contribute to the propensity of plaques to rupture by production of proteases that weaken the fibrous cap, and to thrombosis by generation of tissue factor. Thrombosis provoked by disrupted atheromatous plaques causes most acute coronary events. Blood biomarkers of the inflammatory response correlate with cardiovascular events. Thus, the growing recognition of the importance for atherogenesis of innate immunity in particular, and of inflammation in general, has both theoretical and practical clinical implications.

6.4

THE ADAPTIVE IMMUNE RESPONSE AND ATHEROSCLEROSIS

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Atherosclerosis is a chronic inflammatory disease of the vessel wall. Although some lymphocytes and dendritic cells (DC) constitutively reside in the wall of large arteries, various types of leukocytes infiltrate the adventitia and the subendothelial space, where they form the neointima and eventually much of the atherosclerotic plaque tissue. Experiments in apolipoprotein E-deficient Apoe-/- and in low density lipoprotein receptor-deficient Ldlr-/- mice have shown detrimental effects of T-helper1 (Th1) and Th17 cells, little or no effect of Th2 cells and atheroprotective effects of regulatory Treg cells. Th1 cells mainly produce interferon-γ, Th17 cells IL-17 and Th2 cells IL-4, IL-5 and IL-13. In addition, B cells are found in the adventitia of atherosclerotic arteries, forming organized tertiary lymphoid organs (TLOs) in chronic atherosclerosis. While some of the antibody responses, especially of the IgM type, to atherosclerosis are protective, the role of B cells is unknown. Recent data show that vascular DCs present antigen to T cells in the adventitia, and their number and activity greatly increases in atherosclerosis. We recently identified at least four subsets of vascular DCs, CD11b+CD11c+, CD11b-CD11b+CD103+, CD11b-CD11c+CD8α+ and CD11b-CD11c+CD8α-. In addition, several types of macrophages are found in the vessel wall, both in the adventitia and in the atherosclerotic plaque. Understanding the interplay between the adaptive and the innate immune system will be of key importance for new approaches to the treatment and prevention of atherosclerosis.
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6.5

EXAMINATION OF THE CELLULAR MECHANISMS OF ACCELERATED ATHEROSCLEROSIS IN LUPUS

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Patients with systemic lupus erythematosus (SLE) have accelerated atherosclerosis. In fact, studies have determined that premenopausal women with lupus may have a 50 times greater risk for development of cardiovascular disease, compared to the general population. Unfortunately, the underlying mechanisms are poorly understood. To address this, we transferred lupus-susceptibility to LDL receptor-deficient (LDLr-/-) mice, creating radiation chimeras with NZM2410-derived, lupus-susceptible, B6.Sle1.2.3 congenic or C57Bl/6 control donors (LDLr.Sle and LDLr.B6, respectively). LDLr.Sle mice developed a lupus-like disease characterized by production of double-stranded DNA autoantibodies and renal disease. When fed Western-type or normal chow diet, LDLr.Sle chimeras had increased atherosclerotic lesions. The plaques of LDLr.Sle mice were highly inflammatory and contained more CD3+ T cells than controls. LDLr.Sle mice also had increased activation of CD4+ T cells and B cells and significantly higher antibody to oxidized-LDL and cardiolipin. In addition, we have demonstrated increased molecular activation in lupus T cells reminiscent of reduced activation threshold and increased expression of activation markers such as CD69 and CD44. Studies to determine the minimal lupus-susceptibility genetic interval have demonstrated that genes strictly associated with T cell dysregulation are not sufficient to accelerate atherosclerosis. These data suggest that both T and B cell dysfunction are necessary to accelerate atherosclerotic lesion acceleration in lupus. Collectively, these studies demonstrate that the lupus-susceptible immune system enhances atherogenesis and modulates plaque composition and that T cells are likely necessary but not sufficient for this process.

6.6

REGULATION OF PRO-ATHEROGENIC T CELLS RESPONSES BY THE PD-1 COINHIBITORY PATHWAY

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T lymphocyte-mediated immune responses promote development of atherosclerotic lesions and contribute to plaque vulnerability. We have shown that B7/CD28 family T cell costimulatory pathways influence atherogenesis and lesion phenotype. PD-1 is a CD28 family coinhibitory receptor, which is expressed on activated T cells and is up-regulated on exhausted CD8+T cells in chronic viral infections. PD-1 binds B7-family members PD-L1, expressed on many cell types, and PD-L2 expressed on bone marrow-derived antigen presenting cells. Previously, we found that global PD-L1/L2 deficiency in ldlr-/- mice results in enhanced atherogenesis and lesion inflammation. Using a bone marrow chimeric approach, we now find that PD-L1/L2 expression on hematopoietic cells is critical for limiting lesion inflammation, including CD8+ T cell infiltration. We also find more atherosclerosis and marked increases in lesional CD4+ and CD8+ T cells in PD-1-/-ldlr-/-mice, compared to ldlr-/- mice. PD-1 expression is upregulated on aortic T cells in *ldlr*$^{-/-}$ mice fed a cholesterol-rich diet compared to control diet-fed *ldlr*$^{-/-}$mice. Our results indicate that PD-1 inhibits pro-atherogenic T cell responses, especially those mediated by CD8^{+} T cells. These findings suggest that therapeutic blockade of the PD-1/PD-L pathway, now in development for treatment of cancers and chronic viral infections, may have cardiovascular consequences. (NIH RO1 HL087282). REFERENCES: Gotsman I, Sharpe A., and Lichtman AH. T cell costimulation and coinhibition in atherosclerosis. 2008. Circ. Res. 103:1220-1231.

6.7

IMMUNE CELL RECRUITMENT AND ATHEROSCLEROSIS

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Atherosclerosis is a chronic inflammatory disease of the arterial wall and the innate and adaptive immune responses actively participates in atherogenesis. T and B cells reside within the adventitia of the normal vessels as a consequence of constitutive L-selectin-dependent lymphocyte homing into the aortas. Atherosclerotic conditions accelerate this lymphocyte recruitment. Our recent studies suggested an important role for the CXCL16/CXCR6 axis in the Th1 migration to aortas; others reported an essential role for CCL5, CXCL10, and MIF in T cell influx into the atherosclerotic wall. The mechanisms of monocyte recruitment have been partially characterized and involve selectins, VCAM-1, ICAM-1 and JAM-A as well as several chemokines, such as CXCL1, CCL5, CXCL4, CXCL7 and MIF. Interestingly, Ly6Chigh and Ly6Clow monocyte subsets differently use CCL2, CX3CL1 and CCL5 for their homing into the atherosclerotic aortas. The mechanisms of the migration of NK, NKT cells and mast cells that are found within the aortas are not well understood. Recent studies also highlight the role of adventitia as a possible site of the immune cell influx at least for some leukocyte subsets. Further understanding of the molecular mechanisms of leukocyte trafficking into aortas and their retention in this site will facilitate therapeutic approaches that may regulate homing of different leukocyte subsets into aortas during atherogenesis. Support: AHA Scientist Development Grant 0525532U. References: Galkina,E. and K.Ley. 2007. Leukocyte influx in atherosclerosis. Curr. Drug Targets. 8:1239-1248.

8.0: INFLAMMATION, METABOLIC SYNDROME AND HYPERTENSION

8.1

POTENTIAL ROLE OF INDOLAMINE 2, 3-DIOXYGENASE AND INFLAMMATORY CYTOKINES IN TYPE 2 DIABETIC NEPHROPATHY: EFFECT OF CHROMIUM PICOLINATE

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Indolamine 2,3-dioxygenase (IDO) is an immunoregulatory enzyme that modulates the production of proinflammatory cytokines including interleukin 6 and 17 (IL6, IL17). Utilizing the obese diabetic db/db mouse, we tested the hypothesis that renal expressions of IL6 and IL17, in an IDO-related fashion, contribute to the pathogenesis of type 2 diabetic nephropathy. In addition, we examined the effects of chromium picolinate (CrPic)3, a nutritional supplement purported to improve glycemic control. The db/db mice displayed markedly higher plasma glucose and hemoglobin A1c levels but moderate hyperinsulinemia compared to their lean db/m controls; Cr(Pic)3-treated db/db group showed mild improvement in glycemic status. The db/db mice displayed significant albuminuria that was associated with moderate immunostaining of diabetic renal tissue for IDO but marked IL6 and IL17 immunostaining compared to the kidney of db/m control. By contrast, the kidney of Cr(Pic)3-treated db/db mouse showed intense immunostaining for IDO but reduced IL6 and IL17 expression compared to the untreated db/db group. The reduction in renal IL6 and IL17 immunostaining of the Cr(Pic)3-treated db/db group was accompanied by reduced albuminuria. Collectively, the results suggest potential involvement of renal IDO and inflammatory cytokines in the pathogenesis of renal dysfunction in db/db mice, effects markedly influenced by Cr(Pic)3 treatment. This study was supported by a grant from the NCCAM.

8.2

ACCELERATED INSULIN RESISTANCE AND NEPHRITIS IN A MOUSE MODEL OF LUPUS

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Introduction: Systemic lupus erythematosus (SLE) patients are at increased risk of developing components of the metabolic syndrome. Furthermore, obesity and high-fat diet (HFD) can lead to SLE nephritis exacerbation in humans and mouse models. Study of the interplay between metabolic dysfunction and autoimmunity may inform the care of SLE patients.Objective: We examined the prevalence of HFD-induced insulin resistance (IR) and nephritis in the B6.Sle mouse model of SLE.Methods: B6.Sle mice and B6 controls were placed on low-fat diet or HFD. Fasting serum was collected every 5 weeks and the visceral fat and kidneys were harvested after 10 weeks of feeding.Results: HFD-fed B6.Sle mice gained more weight and had elevated fasting insulin after 5 weeks of HFD feeding as compared to B6 controls. Flow cytometry of gonadal fat revealed significantly higher T cell content and decreased percentages of macrophages. Kidneys from HFD-fed B6.Sle mice showed greater infiltration of natural killer T cells and highly-activated CD86+CD11c+ dendritic cells. Conclusions: HFD-fed B6.Sle mice show

evidence of accelerated systemic IR. Additionally, there is increased T cell infiltration into fat tissue and kidneys of these mice, suggesting that HFD-mediated lymphocyte dysregulation could contribute to the pathogenesis of IR and nephritis in our model. Future experiments will examine the immunological commonalities between the development of HFD-mediated IR and nephritis in B6.Sle mice.

