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Special Program and Abstract Issue

Physiology Is Alive and Well. Just Ask an Undergraduate Student

Erica A. Wehrwein

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When you think of a cutting-edge, exciting area of science, do you think of physiology? If not, you should. Physiology is the basis for medicine. Many important medical advances that we take for granted today are direct or indirect results of research conducted by physiologists. But despite the significance of our area of research, some universities are removing physiology from medical school curricula, shutting down physiology departments, or calling them by another name. Not surprisingly, this causes a great deal of angst among physiologists and is often discussed in our community.

Luckily, physiology is flourishing at the undergraduate level. Despite a smorgasbord of options for college majors in life science disciplines – including bachelor of science degrees in genetics, biochemistry, cell and molecular biology, integrative biology, etc. – more universities have started to offer physiology as a stand-alone undergraduate major instead of offering only a course on physiology. Although the major has existed in a few isolated cases for many years (e.g., University of Arizona and Michigan State University), the past decade has seen many new programs added and the conversion of several programs from kinesiology to human physiology, with 43 programs in the U.S. today. These programs typically have seen three- to fivefold increases in enrollment over the past five years.

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FASEB – In Transition

FASEB is in transition. The FASEB Board began a process of introspection in December 2015 to decide its future. In June 2016, the Board created task forces focusing on governance, membership, campus, and dues. Input to the various task forces is being provided by our FASEB Board representative Hannah Carey as well as a number of other APS members and leaders. Guy Fogleman, the FASEB Executive Director, announced his plans to retire in early 2016 and left FASEB in mid-August. The Board has hired an interim Executive Director who has been charged with evaluating FASEB's programs and activities and to make recommendations on how FASEB should proceed as an organization. Once the interim Executive Director's report is completed and the Task Force Reports are submitted, FASEB will be ready to advertise for and hire a new Executive Director.

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THIS ISSUE:

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Jessica M. Ibarra

A few weeks before the spring term ended, I opened an e-mail informing me I was eligible for promotion. Promotion? Where had the time gone? Within seconds, a lengthy to-do list formed in my mind along with a bit of panic. It was time to get my promotion packet together.

Over the next few days, I reviewed the school's standards for faculty seeking promotion. The guidelines were clear: my promotion hung on demonstrating university fit and highlighting various activities exhibiting my commitment to teaching, research, and service.

The time had come to open up my brag folder (the pile of papers stuffed into a 3-ring binder along with a collection of electronic documents saved as evidence of my commitment to scholarly work) to sort through my pile of academic activities in an effort to categorize my scholarly work into the three main areas of faculty academic responsibility: teaching, research, and service.

The Challenge

Sorting out academic activities into the teaching and research piles was a straightforward task. Instructional activities such as course design, course revision, mentoring, and undertakings involving the classroom and students were easily placed in the "teaching" pile of demonstrated activities. My work in the basic sciences was not difficult to identify and classify into the "research" pile. The guidelines for what was and was not teaching and research scholarship were well defined. Most of us agree on what type of academic work is deemed teaching or research. But what about service? The faculty role most difficult for me to grasp was service.

As I considered the range and frequency of service activities I had undertaken over the years, I realized I had accumulated many service experiences. Simply listing the various service work as "community" or "university" service would not do justice to the scope and magnitude of each of these service commitments.

Much of my service work occurred out in the community and as an individual effort to conduct science outreach, to promote STEM, higher education, health profession careers, physiology, and service learning. The best way to demonstrate my commitment to students, the university, the community, and my profession would require I highlight and showcase my service activities. But how?

This is where I ran into a problem: the guidelines offered by the university were not sufficient to help faculty at my level to document, organize, and show the impact of diverse scholarly service activities in the promotion, rank, and tenure process. It was significantly clear that service was an important aspect of the evaluation process and essential to accomplish the mission of the university; however, more information about scholarly service was required to address service.

Immediately, I looked outside my university's faculty guidelines for a definition of scholarly service and for standards to identify, classify, describe, and show the impact of scholarly service activities. The goal was to understand the most ambiguous component of scholarly work: service. What does the academic world say about service? How frequent should service be conducted? What is the scope of work considered service? What is professional service? Does service matter?

What follows is a brief overview of scholarly service and a challenge to consider professional service as the symbiosis between scholarly service, teaching, and research endeavors in your academic life.

Service

Does your institution consider service for promotion, rank, or tenure? Is service important for your annual faculty review? How does your institution define service? How frequently should service activities occur? Well, the answers depend on whom you ask. The scope of events classified as scholarly service vary from

institution to institution, yet they all value some aspect of service as part of faculty responsibilities. Service remains an important standard of scholarship.

By tradition, service at most universities refers to the compendium of core university responsibilities necessary to meet the mission of the university/institution and to do its work. Internal (university) service is accomplished and evident when faculty conduct committee work, sponsor student organizations, chair a committee, direct a program, chair a department, conduct peer evaluations, mentor, take on course overloads, etc.

Service also occurs when faculty use their knowledge and expertise in public service to the community or to a profession at different levels (local, regional, or national). For example, community service may involve service-learning, consulting, etc. in the city, in the county, or to the public. On the other hand, professional service refers to extramural work on activities that improves the profession or the quality of a discipline/profession.

It may take some digging around to determine the value your institution places on service or the extent to which service is measured for faculty promotion, rank, or tenure.

For faculty new to academia or those interested in increasing their participation in scholarly service, you may begin to realize scholarly service can occur within any area of institutional responsibility: teach, research, and service.

Through service, faculty apply their expertise, skills, and knowledge beyond the walls of the classroom (and often the university). Most importantly, new faculty members demonstrate their commitment to the university, their students, and their professional community through scholarly service.

Service In Teaching

Service in teaching involves instruction activities that take place alongside civic engagement. For example, service learning, outreach, or experiential learning events are commonly recognized forms of service in teaching. In each case, faculty take students outside their classroom and place them in an environment where some form of civic or social issue is addressed. Service in teaching opportunities provides an opportunity for learning to emerge when students engage in service for real-world issues. This type of service experience is not the same as volunteering or performing community service. Service in teaching occurs when learning course content is linked directly to service.

Professional Service

Faculty perform scholarly service for work in their professional society or in organizations. Professional service includes service on commissions, agencies, review panels, organizations, charities, professional societies, advisory groups, and more. Other examples of faculty in professional service roles involves the work required to organize workshops, panels, or meetings; promote the mission of the professional society or organization; review papers, awards applications, and grants; and conduct other civil duties. Sound familiar? The work of the American Physiological Society relies on the professional service contribution of its members. Members' time, skills, knowledge, and experiences benefit the physiology community, help it reach its mission, and contribute to society at large. The collective service efforts of APS members make it possible to realize the strategic aims of the APS to promote physiology and impact the general public.

Why It Matters

Professional service benefits the society or organization you serve, since it relies on the experiences, skills, and talents of its members to move the profession and its programs forward. In the case of the APS, professional service duties performed by its membership leads to "fostering education, scientific research, and dissemination of information in the physiological sciences."

More importantly, the civic duties performed by faculty involved in professional service also benefit YOU (the faculty). Your involvement creates opportunities to make friends; develop your knowledge; form new ideas; become inspired or motivated; develop a support network; and do work you love to do, all while being a part of a national effort to "promote the discipline of physiology and enhance human and animal health."

Amalgam of Academic Life

Intentional, strategic, and well documented professional service aligns the personal, academic, and professional

visions that faculty have for themselves. When done with this in mind, professional service may be the perfect amalgam of faculty academic, professional, and personal life. Service opens the door wide open for faculty to integrate passion for their work, civic duties, leadership, and community impact into their academic duties and responsibilities.

Consequently, scholarly service stands strongly alongside the other criteria for faculty success: teaching and research. Professional service also leads to accomplishment, satisfaction, leadership skills, broadening knowledge, interactions with diverse faculty, collaborations, and influence over decision-making processes.

Professional Service Matters

The bottom line is that professional service matters. Professional service connects university resources (YOU) to pressing social, ethical, cultural, civic, and economic concerns from your communities, schools, cities, states, and country. Without the service of faculty and their involvement in professional societies and groups, the work of these organizations would not get done. For the faculty, there are many tangible (career and professional development) and intangible (friendships, satisfaction, motivation, inspiration, and support) rewards for faculty committed to professional service.

After looking more closely for guidelines and examples of scholarly service, it turns out many of my service experiences met the criteria for service in teaching and in my profession. The exercise of defining scholarly service opened my eyes to the world of the importance of and the impact of professional service. It also made me realize how well documented service in my promotion packet will help me demonstrate my personal commitment to my students, the university, my profession, and society. I am not as panicked as I used to be when thinking about service. Now, I know how to organize, describe, and categorize my service experiences and demonstrate the importance of these activities.

Lastly, I challenge you to invest yourself in professional service not just to demonstrate scholarly service (for promotion, rank, and tenure) but to bring into alignment your academic work and personal goals. ●

Jessica M. Ibarra is an Assistant Professor of Applied Biomedical Sciences in the School of Osteopathic Medicine at the University of the Incarnate Word in San Antonio, TX. Her research experience includes the study of inflammatory factors in cardiac remodeling, arthritis, and diabetes. Her current work focuses on medical education, curriculum development, and science outreach. When she is not teaching or inspiring young scientists, she is mentoring and helping students realize their health profession education/career goals. Ibarra is an active member of the American Physiological Society and is a current member of the Porter Physiology and Minority Affairs Committee, fellow of the Physiology Education Community of Practice, and secretary to the History of Physiology Interest Group. She is also a former APS Life Science Teaching Resource Community Vision & Change Scholars Program and K-12 Minority Outreach Fellow.

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Physiology Is Alive and Well. Just Ask an Undergraduate Student

Undergraduate poster session at EB 2016

Recently, we published a paper on the millennial student view of physiology. We found that:

- 78% of physiology majors have preferences for studying whole-body physiology (with 67% interested in integrative/systems physiology and 11% interested in integrative and cellular physiology;
- Students interested in cell- and molecule-level function are gravitating to other majors such as biochemistry and genetics
- Student interest in integrative physiology is aligned with interest in an applied and holistic view of human health and disease and student aspirations for careers in health care
- Physiology programs are the primary pathway for students heading into medicine, physical therapy and other allied health professions, with 85% to 90% of students in a physiology major stating career aspirations in health care.

Considering these findings, one thing seems clear: millennial undergraduate students intuitively understand that physiology is the basis for medicine. They know that choosing a major in physiology is the best way to learn more about the human body. In my opinion, physiology is alive and well; you just have to know where to look. ●

Erica A. Wehrwein is an assistant professor at Michigan State University. She leads the Physiology Majors Interest Groups (P-MIG), a consortium of physiology undergraduate programs.

This article is reprinted from the I Spy Physiology blog. The blog is geared toward the general public and aims to explain physiology in everyday life. Interested in contributing? Contact communications@the-aps.org.

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FASEB – In Transition

For me, the transition is unsettling. FASEB and the campus that I have known since I arrived at APS in 1985 may be changing. The transition is also important because FASEB and APS have been intimately associated with each other since FASEB was founded in 1912 by physiology, biochemistry, and pharmacology. APS was founded in 1887, and biochemistry and pharmacology split off of physiology in 1906 and 1908, respectively. However, the three societies continued to hold their annual meetings together, ultimately creating the Federation of American Societies for Experimental Biology (FASEB) as the umbrella under which to meet. Pathology, nutrition, and immunology joined FASEB in 1913, 1940, and 1942, respectively.

In 1947, FASEB hired Dr. Milton O. Lee as its first Executive Director, but he also served as the APS publications manager. Lee established the offices for FASEB and the member societies in the corridors of the National Academy of Sciences. However the space was cramped, and in 1954 APS member Louis Katz led a group to find a new home for FASEB and the member societies. The 38-acre Hawley Estate in Bethesda was deemed to be an appropriate future home, but FASEB did not have the funds to complete the purchase. Instead, APS purchased the property for FASEB. FASEB sold off 27 acres of the estate to pay back APS, and the six member societies and FASEB moved to the campus, which was renamed the Beaumont Campus in honor of the GI physiologist William Beaumont.

Unfortunately, the continued existence of the FASEB campus is now in jeopardy as a result of FASEB's decision to pay off a $15,000,000 loan that was used to build the East Wing of the Lee Building. Having paid off the loan, FASEB no longer has the resources needed to upgrade the office building to *class A* status, and with the glut of empty office space in the Maryland suburbs, it is likely more prudent to sell the Campus.

When I arrived at APS in 1985, there were only six FASEB member societies. The societies had representatives on the FASEB Board, but representation was size dependent with APS and ASBMB (biochemistry) having three representatives and the other societies having two each. Similarly, the member societies were assessed a portion of FASEB's expense budget based on the size of the society. FASEB's 1989 Williamsburg retreat changed the expense allocation formula as well as many other aspects of FASEB. The most significant was the decision to begin recruiting new societies to FASEB, and, in order to do so, FASEB had to move away from an assessment formula to a dues structure. The goal was for each member society to pay a fixed dues amount based on the society's number of regular members. At the time of the Williamsburg retreat, member societies were being assessed approximately $62 per regular member.

APS currently pays FASEB dues of $16 per regular member, a more cost-effective proposition for APS and the other member societies. The lower dues structure also enabled FASEB to recruit additional societies. At present, FASEB is comprised of 30 member societies, and each society has 1 voting Board representative. Although the dues amount per member has been reduced significantly, the fact that the member societies pay dues to FASEB based on their regular member count means that the largest societies – ASBMB, APS, and AAI – pay over $100,000/year in dues, whereas the smaller societies pay only $13,000 in dues. Even though there is nearly a 10-fold difference in dues paid between the small and large societies, each society has only one FASEB Board representative.

The Williamsburg retreat also contributed to several other changes. It was decided that FASEB's primary focus and reason for existence was to provide science policy advocacy and support. It was thought that a focus on science policy and advocacy would help FASEB recruit new societies. Clearly, it was the correct decision, because FASEB has now grown to 30 societies representing over 125,000 regular members. It was also decided that FASEB would no longer hold an annual meeting, relinquishing the FASEB meeting to the participating member societies who renamed it the Experimental Biology meeting.

As noted, the decision to focus FASEB on science policy and advocacy has proven to be a success. However, FASEB is not just an advocacy group. It has many other activities, including the management of the FASEB campus, which was mentioned earlier. FASEB also provides benefits management and HR services to the APS staff as well as staff for 11 other societies, including FASEB. It provides meeting management, dues and subscription processing, financial management, and association management on a fee-based, contract basis. FASEB also has a highly successful journal, *The FASEB*

Journal, and an equally successful summer research conference program, both of which provide revenue to FASEB's operational budget.

As noted, to help with the transition, the FASEB Board created a number of task forces and asked them to review and evaluate the programs and activities of FASEB and to help set its future direction. The interim Executive Director is charged with assisting with the task. Ultimately, the FASEB Board will have to review the recommendations and decide which programs and activities to keep and which to discard. In addition, the Board will also have to recruit a new FASEB Executive Director to help lead FASEB into its future.

Although I do not know what will be retained or discarded, I do have some ideas. FASEB should continue to focus on science policy advocacy to represent the 30 societies and their 125,000 member scientists. It should also strive to represent even more by flattening the dues structure and adding new societies. It should also continue to maintain or enhance *The FASEB Journal* and the FASEB Summer Research Conferences. Both are revenue producers for FASEB, and it is clear that FASEB will need revenue to maintain its focus on science policy. It should assess the value of the services that it provides to member societies and decide which are worth keeping. The future of the Beaumont Campus may be the most difficult of all decisions. The FASEB campus has been our home since it was purchased in 1954. The member societies have resided on the campus with FASEB for many years, but as FASEB has grown, fewer have decided to call the campus home. Indeed, 4 of the 6 original member societies have moved off of the Beaumont campus so that only 7 of the 30 FASEB member societies call 9650 Rockville Pike home. If the decision is to sell the campus, APS will be joining FASEB and the remaining resident societies to find a new home in the Maryland suburbs. Given our long history with the campus, many of us will be sad to say goodbye. ●

Martin Frank

Committee Reports

APS Council Holds Summer Meeting in Bethesda

The APS Council held its annual summer meeting in Bethesda, MD, July 20-22, 2016, at the Bethesda Marriott Pooks Hill. Each summer, the Council invites the APS Committee Chairs to the summer meeting to present their annual committee reports to Council. The committee reports begin on page 295 and will be posted to each committee's webpage.

Council approved the request to reserve three positions on the Awards Committee for APS members with experience in education research.

Council approved the request to revise the committee charge for the Career Opportunities Committee.

Council approved the 2015 Audit Report presented by the Finance Committee.

Council approved a request from the Joint Programming Committee to provide registration support for the unfunded APS sessions scheduled for Wednesday afternoon as part of the Society's program at Experimental Biology.

Council approved the request from the Membership and FAPS Committee to add eight (8) APS members, who were members of the National Academy of Engineering, to the inaugural class of APS Fellows. Council also approved the request to change the proposed definition of public service included in the description for the criteria for Fellow status.

Council approved the request for support for $2,000/year for 5 years ($10,500 total) to support two APS undergraduate awards ($1,000 award plus APS membership) at the Society for the Advancement of Chicanos and Native Americans in Science (SACNAS) annual meeting.

Council approved the requests for funds to be allocated for three actions in celebration of the 50th anniversary of the minority focus of the Porter Physiology Development Program: *1)* a commemorative plaque with the names of all Porter Fellows in the first 50 years of the program; *2)* production of an online and printed com-

Committee Chairs. Front *(left to right):* Ida Llewellyn-Smith, Thomas Pressley, Margarita Curras-Collazo, Caroline Rickards, Dexter Lee. Back *(left to right):* Sean Stocker, Robert Brock, Michael Sturek, Eugene Shek, Barbara Goodman, Jeffrey Henegar, Gaylen Edwards, Erica Dale

Council. Front *(left to right):* Irene Solomon, Hannah Carey, Patricia Molina, Jane Reckelhoff, Dennis Brown, Rudy Ortiz. Back *(left to right):* Jennifer Pollock, Lisa Leon, Meredith Hay, Kevin Kregel, Harold Schultz, Bill Yates, Barbara Alexander, David Gutterman, Jeffrey Osborn, Willis Samson, Robert Hester

Staff anniversaries. Front *(left to right):* Krysia Moore, Linda Allen, Peggy Choe, Brooke Bruthers, Claire Edwards, Jane Marklin. Back *(left to right)* Martin Frank, Benjamin Weston, Yang Sipper, Rameela Patel, Christina Bennett, Sean Boyer, Jane Reckelhoff

pendium of Porter Fellows with a description of their research and careers; and *3)* a celebratory event at EB 2017 to provide visibility of the program accomplishments to both Porter Fellows and potential funders.

Council approved the requested extension of membership terms for one member in 2017 and one member in 2019 for the Women in Physiology Committee.

In addition to presenting their reports, the chairs discussed the highlights of their committees' activities and programs during the past year and updated Council on the committee's goals and plans for the coming year. The chairs also submit requests for new committee programs to Council for their approval. If the program requires new financial support, a New Programs Fund request is included with the request.

Each year during the Council/Committee Chairs meeting, APS hosts an employee appreciation reception. The reception provides an opportunity for members of Council and the committee chairs to meet with the APS staff. During the reception, APS President Jane Reckelhoff thanked the staff saying, "It is such a pleasure to be here and present awards for the best staff in the world. APS has a staff average of 12.9 years of service." She stated, "It is a pleasure for me to present the staff with their service awards this year. APS is presenting 14 awards, which is a testament to the Society and its leadership. The staff helps APS run smoothly and ensures that the ideas that Council and the Chairs have become a reality."

The highlight of the reception every year is the recognition of those staff members who have worked for APS for 5 years or more (anniversary is based on 5-year intervals). Each employee celebrating an anniversary receives a certificate of appreciation and a gift certificate. This year, APS President Jane Reckelhoff presented a 35-year certificate to Linda Allen (Director of Membership and Meetings); a 30-year certificate to Krysia Moore (Journal Supervisor); 15-year certificates to Peggy Choe (Copy Editor), C. Brooke Bruthers (Senior Program Manager), Benjamin Weston (Art Editor), Mariam Capers (Peer Review), Sean Boyer (Journal Supervisor); 10-year certificates to Lucia Tayiel (Circulation Manager), Claire Edwards (Web Specialist), Rameela Patel (AR Accountant), Travis Christensen (Copy Editor), Yang Sipper (Web Editor); and 5-year certificates to Christina Bennett (Ethics Manager), Jane Marklin (Marketing Assistant). ●

Animal Care and Experimentation Committee


Jeff Henegar, Chair

During 2015-2016, the Animal Care and Experimentation (ACE) Committee visited Capitol Hill to advocate for research with animals, organized a symposium on reducing regulatory burden, and wrote letters and comments on various animal research issues. Gaylen Edwards chaired the ACE Committee through the end of 2015. Edwards assumed the role of Chair on January 1, 2016.

ACE Committee Meetings

At its fall 2015 meeting, the ACE Committee discussed plans for its EB 2016 symposium and outlined points to address in a letter to AAAS and *Science* concerning the tone of *Science's* recent coverage of animal research issues. After our business meeting, we went to Capitol Hill. ACE Committee members met with legislators and staff from 16 Congressional offices representing Missouri, Florida, North Carolina, Indiana, Georgia, New Jersey, Oregon, Mississippi, and Texas. In our meetings, we underscored the importance of animal research, explained scientists' commitment to humane treatment of animals, and described the deleterious impact of burdensome regulations as well as our concern about having access to rapid and humane transportation for research animals.

When the ACE Committee met at EB 2016, we discussed priorities for the coming year. Because this is an election year, we will not go to Capitol Hill this fall. Rather, we will use our fall meeting for in-depth consideration of several topics of interest. The committee also suggested a number of questions will be raised with OLAW and USDA about how these agencies are interpreting their oversight requirements.

2015-2016 ACE Committee Activities

EB 2016 symposium. The ACE Committee sponsored a symposium at EB 2016 entitled "Having Trouble With your IACUC?" The focus was ways to seek changes to burdensome institutional oversight policies. In my introduction, I pointed out which federal laws and regulations most frequently cause confusion for IACUCs. Anthony Comuzzie (Texas Biomedical Research Institute) focused on ways IACUCs can improve their efficiency, such as by recruiting engaged members and reviewing their policies. J. R. Haywood (Michigan State University) reviewed the highlights of reports on reducing regulatory burden published in recent years by the National Science Board and the National Academy of Sciences. He also suggested one approach IACUCs can use to rein in self-imposed burden. Barbara Hansen (University of South Florida) identified various sources of self-imposed burden and offered suggestions on what institutions should do to resolve disagreements between principal investigators and IACUCs. Linda Yang of the APS Office of Science Policy has created a webpage (*http://www.the-aps.org/mm/SciencePolicy/AnimalResearch/Regulatory-Burden*) with links to these presentations and related materials about reducing regulatory burden.

Chapter advocacy outreach. In 2016, the APS will sponsor three Advocacy Outreach speakers at Chapter meetings. This is part of a program established in 2013 to offer advocacy training outside of the EB meeting. An overview of the program with instructions on requesting a speaker is provided annually to the Chapters. Chapters' requests for speakers are honored on a first-come, first-served basis, with preference given to those that have not had a speaker recently. The talks scheduled for 2016 are:

- "Research Advocacy: Difficult Topics--Animals in Research" (Indiana Physiological Society). *Speaker:* Alicia Schiller
- "Animal Data Reproducibility" (Nebraska Physiological Society). *Speaker:* Gaylen Edwards
- "Advocacy for Science: Making a Compelling and Understandable Case" (Iowa Physiological Society). *Speaker:* Kevin Kregel

Rigor and reproducibility. Galen Edwards, previous ACE Chair, gave a keynote address on "Reproducibility in Research: Redundancy or Requirement" at the Scientists Center for Animal Welfare (SCAW) winter 2015 conference. In his talk, he identified factors that can undermine the reproducibility of animal studies

and discussed the role of veterinarians and animal care staff in addressing them. A pdf of his presentation is available upon request from the Office of Science Policy.

Ketamine. In 2015, the APS submitted a statement opposing a proposal before the World Health Organization (WHO) to classify ketamine as a drug with significant potential for abuse and to place it under international controls. Our statement objected on the grounds that the proposed level of regulation would have deleterious effects on clinical and research settings in both human and veterinary medicine. WHO ultimately rejected the proposal to restrict access to ketamine.

***Science* coverage of animal research.** Last fall, in response to the troubling tone of several articles about animal research issues, the ACE Committee drafted a letter that APS President Patricia Molina sent to AAAS CEO Rush Holt, *Science* Editor-in-Chief Marcia McNutt, and *Science* news editor Tim Appenzeller. Since then, it was announced that McNutt would leave *Science* to become the President of the National Academy of Sciences, and on May 25, it was announced that former NIGMS Director Jeremy Berg (currently at the University of Pittsburgh) would succeed McNutt as Editor-in-Chief of *Science.*

Responding to *Lab Animal* commentaries. *Lab Animal* magazine publishes a monthly column called "Protocol Review." This column presents a scenario that might come before an IACUC and provides suggestions from several IACUC administrators and researchers about how the committee should deal with the situation. ACE Committee member Sonnet Jonker of the Oregon Health & Science University expressed concerns about the responses to the scenario in the February 2016 column, which involved an investigator who resisted switching from open to laparoscopic cholecystectomies for his research on dietary cholesterol metabolism in owl monkeys. Alice Ra'anan worked with Jonker to formulate her concerns into a letter to the editor that was published in the May issue of *Lab Animal.*

Santa Cruz Biotechnology settles with USDA. On May 19, 2016, after nearly 4 years spent contesting USDA allegations of serious Animal Welfare Act (AWA) violations, antibody producer Santa Cruz Biotechnology (SCBT) agreed to settle its case with the USDA. SCBT neither admitted nor denied the violations, but it agreed to pay a record $3.5 million fine. It also agreed that it would stop producing and selling antibodies made from the blood and serum of AWA-regulated species such as goats and rabbits. An article summarizing the case against SCBT was published in *The Physiologist* in January 2016. A year and a half earlier, on July 11, 2014, APS President David Pollock wrote a letter urging SCBT President John Stephenson to bring his company's facilities into compliance with the AWA.

Future Activities

Fall meeting. The ACE Committee will meet October 18-19, 2016.

NIH review of nonhuman primate research. On September 7, 2016, NIH will hold a workshop on "the oversight framework governing the use of non-human primates in NIH-funded biomedical and behavioral research endeavors." The workshop is a response to instructions from the House Appropriations Committee for NIH to "conduct a review of its ethical policies and processes with respect to nonhuman primate research subjects. . . . to ensure it has appropriate justification for animal research protocols." According to NIH's May 24, 2016 announcement of the workshop, participants "will also explore the state of the science involving non-human primates as research models and discuss the ethical principles underlying existing animal welfare regulations and policies." The announcement went on to say that "NIH is committed to ensuring that research with non-human primates can continue responsibly as we move forward in advancing our mission to seek fundamental knowledge and enhance health outcomes."

OLAW and USDA oversight. A letter to OLAW and USDA is being prepared, with a number of questions about their interpretations of certain regulatory requirements.

Support for research animal transportation. Committee members are being encouraged to ask their institutions to adopt statements affirming the importance of research with animals and expressing support for commercial carriers that provide humane and rapid transportation of research animals.

Council accepted the report of the Animal Care and Experimentation Committee. ●

Awards Committee

Ida Llewellyn-Smith, Chair

Action Items

At its 2015 Summer Meeting, Council endorsed the Award Committee's request to increase the value of the Career Enhancement Awards (CEAs) from a maximum of $4,000 per award to a maximum of $20,000 for each Research CEA and a maximum of $10,000 for each Teaching CEA. Because the Awards Committee has generally lacked a body of members with expertise in education research, the Committee requested that Council support two Action Items to ensure the Committee has expertise to judge applications for Teaching CEAs: *1)* reserve three places on the Awards Committee for APS members with experience in education research without increasing the total number of Committee members and *2)* approve the appointment of two to four ad hoc reviewers from the APS education research community to assist with the review of applications tor Teaching Career Enhancement Awards each year. Council approved both Action Items

Career Enhancement Awards

Two categories of Career Enhancement Awards (CEAs) were established in 1995: Research CEAs and Teaching CEAs. Both established and early career professionals are eligible to apply for these awards. Previously, there had been two calls for CEA applications each year. However, with the increase in the value of the CEAs in Summer 2015 (see above), applications from 2016 onward will be submitted once a year.

The Fall 2015 round of applications was the last under the original CEA scheme, which capped funding for each CEA at $4,000. For the Fall 2015 round, the Awards Committee received six applications for Research CEAs and no applications for Teaching CEAs. Four Research CEA applications were awarded funding:

- Blair Johnson, University at Buffalo
- Flavia Souza, Louisiana State University Health Science Center
- Frank Spradley, University of Mississippi Medical Center
- Thad Wilson, Marian University College of Osteopathic Medicine

The increase in value of the Research and Teaching CEAs has resulted in significant changes in how the CEA scheme is run.

1) **Eligibility.** Applicant must now have been a member in good standing of the APS for a minimum of 2 years and hold a rank equivalent to assistant professor or above.

2) **Submission deadlines.** There is now only one call for CEA applications each year rather than two.

3) **Evaluation criteria.** In 2016, the Awards Committee replaced its 1-100 scoring system with a 1-9 NIH-style scoring system. The evaluation criteria for RCEAs were revised by a subcommittee consisting of Li Wang, Minolfa Prieto, and Maggie Alonso-Galicia. Barb Goodman, with help from Marsha Matyas, developed the first set of detailed criteria for assessing applications for Teaching CEAs. The Committee used the revised evaluation criteria to review the first round of applications for awards under the revamped CEA scheme. The Committee thanks the people who were involved in the development of new evaluation criteria for their efforts.

4) **Review process.** David Harris, who has education research experience, joined the Awards Committee immediately after EB 2016. A second person with education research experience, Robert Augustyniak, was endorsed for a term on the Awards Committee beginning in January 2017. Finally, ad hoc reviewers [Jonathan Kibble (University of Central Florida College of Medicine), Tom Pressley (Texas Tech University Health Sciences Center), Penny Hansen (Memorial University of Newfoundland), Johanna Krontiris-Litowitz (Youngstown State University)] participated in the review of 2016 applications for Teaching CEAs. We thank these volunteers for their important contribution to the evaluation of Teaching CEA applications.

For the first round of awards under the revamped CEA scheme in 2016, the Awards Committee received eight applications for Research CEAs and eight applications for Teaching CEAs. Council approved the award to Research CEAs to:

- Pooneh Bagher, Texas A&M Health Science Center
- Sarah Lindsey, Tulane University

- Lucienne Morcillo, Federal University of Rio de Janeiro
- Srinivas Sriramula, LSU Health Sciences Center
- Steven Swoap, Williams College

Teaching CEAs were awarded to:
- Lisa Harrison-Bernard, LSU Health Sciences Center
- Fernanda Marcondes, University of Campinas
- Susan Marsh, Washington State University
- Leslie Sprunger, Washington State University

Young Investigator Awards

APS Young Investigator Awards (YIA) comprise the Arthur Guyton Award for Excellence in Integrative Physiology, the Lazaro J. Mandel Award, the Shih Chun Wang Award, and the Dean Franklin Award. Only one award in each category is given annually to a postdoctoral fellow, medical resident, or early career professional. The awards are presented at the Experimental Biology meeting. In 2015, the Committee received nine applications for the Guyton Award, eight applications for the Dean Franklin Award, five applications for the Shih-Chun Wang Award, and two applications for the Lazaro J. Mandel Award. Many of the applicants for the YIAs were very impressive. The winners of the 2015 YIAs are listed below.

Arthur C. Guyton Award for Excellence in Integrative Physiology

Alain Frigon, University of Sherbrooke

Dean Franklin Young Investigator Award

Timo Reig, Department of Nephrology and Hypertension, University of California San Diego and VA San Diego Healthcare System

Lazaro J. Mandel Young Investigator Award

Jennifer Pluznick, Johns Hopkins University School of Medicine

Shih-Chun Wang Young Investigator Award

Il-man Kim, Georgia Regents University

Measuring Outcomes of the Awards Program

The Awards Committee appreciates the need to measure the success of the Career Enhancement and Young Investigator Awards and to determine whether previous Awardees believe that winning these awards has positively impacted their professional activities. With input from relevant APS staff, the Committee has developed a survey that will facilitate the collection and analysis of relevant data. This survey is going through a final spot check and should be available for Council approval shortly. The Committee aims to send out the survey before the end of the year to people who received CEAs and YIAs 3 and 5 years ago. Information from this first survey should be available in early 2017, with the survey sent out annually to recipients 3 and 5 years after their award. Demographic data on recent applicants and awardees are already available through the Awards website and will also be useful for indicating how successfully the Awards Program is meeting the Society's goals.

Other Activities

1) **Conflict of interest policy.** The first round of applications for $20,000 Research CEAs highlighted the need for the Awards Committee to have a conflict of interest policy. This policy is currently being drafted and should be available for Council approval soon.

2) **Standard operating procedures.** Standard operating procedures (SOPs) for Awards Committee members and for the Chair of the Awards Committee are being developed. The committee thanks Amie Dirks-Naylor, Amy Arnold, and Alexander Staruschenko for drafting an SOP for Awards Committee members. Before the end of the year, the Committee will discuss and finalize the SOP for Members.

3) **Archiving information.** When folders become available in APSConnect, they will be used for storage of current versions of Awards Committee documents.

This is Ida J. Llewellyn-Smith's last year as Chair of the Award Committee, and she thanks Council for the opportunity to serve the Society in this capacity over the past two and a half years. Llewellyn-Smith says it has been a particular privilege to serve as Chair of the Awards Committee when the Career Enhancement Awards Scheme has undergone such a substantial revision, becoming an even more important component of APS's Awards program.

Council accepted the report of the Awards Committee. ●

Career Opportunities in Physiology Committee

Thomas Pressley, Chair

The Career Opportunities in Physiology Committee serves as a resource for current information regarding career options in physiology, professional development at diverse levels, and fostering career interest in physiology.

Career Development at Experimental Biology

In 2016, committee members Christine Schnackenberg and Clintoria Richards-Williams chaired the session entitled "Leadership and Management Skills: What You Might Not See in Your CV." The purpose of the symposium was to explore, identify, and apply inherent and learned leadership/management skills. Experts in leadership and management from various backgrounds spoke for about 25 minutes each and then participated in a short panel discussion. The workshop drew a good-sized group of about 200 attendees. Recording of these talks, as well as additional materials, are being prepared for posting on the APS website.

For 2017, APS members can look forward to "The Many Facets of a 'Teaching' Career." This session is being organized by Committee members Clintoria Richards-Williams and Josef Brandauer.

Career Presentations at APS Conferences

Since 2006, the Committee has worked with the organizers of APS conferences to include career development sessions and/or activities at those meetings. These have included both informal sessions at the APS table/booth and formal sessions with a room assignment. The Education Office budget supports a workshop at one APS conference per year. APS member Jennifer Sasser and committee past-member Erica Wehrwein chaired a career development session at the "Renal and Metabolic Diseases: Physiology and Gender" conference held in Annapolis, Maryland, November 17-20, 2015.

The committee has also encouraged APS chapters to include career development sessions in their annual meetings. Past-member Erica Wehrwein moderated a career panel at the 3rd Annual Meeting of the Michigan Physiological Society, held May 12-13, 2016, in Detroit, Michigan.

Undergraduate Summer Research Fellowship Program

The APS Undergraduate Summer Research Fellowship (UGSRF) Program was established in 2000 with the goal "to excite and encourage undergraduate students worldwide to pursue a career as a basic research scientist." It has developed into a flagship APS program that influences hundreds of undergraduates. Students complete surveys at the start and end of their fellowship year, submit abstracts and research reports during their fellowship, and participate in post-fellowship follow-up surveys 2 and 4 years after completing the program. Committee members Seth Fairfax and Andrew Roberts are working with staff to optimize the survey instruments for more rigorous tracking of participants and evaluation of the program.

In its current form, the UGSRF Program supports up to 24 full-time undergraduate students annually to work in the laboratories of established investigators. Faculty sponsors/advisors must be active members of the APS in good standing. These fellowships provide a $4,000 summer stipend to the student (10-wk support), a $300 grant to the faculty sponsor/advisor, and a $1,300 travel award/reimbursement for the students to attend and present their data (if appropriate) at Experimental Biology. Financial support for the program is provided by the APS. In addition to their laboratory work, the UGSRFs complete interactive online assignments that explore how to structure a research project/series of experiments and that explore/discuss physiology-related careers.

In the application cycle for 2016, 81 eligible applicants were reviewed by the Committee, and the names of 24 suggested awardees were forwarded to Council for approval. Over the 17-year history of the program, the program has received over 1,000 applications with an average funding rate of 32%. Evidence that the program is indeed encouraging interest in research is provided by the continued involvement of UGSRF students in APS activities. All 24 2015 UGSRF students attended EB, and 17 submitted a first-author abstract. Thirteen

of these submitted applications for the 2016 David S. Bruce research awards. Four received a David S. Bruce Outstanding Undergraduate Abstract Award, and two of these, Allison Giuffre and Samuel Norton, went on to win a David S. Bruce Excellence in Undergraduate Research Award.

APS Undergraduate Research Excellence Fellowship Program

The APS Undergraduate Research Excellence Fellowship (UGREF) Program was launched in 2013 with the goal "to encourage undergraduate students worldwide who are already involved in research to continue on that track toward a career as a basic research scientist." Students with previous research experience are encouraged to apply. As with the UGSRF program, UGREF awardees complete surveys at the start and end of their fellowship year, submit abstracts, research reports, and draft manuscripts during their fellowship, and participate in post-fellowship follow-up surveys 2 years after completing the program.

The UGREF Program annually supports up to six full-time second- to fourth-year undergraduate students who have more than 9 months of research experience to continue working in the laboratories of established investigators. Both students and faculty sponsors/advisors must be active members of the APS in good standing. These fellowships provide a $4,000 summer stipend to the student, a $300 grant to the faculty sponsor/advisor, and a $1,300 travel award/reimbursement for the students to attend and present their data at EB. As with the UGSRFs, funding for the UGREF program is provided by the APS, and awardees are selected by the Committee with subsequent approval by Council.

The UGREFs complete interactive online assignments using an online course management platform, exploring how to structure a research project/series of experiments and exploring/discussing physiology-related careers. The UGREF activities are built on UGSRF activities, developing UGREF's writing and presentation skills. UGREFs are also required to submit an abstract to EB.

In the application cycle for 2016, 27 applicants were reviewed by the Committee, and the names of 6 suggested awardees were forwarded to Council for approval. Over the 4-year history of the program, the program has received over 100 applications, with an average funding rate of 20%.

Although a relatively new APS initiative, the UGREF program is also achieving its goal of encouraging continued interest in research while maintaining a rigorous review process. Two of the 2016 awardees, Gabriela Wong and Jacob Young, were participants in the 2015 UGREF program. Five former UGREF students attended EB in 2016 and submitted first-author abstracts. Four submitted David S. Bruce research award applications. One, Slavina Goleva, was ineligible for competition because she was a previous winner.

Undergraduate Orientation Session at EB

Since 2008, all undergraduates who submit a first-author abstract to APS are invited to a special Saturday session. It is a joint project of COPC and the Education and Trainee Advisory Committees. The session includes "how-to" presentations on making the most of the EB meeting, making a poster presentation, and introducing oneself. It has served as the formal recognition session for the UGSRFs. Again this year, for the third time, it also served as the formal recognition session for the UGREFs, STRIDE, and IOSP Fellows. It serves as an informal recognition for the David Bruce Abstract Awardees, as well as for the Video Contest award winners.

The EB 2016 orientation session was organized by committee members Carrie Quinn and Josef Brandauer and attracted well over 100 undergraduate students. As in the past, undergraduate students who submitted a first-author physiology poster were invited, and announcements were posted in e-mails to the Trainee and All-APS listservs. Members of the APS Executive Cabinet welcomed the undergraduate students and presented certificates to the UGSRF, UGREF, STRIDE, and IOSP Fellows. All three committees were well represented, with multiple members attending to talk with the students during and after the session and assist in distributing materials.

APS Careers Poster

COPC sends a physiology careers poster to each U.S. and Canadian undergraduate biology/life sciences department to encourage students to visit the APS website and attract students to physiology graduate work. The most recent poster has been distributed annually since 2012.

A subcommittee from the COPC worked with the APS Marketing Office to create the design. A survey was conducted of the undergraduate departments receiving the poster in 2013. After examining that data, committee member Carrie Quinn reported that career information is being provided primarily by the faculty member or advisor with whom students interact, but some students get information from Internet or career services. A follow-up e-mail includes a digital file of the poster, and a second follow-up e-mail includes links for the undergraduate summer research fellowship programs.

APS Career Cards

Based on suggestions from teachers, the Committee replaced a career brochure previously distributed by the APS with trading cards describing the work of diverse physiologists (career stage, research area, gender, racial/ethnic group, etc.) that are made available online and to teachers on request. COPC coordinates with the Education Committee so that career cards can be distributed as part of PhUn Week activities. During each calendar year, a different group of scientists are featured in a new set of cards. These career cards provide an opportunity for greater interaction with students. If students go to the website on the card, they can answer a question about the physiologist on the card, unlocking an additional biography card that they can print out. Overall, the cards offer a way to be more interactive with the students who receive them.

Feedback was very positive from both physiologists and teachers about the new trading cards. During PhUn Week 2015, "trading card" sets (3-4 biography cards, a physiology career card, and a Physio-Facts card with fun facts about human/animal physiology) were distributed to every 5th to 12th grader taking part in PhUn Week events. Sets were also distributed at the APS booth during the National Association of Biology Teachers Conference, Association of Middle Level Educators Conference, the EB 2016 Teacher-Student Workshop, and in April at the United States of America Science and Engineering Festival. Following these events, there was a spike in web traffic on the APS careers website, suggesting that links on the cards were used. As we accumulate a larger number of cards, we will introduce career-planning "games" and "ask a physiologist" into the site. The development of teaching resources that go along with the trading cards will provide a more effective learning tool for teachers and students.

In 2016, there were five additional cards added: Patricia Molina, David Pollock, and three younger APS researchers. Also, 8.5 × 11 mini-posters for use by teachers on bulletin boards were distributed in the fall as resources for teachers participating in PhUn Week events.

Developing Physiology Interest at the Elementary Level

The COPC works closely with the Education Office to develop materials appropriate for young students. In 2015, 21% of the children participating in PhUn Week events were PreK to 2nd graders (i.e., "pre-readers") for whom there were no APS resources available. To provide career info and physiology engagement for early elementary grades (K-2), the committee expanded the current Phizzy Bear activity booklet. Age-appropriate activities were added that align with new K-12 science education standards. The new activity book is a resource for PhUn Week outreach to early elementary students. With the help of the Puerto Rico Physiological Society (an APS chapter), the activity was also translated into Spanish. It is expected that the artwork can be used for future early elementary projects such as interactive webpages or digital interactive stories or activity books.

Career Outreach Resources

The APS Careers Website was developed by the COPC and launched in March 2003. It provides extensive resources for two major purposes: *1)* to assist students and new and experienced physiologists in the development of their careers and *2)* to help the general public gain a better understanding of the work that physiologists do. The site includes resources for elementary, middle/high school, undergraduate, graduate/professional, postdoctoral fellows, new investigators, established investigators, and the general public. Within each section, the user finds resource categories customized to their needs. The specific resources (such as biographies, hands-on experiments, career resources, etc.) are written at the appropriate educational level. It also serves as a dissemination site for the macromedia flash (PowerPoint plus audio) versions of EB career-related sessions developed by the COPC, Women in Physiology, and Trainee Advisory Committees. It also

includes hyperlinks for career development resources associated with each skill listed in the APS-ACDP List of Professional Skills.