8.3

LYSOPHOSPHATIDYLCHOLINES WITH EICOSAPENTAENOYL OR DOCOSAHEXAENOYL GROUP, AND THEIR OXYGENATION PRODUCTS DECREASE LEVELS OF LTC4 AND CYTOKINES SUCH AS TNF-ALPHA OR IL-6 IN ZYMOSAN A-INDUCED PERITONITIS OF MICE

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Lysophosphatidylcholines with eicosapentaenoyl or docosahexaenoyl group, and their oxygenation products decrease levels of LTC4 and cytokines such as TNF-alpha or IL-6 in zymosan A-induced peritonitis in mice. Lysophosphatidylcholine (lysoPC) is known to be a lipid mediator in various cellular responses. In this study, we examined the effect of lysophosphatidylcholine containing eicosapentaenoyl or docosahexaenoyl group in Zymosan A-induced peritonitis of mice. The i.v. administration of eicosapentaenoyl lysoPC or docosahexaenoyl lysoPC (5-150 ug/kg) prevented zymosan A-induced plasma leakage dose-dependently, with docosahexaenoyl lysoPC being more efficient than eicosapentaenoyl lysoPC. A mechanistic study indicated that such a suppressive action of eicosapentaenoyl lysoPC or docosahexaenoyl lysoPC was at least partially related to the reduction in the levels of LTC4, TNF-alpha or IL-6. Further, in i.p. administration, 1-(15-hydroperoxyeicosapentaenoyl)-lysoPC and 1-(17-hydroperoxydocosahexaneoyl)-lysoPC was more effective than eicosapentaenoyl lysoPC and docosahexaenoyl-lysoPC, respectively in the suppression of plasma leakage. Thus, it is suggested that oxygenation by 15-lipoxygenase may be crucial for a beneficial action of eicosapentaenoyl lysoPC or docosahexaenoyl lysoPC in peritonitis. Separately, docosahexaenoyl lysoPC, in combination with diallyldisulfide, showed an additive cooperation in suppression of NO or IL-6 formation in vitro test. Taken all together, it is suggested that eicosapentaenoy or docosahexaenoyl-lysoPC and oxygenation products may exert anti-inflammatory actions by reducing the levels of TNF-α, IL-6 and LTC4 in vivo systems.

8.4

ORAL ADMINISTRATION OF 2-DOCOSAHEXAENOYL LYSOPHOSPHATIDYLCHOLINE REDUCES LEVELS OF INFLAMMATORY MEDIATORS SUCH AS TNF-ALPHA, IL-6 AND LTC4

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Oral administration of 2-docosahexaenoyl lysophosphatidylcholine reduces levels of inflammatory mediators such as TNF-alpha, IL-6 and LTC4 Lysophosphatidylcholines (lysoPCs) have been known to be bioactive lipid mediators, which take part in various biological and pathological processes. In present study, we examined the effects of 2-docosahexaenoyl lyso-phosphatidylcholine (2-docosahexaenoyl-lysoPC) in vitro as well as in vivo systems. When Raw 264.7 cells were treated with 2-docoshexaenoyl-lysoPC, a concentration-dependent decrease of LPS-induced formation of NO, TNF-α or IL-6 was observed. Additionally, oral administration of 2-docosahexaenoyl-lysoPC was found to inhibit zymosan A-induced plasma leakage dose-dependently in mice with ED50 value of 50 μg/kg and Emax value of about 65%. Moreover, mechanistic study revealed that anti-inflammatory action of 2-docosahexaenoyl-lysoPC seemed to be related largely to LTC4 inhibition, but not PGE2 inhibition. Further, in i.v. administration, 2-(17-hydroperoxydocosahexaneoyl)-lysoPC was more effective than 2-docosahexaenoyl-lysoPC in the inhibition of plasma leakage, suggesting that 2-(17-hydroperoxydocosahexaneoyl)-lysoPC, a product from oxygenation of 2-docosahexaenoyl-lysoPC by 15-lipoxygenase (LOX), may be an active metabolite, intimately responsible for anti-inflammatory action. In support of this, 2-docosahexaenoyl-lysoPC was found to be more efficient than 1-docosahexaenoyl-lysoPC or DHA as substrate for human 15-LOX-2. Taken all together, it is suggested that 2-docosahexaenoyl-lysoPC may be anti-inflammatory after oral intake.

8.5

IRON DEPRIVATION AMELIORATES GLUCOSE TOLERANCE THROUGH REDUCTION OF OXIDATIVE STRESS AND INFLAMMATION IN DIABETIC KKAY MICE

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Background: Recently serum levels of iron or ferritin have been reported to be related to diabetes progression. Here we investigated whether iron deprivation can ameliorate diabetes mellitus through suppression of oxidative stress. Methods and Result: KKAy mice at 8-wks-old age were divided into 2 groups, treatment with 100mg/kg deferoxamine (DFO), an iron chelator agent, or vehicle for 2 wks. After DFO treatment, serum levels of ferritin (125.6 ng/ml) were significantly reduced in KKAy mice (164.3 ng/ml). DFO-treated KKAy mice exhibited 1) low blood glucose levels at 2 h after GTT test, 2) smaller size of epididymal fat and adipocytes area, 3) reduced mRNA expression of TNF-alpha and IL-6, and 4) decreased of urinary 8-OHdG excretion, in comparison to vehicle-treated KKAy mice. Conclusion: These findings suggest that serum iron level affects diabetes and iron deprivation contributes to the improvement of glucose intolerance through reduction of oxidative stress-inflammation.

8.6

DOUBLE NEGATIVE T CELLS IN PERIVASCULAR INFLAMMATION IN ANGIOTENSIN II DEPENDENT HYPERTENSION

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Hypertension is associated with T cell activation. In particular double negative T cells (CD4-CD8-CD3+; DN) are increased. They comprise 1-3% of particularly pro-inflammatory T cell subset in peripheral blood. Their tissue distribution is unknown. Accordingly we cytometrically investigated the characteristics of T cells infiltrating perivascular space, particularly adipose tissue (AT) with emphasis on the mechanisms of DN T cell infiltration in a model of chronic (14 day) angiotensin II minipump infusion. **Results:** Percentage of total leukocytes infiltrating perivascular adipose tissue was over 2- fold higher in Ang II infused mice than in control group (8.86±1.14% vs. 4.4±1.6%; $p<0.05$). Total number of CD3+ cells increased in Ang II hypertension (816±443 vs. 307±151 cells/mg of aorta) with particular increase in aortic DN T cells (191 ±68 vs. 85± 30 cells/mg of aorta). CCR5 expression, which was the highest in DN T cells and lowest in CD4 positive cells. Moreover the expression of CCR5 in DN T cells was higher in Ang II infused mice than in controls (30±10% vs. 21± 5%; $p<0.05$). Chemotaxis assays showed that DN cells migrated most efficiently towards RANTES compared to either CD8 and CD4 positive T cells. Conclusion: In hypertension, the double negative lymphocytes are distinct subset of T cells with the highest migratory properties towards RANTES due to their expression of CCR5. *This work is funded by European Union Structural Grant for the Foundation for Polish Science/Welcome/2009/02*

8.7

ROLE OF NKT CELLS IN THE DEVELOPMENT OF HYPERTENSION

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Our recent evidence strongly implicates T cells in the development of hypertension. The current study investigated the role of a specific subpopulation of T cells known as natural killer T (NKT) cells, in the genesis of hypertension. When activated, NKT cells are potent sources of immunoregulatory cytokines. We hypothesized that NKT cells might modulate the immune response involved in the genesis of hypertension. C57Bl6/J (WT), Jα18-deficient (Jα18-/-) mice that lack invariant NKT cells (Type I) and CD1d-deficient (CD1d-/-) mice that lack both Type I and non-variant (Type II) NKT cells were treated with angiotensin II (200 ng/kg/min) for 14 days via osmotic minipump. In contrast to WT and CD1d-/- mice, angiotensin II-induced hypertension was completely abolished in Jα18-/- mice at day 7 (WT: 155 ± 7; CD1d-/-: 150 ± 5; Jα18-/-: 117 ± 4 mmHg, $P<0.001$), but pressor responses were not different at day 14. FACS analysis revealed that activation of circulating T cells by angiotensin II defined by expression of effector T cell markers CD69 and CD44, was elevated in blood taken from WT and CD1d-/- mice but not Jα18-/- mice. Angiotensin II induced similar degrees of vascular T cell infiltration in all strains of mice. These findings indicate disparate roles of Type I and Type II NKT cells during the development of hypertension. Invariant NKT cells seem to participate in the early phase of hypertension and promote activation of other T cells, perhaps by cytokine elaboration.

8.8

THE INDUCTION OF TH17 IN DOCA-SALT RATS IS PREVENTED BY SPIRONOLACTONE

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High levels of aldosterone cause hypertension, inflammation and damage in heart and kidney, which is prevented by the pharmacological blockade of the aldosterone receptor (MR). Recent findings have implicated Interleukin-17 (IL-17) as a pathogenic mechanism in hypertension. We hypothesized that the Th17 phenotype is activated in mineralocorticoid-dependent hypertension (deoxycorticosterone acetate, DOCA-salt model). We studied three groups of uninephrectomized rats (n=4, 8 and 16 days): Vehicle, DOCA-salt (0.5mg/0.1kg + 0.9% NaCl/0.3% KCl in drinking water), and DOCA-salt+spironolactone (50 mg/kg/d). Our results show increased systolic blood pressure in the DOCA-salt group ($p<0.001$) starting at day 4 of treatment. IL-17 mRNA and protein abundance increased at 8 days of treatment in Peripheral Blood Mononuclear Cells (PBMC), concomitantly with cardiac perivascular and renal infiltration of CD4+ and Th17 lymphocytes. The

expression of Th17 differentiation-expansion cytokines (TGFβ-1, IL-23, and IL-1β;), was elevated in heart and kidney of DOCA-salt rats at 8 and 16 days. IL-17 mRNA and IL-17 protein increased in heart and kidney at 16 days. The MR blocker spironolactone prevented the development of hypertension, the infiltration of heart and kidney by CD4+ and Th17 lymphocytes, and the expression of cytokines characteristic of the Th17 phenotype. These results show the activation of Th17 in hypertension induced by aldosterone, which is dependent on MR activation. (Supported by CONICYT AT-24091044, FONDECYT 1090223, Millennium Nucleus on Immunology and Immunotherapy P07/088-F, and Fondecyt-FONDAP 15010006).

8.9

SALT IS A KEY PLAYER IN ANGIOTENSIN II-INDUCED KIDNEY INJURY AS A TRIGGER PULLER OF INFLAMMATION

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Background: Angiotensin II (AT II) is a major mediator of hypertension and kidney disease, and salt is an aggravating factor. We aimed to investigate what role salt play in AT II-induced kidney injury and clarify the mechanism. Methods: 12 w/o male transgenic tsukuba hypertensive mice (THM), overexpressing human renin and angiotensinogen genes, and wild type C57BL/6(C57) mice were used. THM were divided into three groups, 1) THM+ normal salt (NS); maintained with tap water and standard chow (0.5%NaCl), 2) THM+ high salt (HS); with 1%NaCl water and standard chow, 3) THM+ very low salt (VLS); with tap water and low salt chow (0.05%NaCl), and C57 were maintained with tap water and standard chow for 6 weeks. All experiments followed the Declaration of Helsinki and the APS guideline. Results: Systolic blood pressure (SBP) and 24h urinary albumin excretion were significantly elevated in NS and HS, whereas VLS resulted in the same as C57. Although plasma renin activity, plasma aldosterone concentration and intrarenal AT II concentration were similarly elavated in VLS and HS, only HS showed the enhancement of the renal mRNA expressions of MCP-1 and TNFα, increased urinary malondialdehyde, exacerbated kidney injuries and CD68-positive cells infiltrations. Conclusions: In THM, salt loading elevated BP and oxidative stress, induced some proinflammatory cytokines and macrophages infiltration into the kidneys, and exacerbated renal injuries. However, these findings were suppressed by salt restriction although the renin-angiotensin-aldosterone system was accelerated. This may imply salt is a determinant factor in AT II-induced hypertension and renal injuries through its proinflammatory properties.

8.10

ALDOSTERONE INDUCES CELLULAR SENESCENCE IN THE KIDNEY VIA MINERALOCORTICOID RECEPTOR/P21-DEPENDENT PATHWAY

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Recent studies demonstrated a possible role of aldosterone to mediate cellular senescence. Thus, the aim of this study was to investigate whether aldosterone induces cell senescence in the kidney and whether aldosterone-induced renal senescence affects the development of renal injury. Aldosterone infusion (0.75 μg/h, n=8) into rats for 5 weeks caused senescence-like changes in the kidney, such as increased expression of the senescence-associated β-galactosidase (SA-βGal), p53 and cyclin-dependent kinase inhibitor (p21), and decreased expression of SIRT1. These changes were abolished by eplerenone (100 mg/kg/day, n=10), a mineralocorticoid receptor (MR) antagonist, but unaffected by hydralazine (80 mg/L in drinking water, n=10). Furthermore, aldosterone induced similar changes on SA-βGal, p21 and SIRT1 in cultured human proximal tubular cells (HPTCs), which were normalized by an antioxidant, N-acetyl L-cysteine, or gene silencing of MR. Aldosterone significantly delayed wound healing and increased tumor necrosis factor-α expression (1.6±0.2 fold compared with vehicle treatment, $p<0.05$); gene silencing of p21 diminished the effects (0.9±0.2 fold, $p<0.05$), suggesting that p21-dependent senescence impaired the tubular repair and accelerated the apoptotic changes. These findings indicate that aldosterone induces renal senescence in proximal tubular cells via MR and p21-dependent pathway, which may be involved in the development of aldosterone-induced renal injury.

8.11

INDUCTION OF REGULATORY T CELLS REDUCES ENDOTHELIAL DYSFUNCTION IN A MOUSE MODEL OF HYPERTENSION WITH ELEVATED TH17 CELLS

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Th17 cells secrete the pro-inflammatory cytokine IL-17 and elevated levels of Th17 cells are associated with hypertension in humans and mice. Regulatory T Cells (Tregs) function diametrically to Th17 cells by secreting anti-inflammatory cytokines such as TGF-â and IL-10. Genetic deletion of the immunophilin FKBP12 from hematopoietic cells (FK12EC) increases serum pro-inflammatory cytokine levels; however, it is unknown if Th17 cells and Tregs are altered in FK12EC mice and whether increasing Tregs with all-trans retinoic acid (ATRA) will have beneficial vascular effects. Hypertensive FK12EC mice have decreased endothelium-dependent relaxation responses and increased serum levels of IL-2, IL-6, IL-17A, IL-21, and IL-23 levels compared to controls. Flow cytometry analysis of splenocytes and blood revealed that FK12EC mice have significantly increased Th17 cells and significantly decreased Tregs compared to controls. Treatment of control aortas with plasma from FK12EC mice significantly decreased endothelium-dependent relaxation responses. Treatment of blood from FK12EC mice in vitro with ATRA significantly increased the % of Tregs. Lastly, plasma from ATRA-treated blood from control mice increased endothelium-dependent relaxation responses in aortas from FK12EC mice. These data suggest a novel role for Tregs in modulating endothelial function in inflammatory settings. (Supported by HL084299 to BMM).

8.12

INHIBITION OF THE FK506 TARGET FKBP12 INCREASES TH17 CELL-MEDIATED INFLAMMATION AND HYPERTENSION

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The immunosuppressive drug FK506, which binds the immunophilin FKBP12 then inhibits calcineurin, decreases regulatory T cells (Tregs) and causes hypertension and endothelial dysfunction, however the mechanisms and whether increased Th17 cells play a role is unknown. FK506 treatment of mice for 1 week significantly decreased CD4+/FoxP3+ Tregs and increased CD4+/IL-17+ cells in splenocytes and PBMCs. To determine the mechanisms, we generated mice lacking FKBP12 in endothelial and hematopoietic cells (FK12EC). FK12EC mice exhibited increased vascular expression of genes and proteins related to EC injury and inflammation. Serum pro-inflammatory cytokine levels of IL-6, IL-2, IL-17a, IL-21, and IL-23 were increased significantly suggesting a Th17 cell-mediated inflammatory state. Flow cytometry studies confirmed this as splenocyte and PBMC levels of CD4+/IL-17+ cells were significantly increased in FK12EC mice compared to controls. Furthermore, STAT3 activation, which induces Th17 cells, was increased significantly while STAT5 activation, which induces Tregs, was decreased significantly in spleens of FK12EC mice. These data suggest that FK506, through its inhibition of FKBP12 in endothelial and hematopoietic cells, may cause endothelial dysfunction and hypertension by increasing Th17 T cell polarization and reducing anti-inflammatory Tregs. (Supported by HL084299 to BMM).

8.13

ADOPTIVE TRANSFER OF T-REGULATORY LYMPHOCYTES ATTENUATES ANGIOTENSIN II-INDUCED HYPERTENSION AND VASCULAR INJURY

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Angiotensin (Ang) II induces hypertension by mechanisms in part mediated by T helper (responder) lymphocytes (Tresp). T-regulatory lymphocytes (Treg) suppress Tresp and maintain immune homeostasis. We hypothesized that Treg adoptive transfer could blunt Ang II-induced hypertension and vascular damage. Ten to twelve-week old male C57BL/6 mice were injected i.v. with PBS, 3 x 105 Tregs or Tresp, 3 times, at 2-week intervals, and then infused with or without Ang II (1 μg/kg/min, s.c.) for 14 days (n=7-10). Systolic blood pressure (SBP) was measured by tail-cuff technique. Third order mesenteric artery (MA) endothelial function and stiffness were assessed by pressurized myography. Ang II infusion increased SBP by 40 mmHg ($P<0.001$) and impaired vasodilatory responses to acetylcholine by 70 % ($P<0.001$). L-NAME applied to MA revealed that NO synthase-dependent vasodilatory responses to acetylcholine were impaired by Ang II. Ang II increased MA stiffness, demonstrated by displacement to the left of the stress/strain curve. All the above were prevented by Treg ($P<0.001$) but not by Tresp adoptive transfer. These findings suggest that an immunomodulatory approach that stimulates Treg could prevent Ang II-induced blood pressure elevation and vascular remodeling and endothelial dysfunction. The research is funded by CIHR grants 82790 and 102606.