In 2011-2012, the career website was reconfigured to better fit both the new website content management system and to provide easier access to the growing set of professional development resources APS has developed for trainees. Features include a "Career Development and Mentoring Forum" that provides one-click access to APS resources (webinars, EB symposia, Mentoring Forum articles, etc.). All resources on the site are cataloged in the Life Sciences Teaching Resource Community digital library and pushed to the website as a folder of resources, allowing new materials to easily be added to individual pages. Along similar lines, the Committee has developed APS Career Presentations that consist of downloadable PowerPoint files for use at the middle and high school levels, as well as lower and upper undergraduate levels.

This past year, Committee members Erika Boesen and Norma Ojeda reviewed the content of the website and Career Presentations, respectively. Both raised concerns that the material is becoming out-of-date and, in the case of the website, increasingly plagued by broken links. The Committee and staff have corrected many of the technical problems with the website, but it is clear that both the website and the presentations are in need of significant updates. It is anticipated that a review of the purpose, objectives, and overall concepts for these resources will be a major priority of the Committee over the next year as preparation for redesign and implementation of the overall APS website.

APS Local and Regional Science Fair Awards

This program encourages APS members to make an APS physiology award at their local or regional science fair at the elementary, middle, or high school level. The program provides opportunities for students from elementary through high school to learn what physiology is and to become "associated with the field" through recognition of their work. The program also builds connections between APS members and their local schools. Finally, it encourages local fairs to promote physiology-based projects to their students, since there are potential awards to be won. Student winners receive an APS t-shirt and a certificate for the best physiology project. The teachers of the winning students receive the APS book *Women Life Scientists: Past, Present, and Future* and an APS resource packet. Up to 100 awards are available each year on a first-come, first-served basis. Advertisements are posted in *The Physiologist* and the All-APS News e-mail updates for members and sent to all past participants. The judge (or judges) must be an APS member in good standing who is willing to present the award on behalf of the APS to the student with the best physiology project (one winning student per science fair). Standard conflict of interest policies apply. After the science fair, the judge reports the winner's name and school via an online report form. Winners' names and pictures are posted on the APS Local and Regional Science Fair website.

In the first half of 2016, 15 APS members requested packets for use as Science Fair Awards, and we received 10 follow-up reports. We typically track Science Fair activities on a calendar-year basis (rather than a school year) from January through December to better account for the requests and presentations.

Physiology Video Contest for Undergraduate and Graduate Students

In 2011, the COPC launched the APS video contest *APS Presents...Phantastic Physiology Voyage: "Function Follows Form,* designed to engage undergraduate and graduate students by creating engaging accurate, informative, and entertaining physiology videos for the general public. These videos must creatively demonstrate and/or explore a specific physiological function in 5 minutes or less (including credits). The video can be staged as a short play, commercial, news broadcast, talk show, music video, or documentary. The contest rules are based on a review of video contests sponsored by other professional associations and foundations. They include standards for copyright, use of humans or animals, and use of university facilities.

A selection committee consisting of members of the COPC and past APS Summer Research Teachers (invited) evaluates the videos and identifies a first-place video based on specific criteria for approval by Council. The YouTube links to the finalists' videos are promoted through the APS website and APS Facebook sites. The YouTube viewing statistics are used to name the Viewer's Choice Award winning video (i.e., the video with the most views on YouTube during the period leading up to EB). The contest makes two awards. A $750 first prize

is given to the best video as selected by the Committee, and $250 is given for the Viewer's Choice Award. The best video, selected by the Committee, is shown and announced at the Undergraduate Orientation Session at EB. All of the videos meeting eligibility requirements are shown at the Undergraduate Poster Session at EB, and the winner of the Viewer's Choice Award is announced at this session.

In 2016, the Committee received nine submissions for review. The applicants included both undergraduate and graduate students. The Committee selected and forwarded to Council for approval "Hyperthyroidism: Not as Grave as You Think" by Emilee Friedman and Jennie Wiggins of Augusta University. The video takes advantage of the whiteboard technique popularized by organizations such as the Khan Academy. The video also received the Viewer's Choice award, which was selected by the general viewing public. Both awardees were able to attend EB and accept their award.

One of the advantages of the video contest is that the videos continue to promote physiology long after the contest has ended. Each video remains on YouTube (unless the video creator deletes it), and it can be accessed with links from other sites. For example, each video is cataloged in the Life Science Teaching Resource Community with an embedded YouTube link.

Excellence in Professional Student (MD or DO) Research Travel Award

The APS Excellence in Professional Student (MD or DO) Research Travel Award Program launched in 2013 with the goal "to encourage MD or DO students to attend, present their research, and participate fully in the Experimental Biology (EB) meeting." The award supports up to 10 MD or DO students who are first authors on abstracts to attend EB, present their research, and attend an orientation session. Faculty sponsors/advisors must be active members of the APS in good standing. Applicants are reviewed by the Committee, and suggested awardees are submitted to Council for approval.

In the application cycle for 2016, 17 applicants were reviewed by the Committee, and the names of 10 suggested awardees were forwarded to Council for approval. These students were matched with a mentor for EB, similar to what is done for the APS Minority Travel Fellows Program. Fellows and mentors corresponded via e-mail and then met each other at an Orientation Session on Sunday morning. This session was organized by COPC Members Erika Boesen, Rasna Sabharwal, and Norma Ojeda. APS President Patricia Molina, an MD herself, gave a short presentation encouraging the students and emphasizing the importance of continuing to conduct research. As part of the orientation presentation, three clinician scientists at different career stages gave their perspectives on combining research and medicine. Assigned mentors and additional clinician scientists were also present and participated in one-on-one "speed mentoring" of the students during the session.

Council accepted the report of the Career Opportunites in Physiology Committee. ●

Chapter Advisory Committee


Michael Sturek, Chair

The local APS Chapters promote the future of physiological sciences overall and the APS with grassroots involvement at the graduate, undergraduate, and high school levels. The Chapters contribute to four of the five strategic priority areas and both cross-cutting themes of the APS 2010 Strategic Plan. An overall goal is for the activities of the Chapters to further influence public policy. There are 14 Chapters in varying stages of activity. A main activity is the annual meetings, which typically have ~60-120 participants in a 1- to 2-day meeting. Novel methods for promoting poster attendance, etc. have been used. There is an increasing emphasis on career development in the changing research and education environment. A major strength is the emphasis on trainee involvement in running meetings. Chapters are strongly encouraged to participate in outreach/advocacy activities and are reminded that advocacy speakers are available through APS. Although the APS website has guidelines for establishing and maintaining a Chapter, a more detailed "Best Practices" manual is under construction. Involvement of small colleges in the Chapter activities greatly improves outreach to undergraduates. Following are five major goals for the next year. *1)* Determine how chapters will align with the "Local Physiology Network" concept at institutions that have no formal Physiology Department. *2)* Publish a History of APS Chapters, largely spearheaded by Mari Hopper. The document will include a "best practices" manual derived from this rich history. *3)* Establish a California Chapter. 5) Plan for measurement of long-term outcomes of chapters on the recruitment and development of physiology students and faculty for the future. This will very likely require a very active, centralized database, preferably within the APS. Local APS Chapters can have a major impact on physiology.

Council accepted the report of the Chapter Advisory Committee. ●

Committee on Committees


Bill Yates, Chair

The Committee on Committees (CoC) is composed of a representative appointed by each of the 12 APS Section Steering Committees plus two councilors who serve as Chair and incoming Chair. The primary duty of the CoC is to nominate individuals to serve on APS standing committees and on outside bodies where the APS is represented. The CoC members identify members of their section who might serve on committees. During deliberations by CoC, both the expertise and diversity of the applicant pool are weighed. There is a deliberate attempt to appoint highly qualified members to committees, while maintaining sectional and demographic balance.

Summary of the Process and Deliberations in 2016

For the first time in a number of years, a representative from each section was present at the CoC meeting. It was also evident that every section had been diligent in recruiting members to apply for committee positions. The Section Advisory Committee (SAC) is commended for promoting the importance of every section being highly engaged in the CoC process.

The webpage describing the CoC process (*www.the-aps.org/mm/hp/Audiences/APS-Committees/nominations*) was updated in late 2015 to make the committee appointment process more transparent. There were also efforts to educate CoC representatives about the process to ensure that all sections received the same information.

Although the process worked well, a number of weaknesses were also apparent. Despite efforts to educate applicants, some nominees did not position themselves well for appointment to a committee. Two common mistakes were made by nominees. First, some applied for membership on a committee that already had broad representation from their section, diminishing their chances of being appointed. Second, a number of nominees had not introduced themselves to their section's CoC representative, and thus the representative was hard-pressed to be their advocate. Better education of Society members about the CoC process is recommended.

If an individual did not identify any prior service to the Society (e.g., participation in section or chapter activities), it was difficult for the CoC to muster enthusiasm for their application. As part of continuing education of members, avenues to provide service should be clarified, so a succession route from sectional and chapter engagement, as well as other Society functions (e.g., service on a journal editorial board), to a committee appointment is transparent.

A number of applicants were ending their service on another committee, such that they would rotate from one committee to another. Many of these applicants were known to be highly effective committee members, so it was assured they would provide stellar service if appointed again. However, such migration between committees diminishes the opportunity for others to serve. CoC and Council discussed whether it was appropriate for a member retiring from a committee to be immediately appointed to another committee. It was agreed that a firm policy on this matter would limit flexibility, but in general it was better to provide opportunities for new members rather than to appoint the same individual repeatedly to different committees. However, after a gap in service of 2-3 years, an individual who was previously appointed to a committee can be nominated for a different committee without prejudice.

Another concern is that some individuals are active in more than one section, but only their primary section affiliation is indicated on the application form. It was recommended that the form be revised to provide for transparency in the member's engagement in the Society: all sectional affiliations should be reflected. CoC should consider the totality of the affiliations of the applicant pool when evaluating the diversity of a committee.

The CoC also debated the importance of nominees routinely attending the Experimental Biology meeting. Several applicants had been heavily engaged in APS activities but cannot routinely attend Experimental Biology because of a variety of conflicting obligations. Applications from international members were particularly low this year (2% of the applicant pool), likely because attending Experimental Biology has been emphasized as a requirement for committee service. However, some committees (e.g., Daggs, Publications, Finance) do not have a formal meeting at Experimental Biology, and others have a cursory meeting but conduct most of their business at another time. It was recommended that we poll each committee to determine the importance of attending Experimental Biology. For committees that conduct minimal business at Experimental Biology, CoC should not consider routine attendance at the meeting as a critical factor for appointment.

At present, endorsements for nominations can come from anyone and not just APS members. The CoC noted that endorsements from individuals who are not APS members were often weak, likely because the endorser was unaware of the workings of the Society and the nature of committee service. It is recommended that the CoC process be amended such that only APS members can endorse an applicant. Explicit language should be added to the application, directing the applicant to their section's CoC representative if they have difficulty in finding an endorser.

Characteristics of the 2017 Applicant Pool

This year, 60 positions were open, with a starting date of January 2017, as shown in Table 1.

The CoC was pleased with the pool of applications for committee vacancies. A total of 177 applications were submitted for 60 positions, as indicated in Table 2. In contrast, there were 117 applications the previous year, reflecting a 51% increase. Some committees (e.g., Career Opportunities in Physiology, Education) received a large number of applications, whereas others (e.g., Daggs, Distinguished Physiologists) received few or no applications. In terms of applications per open position, the most competitive committees for

appointments were Career Opportunities, Animal Care, and Education.

Table 3 shows the applicant pool for each committee by primary section affiliation. A few applicants were excluded from the table, since there was no section designation for these individuals. In general, the number of applicants from a section was aligned with the proportion of Society members belonging to that section. However, two sections (Teaching and Water & Electrolyte) received more applications than would be expected based on sectional designation.

Table 4 provides demographic information about the applicant pool in 2017. It should be noted that not all members have designated their age or gender, so some of the values in the table are undoubtedly an underestimate. Nonetheless, these data suggest that we have limited success in soliciting applications from younger members, females, and those living outside the United States. In the future, COC representatives from each section should increase their efforts to encourage applications from these groups.

Action Items for the Future

- Engagement of every section in the committee appointment process was stellar in 2016. It is important to retain this commitment in the future. SAC should continue to remind every section about the importance of the CoC process.
- All committees should be polled about whether attendance at Experimental Biology is critical for active membership. A listing of committees that do not require attendance at Experimental Biology should be published and considered during the CoC process.
- Historically, only an applicant's primary section affiliation was identified on the nomination form, and only the primary section was considered in assuring committee diversity. However, some members are active in more than one section and should be offered the opportunity to designate more than one primary section affiliation.

Table 1. Positions available in 2017

Committee	No. of Positions Available
Animal Care & Experimentation	3 members
Awards	Chair, 4 members
Career Opportunities in Physiology	2 members, 1 Student member, 1 Trainee member
Communications	3 members, 1 Student member
Conference	Chair, 3 members
Daggs	Chair, 2 members
Distinguished Physiologists	2 members
Education	4 members, 1 Trainee member
Finance	2 members
International	Chair, 5 members
Membership	0 members
Perkins	1 members
Physiologist in Industry†	Chair
Porter Physiology Development	Chair, 5 members
Publications	2 members
Science Policy	Chair, 5 members, 1 Trainee member
Women in Physiology	5 members
Totals	***60 total positions: 7 Chairs, 48 members, 2 student members, 3 trainee members**

*This number does not include the positions to be selected by the Executive Cabinet or committees populated by sectional designations. †Only the Chair is selected by COC.

- The committee application process should be revised such that only APS members can endorse a nominee. Applicants should be advised to consult their section(s)' CoC representative if they have difficulty in identifying an endorser.
- As CoC and Council evaluate nominees, preference should be given to qualified individuals who have not recently served on an APS-wide committee. A period of 2-3 years should lapse between appointments to committees.
- Although changes were made to the committee nomination process webpage (*www.the-aps.org/mm/hp/Audiences/APS-Committees/nominations*) last year to clarify the process, further transparency is needed. The following changes are recommended:
- The application form should explicitly state that applicants need to contact the CoC representative of their affiliated sections to secure support during the committee appointment process.
- A webpage should be developed to educate members about avenues for service to the Society, so they are better positioned as committee applicants.
- The website section "Optimizing the Likelihood You Will Be Appointed to a Committee" should be revised to further clarify the CoC process.

Council accepted the report of the Committee on Committees.

Table 2. Number of applicants for each committee

Committee	No. of Applications	Applications per Open Position
Animal Care & Experimentation	14	4.7
Awards	16	3.2
Career Opportunities in Physiology	24	6
Communications	11	2.75
Conference	12	3
Daggs	4	2
Distinguished Physiologists	0	0
Education	21	4.2
Finance	5	2.5
International	16	2.7
Perkins	4	4
Physiologist in Industry†	1	1
Porter Physiology Development	10	1.7
Publications	7	3.5
Science Policy	18	2.6
Women in Physiology	14	2.8
Totals	177*	2.95

*This includes candidates who applied for more than one committee. †Only the Chair is selected by COC.

Table 3. Applicants to each committee by section.

Section	No. of Applications†	% Applications	% Society Membership*
Cardiovascular	38	21.5%	22%
Cell and Molecular	11	6.2%	13%
Central Nervous System	14	7.9%	10%
Comparative & Evolutionary	3	1.7%	4%
Endocrinology & Metabolism	10	5.6%	8%
Environmental & Exercise	20	11.3%	11%
Gastrointestinal & Liver	7	4.0%	6%
Neural Control & Autonomic Reg.	6	3.4%	6%
Renal	13	7.3%	7%
Respiration	20	11.3%	8%
Teaching	19	10.7%	5%
Water & Electrolyte Homeostasis	13	7.3%	2%

*Excludes honorary and affiliate members. †A few applicants had not designated a section affiliation and are not included.

Table 4. Demographic characteristics of applicants in 2016

Group	No. of Applicants (%)†	All APS Members*
Under age 45	32 (18%)	40%
Women	25 (14%)	29%
Reside outside of US	4 (2%)	27%

*Excludes honorary and affiliate members. †Demographic information, particularly gender, is unavailable for some members. Thus some values may be an underestimate.

Communications Committee

Barbara Goodman, Chair

The APS Communications Committee continues to serve in an advisory role to the Communications Office staff. The Committee met most recently at EB 2016 to discuss ongoing projects including the EB 2017 Communications symposium topic, the newly launched and upgraded quiz function on the PhysiologyInfo.org (PIO) website, an update on Facebook promotion of the I Spy Physiology blog, and thinking ahead to ways of highlighting underrepresented minorities in physiology – specifically African Americans and women during Black History Month and Women's History Month 2017, respectively.

For the EB 2017 Communications symposium, the Communications Committee chair (Barb Goodman) has arranged for the Chicago-based Second City comedy troupe to host a session on communicating science through improv. Second City's workshops are geared toward a small-group experience, so our session will be limited to 50 meeting attendees. Attendees must RSVP and will be selected on a first-come, first-served basis.

Communications Committee members and past-Committee members continue to be ongoing contributors to the I Spy Physiology blog. So far in 2016, they have contributed 15 (of 28 total) posts on a variety of physiological topics. As mentioned above, Committee members Barb Goodman, Jessica Faulkner, Emily Johnson (who also contributed four posts in her new role as EB meeting blogger), Ben Miller, Pascale Lane, and Shawn Bender and past-Committee member Dao Ho have written posts.

The Committee also discussed new volunteers requesting to join the Committee. In 2015, the Communications Committee received no new applicants. However, we received 11 applications in 2016, suggesting that the Committee is gaining steam and recognition among the APS membership on the whole.

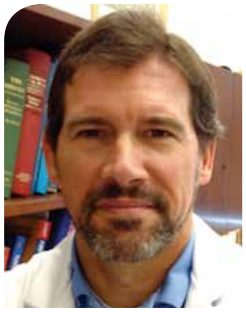
Part of the Committee's charge is to review applications for the APS-sponsored AAAS Mass Media Fellow. Because of a staffing change at AAAS, the Committee was not able to divvy up the review process as usual. However, Committee Chair Barb Goodman reviewed and ranked all of this year's applicants and chose APS member Casey Gilman as this year's Fellow. She originally applied for the 2015 fellowship and reapplied in the 2016 cycle. Gilman received her PhD from the Organismic and Evolutionary Biology (OEB) graduate program at the University of Massachusetts-Amherst. Her undergraduate studies focused on physiological ecology, and she has also written for the *Journal of Experimental Biology,* New England Public Radio, and *Natural History Magazine,* among other publications. She is spending the summer writing for the *Philadelphia Enquirer.* Read her most recent article.

PhysiologyInfo.Org

PhysiologyInfo.org (PIO) – APS's public-facing website – relaunched at the end of 2013. The relaunch was the culmination of concerted efforts among the Communications Committee, APS Communications staff, and APS webmaster. In 2014, the Communications Office made a number of aesthetic changes in response to a user survey conducted earlier in the year. The Communications Office continues to work with and support the Communications Committee in furthering content updates to make the site more robust and informative for a lay audience. Additionally, the Communications staff is in contact with our web-services provider about adding new features to the site.

Council accepted the report of the Communications Committee. ●

Conference Committee


Michael Brands, Chair

The Committee held their annual meeting in Bethesda on October 13, 2015. The Committee discussions included reports from APSCC representatives to the 2015 Endothelin and Bioenergetics conferences, review of the conferences approved for 2016, a motion to "brand" APS conferences with formatting for poster and trainee sessions, a motion to enhance conference promotion within APS, a motion to diversify conference programming slots and allocation of conference support dollars to promote a better balance between senior-, mid-, and junior-level presenters, and a presentation by APS staff to the APSCC about actual costs for putting on a conference followed by a motion to revise the APSCC website to state that "fundraising is not necessary to organize a conference." The last motion was aimed at encouraging junior- or mid-career members to propose interesting new ideas for APS conferences. APS Council endorsed these action items, and also urged the APSCC to develop a "best practices document" that incorporates these items.

Council accepted the report of the Conference Committee. ●

Distinguished Physiologists Committee


Peter K. Lauf, Chair

The Distinguished Physiologists Committee consists of 10 members. One of the primary duties of each Committee member is to "develop and maintain liaison with emeritus members and members about to retire." This liaison is accomplished by submitting, on behalf of the Society, a personal 70th, 80th, 90th, or 100th birthday greeting. Each greeting includes an invitation for the senior recipient to inform APS about his or her current activities, interests, and whereabouts, and requests "words of wisdom" for younger colleagues. The historical and philosophical commentaries evoked by this invitation provide the material subsequently published in "News From Distinguished Physiologists" in each issue of *The Physiologist*. By the end of 2015, the Senior Physiologist Committee members sent birthday wishes to 69 members reaching age 70, 50 members reaching age 80, 30 members reaching age 90, and to 6 members reaching the age of 100! Six response letters were received and published in *The Physiologist*.

Another responsibility of the Distinguished Physiologists Committee is to review applications and recommend to Council the annual awardees of the $500 G. Edgar Folk, Jr., Senior Physiologists Award. This award is designed to support the scientific activities of a senior member. In 2015, we received and awarded one application: Celia D. Sladek of University of Colorado-Denver.

Council accepted the report of the Distinguished Physiologists Committee.

Education Committee


Jeffrey L. Osborn, Chair

ADInstruments Macknight Early Career Innovative Educator Award

The Education Committee received six applications for 2016. The quality of applicants was particularly outstanding in 2016, making choices difficult. The Committee unanimously recommended APS member Stefan Pulver of the University of St. Andrews, St. Andrews, United Kingdom as the 2016 awardee. His application focused on the development of exercises that use the light-activated chloride pump halorhodopsin to identify central pattern generating networks controlling locomotion in *Drosophilia* larvae. The application was particularly outstanding because of its innovative use of optogenetic tools and the ability to adapt and conduct these lab activities from secondary school through graduate physiology education.

EB Refresher Course

The resources from the Refresher Courses are viewed regularly at the APS website and, more importantly, have been downloaded nearly 24,000 times at the LifeSciTRC and have been saved by users to their folders 330 times. The 2015 Refresher Course on "It's All in Your Head: A Refresher Course on the Brain and Systems Control" is now available on the APS website and through the LifeSciTRC.

The 2016 Refresher Course "Keep Your Eye on the Ion: A Refresher Couse on Ionic Homeostasis and Systems Physiology" was very well attended (~350 attendees). Overall, course ratings were very high. There was a good mix of members from all APS membership categories among the attendees. Recording of these talks, as well as additional materials, are being prepared for posting on the APS website. Looking ahead to 2017, the Refresher Course will be titled "Curricular Innovation in Sex and Gender Based Medical Physiology Education." An outstanding group of internationally recognized speakers will present this course at EB 2017 in Chicago, IL.

Professional Skills Courses

A particular highlight was the APS Education Office and Publications Department, collaborating with the Society for Biological Engineers and Biomedical Engineering Society, receiving NSF funding to support development of professional skills training modules on publication ethics. The education modules will serve as tools for use by higher education institutions, laboratory groups, individuals, and professional societies, and will incorporate proven materials and methods, as well as novel approaches. They will be effective for U.S. and international graduate students in science and engineering programs, and will integrate easily into Responsible Conduct of Research (RCR) training.

Medical Physiology and Graduate Program Course Directors

Last year, a new version of the Medical Physiology Course Directors website was launched that includes secure access for course directors. This enhancement allows them to share assessment resources such as case studies and to test items securely. In addition, the LifeSciTRC has added private bulletin boards, which will allow the group to have "members only" online discussion and resource sharing. For Graduate Program Directors, the next NDGS meeting will be held in 2017, with the location to be determined. Stay tuned for upcoming announcements of this joint APS/ASPET graduate course director initiative.

Human Anatomy and Physiology Society (HAPS) Collaboration

The 2016 HAPS Conference was held May 21-23 in Atlanta, GA, with attendees from across the U.S., Canada, and other countries. APS member Lacy Alexander (Pennsylvania State University) gave a presentation entitled "Human Skin as a Model Circulation for Examining Mechanisms of Microvascular Dysfunction."

Liaison With The Physiological Society (TPS), London

The Education Committee has worked for several years to coordinate more effectively with the educational efforts of TPS. The chair of the TPS Education and Outreach Committee (EOC) typically attends the fall and EB APS Education Committee meetings. Education Committee member Jessica Taylor and Director of Education Programs Marsha Matyas attended the PhySoc Education Meeting in Dublin, Ireland in July 2016 to conduct a teaching symposium (Taylor) and plan educational sessions for future PhySoc meetings.

Life Science Teaching Resource Community (Archive of Teaching Resources)

The LifeSciTRC continues to grow as a national and international teaching resource. Resources and collections are submitted by partnering societies as well as individual educators and are peer-reviewed for scientific accuracy and the appropriate use of humans and/or animals in research. In 2015, the LifeSciTRC added 281 new resources and 21 new collections. The LifeSciTRC currently contains more than 7,700 peer-reviewed teaching resources and 273 collections. As users, 1,187 individuals registered to use the LifeSciTRC in 2015, bringing the total number of registered users to 8,406. Registration is not required to view and download materials from the LifeSciTRC, and in 2015 there were 800,335 visits to the LifeSciTRC.

David S. Bruce Awards

In 2016, 97 applications were received and reviewed by the Education Committee. Again, 30 of applications were selected for the "David S. Bruce Outstanding Undergraduate Abstract Award." These students were given the opportunity to present their posters to a panel of judges during the EB meeting. The panel of judges selected 15 to receive the "Bruce Excellence in Undergraduate Research Award" and $400, based on their presentation skills and interviews with the judges. This year's top-ranked student was Lauren Newhouse, from Mayo Clinic working with Mike Joyner's lab. As the Bruce Award program has matured, the overall quality of the posters and presenters continues to improve. This year's David S. Bruce Excellence in Undergraduate Research winners all professed to wanting to enter research-intensive careers, and from their presentations, they are clearly on that trajectory. The Bruce Awards remain an excellent mechanism for enhancing the pipeline to professional careers in physiology.

EB Undergraduate Poster Session

The 2016 Undergraduate Poster Session was held on Sunday, April 3rd, in the San Diego Convention Center. Students were encouraged to attend the exhibitor tables to network with graduate programs. This year, about 140 students attended and presented at the selective UG session. Of those students, 42 were APS UG fellows, and of those in that group, approximately

40 presented a first-author abstract *programmed through their section*. Of note, there were 17 exhibitors present at the Undergraduate Poster Session, most of which were graduate programs from around the country. This number continues to increase annually, and we received excellent feedback from the students and exhibitors about the networking opportunity. There was significant foot traffic through the posters, and we received good feedback from attendees about how a select session focused entirely on undergraduates doing research is important to them.

Frontiers in Physiology Professional Development Program for Teachers

This extramurally funded education program includes three components that are all currently running. Frontiers in Physiology: Integrating Programs to Build Communities of Practice (R25 OD016492) will develop, test, and disseminate a progressive three-program model designed to promote excellence in science education and alignment with state and national Next Generation Science Standards (NGSS). The Six Star Science Online Teacher (OT) Professional Development Program will engage 30 middle/high school teachers annually in a year-long online professional development program that will increase their use of Six Star Science student-centered teaching methods that build students' knowledge and skills in science and their understanding of the role of biomedical research in their lives and careers. The online program will reach teachers in both urban and rural areas and from all school types. Upon completion of the OT Program, teachers can apply for the Frontiers in Physiology Research and Community Leader (RCL) Fellowship that will offer 15 teachers annually an opportunity to engage in a summer of hands-on biomedical research with APS members, work online to complete assignments and gain leadership skills that they will use at workshops and in online communities with colleagues, and experience research sharing by attending the Experimental Biology Meeting.

From a pool of 16 applicants, the Education Committee selected 8 teacher fellows to participate in the 2015 Frontiers Research Teacher Program (50% award rate). These awardees completed their fellowship by participating in EB 2016 in San Diego, CA. The first class of Research Community Leaders was selected from a small pool of past Online Teachers. A requirement for this course is the completion of the online course, so, although the current pool is small, the pool will be increasing each year. These awardees are working in the lab now and will complete their fellowship by participating in EB 2017 in Chicago, IL. Two regional APS chapters (Michigan and Puerto Rico) are in the process of developing regional communities to support K-12 science education. These communities will effectively engage MS and HS science teachers in chapter activities, integrate the Six Star Science principles into chapter K-12 outreach activities, provide content updates and best practice for student-centered learning, involve interactions between both teachers and researchers in the region, and provide information about the APS Frontiers program and Physiology Understanding Week.

EB Workshop for Teachers and Students

Margaret Stieben of the APS Education Office coordinated the workshop for high school teachers, and Education Committee Member Patricia A. Halpin coordinated the 2016 APS Workshop for High School Students. This year's EB workshop had 23 area high school teachers and their 75 students in attendance at the workshop on Monday April 4, along with APS members and 2015-2016 Frontiers Research Teachers. The keynote address "Human Performance in Extreme Environments" was given by Senior Associate Dean, Distinguished Professor of Endocrinology, at the University of Missouri, Warren Lockett. His talk was followed by a Careers Panel that included Lockett along with Anne Whittaker (APS Minority K-12 Outreach Fellow) and Nick Aguirre (this year's Minority Outreach Fellows). The panel was moderated by 2014 Lead Mentor Instructor Robert Manriquez.

The afternoon student session was led by Patricia A. Halpin (University of New Hampshire), along with 2014 Lead Mentor Instructor Robert Manriquez and several APS members. Students used the "Junkyard Digestion" activity on modeling the digestive system with common household items, while their teachers and APS teacher fellows conducted "Using the Next Generation Science Standards," an exercise in using student-centered activities in the classroom led by Barb Goodman (University of South Dakota) and Tonya Smith (Frontiers Mentor Teacher).

Physiology Understanding (PhUn) Week

In November 2015, the APS celebrated the 10-year anniversary of PhUn Week program. In 2015, 16,188 students were reached at 101 event sites across the nation and Puerto Rico. This effort involved 115 APS member lead coordinators and a total of 680 scientists presenting and partnering with 281 classroom teachers and educators. The program continues to exceed its annual goal, which was increased to 16,000 from 14,000 students the year before. Distribution by grades included nearly 31% in high school classrooms, 45% in primary and elementary classrooms, and 24% in middle school classrooms. The number of physiologists (680), teachers (281), and event sites (101) all exceeded projected goals. The Committee is particularly excited about the large increase in the participation of physiologists in PhUn Week events.

In 2016, the PhUn Week Training Session at Experimental Biology session drew over 100 attendees, including physiologists and teachers. Jeff Osborn and Jenny Sasser directed the session in which members (including graduate/postdoctoral trainees) presented 34 posters at the training session. Participants were also encouraged to submit these posters to the LifeSciTri online resource library. ADInstruments continued its tradition of sponsoring a continental breakfast. The incredible success of this society-wide program was celebrated with a poster session and award ceremony for 5- and 10-year participants. Special recognition goes out to Diane Munzenmeier (Center for BioMolecular Modeling, Milwaukee School of Engineering), who not only spearheaded the launching of PhUn week as a member of the education committee but also has conducted PhUn week workshops in each year of the program's existence.

USA Science and Engineering Festival

More than 350,000 children, parents, and teachers attended the 2016 USA Science & Engineering Festival (USASEF) at the Convention Center in Washington, DC, April 15-17. For the first time, the festival included a "Sneak Peek Friday" that allowed school groups to attend the festival. More than 60,000 students and teachers attended this event. On Saturday and Sunday, parents and children of all ages filled the exhibit halls, exploring areas of science, engineering, and technology. APS shared the Medicine and Health area with other professional societies, NIH, NSF, and other federal agencies and companies. APS activities were organized by the Education Committee and Education Office. A demographic and ethnic diversity of visitors to the APS booth included young and old adult individuals, parent-child or family groups, and groups of young children and adolescents. There was a steady and constant flow of guests engaging in both APS booth activities on the "Healthy Heart Race" and a demonstration on insulation using fat, feathers, and skin in cold water. At the APS booth, seven members worked each day to engage thousands of attendees in hands-on physiology activities. Education Committee members included the Committee Chair Jeff Osborn (University of Kentucky) as well as committee members Patricia Halpin (University of New Hampshire) and Jessica Taylor (College of Osteopathic Medicine, William Carey University). APS Minority Outreach Fellows (past and present) included TanYa Gwathmey (Wake Forest University), Elinette Albino-Rodriguez (Ponce Health Sciences University), and Jessica Ibarra (University of the Incarnate Word). Porter Committee Member Stan Andrisse (Johns Hopkins University) also served on the team. Members distributed thousands of APS Career Trading Cards and Phizzy Bear Activity Books to teens and children to promote physiology career exploration.

Undergraduate Program Recognition Project

The Education Committee is continuing its exploration and possible expansion of recognizing undergraduate programs in physiology that serve as exemplars in preparing students for physiology careers. A subcommittee is gathering information and will present their approaches at the fall 2016 committee meeting. The subcommittee examined the availability of undergraduate physiology programs in U.S. institutions and produced a list of "best practices as defined for the development and delivery of undergraduate degrees/programs /curriculum/courses in Physiology for the 21st century." The group is led by Erica Wehrwein (Michigan State University) and Carissa Krane (University of Dayton). The relationships between undergraduate departments and Colleges of Arts and Sciences and Medicine must be carefully explored and vetted. The Committee is committed to focusing on best practices. Smaller institutions might find this program an especially helpful way to show their physiology programs as being competitive on a national scale. The Committee feels it is important for APS to produce

similar work, but equally important for any proposal to use specific language that makes it clear that this is not a certification or accreditation program. This next arena for the discipline of physiology is critical to our defining and branding of the society as a whole. The Chair and Committee are deeply committed and invested in making undergraduate physiology a major focus for APS in the coming years.

Council accepted the report of the Education Committee. ●

Finance Committee


Meredith Hay, Chair

Approve the 2015 Audit Report

On May 17, 2016, the Finance Committee met via conference call with Marina Polyakova of Rogers & Company, the Society's audit firm. Rogers explained that fieldwork for the 2016 audit was performed by Rogers & Company from April 25, 2016 through April 29, 2016 at the APS office in Bethesda. Based on their work, Rogers & Company rendered an unqualified opinion (i.e., clean opinion) on the Society's financial statements affirming that the statements presented fairly, in all material respects, the financial position of the Society at December 31, 2015 and 2014. The 2015 audit report noted no material internal control weaknesses or other areas of concern with respect to the Society's financial processes.

Polyakova discussed the audit report and an accompanying letter to the Finance Committee, Council, and Management of APS (board letter). The board letter is a required communication from the audit firm to the client. Rogers explained that the Society's financial statements were audited in accordance with generally accepted auditing standards. The 2015 audit did not include a review of the Society's records in accordance with government auditing standards, because the Society's income from federal grants was less than $750,000. Only organizations with federal grant (or contract) income of $750,000 require such an audit.

Polyakova discussed all sections of the audit report, including her analysis of the financial statements. She explained that the statements reflected a slight loss for the year of just over $200,000 due to the low return on investments.

A motion was made and seconded to accept the 2015 audit report by Rogers & Company. The motion carried unanimously.

APS Budget

The Finance Committee met in March to review 2015 financial activity and review the Society's 2016 budget. 2015 revenue was $19.85 million, which included $2 million in support from reserves, and expenses over the same period were $18.25 million resulting in a surplus for the year of $1.6 million.

Figure 1

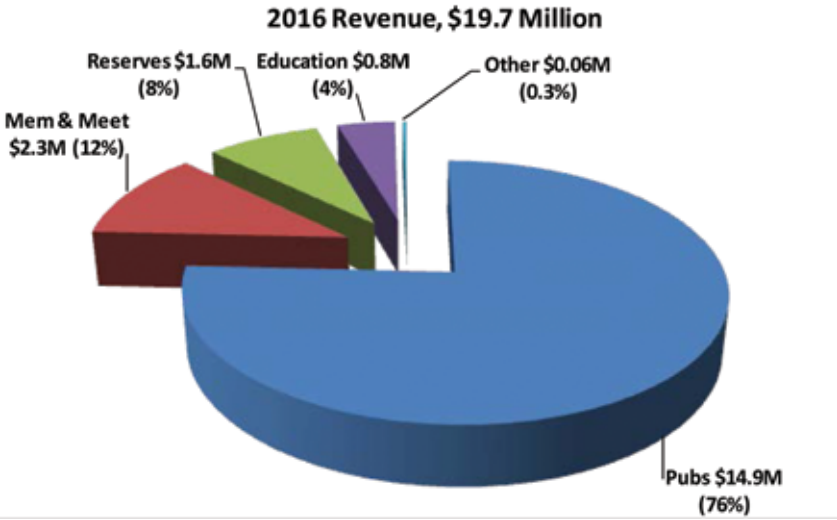
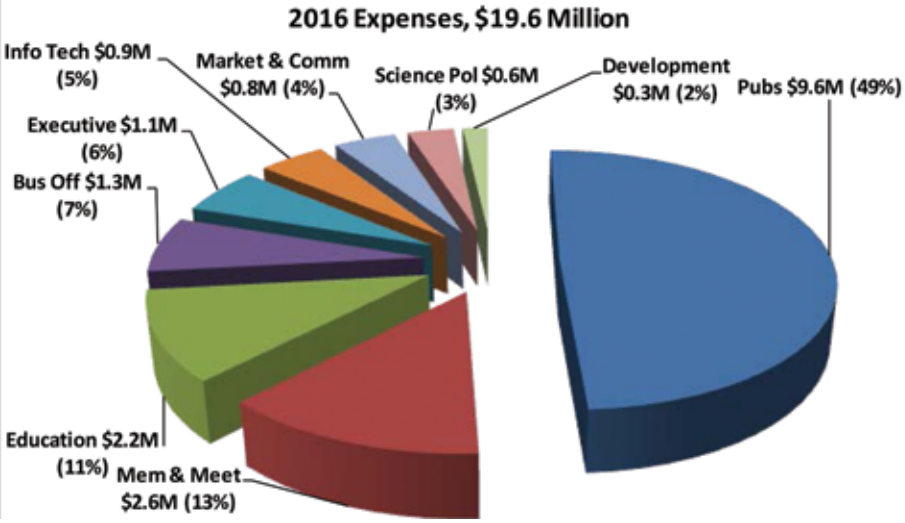


Figure 2

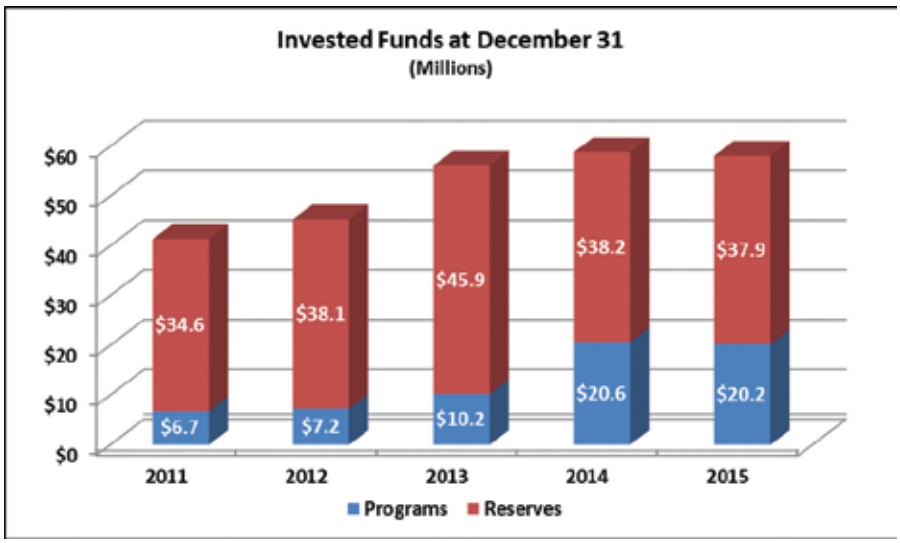


The 2016 operating budget has revenue of $19.7 million, expenses of $19.5 million, and a surplus of $234,000. Figures 1 and 2 represent the sources of revenue and the allocation of expenses, respectively, in the 2015 operating budget. The 2015 report and 2016 budget were approved by Council at its spring meeting.

Investments

The Society's long-term invested funds at December 31, 2015 totaled $58 million. Of those invested funds, approximately $38 million are set aside as reserves, a portion of which is used to support the annual operating budget. The remaining $20 million is used to support the Society's numerous award programs and sections. Figure 3 reflects the overall growth of the Society's investments from 2011 to 2015 and the allocation between reserves and the award programs and section funds. It should be noted that, in 2014, Council authorized moving $9.95 million from reserves to the APS Endowment Fund to support award programs previously included in the operating budget.

Figure 3



Development Office

The development program, now in its second year of operation, has been organized around the need and desire to cultivate additional philanthropic support for the numerous programs and awards offered by the Society. The Development Team, John Van Ness and Megan Mitzelfelt, continues to work with the Society's leadership and staff to build relationships with individuals, corporations, and private foundations so that gifts and grants can be solicited for the Society's current endeavors and, ultimately, build the Society's endowed funds. Thus far, the Development Office has organized a full development program and created an annual operating plan and goals. Considerable headway has been made in identifying prospects, creating online and print communications and marketing materials, initiating annual fund solicitations, approaching foundations and corporations, and making personal calls on major/planned gift prospects.

2015 Annual Report

The Development Office has produced the second APS Annual Report, which reviews the Society's 2015 accomplishments, finances, and lists all donors.

Development Marketing

The communications, marketing, and annual solicitation plan is being implemented; we are presently at mid-year. The activities thus far are given, in brief, in Table 1.

Table 1. Activities in Brief

Giving Program	Appeal	Date(s)	Target	Method	Purpose	Actions and Results
Annual	Membership renewal	January	Members	Letter	To solicit annual gifts	37 gifts; $2,372.50
Planned	Charitable gift annuity appeal	January-February	Members, 65+	Postcard; E-Blast	To cultivate planned gift donors	21% open rate; few prospects identified
Planned	Wills guide appeal	April-June	Members, 45+	Postcard; E-Blast	To identify prospects	140 website visitors; 50 wills guide requests
Annual	Conference sponsorships	January-present	Corporations and foundations	Brochure	To solicit sponsorships and exhibits	Submitted 14 grants and 27 requests; 5 gifts and 2 exhibits; $11,800
Annual	2015 Annual Report	June	Leadership and donors	Report; E-Blast	To recognize and retain donors	Online; mailed to donors and leadership
Annual	The Founders Circle	June	Leadership, prospects, donors	Letter and brochure	To solicit annual gifts and develop prospect pool	Mailed; TBD
Annual	Donor newsletters	July	Donors	Report	To report on gift impact	Mailed; TBD
Annual	Mid-year solicitation	June-July	Members	E-Blast; letter	To solicit annual gifts	E-mailed; TBD
Planned	Bequest appeal	June-July	Members, 65+	Postcard	To cultivate planned gift donors	Mailed; TBD

Corporate And Foundation Relations

Corporate and foundation communications, marketing, and funding proposals are briefly summarized in Table 2. Particular note should be made of two strategic partnership proposals submitted to ADInstruments and DSI, which are currently pending. Also, after considerable time and effort, APS has been approved to submit a proposal to the Hearst Foundations for the Undergraduate Summer Research Fellowship program. Last, a new brochure has been created to market and solicit conference sponsorships.

Major and Planned Giving

Considerable progress had been made in identifying and contacting major/planned gift prospects. At present, we've identified and researched 213 individual members who may be interested in considering a gift. Personal calls on these prospects have been initiated to introduce them to the giving opportunities and to discuss their particular interests and philanthropic objectives. See Table 3 for a summary of these activities.

Table 2. Corporate and foundation proposals

	Total	Donors	Letters of Inquiry	Grants	LOIs and Grants In-Progress	Strategic Partnership Proposals	Sponsorship and Exhibit Requests
As of 6/24	$212,431	33	4	14	2	2	27
Goal	$299,533	50	6	10	n/a	1	10
Percent of goal	71%	66%	67%	140%	n/a	200%	270%

NOTE: There are five outstanding pledges ($27,245).

Table 3. Pipeline report

Prospect Stage	Number
Identified	213
Contacted	158
Visited	73
Promising prospects	31
Founders Circle donors (as a result of visits)	17
Major and planned gifts (pending and completed)	3

Information Technology

The IT Office has numerous projects underway to continue improving the efficiency and effectiveness of the Society's information systems:

- The Personify association management system was upgraded to version 7.5.2 in September 2015. Since that time, IT has been working with the various departments to improve and expand their use of Personify to meet the Society's business processing and information needs.
- In conjunction with Finance and Publications, in March, IT implemented the in-house processing of Author Fees orders that had previously been outsourced to Cenveo, the Society's composition and printing contractor. Savings in excess of $100,000 per year are anticipated as a result of the change. In addition, the Personify-generated documents and e-mail notifications used in the new process have greatly increased processing efficiency resulting in improved customer service to the Society's authors. The project has also increased staff's confidence and reliance in Personify and resulted in their increased use of its functionality and reporting capabilities in place of manual processes. This trend will continue as IT proceeds with the work to consolidate the data existing in our various disparate systems into a central APS enterprise database.
- The Personify 7.5.2 upgrade also brought a number of improvements to the My Profile and APS store website, and additional changes are in progress to make further improvements to the functioning and responsiveness of those webpages.
- The project for redesigning the APS website and implementing a new content management system has begun, and is in the initial phase of fleshing out the details for the request for proposal to be distributed to interested vendors.
- Recently, the IT Office completed moving the APS servers and network to the colocation site of our managed services provider, ZyEdge. In addition to improving the Society's disaster recovery and business continuity capabilities, having our technology infrastructure at a colocation center readily provides access to the scalable resources needed for anywhere office capabilities. As a part of this project, IT completed moving the e-mail system to Microsoft's Office 365 Exchange Online service in February and has replaced 75% of the staff's desktop computers with laptops running Window 10 Pro. IT is in the process of rolling out the remaining subscribed Office 365 services of Skype for Business and OneDrive for Business to provide for business class audio/web conferencing and sharable cloud drive services.
- IT is investigating options for providing phone systems and Internet access to the APS office, which would result in increased phone system capabilities and faster internet service compared with the same services currently provided by FASEB.

2015 Financial Statements

What follows are facsimiles of the Society's 2015 audited financial statements.

Council accepted the report of the Finance Committee. ●

The American Physiological Society

Statements of Financial Position

December 31, 2015 and 2014

Assets	2015	2014
Cash and cash equivalents	$ 975,423	$ 769,723
Investments	63,560,541	61,774,706
Certificates of deposit	217,064	412,886
Accounts receivable, net	895,401	1,028,127
Pledges receivable, net	412,523	323,325
Accrued interest receivable	132,189	128,326
Advances to section editors	69,463	92,162
Prepaid expenses	323,448	273,068
Inventory	54,900	56,630
Property and equipment, net	1,451,658	1,551,514
Total assets	$ 68,092,610	$ 66,410,467
Liabilities and Net Assets		
Liabilities		
Accounts payable and accrued expenses	$ 1,967,462	$ 1,479,656
Deferred subscriptions	6,846,161	5,423,621
Deferred dues and other	594,587	580,638
Capital lease obligations	106,053	140,062
Total liabilities	9,514,263	7,623,977
Net Assets		
Unrestricted	56,934,714	57,172,250
Temporarily restricted	1,128,324	1,098,931
Permanently restricted	515,309	515,309
Total net assets	58,578,347	58,786,490
Total liabilities and net assets	$ 68,092,610	$ 66,410,467

The American Physiological Society

Statement of Activities

For the Year Ended December 31, 2015

	Unrestricted	Temporarily Restricted	Permanently Restricted	Total
Operating Revenue				
Subscriptions	$ 11,153,862	$ -	$ -	$ 11,153,862
Author charges	3,066,418	-	-	3,066,418
Membership dues	1,123,272	-	-	1,123,272
Grants and contracts	739,525	-	-	739,525
Conferences and meetings	802,401	-	-	802,401
Contributions	495,293	161,685	-	656,978
Advertising	183,226	-	-	183,226
Other income	405,937	-	-	405,937
Released from restrictions	129,059	(129,059)	-	-
Total operating revenue	18,098,993	32,626	-	18,131,619
Operating Expenses				
Publications	10,832,776	-	-	10,832,776
Society general	3,733,936	-	-	3,733,936
Society programs	1,350,961	-	-	1,350,961
Education	1,791,287	-	-	1,791,287
Marketing	1,051,241	-	-	1,051,241
Total operating expenses	18,760,201	-	-	18,760,201
Operating Change in Net Assets	(661,208)	32,626	-	(628,582)
Non-Operating Activities:				
Investment Income				
Net realized gain on investments	2,594,420	-	-	2,594,420
Net unrealized loss on investments	(2,650,241)	(3,233)	-	(2,653,474)
Interest and dividends	1,166,478	-	-	1,166,478
Investment management fees	(686,985)	-	-	(686,985)
Total investment income	423,672	(3,233)	-	420,439
Change in Net Assets	(237,536)	29,393	-	(208,143)
Net Assets, beginning of year	57,172,250	1,098,931	515,309	58,786,490
Net Assets, end of year	$ 56,934,714	$ 1,128,324	$ 515,309	$ 58,578,347

International Physiology Committee


Michael Hill, Chair

APS members represent 88 countries from 6 continents with over a quarter of the Society's membership being international. International members represent a large and important constituency within The Society, and one that is growing – over 30% of new members are internationals. At recent Experimental Biology meetings, international registrants comprised over 20% of the total registration (some two-thirds of whom are APS members). These figures clearly indicate the international nature of APS.

The role of the International Physiology Committee (IPC) is to assist APS by identifying and implementing ways in which APS can best serve its international members, achieve globally its goals of fostering education, scientific research, and dissemination of information in the physiological sciences, and raise its global stature.

The IPC assists APS in the review of international awards programs. The International Early Career Physiologist (IECP) travel awards program provides support for students, trainees, and junior faculty working outside the U.S. to attend Experimental Biology (EB). The IPC reviewed 36 applications, and APS made 11 awards of $1,000 each for EB2015. Successful applicants represented diverse regions, including South and North America, Europe, and Australia.

The IPC also reviewed applications for two APS travel awards this year: the APS-The Physiological Society Joint Meeting and the 2016 ICPS-Beijing Conference Travel Award. The IPC has promised support to 39 awardees for the APS-TPS Joint Meeting to be held in Dublin Ireland from July 29-31. We will also be supporting six awardees for the 2016 ICPS meeting to be held on September 25-28, 2016 in Beijing, China.

The International Opportunity Program (IOP; formerly the Latin-America Initiative) aims to strengthen ties between APS and international societies on a global level, with a particular emphasis on underrepresented regions. We had two rounds for 2016, with the program providing support for six applications total, with each receiving $7,500.

IOP – Round 1 Awardees:

- Tania Zenteno-Savin (Centro de Investigaciones Biologicas del Noroeste). Organization of a 3-day conference entitled "Second International Conference on Oxidative Stress in Aquatic Ecosystems" to be held in La Paz, Baja California Sur.
- Mubarak Hussain Syed (HHMI-Institute of Neuroscience). Organization of a 2-wk course entitled "Insect Neuroscience from Stem Cells to Neural Circuits" at the Institute of Biomedical Sciences Mekelle University, Mekelle, Ethiopia.

IOP – Round 2 Awardees:

- Lara De Ruisseau (Le Moyne College, Syracuse, NY). Organization of a day-long symposium entitled "The Physiology that Unites Exercise and Cognition" to be held at the University of Eastern Finland, Kuopio, Finland.
- Willis Samson (St. Louis University, St. Louis, MO). Organization of a workshop entitled "Designing Molecular Tools for the Study of Integrative Physiology" to be at the Institute of Medical Investigation Mercedes y Martin Ferreya, Cordoba, Argentina.
- Talant Sooronbaev (Kyrgyz National Center for Cardiology and Internal Medicine, Kyrgyz Republic). Support for the organization of the Issyk-Kul Leh Symposium to be held in Issyk Kul Lake, Cholpon Ata, Kyrgyzstan.
- Jun Sun (University of Illinois, Chicago, IL). Organization of a 2-day international symposium/workshop entitled "East Meets West: Advances in Gut Microbiome effects on Pathophysiology of Human Diseases" in Yangzhou, China.

2015 IOP supported initiatives (representing programs undertaken in South America and Africa) were undertaken, and outcome reports were submitted to APS.

Application for IOP support for 2017 was advertised, with a deadline for submission of June 30, 2016. So far, seven applications have been started for this award.

Sincere thanks are extended to Esther Samuel (APS), for her excellent administrative support. Thanks are also given to the members of the International Committee for review and ranking of the various submissions.

Council accepted the report of the International Physiology Committee. ●

IECP Awardees 2016

Awardee	Institution
Thales Barbosa	Fluminense Federal University, Brazil
Jan Czogalla	University of Zürich, Switzerland
Rodrigo Del Rio	Universidad Autonoma de Chile, Chile
Igor Fernandes	Federal Fluminense University, Brazil
Linda Gallo	Mater Research Institute-University of Queensland, Australia
Mohammad Jafarnejad	Imperial College London, UK
Simon Malenfant	Quebec Heart and Lung Institute, Canada
Jamie Mayo	University of Melbourne, Australia
Aaron Phillips	University of British Columbia, Canada
Bethan Phillips	University of Nottingham School of Medicine, UK
Amin Shah	University of Alberta, Canada

How can APS better serve its international members? How can APS serve in outreach to the international physiology community? If you have suggestions, ideas, or concerns, please contact Michael Hill, Chair of the APS International Physiology Committee, at *hillmi@missouri.edu*.

John F. Perkins Jr. Memorial Award Committee

Paul Davenport, Chair

The John F. Perkins Jr. Memorial Award for International Physiologists promotes cultural exchange and scientific collaborations by providing supplementary aid to families of foreign scientists working for a minimum of 3 months in the U.S. In this way, young scientists are able to bring spouses and children, and thus make full use of the cultural exchange as well as the scientific benefits associated with an international collaboration. This award is intended to support spouses' and children's visits to the U.S. for postdoctoral fellows and junior faculty from overseas. Application for the Perkins Award must be made jointly by the host, who must be an APS member, and the visiting scientist. The recipient receives funds generally not exceeding $5,000.

Applications for the Award are accepted in the spring and fall, with application deadlines of April 15 and October 15. For the October 2015 deadline, the Committee received two applications and funded both with a stipulation for one applicant. For the April 2015 deadline, the Committee did not receive any applications. The committee revised the description of the award to clarify that the award is intended to bring over a family consisting of a spouse and children.

Council accepted the report of the John F. Perkins Jr. Memorial Award Committee. ●

Joint Program Committee


Robert Hester, Chair

Overview of Committee Activity

Experimental Biology 2016

The 2016 EB Meeting was held in San Diego from April 2 to April 6. Participating societies were APS, AAA (anatomy), ASBMB (biochemistry), ASIP (pathology), ASN (nutrition), and ASPET (pharmacology). The APS hosted five guest societies: the Microcirculatory Society (MCS), the Biomedical Engineering Society (BMES), American Federation for Medical Research (AFMR), and The Physiology Society (Physoc). Overall paid scientific registrants across all societies was 11,488, with 1,253 ancillary registrants. APS had 395 sessions in total: 249 poster sessions, 62 symposia, 51 featured topics, 19 lectures, 2 workshops, 1 refresher course, 2 awards sessions, and 9 special sessions.

The meeting opened with several unique sessions on Saturday, April 2, including the traditional Refresher Course this year focusing on "Keep Your Eye on the Ion: Refresher Course on Ionic Homeostasis and Systems Physiology." APS sponsored two unopposed Techniques and Technology in Physiology Workshops on Saturday, April 2, entitled "Novel Methods to Perturb Genes for Physiological Examination" and "Advanced Microscopy Techniques for the Study of Physiology." In addition, Saturday featured *Physiology in Perspectives: The Walter B. Cannon Memorial Award Lecture* presented by Amira Klip from the Hospital for Sick Children, Toronto, Canada. The lecture was followed by an opening reception free to all physiology registrants.

APS also held the *Henry Pickering Bowditch Memorial Award Lecture* featuring Sean Stocker of Penn State University and 12 section-sponsored distinguished lectures. APS also sponsored four Integrative Symposia Systems:

- Role of Epithelium in Innate Defense: More than a Barrier
- Early Life Stress and Sex-Specific Manifestations of Cardio-Respiratory Dysfunction: Insight from Microglial Cells
- Mechanobiology of Fibrosis Across Organ Systems
- Reprogrammed Cells as Models for Disease

The APS President's Symposium Series was organized by Patricia Molina around the theme *Physiological Mechanism Responsive to Behavioral and Environmental Challenges* and included three symposia and one lecture:

- "Physiological Processes Underlying Organ Injury in Alcohol Abuse"
- "Dietary Influences on Physiological Control Mechanisms: How Much, When and What"
- "Physiological Adaptation to Behavioral, Environmental, and Chronological Stress"
- *Nobel Prize in Physiology or Medicine Lecture* presented by Dr. Roger Tsien (Howard Hughes Medical Institute, Univ. of California, San Diego)

A total of 7,761 abstracts, 6,979 regular and 782 late-breaking, were programmed by the six EB 2016 sponsoring societies. Table 1 provides a breakdown of on-time and late-breaking abstracts programmed over the past 5 years. Total abstracts submitted for EB 2016 decreased by slightly more than 4% over EB 2015, which received a total of 8,159 abstracts, and less than EB 2014 with total of 7,907 abstracts.

Table 1 provides the breakdown of total attendance at EB 2016 and 2015. EB 2016 represented 730 fewer scientific registrants than EB 2015. The total number of undergraduate/high school members, teachers, and students was 1,919, a decrease of 590. Ancillary registration decreased by 260. Total registration decreased by 1,580 compared with EB 2015. There has also been a slow decrease in number of exhibitor booths and companies over the last 4 years.

Experimental Biology 2017

EB 2017 to be held Saturday, April 22 through Wednesday, April 26 in Chicago, IL.

The JPC met again on June 13-14, 2016 in Crystal City, VA to schedule rooms by day and time for the platform sessions, and to minimize scientific overlap.

Table 1. Registration rreakdown EB 2016 and EB 2015

	EB 2016-San Diego		EB 2015-Boston	
Registration Type	**Total**	**Percent**	**Total**	**Percent**
Member	4,648	36%	4,975	36%
Retired member	144	1%	120	1%
Retired nonmember	24	0%	0	0%
Nonmember	834	7%	956	7%
Postdoc member	612	5%	658	5%
Postdoc nonmember	270	2%	285	2%
Grad student member	1,739	14%	1,782	14%
Grad student nonmember	1,047	8%	1,229	8%
One-day member	151	1%	179	1%
One-day nonmember	100	1%	115	1%
Total Paid Scientific	**9,569**	**75%**	**10,299**	**75%**
Undergraduate member	529	4%	537	4%
Undergraduate nonmember	986	8%	1,324	8%
High school student	319	3%	477	3%
High school teacher member	36	0%	97	0%
High school teacher nonmember	49	0%	74	0%
Total Paid Undergrad/High School	**1,919**	**15%**	**2,509**	**15%**
Exhibitors	943	7%	1,121	7%
Guest of exhibitors	199	2%	277	2%
Guest	86	1%	68	1%
Press	21	0%	42	0%
Blogger	4	0%	5	0%
Total Paid Ancillary Registration	**1,253**	**10%**	**1,513**	**10%**
Grand Total Registration	**12,741**		**14,321**	

We had 32 Section and Interest Group JPC Reps attend the meeting. We developed 14 tracks for the EB 2017 meeting and then programmed the scientific sessions. JPC representatives were informed that speakers and chairs were not allowed to speak in or chair multiple sessions. Several APS Sections and Interest Groups added "Nutrition" to abstract categories to attract other members of EB to APS sections.

Translational Physiology Interest Group will again be selecting translational abstracts for an unfunded session where investigators will provide 5-minute presentations on translational physiology. The meeting will also feature sessions organized by the APS Publications, Careers in Physiology, Public Affairs, Women in Physiology, Trainee Advisory, Physiologists in Industry, and Education Committees.

The Call for Abstracts and online abstract submission site will be available by September 2016. The abstract deadline will be November 17, 2016. There will be no extension to the November 17, 2016 abstract deadline. Abstracts submitted after November 17 will be considered late-breaking and scheduled as posters on Wednesday, April 26, 2017. EB 2017 will provide a late-breaking abstract deadline of February 8th, 2017.

The EB Management Committee approved discontinuing distribution of the printed meeting program. The entire program will be available online as a printable pdf and on a downloadable mobile app.

The JPC received 11 integrative physiology symposium proposals for EB 2017 of which the following were been selected.

- Exercise Intolerance in Metabolic, Pulmonary, and Cardiovascular Diseases: Did We Forget the Brain?
- Hypoglycemia: Neural Pathways of the Glucose Counter-Regulatory Response, Hypoglycemia Unawareness and its Treatment
- Engineering for Health: Integrating Engineering and Physiology to Combat Disease
- The Impact of Exosomes on Muscle Atrophy, Hypertrophy and Myogenesis

In addition, two workshops are scheduled on the first day of EB 2017:

- Choosing the Right Diet for Your Animal Model (Lund/Ney)
- Using Ultrasound to Teach Cardiovascular Physiology and Anatomy (Silverthorn/Guttmann) – cosponsored by AAA

The President's Symposium Series, organized by Jane Reckelhoff, entitled *Research Advances in Sex/Gender and Developmental Programming of Chronic Diseases*, will feature a series of three symposia and a Nobel Lecture:

- Developmental Programming of Diseases (Alexander)
- Sex Differences in Physiology and Pathophysiology (Prieto)
- Women's Health Research (Brooks)
- Nobel Prize Award Lecture: Louis Ignarro, UCLA

The Cannon Lecture will be given by Michael Welsh. The Bowditch Lecture will be given by Brant Isackson.

Action Items

In an effort to stabilize Wednesday afternoon programming and ensure attendance at the Nobel Lecture, Council approved providing complimentary registration to presenters in Wednesday afternoon sessions as follows: all speakers and chair(s) on eight symposia sessions and up to two registrations on one featured topic session.

Council accepted the report of the Joint Program Committee. ●

Membership Committee

Robert Brock, Chair

Overview of Committee Activities

The majority of the efforts put forth by the Membership Committee over the previous year have centered on the continued development and implementation of the new Fellow of the APS (FAPS) Program. The inaugural review and approval process was completed with little issue. A few small changes for clarification and efficiency purposes have been proposed to Council for approval. In addition, a cohort of APS members from the National Academy of Engineering has also been included in the inaugural class of APS Fellows (FAPS). They are:

- James B. Bassingthwaighte (University of Washington, Seattle, WA)
- Emery N. Brown (Massachusetts General Hospital, Harvard Medical School, Boston, MA)
- Yuan-Cheng B. Fung (University of California, San Diego, CA)
- Morteza Gharib (California Institute of Technology, Pasadena, CA)
- Geert W. Schmid-Schoenbein (University of California, San Diego, CA)
- Watt W. Webb (Cornell University, Ithaca, NY)
- Sheldon Weinbaum (City University of New York)
- Ajit P. Yoganathan (Georgia Institute of Technology, Atlanta, GA)

Furthermore, the Committee continues to advocate for a promotional campaign to better communicate the benefits of APS membership to aid with member recruitment and retention. There is an on-going interaction with Veronica Purvis, the APS Director of Marketing, toward this goal of effectively recruiting and retaining target membership sub-groups.

Membership Statistics

The Chair reviewed the spring membership status report. The total number of members is 10,830. [Note: 1,254 (912 regular) members were dropped on April 1, 2016 as a result of unpaid dues.] There was a net gain of 209 regular, 196 graduate student, and 36 undergraduate student members since the Fall 2015 status report. The current retention rate is 91%.

Council accepted the report of the Membership Committee. ●

Physiologists in Industry Committee

Eugene Shek, Chair

EB2016 PIC Symposium

The 2016 symposium, entitled "Metabolic Syndrome and the Pathway of Drug Development: From Bench to Bedside," was held on Sunday, April 3, 10:30-12:30 PM at San Diego Convention Center, Room 22. This symposium focused on the known risk factors involved in the metabolic syndrome. It also covered basic research and various steps (ranging from early drug discovery, applied preclinical, development, safety, and the clinical and regulatory hurdles) that are required to get a drug from conception to FDA approval. This symposium was chaired by Matthew R. Zahner and co-chaired by Denise C. Cornelius, who was our 2015 PIC Novel Disease Model Award Postdoctoral recipient.

EB2016 Novel Disease Awards

The PIC Novel Disease Model Award is sponsored by Novo Nordisk Research Centre China. Two awards are granted each year to a graduate student ($500) and a postdoctoral fellow ($800) who submit the best abstracts at EB that describe a novel disease model (either in vivo or in vitro). Applicants do not have to be APS members, and there are no restrictions on how the award is spent. Moreover awardees can only receive the Novel Disease Award once as a postdoctoral fellow and once as a predoctoral student. Our 2016 predoctoral and postdostoral awardees, respectively, are Renato Gaspar (Federal University of Maranhão, Sao Luis, Brazil) and Kavaljit Chhabra (University of Michigan, Ann Arbor, MI). Awardees were recognized at the 2016 annual APS Business Meeting.

Physiologists in Industry Committee Mixer

The 16th Annual Physiologists in Industry Committee Mixer was held at the San Diego Marriott Marquis & Marina (Sunday, April 3, 6:45-8:00 PM, Malibu Room). The Industry Mixer is traditionally a great opportunity for our students, postdoctoral fellows, and academic colleagues to network with APS industry members. It is designed to attract trainees and engage them in discussion about career, research, and opportunities in industry positions. The mixer was well received and attended by students and postdoctoral trainee.

Annual Physiologists in Industry Committee Meeting

The annual APS PIC meeting was scheduled on Sunday, April 3, 2:30-4:00 PM in the San Diego Marriott Marquis & Marina, Carlsbad Room; this was the committee's annual opportunity to have a face-to-face meeting to discuss committee business and new initiatives. Eugene W. Shek, the current PIC chair, will complete his 3-year term at the end of 2016. Matthew R. Zahner will become the new PIC Chair starting on January 1, 2017. Please join me to welcome and congratulate Zahner for his new role.

EB 2017 PIC Symposium

The Physiologists in Industry Committee is sponsoring an exciting Symposium with Environmental & Exercise Physiology Section at EB 2017, Chicago, entitled "Nitric Oxide Pathway Modulation for Therapeutic Intervention and Exercise Enhancement," on Sunday, April 23, 10:30-12:30 PM at Chicago Convention Center. This symposium will explore recently discovered therapeutic compounds and nutritional supplementation as a way to enhance NO pathway signaling in health and disease. It will provide a great opportunity for interaction and knowledge exchange

between our academia and industry colleagues. This section will be chaired by John Mattson and co-chaired by Kavaljit Chhabra, who is our 2016 PIC Novel Disease Model Award Postdoctoral Recipient. Looking forward to seeing you all at the EB 2017 PIC Symposium.

Council accepted the report of the Physiologists in Industry Committee. ●

Porter Physiology Development and Minority Affairs Committee

Maggie Curras-Collazo, Chair

Clifford Barger Underrepresented Minority Mentorship Award

The Porter Physiology Development and Minority Affairs Committee (PPD-MAC) was pleased to launch the new A. Clifford Barger Underrepresented Minority Mentorship Award (*www.the-aps.org/barger*) during EB 2016. The Barger Award promotes and embodies the APS's goal of broad diversity among physiologists by recognizing outstanding mentors who make significant impacts on diversity in physiology. It recognizes mentoring as a highly valued professional activity that merits a high level award by the Society.

APS-Sponsored Local Undergraduate Research Awards in Physiology

Council approved this new program in Summer 2015, conceived as a joint proposal with COPC, which will provide up to 50 awards per year to undergraduate award winners of university poster competitions in physiology judged by APS members. The program was launched in April 2016. APS members are encouraged to request an award packet at *www.the-aps.org/LURAP*.

Porter Physiology Development Fellowship Program

The goal of the Porter Physiology Development Program is to encourage diversity among students pursuing full-time studies toward the PhD in the physiological sciences and to encourage their participation in the APS. The program provides 1- to 2-year, full-time graduate fellowships. The program is open to underrepresented ethnic minority applicants who are citizens or permanent residents of the U.S. or its territories. Fellows are expected to be/become APS members, participate in EB, complete online professional development activities, and participate in K-12 outreach. The PPD-MAC is pleased to announce the 2016-2017 Porter Fellows:

Table 1. 2016-2017 Porter Fellows

Name	Institution	Fellow Year
Olubusayo Awe	Johns Hopkins School of Medicine	2016-2017
Mariano Colon-Caraballo	Ponce Health Sciences University	2016-2017
Victoria Parker	University of Iowa	2016-2017
Candy Rivas	University of Arizona	2016-2017
Diarra Williams	University of Arkansas for Medical Sciences	2016-2017
Ijeoma Obi	University of Alabama, Birmingham	2015-2017
Jinae Roa*	Scripps Institution of Oceanography	2015-2017
Lindsey Stavola	Yale University	2015-2017

*J. Roa has been designated the Eleanor Ison Franklin Fellow for having the highest ranked renewal application.

Since 1967, the program has provided more than 246 fellowships to 145 trainees. For 2016-2017, the PPD-MAC selected a total of eight fellowships to be awarded. The stipend paid to the Porter Fellows for 2016-2017 will again be $28,300/year, consistent with the NIH scale. In 2014, Council approved increased funding for the Porter Physiology Development Fund from $40,000 to $80,000/year. The Committee expresses its sincere appreciation for the continued support of the William Townsend Porter Foundation, APS member contributions, and the APS Council that makes these fellowships possible.

Beginning with the 2016-2017 Porter fellow cohort, PPD-MAC members will correspond with these students on a quarterly basis with encouragement and coaching. PPD-MAC members will be able to confidentially discuss any issues as they arise and will select the fellows they will coach/mentor. This new program component has been added to the Condition of Award.

Another initiative is to engage Porter fellows in APS-sponsored opportunities. PPD-MAC will continue to discuss new approaches to reach this goal, including the idea of showcasing Porter Fellow accomplishments and promoting Porter fellows as new trainee members within their respective Section's activities.

The Porter Fellowship program will be celebrating its 50th anniversary in 2017! Please watch for celebratory initiatives at EB 2017.

Social Media Outreach

The PPD-MAC manages a Facebook fan page to promote networking among minority physiologists. The Committee posts news related to professional development opportunities one to two times weekly. The group currently has 383 followers. The direct link to the page is *www.facebook/APSMinorityPhysiologists*.

Minority Travel Fellows Program

The PPD-MAC reviewed and recommended 40 award recipients for Minority Travel Fellowships to attend EB 2016. This year, 6 of those 40 travel fellows were supported with funding from Janssen Pharmaceutical Companies of Johnson & Johnson. The latter were facilitated by past Porter fellow, Raul Camacho. Again this year, PPD-MAC was pleased that former Porter Fellows and past Travel Fellows volunteered to be mentors for the "younger" Travel Fellows. Since its inception in 1987, the APS Minority Travel Fellowship Program has awarded 823 travel fellowships to 582 undergraduate students, graduate students, postdoctoral fellows, and faculty members at minority institutions. As in the past, the Committee held a reception for Travel Fellows, their meeting mentors, and past and current Porter Fellows. This reception builds stronger connections between minority students and the larger community of APS scientists, especially other minority scientists. The Porter reception again this year was extremely successful. The Travel Fellows Luncheon was held on Wednesday of the EB meeting. The Fellows heard from keynote speaker-Sue Bodine (University of California, Davis). Bodine's talk, "Fostering Successful Scientific Collaborations Between Academia and Industry," provided insights and perspectives on her career path from academia to industry and back again to academia.

Annual Biomedical Research Conference for Minority Students

APS staff and PPD-MAC members exhibited at the November 2015 meeting in Seattle, WA to promote undergraduate programs, graduate study in physiology, and the APS programs for minority students. The APS was pleased to again provide $2,500 for cash awards for the most outstanding undergraduate presentations in physiology research. Ten undergraduate students received APS-sponsored awards for the best oral and poster presentations in the physiological sciences. Students also received a complimentary 1-year undergraduate membership to APS. Awardees were added to the APS Minority Physiologists and APS Trainee Communities on APS CONNECT.

Society for the Advancement of Chicanos and Native Americans in Science

In 2015, the theme for the Society for the Advancement of Chicanos and Native Americans in Science (SACNAS) annual conference was "Science, Culture, and Community." APS exhibited at this conference, which took place October 29-31 in National Harbor, MD (outside Washington, DC). Over 3,700 attendees participated in the conference. Most attendees were undergraduates. APS staff and PPD-MAC members manned the APS booth to promote APS diversity programs, K-12 resources and materials, and Undergraduate Summer Research Fellowships, as well as other APS awards, grants and fellowships, trainee and career information,

and APS membership. This summer, Council approved funding for presentation awards, plus APS membership, to be given for outstanding undergraduate student poster and graduate student oral presentation awards. A total of $2,100/year for 5 years ($10,500 total) was committed.

K-12 Minority Outreach Fellows Program

This program fosters communication between URM graduate and postdoctoral students and middle/high school URM life sciences students. Two awardees were selected for 2016-2017: Carmen de Miguel, a postdoctoral fellow at University of Alabama at Birmingham, and Joshua Streak, a graduate student at University of New Mexico. Congratulations!! Fellows attended EB 2016 to learn about outreach techniques and activities, will conduct outreach and attend either SACNAS or ABRCMS this fall, and will attend EB again in 2017 to conduct outreach activities.

"Be Counted" Campaign

The Porter Committee continues to seek the most current information about APS members including gender, race, and ethnicity to better tailor its programmatic initiatives and track successes in current ones. The Committee once again promoted this campaign at EB 2016 and other APS-sponsored conferences by providing informational and promotional materials at APS-sponsored booths. At the Committee's request, President Molina sent a brief survey to APS members with missing profile information. Overall, the combined efforts have increased member compliance. PPD-MAC will continue to monitor URM and ethnicity profiles in an effort to better inform other APS committees and Sections.

Council accepted the report of the Porter Physiology Development and Minority Affairs Committee. ●

Publications Committee

Curt Sigmund, Chair

Editor Reappointments

The first terms of Editors-in-Chief P. Darwin Bell (*AJP-Renal Physiology*), Willis K. Samson (*AJP-Regulatory, Integrative, Comparative Physiology*), and David Pollock (*Comprehensive Physiology*) were evaluated for reappointment to a second 3-year term, effective July 1, 2016. The Publications Committee unanimously reappointed all three Editors-in-Chief to a second and final 3-year term. At the fall 2016 meeting, the Publications Committee will interview for the Editorship of the *Journal of Applied Physiology*. The deadline for nominations is August 15, 2016.

New Editors 2016

AJP-Endocrinology and Metabolism: André Marette, University Laval, Québec replaced Charles Lang as of July 1, 2016.

Scientific Impact/Attractiveness to Authors

Journal Statistics

Impact Factor. The 2015 Journal Impact Factors (IF) published in Journal Citation Reports by Thomson Reuters released June 2016 has APS Journals generally holding their rankings. The following journals increased appreciably in Impact Factor: *Physiology* (from 4.857 to 6.541), *AJP-Lung* (from 4.080 to 4.721). *Comprehensive Physiology* fared well with its second full-year IF of 6.364. *Physiological Reviews* once again ranked no. 1 in the field of physiology, with an IF of 30.924. *AJP-Endo* (3.825), *AJP-Renal* (3.390), *AJP-Regu* (3.168), *PG* (2.995), and *Advances* (1.481) also increased their rankings. See *http://www.the-aps.org/mm/Publications/Journals/Impact-Factors.html* for full list of Impact Factors.

Manuscripts received. Manuscript submissions in 2015 decreased overall by 1% vs. 2014 across all original research journals and all manuscript types. (There was a 3% decrease in 2014 vs. 2013.) Submissions to *AJP-Lung* increased by 20%; *JN* by 15%; *AJP-Heart* by 2%; and *Advances* by17% compared with 2014.

Articles and pages published. The number of regular research articles published increased by 5% to 2,701 in 2015 from 2,568 in 2014; published invited articles increased by 14% to 541 in 2015 from 473 in 2014. In 2015, 381 articles were published in *Physiological Reports*. The number of records accepted for AiPS decreased by 6% from 2,801 in 2014 to 2,782 in 2015. Journal pages published increased by 1% compared with 2014. The number of published pages was 20% under the 2015 page cap limit.

Supplemental data. A total of 372 data supplements were published in 2015, 140 of which contained video files. This represents a 53% increase in total data supplements from 2014 and a 59% increase in video files.

Podcasts. *AJP-Heart* released 29 podcasts in 2015 and 14 podcasts YTD May 2016, and *JAPPL* released 17 podcasts in 2015 and 6 podcasts YTD May 2016, highlighting published articles. *AJP-Cell* published one podcast in April 2016. *JN* launched a podcast series this year and has published eight podcasts through May 2016.

Time to first decision. Time to first decision averaged 21.2 days in 2015 across all of the original research journals, 0.6 days fewer compared with 2014. Of note, *AJP-Cell* time to first decision was fastest at 15.4 days, followed by *AJP-GI* at 17.4 days. The average time to first decision year to date May 2016 is 20.8 days.

Peer review system. Enhancements to the peer review system are made as requested by the EICs, and as needed by staff. These enhancements are ongoing. In 2015, there were approximately 12 enhancements developed and implemented.

Strategic Planning 2011-15

Following are the five key strategic objectives derived at the Strategic Planning retreat held January 2011:

- Increase efforts to ensure awareness of, and advocacy for, the discipline of physiology
- Actively work to attract, meet the needs of, engage, and retain membership subgroups
- Develop strategies to strengthen the Society's publications in a changing world
- Enhance opportunities for scientific interaction and exchange
- Increase the visibility of physiology in life sciences and health sciences education

Additionally, two themes were identified that cut across all five strategic priority areas:

- Physiology as critical in translational research
- Chapters as an underutilized resource for the strategic directions

Comprehensive Physiology

Commitment to *Comprehensive Physiology* (CPHY) will be enhanced by *1)* continuing it as open access for another year, *2)* increasing the marketing of CPHY, and *3)* encouraging the various APS science educational programs to use CPHY in their curriculum to increase its visibility among young investigators. To do this:

- Selections are made publicly available on a rotating basis.
- To enhance visibility, CPHY remains accessible to APS members via single sign on for members, and the initiative is being assessed.
- Enhanced marketing and sales efforts: gap analysis Wiley / APS Digital Library customers.

Best efforts must be exerted such that CPHY appears on all relevant indexing databases:

- CPHY is indexed in Scopus and included in PubMed. It received its second full year 2015 Impact Factor of 6.641. It is indexed and abstracted in SciSearch®, Journal Citation Reports/Science Edition, BIOSIS Previews, BIOSIS Reviews Reports and Meetings from volume 1, issue I (see also Comprehensive Physiology)

AE and Editorial Board Submissions to APS Journals

Editorial Board members should be encouraged to publish in APS journals by:

- EIC suppling a semi-annual or annual report to the AEs on the number and type of submissions from their Editorial Board members, and the EICs will include this information in their annual report to the Publications Committee
- Requiring editors to send an annual letter to Editorial Board members detailing how many manuscripts they have reviewed, how many they declined, and how many they submitted and published in the journal
- Strongly encourage AEs and Board to solicit work from their peers

EICs and AEs are encouraged to publish their best work in APS journals. EICs supply a semi-annual or annual report regarding their submissions.

Acceptance/Rejection Rates

Editors of journals with higher acceptance rates continue to be encouraged to bring their acceptance rates down to provide some consistency between journals. This will be continuously monitored, and EICs/AEs will be encouraged to continue this process. In addition to improvement in IF, this will provide a pool of potential cascade-down submissions for the new OA journal. AEs should be given more encouragement to reject mundane work.

Acceptance rate in some of the AJP sections whose acceptance rate is >50% should be reduced. The Publications Committee has requested that the APS journals' rejection rate be brought up to 60%, where appropriate. Across the board, the overall rejection rate in 2015 is 57%.

Publicize Our Best Work in a Virtual Selection

Mechanisms should be developed to recognize authors of highly cited/downloaded articles to encourage them to resubmit. Authors could receive a letter from EIC congratulating them on their work and asking them to consider submitting their next paper to the journal. This could also make the most viewed paper from each issue freely available (and the author notified that this has been done).

"Manuscript of the Year Awards" could be created for reviews and original research for each journal, with criteria to be established by consensus by the EICs.

The APS*select* collection, which selects the best articles from all APS original research journals and presents these as a special collection was launched January 2014 (see also *APSselect*)

Each Journal may offer an award (from "fungible" office funds) to a Best Paper by a Young Investigator Award.

A Conference Proceedings initiative was established in 2015, providing the option for APS conference organizers to arrange with an Editor-in-Chief to publish selected proceedings.

This initiative has been established (see *Editorial Initiatives*).

Revenue

Membership should be encouraged to make sure their institutional libraries maintain their subscriptions, acquire the complete Digital Library, and subscribe to the Legacy Collection if they have not already.

Notice is placed on member login page to encourage members to ask their librarian to subscribe to APS journals if their institution is not a subscriber

Annual Reviews Award for Scientific Reviewing

The *Annual Reviews* Award for Scientific Reviewing was introduced in 2012. The Award is given for excellence in providing systematic, periodic examinations of scholarly advances and provoking discussion that should lead to new research activity. The award recognizes an APS member who has written scientific reviews and has helped provide an enhanced understanding of the area of physiology reviewed. The successful candidate, chosen by the Publications Committee, is awarded $2,000 and up to $2,000 reimbursement toward travel to attend the annual Experimental Biology meeting to receive the monetary award and a recognition plaque.

The 2016 Award was given to two recipients who are recognized for their published research collaborations, namely Barbara Cannon (Wenner-Gren Institute, Stockholm University) and Jan Nedergaard (Wenner-Gren Institute, Stockholm University).

Annual Reviews has informed the APS that they will not renew the award after 2017, having determined that "there are more cost-effective ways . . . to recognize and promote excellence in scientific reviewing."

Publications Integrity and Policy

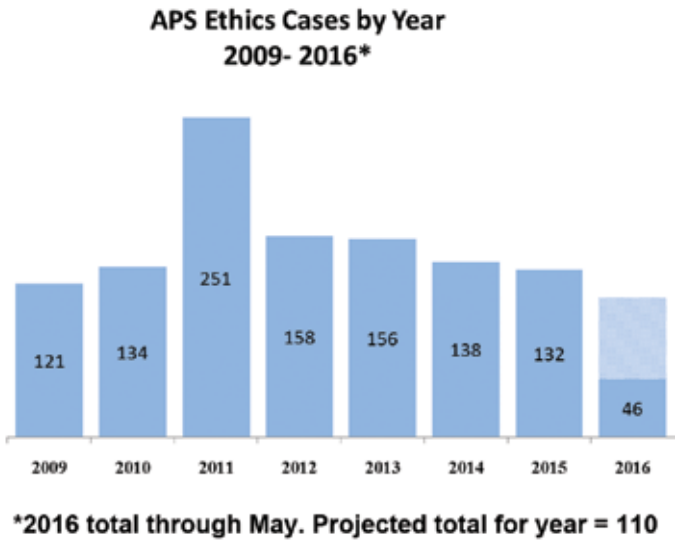
NSF grant. In 2012, the Publications and the Education Departments jointly submitted a successful application to the NSF for the development of modular course materials on publication ethics. The materials are to be used in Responsible Conduct of Research courses in STEM graduate programs. Additional collaborators on the project are the Biomedical Engineering Society (BMES) and the Society of Biological Engineers (SBE). The amount of the grant was $400,000 awarded over a

3-year period. Currently, the grant is on a 1-year no cost extension through summer 2016 to complete the grant goals and submit the final report.

NIH grant. In fall 2014, the Publications and Education Departments jointly submitted a grant proposal, solicited by NIH, to develop a training module to enhance data reproducibility (RFA-GM-15-006). The proposal, entitled "Controls in Animal Studies Professional Skills Course," utilizes APS expertise in developing professional skills training courses as well as the expertise of physiologists (Advisory Board) in designing animal studies. The proposed module focuses on the general factors that a researcher should consider when designing, analyzing, and reporting results from animal studies. Topics include sex, longitudinal studies, drug treatments, large animal models, and surgery. The proposal was well received and funded ($150,000). The 3-year grant period began on September 18, 2015.

Ethics cases. After a spike in 2011 (251 cases), the total number of ethics cases arising during peer review and production has decreased (from 158 cases in 2012 to 132 cases in 2015). Most cases were presentation errors that were fixed before early publication, with the majority of the balance of cases categorized as duplication of data and falsification of data. Other cases were categorized as duplicate publication, human/animal subject protocol, authorship concerns, and plagiarism.

In 2015, 71% of all cases were identified in the accepted stage before manuscripts were sent to AiPS. This compares to 75% in 2014. In 2015, the percentage of cases identified in AiPS was 2% (vs. 3% in 2014). The percent identified in the submitted/review stage was 17%, which compares to 13% last year. In 2015, 13 cases (10%) were identified after final publication compared with 12 cases (9%) in 2014. Six corrigenda and seven retractions were published. Sanctions were applied to three cases. Location of corresponding authors' institutions of 2015 ethics cases was 52% U.S. vs. 48% non-U.S. institutions, which is a switch from last year when slightly more international institutions were listed. Projected total cases for 2016 is 110, a 17% decrease vs 2015.



Publication ethics policy updates. In 2015, APS Information for Authors was amended as follows:

- October 2015: Added recommendations for databases appropriate for genome-wide datasets using next generation sequencing, i.e. Gene Expression Omnibus (GEO) (*http://www.the-aps.org/mm/Publications/Info-For-Authors/Data-Repositories*).
- November 2015: Authors are now permitted to deposit their accepted, peer-reviewed manuscript to an institutional repository with the following conditions: APS retains copyright, they observe a 12-month embargo, and a link to final published article on the APS website is featured (*http://www.the-aps.org/mm/Publications/Info-For-Authors/Copyright*).
- December 2015: Removed the *Journal of Neurophysiology* pre-print server exception to the Prior Publication policy (*http://www.the-aps.org/mm/Publications/Info-For-Authors/Ethical-Policies*).

December 2015: Removed the Ethical Process flowcharts from the Ethics Policy resource section and added a statement to the Ethical Procedure section of the Ethics Policy stating that the journals of the APS are members of the Committee on Publication Ethics (COPE) and support their principles. Issue-specific flowcharts are on the COPE website (*http://www.the-aps.org/mm/Publications/Info-For-Authors/Ethical-Policies*).

Changes to Instructions to Authors. In February 2015, changes were made to the description of the Letters to the Editor article type to emphasize that the decision to publish rests with the EIC (*http://www.the-aps.org/mm/Publications/Journals/AJP-Heart/Article-Types*).

In June 2015, the same change was made to the description of the Perspectives article type as was

made to the LTE article type (*http://www.the-aps.org/mm/Publications/Journals/AJP-Heart/Article-Types*).

In July 2015 detailed requirements for description of cell lines and other source materials were added (*http://www.the-aps.org/mm/Publications/Info-For-Authors/Formatting#cell_lines*).

Finance

Subscription sales. In 2012, a tiered pricing model was implemented for journals and the Legacy Content. Tiers were assigned based on the value of the journal to the institution. The goal was to have a pricing variance of 10% between tiers 1, 2, and 3 by 2014, which has been achieved.