8.14

INTERLEUKIN-10 ATTENUATES THE ACUTE PRESSOR RESPONSE TO A LOW DOSE OF ANGIOTENSIN II

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Interleukin-10 (IL-10), an anti-inflammatory cytokine, inhibits the expression of pro-inflammatory cytokines including interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α). IL-10 inhibits the activation of nuclear factor kappa-beta ($NF-\kappa B$). Endogenous IL-10 also limits Angiotensin II (Ang II) – mediated oxidative stress and vascular dysfunction both *in vitro* and *in vivo*. We tested whether the acute administration of IL-10 would attenuate the blood pressure and

heart rate responses to Ang II. Male Swiss-Webster mice were treated with either IL-10 (16 ng/h sc) or vehicle for 7 days and the acute blood pressure and heart rate responses to Ang II were assessed under isoflurane anesthesia. The blood pressure response to Ang II (0.1 μg/kg) was attenuated in IL-10 infused mice (2.5 ± 1 mmHg) when compared to vehicle (5.3 ± 1 mmHg). Similar heart rate changes were observed in IL-10 (-5.2 ± 3.0 bpm) and vehicle (-8.0 ± 6.0 bpm) infused mice. The blood pressure responses to Ang II (0.6 and 1 μg/kg) were similar in vehicle (10.4 ± 1.0 and 13.2 ± 4.1 mmHg) and IL-10 (9.3 ± 2.0 and 15 ± 4.2 mmHg) infused mice. During the highest dose of Ang II (1 μg/kg), IL-10 infusion caused a greater attenuation in heart rate (-45 ± 22 bpm), when compared to vehicle (-9 ± 6 bpm). The findings indicate that elevated plasma IL-10 concentration attenuates the acute pressor response to a low dose of Ang II and enhances the reflex-mediated fall in heart rate at a higher dose.

8.15

IL-10 DEFICIENCY AUGMENTS INFLAMMATION, ENDOTHELIAL DYSFUNCTION, AND BLOOD PRESSURE IN A MOUSE MODEL OF GESTATIONAL HYPERTENSION

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Gestational hypertension (GH) may result from over-activation of the maternal immune system and is characterized by endothelial dysfunction and excessive inflammation. Given the importance of anti-inflammatory cytokines in successful, normotensive pregnancies, we hypothesized that deficiency of the anti-inflammatory cytokine IL-10 would exacerbate symptoms in our mouse model of GH. Maternal immune system activation with poly I:C causes systemic and placental inflammation, endothelial dysfunction, and hypertension in mice only when pregnant. In the absence of poly I:C, pregnant IL-10 KO mice exhibited no significant differences in systolic blood pressure, aortic relaxation responses, or serum and placental IFNã levels compared to pregnant WT mice. However, poly I:C-treated pregnant IL-10 KO mice had significantly increased blood pressure, endothelial dysfunction, fetal demise, pro-inflammatory T cells, serum and placental levels of IFNγ, as well as aortic and placental PECAM expression compared to poly I:C-treated pregnant WT mice. Additionally, sera from poly I:C-treated WT mice significantly decreased relaxation responses and increased PECAM expression in control aortas, which were normalized by recombinant IL-10. Collectively, these data show that the absence of IL-10 exacerbates the inflammation, endothelial dysfunction, and hypertension induced by maternal immune system activation during pregnancy and that IL-10 treatment may be beneficial in women with GH.

8.16

HYPERTENSION IN RESPONSE TO PLACENTAL ISCHEMIA IN PREGNANT RATS IS ASSOCIATED WITH INCREASED CD4+ T HELPER CELLS; A LINK IN THE PATHOPHYSIOLOGY OF PREECLAMPSIA

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Increased cytokines such as TNFα, IL-6, IL-17 and sFlt-1 are associated with an imbalance between regulatory and effector T cells, recently observed in preeclamptic patients. However, it is unknown if placental ischemia stimulates this T cell imbalance. We hypothesize that hypertension in response to chronic placental ischemia stimulates CD4+ T cells to produce inflammatory cytokines and anti-angiogenic factors during pregnancy.We measured blood pressure (MAP) and circulating T cells at day 19 of gestation from normal pregnant (NP) rats and placental ischemic pregnant RUPP rats. We utilized FACS to analyze CD4+T cells from RUPP and NP plasma. CD4+ T cells were cultured overnight in RPMI media with IL-2 and IL-12 under atmospheric conditions and media used to determine sFlt-1, TNF-α, IL-17, and IL-6 via ELISA.MAP increased from 106 +/- 2 mmHg (n=10) NP to 125 +/- 2 mmHg (n=15) RUPP (P<0.001). CD4+T cells increased 28 +/- 2% gated cells RUPP compared to 20 +/- 1.7% gated cells NP. TNF-α, IL-6 and IL-17 increased from 1333 +/- 230 NP to 2500 +/- 500 pg/ml RUPP; 28780 +/- 12831 to 77830 +/- 29361 pg/ml RUPP; 2450 +/- 450 to 7144 +/- 3300 pg/ml RUPP rats, respectively. sFlt-1 increased from 10468 +/- 2800 NP to 25000 +/- 6500 pg/ml RUPP (p<0.05).These data indicate that T cells are increased in response to placental ischemia, secrete inflammatory cytokines, anti-angiogenic factors and are therefore one potential link in the pathophysiology of hypertension during preeclampsia.

8.17

ASSESSMENT OF INDOLAMINE 2, 3-DIOXYGENASE (IDO) AND FOXP3+ REGULATORY T CELLS (FOXP3+ TREGS) DURING EARLY PHASE OF RENAL ISCHEMIA REPERFUSION INJURY

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IDO is a major regulator of immune and inflammatory functions, in part through recruitment of FoxP3+ Tregs. We tested the hypothesis that a renal ischemia reperfusion (IR) insult causes renal expression of IDO and recruitment of Tregs during the acute phase of injury. Accordingly, the right kidney of the transgenic mice expressing Act-mOVA FoxGFP was subjected to 45 min. of ischemia followed by 4 hrs. of reperfusion; the left kidney served as sham control. Thereafter, utilizing flow cytometry, blood samples and cells prepared from each kidney were examined for FoxP3+ Tregs for comparison to those of mice that were not subjected to an IR insult. The percent of FoxP3+ Tregs in the peripheral blood was significantly elevated in mice subjected to an IR insult compared to their non-IR counterparts. On the other hand, the ischemic-reperfused (right) kidney showed significantly lower percent of FoxP3+ Tregs compared to the non-ischemic-reperfused (left) kidney. Interestingly, however, the ischemic-reperfused kidney showed intense immunostaining for IDO compared to either the sham kidney or that of the unmanipulated animal thereby suggesting an uncoupling between IDO expression and conversion of effector T cells to FoxP3+ Tregs following a renal IR insult. Collectively, the results suggest that impaired recruitment of FoxP3+ Tregs to the ischemic-reperfused kidney during the early acute phase of an IR insult likely contributes to the pathogenesis of acute renal failure.

8.18

ROLE OF INFLAMMATION IN HYPERTENSION IN MALE AND FEMALE SPONTANEOUSLY HYPERTENSIVE RATS

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Accumulating evidence suggests a key role for inflammation in the development and progression of hypertension. We recently reported that female SHR have higher urinary excretion of inflammatory markers and greater tumor necrosis factor-α (TNF-α) levels in the mesenteric arterial bed compared to male SHR. We hypothesize that TNF- and inflammation contribute to the hypertension in female SHR to a greater extent than in males. To test this hypothesis, we assessed protein expression of the TNF-α receptor 1 in mesenteric arteries, plasma C-reactive protein (CRP) levels, and measured BP in young (12 - 16 week old) male and female SHR treated with the competitive TNF-α inhibitor, etanercept, or the immunosuppressive agent mycophenolate mofetil (MMF). There were no significant differences in TNF-α receptor 1 protein expression between males (5.0 ±1.5 relative densitometry units (RDU)) and females (4.1±0.6 RDU) or in CRP levels (males: 2349±341 μg/ml; female: 2499±571 μg/ml). Etanercept was administered by IP injection in increasing doses (0.4, 0.8, 1.6 mg/kg/day) and BP was continuously monitored by telemetry. Treatment with etanercept did not alter the BP in either sex. However, 2 week treatment with MMF (p.o., 20 mg/kg/day) significantly decreased mean arterial BP in both male (151±1 to 141±1, p<0.05) and female SHR (133±1 to 124±2, p<0.05). Although males had significantly higher BP than female SHR (p<0.05), the percent decrease in BP in response to MMF was comparable between the sexes (males: -4±2% vs females: -6±3%, NS). Therefore, while the data do not support a role for TNF-α in the hypertension in SHR, the MMF data suggests an inflammatory component to the hypertension in SHR irrespective of sex.