The online-only 2015 price for tier 1 was set at 0% increase; the tier 2 price was set at 2% increase, and tier 3 at 5% increase. Print and online journal prices for 2015 were set at 2.5% increase for tier 1, a 5.5% increase was set for tier 2, and a 7.5% increase was set for tier 3. The print-only price increased by 10%. Subscription count decreased in 2015 by 4% compared with 2014.The number of institutions subscribed, based on consortia equivalents to individual institutional counts, are comparable to prior years. Sales representation covers Europe, the Mideast, Brazil, and Latin America, India, and China.

Color figures. Authors continue to take advantage of the benefit of scientifically warranted color made free of charge to first or last authors of original research articles who are APS members. (Scientifically necessary color is published free of charge in all review articles.) The number of color figures in research articles published in 2015 increased by 3%, from 5,135 in 2014 to 5,288 in 2015, of which 2,835 (54%) were free to APS members.

Advertising. The APS sells commercial advertising in its online and print journals through AdNet, a FASEB-managed advertising group.

Author fees. The author survey conducted by the Publications Department in 2014 raised several important issues that resulted in recommendations from the Publications and Finance Committees to implement the following revenue-neutral revisions to author fees applicable to manuscripts submitted as of January 1, 2016:

- Color fees for nonmembers have been reduced to $200 per figure from $400 per figure (this has been made possible through passing on to our authors cost savings resulting from production changes and a renegotiation of the APS contract with its composition and printing vendor)
- Elimination of page charges for the *AuthorChoice* option, whereby only the fixed fee ($2,000 for original research articles; $3,000 for review articles) is payable. *AuthorChoice* enables the article to be made freely available on publication without embargo
- Increase in page charges from $75 to $85 per page

AuthorChoice. The APS *AuthorChoice* program was introduced to all APS research journals in July 2007. For a fee of $2,000 ($3,000 for review articles) in addition to other author fees, an article is made freely available immediately upon payment and is delivered by the APS on behalf of the author to PMC to be uploaded upon publication. Starting with manuscript submissions in 2016, author pages charges that were in addition to the $2,000 (or $3,000 for review articles) are eliminated. This change brings the APS within community norms. In 2015, APS received 57 requests for this option vs. 51 requests for this option in 2014, which represents approximately 1.5% of all published articles during 2015.

As of 2013, to enable authors to comply with the policies of many funding agencies, APS has instituted the application of the CC-BY Creative Commons attribution license (*http://creativecommons.org/licenses/*) to *AuthorChoice* articles governing reuse in place of standard copyright.

Copyright and permissions. Since 2011, the Copyright Clearance Center (CCC) administers on behalf of APS permissions for reuse of APS copyrighted materials in electronic format. The CCC is the primary provider to academic publishers of copyright permissions services, and the APS has been working with this organization for many years for administration of print copyright permissions.

Editor budgets. Expense for running the Editors' offices remains at approximately $150 per manuscript. Almost all editors are in alignment, and some realize savings. The ability to make office funds "fungible" for other specific purposes was implemented as a 2-year trial as

of 2013. The guidelines were reviewed and revised by the Publications Committee at its March 2014 meeting and again at its 2015 meeting, when it was agreed:

- editors may use their expense budget to benefit the journal as they see fit
- EICs would include in their reports and presentation to the Publications Committee at their joint March meeting how the EIC and AE budgets were used to benefit their journal.

Online Journals Hosting New Initiatives

Article-level metrics. Launched in December 2015, usage data for each article (archive and current) are updated a on a monthly basis and presented on the "Info" tab on each article. Metrics shown are for abstract and full-text HTML views as well as PDF downloads, and include data for both the early view (AiPS) and final versions. This feature has been live since 2014 on *Physiological Reports,* presenting combined Wiley and HighWire usage data (since the journal is hosted on both platforms). The data are updated monthly on approximately the 15th of each month.

TOC annotations. Tables of contents carry a brief description of each research article. This feature was launched on *AJP-Heart* in July 2015 and went live on *JAPPL* in early 2016. It will be rolled out to other journals as desired throughout the year.

TOC thumbnail images. Tables of contents will carry thumbnail-sized images associated with each article. *Physiological Reviews* is the pilot journal before rolling out to all other journals in 2016. This feature appeared in Physiological Reports from launch.

Journal collections. APS has been hosting individual journal collections, and we now have a tool, Custom Collections Toolkit, enabling collection development across all journals. We see this tool as a precursor to the larger cross-journal taxonomy project we would like to develop with the editors in the next year or so. In general, cross-journal collections would provide increased discoverability of APS journal content by topic via search engines as well as browsing. To further enrich a collections initiative, categories and keywords can be identified within and across journals, including at submission and in the metadata. The Physiological Society has shared their taxonomy with us, which serves as a starting point.

Continuous Publication. The continuous publication interface enables publication of articles as they become available. This workflow was rolled out gradually and was completed for all journals with June 2016 issues.

Article versions information. Version information for articles has always been available in the Info tab, but the fact that one might be viewing an early version of a manuscript as opposed to the final version was not visible enough. We have added a box in the sidebar on the AiPS version of the article pages for version information. If the reader is not viewing the most recent version of an article, you can click on "View the most recent version of this article" from the sidebar.

Consolidated tables of contents. As requested by the Publications Committee, the APS Publications Department is trialing a consolidated table of contents (TOC) on the APS website (*http://www.the-aps.org/mm/Publications/Journals/Journal-TOCs*). This consolidated TOC is composed of the current issues all APS journal TOCs in a single PDF and is updated twice monthly. The PDF is keyword searchable. Usage of these consolidated TOCs can be tracked and reported via Google Analytics. The TOC webpage carries an opt-in RSS feed.

Editorial New Initiatives

Reviewer CME

Since 2013, APS has offered Continuing Medical Education (CME) credit to medical doctors who serve as manuscript reviewers and meet qualitative criteria that have been set by our editors. This was introduced in *AJP-Endo,* administered by The Endocrine Society. Since July 2015, CME has been offered to eligible reviewers for *AJP-Heart* and *JAPPL*, administered by Washington University School of Medicine CME Department. *AJP-Lung* and *AJP-Renal* have rolled out the program to their physician reviewers starting in early 2016. Details about Manuscript Review CME are at *http://www.the-aps.org/mm/Publications/Journals/Reviewer-Guidelines.html#CME*.

In 2015 CME Credit for manuscript reviewing was awarded to 25 reviewers for 27 papers in *AJP-Endo;* 34 reviewers for 41 papers in *AJP-Heart;* and 21 reviewers for 26 papers in *JAPPL*. Year to day May 31, 2016, CME Credits for manuscript reviewing to 9 reviewers for 15 papers in *AJP-Endo;* 25 reviewers for 45 papers in *AJP-Heart;* and 22 reviewers for 35 papers in *JAPPL*.

Conference Proceedings

At its fall 2014 meeting, the APS Publications Committee approved a plan to publish articles from APS-sponsored conferences at no cost to authors. As directed by APS Council, an appropriate method to account for potential revenues, costs, and losses within the APS budget is in place.

This initiative was proposed by Hershel Raff, then-APS Publications Chair, because of its potential for attracting new authors and readers, increasing submissions, establishing a durable record of conferences, and as an overall aid in to publicize conferences and raise enthusiasm.

The key components of this initiative are:
1) Selected participants are invited to submit short reviews or original research.

2) Submission length up to 4 printed pages, 2 figures (or 1 table and 1 figure), 1 in color.

3) Standard ethical/scientific considerations apply. Overall impact is not a criterion. Extensive revisions or additional experiments should not be requested.

4) Articles included in standard journal issue and appear in a separate online TOC.

5) Host EIC is in agreement with organizers on all details established for the conference articles.

6) No author charges apply; publication costs are supported by APS.

7) Applies to APS conferences in 2015-2016 (up to 2 per year; first-come, first-served); assessment at 2017 Spring Publications meeting.

This new conference proceedings program has been initiated by *AJP-Regu* for the Cardiovascular, Renal and Metabolic Diseases: Physiology and Gender Conference, Annapolis MD, November 17-20, 2015 (*http://www.the-aps.org/mm/Publications/Journals/AJP-Regu/Special-Calls/Sex-and-Gender.html*). Articles have been commissioned with an extended submission deadline of September 3, 2016. Submissions for a Call for Papers on the topic have also been invited as of January 1, 2016, with a submission deadline of June 30, 2016. An Editorial, jointly authored by the organizer, Jane Reckelhoff, and EIC Rick Samson has been published in the November 2015 issue of *AJP-Regu*. Furthermore, a dedicated webpage has been created to showcase these articles (see *http://ajpregu.physiology.org/gender*).

Advances in Physiology Education has commissioned nine articles from speakers from the Institute on Teaching and Learning Workshop held in Madison, WI, June 20-24.

AJP-Heart is commissioning articles from speakers from the Inflammation, Immunity and Cardiovascular Disease Conference, Westminster, CO, August 24-27.

Module on Best Practice in Peer Reviewing

The author survey conducted by the Publications Department in 2014 raised several important issues that the Publications Committee was keen to address. One of these issues was the creation of educational materials to support best practice for APS reviewers. To achieve this, a working group of Editor-in-Chief volunteers (Irv Zucker, Chair; Sadis Matalon, Rick Samson, and Peter Wagner) was established, which developed these materials. The materials represent the basic principles for peer reviewing. The goal established is for each EIC to host webinars for their Associate Editors and Editorial Boards on a regular basis.

The working group developed and the Publications Committee reviewed and edited the module. The PPT module is complete and has been distributed to the EICs for use with their editorial boards. This module, along with a version narrated by Irv Zucker (EIC, *AJP-Heart*) is available at *http://www.the-aps.org/mm/Publications/Journals/Reviewer-Guidelines.html.*

Recent Publications

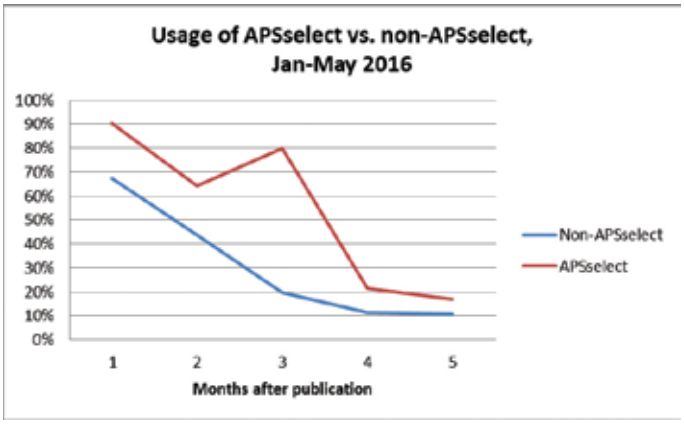
APSselect

APS*select* (*http://apsselect.physiology.org/*) launched January 2014. APS*select* highlights and promotes the top APS original research papers that are published each month across all 10 APS original research journals. APS*select* is edited by Joe Metzger (Editor-in-Chief) and Linda Samuelson (Associate Editor).

As of January 2016, a new group (Selection Board) has been invited to take the place of the Selection Committee. The new Selection Board members were recommended

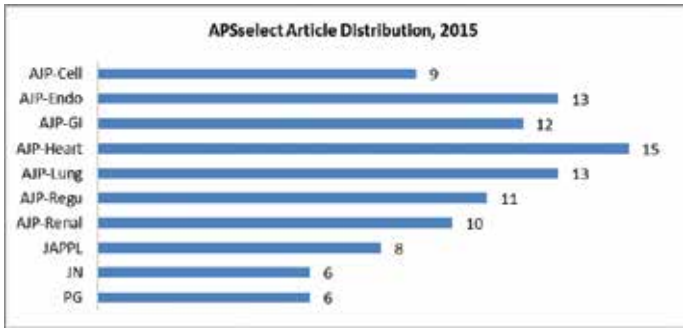
by the EICs and approved by the Publications Committee. The Selection Board is representative of disciplines within physiology and is a larger group, providing greater flexibility at voting.

Usage data continue to show that articles chosen for APS*select* experience a spike in usage on publication vs. non-APS*select* articles (see graph below).



The data have been normalized across all 10 research journals; values are given as a relative percentage of the maximum amount of usage that an average non-selected or selected article received during this 5-month period.

APS*select* articles in 2015 as represented across all APS original research journals. *AJP-GI* and *Lung* had the highest representation of APS*select* articles as a percentage of published articles (7.5%).



Comprehensive Physiology

Comprehensive Physiology (*www.comprehensivephysiology.com*), edited by David Pollock, is a digital update of the *Handbook of Physiology*. The first issue, published in January 2011, included all content of the most recent edition of the printed *Handbook* digitized and presented as "Classic Content," plus 25 new articles. *Comprehensive Physiology* is published by Wiley on behalf of the APS on a quarterly basis.

As of June 1, 2016, 841 articles have been invited, of which 401 have been accepted, 22 are in review or revision, and 370 have been published. The invited articles are from 11 of the 13 sections, covering 23 topics; "topics" correspond to a volume of the *Handbooks*.

The Journal has been accepted in all of the major abstract and indexing services including Scopus, PubMed, SciSearch®; Journal Citation Reports/Science Edition; BIOSIS Previews; BIOSIS Reviews Reports and Meetings; Thomson Reuters. The Journal was accepted into PubMed in 2012, and indexing began in July 2013. Its 2015 Impact Factor is 6.364.

Book Monograph Series

In June 2011, The APS and Springer signed an agreement for Springer to publish books in its monograph series on behalf of the APS. These series are *Perspectives in Physiology, Methods in Physiology, Physiology in Health and Disease.* As part of the Agreement, 33 backlist monograph titles have been digitized and are now freely available to APS Members (see *http://www.the-aps.org/mm/Publications/Books*). The books are hosted on the Springer website and can be downloaded as a PDF or viewed in the reader's browser. Fifteen books have been accepted into the program to date, of which seven have been published:

APS Book Chapter Downloads 2013–YTD 2016

Title	Author	2013 Total	2014 Total	2015 Total	2016 Total	Grand Total
Circadian Clocks: Role in Health and Disease	Gumz				2,183	2,183
Ion Channels and Transporters of Epithelia in Health and Disease	Hamilton & Devor			3,853	7,403	11,256
Sodium and Water Homeostasis	Hyndman & Pannabecker			1,636	1,441	3,077
Marine Physiology Down East: The Story of the Mt. Desert Island Biological Laboratory	Evans			910	471	1,381
Mechanism of Muscular Contraction	Rall		1,698	5,257	1,089	8,044
Essays on the History of Respiratory Physiology	West		60	9,340	2,703	12,103
The Rise of Fetal and Neonatal Physiology	Longo	4,451	8,319	5,286	2,243	20,299
ALL-TIME TOTALS		4,451	10,077	26,282	17,533	58,343

Physiological Reports

Physiological Reports is a general physiology open access journal owned jointly by the APS and The Physiological Society and published on their behalf by Wiley. The Journal is tasked to achieve a quick time to publication while upholding a quality standard of sound research that constitutes a useful contribution to the field. The Journal uses a "cascading peer review model," whereby manuscripts considered unsuitable for publication in an APS/TPS journal, yet deemed to be of publishable value, are "referred" to *Physiological Reports,* with author agreement to "transfer" the manuscript and reviewer agreement to transfer the manuscript reviews. Peer review for de novo submissions is comparable to that of APS journals, and manuscripts transferred to *Physiological Reports* should require no more than minor revisions, which the author must address.

Article types. The journal accepts only original research articles. In July 2015, the journal began accepting a new article type, Case Reports, which are clinical cases that must have a physiological focus (see *http://physreports.physiology.org/author-guidelines*). In 2016, the journal began accepting reviews.

Joint Managing Board. *Physiological Reports* is managed by a Joint Managing Board composed of representatives from both societies.

Editors. Susan Wray (University of Liverpool) is Editor-in-Chief, and Thomas Kleyman (University of Pittsburgh) is Deputy Editor-in-Chief.

Journal business model. *Physiological Reports* is funded by an author processing charge (APC) of $1,500 paid by the author upon acceptance. There are waivers in place for submissions from authors based in countries that cannot afford the fees (as there are for all APS journals). The first 100 manuscripts accepted in 2013 were free to the authors. The APC for Case Reports and Reviews is $1,000. Fees for Case Reports accepted for publication through December 2016 are waived.

Finances. The societies receive a profit share, split 50:50, for articles published via de novo submissions and split by actual percentage of articles published via transfer from each society. The journal was profitable in each of its first 2 full years (2014 and 2015) of publication.

Editorial status. *Physiological Reports* launched in March 2013. The first article appeared online in May 2013, and the first issue was compiled in June 2013 and subsequently compiled monthly. In 2015, 381 articles were published. The cascading peer review model accounts for ~81% of the total submissions, with ~93% of these articles accepted for publication. From January to May 2016, 144 articles were published, with the cascading peer review model accounting for ~96% of the total submissions, and ~93% of these articles accepted for publication.

In June 2015, the societies signed an agreement with The Scandinavian Physiological Society to include their journal *Acta Physiologica* as a supporter journal to *Physiological Reports,* from which suitable manuscripts are referred. In 2015, 14 (19%) of 75 manuscripts referred from *Acta Physiologica* were transferred by the authors. YTD May 2016, 17 (35%) of 48 manuscripts referred from Acta were transferred by the authors.

Module on Best Practice in Peer Reviewing

The author survey conducted by the Publications Department in 2014 raised several important issues, which the Publications Committee agreed to address. One of these issues was the creation of educational materials to support best practice for APS reviewers. To achieve this, a working group of EIC volunteers [Irv Zucker (Chair), Sadis Matalon, Rick Samson, and Peter Wagner] was established, which developed these materials. The materials represent the basic principles for peer reviewing. The goal established is for each EIC to host webinars for their associate editors and editorial boards on a regular basis.

The working group developed and the Publications Committee reviewed and edited the module. The PPT module is now complete and has been distributed to the EICs for use with their editorial boards. This module, along with a version narrated by Irv Zucker (EIC, *AJP-Heart*), is available at *http://www.the-aps.org/mm/Publications/Journals/Reviewer-Guidelines.html*.

2017 Journal Subscription Pricing

Journal subscription pricing for 2017 was presented to and accepted by the Publications Committee and Finance Committee at their March meetings and subsequently approved by Council at its April 2016 meeting. Increases in 2016 were 0% for tier 1, 3% for

tier 2, and 5% for tier 3. The journals program achieved significant savings in printing costs during the past year, so it was agreed that by implementing lower price increases in 2017, the savings will be passed on to our subscribers. Pricing was therefore unanimously agreed as follows:

- 1.0% for tier 1 (smallest organizations)
- 1.5% for tier 2 (larger organizations representing over 70% of the society's subscribers)
- 2.5% for tier 3

Marketing and Communications

The Communications Office posts daily on APS National Facebook and Twitter feeds. This includes a wide variety of journal-related content such as monthly APS*select* article announcements, press releases on APS*select* and Articles in PresS, news articles that mention research published in the APS journals, and content shared from journals that have their own social media feeds. We post 10-15 journal-related posts each month on Facebook and 20-25 on Twitter. Journal-related content remains some of the most popular on the APS National social feeds, with total views of this content in the tens of thousands.

The publications marketing main objective in 2016 has been to promote submissions for the journals. Promoting APS*select* articles continued to be a priority in 2016. Collaboration with Wiley's marketing representatives for *Physiological Reports* was also strengthened along with *Comprehensive Physiology*. More time was devoted to promoting the APS books program (managed by Springer). The MarComm Department approached these needs by increasing paid advertising while highlighting key journal features (e.g., 2016 price changes and IF increases), featuring journals at specific exhibit meetings, highlighting special calls and proceedings, while integrating more communications and social media aspects. The remainder of the year will focus on equipping the journals' editors and authors with more promotional marketing tools to enable them to further self-promote their journals and articles.

In previous years, the Communications report has focused on press release outreach for the journals. Since early 2014, increased effort has been placed on sharing social media content related to APS journals. YTD 2016, 17 of 28 press releases were related to journal content. The top five most popular releases in 2016 have addressed

- Detection and treatment of Sjögren's syndrome which causes dry eyes (*AJP-Cell*)
- The link between early life trauma and anxiety and digestive problems (*AJP-GI*)
- How estrogen may protect women against the flu (*AJP-Lung*)
- How chronic drinking affects pancreatic absorption of critical vitamins (*AJP-Cell*)
- How a small reduction in food can be enough to slow polycystic kidney disease (*AJP-Renal*)

Data Reproducibility

Since 2014, there has been intense discussion by NIH, NAS, FASEB, and other funders on the lack of experimental reproducibility in research. In response, NIH is making changes to graduate student education, grant preparation, and publishing guidelines in effort to promote better experimental design, analysis, and reporting (*https://www.nih.gov/research-training/rigor-reproducibility*). In 2015, FASEB hosted three roundtable discussions on experimental reproducibility, of which APS was an active participant. The first two discussions focused on the reproducibility of animal models and experiments using antibodies (Westerns, diagnostics, etc.). The third discussion synthesized the first two sessions and discussed ways to improve reporting. As a result of these meetings, FASEB prepared a set of recommendations to the scientific community published in January 2016 and available at *www.faseb.org/Portals/2/PDFs/opa/2016/FASEB_Enhancing%20Research%20Reproducibility.pdf*. In particular, FASEB would like societies to engage their journals to increase directives for reporting. In support, APS Council and Publications Committee want to consider whether there are ways our journals can help address the problems with experimental reproducibility and transparency.

In December 2015, Curt Sigmund arranged teleconferences with the EICs to introduce the three main issues to consider regarding enhancing data reproducibility and transparency in APS journals. The issues are:

- Enhance reporting of research methodologies (i.e., animal husbandry conditions, e.g., light dark cycle, temperature; reagent details, e.g., catalog and lot numbers; detailing protocol for each experiment, e.g., figure by figure, panel by panel)

- Enhance validation of antibodies and reagents and provide proof of validation (e.g., proof of antibody selectivity, proof of gene knockdown)
- Access to source data for *1)* reviewers of manuscript and/or *2)* readers of published articles: *1)* What is source data? *2)* What data are necessary to review or publish? *3)* Who would review the data?

The Editors were charged with presenting these issues to their associate editors and/or editorial boards and providing bulleted recommendations to the Publications Committee by January 29, 2016. The recommendations from these reports were discussed at the Spring EIC/Publications Committee meetings. Recommendations from the meetings are presented here. These recommendations will contribute to the development of a set of guidelines for all journals and a set of guidelines that are specific for particular protocols.

Summary of Publications Committee Discussions on Reproducibility Resulting From Editor Recommendations

Enhance author guidelines to encourage transparent reporting of methods. The changes will be consistent with requirements set by the NIH and will follow the lead of high-quality biological journals where guidelines are already established.

Start with *encouraging* authors to submit enhanced reporting for antibodies/reagents and use of animals in experiments. Over time, ramp up from *encouraging* toward *requiring*, as authors become familiar with expectations and the philosophy of enhanced reports gains acceptance.

Enhance reporting of animal experiments. Reinforce ARRIVE Guidelines and refer authors to the summary version *http://www.nc3rs.org.uk/sites/default/files/documents/Guidelines/NC3Rs%20ARRIVE%20Guidelines%202013.pdf*.

Enhance reporting of antibodies. Authors submit one representative full blot per antibody that generated the data for the paper, exclusive of loading controls; labeling nonspecific and specific bands as appropriate, labeling lanes, and exposure time.

Provide guidance on reporting immunohistochemistry data.

Develop a small cadre of statistics consultants with whom editors may consult for some papers during review process *(in development).*

Publish a "Cores of Reproducibility in Physiology Series" (CORPS) to describe and encourage best practice. *Update:* The series has been established initially in JAPPL and is being rolled out to all journals.

Publish an editorial on APS Journal Reproducibility Initiatives and market broadly.

Refer to CONSORT Guidelines for Clinical studies.

Information to be Added to the Publications Homepage/Instructions for Authors«

Promoting transparent reporting in APS publications to enhance data reproducibility. Concerns about the lack of rigorous reporting of, and the failure to reproduce, studies published in research articles have been the topic of discussion at NIH, FASEB, APS, and other science organizations. In particular, APS Council and the APS Publications Committee have considered how APS journals could facilitate better reporting of experiments reported in the APS Journals and, in turn, increase the likelihood that studies could be reproduced.

As of 2016, several guidelines in the APS Information for Authors have been updated, including the addition of a new section entitled "Experimental Details to Report in Your Manuscript" in effort to promote greater transparency in reporting relevant experimental information. As such, authors are encouraged to enhance in the reporting of the following details.

Animal experiments. Authors are encouraged to refer to the ARRIVE Guidelines Checklist in preparation for reporting the methods and results of animal studies. Particular attention should be given to providing detailed information regarding:

- The animals used in the study (species, strain, sex, age, source of animals, genetic modification status, housing, diet, etc.)
- The controls used in the study (littermate, purchased, identical conditions, contemporaneous, historical, etc.)

- Precise details of all experimental procedures (drug formulation and dose, anesthesia and analgesia used, method of euthanasia, etc.)
- Steps taken to minimize subjective bias in the study design (randomization, blinding, etc.)

Antibody validation. Authors are encouraged to submit one representative full blot per antibody that generated the data for the paper, exclusive of loading controls. Lanes on the blot should be labeled to note the nonspecific and specific bands, and exposure time should be indicated. These materials will be assessed during review and will be published online as "Supporting Data."

Authors are encouraged to describe how antibodies were validated for specificity. Authors can reference prior publications or show positive or negative controls on the blot, as noted above, which is submitted as "Supporting Data."

Experimental details in figures and legends. Information reported in figures and legends should describe each individual experiment presented, including the number of samples or animals used per treatment. The statistics performed for each experiment should be reported as well. If concerns regarding the statistical analyses reported in the manuscript are raised during peer review, the associate editor may consult a statistician for guidance.

Authors are encouraged to present gels or blots in figures with the following:
- a molecular-weight size marker
- space above and below the band of interest from the original image

Authors are encouraged to provide detailed legends to all figures including:
- specific *n* value for each treatment group in each experiment
- description of statistics used to analyze each experiment

Council accepted the report of the Publications Committee. ●

Science Policy Committee

Kevin Kregal, Chair

SPC Meetings

The SPC held its ninth face-to-face meeting in Bethesda, MD on September 20-21, 2015. Discussion focused on the role of committee members in initiating advocacy activities and what the committee can do to further the Society's efforts to address problems with reproducibility. A subcommittee was formed to draft a list of potential activities that committee members could undertake. This action item was completed and presented to the committee at the EB 2016 meeting San Diego, CA. At the EB meeting, committee members continued discussion of advocacy activities and reproducibility.

SPC Advocacy Activities

Fall Committee Meeting Hill Visits

When the SPC met in Bethesda last September, there were 20 members in attendance, including current and incoming committee members, early career advocacy fellows, a representative from Council, and former committee members. With the help of APS and FASEB staff, committee members met with staff in 27 Congressional offices to discuss sustainable funding for research; pending legislation to reauthorize the NIH and NSF; and legislative language that would relax restrictions on travel for federal employees, including scientists.

APS Leadership Visits Capitol Hill With FASEB

On March 3, 2016, I joined two other APS members to participate in FASEB's annual Capitol Hill Day. Representing APS were FASEB Board Member Hannah Carey and FASEB Past-President J. R. Haywood. Charles Wood also attended as President-elect of the ACDP.

CNSF Exhibition

APS and FASEB co-sponsored an exhibit at an April 27, 2016 Coalition for National Science Funding event on Capitol Hill. APS member and Early Career Advocacy Fellow Allyson Hindle presented her research on naturally occurring protection for the hearts and brains of diving seals in Antarctica to an audience that included Members of Congress and Congressional staff. The exhibit was meant to highlight the value of NSF-funded research and to make the case for future budget increases. APS representative to the FASEB Board Hannah Carey also attended the event.

APS Early Career Advocacy Fellows

We received six eligible applications for the November 2015 deadline for the APS Early Career Advocacy Fellowship (ECAF). Each application was reviewed by members of the committee, and a final decision was made on a conference call. The committee selected two awardees: LaShauna Evans (Postdoctoral Fellow at the University of Texas Health Science Center at San Antonio) and Heidi Medford (Postdoctoral Fellow at Washington State University). After the APS Council unanimously approved the committee's selection, Evans and Medford joined the two second-year fellows, Allyson Hindle and Giovanna Collu. The fellows were invited to attend the committee's meeting at Experimental Biology in San Diego. An ECAF orientation luncheon was also held at EB, where they had the opportunity to interact with members of the committee, Society leadership, past Fellows, and staff.

The SPC committee will forgo visiting Capitol Hill this fall since Congress will be out of session for the election. Therefore, a special Hill Day was held on May 16-17, 2016 to provide the ECAF with an opportunity to meet with Members of Congress. The Fellows, accompanied by myself, incoming SPC Chair Laura McCabe, and the APS Science Policy team, visited the offices of their Senators and Representatives to discuss the importance of federal funding for research and to raise concerns about the pending reauthorization for the Small Business Innovation Research (SBIR) program. The issue of SBIR reauthorization came up just before the fellows visited Capitol Hill, so we took the opportunity to convey our concerns about the potential negative impact of increasing the SBIR set aside.

Chapter Advocacy Partnerships

APS will sponsor three Advocacy Outreach speakers at APS Chapter meetings in 2016 under this program that was established in 2013 to offer advocacy training outside of the EB meeting. Council allocated $6,000 per year to cover the travel expenses of three such speakers. An overview of the program and instructions on requesting a speaker is provided annually to the Chapters. Their requests are honored on a first-come, first-served basis, with preference given to Chapters that have not recently had a speaker. These are the talks scheduled for 2016:

- "Research Advocacy: Difficult Topics-Animals in Research" by Alicia Schiller; Indiana Physiological Society
- "Animal Data Reproducibility" by Gaylen Edwards; Nebraska Physiological Society
- "Advocacy for Science: Making a Compelling and Understandable Case" by Kevin Kregel; Iowa Physiological Society

Leadership Interactions With FASEB

The APS is currently represented on the FASEB Board of Directors by Hannah Carey, who will begin service as Vice President-elect for Science Policy of FASEB in July. APS will need to identify a new Board Representative when Hannah begins serving as Vice-President for Science Policy in July of 2017. Kevin C. Kregel serves as the APS representative to the FASEB Science Policy Committee (SPC), and he will continue his service as the Chair of FASEB's Animals in Research and Education Committee. J.R. Haywood Served as FASEB Past-President in 2015-2016.

Hannah and J.R. both attended the FASEB Board meeting in June, which focused on the future of FASEB. The FASEB SPC meeting has been rescheduled for August 11, and the symposium topic will be "Evaluating Research and Researchers."

The APS is represented on various FASEB SPC subcommittees by the following individuals: Hannah Carey, John Chatham, Gaylen Edwards, J. R. Haywood, Bill Talman, Tim Musch, Bill Yates, Virginia Miller, Carrie Northcott, Gina Yosten, Tom Pressley, Chris Westby, TanYa Gwathmey-Williams, Amy Davidoff, Brooks Robey, and Phil Clifford.

EB 2017 Public Affairs Symposium

The SPC is planning the Public Affairs Symposium for EB 2017 in Chicago. The committee selected the topic of reproducibility in research with a focus on what researchers need to do to comply with new requirements for grant applications and publications. Committee members Roy Sutliff and Alicia Schiller are chairing the session, entitled "Why Scientific Rigor Matters and How to Improve It."

Council accepted the report of the Science Policy Committee. ●

Section Advisory Committee


Wolfgang Kuebler, Chair

On an annual basis, the Section Advisory Committee (SAC) undergoes changes in membership. In the past year, Jennifer Pollock (WEH), Martha O'Donnell (CAMPS), and Merry Lindsey (CVS) completed their service as section chairs. We recognize and thank Jennifer, Martha, and Lindsey for their leadership and dedication to the APS. Concomitantly, we welcome Carmen Hinojosa-Laborde, Ken Gagnon, and William Jackson as new section chairs for WEH, CAMPS, and CVS to SAC.

In November 2015, SAC and Council held their second Joint Meeting outside the regular EB meetings in New Orleans, LA. At the Joint Meeting, council and SAC discussed the Society's Strategic Plan, Opeational Guide Write-ups related to Sections, Guidelines and Procedures for future EB programming, the role of SAC in COC operations and in governance of PAS, and new initiatives including the Reprograming/Rebranding Initiative and the establishment of a Leadership Academy. SAC members welcomed the opportunity for a concerted exchange with Council and strongly support the continuation of the Joint Fall meeting.

SAC has discussed extensively about ways to increase the attractiveness and diversity of our scientific program at Experimental Biology and to leverage on the unique advantages of a more generalistic compared with an organ-specific scientific society. A task force consisting of the Chairs of the Joint Programming Committee, Robert Hester, the Chair of SAC, Wolfgang Kuebler, and SAC members and Section chairs Donald Kohan and Sean Stocker was established to discuss future options for EB programming. At the 2014 Fall Meeting, SAC was scheduled to review session allocations for the 2017 EB meeting. In the previous SAC evaluations, the number of session allocations for each section was determined based on a relative average of the number of abstracts submitted to each section from the previous three EB meetings. SAC members shared considerable concerns about the current evaluation system. SAC therefore decided to keep session allocations unchanged for the time being and to work with Council to develop alternative metrics.

Most sections continue to see a continuous increase in applications for their awards. In response, many sections have increased their number of awards, as well as award endowments to adjust for increased travel and housing expenses, and several sections have established APS Endowed Matching Funds to sustain the funding of existing awards or increase the number of awards for established programs.

Finances remain an important concern, and, for some sections, expenses have exceeded income in the past years. Major cost factors remain the social events/banquets at the EB meeting and the support for trainee activities.

Council accepted the report of the Section Advisory Committee. ●

Trainee Advisory Committee


Erica Dale, Chair

TAC Trainee Survey

The Trainee Advisory Committee (TAC) began planning for next year's survey and has decided to follow the lead of Council and the strategic planning process to incorporate the goals and interests of the Society and how they may best serve the trainee community. The survey is not limited to APS members but casts a broader net to solicit information from many trainees who are involved in APS activities and meetings. Since there has been a change in timeline for APS strategic planning, the TAC has put the finalization of the survey on hold to coordinate with Council planning activities.

EB Symposia

The EB2016 TAC Symposium was entitled "Now Hiring Ph.D.s: Post Doc Not Required" and was organized by Angelina Hernandez-Carretero and Brendan Dougherty. The presentations focused on exposing trainees to the diverse career paths available in today's job market for those with PhDs. Those presentations were then followed by a panel discussion with the invited speakers (Philip Clifford, Sonia Zarate, Jeremiah Mitzelfelt, Carrie Wolinetz, Maria Urso, and Andrea Gwosdow). Attendance at this morning session was impressive and peaked at over 200 people. The speakers also received very high ratings from the attendees.

The EB2016 Trainee Cross-Sectional Featured Topic was entitled "Sex Differences in Health and Disease" and encouraged submissions from all sections. The session was well attended and gave trainees a chance to showcase their work.

The EB2017 TAC Symposium is entitled "Kick Start Your Funding: Looking Beyond NIH and NSF" and is being organized Jennifer Steiner and Kristi Streeter. The goal of this symposium is to help trainees and early career scientists identify additional funding opportunities to support their research as well as provide them with information on how to apply and write successful applications. There will be a speaker from the following four categories: *1)* private foundation, *2)* military, *3)* crowd sourcing/kickstarter, and *4)* industry.

Career Webinar Series

The Career Webinar subcommittee (Brendan Dougherty and Christopher Banek) is working closely with Aaron James Polichnowski from the Porter Fellowship committee and Seth Fairfax of COPC to move the project forward. They have identified Rudy Ortiz at UC-Merced as the first webinar participant. We anticipate launching this webinar series in 2016 and are exploring options for creating a permanent home for the webinar series on the APS website.

APS Trainee Facebook Site and Twitter

The APS Trainee Facebook page currently has 1,984 Facebook "Likes" (a 19% increase from 2015), and the Twitter account has 192 followers (a 22% increase from 2015). Postings come from staff, TAC members, and APS Twitter feeds, providing regular communication to the trainee fans from the APS and the TAC. At the fall meeting, TAC establishes monthly topics relevant to trainees and assigns members responsible for posting materials.

Dale J. Benos Early Career Professional Service Award

The TAC received seven completed applications for the 2016 award. The Committee was extremely pleased to select Keisa Mathis, Assistant Professor at the University of North Texas Health Science Center (UNTHSC). Mathis showed a remarkable level of professional service, outreach, and mentoring/teaching activities with students at the graduate/professional, undergraduate, and K-12 levels, as well as with members of her community. She had been nominated for or won awards for both her mentoring and research throughout her career to date. The Council approved this selection, and Mathis received the award at EB 2016. She will also prepare an article for a future issue of *The Physiologist*.

The TAC has begun to follow up with past award winners to determine whether they have continued their service activities and to ask about the impacts of receiving the award on their careers and professional activities. The first recipient of this award was surveyed at the end of 2013, and the fourth recipient will be surveyed this year.

TAC Outreach

TAC members continue to use social media tools (Facebook and Twitter) along with the APS website and newsletter to engage both undergraduate students and physiology trainees. TAC business cards were distributed again at EB 2016 to increase awareness of these social media websites. TAC members attended the EB 2016 Undergraduate Poster Session and engaged many of the undergraduate students in discussions of their research, encouraged them to become APS members, and promoted the APS social media sites (Facebook, etc.). The TAC also collaborated with the Career Opportunities in Physiology Committee on the 2015 EB Undergraduate Orientation Session, with Angelina Hernandez-Carretero presenting a portion of the orientation talk.

Undergraduate Research Advisory Board

To gain a broader perspective of the involvement of undergraduates in APS, the TAC sent a survey to all undergraduates that submitted an abstract to a physiology topic at the EB 2016 meeting. As an incentive to increase participation, the undergraduates were given the opportunity to receive a certificate recognizing them as members of the Undergraduate Research Advisory Board. Almost half of the undergraduate students presenting at EB2016 responded to the survey. Results will be discussed by TAC at the fall meeting and presented to Council. A manuscript has been prepared on the first 2 years of results and will be submitted soon for publication.

APS Graduate Student Ambassador Program

The GSA subcommittee (Kristi Streeter, Jennifer Steiner, and Steven Copp) is working with APS staff to finalize the GSA application and launch the website. Suggestions from Council for including appropriate metrics for success were included in the revisions for this new program. The goal is to start advertising for applications at the end of the summer/early fall of 2016. The subcommittee will also be working this summer to develop an online training webinar.

Communication With Sections

TAC representatives updated their section activity information in 2015-2016 and shared information with their Section Steering Committee. The most common trainee activities for Sections were research awards for trainees and junior faculty, discounted banquet tickets for trainees, involvement of junior faculty as symposia speakers, and e-media use. About two-thirds of the Sections have a trainee section in their newsletter and have trainees co-chair symposia or featured topics at EB, and some sections also include trainee-driven sessions at EB, trainee subcommittees within the section, and awards for undergraduate students.

Communication With APS Committee Trainee Members

Council has approved funding for a breakfast meeting at EB for all trainees who are members of the APS standing committees and APS Chapter Steering Committees. The idea of this breakfast is to maintain contact with these trainees to understand their needs and concerns in regard to committee service. Attendance at these meetings has steadily climbed each year. In addition, the TAC has begun to develop a survey to be distributed to trainees on APS committees, which will be sent this summer.

Council accepted the report of the Trainee Advisory Committee. ●

Women in Physiology Committee


Caroline Rickards, Chair

Strategic Planning Meeting

The Women in Physiology (WIP) Committee and invited guests met at APS headquarters from May 2 to 3, 2016 for a Strategic Planning Meeting. In accordance with our request for funding to Council in July 2015, the primary objective of this meeting was to develop a 5-year strategic plan for the WIP Committee to:

- Increase gender equality among APS membership
- Increase recruitment and retention of female students in physiology
- Promote the success of women physiologists across career stages

Nine WIP Committee members, two invited guests, and four APS staff were in attendance. We had an exceptionally productive meeting where we were able to thoroughly review our current charge and committee activities, and explore potential new initiatives for the advancement of women within the Society and the wider scientific community, including development of options for new mentoring programs within the Society. The final report from this meeting will be submitted to Council for discussion at their fall meeting.

Bodil Schmidt-Nielsen Distinguished Mentor And Scientist Award

Four excellent nominations were received for the 2016 Bodil Schmidt-Nielsen Distinguished Mentor and Scientist Award. The WIP Committee selected Ormand MacDougald (University of Michigan) as the awardee. MacDougald gave an excellent talk, providing an overview of his best tips on mentoring that he has developed over his prestigious career. MacDougald will develop a Mentoring Forum column based on his lecture, to be published in The Physiologist over the next year; the multimedia presentation (slides and audio) will also be posted on the APS website. The lecture was followed by an afternoon tea reception, attended by APS Council members, former Schmidt-Nielsen Awardees, MacDougald's nominators, colleagues, and mentees, awardees of the various APS award programs (tum Suden, Minority Travel Fellows, Porter Fellows, etc.), other trainees, and guests specified by the awardee.

APS Professional Opportunity Awards: Caroline Tum Suden/Frances Hellebrandt, Steven M. Horvath, and Fleur L. Strand Awards

The WIP Committee received 117 initial applications for the 2016 tum Suden Professional Opportunity Awards, which is slightly lower than the number of applications in 2015 (125). The Committee was able to fund 50 tum Suden Awards (up from 46 previously), 2 Horvath Awards (awarded to the top two URM applicants), and 1 Strand Award (awarded to the top overall applicant) for an overall funding rate of 45% (53 awards in total).

In an effort to promote the namesakes of each of these awards (tum Suden, Hellebrandt, Horvath, and Strand), short biosketches were developed by the WIP Committee providing background on the origin of the awards and their funding sources. These biosketches have been published on the APS website and were printed on cards that were distributed to the awardees with their award certificates at the APS Business Meeting.

Mentoring Programs

Mentoring Forum

Over the past 12 months, the WIP Committee has coordinated the development of six new Mentoring Forum columns published in *The Physiologist*. These columns cover a wide range of topics, from how to respond to inappropriate behavior, to mentoring for collaborations, making the transition from teaching in the lab to the classroom, switching from applied to basic research, and time management. Each article has related bulletin board discussion topics at the mentoring website (*www.the-aps.org/mentoringforum*).

Online Mentoring Programs

The WIP Committee continues to provide information about MentorNet on the APS website, and the APS is also a Scientific & Professional Society partner with the National Research Mentoring Network (NRMN) (*https://nrmnet.net/*), which provides comprehensive mentoring services for APS members, including a "Virtual Mentoring Program," similar to the MentorNet approach. A variety of mentoring resources are also available on the APS website under the "Career Development & Mentoring" link. During the recent Strategic Planning Meeting, the WIP Committee explored a number of innovative options that would provide much needed mentoring experiences across career stages, without incurring the logistical and fiscal expenses of establishing and maintaining a formal mentoring program, such as MentorNet, or a similar APS-specific program. These program ideas will be detailed in the final strategic planning meeting report.

WIPC Facebook Page

The WIP Committee has a very active Facebook page, with 633 "likes" and many posts reaching more than 300 views. The specific goals of this page are to share information that is relevant to the mission of the WIP Committee with both men and women, including content on gender issues in science, promotion of physiology to early career scientists, and mentoring. The page can be found at *www.facebook.com/APS.WIPC*.

Experimental Biology Mentoring Workshop

For EB 2016, the workshop was entitled "Negotiating for Success." The workshop was held on Monday from 3:15 to 5:15 PM and was attended by approximately 150 participants! Twenty-five attendees (~17%) completed a survey, and, based on these responses, the audience was primarily made up of trainees (undergraduate students, graduate students, postdoctoral fellows); however, there was a wide range of audience members, including junior and established investigators. The speaker presentations were rated highly, and there were several questions and issues raised during the discussion period (30 minutes after the presentations) as well as for several minutes following the end of the symposium.