8.19

BONE MARROW MACROPHAGES (MΦ) INFILTRATE MESENTERIC ARTERIES (MA) IN DOCA-SALT HYPERTENSIVE RATS

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The recruitment and activation of Mϕ in the vascular adventitia is associated with hypertension. Mice without Mϕ colony-stimulating factor, have reduced inflammation and are protected against hypertension induced vascular damage. We reported an increased number of Mϕ with high level of p22Phox and TNF-α in the MA adventitia in DOCA-salt rats. We tested the hypothesis that infiltrated Mϕ are derived from the bone marrow and not the spleen. Using flow-cytometry we found no difference in CD163 and CD11b level on splenic Mϕ from DOCA-salt rats compared to sham. We found the percent of CD11blow/CD163- blood Mϕ in DOCA-salt rats was higher compared to sham. In CD11blow Mϕ population, CD163 level was higher in DOCA-salt rats compared to sham (p<0.05). The percent of CD11bhigh/CD163high and CD11blow/CD163low peritoneal Mϕ were higher and lower respectively in DOCA-salt rats compared to sham. In CD11bhigh Mϕ population, CD163 level was higher in DOCA-salt rats compared to sham (p<0.05). The data suggests the infiltrated Mϕ in the MA of DOCA-salt rats were bone marrow-derived. Mϕ change from CD11blow/CD163-to CD11bhigh/CD163high as they migrate from the blood into the peritoneal cavity in DOCA-salt rats. High level of CD11b, a subunit of αMβ2 integrin, allows Mϕ adhering to MA adventitia, and cross-linking of Mϕ CD163 can induce the production of pro-inflammatory cytokines. (Supported by HL70687)

8.20

TNF-A PROMOTES HYPERTENSION AND RENAL INJURY IN THE CHRONIC INFLAMMATORY DISORDER SYSTEMIC LUPUS ERYTHEMATOSUS

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Immune system activation has been implicated in the progression of hypertension in both humans and experimental models. The role that the cytokine TNF-□ has in the development of hypertension remains unclear. We tested whether TNF-□ mechanistically contributes to hypertension and renal injury in a chronic autoimmune inflammatory disorder, systemic lupus erythematosus (SLE). A mouse model of SLE (Female NZBWF1) and controls (Female NZW) were treated with vehicle (saline) or the TNF-□ antagonist etanercept (ETAN 0.8 mg/kg, S.C.) for 4 weeks. SLE mice have increased MAP compared to controls (in mmHg, 150±5 vs. 113±5, p<0.05). SLE mice treated with ETAN had lower pressure (132±3, p<0.05) compared to vehicle treated SLE mice. MAP in control mice was not affected by ETAN treatment (117±5). Urinary albumin (in □g/mg creatinine, 28742±9032 vs. 1075±883 p<0.05), glomerulosclerosis index (2.5±1.6 vs. 0.0±0.0, p<0.05), and renal cortex CD68+ staining (% area, 4.75±0.80 vs. 0.79±0.12, p<0.05) were greater in SLE mice compared to controls. ETAN treated SLE mice had significantly lower urinary albumin (8154±3899), glomerlosclerosis index (0.1±0.1) and CD68+ staining (2.28±0.32). Control animals were not affected. Renal cortex NADPH oxidase activity was greater in SLE mice compared to controls (in RLU/mg protein, 10718±1276 vs. 7584±229, p<0.05) and this was blunted in ETAN treated SLE mice (6645±490, p<0.05). Phosphorylated NF□B in the renal cortex was greater in SLE mice compared to controls (p<0.05) and was reduced after ETAN treatment. These data support TNF-□ as an important mediator of hypertension and renal injury during chronic inflammatory disease, likely through NF□B activation and the generation of superoxide in the renal cortex.

8.21

AGE-ASSOCIATED INFLAMMATION AND OXIDATIVE STRESS ALTER RENAL ANGIOTENSIN II AT1 AND DOPAMINE D1 RECEPTOR FUNCTION AND CONTRIBUTE TO HYPERTENSION IN AGING

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Kidney AT1 (AT1R) and D1 (D1R) receptor play key role in maintaining sodium homeostasis and blood pressure (BP). Earlier we reported that age-related increase in inflammation (IF) and oxidative stress (OS) are associated with reduced D1R function in old Fischer 344 (F344) rats. Also, mimicking age-associated IF and OS in adult Sprague-Dawley rats with lipopolysaccharide reduces D1R function and causes salt-sensitive hypertension. However, AT1R function in these studies was not explored. Therefore, we hypothesized that kidney AT1R function is enhanced in aging. To test this, we used Fischer 344 x Brown Norway (FBN) adult and old rats. BP and natriuretic responses to AT1R antagonist candesartan and D1R agonist SKF38393 were measured. Also, OS marker, transcription factors (NFκB, Sp3) and AT1R and D1R mRNAs were determined in proximal tubules (PTs). We found higher BP in old FBN rats. OS marker in PTs increased and natriuretic response to SKF38393 decreased in old FBN rats similar to old F344 rats. And, natriuretic response to candesartan was potentiated in old than in adult FBN rats. The nuclear NFκB and Sp3 levels were higher together with higher AT1R and lower D1R mRNA levels in PTs of old FBN rats. Taken together, studies in F344 and FBN rats suggest that age-associated IF and OS by altering gene expression increase AT1R and decrease D1R function contributing to hypertension in aging. And, NFκB as well as Sp3 seem to play critical role in this phenomenon. Funding source: NIH/NIA AG25056, AG29904.

8.22

UPREGULATION OF CCR2 AND ITS LIGANDS CCL7 AND CCL8 IN THE VASCULAR WALL DURING HYPERTENSION IN MICE

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Leukocytes accumulate in the vascular wall during hypertension (HT) and contribute to the vascular inflammation associated with this condition. Chemokine ligand-receptor interactions are key mediators of leukocyte trafficking into the vessel wall and thus represent promising therapeutic targets. In this study we characterized the changes in expression of chemokine receptors and their ligands that accompany angiotensin (Ang) II-induced HT in mice. C57BL6J mice were infused with Ang II (0.7 mg.kg-1.d-1) or saline for 14 d via osmotic micropumps. Systolic blood pressure (SBP) was measured by tail cuff and aortas were removed, with perivascular fat intact, for analysis of chemokine ligand and receptor expression by SYBR green-based PCR array and Taqman qPCR. SBP was elevated at 14 d in Ang II (146.2±8.3 mmHg) vs saline (117.1±4.4 mmHg) -treated mice. PCR array showed that mRNA expression of CCR2, and its ligands CCL7 and CCL8, were elevated in aortas of Ang II-treated mice. Taqman qPCR confirmed that expression of CCR2, CCL7 and CCL8 was 2.2-, 2.4- and 3.0-fold higher respectively in Ang II- vs saline-treated mice (n=5). However, expression of CCL2, the conventional ligand for CCR2, was not altered. These findings highlight the CCR2-CCL7/CCL8 axis as a potential therapeutic target in HT. Future studies will determine which leukocyte subsets depend on this interaction for entry into the vessel wall and whether disruption of CCR2-CCL7/CCL8 prevents vascular inflammation in HT. Funded by the National Health and Medical Research Council of Australia and the Heart Foundation of Australia.

8.23

A ROLE FOR AT1 RECEPTORS ON T LYMPHOCYTES IN ANGIOTENSIN II-DEPENDENT HYPERTENSION

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To examine the role of AT_1 receptors on T lymphocytes in the pathogenesis of Ang II-dependent HTN, we intercrossed mice carrying a floxed gene for the AT_{1A} receptor ($Agtr1a^{flox/flox}$) with mice harboring Cre recombinase under control of the CD4 promoter, thereby removing AT_{1A} receptor-mediated responses from T cells alone (Cre^+ $Agtr1a^{flox/flox}$=TKO). Compared to Cre^- littermates (Controls), the TKOs had a 90% reduction in AT_{1A} mRNA expression in $CD4^+$ and $CD8^+$ T cells ($p<0.0001$) but had preserved AT_{1A} expression in B cells, kidney, and heart. Following uni-nephrectomy, the TKO and Control mice ($n\geq11$) had similar blood pressures measured by radiotelemetry at baseline (115±1 vs. 116±2 mm Hg; p=NS) and throughout 4 weeks of Ang II infusion (162±6 vs. 161±4;p=NS). Despite similar blood pressures, the TKOs had 40% more albuminuria than Controls following Ang II infusion (1274±90 vs. 907±92μg/mg creatinine;p<0.009). TKO kidneys also showed enhanced mRNA expression of the T cell chemokine CCL5 (p=0.03) leading to a higher proportion of dense perivascular T cell infiltrates in the TKO kidneys (37.5 vs. 4.3%; p=0.004, Fisher's Exact). These T cell infiltrates were associated with exaggerated renal mRNA expression of IL-1β, TNF-α, and IFN-γ ($p\leq0.04$ for each). Thus, in Ang II-induced hypertension, activation of AT_1 receptors on T cells paradoxically limits their accumulation in the kidney, constrains renal expression of pro-inflammatory cytokines, and ameliorates glomerular proteinuria.

8.24

EFFECTS OF ANG II –INDUCED HYPERTENSION ON THE HEART IN MICE WITH TH1/TH2 PROFILES

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We hypothesized that angiotension II (Ang II)–induced hypertension causes more severe cardiac damage in Th2 responder mice than in Th1 mice. We used C57BL/6J mice (Th1) and Balb/c mice (Th2). Mice were split into 4 groups: 1) C57 BL/6J vehicle, 2) C57BL/6J Ang II, 3) Balb/c vehicle, and 4) Balb/c Ang II. Ang II (1.4 mg/kg/day) was given for 8 weeks s.c. Balb/c mice showed 1) enlarged left ventricular (LV) chamber reflected by higher LV diastolic dimension (LVDd), thinning posterior wall thickness (PWT), 2) lower LV ejection fraction (LVEF), and 3) 2-fold higher LV collagen compared to C57 BL/6J at baseline. Both strains had similar baseline systolic blood pressure (SBP) at 112 ± 3 mm Hg for C57BL/6J mice and 108 ± 2 for Balb/c, which were increased to similar levels after Ang II infusion with SBP values of 156 ± 2 and 150 ± 4 mmHg, respectively. They also had similar cardiac hypertrophy. However, in Balb/c vs C57Bl/6J mice, Ang II induced greater LV chamber dilatation (3.7±0.2 vs 2.8±0.1 mm), wall thinning (0.85±0.02 vs 1.03±0.02 mm) and fibrosis (18.1±2.0 vs 7.55±0.55 μg/mg dry LV), leading to congestive heart failure shown by dramatically decreased LVEF (49±3 vs 77.5±1.9 %) and lung congestion indicated by almost a 2-fold increase in lung weight (LW)/BW ratio (p = 0.001). Ang II-hypertension was also associated with severe deterioration of endothelium-dependent relaxation of aorta from Balbc compared to C57Bl/6J mice. Thus, dominant Th2 function appears to be associated with chronic cardiac and arterial damage; and hypertension accelerates LV remodeling and dysfunction, leading to dilated cardiomyopathy (DCM) in Th2 mice. Ang II-induced hypertension in Balb/c mice may be an ideal model of DCM caused by immune imbalance.