Representation Of Women in APS and Scientific Community Leadership

One of the charges of the WIP Committee is to support advancement of women in APS and in the scientific community at large. The WIP Committee annually reviews the number of women serving on APS Committees and Section Steering Committees. Of the members who include gender in their membership profile (n = 9,106), women currently comprise 29% of the APS membership [27% of the regular membership (n = 6,822) and 49% of the graduate/undergraduate student membership (n = 1,406)]. Most of these numbers either remain the same or have increased slightly from last year.

In reviewing the membership of the APS Section Advisory Committee (SAC) and other Society committees, we found that the representation of women on the general Society committees continues to be very good. The WIPC commends the Committee on Committees for its ongoing attention to gender diversity on APS-appointed committee positions. The Committee is also pleased to note that in 2016-2017, the APS Council has four women out of nine elected members (44%) as well as women in both the President and Past-President positions. Thus, overall, 6 of the 12 (50%) elected members of Council are women. The Committee contends that this continues to reflect the increasing role of women in the Society as a whole and especially in Committee leadership positions, which provide important visibility and service opportunities. The SAC, however, has only 2 women representatives out of 13 (15%), down from 3 in 2015, and 6 in 2014; this underscores the importance of encouraging all Sections to involve women in their leadership positions, particularly as Section chairs.

In reviewing the Section Steering Committees, on average, we found about the same proportion of women as last year (38% vs. 37%). Currently, all but three Sections (CNS, EEP, Respiration) have Steering Committees where at least 25% of the members are women, and 7 of the 12 Sections have Steering Committees where at least 40% of the members are women. Similarly, 15 of 22 APS Committees are comprised of at least 25% women members; these numbers are similar to 2015. The Committee encourages the Committee on Committees and the

Section Steering Committees to continue their efforts to identify and select women members for APS Committees.

Although historically there have been very few women selected for the major APS awards, the WIPC is pleased to report that 3 of the 14 (21%) Distinguished Lectures at EB 2016 were given by women. Although this is down from 6 of 14 Lectureships in 2015, these numbers do closely represent the proportion of eligible women members in the APS (27% of regular membership). The Committee continues to encourage Sections to include women on their slate of candidates for their Distinguished Lectureships in the future.

Concluding Remarks

The WIP Committee continues to work to promote women within the Society and the scientific community and to provide mentoring for early career investigators and trainees. We look forward to additional involvement in new APS programs and activities, and we will strive to remain instrumental in the implementation of the New Strategic Plan developed by Council.

Council accepted the report of the Women in Physiology Committee. ●

APS News

Physiology Quiz Show

Susan M. Barman
Michigan State University, East Lansing, Michigan

On July 29-30, 2016, about 350 students and 100 faculty members from 81 medical schools representing 23 countries gathered at the Universitas Gadjah Mada (UGM) in Yogyakarta, Indonesia to participate in the 14th Annual Inter-Medical School Physiology Quiz (IMSPQ). IMSPQ is a science Olympiad in which enthusiastic medical students compete for the winning trophy given to the team that shows the best proficiency in physiology based on their performance in a written test and a fast-paced oral quiz. Medical schools across the globe are eligible to send a team of 3-5 students and faculty mentors to participate in the IMSPQ. This year's participants included medical students from Australia, Bangladesh, Bosnia and Herzegovina, Cambodia, China, Egypt, Hong Kong, India, Indonesia, Iran, Japan, Laos, Malaysia, Mongolia, Myanmar, Nepal, Singapore, Sri Lanka, Sudan, Taiwan, Thailand, The Philippines, and Vietnam.

The IMSPQ was the brainchild of Hwee-Ming Cheng, Professor of Physiology of the Faculty of Medicine at the University of Malaya in Kuala Lumpur Malaysia. In 2003, he organized a small group of competitors from several Malaysian medical schools in an effort to encourage medical students to get excited about learning physiology. This year's event was the first time that IMSPQ has been held at a location other than the University of Malaya. The event ran flawlessly under the very capable leadership of Denny Agustiningsih and Dicky Moch Rizal, both Professors of Physiology at the Faculty of Medicine of UGM.

Nearly 350 students from 81 medical schools across the globe took a written exam on *Day 1* of the 14th Annual Inter-Medical School Physiology Quiz (IMSPQ) held on July 29-30, 2016 at the Medical Faculty of Universitas Gadjah Mada in Yogyakarta, Indonesia

After months of intense preparation to strengthen their knowledge of cellular and systems physiology, all of the medical students took a 2-hour written exam composed of 100 true/false questions on the first morning of the IMSPQ. The composite score of each team was used to determine the 40 teams that qualified for the oral quiz on the second day. While the students were gathered in the exam hall anxiously answering the true/false questions, the faculty mentors that accompanied them to UGM participated in a neurophysiology refresher course. I was honored to be the invited speaker for this event. My presentation entitled "Integrative Neurophysiology: A Link Between the Brain, Sympathetic Nerves, and the Cardiovascular System" was well received by the participants. The intent of this refresher course was to provide the faculty with information that they can use to teach undergraduate medical students and graduate students in their home institutions. I emphasized fundamental features of the autonomic nervous system that are often missed in physiology and neuroscience textbooks (6). My presentation was followed by a hands-on workshop led by ADInstruments, in which faculty participants could test devices that can be used to enhance their teaching of physiology.

Day 2 was definitely the highlight of the IMSPQ. This is when three students from each of the 40 top teams (based on the written quiz) took the stage to answer questions in a rapid-paced oral quiz with Cheng serving as a talented Quiz Master. I had the privilege of serving as one of two judges for the oral quiz. Luckily, the judges are provided with the ideal answers for each question. In the event that the Quiz Master was not certain that the answer given by the student was correct, he turned to the judges for advice. We had the opportunity to ask for clarification and had the daunting task of declaring whether the answer was right or wrong.

The oral quiz occurred over an 8-hour period in a room filled with nervous students and faculty, hoping their team would score the most points in each round. For Round 1, the top 40 teams were organized into eight groups of five teams each. Five teams at a time took the stage, and each student was given a question to answer within 15 seconds or risk having a student on the opposing team answer the question and thus accumulate more points. By Round 2, only 12 teams remained; then the Semifinal Round whittled the top six teams down to three. The Final Round determined the first-, second-, and third-place finishers.

For the second year in a row, the first place prize went to the team from UGM, this year's hosting institution. They were nearly perfect in the final round! Second and third place went to the teams from Mahidol University Siriaj Hospital (Thailand) and De LaSalle University Health Sciences Institute (The Philippines). Although only one team can reach the top, all of the student participants were winners since they had competed at their home institutions to gain the opportunity to come to the 14th annual IMSPQ and interact with students and faculty from across the globe. These future medical doctors realized the importance that physiology has in their careers, and they were willing to spend long hours learning important aspects of medical physiology.

Another highlight of the event was the cultural evening on the first day. Medical students competing in the IMSPQ proved their talents extend beyond their competency in physiology. On this evening, they danced, sang, and performed artistic maneuvers to delight the audience as we learned about their national and regional identities.

To learn more about the types of questions and format of the IMSPQ, I encourage you to read several articles by the Quiz Master Cheng and published in *Advances in Physiology* (1, 3, 5). Also, Cheng has published a book that includes a collection of IMSPQ-related questions (2). As described by Cheng (3), many of the faculty who have brought a team of medical students to participate in the IMSPQ in Malaysia have been inspired by the dynamics of the program and have developed a similar quiz in their home country. Kim Barrett was the invited speaker for the GI refresher course in 2015 (4). She and I were both so impressed by the enthusiasm to learn physiology expressed by the medical students that we are now in the process of developing a plan to work with the APS Chapters program to hold a physiology quiz in the U.S. We are hopeful that the APS can send a team to the next IMSPQ that again will be held in Malaysia. Walter Boron will be the instructor for the physiology refresher course held at the host institution, University of Malaya.

Medical students from the host institution, Universitas Gadjah Mada, won first place at the IMSPQ 2016. Team members were (*left* to *right*) Aditio Artyasa, Alfin Harjuno, Fitrawan Silvano, Maria Patricia, and Yolanda Kadir; Dr. Hwee-Ming Cheng, Quiz Master, is seen at *right*.

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Education

APS STEP UP Fellows Attend 2016 Summer Research Symposium

APS was pleased to be a host of the 2016 APS Short-Term Education Program for Underrepresented Persons (STEP-UP) Summer Research Symposium from July 31 to August 4 at the National Institutes of Health (NIH) in Bethesda, MD.

Sixty Fellows from three coordinating centers (APS, Children's Hospital-Los Angeles, and Pennsylvania State University-Hershey) participated in the symposium. Each fellow gave an oral and poster presentation on their summer research project, and attended career roundtable discussions and networking and professional development sessions. Dr. Sharon Milgram, Director, NIH Office of Intramural Training & Education, gave a talk on "It's Your Life: Design It Wisely." The symposium ended with a celebratory awards banquet.

2016 APS STEP UP Fellows at the Summer Research Symposium

Of the 60 fellows in attendance, 25 were in the APS cohort, and 15 were working in labs with APS Members (see Table 1).

During the program, each Fellow participated in hands-on research experience in the lab of an established investigator to develop a hypothesis, design and troubleshoot experiments, collect and analyze data, and write and present results. They networked with other undergraduates interested in biomedical research, explored the nature of research and how scientists think about their specific question, explored career options and what it takes to be successful in those careers, and had their career questions answered by members of the grant Advisory Board and the Porter Physiology Development and Minority Affairs Committee.

Table 1. 2016 APS STEP-UP Fellows and Research Hosts

Student/Student Institution	Host/Host Institution
Nana Acquah University of Pennsylvania	**Grace Zhang, PhD** George Washington University
Pravleen Bains Illinois State University	**Peter Aronson, PhD*** Yale University
Benchaa Boualam University of Maryland, College Park	**Grace Zhang, PhD** George Washington University
Rosemary Carter Rochester Institute of Technology	**Jason Kim, PhD*** University of Massachusetts Medical School
Efe Disi University of Kentucky	**Jeffrey Osborn, PhD*** University of Kentucky
Elizabeth Escobar University of Notre Dame	**Xiaonan Wang, PhD*** Emory University School of Medicine
Ibrahim Finley Jackson State University	**Frank A. Anania, MD*** Emory University School of Medicine

Laura Galarza-Paez Emory University	**Douglas Eaton, PhD*** Emory University
Juan Infante Harvard University	**Brian R. Noga, PhD*** University of Miami School of Medicine
Daenique Jengelley Tuskegee University	**Carmella Evans-Molina, MD, PhD** Indiana University
Maria Andrea Jurado Columbia University	**Benjamin Fogelgren, PhD*** University of Hawaii
Zulikayida Maimaiti Massachusetts Institute of Technology	**Solange P. Brown, MD, PhD** Johns Hopkins University School of Medicine
Marda Mengesha University of Maryland, College Park	**Rebecca Brown, MD** National Institute of Diabetes & Digestive & Kidney Diseases
Clayton Moralez University of Arizona	**Heddwen L. Brooks, PhD*** University of Arizona
Prisca Obi Harvard College	**Gordon Freeman, PhD** Dana-Farber Cancer Center
Alexander Pabón University of Puerto Rico in Bayamón	**Jorge Duconge , PhD** University of Puerto Rico, Medical Sciences Campus
Cayra Ramirez Santiago University of Puerto Rico, Río Piedras Campus	**Mark W. Miller, PhD*** University of Puerto Rico School of Medicine
Mariela Rosa University of Puerto Rico, Río Piedras Campus	**Polina Lishko, PhD** University of California, Berkeley
Maia Schumacher Seattle University	**Wendy Thomas, PhD*** University of Washington
Whitney Sheets West Virginia University	**Paul Chantler, PhD*** West Virginia University
Fuad Sirmolo University of Maryland at Baltimore	**Sheng Wu, PhD*** Johns Hopkins University School of Medicine
Lean Teodoro Northern Marianas College	**Alan Hargens, PhD*** University of California, San Diego
William Wey Vanderbilt University	**Alan Hargens, PhD*** University of California, San Diego
Jasmine Williams Emory University	**Clintoria Richards Williams, PhD*** Emory University
Alexander Young Howard University	**Rasheed Gbadegesin, MD** Duke University

*APS Member

This program, funded by the National Institute of Digestive and Diabetes and Kidney Diseases (NIDDK; 1 R25 DK-95492-01), provides hands-on research experiences for underrepresented undergraduate students interested in exploring research careers. It also aims to provide exposure to the core NIDDK mission areas of diabetes, endocrinology, and metabolic diseases; digestive diseases and nutrition; kidney, urological, and hematological diseases. For more information, visit *www.the-aps.org/stepup*. ●

Education Office Awards for Experimental Biology 2017 (April 22-26 in Chicago)

To apply for any of these awards, go to the-aps.org/awardapps.

Novel Disease Model Awards for Pre-doctoral Students and Postdoctoral Fellows

Application deadline: November 17, 2016 (coincides with EB abstract deadline)
the-aps.org/mm/awards/Other-APS-Awards

This award recognizes a postdoctoral trainee ($800 award) and a pre-doctoral student ($500 award) who submit the best abstracts describing a disease model at EB. Although the model may be cellular or in vivo, the applicant should clearly emphasize the novelty of the model and the potential utility of the system for future research related to a disease process. The applicant does not have to be an APS member, and there are no restrictions on how the award is spent; however, the sponsor must be an APS member in good standing at the time of application. For the pre-doctoral award, the applicant must be a U.S. graduate student or equivalent, that is, they must have earned a bachelor's degree and be enrolled in subsequent training. Previous awardees are not eligible to apply at the same education level. Use the link above, then select either "Graduate or Medical Student" or "Postdoctoral Fellow or Medical Resident" for more details.

Caroline tum Suden/Frances A. Hellebrandt Professional Opportunity Awards

Steven M. Horvath Professional Opportunity Awards

Fleur L. Strand Professional Opportunity Award

Application deadline: November 17, 2016 (coincides with EB abstract deadline)
the-aps.org/mm/awards/Other-APS-Awards

To be considered for these awards, the applicant must be either a graduate student or a postdoctoral fellow and the first author of an abstract submitted to APS, and must be a member of APS in good standing at the time of application (either Student or Regular). To receive the award, recipients must attend EB, present a poster at the meeting, and attend the APS Business Meeting. The top applicant will be designated as the Strand Awardee and will receive $1,000 plus complimentary EB registration. The top two underrepresented racial/ethnic minority recipients will be designated as Steven M. Horvath Awardees. The tum Suden/Hellebrandt and Horvath awardees receive $500 and complimentary EB registration. Awardees will only be allowed to receive a tum Suden, Strand, or Horvath Award once as a pre-doctoral student and once as a postdoctoral trainee.

Excellence in Professional Student (MD or DO) Research Travel Awards

Application deadline: November 28, 2016
the-aps.org/md-do

The Excellence in Professional Student Research Travel Award provides funds for up to 10 MD or DO students who are first authors on an abstract submitted to the EB meeting to attend, present their research, and participate fully in the EB meeting. To be considered for these awards, the applicant must be working with an APS member. The applicant does not need to be an APS member at the time of application. U.S. residency is NOT required. See the website for more details and apply online at *the-aps.org/awardapps*.

APS Minority Travel Fellowship Awards

Application deadline: November 28, 2016
the-aps.org/minoritytravel

The APS is offering travel awards for individuals who traditionally have been underrepresented in science to attend EB 2017. The specific intent of the APS Minority Travel Fellowship is to increase active participation in and networking at scientific meetings among pre- and postdoctoral students, and early career faculty who are from groups underrepresented in the physiological sciences.

The APS Minority Travel Fellowship Awards are open to:

- underrepresented minority (URM) graduate students, postdoctoral fellows, and early career faculty (recently transitioned or within 5 years of receiving a PhD)
- graduate students, postdoctoral fellows, and early career faculty (recently transitioned) with disabilities.

Applicants must be attending U.S. institutions and conducting research within the 50 states and U.S. Territories. Membership is not required for APS Minority Travel Awards. Porter Physiology Development Fellows are encouraged to apply. Incomplete applications will not be reviewed. Awardees will receive up to $1,800 in expense reimbursement for registration, housing, meals, air and ground transportation, and tips. These awards do not provide funds for international travel.

ADInstruments Macknight Early Career Innovative Educator Award

Application deadline: December 1, 2016

the-aps.org/adi

The ADInstruments Macknight Early Career Innovative Educator Award honors an early career APS member who demonstrates the greatest potential for incorporating innovative teaching techniques and effectively utilizing technology resources in engaging undergraduate students in physiology education. The awardee receives up to $1,500 in travel expense reimbursement and complimentary advanced registration to attend the EB meeting and an Institutional Grant providing the awardee's institution with a PowerLab PTB4152 Physiology Teaching Bundle or equivalent.

EB Undergraduate Research Awards

Abstract deadline: November 17, 2016
Application deadline: January 12, 2017
the-aps.org/bruce

The EB Undergraduate Research Awards are presented annually to undergraduate students who submit a first-author abstract to the EB meeting and present their research at the meeting. There are two types of EB Undergraduate Awards that students can apply for through a single application. See the website for more details and apply online at *the-aps.org/awardapps*.

Barbara A. Horwitz/John M. Horowitz Outstanding Undergraduate Abstract Awards

This award provides $100 and a 2-year complimentary membership with APS. The student must be enrolled as an undergraduate at the time of the abstract submission, be the first author on a submitted abstract for the EB meeting, and be working with an APS member. Selection of awardees is based on the abstract, letter of application, and letter of support from the research host. Receipt of the award is contingent on presenting the research at EB.

David S. Bruce Excellence in Undergraduate Research Awards

To be considered for this award, students must be a Barbara A. Horwitz/John M. Horowitz Outstanding Undergraduate Abstract Awardee and must attend and present a poster at the EB meeting. The recipients receive $400 and a certificate. The highest ranked awardee receives another $250, thanks to the generous contribution of an APS member. Selection of awardees is based on the quality of the poster and oral presentation of the poster to the Award Selection Committee.

Dale J. Benos Early Career Professional Service Award

Application deadline: January 24, 2017
the-aps.org/benos

The Dale J. Benos Early Career Professional Service Award honors an early career stage (graduate student, postdoctoral fellow, assistant professor or equivalent position) member of APS. The award will honor someone who is judged to have made outstanding contributions to the physiology community and demonstrated dedication and commitment to furthering the broader goals of the physiology community. This can be by serving on professional committees, participating in K-12 education outreach, participating in scientific advocacy and outreach programs, or by otherwise strengthening and promoting the physiology community. See the website for more details and apply online at *the-aps.org/awardapps*. ●

Other Education Office Awards

Applications Open Through Winter 2017
To apply for any of these awards, go to *the-aps.org/awardapps*.

Minority Outreach Fellowships

Application deadline: December 1, 2016
the-aps.org/minorityoutreach

The APS Minority Outreach Fellowship seeks to foster communication between minority graduate and postdoctoral students and middle/high school minority life sciences students. Program activities include year-long outreach fellowships for senior graduate students and postdoctoral fellows to visit K-12 classrooms, help conduct teacher professional development workshops, and attend scientific meetings. Funds are provided to attend two EB meetings and one fall conference (ABRCMS or SACNAS), a value of $5,400.

Porter Physiology Development Fellowships

Application deadline: January 15, 2017
the-aps.org/porter

The goal of the Porter Physiology Development Program is to encourage diversity among students pursuing full-time studies toward the PhD in the physiological sciences and to encourage their participation in the Society.

The Porter program provides a full-time graduate fellowship ($28,300 during the academic year) to students in programs leading to the PhD in the physiological sciences at U.S. institutions. The program is open to underrepresented racial and ethnic minority applicants who are citizens or permanent residents of the U.S. or its territories and student members of the Society.

Undergraduate Summer Research Fellowships

Application deadline: February 1, 2017
the-aps.org/summerresearch

APS is proud to offer four programs that allow undergraduate students to participate in research during the summer. Recipients spend an average of 10 weeks in the laboratory of an established scientist and APS member. Each program recruits undergraduate students nationwide, two internationally. Two targeted programs are open to students from disadvantaged backgrounds, from underrepresented racial and ethnic groups, and students with disabilities. Each Fellow receives a stipend plus additional funds for travel to present his or her research at a scientific meeting. Research hosts receive funds for student lab supplies. See the website for more details. ●

Create a Short Physiology Video and You Could Win a Cash Prize and EB Travel Award

Application deadline: December 15, 2016
the-aps.org/video

APS Presents...Phantastic Physiology Voyage: "Function Follows Form" video contest encourages undergraduate and graduate students to creatively connect with physiology and engage with the broader public through a short video contest.

First place awardee(s) receives a total of $750 and up to $1,000 to partially cover travel costs to attend the Experimental Biology 2017 meeting to accept the award. The award-winning video is selected by members of the Career Opportunities in Physiology Committee.

Viewer's Choice awardee(s) receives a total of $250 (determined by total views on YouTube site as of the first day of the Experimental Biology meeting).

Videos should creatively demonstrate and/or explore a specific physiological function in 5 minutes or less (including credits). The target audience is the general public. Video can be staged as a short play, commercial, news broadcast, talk show, music video, documentary, etc. No professional assistance can be provided to produce the video in any manner. Videos are uploaded to YouTube for viewing. Application materials are submitted online to APS.

To view previous award-winning videos and find more information, visit *the-aps.org/video*.

Start filming now and submit your award-winning video by December 15 at *the-aps.org/awardapps*.

Science Policy

APS Offers NIGMS Suggestions on Biomedical Graduate Education

On June 8, 2016, the National Institute of General Medical Sciences (NIGMS) released a request for information (RFI, NOT-GM-16-109) seeking input on several issues related to "Strategies for Modernizing Biomedical Graduate Education." The RFI focused on how NIGMS could identify and implement new strategies to enhance graduate education through the existing institutional predoctoral training grants program. The APS Science Policy and Education Committees jointly developed a response emphasizing the need for students to develop a range of core skills to serve them in their future careers. Below are excerpts of the APS comments. The full response is available on the APS website.

Topic 1: Current Strengths, Weaknesses, and Challenges in Graduate Biomedical Education

Strengths

The current system, which is essentially an apprenticeship model, is very good at training people to be academic researchers. Students are major contributors to academic research and by and large go on to successful careers. NIGMS training grants are essential to continue to encourage the best and the brightest students to enter the field of biomedical research. These programs also provide the best potential to increase diversity and address the current disparities in the biomedical workforce, which is an ongoing goal for the NIH and the research community as a whole.

Weaknesses

Many students pursue careers outside of the traditional academic laboratory setting, either because they choose to do so or because the changing job market has reduced the number of available tenure-track academic positions. Since most of those who now serve as mentors were trained in academic settings under the apprenticeship model, few are equipped to help train their students for jobs outside of academia. Consequently, their students may not be acquiring the skills that they will need to be successful.

Challenges

Scientific fields change quickly, and it is a significant challenge for graduate programs to adapt and provide access to training for the broad range of skills that students will need to succeed in the workforce, whether they pursue positions within or outside of academia.

NIH's training programs have not fully adapted to the reality that only a minority of PhDs remain in academic research. As a result, when training grants are reviewed, there continues to be a heavy emphasis on the proportion of a program's trainees who go on to academic research careers. To remedy this, when training grants are reviewed, emphasis should be placed on the training environment that the program provides, and success in terms of career outcomes should be broadly defined to include a diversity of employment sectors. NIH should also consider modifying language in requests for applications for training grants to indicate that the overall goal of the program is to train the next generation of scientists for a broad range of scientific careers.

Topic 2: Changes (To NIGMS Training Programs) That Could Enhance Graduate Education to Ensure That Scientists of Tomorrow Have the Skills, Abilities, and Knowledge They Need to Advance Biomedical Research as Efficiently and Effectively as Possible.

Graduate training should move toward a model of formal education for foundational scientific skills, especially scientific method and design. Not only would this type of training improve the rigor of scientific discovery, it would also allow greater flexibility in the next stage of a student's career since a sound understanding of scientific methods or statistics can be applied in multiple fields.

Students should be encouraged to make use of Individual Development Plans (IDPs) to help them focus their career plans and acquire the skills they will need to be successful on their chosen path.

Topic 3: The Major Barriers to Achieving These Changes and Potential Strategies to Overcome Those Barriers

Most graduate programs lack general, standardized training for laboratory techniques. For students to be able to identify the best experimental approaches or come up with a good testing paradigm, it is essential that they know and consider interaction between systems and methods. Most graduate programs also lack training in teaching, budgeting, and many of the other skills listed below. Although some aspects are taught through the mentor/mentee relationship, it is not formalized or standardized in many cases.

As we see in a discipline such as physiology, scientific research can be highly specialized within and between fields. This presents a significant challenge in providing any kind of standardized training. For example, trying to provide standardized training in laboratory techniques, experimental design, or statistical analysis would be a huge challenge in many graduate programs, especially the large "umbrella" type of programs that are now quite common at large research institutions.

In general, there should be more of an investment in developing courses in graduate training programs that would offer the opportunity to develop some of the skills described below. Instead of having each institution develop its own courses for each of the skills, it makes more sense for NIH to sponsor the development of online courses that can be shared.

Topic 4: The Key Skills That Graduate Students Should Develop to Become Outstanding Biomedical Scientists, and the Best Approaches for Developing Those Skills

These key skills could include but not be limited to *1)* essential skills applicable to all fields that ensure ability to design meaningful experiments and critically analyze data, *2)* ability to adapt new and emerging technologies or approaches, and *3)* other skills such as team science.

Among the skills that students need are scientific method and process; training related to rigorous experimental design; statistical analysis; data management; journal article writing and review; grant writing and review; budgeting; teaching; mentoring and leadership; educational outreach; laboratory and project management; time management; team science; communications; tech transfer; and business/finance for scientific lab management. These skills are necessary not only for success as an academic researcher, but they are also essential for anyone who is looking to pursue a career outside of a laboratory setting. Many of these skills could be offered in foundational coursework early in PhD programs. The teaching of some skills may be well suited to presentations offered via online courses that could be made available to multiple grantee institutions.

The NIGMS supports a diverse set of institutional training grant programs, each with their own particular strengths. NIGMS should encourage programs to develop training modules or workshops based on their existing areas of strength. These modules or workshops could then be shared with other NIGMS-supported trainees.

Topic 5: Potential Approaches to Modernizing Graduate Education Through the Existing NIGMS Institutional Predoctoral Training Grants Program to Ensure That Trainees Have the Skills and Knowledge They Need to be Prepared to Enter the Biomedical Research Workforce

NIGMS should encourage grantees to include foundational training that includes the skills listed above into their training programs.

Institutional training grant review should focus less on any particular career outcome of students and more on the research environment being provided by the institution. Institutional training grant programs should clearly demonstrate how they will provide students with the skills they will need to pursue various career pathways.

Reviewers of training grants should not equate success with a high proportion of graduates going into academic research settings. Instead, the definition of career success should be broadened to include non-bench research-related academic and non-academic career choices. ●

Spotlight on Nonhuman Primate Research

NIH held a workshop September 7, 2016 on "Ensuring the Continued Responsible Oversight of Research with Non-Human Primates." In advance of the workshop, experts from nine scientific societies including the American Physiological Society released a white paper on *The Critical Role of Nonhuman Primates in Medical Research* to highlight some of the important findings from this research.

NIH Workshop

The NIH workshop on nonhuman primate (NHP) research was organized at the behest of the House Appropriations Committee. In the report accompanying NIH's FY 2016 funding legislation, the House panel asked the agency to "conduct a review of its ethical policies and processes with respect to nonhuman primate research subjects, in consultation with outside experts, to ensure it has appropriate justification for animal research protocols." In July 2015, when this language was added to the funding report, PETA was in the midst of a campaign to shut down the work of a National Institute of Child Health and Human Development (NICHD) researcher studying infant development in monkeys. In December 2015, NICHD announced that that this research program would be phased out over 3 years, citing what it said were financial reasons (see "NIH to end research targeted by PETA" *http://www.the-aps.org/mm/Publications/Journals/Physiologist/Archive/2016-Issues/March-2016-Vol-59No-2/Science-Policy/NIH-to-End-Research-Targeted-by-PETA*).

On the morning of September 7, NIH Director Francis Collins welcomed the workshop participants and said that the agency was looking forward to their input. He noted that NIH's mission-expanding fundamental knowledge and using that knowledge to improve human health also includes responsible use of animals. Carrie Wolinetz, Associate Director for Science Policy, said that NIH takes its stewardship of animals seriously. She also indicated that, although she was confident that the current oversight framework for NHP research is robust, periodic reviews are healthy because "[th]e best science is informed by good ethical thinking."

The theme of the morning session was the state of NIH-supported research involving NHPs. Five speakers discussed the role NHPs play in research on infectious diseases such as HIV/AIDs, Ebola, and Zika; the neurobiology of social disorders such as autism; developing techniques for heart muscle regeneration; issues related to fertility and infertility; and the development of brain-machine interfaces and Deep Brain Stimulation. In the ensuing discussion, workshop participants were asked to identify what can be done to improve utilization of NHPs. Suggestions included publishing negative data to prevent unnecessary duplication of studies; establishing a repository of research findings to maximize their impact; obtaining more data from each animal; ensuring that studies are reproducible; and better characterization of NHP models.

The afternoon session addressed the current ethical oversight framework for NIH-supported research with NHPs. Topics included an overview of the Animal Welfare Act Regulations and its requirements to ensure the psychological well-being of NHPs, as well as resources for reducing, refining, and replacing NHPs that are available through the USDA's Animal Welfare Information Center. There was a presentation on NIH's own oversight requirements for NHP research as mandated by the Health Research Extension Act of 1985, which include the PHS Policy on the Humane Care and Use of Animals in Research; U.S. Government Principles for the Utilization and Care of Vertebrate Animals in Teaching, Research, and Training; and the *Guide for the Care and Use of Laboratory Animals*. Other presentations described specific ethical considerations with respect to NHP research; the veterinarian's perspective on managing NHPs in research; and how IACUCs implement regulations, guidance, and policies when they evaluate protocols.

During the workshop, Wolinetz also shared comments submitted online. A number of these were critical of the workshop, asserting that it should have devoted more time to topics such as alternatives and alleging that participants who work with NHPs have a conflict of interest. Several of these commentators made it clear that they were inalterably opposed to NHP research.

The entire workshop is archived on the NIH website at *https://videocast.nih.gov/summary.asp?Live=19663&bhcp=1*. In addition, the APS Office of Science Policy provided reports and commentary via the @SciPolAPS Twitter feed using the hashtag #NIHPrimateWorkshop.

Scientific Societies' White Paper

In advance of the NIH workshop, the APS joined with other scientific societies in developing a white paper on NHP research. Experts from the APS, American Academy of Neurology, American Transplant Foundation, Federation of American Societies for Experimental Biology, Society for Neuroscience, American Society for Microbiology, American College of Neuropsychopharmacology, the Endocrine Society, and the Foundation for Biomedical Research contributed to the development of *The Critical Role of Nonhuman Primates in Medical Research.*

Although scientists can address many research questions by studying species such as fruit flies, zebrafish, and rodents, NHPs are needed to answer others. That is because these species' immune systems, metabolism, and brain structures more closely resemble those of humans. Promising areas of research highlighted in *The Critical Role of Nonhuman Primates in Medical Research* include:

- Treating glioblastoma with a modified version of the polio virus to help the body's immune system mobilize against this deadly brain cancer
- Using antibodies to prevent HIV-positive mothers from transmitting the virus to their babies
- Boosting the effectiveness of treatments for HIV and chronic hepatitis B with a combination of drugs that stimulate the immune system
- Preventing the transmission of the Zika virus from a pregnant woman to her fetus via the placenta
- Coaxing patients' immune systems to tolerate organ transplants to remove the need for immune-suppressant drugs
- Mapping how the brain works to improve treatments for such diseases as Parkinson's, other movement disorders, Alzheimer's, alcoholism, drug addiction, bipolar disorder, and autism
- Developing technology to enable paralyzed individuals to control a prosthetic arm through brain-machine interfaces
- Understanding why blood pressure rises as we age and the mechanisms that underlie an inherited form of hypertension in baboons that is similar to a condition seen in humans
- Finding better treatments for obesity and diabetes. ●

OLAW Provides Guidance on Changing Animal Activities

The NIH Office of Laboratory Animal Welfare (OLAW) has a new webinar providing guidance on implementing its policy on significant changes to animal activities (NOT-OD-14-126). During the September 8, 2016 webinar, speakers from Colorado State University (CSU) discussed how their institution incorporated veterinary verification and consultation (VCC) into their oversight of animal protocols. They also shared examples of animal activities that were appropriate candidates for VCC. To access the archived webinar and slides, as well as supporting materials from CSU, go to *http://grants.nih.gov/grants/olaw/educational_resources/webinar_09082016.htm*. ●

Meetings

The APS Institute on Teaching and Learning

Madison, Wisconsin, June 20-24, 2016

In the early summer of 2016, APS held its second Institute on Teaching and Learning in the vibrant university-town of Madison, Wisconsin. The workshop allowed attendees to learn new skills and techniques so they can teach physiology in their own classrooms. The workshop was organized by Barbara Goodman (Chair; University of South Dakota), along with an Organizing Committee that is equally dedicated to the art of teaching. The committee members included Beth Beason (Rice University), Gregory Brower (Texas Tech. University Health Science Center), Robert Carroll (East Carolina University), Chaya Gopalan (Southern Illinois University, Evansville), Patricia Halpin (University of New Hampshire, Manchester), Rachael Hannah (University of Alaska, Anchorage), Jonathon Kibble (University of Central Florida College of Medicine), Maureen Knabb (Westchester University), Jenny McFarland (Edmonds Community College), Dee Silverthorn (University of Texas at Austin), Mary Pat Wenderoth (University of Washington), Ann Wright (Canisius College), Bruce Wright (Ross University), and J. Michael Wyss (University of Alabama at Birmingham). The committee organized a program that included a packed schedule of dynamic plenary lectures, workshops, interactive poster sessions, and networking opportunities that made the workshop a valuable experience not only for those who are interested in teaching physiology but also for attendees who wanted to learn some new teaching techniques to engage their students.

The workshop was attended by 124 total registrants, of whom 64 (52%) attendees identified themselves as APS members, 20 (16%) registered as nonmembers, 9 (7%) attendees were identified as postdoctoral fellows, 4 (3%) were considered as students attendees, and invited speakers made up the remaining 24 (19%) attendees. Table 1 shows the breakdown of the different registration types. This workshop mainly attracted individuals from the U.S.; however, of the 124 registrants, 8 (9%) were from outside of the U.S. Table 2 depicts the breakdown of attendees by country.

Table 1. Registration statistics

Registration Type	Number of Attendees (%)
APS member	64 (52%)
Nonmember	20 (16%)
Postdoctoral fellows	9 (7%)
Students	4 (3%)
Exhibitors	3 (3%)
Invited chairs/speakers	24 (19%)
Total	**124 (100%)**

Table 2. Registration geographic region statistics

Region	Number of Attendees (%)
U.S.	114 (91%)
Australia	1 (1%)
Brazil	1 (1%)
Canada	1 (1%)
Columbia	1 (1%)
Grenada	2 (1%)
Israel	1 (1%)
Mexico	1 (1%)
St. Maarten	1 (1%)
Total	**124 (100%)**

The workshop program consisted of 1 keynote lecture, 10 plenary lectures, and 18 unique workshops on a wide variety of topics related to the teaching of physiology and classroom teaching techniques. The audience was encouraged to share their ideas and thoughts with the speakers at the end of their talks. The workshop 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 desserts and beverages before the opening keynote lecture. There were also three afternoon poster sessions where scientists presented their work and discussed their teaching styles and techniques with other attendees. Moreover, the workshop had daily group meals that facilitated further discussions between the attendees.

A total of 60 abstracts were submitted for the workshop. Thirty-three of these abstracts were programmed as poster presentations. The remaining 27 abstracts were submitted by invited speakers. Of the abstracts submitted for the workshop, 34 (57%) were submitted by a female first author; 4 (7%) were submitted from institutions outside of the U.S., including abstracts from Brazil, Canada, Columbia, and Grenada.

At the workshop, attendees Kelli Carter (University of South Florida) and C. J. Urso (Seton Hall University) were the recipients of the American Physiological Society Abstract Travel Award. Carter and Urso were congratulated by the attendees and presented with a certificate and cash prize. In addition, Daniela Terson de Paleville (University of Louisville) was awarded the Minority Travel Fellowship Award, which is provided to encourage participation of under-represented minority individuals in the physiological sciences. The fellowship provides reimbursement of all expenses associated with travel and participation in the workshop.

The American Physiological Society and the Organizing Committee gratefully acknowledge the financial support provided though a generous educational grant from ADInstruments.

Workshop attendees engage in lively discussion during a poster session

Camilo Lellis-Santos *(middle)* discusses his teaching techniques with attendees

Workshop Organizer, APS Member Barb Goodman *(left)* presents Kelli Carter *(right)* with her Abstract Travel Award certificate

Goodman *(left)* presents C. J. Urso *(right)* with his Abstract Travel Award certificate

Inflammation, Immunity, and Cardiovascular Disease

Westminster, Colorado, August 24-27, 2016

Late this summer, the second 2016 APS Conference, "Inflammation, Immunity, and Cardiovascular Disease," was held in the suburban town of Westminster, Colorado. The conference was organized by APS Members, David Harrison (Chair; Vanderbilt University) and David Mattson (Co-Chair; Medical College of Wisconsin, Milwaukee). Helping Harrison and Mattson were the Organizing Committee members, including Meena Madhur (Vanderbilt University), Brett Mitchell (Texas A&M University), Ernesto Schiffrin (McGill University, Canada), and Cornelia Weyand (Stanford University). The committee organized a program that included a packed schedule of symposia covering a wide breadth of topics from basic immunity to inflammation in the body.

The conference was attended by 135 total registrants, of whom 26 (19%) attendees identified themselves as APS members, 23 (17%) registered as nonmembers, 17 (13%) attendees were identified as postdoctoral fellows, 30 (22%) were considered as students attendees, and invited speakers made up the remaining 39 (29%) attendees. Table 1 shows the breakdown of the different registration types. This conference mainly attracted professors from the U.S.; however, of the 135 registrants, 27 (20%) represented countries from Australia, Canada, Germany, Ghana, Israel, Japan, Spain, Sweden, and the UK. Table 2 depicts the breakdown of the different regions that attendees came from.

The conference program consisted of one plenary lecture, nine symposia sessions, as well as two interactive poster sessions, opportunities for oral abstract presentations presented by student and postdoctoral fellows, and a career workshop. The audience was encouraged to share their ideas and thoughts with the speakers at the end of their talks. The conference included a Welcome and Opening Reception, which was designed to give attendees a chance to meet with long-time colleagues and create new friendships before the opening plenary lecture.

Table 1. Registration statistics

Registration Type	Number of Attendees (%)
APS members	26 (19%)
Nonmembers	23 (17%)
Postdoctoral fellows	17 (13%)
Students	30 (22%)
Invited chairs/speakers	39 (29%)
Total	**135 (100%)**

Table 2. Registration geographic region statistics

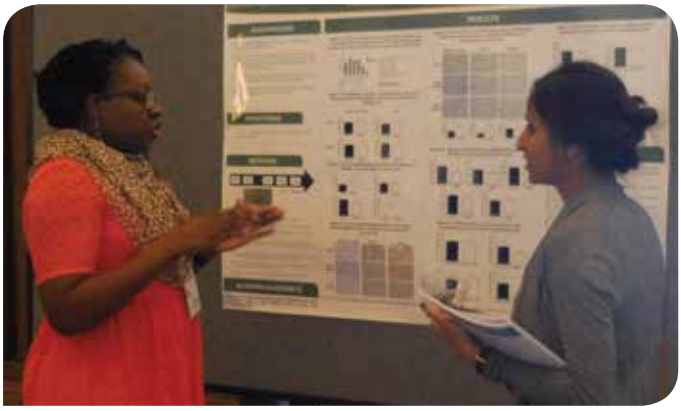
Region	Number of Attendees (%)
U.S.	108 (80%)
Australia	5 (3%)
Canada	3 (2%)
Germany	5 (4%)
Ghana	1 (1%)
Israel	1 (1%)
Japan	4 (3%)
Spain	1 (1%)
Sweden	4 (3%)
UK	3 (2%)
Total	**135 (100%)**

A total of 97 abstracts were submitted for the conference. Sixty-seven of these abstracts were programmed as poster presentations. Moreover, 15 authors of these abstracts had the opportunity to present their work as an oral presentation during the conference. The remaining 30 abstracts were submitted by invited speakers. Of the abstracts submitted for the conference, 40 (41%) were submitted by a female first author; 22 (22%) were submitted from institutions outside of the U.S., including abstracts from Australia, Canada, China, Germany, Ghana, Japan, Spain, Sweden, and the UK.

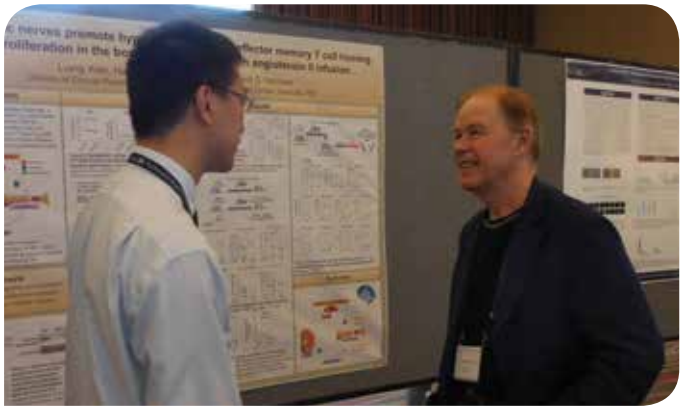
At the conference, the attendees Ellen Gillis (Augusta University), Fanny Laroumanie (Vanderbilt University), Jonghae Lee (University of Missouri, Columbia),

Maggie Lieu (Monash University, Australia), Paola Marcovecchio (La Jolla Institute), Bindiya Patel (University of Alabama at Birmingham), Cayla Rodia (University of Connecticut), Amrita Sarkar (Thomas Jefferson University), Anna Wang (Monash University, Australia), and Liang Wang (Vanderbilt University) were the recipients of the American Physiological Society Abstract Travel Award. The awardees were congratulated by the conference organizers and attendees, and presented with a certificate and cash prize at the Closing Banquet and Awards Ceremony. In addition, Ruben Banuelos (A. T. Still University), Shirley Dehn (Northwestern University), Ijeoma Obi (University of Alabama at Birmingham), and Lia Taylor (Augusta University) were awarded Minority Travel Fellowship Awards, which are provided to encourage participation of under-represented minority individuals in the physiological sciences. The fellowship provides reimbursement of all expenses associated with travel and participation in the workshop. Moreover, thanks to a generous grant from the National Institutes of Health, National Heart, Lung, and Blood Institute, 28 attendees were recipients of a travel award for the conference.

The American Physiological Society and the Organizing Committee gratefully acknowledge the financial support provided though generous educational grants from ADInstruments, the American Heart Association's Council on Hypertension and the Kidney in CV Disease, Data Sciences International, DMT-USA, Miltenyi Biotec, Inc., and NIH, National Heart, Lung, and Blood Institute.



Conference attendees engage in lively discussion during a poster session



Conference Organizer David Harrison *(right)* discusses a poster with an attendee

Conference Organizers and APS Members David Harrison and David Mattson congratulate the Abstract Travel Awardees at the Conference Banquet and Award Ceremony

Experimental Biology

Experimental Biology 2017 Distinguished Lectures

Physiology in Perspective: The Walter B. Cannon Memorial Award Lecture

Michael Welsh

Univ. of Iowa Roy J. and Lucille Carver Col. of Med.