8.25

PROTEIN TYROSINE PHOSPHATASE 1B DELETION INCREASES BLOOD PRESSURE IN TH2 RESPONSIVE MICE ONLY BY INCREASING PRO-INFLAMMATORY CYTOKINES SECRETION

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Hypertension is associated with an inflammatory process involving T cells-induced cytokines release. Whereas cytokine secretion and signaling require tyrosine phosphorylation, the role of protein tyrosine phosphatase 1B (PTP1B) in cytokine signaling and blood pressure (BP) regulation is unknown. To determine its role in these two latter processes, PTP1B was deleted in mice on either a Th1 (C57) or a Th2 (Balb/C) responsive background. BP recording in these 2 strains demonstrated that PTP1B deletion elevated BP in Th2 responsive mice (Balb/C: 99±5 vs

PTP1BxBalb/C: 120±3) while it had no effect on the Th1 responsive mice (C57: 105±3 vs PTP1BxC57: 110±3 mmHg). Plasma cytokine profile showed that PTP1B deletion suppressed Th2-derived anti-inflammatory cytokine secretion (IL-10, Balb/C: 53±7 vs PTP1BxBalb/C: 19±2 pg/mL) and increased Th1-derived inflammatory cytokine production (IL- 6, Balb/C: 11±1 vs PTP1BxBalb/C: 24±5, TNF[alpha], Balb/C: 45±4 vs PTP1BxBalb/C: 99±26 pg/mL) in the PTP1BxBalb/C mice. IL-10 and TNF[alpha] secretion were not affected by PTP1B deletion in mice on Th1 responder background but levels of Th1 derived pro-inflammatory cytokines were reduced. (IL-6, C57: 25±1 vs PTP1BxBxC57: 4±1, INF[gamma], C57: 34±5 vs PTP1BxC57: 19±3 pg/mL). These data suggest that a Th2 responder background is required to produce hypertension in response to PTP1B deletion. This differential effect may reflect that different contributions of Th1 and Th2 cells to hypertension secondary to deletion of PTP1B.

8.26
DIFFERENCES IN PULMONARY HYPERTENSIVE CONDUIT ARTERY MATERIAL PROPERTY CHANGES OF THE HUMAN, CALF AND RAT

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We hypothesize that rat conduit pulmonary arteries remodel and stiffen in pulmonary hypertension through a more collagen-dependent mechanism compared to calves or humans, and that these large mammals remodel predominantly through more elastin-dependent mechanisms. Pulmonary vascular physiology of small mammals has been shown to respond to hypoxia-induced pulmonary hypertension (PH) differently than large animals (e.g. calf and pig)1. Given the physiological similarities in vascular remodeling of large animal models to the changes typically seen in humans, we investigated the possibility that these differences may result in physiologically disparate material property changes in-vivo between rodent and human vascular tissues. Arterial tissues from humans, calves and rats were studied. PH was induced by hypobaric-hypoxia for the rat and calf models, PH etiology of human tissues was not considered. Tissue mechanical properties were tested by uniaxial loading of arterial strips in a materials testing system. Our results show that PH rats demonstrate a statistically significant reduction in the material stretch at which collagen begins to carry load. However, this phenomenon is not present in the human or calf, indicating that important inter-species differences exist between the large and small mammalian PH model systems. 1) K.R. Stenmark et.al., Circulation Research, vol. 99, 2006.

8.27
IMPACT OF RESIDUAL STRETCH ON COLLAGEN ENGAGEMENT IN THE MAIN PULMONARY ARTERIES OF HEALTHY AND PULMONARY HYPERTENSIVE CALVES AT PHYSIOLOGICAL PRESSURES

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Methods: In-vivo pressure-diameter data for the main pulmonary artery (MPA) were obtained from newborn calves exposed to 2 weeks of normoxia or hypobaric hypoxia [1]. After sacrifice, harvested calf MPA rings were cut radially and photographed for open ring dimensions. MPA in-vivo circumferential stretch (λc) was obtained from the in-vivo diameter data using the incompressibility condition and open ring as the reference state. Using a two-family collagen fiber model [2] and the transition λc (at which collagen begins to carry load) estimated from uniaxial tests [3], the transition stretch in the fiber direction can be estimated. In vivo, the critical λc occurs when the fiber stretch reaches the transition stretch. Results: In-vivo λc increased from the inner to outer wall. For the control group, λc were always less than the critical λc, while for the hypoxic group, λc at the outer wall were close to the critical λc at end-diastole and □c in the whole artery were larger than or around the critical λc at end-systole. Conclusions: Collagen engages only in the hypoxic group, implying higher right heart afterload. Considering residual stretch causes collagen near the outer wall to engage earlier than that close to the inner wall.

8.28
IMMUNE RESPONSES IN THE LUNG TO SECOND HAND CIGARETTE SMOKE

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Aim: The main risk of chronic obstructive pulmonary disease is cigarette smoke (CS) exposure. We investigate the possible impaired regulation of inflammatory responses upon CS exposure. Methods and Results: We exposed male Spraque Dawley rats with a mixture of side- and mainstream CS. Significant alveolar airspace enlargements were observed after 2 mo of CS exposure versus in room air exposed controls, further augmented after 4 mo. Moreover, flow cytometric staining showed an increase in regulatory T cells (CD25+CD4high) in blood of CS exposed rats. We saw a significant increase in CD39+ macrophages (CD4low) in the lung, indicating an important role of adenosine signaling. Lung sections showed increased foamy macrophages in the alveolar space of CS exposed rats. Interleukin-18 was upregulated in the bronchoalveolar lavage fluid of CS exposed rats. Exposure of CS also upregulated pro-inflammatory cytokines in the plasma, whereas Rantes and leptin were downregulated. In the lung IL-6, IL-1a, IL-1b as well as leptin were downregulated. Moreover, CS differentially inhibits lung macro and microvascular endothelial cell proliferation in vitro and alpha-antitrypsin treatment exhibits a protective effect. Conclusions: Exposure to CS leads to significant differences in the regulation of activated lymphocytes and cytokines, and our data suggest new targets for therapeutics to interfere with the disease progression. Funded by AHA 0735388N, FAMRI CIA 072053 and Bixler Family Foundation.

8.29
AC-SDKP REDUCES CARDIAC CROSS-LINKED COLLAGEN, NFKB EXPRESSION, INFLAMMATION AND LYSYL OXIDASE EXPRESSION IN ANGIOTENSIN II INDUCED HYPER-TENSION

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We hypothesized that N-acetyl-Ser-Asp-Lys-Pro (Ac-SDKP) not only reduces cardiac fibrosis but also collagen cross-linking and that these effects are in part due to reduction in cardiac NFκB, inflammation (T-lymphocyte) and lysyl oxidase (LOX) expression. Male Lewis rats were divided into 4 groups (n=6-8, each): 1 (vehicle); 2 (Ac-SDKP 800μg/kg/day); 3 (AngII 750μg/kg/day) and 4 (AngII+Ac-SDKP). Systolic blood pressure (mmHg) was recorded for 3 weeks, after which collagen cross-linking was quantified by measuring insoluble (IC), soluble (SC) and total (TC) collagen (μg/mg dry weight tissue). Moreover, NFκB activity (EMSA assay), CD4+ and CD8+ lymphocytes (immunohystochemestry; #/mm2) and LOX expression by Western blot were quantified.
Results (X±SEM)

	Vehicle	Ac-SDKP	AngII	Ac-SDKP+AngII
BP	125±3	122±3	202±15*	205±8*
IC	9.5±1.3	10.7±1.3	16.3±1.6*	10.0±0.9#
SC	2.0±0.2	1.7±0.4	2.7±0.7	2.9±0.7
TC	11.7±1.3	12.5±1.5	19.0±1.8*	13.0±0.9#
NFκB	11.5±1.4	15.0±3.5	24.4±1.8*	15.7±2.0#
CD4+	29.4±6.5	19.7±4.52	54.9±4.6*	37.9±6.6#
CD8+	3.2±1.4	4.9±1.26	12.4±1.2*	3.8±0.9#
LOX	0.1±0.03	0.1±0.04	0.2±0.03*	0.1±0.03#

* and # p<.05 ANGII vs Vehicle and ANGII+AC-SDKP respectively.
Conclusion: Ac-SDKP decreased cardiac cross-linked collagen (IC) which is important for reducing myocardial stiffness in hypertension. This antifibrotic effect of Ac-SDKP may be mediated by decreasing NFκB, CD4+ and CD8+ lymphocyte infiltration and LOX expression in the heart. Grant#: NIH HL028982 to OAC.