Insights into the Pathogenesis of Cystic Fibrosis Lung Disease

Saturday, April 22, 2017, 5:30 PM

Claude Bernard Distinguished Lectureship of the APS Teaching of Physiology Section

Jeffrey D. Karpicke

Purdue Univ.

Retrieval-Based Learning: Simple Strategies for Helping Students Learn

Sunday, April 23, 2017, 10:30 AM

Hugh Davson Distinguished Lectureship of the APS Cell and Molecular Physiology Section

Jennifer Lippincott-Schwartz

HHMI Janelia Research Campus

Emerging Fluorescence Technology to Study Cell Architecture and Dynamics

Sunday, April 23, 2017, 2:00 PM

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

Peter Bie

Univ. of Southern Denmark-Odense

Mechanisms of Sodium Balance

Sunday, April 23, 2017, 4:15 PM

Henry Pickering Bowditch Award Lecture

Brant Isakson

Univ. of Virginia

Coordinating Tissue Function Through Heterocellular Communication

Sunday, April 23, 2017, 5:45 PM

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

Robin McAllen

Florey Inst. of Neuroscience and Metal Health

Mapping the Diversity of Central Autonomic Control

Monday, April 24, 2017, 8:00 AM

Solomon Berson Distinguished Lectureship of the APS Endocrinology and Metabolism Section

Wolfgang Langhans

ETH Zurich

GLP-1 in Energy Homeostasis: There is More than Meets the *Eye*

Monday, April 24, 2017, 10:30 AM

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

W. Larry Kenney

Penn State Univ.

Skin-Deep Insights into Vascular Aging

Monday, April 24, 2017, 2:00 PM

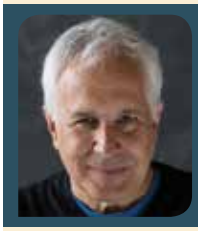
Carl W. Gottschalk Distinguished Lectureship of the APS Renal Section

Paul Welling

University of Maryland Med. Sch.

Protecting Potassium Balance at All Costs: Molecules to Man

Monday, April 24, 2017, 3:15 PM



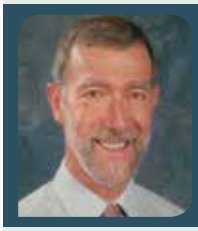
Joseph Erlanger Distinguished Lectureship of the APS Central Nervous System Section

Jeff Lichtman

Harvard Univ.

Does Connectomics Make Sense?

Monday, April 24, 2017, 3:15 PM



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

Peter D. Wagner

Univ. of California, San Diego

Lung and Muscle Heterogeneity: Consequences for Oxygen Transport Limitation

Tuesday, April 25, 2017, 10:30 AM

History of Physiology Lecture

Jack Rall

Ohio State Univ.

The XIIIth International Physiological Congress in Boston in 1929: American Physiology Comes of Age

Tuesday, April 25, 2017, 1:00 PM

Robert M. Berne Distinguished Lectureship of the APS Cardiovascular Section

Benjamin D. Levine

Texas Health Presbyterian Hospital Dallas

Astronauts, Athletes, and Aging. A TripTik Guide to the Cardiovascular Adaptation to Physical Activity

Tuesday, April 25, 2017, 2:00 PM

August Krogh Distinguished Lectureship of the APS Comparative and Evolutionary Physiology Section

Warren Burggren

Univ. of North Texas

Developmental Plasticity, Epigenetics and Evolution: A Comparative Physiologist's Od(d)yssey

Tuesday, April 25, 2017, 3:15 PM

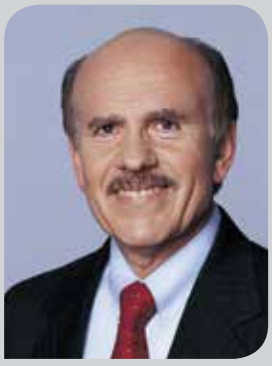
Horace W. Davenport Distinguished Lectureship of the APS Gastrointestinal and Liver Physiology Section

John L. Wallace

Antibe Therapeutics and Univ. of Calgary

Why the GI Tract Does Not Digest Itself: A Davenport-Inspired Journey

Tuesday, April 25, 2017, 3:15 PM



APS Nobel Prize Award Lecture

Louis Ignarro

UCLA Sch. of Med.

The Road to Stockholm: A Nobel Mission

Wednesday, April 26, 2017, 4:45 PM

Upcoming EB Symposia

Mark your calendars for professional development symposia at Experimental Biology 2017! Check the symposia web sites for more information.

Curricular Innovation in Sex and Gender Based Medical Physiology Education

Saturday, April 22, 2017, 8:00 AM-12:00 PM

the-aps.org/refresher-sexandgender

(Medical Education Refresher Course sponsored by the Education Committee)
Organizers: Karie Scrogin, Jennifer Sasser
Location: Convention Center, Room W190A

Get an update on curricular innovation in sex- and gender-based medical physiology education content from leading experts in the field: **Introduction of Sex and Gender Health Differences in Medical Curricula** (Marjorie Jenkins, Internal Medicine, Texas Tech University Health Sciences); **Ovarian Hormones Mood and Cognition** (Gillian Einstein, Psychology, University of Toronto, Dalla Lana School of Public Health); **Sex Differences in Fetal Programming: Consequences for Multiple Generations** (Kent Thornburg, Medicine, Oregon Health and Sciences University); **Update on Sex and Gender Differences in Cardiovascular Physiology** (Virginia Miller, Surgery, Mayo Clinic)

2017 Mentoring Symposium: Choosing the Right Lab and Personnel for Your Career

Sunday, April 23, 2017, 10:30 AM-12:30 PM

the-aps.org/choosing-a-lab-and-personnel

(Sponsored by the Women in Physiology Committee)
Organizers: Karen L. Edelblum, Brandi Wynne
Convention Center, Room W192A

Every new graduate student, postdoc, or faculty member must make difficult decisions regarding the people we choose to work with. These choices will affect not only productivity and chances for success, but also our sense of well-being. Thus these decisions are among the most important we make. As a trainee, you will choose which mentor to work with to complete your studies, and, as an investigator, you will choose which trainees to employ in your laboratory. There are many factors associated with identifying an ideal mentor/mentee relationship, whether you are the applicant looking for a position or the investigator hiring to fill a position. The Women in Physiology Symposium for 2017 will focus on the key aspects of this complicated decision-making process. To address this, we will discuss the crucial points that newly minted PhD students will want to consider when choosing a lab to facilitate success as a postdoctoral fellow, whether they follow career paths in academia or in industry. For a new faculty member, the decision of whom to hire as a postdoc is just as critical for the success of a fledgling lab; therefore, we will also discuss important factors to be considered when interviewing postdoc candidates. Last, despite the best effort of both the applicant and the investigator, not every candidate or prospective mentor will be the perfect match. Our final speaker will discuss the best exit strategies to employ when it is time to sever the mentee/mentor relationship. This will be discussed from both the perspective of the mentor in letting trainees move on to a different position, as well as the position of the trainee, whose needs may not be fulfilled by their current mentor. A panel discussion will take place following the last seminar. The goal of this symposium is to provide you with the tools to help make these important decisions of your career. Presentations include: **Selecting a Good Mentor and Lab for an Academic Postdoc** (Karen Sweaze, Arizona State University); **Selecting a Good Lab for a Postdoctoral or Research Experience in Industry** (Shaila Basavappa, Takeda Pharmaceutical); **Selecting a Postdoc for Your Laboratory** (R. Clinton Webb, Medical College of Georgia at Augusta University); **How to Let Go and Make Go** (Marshall "Chip" Montrose, University of Cincinnati).

Monday, April 24, 2017, 10:30 AM-12:30 PM

the-aps.org/teachingcareer

Panel Discussion

The Many Facets of a "Teaching Career"

(Career Symposium)

Organizers: Clintoria R. Williams, Josef Brandauer

Convention Center, Room W192C

This symposium is targeting scientists pursing an academic career that includes teaching. Teaching has become a desired and required component of many academic careers. Teaching requirements and expectations vary significantly between institutions and even departments. In order for job candidates to reach an informed decision regarding the institution that is the best fit for their career goals, an understanding of these requirements and expectations is needed. The specific goals of this symposium are for participates to be able to *1)* identify clues within job announcements that will provide hints about teaching expectations, *2)* learn about teaching expectations at various institution types (regional, research intensive, liberal arts, and historically black colleges and universities), and *3)* provide insight into strategies for "successful teaching" from instructors at various career stages and institution types. Specific questions that panelists will address include: How to determine whether a teaching career is the right choice? How to prepare for a teaching career at any level? How to find the right fit in regard to institution and department? How to balance teaching-research life? How to determine the value of teaching in the evaluation process for promotion? Presentations include: **How to Dissect a Job Announcement: Dirty Little Secrets** (Erica Wehrwein, Michigan State University, East Lansing); **Teaching-Research Balance in the Changing Classroom - HBCU** (Dexter Lee, Howard University); **Perspective from a Liberal Arts College** (Lara Deruisseau, LeMoyne College); **Educating the Educator for Successful Teaching - R1 Institution** (David Harris, University of Central Florida); **Mentoring: Bridging the Gap Between the Classroom and the Laboratory - Regional University** (Carissa Krane, Dayton University).

Wednesday, April 26, 2017, 10:30 AM-12:30 PM

the-aps.org/kick-start-your-funding

Kick Start Your Funding: Looking Beyond NIH and NSF

(Trainee Symposium)

Organizers: Jennifer Steiner, Kristi Streeter

Convention Center, Room W192C

Funding for scientific research is becoming progressively harder to obtain, and competition continues to grow. Despite the increased challenge to gain federal funding (i.e., NIH), many universities and other institutions require their applicants to have funding when applying for faculty positions, regardless of their career stage. As such, an enormous amount of pressure is placed on trainees to obtain funding before looking for a position as an independent scientist. In addition, early career investigators who have already transitioned to independent positions also experience similar pressures and difficulties in obtaining funding. In recognition of the funding crisis as well as the increasingly competitive job market for trainees and early career investigators, the goal of this symposium is to provide information on funding sources outside of the NIH and NSF. We will have four speakers, with each representing less tradition funding mechanisms including *1)* industry, *2)* private foundations, *3)* crowd-funding, and *4)* military funding. Each speaker will identify how to find funding within their genre, provide information and tips for writing successful grant proposals, and compare and contrast their organization with how other funding mechanisms (i.e., NIH) work. To provide such information, we have invited speakers who have successfully obtained funding (Dr. Leon, military) as well as representatives from companies or private foundations that either have grant programs or regularly fund product research. We will also have a representative to discuss fund-raising opportunities via crowd-funding websites. This symposium will be conducted in the typical format of 25-minute presentations followed by 5 minutes of questions, with additional interactions to be encouraged following all four speakers' presentations. The presentations are: **Seeking Funding Outside the Norm: Unique Opportunities Within Military Research Programs** (Lisa Leon, Thermal Mountain Medicine Division, Thermal Mountain Medicine Division); **Working With the Nutrition Industry to Promote Scientific Advancement** (Abbott Nutrition Representative, Abbott Nutrition Company); **Cancer Funding from a Private Foundation** (American Cancer Society Representative, The American Cancer Society); **Crowd Funding Your Science** (Christina Tran,- Community Manager, Experiment.com).

Experimental Biology 2017

April 22–26, 2017, Chicago, IL

PHYSIOLOGY PLATFORM SESSIONS

Saturday, April 22, 2017

Room			
W375A			5:30 PM–6:30 PM *APS Lecture* Physiology in Perspective: The Walter B. Cannon Memorial Award Lecture **Welsh**
W190A	8:00 AM–12:00 PM *Education Committee Refresher Course* Curricular Innovation in Sex and Gender Based Medical Physiology Education **Scrogin/Sasser**	2:15 PM–5:15 PM *WEH Section Award Session* WEH Trainee Award Finalists and Data Diuresis	
W196B	9:30 AM–11:30 AM *MCS Symp* President's Symp: (Not) Lost in Translation: Bridging the Gaps Between Microcirculatory Basic Science and Clinical Science **Chillian**	1:00 PM–3:00 PM *MCS Symp* Depression and its Effects on Microvascular Function and Vascular Adaptation **Chantler/Olfert**	6:00 PM–8:00 PM MCS Poster Discussion amd Reception **Jackson**
W196C	9:00 AM–5:00 PM *ETG Group Special Session* Pre-EB Meeting of the ETG Group Young Investigators Symp		
W196A		1:00 PM–3:00 PM *Techniques Workshop Program Workshop* Choosing the Right Diet for Your Animal Model **Lund/Ney**	
W190B			3:15 PM–5:15 PM *Techniques Workshop Program Workshop* Using Ultrasound to Teach Cardiovascular Physiology and Anatomy **Silverthorn/Guttmann**
W192B	1:00 PM–3:00 PM *AFMR Symp* Small GTPases: Basic Science to Translational Research **Zhao/Birukova**	3:15 PM–5:15 PM *AFMR Symp* New Insights into Insulin Resistance-Molecular Mechanisms and Therapeutic Implications **Prabhakar/Hawkins**	3:30 PM–5:30 PM *Microcirculatory Society (MCS) Symp* Investigating Microvascular Physiology and Pathophysiology Using Novel Techniques **Stokes/Gavins**
W193			3:00 PM–5:00 PM *NCAR Section Award Session* DATA NCARnation

Saturday, April 22, 2017, *cont.*

Room			
W194B		1:00 PM–5:15 PM *Physiological Genomics Group Special Session* Fourth Annual Physiological Genomics Group Conference	
W192C		1:00 PM–3:00 PM *Science Policy Committee Symp* Why Scientific Rigor Matters and Ways to Improve It **Sutliff**	
W192A			3:00 PM–5:00 PM *Communications Committee Symp* New Models of Science Communication: Increasing Reproducibility and Transparency **Crecelius/Serrador**

Sunday, April 23, 2017

Room	8:00-10:00 AM	10:30 AM-12:30 PM	3:15-5:15 PM
W375A	*ACSM Symp* A Roadmap for the Future of Exercise Science: Key Research Directions **Bouchard**	*President's Symp Series* Sex Differences in Physiology and Pathophysiology **Prieto**	*Integrative Physiol Symp Series* Hypoglycemia: Neural Pathways of the Glucose Counter-Regulatory Response, Hypoglycemia Unawareness and Its Treatment **Verberne** 5:45 PM–6:45 PM Henry Pickering Bowditch Award Lecture **Isakson**
W190A	*GIL Section Special Session* John Forte Session of the GIL Session	10:30 AM–11:30 AM Teaching Section Bernard Lecture **Karpicke** 2:00 PM–3:00 PM Cell Section Davson Lecture **Lippincott-Schwartz**	3:15 PM–4:15 PM *WEH Section Lecture* WEH New Investigator Award Lecture 4:15 PM–5:15 PM WEH Section Lecture WEH Section Starling Lecture **Bie**
W196B	*CV Section FT* Sex Differences in Obesity and Cardiovascular Disease **Belin de Chantemele/Loria**	*EEP Section Symp* Cerebral Function of Permanent Residents of High Altitude: Problems and Solutions **West/Simonson**	3:15 PM–4:15 PM MCS Landis Award Lecture 4:30 PM–5:30 PM MCS Business Meeting **Chillian**
W196C	*NCAR Section Symp* Cardiac Sensory Afferents: The Cornerstone for Autonomic Reflex Processing in Health and Disease **Shivkumar/Wang**	NCAR Section Young Investigator Awards **Young/Sabharwal**	*CV Section FT* Immune Cells Involved in Cardiovascular Disease **Mitchell/Madhur**

Sunday, April 23, 2017, *cont.*

W196A	*Nutrition Group Symp* From Cancer-Causing Villain to Health-Promoting Hero: Taking a U-turn on Dietary Nitrite and Nitrate? **Keen/Hast**	*Resp Section Symp* Ventilatory Control and Function Following Perinatal Insults **Bailey/Revill**	*Resp Section FT* Epigenetic Regulation of Lung Development, Injury, and Repair **Rogers/Tipple**
W190B	*EEP Section Symp* SIRT1 in Muscle Health and Disease **Ljubicic**	*CV Section FT* Vascular-Mediated Mechanisms of Cognition and the Role of Exercise **Baynard**	*E&M Section Symp* Stressing the Beta Cell: The Good and The Bad **Corbett**
W192B	*CV Section Symp* Mathematical Modeling of Cardiac Excitability and Arrhythmias **Hund/Ripplinger**	*Physiol in Industry Committee Symp* Nitric Oxide Pathway Modulation for Therapeutic Intervention and Exercise Enhancement **Mattson**	*NCAR Section FT* Ion Channel Modulation: Contributions to Autonomic Dysfunction in Cardiovascular and Metabolic Diseases **Chen/Toney**
W193	*Teaching Section Symp* Inclusive Practices for Diverse Student Populations **Johnson**	*Muscle Biology Group Symp* The Role of Ribosome Biogenesis in Skeletal Muscle Hypertrophy **McCarthy**	*ETG Group Symp* MicroRNAs in Kidney and Epithelial Transport Physiology, Development and Disease **Loffing/Cai**
W194B	*Renal Section FT* Renal Section Young Investigator Award FT **Pochynyuk**	*Renal Section Symp* Mining Protein-Protein Interactions to Study Renal Transporter Regulation **Chen/Brown**	*PG Group Symp* Single Cell Analysis and Genomic Approaches **Coller**
W192C	*WEH Section FT* Stress and Physiological Responses Throughout Life **Pollock/Taylor**	*PG Group Award Session* Trainee Highlights in Physiological Genomics	*Cell Section FT* Cellular Mechanisms of Stress and Inflammation **Bomberger/White**
W192A	*TransPhys Group FT* Translation Physiology Showcase: Focus on Sex differences in Obesity and Blood Pressure, Developmental Programming, and Women's Health	*Women in Physiol Committee Symp* Choosing the Right Lab and Personnel for Your Career **Edelblum/Wynne**	CEPS Section Trainee-Driven FT

Monday, April 24, 2016

Room	8:00-10:00 AM	10:30 AM-12:30 PM	3:15-5:15 PM
W375A		*President's Symp Series* Women's Health Research **Brooks**	*Integrative Physiol Symp Series* Engineering for Health: Integrating Engineering and Physiology to Combat Disease **Yosten**
W190A	8:00 AM–9:00 AM NCAR Section Ludwig Lecture **McAllen** 9:00 AM–10:00 AM NCAR Section Ludwig MiniSymp	E&M Section Berson Lecture **Langhans**	2:00 PM–3:00 PM EEP Section Adolph Lecture **Kenney** 3:15 PM–4:15 PM Renal Section Gottschalk Lecture **Welling**

Monday, April 24, 2017, *cont.*

Room	8:00-10:00 AM	10:30 AM-12:30 PM	3:15-5:15 PM
W196B	*CV Section Symp* Vascular Autophagy and Endothelial Cell Nitric Oxide Generation **Symons/Gutterman**	*CV Section Symp* Adipose Tissue and the Cardiovascular System: Interactions with Sleep and Cardiometabolic Risk Factors **Covassin/Gollasch**	3:15 PM–4:15 PM CNS Section Erlanger Lecture **Lichtman** 4:15 PM–5:15 PM CNS Section Erlanger MiniSymp
W196C	*WEH Section FT* Hypertension and Sodium Homeostasis-Developing Concepts **Sandberg/Gohar**	*WEH Section FT* Neural and Hormonal Modulation of Fluid Balance and Ion Homeostasis in Health and Disease **Gao/Schiller**	*Teaching Section Symp* Examining the Changing Landscape of Course Delivery and Student Learning **Halpin/Gopalan**
W196A	*E&M Section FT* Brown Fat versus Skeletal Muscle as Metabolic Targets: Activation of Thermogenesis or Glucose Oxidation **Symonds**	*Resp Section FT* Respiratory Control: Beyond the Diaphragm **Fuller/Turner**	*Resp Section Symp* The Emerging Role of Lipids in Lung Physiology and Disease **Kuebler/Letsiou**
W190B	*Hypoxia Group FT* Phyisological Adaptation to Hypoxia and High Altitudes **Iturriaga**	*CV Section FT* Carl J. Wiggers Award FT **Heusch/Meininger**	*E&M Section Symp* Sleep, Circadian Clocks and Metabolism **Van Cauter/Samson**
W192B	*CV Section Symp* Ubiquitin and Ubiquitin-Like Proteins in Cardiovascular Physiology and Disease **Weisleder/Wang**	*CEP Section Symp* Consequences of Physiological Stressors on the Development and Function of the Cardiovascular System **Gillis**	*CV Section Symp* Emerging Concepts in the Local Regulation of Blood Flow **Freed/Duncker**
W193	*Teaching Section FT* Innovation in Teaching: Abstract Driven **Osborne**	*NCAR Section FT* Circumventricular Organs: A Blood-Brain Interface for Modulation of Cardiovascular and Immune Function and Fluid and Electrolyte Balance **Yao**	*WEH Section FT* New Animal Models and Their Utility for WEH Research **Palygin/Pochynyuk**
W194B	*GIL Section Symp* Posttranscriptional Regulation of Intestinal Homeostasis: Stem Cells to Cancer **Hamilton/Chatterji**	*GIL Section FT* Cytoskeletal Proteins in GI and Liver Diseases **Snider/Toivola**	*Cell Section Symp* Transport Proteins and Cellular Signaling in Choroid Plexus Epithelia **Blazer-Yost/Praetorius**
W192C	*Renal Section FT* Advances in Renal Physiology I **Ortiz**	*Careers Committee Symp* The Many Facets of a Teaching Career **Williams/Brandauer**	*PG Group FT* Novel Genetic Risk Factors and Early-Pregnancy Mechanisms Contributing to Preeclampsia **Grobe/Young**
W192A	*Pubs Committee Symp* Publishing 101: How to Get Your Work Published and Avoid Ethical Minefields **Sigmund/Scheman**	*ETG Group FT* Steve Hebert Lecture FT **Moeller**	*Hypoxia Group Symp* Intermittent Hypoxia: Molecular, Integrative, and Clinical Implications **Svatikova/Schreihofer**

Tuesday, April 25, 2017

Room	8:00-10:00 AM	10:30 AM-12:30 PM	3:15-5:15 PM
W375A		*President's Symp Series* Developmental Programming of Diseases **Alexander**	*Integrative Physio Symp Series* Exercise Intolerance in Metabolic, Pulmonary and Cardiovascular Diseases: Did We Forget the Brain? **Brassard/Bailey** 5:45 PM–7:30 PM APS Business Meeting
W190A	*PG Group and ASHG Symp* Atttaching Physiology to the Genome: Spotlight on Cardiovascular Genetics **Lettre/Joe**	10:30 AM–11:30 AM Resp Section Comroe Lecture **Wagner**	2:00 PM–3:00 PM CV Section Berne Lecture **Levine** 3:15 PM–4:15 PM GIL Section Davenport Lecture **Wallace**
W196B	*Resp Section Symp* Embracing the Heterogeneity of Lung Injury: A Bedside to Bench to Bedside Perspective **Dudek/Shaver**	*CV Section Symp* Mechanisms of Stiffening in Large and Small Vessels: From the Cytoskeleton to the Extracellular Matrix **Martinez-Lemus**	3:15 PM–4:15 PM CEP Section Krogh Lecture Supported by Novo Nordisk Foundation **Burggren**
W196C	*NCAR Section Symp* Brain Secrets Revealed by Optogenetics/Chemogenetics Approaches **Rahmouni/De Kloet**	*WEH Section Symp* Sodium Homeostasis in Metabolic Syndrome: Sorting out New and Old Players **Ecelbarger/Tiwari**	*Resp Section FT* Central Neuromodulation in the Control of Breathing **Garcia/Mitchell**
W196A	*CV Section FT* Novel Insights in Vascular Pathophysiology **Isakson/Bagher**	*CNS Section FT* New Insights Into the Role of Hypothalamic Peptides in the Control of Appetite and Energy Balance **Blevins**	*CNS Section Symp* Role of CNS Osmosensory Networks in the Regulation of Cardiovascular Function in Health and Disease **Stern/Pires da Silva**
W190B	*EEP Section Symp* Exercise Training versus Metformin for the Treatment of Cardiometabolic Diseases **Schrage/Jenkins**	*CV Section FT* Kaley Lecture FT: The Complications of Diabetes and the Role of Inflammation: Mechanisms and Therapeutic Opportunities **Schmidt**	*History Group Symp* Twenty Years of Physiological Genomics **Mattson**
W192B	*CEP Section Symp* Developmental Programming of Vertebrate Health and Disease by Hypoxia **Galli**	*EEP Section FT* Impact of Dairy Based Nutritional Interventions on Cardiometabolic Health **Alexander/Miller**	*E&M Section Symp* Central GLP-1 Receptors in the Regulation of Energy Homeostasis **Langhans**
W193	*CNS Section FT* Neuroglial Interactions in Health and Disease **Gulbransen**	*NCAR Section FT* Epigenetic Modifications and Autonomic Regulation **Sriramula**	*EEP Section FT* The Integrated Physiological Response to Heat Stress in Vulnerable Populations **Sawka**
W194B	*GIL Section FT* Autophagy: Driver of Microbiome Composition and Intestinal Homeostasis **Theiss**	*Cell Section FT* Mechanisms and Influences on Cell Signaling **Worrell**	*Cell Section Symp* Multi-omics to Study the Cellular Pathophysiology of Neurodegenerative Diseases **Pimplikar/Tsai**

Tuesday, April 25, 2017, *cont.*

Room	8:00-10:00 AM	10:30 AM-12:30 PM	3:15-5:15 PM
W192C	*Renal Section FT* Advances in Renal Physiology II **Inscho**	*Renal Section Symp* Regulation of Glomeruli Physiological Function: Podocytes and Beyond **Ilatovskaya/Reiser**	*Sex Group Symp* Sex Differences in Diabetes, Obesity and Blood Pressure Control **Brooks**
W192A	*Cell Section FT* Ion Channels and Transporters in Health and Disease **Hamilton/Rodrigues**	*Muscle Biology Group FT* The Hot Zone: Skeletal Muscle Changes Caused by Hyperthermia and Heat Stress **Selsby**	*ETG Group FT* Hans Ussing Lecture FT **Klein**
Location TBD		1:00 PM-3:00 PM 2014 Tang Prize in Biopharmaceutical Science **Charpentier**	1:00 PM–2:00 PM History of Physiology Group Lecture

Wednesday, April 26, 2017

Room	8:00-10:00 AM	10:30 AM-12:30 PM	2:30-4:30 PM
W375A	*Integrative Physiol Symp Series* The Impact of Exosomes on Muscle Atrophy, Hypertrophy and Myogenesis **Wang/Mitch**		4:45 PM–5:45 PM APS Nobel Prize Award Lecture **Ignarro**
W190A	*CV Section FT* Mitochondrial Physiology in Cardiovascular Disease **Calvert/Elrod**	*CV Section FT* Inflammation and Immunity and Heart Disease **Halade/Thorp**	*WEH Section Symp* Hydration and Vasopressin Beyond the Kidney **Armstrong**
W196B	*Physoc/APS Symp* The Modulation of Aging Through Altered Proteostasis **Miller**	*CV Section Symp* MiRNA Regulation of the Mitochondrion in Cardiovascular Disease **Hollander/Thapa**	*WEH Section Symp* Mechanisms of Hypertension Risk **De Miguel/Abais-Battad**
W196C	*Resp Section FT* Organelle Dysfunction in Pulmonary Vascular Disease: Mitochondria, Endoplasmic Reticulum, Lysosomes and Beyond **Shimoda/Ryan**	*EEP Section Symp* Blood Doping: Physiology, Pharmacology and Detection Challenges **Bowers**	*TransPhys Group Symp* Leptin Beyond Appetite Regulation **Singh/Cohen**
W196A	*Muscle Biology Group FT* Satellite Cells: External and Systemic Influences **Brooks/Dupont-Versteegden**	*Physoc Symp* Sexual Dimorphism, Plasticity and Genomic Diversity of the Paraventricular Nucleus **Samson/Yosten**	*TransPhys Group Symp* Vasodilation in Human Microvessels: From Bed to Bench and Back **Kleinbongard/Ait-Aissa**
W190B	*Resp Section Symp* Mechanisms Preserving Breathing Stability **Watters/Braegelmann**	*CEP Section FT* Comparative Perspectives on Hypoxia Signaling and Tolerance **Scott**	*EEP Section Symp* Patent Foramen Ovale: The Hole Story of Environmental and Exercise Physiology **Halliwill/Lovering**

Wednesday, April 26, 2017, *cont.*

Room	8:00-10:00 AM	10:30 AM-12:30 PM	2:30-4:30 PM
W192B	*Cell Section Symp* Glial Cell Development, Migration, and Pathophysiology **Gagnon/Sontheimer**	*CV Section FT* Cardiovascular Control after Spinal Cord Injury **Phillips/West**	*E&M Section Symp* Fighting the Fat: Lifestyle Approaches to Protect Against Adipose Tissue and Liver Dysfunction **Wright**
W193	*CNS Section Symp* Non-motor Dysfunctions in Parkinson's Disease **Travagli**	*NCAR Section FT* Neural Control of the Heart: New Tools, New Mechanisms **Chapleau**	*EEP Symp* Novel Imaging Technologies in Reproductive Physiology **Yamaleyeva/Lindsey**
W194B	*GIL Section Symp* Environmental, Microenvironmental and Nanoenvironmental Factors Which Drive Liver Disease Pathogenesis and Progression **Pritchard**	*Renal Section Symp* New Insights into the Regulation of Renal Blood Flow in Health and Disease **Polichnowski/Williams**	*Cell Section Flex Session* Omics Approaches to Understanding Muscle Biology **Burniston/Chen**
W192C	*E&M Section FT* Central and Peripheral Mechanisms Regulating Body Weight and Glucose Homeostasis **Zsombok**	*Trainee Committee Symp* Kick Start Your Funding: Looking Beyond NIH and NSF **Steiner/Streeter**	Pan-American Symp **TBD**
W192A	*EEP Section FT* Exploiting Environmental Stressors to Improve Health and Performance **Minson/Romero**	*TransPhys Group Symp* Peptides and Metabolites in Cardiovascular Disease **de Castro Brás/Halade**	*ETG Group FT* New Insights into Renal Transport: Application of Genetic Models **Rieg/Fenton**

Membership

New Regular Members

***transferred from student membership**

Gerard Ahern
Georgetown Univ., Washington, DC

Joanne S. Allard
Howard Univ. Coll. of Med., Washington, DC

William Arthur Altemeier
Univ. of Washington, Seattle, WA

José Lisandro Areta
Norwegian Sch. of Sport Sci., Oslo, Norway

Adriana Simionescu Bankston
Univ. of Louisville, Louisville, KY

Andrea Becchetti
Univ. of Milano Bicocca, Milano, Italy

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Matthew Cramer
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Marta Fiorotto
Baylor **Coll**. of Med., Houston, TX

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New Graduate Student Members

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Publications

Current Calls for Papers

Physiological Genomics

- Genetics of Metabolic Syndrome
- Gut Microbiota in Health and Disease
- Systems Biology and Polygenic Traits
- Single Cell Analysis
  Submission deadline: May 31, 2017

Journal of Neurophysiology

- Spinal Control of Motor Outputs
  Submission deadline: December 31, 2016
- Building Neural Circuits: Wiring and Experience
  Submission deadline: December 31, 2016
- Biology of Neuroengineering Interfaces
  Submission deadline: December 31, 2016
- Where Are You Going? The Neurobiology of Navigation.
  Submission deadline: June 30, 2017
- The Mouse Visual System
  Submission deadline: June 30, 2017
- Central Pattern Generators
  Submission deadline: June 30, 2017

Advances in Physiology Education

- Historical Perspectives and Living Histories

American Journal of Physiology – Cell Physiology

- Cell-to-Cell Communication and Signaling Pathways
  Submission deadline: December 31, 2016
- Cellular Mechanisms of Proteostasis
  NEW Deadline: December 31, 2016
- Gasotransmitters
  NEW Deadline: December 31, 2016
- Pathophysiology of Skeletal Muscle Loss
  Submission deadline: December 31, 2016
- Single Cell Physiology
  Submission deadline: December 31, 2016
- Stem Cells: Physiology and Microenvironment
  NEW Deadline: December 31, 2016

American Journal of Physiology – Endocrinology and Metabolism

- Role of Gut Microbiota and Gut-Brain and Gut-Liver Axes in Physiological Regulation of Inflammation, Energy Balance, and Metabolism
  Submission deadline: September 30, 2017
- Role of Fetal Programming and Epigenetic Regulation on the Development of Endocrine and Metabolic Alterations
  Submission deadline: September 30, 2017
- Browning and Beiging of Adipose Tissue, Its Role in the Regulation of Energy Homeostasis and as a Potential Target for Alleviating Metabolic Diseases
  Submission deadline: September 30, 2017
- Mechanisms of Effects on Sleep Disruption on Adipocyte/Obesity Metabolism and Their Relation to Other Metabolic Disease
  Submission deadline: September 30, 2017
- Metabolism and Signaling Functions of Amino Acids in the Regulation of Cell/Tissue Function in Health and Disease
  Submission deadline: September 30, 2017
- Role of Adipose Tissue Nutrient/Vitamin Metabolism in Physiological and Altered Metabolic Settings
  Submission deadline: September 30, 2017
- Endocannabinoids and Cannabinoid Receptors as Regulators of Endocrine Functions and Tissue Metabolism
  Submission deadline: September 30, 2017
- Role of Myokines and Adipokines and Other Cross-Talk Mechanisms of Regulation of Endocrine and Metabolic Functions
  Submission deadline: September 30, 2017
- Mitochondria Dysfunction in Aging and Metabolic Diseases
  Submission deadline: September 30, 2017

American Journal of Physiology – Gastrointestinal and Liver Physiology

- Gut-Brain Interactions and Brain Imaging
- Physiology of Gastrointestinal, Hepatic, and Pancreatic Cancer
- Metabolomics and Physiological Systems
- Systems Biology in Gastrointestinal Physiology and Diseases

American Journal of Physiology – Heart and Circulatory Physiology

- Advances in Cardiovascular Geroscience
  Submission deadline: November 30, 2016
- Heart Failure--Novel Therapeutic Pathways Emerging from Basic Science
  Submission deadline: February 15, 2017

American Journal of Physiology – Lung Cellular and Molecular Physiology

- Electronic Cigarettes: Not All Good News?
  Submission deadline: October 1, 2017
- Ion Channels and Transporters in Lung Function and Disease
- Age-Related Dysfunction in Lung Barrier Function in Health and Disease
- Real-Time Visualization of Lung Function: from Micro to Macro
  Submission deadline: January 2017
- Biomarkers in Lung Diseases: from Pathogenesis to Prediction to New Therapies
  Submission deadline: January 2017
- Translational Research in Acute Lung Injury and Pulmonary Fibrosis
  Submission deadline: January 2017

American Journal of Physiology – Regulatory, Integrative and Comparative Physiology

- Hypertensive Disorders of Pregnancy: Effects on Mother and Baby
  Submission deadline: December 1, 2016
- Exploiting Environmental Factors to Improve Health and Performance
  Submission deadline: March 31, 2017
- Model Systems for the Study of Integrative Physiology: The Rebirth of Translational Biology
  Submission deadline: May 1, 2017
- Oxygen Signaling
  Submission deadline: December 31, 2017

American Journal of Physiology – Renal Physiology

- Endothelin in Renal Physiology and Disease
  Submission deadline: June 30, 2017
- Imaging Techniques in Renal (Patho)physiology Research
  Submission deadline: June 30, 2017
- Inflammation and Inflammatory Mediators in Kidney
  Submission deadline: June 30, 2017
- Mechanism and Treatment of Renal Fibrosis and Treatment
  Submission deadline: June 30, 2017
- Transport Proteins as Regulators of Blood Pressure Homeostasis
  Submission deadline: June 30, 2017
- Renal Hemodynamics
  Submission deadline: June 30, 2017
- Gender and Hormones in Lower Urinary Tract Function
  Submission deadline: June 30, 2017

For a complete list of current Calls for Papers, visit the APS website.

Books Received

Lucky Choices: The Story of My Life in Sciences

Jens Christian Skou

Copenhagen, Denmark: UPress, 2016, 255 p., kr. 299.95 (hardback), $31.99 (kindle)

ISBN: 9788793060357

News From Distinguished Physiologists

Letter to Lois Heller

Samuel W. French writes: "Thank you for your very thoughtful note and happy 90th birthday greetings from the American Physiological Society. I want to write a brief letter regarding my adventures as an academic and practicing pathologist with physiological, nutritional, and cell biology components. To make sense of a disease process, many different approaches are necessary. This is especially true in the study of liver disease, which has been the focus of my research program.

"Studies on rodents models of alcoholic liver diseases

"I was first funded by NIH to study the lobular distribution of mitochondrial succinic dehydrogenase in the liver biopsies from patients suffering from alcoholic hepatitis in 1960. This was followed by nutritional studies on liver enzymes using rats fed a choline-deficient diet and alcohol to induce cirrhosis. The effect of alcohol plus thiamine, B6, pyridoxine, and a riboflavin-deficiency diet fed to rats where changes in liver enzyme activities occurred were evaluated. Then the activity levels in the liver were measured in rats fed ethanol. The results showed that alcohol ingestion altered these parameters and also caused liver hypoxic changes. Later on, we demonstrated in vivo that liver hypoxia was caused by ethanol. This was done by using Doppler and PO2 levels measured on the surface of the liver in vivo.

"Following this, we began to study the effect of alcohol on rat liver phospholipids in the liver cell membranes. Ethanol feeding induced mitochondrial fragility as a result of changes that developed in the phospholipid and fatty acid composition of the mitochondrial inner and outer membranes. Similar changes were noted in the brains of rats fed ethanol and during alcohol withdrawal.

"Studies of the brain adrenergic sensitivity during alcohol withdrawal in rats established the phenomenon of adrenergic super-sensitivity and subsensitivity while alcohol was being ingested. Subsensitivity was also observed in the liver during alcohol ingestion. Ethanol ingested in rats caused subsensitivity of plasma cAMP to glucagon injections.

"With Dr. Tsukamoto, we developed the experimental intragastric tube feeding rat model of alcoholic liver disease. Alcohol is fed 24 h/day continuously, but the blood alcohol levels cycle up and down over a 7- to 10-day period. At the peak blood alcohol levels (500 mg/% blood alcohol), a binge-drinking equivalent is achieved. When SAMe or betaine methyl donors are fed with alcohol in this model, the cycle is prevented and the blood alcohol levels remain at 100-200 mg%. This prevents liver pathology of fatty liver and liver fibrosis from developing by epigenetic means. Dietary saturated fat (tallow) prevented the fatty liver and fibrosis caused by alcohol. Unsaturated fat (linoleic acid) fed with alcohol to rats induced fatty liver caused by alcohol-fed rats. A diet high in unsaturated fatty acids, especially fish oil, produced the most advanced liver injury in the rats fed ethanol intragastrically.

"We published several highly cited papers on the role of CYP2E1 stabilization by alcohol as the cause of lipid peroxidation, serum protein adduct formation, hydroxyethyl free-radical formation, and protein oxidation as the cause of alcohol-induced liver oxidative stress.

"By feeding inhibitors of CYP2El such as chlormethiazole to rats using the intragastric tube feeding model, we showed that chlormethiazole prevented the pathology of alcoholic liver disease, indicating that oxidative stress caused by ethanol feeding is due to CYP2El stabilization.

"This stabilization of CYP2E1 by ethanol feeding is due to the reduced activity of the 26S proteasome. We further showed that the loss of 26S proteasome activity caused by alcohol feeding was the result of a shift from the 26S proteasome to the immunoproteasome. This shift was prevented by feeding SAMe or betaine, both methyl donors, which are required for the enzymatic conversion of norepinephrine to epinephrine. Epinephrine is 5-10 times more effective in increasing the metabolic rate and NAD^+ generation. NAD+ is required for alcohol dehydrogenase for the oxidation of ethanol and the increase in the rate of elimination of the blood alcohol levels in rats with high blood alcohol levels (1 > 500%mg). This provided the explanation as

to how SAMe or betaine feeding kept the blood alcohol levels low (100 mg%) in the intragastric tube feeding model of chronic alcoholic liver disease. This provided the rationale for therapy and prevention of alcoholic liver disease in man. Betaine was shown in vitro to be a very effective antioxidant for liver cells in cultures that were exposed to 100 mM ethanol. In vitro ethanol (100 mM)-treated liver cells transfected with CYP2E1 (hepatoblastoma-derived liver cells) developed Mallory-Denk-like bodies after 24-48 h of culture.

"The role played by catecholamines in the pathogenesis of the blood alcohol cycling phenomenon was established in the rat intragastric tube feeding model of chronic alcoholic hepatitis by measuring the blood catecholamine levels daily during the cycle.

"Using proteomics through a collaboration with a French investigator, we determined the molecular modifications of the 26S proteasome isolated from rats fed ethanol intragastrically. The epigenctics of proteasome inhibition in the livers of rats fed alcohol chronically was reported.

"We reported the role of the stellate cell as the source of liver fibrosis in rats fed a high fat-low protein diet with alcohol, and, with Dr. Tsukamoto, we developed an alcoholic pancreatitis model in rats. The tonnation of 4-hydroxynonenal formation plays a role in the oxidative stress caused by ethanol in the ethanol intragastric tube fed rat model. Microarrays were performed on the liver of these rats during the cycle. The results were compared with those derived from liver cell cultures exposed to ethanol (100 mM). At the peak of the cycle, histone acetyltransferase p300 expression increased. An increase in acetylation and a decrease in methylation of DNA methyl 5'cytosine occurred.

"Studies on human liver biopsies in cases of alcoholic hepatitis

"Regarding protein quality control in the liver of biopsies from patients with alcoholic hepatitis and mice fed DDC, we found that ufinylation and FAT10 methylation are downregulated. There is an increase in the activity of the complement systems in the liver of alcoholic hepatitis patients, which may contribute to the inflammation. Levels of metacaspase 1 and chaperones related to protein quality control are increased in alcoholic hepatitis. TLR3/4 signaling is mediated via the NFκB-CXCR4/7 pathway in human alcoholic hepatitis when MDBs are formed. The inflammasome forms in alcoholic hepatitis when MDBs form.

"We just published our studies on the expression of autophagocytosis genes in the alcoholic hepatitis biopsies. There is upregulation of many components of autophagocytosis in the liver biopsies compared with normal control livers. Upregulation of autophagocytosis should improve the elimination rate of misfolded and damaged proteins, which accumulate to form the MDB aggresomes. We found MDBs within the autophagosomes by electron microscopy.

"We found that IL-8 signaling is upregulated in alcoholic hepatitis and in DDC mice forming MOBs. Also, we found that the regulation of miR-340 and miR483-3p in alcoholic hepatitis and DDC-fed mice were changed compared with controls. This accounted for the downregulation of p53 and upregulation of BRCA1. RNA sequencing of the liver biopsies from patients with alcoholic hepatitis showed upregulation of many ingenuity pathways, most prominent being the BRCA1 and the G1/S pathways. Several cell cycle inhibitors were upregulated, accounting for the lack of liver cell regeneration in alcoholic hepatitis. Similar changes were found in the DDC-fed mice livers, forming MDBs using PCR. We published the data showing inhibitors of the cell cycle were upregulated in the alcoholic hepatitis liver biopsies, including p2l, p27, p15, ATM, and TGF-β.

"The type of macrophages that populate the liver sinusoids is changed in human alcoholic hepatitis liver biopsies from M1 to M2a, b, and c. These are anti-inflammatory phenotypes.