8.30
ETA RECEPTOR ACTIVATION INCREASES RENAL T CELLS AND CELLULAR PROLIFERATION DURING CHRONIC ANG II INFUSION INDEPENDENT OF HYPERTENSION

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T-cells and endothelin (ET-1) both contribute to angiotensin II (Ang II)-dependent hypertension. We hypothesized that ET-1, via the ETA receptor, facilitates T-cell infiltration in the kidney during Ang II-dependent hypertension. C57Bl/6 mice were divided into four treatment groups: saline-infused via an osmotic mini-pump (saline); Ang II-infused mice (490 ng/kg/min; Ang II); Ang II-infused mice treated with an ETA receptor antagonist (ABT-627 5 mg/kg/day; Ang II+ABT-627); or Ang II-infused mice given triple antihypertensive therapy (hydralazine 30 mg/kg/day, reserpine 0.6 mg/kg/day, and hydrochlorothiazide 12 mg/kg/day; Ang II+TTx). After 14 days, Ang II increased blood pressure and the number of CD3+ and proliferating cells in the kidney. Mice treated with ABT-627 had lower pressures, decreased CD3+ cells, and less proliferating cells in the renal cortex. Triple therapy treated mice had similar reductions in pressure, but no change in renal cortical CD3+ cells compared to kidneys from Ang II hypertensive mice. In the outer medulla, both ABT-627 and TTx reduced the number of CD3+ cells and macrophages. These data suggest that ETA receptor activation in Ang II

hypertension increases CD3+ cells and proliferation in the renal cortex independent of changes in blood pressure, while changes in inflammatory cell numbers in the renal medulla are blood pressure-dependent.

8.31

SUPPRESSION OF ANGIOTENSIN II INDUCED HYPER-TENSION WITH ADOPTIVE TRANSFER OF REGULATORY T-LYMPHOCYTES

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The $CD4^+$ lymphocyte function has been shown to be an integral element of angiotensin II (AngII) induced hypertension. $CD4^+$ lymphocytes can be grouped into four subsets, namely TH1, TH2, TH17, and regulatory T-lymphocytes (Treg). The Treg lymphocyte suppresses the immunological activity of the TH1, TH2, and TH17. Therefore, our hypothesis is that adoptive transfer of Treg lymphocytes may suppress AngII induced hypertension. In C57BL/6j male mice, 490 ng/min/kg of AngII with infused over 21 days. Arterial blood pressure was measured with the invasive telemetry system (DSI). On day 21 the systolic blood pressure of the AngII group was165±4 mmHg compared with 110±3 mmHg in the control. The aortic infiltrate Th17 (RORγT) was increased by 5-fold and Treg (FoxP3) decreased by 2-fold with AngII infusion compared with control ($P<0.01$). Adoptive transfer of 3,500 $FoxP3^{GFP}$ $CD4^+$ lymphocytes/recipient mouse reduced the systolic blood pressure to 136±5 mmHg, decreased the TH17 (RORγT) by greater than 10-fold and increased the Treg (FoxP3) by 16-fold ($P<0.001$). Similarly, the TLR2 agonist, Pam3cys-SKKKK at 50µg/mouse/week induced Treg (FoxP3) by 5-fold and reduced the splenic Th17 (RORγT) by greater than 10-fold in AngII treated mice compared with AngII treated only ($P<0.001$) and reduced the arterial blood pressure by 41 mmHg. The percentage of $FoxP3^{GFP}$ aortic lymphocytic infiltrates was 2.5% for control, 0.9% for AngII only and 7.1% for Pam3cys + AngII. Finally, to confirm that the TH17 (RORγT) lymphocyte is critical in the development of AngII induced hypertension, we infused AngII into ROR□T KO mice and no hypertension was detected. These data underscore the role of the $CD4^+$ subtypes, Treg and TH17 in the pathogenesis of arterial hypertension.

8.32

ADOPTIVE TRANSFER OF HYPERTENSION WITH $CD4^+$ LYMPHOCYTES

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Recently pathogenesis of hypertension has recently been understood to be reliant on the $CD4^+$ lymphocyte. The aim of this murine study was to determine if hypertension (HTN) could be adoptively transferred (AT) with $CD4^+$ lymphocytes. C57BL/6j female mice were given a low dose of the nitric oxide inhibitor L-NAME (12.5 mg/L) in the drinking water plus an 8% NaCl diet. The L-NAME/salt mice became progressively hypertensive and on day 28 and the systolic blood pressure was 169±4 mmHg compared with 114±2 mmHg in the untreated normotensive controls ($P<0.01$). On day 28 the splenocytes were harvested from these 2 groups, purified with negative selection beads, and adoptively transferred into naive syngeneic severe combined immunodeficient (SCID) mice at a dose of $8x10^6$ $CD4^+$ lymphocytes/mouse. The systolic blood pressure of the sham SCID was 116±2 mmHg, 114±2 mmHg for the normotensive-AT, and 152±2 mmHg for the hypertensive-AT on day 28 post-AT. The recipient SCID mice received no other treatments beyond that of the AT. The donor lymphocytes showed an increased TH1 and Th17 immune profile. The lymphocytic infiltrates of the recipient aorta showed an increase of TH1 lymphocytes from 33% to 51% and Th17 lymphocytes from 11% to 36% with no change in the TH2 when comparing the normotensive-AT with the hypertensive-AT groups, respectively. Analysis of the arterial vasculature extracellular matrix showed an increase in collagen types I and III, TIMPs, and lysyl oxidase expression and enzymatic activity with mixed expression of MMPs when comparing the hypertensive-AT with normotensive-AT groups. Renal function analysis of all recipient-AT mice was within normal limits. These data support that hypertension can be adoptively transferred with purified $CD4^+$ lymphocytes and which is associated with vascular extracellular matrix remodeling.

8.33

INTERFERON-GAMMA RECEPTOR KNOCK-OUT MICE DEVELOP LESS CARDIAC AND TUBULAR DAMAGE DE-SPITE ALBUMINURIA IN ANGIOTENSIN II-INFUSED MICE

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Angiotensin (Ang) II, a major mediator of hypertension-induced cardiac and renal damage, has proinflammatory properties. Innate immunity is important to this process. However, the role of interferon (INF)-g signaling is unexplored. We analyzed INF-g receptor deficiency (INF-gR-/-) in Ang II-infused hypertensive mice and included sham controls. Ang II-infused IFN-gR-/- mice were markedly resistant to cardiac hypertrophy compared to Ang II-infused wild-type mice. Both groups developed a similar degree of (telemetric) hypertension (189±3 vs. 187±3 mm Hg, respectively). Amelioration of cardiac morphology in Ang II-infused IFN-gR-/- mice was accompanied by an improvement in arrhytmogenic electrical remodeling, indicating the functional significance of the enhanced cardiac morphology. In the kidney, tubular damage was reduced in Ang II-infused IFN-gR-/- mice, as indicated by decreased neutrophil gelatinase-associated lipocalin (Ngal) mRNA expression. Surprisingly, IFN-gR-/- mice showed significantly increased albuminuria compared to Ang II-infused wild-type mice (2053±506 vs. 406±71 µg/d, respectively). Both sham groups showed no albuminuria (4±1 vs. 2±0.5 µg/d). In conclusion, IFN-gR deficiency ameliorates Ang II-induced cardiac damage and accounted for the improved electrical remodeling, independent of blood pressure effects. In the kidney, IFN-g signaling acts in a cell type-specific manner. Tubular cells are protected by the absence of the IFN-gR, while cells regulating the glomerular barrier appear to require the IFN-gR in the presence of Ang II. The results underscore a novel but complex role of signaling in the immune response to Ang II.

10.0: INFLAMMATION AND THE METABOLIC SYNDROME

10.3

INABILITY TO MANAGE GUT MICROBIOTA CAN RESULT IN CHRONIC LOW-GRADE INFLAMMATION THAT RESULTS IN METABOLIC SYNDROME

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Metabolic syndrome is a constellation of metabolic abnormalities (hyperglycemia, hyperlipidemia, insulin resistance, obesity, and hypertension) that increase the risk of type 2 diabetes and cardiovascular disease. Here, we show that metabolic syndrome develops in mice genetically deficient in toll-like receptor 5 (TLR5), a component of the innate immune system that recognizes bacterial flagellin present on flagellated pathogenic and commensal bacteria. Loss of TLR5 rendered the mice unable to protect the intestine from challenge by Salmonella typhimurium and, moreover, made them prone to developing spontaneous colitis in the absence of pathogenic challenge. However, the majority of the mice developed low-grade chronic inflammation that lacked histopathologic evidence of inflammation but, rather was characterized by elevated expression of a variety of genes associated host defense and inflammation. Compared to wild-type littermates, the mutant mice displayed an increase in body mass, fat mass, were hyperphagic, and showed alterations in the bacterial species comprising their gut microbiota. Transfer of gut microbiota from TLR5-deficient mice to wild-type germ-free mice conferred many features of metabolic syndrome to the recipients. Food restriction prevented obesity but not insulin resistance in the TLR5-deficient mice. Thus, innate immune-mediated changes in gut microbiota can contribute to the development of insulin resistance and other hallmarks of metabolic syndrome in mice, supporting the emerging view that gut microbiota play a key role in metabolic disorders.

12.0: INFLAMMATION, IMMUNITY, HYPERTENSION AND RENAL DISEASE

12.2

IMMUNE MECHANISMS IN SALT-SENSITIVE HYPER-TENSION

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Impaired pressure natriuresis and infiltration of immune cells in renal tubulointerstitium are features of experimental SSHT. Since immunosuppression ameliorates or prevents SSHT and Heat Shock proteins (HSP) are overexpressed in the kidney in SSHT, we studied if HSP70 participates in the pathogenesis of SSHT acting as a self-antigen, thereby causing a chronic low-grade inflammation of the kidney. SSHT was induced in male Wistar rats with transient (3 weeks) oral L-NAME administration prior to a high salt diet. HSP70 induced a proliferative response in T lymphocytes of rats with SSHT. Immune tolerance to HSP70 (confirmed by skin tests and suppression of T cell proliferative response) was induced with intraperioneal injections of a highly preserved HSP aminoacid sequence. Tolerization resulted in reduced renal immune cell infiltration and maintenance of systolic blood pressure (Tail cuff and intraarterial) within normal limits (133±6.7 mmHg) during 12 weeks with a 4% salt diet, in contrast ($p<0.001$) with the non-tolerized group that presented renal inflammation (lymphocyte and macrophage infiltration and increased Il6 abundance) and hypertension (160±8.07 mmHg). Results indicate that HSP70-induced autoimmunity plays a role in the development of renal tubulointerstitial inflammation and in hypertension driven by a high salt diet. Further investigations may provide novel pathogenic insights and therapeutic approaches to the treatment of hypertension. (FONACYT grant 2005000283). Rodriguez-Iturbe B et al (2004). Am J Physiol Renal Physiol

286,F606-F6166. Vaziri ND & Rodriguez-Iturbe B (2006). NATURE Clin Pract Nephrol 2, 582-293.