"Studies on the pathogenesis of Mallory-Denk body aggresomes formation

"Mallory-Denk body experimental models were established in mice fed grisiofulvin DDC or dieldrin. Using the DDC model, we established that FAT10, p62, p38, NF-κB, and ERK were required for Mallory-Denk body formation and that feeding mice betaine or SAMe (both arc methylating agents) with DDC prevented Mallory-Denk body formation as a result of epigenetic changes in the liver.

"Hepatocytes formed cytokeratin aggregates when rats were fed the proteasomc inhibitor PS341. We began to focus on the protein quality control system of the liver where alcohol abuse leads to liver cell aggresome formation due to the inhibition of the 26S proteasome by alcohol ingestion.

"The aggresomes formed are due to the accumulation of many proteins due to failure of the proteins to turn over. The aggresome is called a Mallory-Denk body (MDB). We first developed a method to isolate and purify MDBs and determined their protein composition, which was predominantly cytokeratins, heat shock proteins, and ubiquitin.

"The FAT10 promoter region was discovered to include an IFN-γ response element that responded to IFN-γ and TNF-α to increase FAT10, NF-κB, and the immunoproteasomal catalytic subunits expression (LMP 2 and 7). This explained the mechanism of the shift from the 26S proteasome to the immunoproteasome, which resulted in the loss of the 26S proteasome activity in response to DDC induction of MDBs.

"We showed that RNA interference of valosin-containing protein (vcp/p97) increases MDB formation, most likely by inhibiting autophagocytosis.

"The role of heat shock proteins in MDB formation in drug-primed (DDC) mouse liver was shown by heating the mice on a rafl on hot water (43°C for one-half hour). Heat shock proteins became elevated in the blood, and MDBs began to form in 2 days and were numerous by 6 days post-heat treatment.

"The role of the frameshift mutant of ubiquitin in the MDB formation was established by involving the ubiquitin-proteasome pathway.

"We found that SAMe prevented the upregulation of toll-like receptor signaling, which prevents mice fed DDC from developing MDBs. MDBs formed when EZH2/H3K 27me3 failed to methylate DNA in the nuclei of human and mice liver cells.

"Using infrared spectroscopy examination of isolated and purified fractions of MDBs, we characterized the protein conformation as showing an increase in the formation of beta sheets.

"Using autoradiography electron microscopy, we determined the distribution of newly synthesized cytokeratins in the MDB filaments and the normal intermediate filaments by using radiolabeled methionine uptake in the extracted liver cells from DDC-refed mice. The ^{35}S-labeled proteins went to the MDB filaments rather than to normal intermediate filaments in the liver cells, which explains where the MDB-forming cells lack cytokeratins in their cytoplasm. They normally have cytokeratins in their cytoplasm.

"DDC-fed mice-induced MDBs forming hepatocytes expressed a preencoplastic phenotype. The MDBs arc formed by the hepatomas that develop beginning 9 months after DDC is withdrawn.

"Miscellaneous reports of interest

"Together with George Perry, using immunogold labeled ubiquitin staining of extracted liver intermediate filament preparations and electron microscopic visualization, we showed that ubiquitin was bound to the cytokeratin filaments. We also showed the connections of the liver cell intermediate filaments to the nuclear membrane envelope and the plasma membrane. We showed the role of cytokeratin intermediate filaments in the liver in the trans-hepatic transport and canalicular secretion.

"We identified the rings of actin that follow the bile canaliculus, which propels the bile and the bile pericanicular sheath of intermediate filaments that surrounds the bile canaliculus.

"We wrote the second paper to report the importance of nonalcoholic fatty hepatitis (now called NASH) (reported in 1989).

"To answer the question regarding the pattern of metastasis for the different types of malignant tumors derived from 4,000 autopsies performed, where accurate records were kept and analyzed, we published the results to help clinicians to understand where they can determine the source of unknown primary malignancies. This article is highly cited." ●

Positions Available

Assistant Professor: The Department of Integrative Biology at the University of California, Berkeley, is embarking on a broad search for a scientist to fill a tenure-track position (Assistant Professor) in Vertebrate Physiology. The expected start date is July 1, 2017. We welcome applicants who study any vertebrate taxon (including humans) and who can relate their research to human physiology. We seek a colleague to join a department with strong multidisciplinary emphases, and to complement and bridge strongly represented fields such as human health sciences, ecology, comparative biomechanics, endocrinology, genomics, and evolutionary biology. Candidates will also be expected to have a strong interest in both undergraduate and graduate teaching and to contribute to instruction in core courses of vertebrate physiology as well as in their specific area of expertise. *Required minimum basic qualifications:* Applicants with a PhD or equivalent degree and an exceptional research record in any area of vertebrate physiology are encouraged to apply. Specific areas of interest include (but are not limited to) the physiology of behavior, interactions between humans (and other primates) and pathogens, microbiome effects on organismal physiology and health, comparative metabolic physiology, systems neuroscience in an evolutionary or ecological context, mechanisms of physiological adaptation, and mechanisms of aging. The Department is interested in candidates who will contribute to diversity and equal opportunity in higher education through their teaching, research, and service. A PhD or equivalent degree is required by date of application. *Additional qualifications:* Demonstrated excellence, originality, and productivity in research, and interest in undergraduate and graduate teaching as well as public outreach. To apply, please go to the following link: *http://apptrkr.com/860196*. Please direct all questions to *ib_ap_assist@berkeley.edu*. Applicants should include the following required documents: cover letter; curriculum vitae; brief description of research accomplishments; statement of research objectives; statement of teaching interests; list of publications; statement of contributions to diversity: statement addressing past and/or potential contributions to diversity through research, teaching, and/or service; 3 significant publications. Applicants should arrange to have three letters of reference submitted online. All letters will be treated as confidential per University of California policy and California state law. Please refer potential referees, including when letters are produced via a third party (i.e., dossier service or career center), to the UC Berkeley statement of confidentiality (*http://apo.berkeley.edu/evalltr.html*) before submitting their letters. The Department of Integrative Biology, UC Berkeley, is committed to addressing the family needs of faculty, including dual career couples and single parents. For information about potential relocation to Berkeley or career needs of accompanying partners and spouses, please visit *http://ofew.berkeley.edu/new-faculty*. The University of California is an affirmative action/equal opportunity employer. All qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, disability, age, or protected veteran status. For the complete University of California nondiscrimination and affirmative action policy, see *http://policy.ucop.edu/doc/4000376/NondiscrimAffirmAct*.

Associate/Full Professor: We are seeking an experienced investigator and teacher of medical physiology or pharmacology for the CUNY School of Medicine at the City College of New York for a tenured/tenure-track position in the Department of Physiology, Pharmacology, and Neuroscience. *Responsibilities/duties:* The candidate must be an independent investigator with experience in medical school teaching of physiology and pharmacology. Preference will be given to investigators who have a strong record of funded scholarly research and publications in molecular or cellular physiology and/or pharmacology with teaching experience in systemic physiology and/or pharmacology. The Department is responsible for teaching Medical Physiology, Pharmacology, and Neuroscience to medical, graduate, and physician assistant students. A competitive salary commensurate with experience and a startup package will be offered. Applications will be reviewed as they are received until the position is filled. Requirements: MD and/or PhD with a minimum of 4 years of postdoctoral research and teaching experience in medical physiology or pharmacology. The School of Medicine and the City College of New York highly value diversity. Candidates of all backgrounds and genders are encouraged to apply. *To apply*, please view the original posting at *http://www.cuny.edu/employment/jobsearch.html* and search for the Job ID (15212) or title. Please upload a

current curriculum vitae, statement of research plan, and contact information of 3 potential references.

Chair: The Department of Biology (*www.twu.edu/biology*) at Texas Woman's University (*www.twu.edu*) invites applications and nominations for the position of Department Chair. The starting date is flexible. Applicants must have a PhD in Molecular or Cellular Biology or a closely related discipline. All applicants should currently hold the rank of Professor or, if applying as an Associate Professor, should meet the qualifications for promotion to Professor at TWU. Applicants need a record of research, external funding, teaching, and service appropriate for a tenured appointment at the rank of Professor. Preference will be given to candidates with previous chair or other related administrative experience in a doctoral-level training program. The successful candidate will have appreciation for a multidisciplinary environment and a commitment to excellence in undergraduate and graduate education. The Chair will teach in her/his area of specialization and maintain an active research program. The Chair position is a 12-month administrative appointment with responsibilities for overseeing programs leading to the Bachelor of Science in Biology (including a Biology degree with an emphasis in research), the Bachelor of Science in Medical Technology, the Master of Science in Biology degree, and the PhD in Molecular Biology. The Department of Biology currently has 10 full-time faculty, 3 laboratory coordinators, approximately 475 Biology undergraduates, 30 Master's degree and 25 PhD degree-seeking students (*www.twu.edu/biology*). Review of applications will begin immediately and will continue until the position is filled. To apply: Please submit a cover letter, curriculum vitae, copy of graduate transcripts, statement of administrative philosophy, research agenda, and names with e-mail addresses and phone numbers of three references via e-mail to *Facultyjobs@twu.edu* (e-mail header or subject line must include job title and job code 16AS-BIO05). Confidential inquiries may be sent to Dr. Claire Sahlin, Associate Dean, at *csahlin@twu.edu*. Texas Woman's University, an AA/EEO/VEVRAA employer, supports diversity. Men and women, and members of all racial/ethnic groups and abilities, are encouraged to apply. All offers of employment will be contingent on the candidate's ability to provide documents that establish proof of identity and eligibility to work in the U.S. All positions at Texas Woman's University are deemed security-sensitive and require background checks and verification of academic credentials.

Faculty Position: The Department of Molecular and Cell Biology at the University of California, Berkeley, is soliciting applications for a new faculty position in Physiology. This position is open at any level (tenure-track or tenured). Rank will be determined by qualifications and experience. The expected start date is July 1, 2017. We seek applicants studying how communication within and between cells supports physiological function and enables cells, tissues, and organisms to respond to changes in their environment and maintain homeostasis. Our preference is for applicants who use quantitative molecular and cellular techniques, as well as tissue and animal models, to uncover the mechanism of normal function as well as dysfunction that may lead to human disease. We seek candidates who have demonstrated excellence, originality, and productivity in research. A PhD and/or MD or equivalent degree in Molecular Biology or a related field is required at the time of application. Preferred qualifications (by start date) include interest and experience in undergraduate and graduate teaching in Physiology. *Application procedure:* Applicants who are not currently tenured at an institute of higher education/are not a senior independent investigator must complete an online application via the following link: *http://apptrkr.com/866406*. You must arrange to have three letters of reference submitted online. All letters will be treated as confidential per University of California policy and California state law. Please refer potential referees, including when letters are produced via a third party (i.e., dossier service or career center), to the UC Berkeley statement of confidentiality (*http://apo.berkeley.edu/evalltr.html*) before submitting their letters. Applicants who are currently tenured at an institute of higher education or are an independent investigator must complete an online application via the following link: *http://apptrkr.com/866406*. You must provide contact information for 3-5 referees. We will only contact your referees if you are a finalist for the position, and we will seek your permission before doing so. Applicants should include cover letter; curriculum vitae; summary of major research accomplishments (approx. 250 words); publications list; a summary of approximately 1,500 words of the applicant's ongoing and planned research program,

plus up to one page of figures and a list of essential publication citations); three significant publications (in addition to your publications, provide statements for each that begin with the manuscript title and author list and then summarizes, in approximately 300 words, the significance of the selected publication); a statement of teaching (summary of teaching interests, as well as any prior teaching and mentoring experience, of approximately 500 words); statement of contributions to diversity. The final deadline for applications is October 31, 2016. To receive full consideration, please submit a completed application by this date. Please direct questions to *MCBsearch.committee@berkeley.edu*. The Department of Molecular and Cell Biology, UC Berkeley, is committed to addressing the family needs of faculty, including dual career couples and single parents. The Department is interested in candidates who will contribute to diversity and equal opportunity in higher education through their teaching, research, and service. For information about potential relocation to Berkeley or career needs of accompanying partners and spouses, please visit *http://ofew.berkeley.edu/new-faculty*. The University of California is an equal opportunity / affirmative action employer. All qualified applicants will receive consideration for employment without regard to race, color, religion, sex, sexual orientation, gender identity, national origin, disability, age, or protected veteran status. For the complete University of California nondiscrimination and affirmative action policy, see *http://policy.ucop.edu/doc/4000376/NondiscrimAffirmAct*.

Industrial Position: University of Vermont Medical Center, Vermont's academic medical center, seeks an Exercise Physiologist Sr. The University of Vermont Medical Center is located in Burlington, Vermont, a small vibrant city situated on the shore of beautiful Lake Champlain. The Champlain Valley region offers an incredible range of educational, entertainment, and recreational opportunities. *Position summary:* The Exercise Physiologist Sr. demonstrates advanced knowledge pertaining to the design, evaluation, and standards of exercise programs for patients who qualify or are undergoing cardiac rehabilitation; functions as an essential member of a multidisciplinary team directing, coordinating, and providing special expertise to staff, patients, and families participating in the Cardiac Rehabilitation Program; demonstrates leadership skills through mentoring, orienting new staff, providing educational programs for staff, involvement in FAHC committees and / or professional organization. The Exercise Physiologist Sr. functions within the framework of the University of Vermont Medical Center standards of patient care. Please note: This is a part time (32 hr / wk) day shift with staggered hours. One, 2-hour Saturday shift (7:30 AM to 9:30 AM), with a rotation of every 9-10 weeks. *Education:* Master's degree in exercise physiology from an accredited school. *Experience:* Experience in a rehabilitation setting preferred. *The Organization:* University of Vermont Medical Center has a medical staff of nearly 800 physicians, including more than 500 University of Vermont Medical Group physicians (jointly employed by University of Vermont Medical Center and the UVM College of Medicine) and approximately 200 community-based physicians. For more information about University of Vermont Medical Center, find us online at *http://www.uvmhealth.org/medcenter* or on our Facebook, Twitter, YouTube, and blog sites. Apply online at: *http://bit.ly/2c3W5hn*. University of Vermont Medical Center offers relocation assistance for this position. University of Vermont Medical Center proudly offers a non-smoking work environment. We are an equal opportunity / affirmative action employer. Applicants will receive consideration for employment without regard to race, color, religion, sex, national origin, disability, or protected veteran status.

Lecturer: School of Exercise and Rehabilitation Sciences at University of Toledo is looking for an assistant lecturer, specializing in exercise physiology, clinical exercise physiology, or strength and conditioning. The non-tenure-track position is for 9-months at the rank of assistant lecturer with a salary of $45,000. Position starts in August 2017. *Required qualifications:* Master's degree in Exercise Science or closely related program; clinical certification(s), credentials from NSCA and / or ACSM; previous instructional experience. *Preferred qualifications:* terminal degree, earned doctorate in Exercise Science or closely related program; emphasis in one or more of the following areas: Exercise Physiology, Clinical Exercise Physiology, Biomechanics, Neuromuscular Control; 1-2 years of collegiate level teaching experience, with innovative teaching technologies, including development of online or hybrid courses; knowledge of, or experience in, working with diverse populations; professional knowledge in clinical science. *Responsibilities:* Teach

undergraduate courses in one of the following or related areas: Strength and Conditioning, Exercise Testing and Programming, Biomechanics, Neuromuscular Control for the HPFP concentration; collaborate with faculty and students in the School of Exercise and Rehabilitation Sciences, as well as other appropriate schools/departments across the college and university; participate in school, college, and university service. Exercise Science is a program in the School of Exercise and Rehabilitation Sciences, which is one of four schools in the College of Health and Human Services. Approximately 500 students are actively pursuing majors at the BS, MS, and PhD levels. Undergraduate students may choose to major in Exercise Science, Recreation Therapy, or Respiratory Care. Specializations include Human Performance and Fitness Promotion and Pre-Health Care (pre-PT, pre-OT, pre-PA, and pre-med). Instruction in microbiology, pathophysiology, and anatomy and physiology is also provided to many non-majors. Areas of emphasis at the MS level include Athletic Training 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. Additional information can be found online at *http://www.utoledo.edu/hhs/schools/*. *Application procedure:* To apply, go to *https://jobs.utoledo.edu/* to submit the following required items: *1)* letter of application; *2)* complete, current curriculum vitae; *3)* statement of teaching. Letters of recommendation are to be provided upon request. Any questions should be directed to: Elyce Ervin, Search Committee Co-Chair, School of Exercise and Rehabilitation Sciences, Mail Stop 119, College of Health and Human Services, University of Toledo, Toledo, OH 43606; e-mail: *elyce.ervin@utoledo.edu*; office phone: (419) 530-2457; fax: (419) 530-2477. *Deadline:* Review of applicants will begin immediately and continue until the position has been filled. *Appointment date:* The position will begin August 2017. Although the position will remain open until filled, applicants are encouraged to submit their materials by December 2, 2016 for full consideration. The University of Toledo is an equal access, equal opportunity, affirmative action, Title IX employer, committed to excellence through diversity.

Postdoctoral Position: The laboratory of Dr. Sandra Davidge at the University of Alberta/Women and Children's Health Research Institute has a Postdoctoral Fellow position available immediately. Dr. Davidge is a Tier 1 Canada Research Chair in Maternal and Perinatal Cardiovascular Health. The focus of the Davidge laboratory is to understand the mechanisms of cardiovascular dysfunction in complicated pregnancies for both the mother and her offspring. The laboratory has three complementary CIHR-funded programs that include the investigation of the causes and consequences of preeclampsia, determining the consequences of maternal aging on pregnancy outcomes, and addressing the mechanisms of cardiovascular pathophysiology in offspring born from complicated pregnancies [a field termed the Developmental Origins of Health and Disease (DOHaD)]. Applicants will be required to have a PhD or equivalent qualification. Applicants will be expected to be motivated and self-driven, and should work well both independently and as part of a team. Collaboration and interactions with other investigators will be encouraged. The successful applicant will be expected to participate in all aspects of the research project, including experimental design, data collection, data analysis, preparation of graphs and figures, and preparation of manuscripts. Strong written and oral communication skills are highly desired. Stipends and benefits paid to postdoctoral fellows shall be in accordance with University of Alberta policies (*http://www.postdoc.ualberta.ca*) and funding agency guidelines. It is expected that the successful candidate will, with assistance, seek to procure external funding through any available opportunities. *To apply:* Interested candidates should submit their CV to *resadmin@ualberta.ca* and include a brief cover letter summarizing their research interests and career goals, along with the names of at least 2 references. *Closing date:* position open until filled. All qualified candidates are encouraged to apply. We thank all applicants for their time and effort, but only those selected for an interview will be contacted. Applicants may be considered for future vacancies. The University of Alberta offers appointments on the basis of merit. We are committed to the principle of equity in employment. We welcome diversity and encourage applications from all qualified women and men, including persons with disabilities, members of visible minorities, and Aboriginal persons.

Postdoctoral Position: A postdoctoral position is available in the laboratory of Dr. Douglas Tilley at the Lewis Katz School of Medicine at Temple University.

Dr. Tilley's lab is housed within the Center for Translational Medicine and focuses on novel aspects of GPCR regulation of cardiac function and remodeling during heart failure or following acute injury. The postdoctoral fellow will be involved in designing and coordinating molecular biology and in vivo experiments with the goal of elucidating the impact of receptor signaling in the heart. As such, they would be responsible for the evaluation of cardiac function in mouse models of heart failure with follow-up biochemical, histological, and morphological assays, and would perform a wide array of experimental techniques including (but not limited to) the synthesis and use of viral constructs, fluorescent/FRET microscopy, histology, RT-PCR arrays, FACS, immunoprecipitation, and immunoblotting. The postdoctoral fellow will be involved in data analysis, preparation of manuscripts for publication, and presentation of their research at scientific meetings. Some experience in molecular pharmacology and/or in vivo models of heart failure is desired, and eligible candidates will have a PhD or MD/PhD by the time they commence postdoctoral work in Dr. Tilley's lab. Please send your updated CV and letter of interest to Dr. Tilley at *douglas.tilley@temple.edu*.

Postdoctoral Position: The Medical College of Wisconsin-Milwaukee is looking to fill a postdoctoral position in mitochondrial and vascular biology of aging cardiovascular disease. This is a 2-year, NIH-funded position with possibility of extension. The position is a joint hire between the laboratory of Andreas Beyer and David Gutterman as part of a collaborative environment in the cardiovascular center. The lab focuses on cardiovascular disease with a focus on microvascular function. A multidisciplinary approach combining, genetics, biochemistry, and molecular biology are used to evaluate the mechanism of how coronary microvascular function contributes to the development of CAD and other heart conditions. We study several relevant pathways that contribute to the regulation of vascular tone, dilator capacity, and metabolism, including autophagy, telomerase biology, mitochondrial signaling/function mechanosemsetive miRNAs. Multiple projects are available to be studied. We seek a highly motivated, ambitious, and talented scientist to join an enthusiastic and collaborative team of outstanding scientists. The successful applicant will hold a PhD or equivalent in a relevant research area such as biology, molecular biology, genetics, or physiology and have a strong track record of, and potential for, success. The applicant should have a keen interest in the mitochondrial effects on aging and/or cardiovascular disease and excellent written and oral communication skills. To apply, send CV and names of references (preferably by e-mail) to: Andreas M. Beyer PhD, Medical College of Wisconsin, Assistant Professor of Medicine and Physiology, HRC H4875, 8701 Watertown Plank Rd., Milwaukee, WI 53226. ●

Meetings & Congresses

2016

November 2-4

APS Intersociety Meeting: The Integrative Biology of Exercise VII, Phoenix, Arizonia. *Information:* internet: *http://www.the-aps.org/mm/Conferences/APS-Conferences/2016-Conferences/Exercise*; #IBExercise7

2017

February 11-15

Biophysical Society 61st Annual Meeting, New Orleans, LA. *Information:* Internet: *http://www.biophysics.org/2017meeting/Home/tabid/6672/Default.aspx*

April 22-26

2017 Experimental Biology, Chicago, IL. *Information:* Internet: *http://apsebmeeting.org/*

May 27-June 1

International Neuromodulation Society 13th World Congress, Edinburgh, Scotland. *Information*: internet: *http://www.neuromodulation.com/ins-congress*

August 1-5

IUPS 38th World Congress: Rhythms of Life, Rio de Janeiro, Brazil. *Information:* Internet: *http://iups2017.com/*

August 30-September 2, 2017

APS Conference: Physiological Bioenergetics: Mitochondria from Bench to Bedside, San Diego, CA. *Information:* Internet: *http://www.the-aps.org/mm/Conferences/APS-Conferences/2017-Conferences/Bioenergetics; #Bioenergetics17*

Meetings and Conferences

Experimental Biology 2017
April 22-26, 2017 • Chicago, Illinois

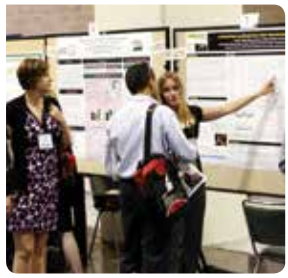
APS Conference: Cardiovascular Aging, New Frontiers and Old Friends
August 11–14, 2017 • Westminster, Colorado

APS Conference: Physiological Bioenergetics: Mitochondria from Bench to Bedside
August 30–September 2, 2017 • San Diego, California

APS is also participating in the following meeting

IUPS 38th World Congress: Rhythms of Life
August 1–5, 2017 • Rio de Janeiro, Brazil





For more information on APS meetings, please visit:
the-aps.org/conferences

APS Members receive discounted registration to EB and APS Conferences!
The American Physiological Society usually holds one or more specialty conferences each year. In addition, APS joins with other societies to sponsor Intersociety Meetings as interest warrants. Please send an email to meetings@the-aps.org for questions or to propose APS Conference ideas.

American Physiological Society • 9650 Rockville Pike • Bethesda, MD 20814
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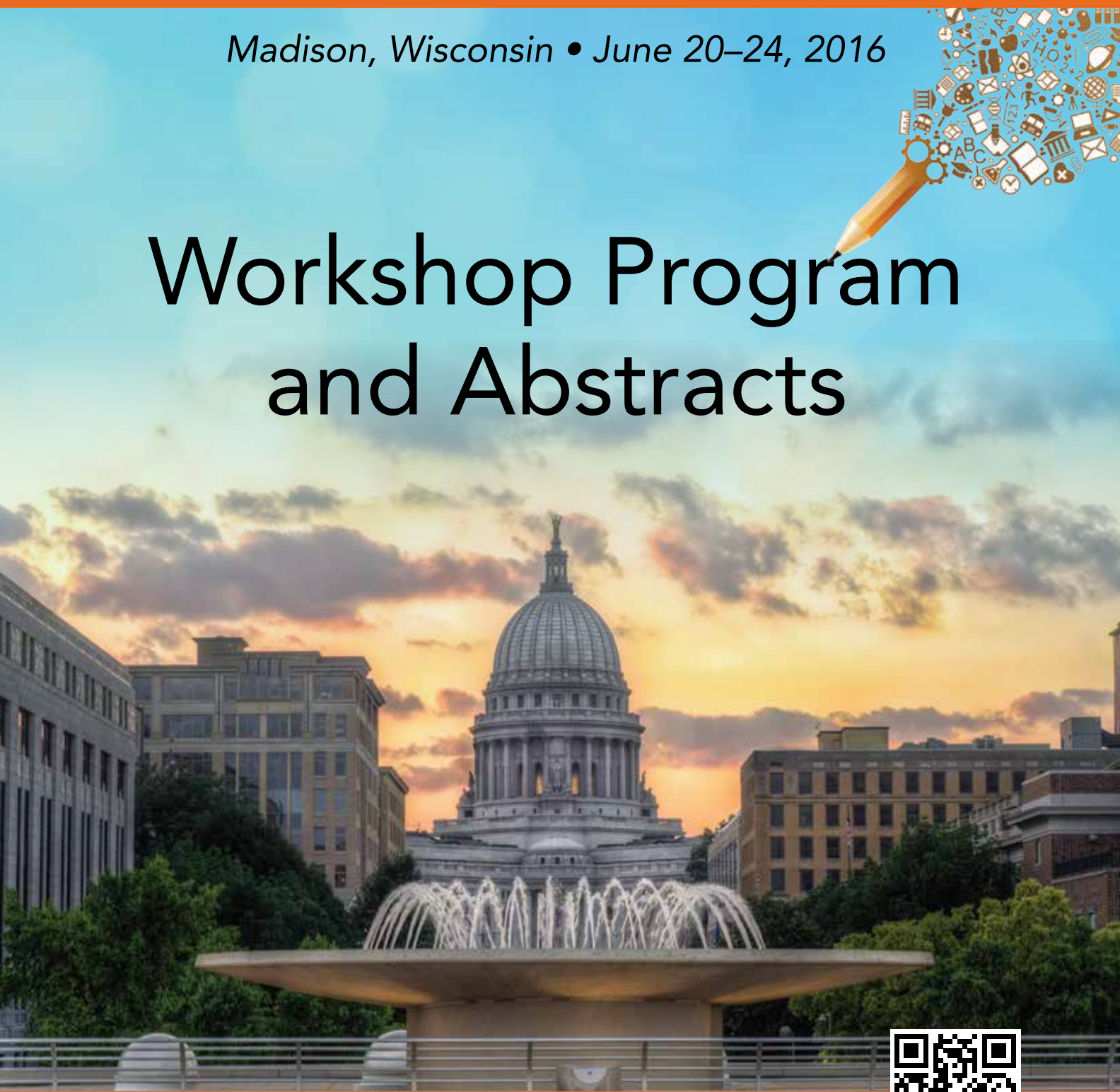
aps Workshop:
Institute on Teaching and Learning

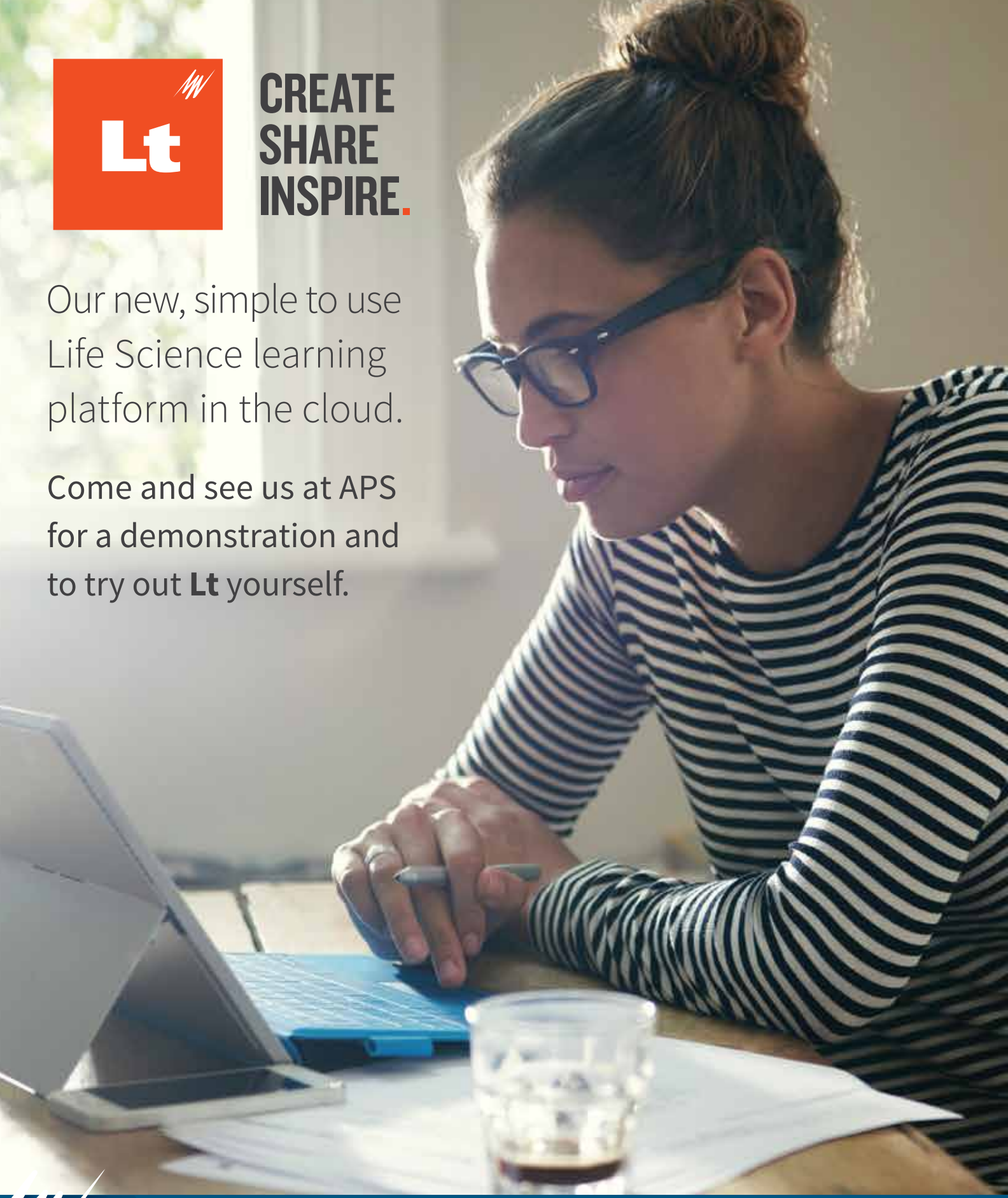
Madison, Wisconsin • June 20–24, 2016



Workshop Program and Abstracts

the-aps.org/ITL

#ITLPhysiology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APS Institute on Teaching and Learning

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Acknowledgements

The Workshop Organizers and The American Physiological Society gratefully recognize the generous financial support from the following:

ADInstruments, Inc.

2016 APS Teaching Workshop: The APS Institute on Teaching and Learning
Week-at-a-Glance Schedule

	Monday, June 20, 2016	Tuesday, June 21, 2016	Wednesday, June 22, 2016	Thursday, June 23, 2016	Friday, June 24, 2016
7:00 AM	Registration for Ultrasound Workshop (7:00 AM-4:00 PM)	Registration for ITL (7:00 AM-5:00 PM)	Registration for ITL (7:00 AM-5:00 PM)	Registration for ITL (7:00 AM-5:00 PM)	Registration for ITL (7:00 AM-12:00 Noon)
7:30-8:30 AM		Breakfast	Breakfast	Breakfast	Breakfast
8:45-9:00 AM		Daily Orientation and Announcements	Daily Orientation and Announcements	Daily Orientation and Announcements	Daily Orientation and Announcements
9:00-10:00 AM		Plenary Lecture I: **Defining the Role of the Instructor in an Active Learning Environment**, Speaker: Harold Modell	Plenary Lecture III: **A New Paradigm for Student Learners**, Speaker: Terry Doyle	Plenary Lecture V: **Supporting Faculty: Resources from Professional Societies and On-line Communities**, Speaker: Marsha Lakes Matyas	Plenary Lecture VII: **Educational Leadership: Benefits of Stepping Outside the Classroom**, Speaker: Tom Pressley
10:00-10:30 AM		Networking Break	Networking Break	Networking Break	Networking Break
10:30 AM-12:00 Noon		Concurrent Workshop I: **Using Recorded Lectures for Flipped Classrooms**, Speaker: Chaya Gopalan	Concurrent Workshop I: **Tips for Team-based Learning**, Speakers: Jon Kibble, and Lisa Barkley	Concurrent Workshop I: **Teaching Physiological Concepts by Enhancing Student Visual Literacy**, Speaker: Katie Johnson	Plenary Lecture VIII: **Implementing and Managing Change**, Speaker: Barbara Goodman
10:30 AM-12:00 Noon		Concurrent Workshop II: **Strategies for Creating a Culture of Academic Integrity**, Speaker: Kim Henige	Concurrent Workshop II: **Student Role in Learning: What is the Goal and How Do We Get There?** Speaker: Beth Beason-Abmayr	Concurrent Workshop II: **Statistics in Educational Scholarship**, Speaker: Doug Curran-Everett	
10:30 AM-12:00 Noon		Concurrent Workshop III: **Integrating Discovery-based Research into the Undergraduate Curriculum**, Jay Labov, *and Catherine Middlecamp*	Concurrent Workshop III: **Educational Scholarship: A Step by Step Guide to Implement and Publish Your Classroom Research**, Speaker: Valerie O'Loughlin	Concurrent Workshop III: **Instructors' Understanding of Student's Misconceptions can Improve Meaningful Learning**, Speaker: Ann Wright	
12:00 Noon-1:00 PM		Lunch	Lunch	Lunch	Lunch
1:00-2:00 PM		Plenary Lecture II: **Team-based Learning in a Large Enrollment Class**, Speaker: Jon Kibble	Concurrent Plenary Lecture IVa: **The Changing USMLE and NBME Medical Scool Services**, Speakers: Steven Haist & Agata Butler	Concurrent Plenary Lecture VIa: **Central Role of Physiology in the Professional Curriculum**, Speaker: Rob Carroll	
1:00-2:00 PM			Concurrent Plenary Lecture IVb: **Funding for Educational Research and Curriculum Change,** Facilitator: Barbara Goodman	Concurrent Plenary Lecture VIb: **The Pipeline of Physiology Courses in Community Colleges,** Speakers: Jenny McFarland, and Pamela Pape-Lindstrom	
2:00-2:30 PM		Networking Break	Networking Break	Networking Break	
2:30-4:00 PM		Concurrent Workshop IV: **Interactive Teaching and Learning**, Speaker Dee Silverthorn	Concurrent Workshop IV: **Using Social Media to Enhance Student Learning**, Speaker: Patricia Halpin	Concurrent Workshop IV: **Physiology Majors Interest Group**, Speaker: Erica Wehrwein	
2:30-4:00 PM		Concurrent Workshop V: **Using PULSE rubrics to Assess Departmental Transformation to Student-centered Learning**, Speaker: Pamela Pape-Lindstrom	Concurrent Workshop V: **Using Conceptual Frameworks in Teaching and Learning Physiology**, Speaker: Jenny McFarland	Concurrent Workshop V: **Facilitating Small Group Discussion-Basic,** Speakers: Sydella Blatch and Carol Schmidhauser	
2:30-4:00 PM		Concurrent Workshop VI: **Accreditation (Role of Course/Block Directors)**, Speaker: Mike Levitzky	Concurrent Workshop VI: **Preparing Students for Board Exams**, Speaker: Rob Carroll	Concurrent Workshop VI: **Facilitating Small Group Discussion-Advanced,** Speaker: Betsy Jones	
3:00-8:00 PM	**ITL Registration Opens**				
4:00-5:30 PM		**Best Practices in Professional School Physiology Poster Session**	**Innovative Curricula Throughout Educational Levels Poster Session**	**Best Practices in Undergraduate Physiology Poster Session**	
5:30-7:00 PM	Group Dinner (5:30-6:30 PM)	Group Dinner	Dinner on own	Group Dinner	
6:30-8:00 PM	Welcome and Opening Reception				
7:00-8:30 PM		Collaborations and Special Topics	Collaborations and Special Topics	Collaborations and Special Topics	
8:00-9:00 PM	Keynote Lecture: **The Changing National Landscape in Undergraduate STEM Education: Connecting the Dots**, Speaker: Jay Labov				

Location:

The 2016 APS Workshop: The APS Institute on Teaching and Learning will be held June 20—24, 2016 at the Madison Concourse Hotel located at: 1 W. Dayton St., Madison, Wisconsin, 53703, telephone (608) 294-3022.

Onsite Registration Hours:

Monday, June 20……..……3:00—8:30 PM
Tuesday, June 21…….....7:00 AM—5:00 PM
Wednesday, June 22…….....7:00 AM—5:00 PM
Thursday, June 23……7:00 AM—5:00 PM
Friday, June 27……..7:00—11:00 AM

On-Site Registration Fees:

APS Member	$750
APS Retired Member	$550
Invited Presenter	$750
Nonmember	$950
APS Postdoctoral Member	$600
Postdoctoral Nonmember	$700
APS Student Member	$550
Student Nonmember	$600

Payment Information:

Registrants may pay by institutional or personal check, traveler's check, MasterCard, VISA or American Express or in United States Dollars. 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 bio-medical 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 onsite 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 four years** of this workshop, 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.**

Included in your Registration:

Your registration to this workshop includes entry into all scientific sessions, program book, opening reception, poster sessions, networking socials, and meals. **There are no substitutions or refunds.** You must pay the registration fee regardless of whether you are not able to stay for the entire workshop or partake in any of the meals during the work-shop program. Guests of attendees are not permitted in the scientific sessions.

Press Registration:

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.

Meal Service:

Meals will be served promptly in the Madison Ballroom according to the following schedule:

Breakfast ……...7:30—8:30 AM
Lunch ……12:00 Noon—1:00 PM
Dinner*: ……....5:30—7:00 PM
**Except Wednesday, June 22. Dinner is on your own.*

Photograph/Video Recording:

The photographing and/or the video recording of any the workshop sessions for personal or private use is strictly prohibited.

Code of Conduct:

APS is committed to providing a friendly, safe, and welcoming environment for all, regardless of gender, sexual orientation, disability, race, ethnicity, religion, national origin, or other protected characteristics. We expect all attendees, media, speakers, volunteers, organizers, venue staff, guests, and exhibitors to help us ensure a safe and positive workshop experience for everyone. Alert the APS Registration Desk if you notice a dangerous situation, someone in distress, or violations of this Code of Conduct.

Program Objective:

The purpose of this workshop and the APS Institute on Teaching and Learning (ITL) is to engage educators (community/4-year college through professional school) in interactive sessions on best practices in teaching, learning, and assessment. Whether you are an experienced educator or new to teaching, it will challenge you to gain skills in designing and implementing educational research in your classroom and in learning how to share your findings with colleagues.

Target Audience:

The intended audience for this workshop includes educators teaching in community colleges, 4-year colleges, as well as those who are experienced educators or new to teaching.

Did you register early?
The drawing for the iPad Pro will be held on Thursday, June 23 at 8:45 AM during the Daily Orientation and Announcements

MONDAY, JUNE 20, 2016

1.0 KEYNOTE LECTURE
Mon., 8:00—9:00 PM, Madison Ballroom.

8:00 PM **1.1** The Changing National Landscape in Undergraduate STEM Education: Connecting the Dots. **Jay Labov,** *Natl. Acd. of Sci.*

TUESDAY, JUNE 21, 2016

2.0 DAILY ORIENTATION AND ANNOUCEMENTS
Tues., 8:45—9:00 AM, Madison Ballroom.

8:45 AM **2.1** Daily Orientation and Annoucements.

3.0 PLENARY LECTURE I
Tues., 9:00—10:00 AM, Madison Ballroom.

9:00 AM **3.1** Defining the Role of the Instructor in an Active Learning Environment. **Harold Modell,** *Physiology Edu. Res. Consortium, Seattle, WA.*

NETWORKING BREAK
Tues., 10:00—10:30 AM, Wisconsin Ballroom.

4.0 WORKSHOP I*
Tues., 10:30 AM—12:00 Noon, Madison Ballroom.

10:30 AM **4.1** Using Recorded Lectures for Flipped Classrooms. **Chaya Gopalan,** *Southern Illinois Univ., Edwardsville.*

5.0 WORKSHOP II*
Tues., 10:30 AM—12:00 Noon, University AB.

10:30 AM **5.1** Strategies for Creating a Culture of Academic Integrity. **Kim Henige,** *California State Univ., Northridge.*

6.0 WORKSHOP III*
Tues., 10:30 AM—12:00 Noon, University CD.

10:30 AM **6.1** Integrating Discovery-based Research into the Undergraduate Curriculum. **Jay Labov,** *Natl. Acd. of Sci.,* and **Catherine Middlecamp,** Univ. of Wisconsin, Madison.

*Concurrent workshops/lectures.

7.0 PLENARY LECTURE II
Tues., 1:00—2:00 PM, Madison Ballroom.

1:00 PM **7.1** Team-based Learning in a Large Enrollement Class. **Jon Kibble,** *Univ. of Central Florida.*

NETWORKING BREAK
Tues., 2:00—2:30 PM, Wisconsin Ballroom.

8.0 WORKSHOP IV*
Tues., 2:30—4:00 PM, Madison Ballroom.

2:30 PM **8.1** Interactive Teaching and Learning. **Dee Silverthorn,** *Univ. of Texas at Austin.*

9.0 WORKSHOP V*
Tues., 2:30—4:00 PM, University AB.

2:30 PM **9.1** Using PULSE Rubrics to Assess Departmental Transformation to Student-centered Learning. **Pamela Pape-Lindstrom,** *Everett Comm. Coll., WA.*

10.0 WORKSHOP VI*
Tues., 2:30—4:00 PM, University CD.

2:30 PM **10.1** Accreditation: Role of Course-Block Directors. **Mike Levitzky,** *Louisiana State Univ.*

Poster Session I

11.0 BEST PRACTICES IN PROFESSIONAL SCHOOL PHYSIOLOGY
Tues., 4:00—5:30 PM, Wisconsin Ballroom.
Facilitator: **Thad E. Wilson,** *Marian Univ. Coll. of Osteopathic Med., Indianapolis.*

Poster Board

1 **11.1** A Flipped Classroom Approach With Emphasis on In-Class Active Learning Improves Student Performance on Application Style Questions in a Graduate Level Anatomy Class. **S. Inglis,** *Univ. of South Dakota.*

2 **11.2** Facilitating Peer-to-Peer Interaction in a Distant Learning Program Using Interactive Television. **D. Geenen.** *Grand Valley State Univ.*

3 **11.3** Impact of Pre-testing in Laboratory Performance in Optometry. **M.**

Poster Board

L. A. Fortepiani. *Univ. of the Incarnate Word, San Antonio, TX.*

4 **11.4** Use of Ultrasound For Assessment of Liver Measurement Accuracy During Early Medical Training. **G. Brower, R. McDonald-Thomas, V. Tipton, V. Lee, and F. Prabhu.** *Texas Tech Univ. Hlth. Sci. Ctr.*

5 **11.5** Promoting Student Engagement in a Medical Pharmacology Course: An Experience with Learning Catalytics. **R. Pena Silva, J. Mantilla Rivas, and A. Rodriguez Pulido.** *Univ. de los Andes, Bogata, Columbia.*

6 **11.6** Exam Blueprinting in a First-year Medical School Physiology Course. **T. Pressley.** *Texas Tech Univ. Hlth. Sci. Ctr.*

7 **11.7** Effectiveness of Lecture Compared to Independent and Active Learning Modalities in a Medical School Curriculum. **M. Sheakley.** *Western Michigan Univ. Sch. of Med.*

8 **11.8** Linking IRAT and GRAT Questions Through Clinical Concepts. **D. Averill, M. Cornacchione, and M. Shoemaker.** *The Commonwealth Med. Coll., Scranton, PA.*

9 **11.9** Clinically Relevant Application Exercise which Emphasizes Decision Making. **D. Averill.** *The Commonwealth Med. Coll., Scranton, PA.*

10 **11.10** How to Train a Doctor? Merging Clinical Reasoning with Physiological Concepts. **G. Walcott Bedeau, I. Murray, J. Swope, J. Modica, J. Powell, and M. Clunes.** *St. George's Univ., Grenada.*

WEDNESDAY, JUNE 22, 2016

12.0 DAILY ORIENTATION AND ANNOUCEMENTS

Wednes., 8:45—9:00 AM, Madison Ballroom.