12.3
TREG LYMPHOCYTES IN VASCULAR AND KIDNEY DISEASE

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Recent data suggest that T suppressor lymphocytes including regulatory T cells (Treg), which express the transcription factor forkhead box P3 (Foxp3), play critical roles in hypertension and progression of vascular remodeling as well as in atherosclerosis and kidney disease. We investigated the potential implication of Treg in consomic rats bearing chromosome 2 of Brown-Norway rats on a background of Dahl salt-sensitive rats. Blood pressure was lower and vascular remodeling less pronounced in the consomic, vascular inflammatory mediators were decreased, Treg and immunosuppressant cytokines such as IL-10 were increased, whereas INF-γ and IL-6 were reduced. Infusion of angiotensin II raised BP and induced vascular remodeling, inflammatory mediators and oxidative stress in mice, al of which were blunted by Treg adoptive transfer. Transferred Treg cells also ameliorated cardiac damage and improved electric remodeling in angiotensin II-infused mice, independently of BP-lowering effects, which suggests a role of Treg in the pathogenesis of hypertensive cardiac damage. Renal ischemia/reperfusion injury was also improved by Treg adoptive transfer. In summary, Treg suppress cardiovascular and renal injury and BP elevation in part through anti-inflammatory actions.

12.4
INTERLEUKIN 17 AND HYPERTENSION

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T cells are required for the full development of angiotensin (Ang) II-induced hypertension. However, the specific subsets of T cells that are important in this process are unknown. Th17 cells represent a novel subset that produces the proinflammatory cytokine interleukin 17 (IL17). We found that Ang II infusion increased IL17 production from T cells and IL17 protein in the aortic wall. Blood pressure in response to 4 weeks of Ang II infusion was significantly blunted in IL17-/- mice. Vessels from IL17-/- mice displayed preserved vascular function, decreased superoxide production, and reduced aortic T cell infiltration in response to Ang II. Gene array analysis on cultured aortic smooth muscle cells revealed that IL17, in conjunction with TNFalpha, modulated expression of over 30 genes, including a number of inflammatory cytokines/chemokines. Serum levels of IL17 in diabetic humans were significantly increased in those with hypertension compared to normotensive subjects. We then examined the effect of IL17 on atherosclerosis by crossing IL17-/- mice with ApoE-/- mice. Three months of high fat diet induced a marked increase in production of IL17 from T cells of ApoE-/- mice. Despite favorable effects on weight, cytokine profile, and vascular reactive oxygen species, atherosclerotic lesion area was unchanged in IL17/ApoE-/- mice. We conclude that IL17 does not affect plaque size but is critical for the maintenance of Ang II-induced hypertension and vascular dysfunction and might be a therapeutic target for this widespread disease.

12.5
T-LYMPHOCYTES IN THE KIDNEY EXAGGERATE HYPERTENSION AND RENAL DAMAGE IN SALT-SENSITIVE HYPERTENSION

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Elevated dietary NaCl intake leads to hypertension and renal damage in the Dahl Salt-Sensitive (SS) rat. Experiments demonstrated a significant increase in infiltrating T-cells and elevated oxidative stress in the kidney of Dahl SS rats following an elevation of dietary NaCl. Studies were performed to examine the functional role of the infiltrating cells in Dahl SS hypertension. Chronic administration of mechanistically-different immunosuppressive agents, mycophenolate mofetil or tacrolimus, blocked the infiltration of T-cells that occurs in the kidneys of Dahl SS rats fed elevated NaCl. The decrease in infiltrating cells was associated with decreased oxidative stress in the kidney and an attenuation of hypertension and renal damage. Further studies demonstrated that infiltrating T-cells are enriched in NADPH oxidase. Finally, it was demonstrated that tempol, a superoxide scavenger, blunts salt-sensitive hypertension in the Dahl SS, indicating that oxidative stress participates in this disease process. Together, these data indicate that infiltrating T-cells in the kidney of Dahl SS rats fed high salt provide a source of free radical production that participates in the elevation of arterial blood pressure and the development of proteinuria and renal histological damage. (Supported by HL-29587 and DK-62803). References: De Miguel C, S Das, H Lund, and DL Mattson. T-lymphocytes mediate hypertension and kidney damage in Dahl salt-sensitive rats. Am J Physiol 298:R1136-R1142.

12.6
EARLY LIFE STRESS REDUCES RENAL FUNCTION AND ENHANCES ANGIOTENSIN II-DEPENDENT RENAL T CELL INFILTRATION AND HYPERTENSION

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Epidemiological studies have reported that an adverse environment during childhood increases the susceptibility to develop cardiovascular disease. To study this phenomenon, we have utilized a model of early life stress, maternal separation (MS), in rats. We hypothesized that MS induced increased angII-induced infiltration of T cells and macrophages, renal function and proteinuria. MS was performed in male WKY rats 3 hrs/day from day 2 to 14 of life. Aortic T cell and macrophage cell counts were similar in MS and control rats under baseline conditions at 14 weeks. Basal creatinine clearance was reduced and proteinuria was increased in MS compared to control rats. Chronic angII infusion produced a significantly larger increase in blood pressure in MS rats. AngII-induced infiltration of T cells and macrophages were significantly higher following angII infusion in aortic tissue from MS rats, while only T cells were significantly higher in renal cortical tissue from MS rats. Creatinine clearance was also reduced in MS rats infused with angII. Our findings suggest that early life stress induces an enhanced inflammatory response to angII and together with reduced renal function, mediating the increased risk to hypertension. Funding support provided by NIH P01 HL69999 and AHA.

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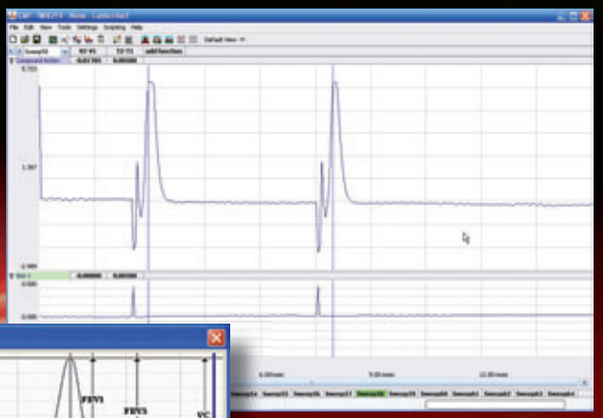

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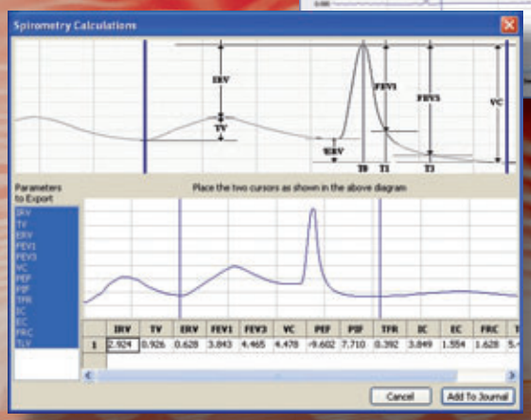

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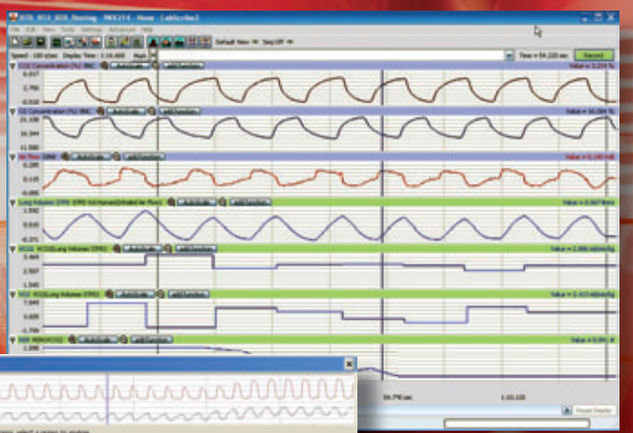

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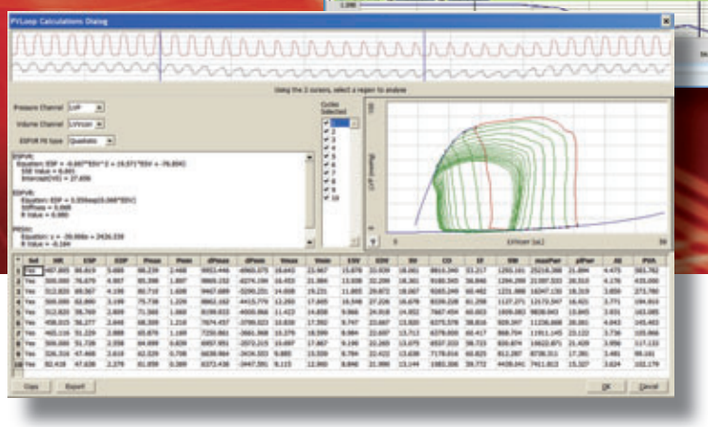

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