8:45 AM **12.1** Daily Orientation and Annoucements.

13.0 PLENARY LECTURE III

Wednes., 9:00—10:00 AM, Madison Ballroom.

9:00 AM **13.1** A New Paradigm for Student Learners. **Terry Doyle.** *Ferris State Univ., MI.*

NETWORKING BREAK

Wednes., 10:00—10:30 AM, Wisconsin Ballroom.

14.0 WORKSHOP I*

Wednes., 10:30 AM—12:00 Noon, Madison Ballrooom.

10:30 AM **14.1** Tips for Team-based Learning. **Jon Kibble,** and **Lisa Barkley,** *Univ. of Central Florida.*

15.0 WORKSHOP II*

Wednes., 10:30 AM—12:00 Noon, University AB.

10:30 AM **15.1** Student Role in Learning: What is the Goal and How Do We Get There? **Beth Beason-Abmayr,** *Rice Univ., Houston, Texas.*

16.0 WORKSHOP III*

Wednes., 10:30 AM—12:00 Noon, University CD.

10:30 AM **16.1** Educational Scholarship: Step-by-Step Guide to Implement and Publish Your Classroom Research. **Valerie O'Loughlin,** *Indiana Univ.*

17.0 PLENARY LECTURE IVa*

Wednes., 1:00—2:00 PM, Madison Ballroom.

1:00 PM **17.1** Changing USMLE and NBME Medical School Service. **Steven Haist,** and **Agata Butler,** *Natl. Brd. of Med. Examiners.*

18.0 PLENARY LECTURE IVb*

Wednes., 1:00—2:00 PM, University AB.

1:00 PM **18.1** Funding for Educational Research and Curriculum Change. **Barbara Goodman.** *Univ. of South Dakota.*

*Concurrent workshops/lectures.

Join your colleagues for the Opening Reception on Monday, June 20, 2016 from 6:30—8:00 PM

NETWORKING BREAK

Wednes., 2:00—2:30 PM, Wisconsin Ballroom.

19.0 WORKSHOP IV*

Wednes., 2:30—4:00 PM, Madison Ballroom.

2:30 PM **19.1** Using Social Media to Enhance Student Learning. **Patricia Halpin.** *Univ. of New Hampshire, Manchester.*

20.0 WORKSHOP V*

Wednes., 2:30—4:00 PM, University AB.

2:30 PM **20.1** Using Conceptual Frameworks in Teaching and Learning Physiology. **Jenny McFarland.** *Edmonds Community Coll., Lynnwood, WA.*

21.0 WORKSHOP VI*

Wednes., 2:30—4:00 PM, University CD.

2:30 PM **21.1** Roundtable Disucssion of Preparing Students for Board Exams. **Rob Carroll.** *East Carolina Univ.*

Poster Session II

22.0 INNOVATIVE CURRICULA THROUGHOUT EDUCATIONAL LEVELS

Wednes., 4:00—5:30 PM, Wisconsin Ballroom.

Facilitator: **Erica Wehrwein,** *Michigan State Univ.*

Poster Board

11 **22.1** Unpacking the Core Concept of Cell-cell Communications for Undergraduate or Professional Level Physiology Courses. **J. Michael, J. McFarland, A. Wright, H. Modell, and W. Cliff.** *Rush Med. Coll., Chicago, Edmonds Comm. Coll., Lynnwood, WA Canisius Coll., Buffalo, Physiology Res. Consortium, Seattle, and Niagara Univ.*

12 **22.2** Learning to Work Together: In-class Training to Enhance Student Collaboration and Increase Learning Outcomes. **P. Sonner, and M. Newsome.** *Wright State Univ.*

13 **22.3** Exercise Physiology Mob-Lab-based Protocol Promotes Learning and Student Engagement. **P. Inamori, R. Nadal, and C. Lellis-Santos.** *Univ. Fed. de São Paulo, Brazil.*

Poster Board

14 **22.4** Big Brother is Watching. **C. J. Urso and Edward Tall.** *Seton Hall Univ., Orange, NJ, and Montclair State Univ., NJ.*

15 **22.5** Moving from Memorization to Curiosity: Encouraging Student-Directed Learning in an Advanced Physiology Class. **J. Fry.** *Curry Coll., Milton, MA.*

16 **22.6** Physiology Understanding Week (PhUnWeek): K-12 Outreach Program Promotes Both Engagement and Impact. **M. Stieben, and M. Lakes Matyas.** *APS.*

17 **22.7** Educators Rate Life Science Teaching Resource Community as Both Useful and Usable. **M. Byse, and M. Lakes Matyas.** *APS.*

18 **22.8** Expansion of Undergraduate Programs during the Vision and Change Era: Physiology and Other Life Science Fields. **M. Lakes Matyas.** *APS.*

19 **22.9** Agents of Change-Moving from Survey to Experiential Learning: Revamping University of Alaska, Anchorage's Introductory Biology Course Along Vision and Change Guidelines. **R. Hannah, J. Burns, K. Duddleston, and S. Gerken.** *Univ. of Alaska, Anchorage.*

20 **22.10** Reinforcing and Tracking Prerequisite Concept Mastery in Physiology Using Two-Stage Collaborative Testing. **K. Hull, and S. Wilson.** *Bishop's Univ., Sherbrooke, Canada.*

21 **22.11** Team-based Assessment of Nephron Structure and Urine Formation Used as a Forced Retrieval Activity to Improve Student Learning. **J. Chapman.** *Heartland Comm. Coll., Normal, IL.*

*Concurrent workshops/lectures.

Did you register early?
The drawing for the iPad Pro will be held on Thursday, June 23 at 8:45 AM during the Daily Orientation and Announcements

THURSDAY, JUNE 23, 2016

23.0 DAILY ORIENTATION AND ANNOUCEMENTS
Thurs., 8:45—9:00 AM, Madison Ballroom.

8:45 AM **23.1** Daily Orientation and Annoucements.

24.0 PLENARY LECTURE V
Thurs., 9:00—10:00 AM, Madison Ballroom.

9:00 AM **24.1** Supporting Faculty: Resources from Professional Societies and Online Communities. **Marsha Lakes Matyas.** *APS.*

NETWORKING BREAK
Thurs., 10:00—10:30 AM, Wisconsin Ballroom.

25.0 WORKSHOP I*
Thurs., 10:30 AM—12:00 Noon, Madison Ballroom.

10:30 AM **25.1** Teaching Physiological Concepts by Enhancing Student Visual Literacy. **Kathryn Johnson.** *Beloit Coll., WI.*

26.0 WORKSHOP II*
Thurs., 10:30 AM—12:00 Noon, University AB.

10:30 AM **26.1** Statistics in Educational Scholarship. **Douglas Curran-Everett.** *Natl. Jewish Hosp., Denver*.

27.0 WORKSHOP III*
Thurs., 10:30 AM—12:00 Noon, University CD.

10:30 AM **27.1** Instructors' Understanding of Student's Misconceptions can Improve Meaningful Learning. **Ann Wright.** *Canisius Coll., Buffalo.*

28.0 PLENARY LECTURE VIa*
Thurs., 1:00—2:00 PM, Madison Ballroom.

1:00 PM **28.1** The Central Role of Physiology in the Professional Curriculum. **Robert Carroll.** *East Carolina Univ.*

*Concurrent workshops/lectures.

29.0 PLENARY LECTURE VIb*
Thurs., 1:00—2:00 PM, University AB.

1:00 PM **29.1** The Pipeline of Physiology Courses in Community Colleges. **Jenny McFarland, and Pamela Pape-Lindstrom.** Edmonds Comm. Coll., Lynnwood, WA, and *Everett Comm. Coll., WA.*

NETWORKING BREAK
Thurs., 2:00—2:30 PM, Wisconsin Ballroom.

30.0 WORKSHOP IV*
Thurs., 2:30—4:00 PM, Madison Ballroom.

2:30 PM **30.1** Physiology Majors Interest Group. **Erica Wehrwein.** *Michigan State Univ.*

31.0 WORKSHOP V*
Thurs., 2:30—4:00 PM, University AB.

2:30 PM **31.1** Facilitating Small Group Discussion: Basic. **Sydella Blatch, and Carol Schmidhauser.** *Stevenson Univ., MD.*

32.0 WORKSHOP VI*
Thurs., 2:30—4:00 PM, University CD.

2:30 PM **32.1** Faciliatating Small Group Discussion: Advanced. **Betsy Jones.** *Texas Tech. Univ.*

Poster Session III

33.0 BEST PRACTICES IN UNDERGRADUATE PHYSIOLOGY
Thurs., 4:00—5:30 PM, Wisconsin Ballroom.
Facilitator: **Lynn M. Diener,** *Mount Mary Univ., Milwaukee, WI.*

Poster Board

21 **33.1** Original Course-Based Undergraduate Research Experience to Detect Parasitic Worms in Local Parks in Two Undergraduate Courses. **C. Schmidhauser, and S. Blatch.** *Stevenson Univ., Owings Mills, MD.*

22 **33.2** Effect of Collaborative Exams on Retention in an Upper Division Physiology Class. **J. Cooke.** *Univ. of California, San Diego.*

Poster Board

23 **33.3** An Interrupted Case Study Approach for a Diabetes Lab Exercise. **N. Aguilar-Roca.** *Univ. of California, Irvine.*

24 **33.4** Use of Animal Gastrointestinal Tracts in Undergraduate Physiology Courses. **C. Caldari.** *Centenary Coll. of Louisiana, Shreveport.*

25 **33.5** Phys-Maps: An Assessment Tool to Measure Student Learning in Undergraduate Physiology Programs. **K. Semsar, M. Smith, S. Brownell, B. Couch, A. Crowe, S. Freeman, M. Summers, C. Wright, and J. Knight.** *Univ. of Colorado, Boulder, Univ. of Maine, Orono, Arizona State Univ., Tempe, Univ. of Nebraska, Lincoln, and Univ. of Washington, Seattle.*

26 **33.6** Revisiting the Physiological Cost of Negative Work: A Team-based Activity for Undergraduate Exercise Physiology Students. **S. Elmer, and M. Kilgas.** *Michigan Tech. Univ., Houghton.*

27 **33.7** Teaching Undergraduate Online Science Courses with Laboratory Components. **J. Lafuze.** *Indiana Univ. East.*

28 **33.8** Technology and Publishers: Materials in Teaching Physiology to Undergraduate Learners. **J. Lafuze.** *Indiana Univ. East.*

29 **33.9** A Grading Scheme to Inspire Hope and Foster Grit in a Large Undergraduate Physiology Course. **K. Strang.** *Univ. of Wisconsin, Madison.*

30 **33.10** Investigating the Effect of Question Order on Student Understanding of Structure and Function. **K. Carter, and L. Prevost.** *Univ. of South Florida.*

31 **33.11** Assessing the Effectiveness of Student Learning and Engagement in a Content Heavy Flipped Class. **Joost Monen.** *Ramapo Coll. of New Jersey.*

32 **33.12** Using Confluent Classroom Design to Teach Undergraduate Anatomy and Physiology. **A. Petzold, and R. Dunbar.** *Univ. of Minnesota Rochester.*

FRIDAY, JUNE 24, 2016

34.0 **DAILY ORIENTATION AND ANNOUCEMENTS**
Fri., 8:45—9:00 AM, Madison Ballroom.

8:45 AM **34.1** Daily Orientation and Annoucements.

35.0 **PLENARY LECTURE VII**
Fri., 9:00—10:00 AM, Madison Ballroom.

9:00 AM **35.1** Educational Leadership: Benefits of Stepping Outside the Classroom. **Tom Pressley.** *Texas Tech. Univ.*

NETWORKING BREAK
Fri., 10:00—10:30 AM, Wisconsin Ballroom.

36.0 **PLENARY LECTURE VIII**
Fri., 10:30 AM—12:00 Noon, Madison Ballroom.

10:30 AM **36.1** Implementing and Managing Change. **Barbara Goodman.** *Univ. of South Dakota.*

Don't forget to join in the discussions during the daily Poster Sessions from 4:00—5:30 PM

Thank you to the generous support of this workshop from ADInstruments

2016 APS Workshop
APS Institute on Teaching and Learning

Abstracts of Invited and Contributed Presentations

1:0 KEYNOTE LECTURE

1.1
THE CHANGING NATIONAL LANDSCAPE IN UNDERGRADUATE STEM EDUCATION: CONNECTING THE DOTS

Jay Labov[1]
[1]Brd. on Life Sciences, Natl. Academies of Sci., Engineering, and Medicine, Keck Ctr. 638, 500 Fifth St., NW, Washington, DC, 20001.

This session will focus on recent national trends to improve undergraduate and K-12 STEM education and how understanding both the education system and education policy can lead to more informed decision making at the local and state levels. Learning goals for this session include: 1.) Briefly reviewing recent national reports on the improvement of undergraduate education in STEM and how they might inform discussions on your campus. 2.) Exploring the changing relationships among several components of the undergraduate STEM education "ecosystem." 3.) Recognizing and understanding the growing influence of K-12 education on what you do and your role in influencing K-12 education to increase the number of college-educated STEM graduates.

3:0 PLENARY LECTURE I

3.1
DEFINING THE ROLE OF THE INSTRUCTOR IN AN ACTIVE LEARNING ENVIRONMENT

Harold Modell[1]
[1]Director, Physiology Educational Research Consortium, P.O. Box 51187, Seattle, WA, 98115-1187.

An active learning environment is one in which the learners engage the material in ways that help them build, test, and refine their mental models of the system being studied. The primary goal in this environment is to help students use the information they are acquiring to solve problems. Although most faculty agree that the role of the instructor in this environment is to facilitate learning by helping the learner to learn, classroom practice is not always consistent with this role. This interactive discussion will focus on exploring the role of the instructor in the active learning environment by examining a number of questions that must be considered before faculty can adopt a mindset that directs appropriate classroom practice. In addition, currently advocated "active learning tools" (e.g., group work, think-pair-share, clickers) will be discussed in the context of the "helping the learner to learn" mindset to develop criteria for choosing appropriate tools for specific classroom situations. Reference: Michael, JA. and Modell, HI. Active Learning in Secondary and College Science Classrooms: A working model for helping the learner to learn. (2003) Mahwah, NJ: Routledge. ISBN 978-0-8058-3948-7.

4:0 WORKSHOP I*

4.1
USING RECORDED LECTURES TO FLIPPED CLASSROOMS

Chaya Gopalan[1]
[1]Applied Health & Nursing Dept., Southern Illinois Univ., Edwardsville, Box 1126, Edwardsville, IL, 62026.

Recorded lectures play a vital role in the flipped classroom model of teaching as these are the ones students depend on and use multiple times to prepare for the active learning sessions during class period. Although there are a variety of well known high quality resources to choose from, instructor-recorded lectures are favored due to the fact that the content and the vocabulary being consistent with assessments. There are a number of gadgets available to record lectures today but the technological skills required and the ability to lecture without audience feedback are challenges to consider while creating these videos. The focus of this workshop is to address the advantages and challenges of using lecture videos in the flipped classroom.

5:0 WORKSHOP II*

5.1
STRATEGIES FOR CREATING A CULTURE OF ACADEMIC INTEGRITY

Kim Henige[1]
[1]Kinesiology, California State Univ., Northridge, 18111 Nordhoff St., Northridge, CA, 91330-8287.

Within American universities, the typical approach to academic integrity involves the "3 Ps of Academic Integrity": Prevention, Policing, and Punishment. In this workshop, participants will discuss an enhanced approach, with a focus on creating a general culture of integrity. Such an approach may have positive consequences beyond the college years. The session will begin with a review of recent statistics on cheating in school and its association with other types of dishonesty. The 3 Ps of Academic Integrity will be briefly addressed, with an emphasis on introducing and discussing recommended general and concrete strategies for creating a culture of integrity. The strategies will be divided into five categories: 1.) Communicate about integrity, 2.) Role model integrity, 3.) Create space for integrity, 4.) Integrate ethics into the curriculum, and 4.) Respond to cheating. References: Bertram Gallant, T. Creating a culture of integrity: An alternate proposal for educators. Workshop presented at California State University, Northridge April 17, 2015. Josephson Institute. Survey results, 2009. Retrieved: February 26, 2016 from http://josephson-

institute.org/surveys. McCabe, D.L. Cheating among college and university students: A North American perspective. The International Journal for Educational Integrity, 1(1), 1-11, 2005.

6:0 WORKSHOP III*

6.1
INTEGRATING DISCOVERY-BASED RESEARCH INTO THE UNDERGRADUATE CURRICULUM

Jay Labov[1], and Cathy Middlecamp[2]
[1]Brd. on Life Sciences, Natl. Academies of Sci., Engineering, and Medicine, Keck Ctr. 638, 500 Fifth St., NW, Washington, DC, 20001, [2]Nelson Inst. for Environmental Studies, Univ. of Wisconsin, Madison, 550 North Park St., Madison, WI, 53706.

The 2012 report, "Engage to Excel," from the President's Council of Advisors on Science and Technology (PCAST) urges the STEM education community and funding agencies to "Advocate and provide support for replacing standard laboratory courses with discovery-based research courses." Emerging evidence suggests that engaging undergraduates in discovery research as early as possible during their undergraduate years is one of the best strategies for supporting and retaining STEM students and improving aspects of scientific literacy. But providing all students with individualized mentored research experiences, one of the traditional routes to a career in science, is not possible given the large numbers of beginning STEM students and limitations in lab space, supply budgets, and available research mentors. Acting on the PCAST recommendation, many undergraduate STEM educators are now experimenting with various strategies for engaging more students in research, and a variety of tested models are emerging. These successes are catalyzing interest in replacing standard "cook-book" laboratories with discovery-based research and related activities in labs associated with lecture courses or in stand- alone laboratory courses, utilizing on-campus, off-campus, and on-the-web resources. This presentation will report on a convocation around these issues that was organized by a committee of the National Academies of Sciences, Engineering, and Medicine. It will summarize successes and challenges that were discussed during the convocation and are presented in the report. The report is available for electronic download without cost at http://www.nap.edu/catalog/21851. Session participants will receive a printed copy of the report.

7:0 PLENARY LECTURE II

7.1
TEAM-BASED LEARNING IN A LARGE ENROLMENT CLASS

Jonathan Kibble[1], and Lisa Barkley[2]
[1]Med. Education, Univ. of Central Florida, 6850 Lake Nona Blvd., Orlando, FL, 32827, [2]Clinical Sci., Univ. of Central Florida, 6850 Lake Nona Blvd., Orlando, FL, 32827.

According to the TBL Collaborative[1], *" [TBL].. is an evidence based collaborative learning teaching strategy designed around units of instruction, known as "modules," that are taught in a three-step cycle: preparation, in-class readiness assurance testing, and application-focused exercise. A class typically includes one module."* In this session we will provide an overview of each of these major components of TBL; we will present ideas about implementation and will discuss common challenges from our perspective as TBL practitioners. Our aim is to have an interactive session to explore the four underlying principles of TBL[2]: 1.) Proper formation of diverse groups, 2.) Student accountability for preparation and team performance, 3.) Use of assignments that promote learning and team development, 4.) Use of feedback (2). References: 1. Team-Based Learning Collaborative http://www.teambased-learning.org/definition/ [2/24/2016]; Michaelsen LK, Sweet M. Fundamental Principles and Practices of Team-Based Learning. In *Team-Based Learning for Health Professions Education: A Guide to Using Small Groups for Improving Learning* edited by Michaelsen LK, Parmelee DX, McMahon KK, Levine RE, Sterling Virginia: Stylus Publishing, 9-31, 2007.

8:0 WORKSHOP IV*

8.1
INTERACTIVE TEACHING AND LEARNING

Dee Silverthorn[1]
[1]Dell Med. Sch., University of Texas at Austin

In this session we will discuss different ways to create interactive classrooms, from clickers and small group learning to pre-recorded lectures for 'flipped classrooms.' Participants should come prepared to share both their best practices and their lessons learned.

9:0 WORKSHOP V*

9.1
USING PULSE RUBRICS TO ASSESS DEPARTMENTAL TRANSFORMATION TO STUDENT CENTERED LEARNING

Pamela Pape-Lindstrom[1]
[1]Life Sciences, Everett Comm. Coll., 2000 Tower St., Everett, WA, 98201.

Workshop participants will use the PULSE Vision & Change Rubrics 2.0 available at http://www.pulsecommunity.org/page/recognition. The rubrics were developed for departmental self-assessment based on recommenda-

tions in *Vision and Change in Undergraduate Biology Education: A Call to Action (V and C). V and C* details the consensus among life sciences educators regarding student-centered learning of core concepts and competencies based on evidence from cognitive science, neuroscience and science education research. The *V and C* recommendations emphasize scientific reasoning and ability to think critically within a discipline, instead of mastery of disciplinary facts alone. Laboratory work, hypothesis creation and testing assume a new prominence, and this report shifts the focus from teacher-directed exercises to student-centered efforts. The rubrics were developed to assess the degree to which departmental work and support align with the *V and C* recommendations. They also form the basis of a tiered recognition program, with an underlying assumption that exemplar levels of achievement will lead to improved student learning outcomes. The Faculty Practice/Support rubric has 20 criteria reflecting issues that are driven by or affect faculty. The Student Higher Level Learning section of this rubric evaluates faculty efforts to provide engaging, evidence-based, student-centered exercises. Student willingness to reflect on and engage in activities that require higher level cognitive efforts is also assessed. The rubrics were developed to be applicable to all institution types and statistical analysis demonstrates that the instrument shows no bias by institution type. Participants will work with an abbreviated form of the recognition rubrics (PULSE Progress Snapshot Rubric) and be introduced to the full rubrics. Use of the rubrics will allow faculty and their departments to move toward more student-centered learning of biological concepts and competencies. (NSF EF-1350120 & DBI-1323223). References: Brewer, Carol A., and Diane Smith. "Vision and change in undergraduate biology education: a call to action." *American Association for the Advancement of Science, Washington, DC* (2011). Pape-Lindstrom, Pamela, et al. "PULSE Pilot Certification Results." *Journal of Microbiology & Biology Education* 16.2 (2015): 127. Aguirre, Karen M., et al. "PULSE Vision & Change Rubrics." *CBE-Life Sciences Education* 12.4 (2013): 579-581.

10:0 WORKSHOP VI*

10.1
ACCREDITATION: ROLE OF COURSE /BLOCK DIRECTORS

Michael Levitzky[1]
[1]Physiology, LSU Hlth. Sci. Ctr., 1901 Perdido St., PO Box P7-3, New Orleans, LA, 70112-1393.

In this workshop we will discuss the role of course directors and block directors in the accreditation of higher education institutions. We will first give an overview of higher learning institution accreditation in the United States. We will then discuss accreditation by the seven regional accrediting bodies recognized by the U.S. Department of Education and by the Council for Higher Education Accreditation (CHEA). We will then briefly mention some national accreditors, before discussing specialized and professional accreditors, such as the Liaison Committee on Medical Education, the American Dental Association, and other accrediting bodies for allied health and nursing schools. We will then discuss the importance of Continuous Quality Improvement and the assessment of Institutional Effectiveness in regional accreditation, including the feedback loop of stating expected outcomes, assessment of achievement of the outcomes, and evidence of improvement based on analysis of the assessment. Finally, we will discuss the roles of course directors or block directors in preparing for accreditation self-studies and site visits. These include the important and sometimes controversial subject of faculty qualifications to teach courses (and how to justify those faculty who may not meet accrediting body guidelines) and the importance of demonstrating that course content meets the requirements of specialized accrediting bodies.

11:0 BEST PRACTICES IN PROFESSIONAL SCHOOL PHYSIOLOGY POSTER SESSION

11.1
A FLIPPED CLASSROOM APPROACH WITH EMPHASIS ON IN-CLASS ACTIVE LEARNING IMPROVES STUDENT PERFORMANCE ON APPLICATION STYLE QUESTIONS IN A GRADUATE LEVEL ANATOMY CLASS

Stuart Inglis[1]
[1]Basic Biomed. Sci., Univ. of South Dakota, 414 E. Clark St., Vermillion, SD, 57069.

A flipped classroom design was implemented for the musculoskeletal unit of a graduate level gross anatomy class that had been previously taught using a traditional didactic design. Prior to the redesign, first year OT, PT, and PA students attended a 1 hour lecture with no structured review activities between classes. With the flipped classroom, students were assigned a prerecorded podcast to view the night before class, containing the material previously presented in lecture. Class time was reorganized as active learning sessions, in which students were presented with clinical scenarios and, working in groups, applied their understanding of anatomy presented in the podcasts to make a diagnosis. At the conclusion of the unit, identical test questions used in the unit tests for both groups were identified, characterized, and compared between groups. In both traditional didactic and flipped classroom course designs, students scored distinctively better on questions requiring the recall of memorized material when compared to the appli-

cation of acquired knowledge to novel situations. When comparing between the two groups, there was little appreciable difference between students on rote memory style questions, but the students in the flipped class design scored significantly better on application style questions when compared to students in the traditional classroom setting. Student evaluations of the flipped classroom design were generally positive, with most students agreeing that the flipped classroom design required more work, but resulted in greater understanding and retention of material when compared to a traditional classroom setting. These data suggest that students may struggle to apply acquired knowledge to unfamiliar situations, and that appropriately designed active learning sessions may assist students in developing these application skills necessary to address these sorts of questions.

11.2
FACILITATING PEER-TO-PEER INTERACTION IN A DISTANT LEARNING PROGRAM USING INTERACTIVE TELEVISION

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The Physician Assistants Department in the College of Health Sciences at Grand Valley State University is incurporating a distant learning format into their curriculum. The purpose of this format is to develop and implement the delivery of its didactic courses to graduate students in a Grand Rapids, Michigan cohort while broadcasting the live lectures to a second student cohort in Traverse City, Michigan through Interactive Television (ITV). Presentation of the physiology and pathophysiology lectures by the instructor using ITV is challenging because of the difficulty of noticing visual cues from students at the distant learning site and the desire to incorporate peer-to-peer interaction between students at both sites. In addition, it is necessary to present information to both student cohorts simultaneously when the ITV system is unavailable, for example, during a review session. Thus, we used two different electronic approaches to address these concerns (1) an online interactive question and answer format called "*Socrative*" available free of charge to both students and instructor, and (2) *Blackboard Collaborate*. Using *Socrative*, the instructor creates quizzes with multiple choice, true/false, or open-ended questions and presents these to both sites using the ITV system. The students log on to *Socrative* with smart phones, laptops, or tablets and answer the questions loaded onto the site by the instructor. One of the advantages of using this internet-based system is that groups of students can compete against each other and groups can be formed within or between the cohorts. This interactive program allows the instructor to observe students working through problems and to receive student responses in real time. *Blackboard Collaborate* has been used to review for exams at times when the ITV system is unavailable to both cohorts and instructtor. PowerPoint slides and lecture material can be displayed on screen at remote sites and presented to the students wherever they can connect online. Students then "join a session" and interact through audio and video feeds with the instructor and each other during the review. We frequently have students working in small groups during *Blackboard Collaborate* sessions, which also facilitates peer-to-peer interaction. These two resources for interactive learning have helped the program promote an *esprit de corps* between the two ITV sites and enabled student interaction with the distant learning format.

11.3
IMPACT OF PRE-TESTING IN LABORATORY PERFORMANCE IN OPTOMETRY

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In health professions, training in preclinical curricula such as physiology and pathology is fundamental for clinical practice. However, many students find the transition from pre-clinical to clinical training challenging. In an attempt to increase understanding and performance during this transition, we targeted a course with clinically relevant techniques and evaluations but without a direct patient care component. We hypothesized that testing students on laboratory skills prior to the laboratory session (pre-lab testing or PLT) may increase understanding and retention of the information presented and impact their performance in practical or theoretical testing when compared to testing during the laboratory session (in-laboratory testing or ILT). This study was performed using four (two ILT and two PLT) cohorts. There were a total of four laboratory sessions included in the course, and all students participated in all the sessions. All the students enrolled in the course also took a test based on the theoretical and practical facts included in the lecture portion and handout for each laboratory session. The two PLT groups submitted the tests prior to laboratory, while the ILT groups completed the assignments during the laboratory session. At the end of the semester the students took a final theoretical test and a final practical test that included activities from every laboratory session. While the scores on the pre-test in the PLT groups appeared to better predict scores on the practical portion of the final exam than did the laboratory assignment scores for the ILT groups, the overall performance of both student groups (PLT and ILT) was similar for the laboratory testing and final practical and theoretical exams. These results indicate that the time of testing does not seem to modify the score of

the students in either practical or theoretical portions of an optometry preclinical course.

11.4
USE OF ULTRASOUND FOR ASSESSMENT OF LIVER MEASUREMENT ACCURACY DURING EARLY MEDICAL TRAINING

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Documentation of competency in performing entrustable professional activities requires the incorporation of new techniques and technologies in medical education. Abdominal examination is a challenging skill for First Year medical students to master. A specific learning objective in our Development of Clinical Skills (DOCS) course is that students develop proficiency in evaluation of subcutaneous structures when performing physical examinations. Assessment of the abdominal system with respect to the liver typically includes palpation, percussion and/or the scratch test to determine localization of the liver edge and measurement of liver span. Historically it has been difficult to establish the student's proficiency in performing this aspect of the physical examination. Moreover, studies show that clinicians routinely underestimate the liver span as compared to that measured by ultrasonography. Thus, more effective methods to enhance learning of abdominal examination techniques are needed. Thus, we have implemented an approach that incorporates real-time ultrasound imaging to provide immediate feedback when teaching examination of the liver. The dimension of liver span and location of the liver edge along the right midclavicular line are determined by percussion and palpation in a standardized patient. The accuracy of the student's estimated measurements are then compared to the liver span as determined by ultrasound imaging (GE LOGIQ ultrasound). We found ultrasound-assisted determination of liver edge localization and span to be a useful adjunct to traditional means of teaching abdominal examination skills. A distinct advantage of utilizing this approach is the student can obtain immediate feedback with respect to their percussion skills, thus improving their accuracy for times when ultrasound is not available. Finally, it contributes to providing our students with a solid foundation in point-of-care ultrasonography.

11.5
PROMOTING STUDENT ENGAGEMENT IN A MEDICAL PHARMACOLOGY COURSE: AN EXPERIENCE WITH LEARNING CATALYTICS

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Student participation is an essential component of active learning. Student response systems promote student engagement and have been used for years with good results. Learning Catalytics (LC) is a recently developed alternative, to which students can connect using their personal communication devices and Internet. In contrast to other systems, LC includes multiple types of questions that facilitate assessment of different components of learning. The purpose of this study was to assess the effectiveness of LC as a teaching and learning tool in a medical pharmacology course. We included data from 3 cohorts of second year medical students (2014-2, 2015-1 and 2015-2) corresponding to 184 students. First, we surveyed the perceived benefit of using LC to students and teachers. This survey assessed satisfaction with the tool, commitment to study and prepare lectures before class, interactions between students and professors, perceived learning during the activities, and limitations of the tool. Second, we also asked for a numerical grade (1-5 scale) and comments about the perceived benefit of the experience with LC. We compared this perceived benefit to academic performance (final grade), educational environment (DREEM questionnaire) and gender. Data was analyzed with chi square test or one way ANOVAs followed by post hoc test. Results: More than two thirds of students agreed with statements related with high satisfaction, increased interactivity, more commitment to the preparing lectures and better learning using LC. These statements almost always agreed with the perceptions of the course director. Perceived benefit of LC in the learning process was high among students (4,37 ± 0,72 mean±SD, 4,5 median). We did not find a correlation between final grades or gender, and the perceived benefit of LC. Students that scored in the upper quartile (Q4) in the learning environment questionnaire, perceived a higher benefit of LC than students in the lower quartile (Q1) ($P<0,05$). Technical issues such as network connectivity during the sessions were the most reported limitation of LC. In summary, students perceive that the learning process and social interactions improve while using LC. Most students appear to benefit from using LC and no differences were found between genders of between different levels of performance in the course. Learning Catalytics appears to be a useful tool for the teaching of pharmacology. Funding: Conecta-TE, Universidad de los Andes.

11.6
EXAM BLUEPRINTING IN A FIRST-YEAR MEDICAL SCHOOL PHYSIOLOGY COURSE

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Most educators would agree that the assessment of a student's mastery of a subject should be based on sound, quantitative principles. Although statistics allow the analysis of exams after students take them, faculty often do an inconsistent job when building the exam in the first place. Developing the skills needed to improve exams would increase longitudinal consistency and minimize the need to revise scores after the fact. Blueprinting, a practice more traditionally associated with standardized, high-stakes exams, offers a potential mechanism for achieving balance and reproducibility. Accordingly, I used exam blueprinting for systematic help in developing the cardiovascular exam for our first-year medical course in physiology. A simple spreadsheet was created that listed the various subjects covered, as well as the approximate amount of time devoted to each. With a target of 55 questions for the exam, I could use the relative amounts of time to estimate the number of questions that I needed for each subject. Of course, the estimates were not always integers, so some leeway was required when allocating the actual number of questions. The resulting blueprint provided a guide when assembling the exam from a databank of questions used in previous years. I also used blueprinting to estimate how difficult the exam might be for students. This was accomplished in two ways. The first was to take advantage of the cognitive domains outlined in Bloom's taxonomy of educational objectives (Bloom 1956). I divided questions into those that rely on knowledge, comprehension, or application/analysis using the guidelines outlined by Crowe et al. (2008). The second was based on previous success rates for the questions. These were simply averaged to provide an overall estimate of the outcome for the exam. My predicted score was 82.8%, and the actual outcome for the exam was 83.3%. Moreover, a retrospective analysis of last year's cardiovascular exam using a similar blueprint showed similar success in predicting the outcome of that older exam. These results suggest that blueprinting provide a robust means of predicting the exam's outcome, and they argue that the routine use of blueprinting for in-house exams offers the potential to generate more consistent assessments across multiple years and classes. (Supported by an APS Teaching Career Enhancement Award). References: Bloom B. S. (1956). *Taxonomy of Educational Objectives, Handbook I: The Cognitive Domain*. New York: David McKay Co. Crowe, A., Dirks, C., and Wenderoth, M.P. (2008) Biology in Bloom: Implementing Bloom's Taxonomy to Enhance Student Learning in Biology. *CBE& Life Sci. Educ.* 7:368-381.

11.7
EFFECTIVENESS OF LECTURE COMPARED TO INDEPENDENT AND ACTIVE LEARNING MODALITIES IN A MEDICAL SCHOOL CURRICULUM

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Introduction/Rationale: Didactic lectures, once the mainstay of undergraduate medical education, are diminishing in favor of more student-centered, active forms of classroom learning in many medical curricula. Nonetheless, the material formerly taught in didactic lectures is still important to the foundation of medical knowledge, and still must be mastered by students. To serve this purpose, a variety of vehicles to deliver this content for independent learning outside of the classroom have been developed and implemented at a variety of medical schools. However, the question regarding the efficacy of student learning in didactic lectures compared to independent learning events remains. Methods: The curriculum at Western Michigan University Homer Stryker M.D. School of Medicine (WMed) was designed to include didactic lectures, independent learning events, and active learning events each week. In this curriculum, an independent learning event is an eBook developed by a faculty member, containing learning objectives and specific content, which the students are required to master outside of the classroom. An active learning event is a classroom session where an interactive form of learning occurs, such as problem solving tutorials, case studies, debates, and team-based learning. A typical academic week consists of approximately 6 hours of didactic lecture, far less than a traditional medical school curriculum, 3-5 hours of active learning, and 5-7 hours of independent learning events. To assess whether learning is different between didactic lectures, active learning events, and independent learning events within our curriculum, statistical analysis is being used to evaluate student performance on end-of-course (summative) exams for each Foundations of Medicine course in the undergraduate medical curriculum (years M1 and M2). This serves to compare individual students' performance on assessments from didactic lectures versus independent learning events versus active learning events. In addition, the data is further elucidated by comparing the performance on assessments for students in the upper and lower ranks of the class. Outcomes: Initial analysis of the data shows that students perform significantly better on the summative assessment questions from Independent and Active Learning events, relative to didactic lecture events.

11.8
LINKING IRAT AND GRAT QUESTIONS THROUGH CLINICAL CONCEPTS

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Background: Many programs which employ Team Based Learning (TBL) use individual readiness assurance tests (IRAT) and group readiness assurance tests (GRAT) to evaluate students' initial comprehension of foundational information. Michaelson and colleagues originally proposed that the same set of questions should be used in the IRAT and GRAT in a TBL session. In part, the repetition of the same questions in the group setting was proposed as a way to foster collaborative thinking and team development. In some programs such as medical education these questions emphasize higher order cognitive thinking in the domains of application, synthesis, prediction, and prioritization. Description: At The Commonwealth Medical College the third year clinical curriculum is delivered through a combination of longitudinal integrated clerkships (LIC) and block clerkships. Internal and Family Medicine have used TBL as their primary instructional and assessment format during didactically oriented learning activities which occur every 4-5 weeks. In previous years the IRAT and GRAT used identical questions. Upon review of these learning activities there was a perceived need to enrich the learning of students. To extend students' learning in clinical medicine, the IRAT and GRAT were redesigned such that the IRAT and GRAT used different questions but each pair of IRAT and GRAT questions were linked to a common concept or topic. For example, IRAT questions may focus on specific aspects of differential diagnosis whereas the GRAT questions might focus on development of treatment plans pertinent to the diagnosis of the IRAT question.

11.9
CLINICALLY RELEVANT APPLICATION EXERCISE WHICH EMPHASIZES DECISION MAKING

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Background: Team Based Learning encourages a pedagogical approach composed of three sequential steps. The first step, individual readiness assurance test (IRAT), evaluates how well individual students assimilate foundational information. The second step, group readiness assurance test (GRAT), evaluates the collaborative efforts of group members to answer the same questions used in the IRAT. The third step, application exercise, requires the group to apply foundational knowledge in the solution of a complex problem. Application exercises used during the initial years of medical student education can be a powerful strategy to consolidate foundational information in a clinically relevant setting. Another important skill set which medical students need to develop is the ability to make knowledge-based decisions in a team setting. We are in the process of developing a set of self-directed application exercises which can be used to evaluate the decision making process of medical students. Description: The application exercise begins with an initial brief description of a clinical problem. Each group can request additional information to formulate an initial differential diagnosis. Since the number of choices is limited, the group needs to prioritize the type of information they believe is important. Based upon the development of the differential diagnosis, the group can request various clinical tests or procedures to further refine the differential diagnosis. Once again, the number of choices is limited and the group must make knowledge-based decisions in the prioritization of tests or procedures "ordered". The application exercise ends with groups discussing the rationale used in solving the clinical problem. Results: The application exercise described above has been implemented as a self-directed presentation. Depending on the clinical case and the design of the application exercise, "break points" may be inserted when groups suspend their movement through the activity so discussion among groups may take place. Movement through the activity resumes after a brief discussion. Future plans include the implementation of this self-directed activity on a platform which will allow us to analyze the decision making process of medical students. Conclusion: Based upon results of initial surveys, medical students value this type of application exercise because it requires them to make knowledge-based decisions in a clinically relevant setting.

11.10
HOW TO TRAIN A DOCTOR? MERGING CLINICAL REASONING WITH PHYSIOLOGICAL CONCEPTS

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Objective: Basic science knowledge and clinical reasoning are two essential elements in medical education. Early integration of both elements in medical courses is a challenge in terms of the focus, emphasis and balance of both objectives. In an attempt to integrate these ideas, we developed an online interactive clinical case module and measured the

level of enthusiasm for learning and the application of physiological concepts during a first year medical student basic sciences course. **Design & Methods:** Students enrolled in Medical Physiology (n=439) completed a St. George's University and Smart Sparrow™ designed online interactive clinical case module as a prerequisite to a facilitated small group session. The module consisted of four components: patient history, physical exams, history notes, and test results. Student progress through several decision branch points was recorded, and student performance quantified during and after the module. Formative assessment was via questions at key branch points, with immediate feedback. Cumulative assessment was via rubric scoring of the patient notes, and incorporation of two multiple choice questions in the survey and midterm exam. Student feedback was obtained via an exit Likert scale survey. **Results:** Students spent an average of 1 hour on the module, with analysis of formative branchpoints. Findings indicated an increased understanding of the diagnosis: 68% of the students made the correct diagnosis after the interview phase, 86% were able to make the correct diagnosis after the interview, examination, notes & tests phases, and 76% of the students were able to correctly answer the post module Multiple Choice Questions. In the Likert survey, 70-85% of the students indicated that the module stimulated an increased enthusiasm in critical thinking, related to lecture content, basic science knowledge and clinical reasoning. **Conclusion:** The innovative online module effectively integrated basic science knowledge with clinical skills, as determined both by student feedback and MCQ performance. It also improved inclass dynamics and identified key conceptual difficulties. **Acknowledgements:** 1. The Clinical Tutors of the Department of Physiology and Neuroscience, School of Medicine, St. George's University. 2. Allison Murray and Smart Sparrow™ Team.

13:0 PLENARY LECTURE III

13.1
A NEW PARADIGM FOR STUDENT LEARNERS

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The challenges facing American Higher Education cannot be solved by teachers alone. If educational outcomes are ever going to improve in the United States, it is essential that our students, both those who struggle and those who do well, learn how to be better learners. This session will discuss how the findings from neuroscience, cognitive science and biology research, over the past fifteen years; demand a new paradigm for student learners. This new paradigm requires students first, to prepare themselves for learning and second, to apply the findings from brain research on how the human brain learns best to their learning and studying practices. The specific actions students need to take to adopt this new paradigm will be shared in this session. References: Doyle, T. & Zakrajsek, T. (2013) The New Science of Learning. Sterling, Virginia: Stylus. Strickgold, R. (2005, October 27). Sleep dependent memory consolidation. Nature, 437, 1272-1278. doi: 10.1038/nature-04286. Ratey, J. (2008). Spark: The revolutionary new science of exercise and the brain. New York: Little Brown.

14:0 WORKSHOP I*

14.1
TIPS FOR TEAM-BASED LEARNING

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This is a hands-on workshop in which participants will experience each component of TBL. The session will cover proper small group formation, the conduct of readiness assurance testing and deployment of application focused exercises. The session will culminate with discussion about key topics such as conduct of appeals, incorporation of peer evaluation and establishment of grading schema in collaboration with learners.

15:0 WORKSHOP II*

15.1
STUDENT ROLE IN LEARNING: WHAT IS THE GOAL AND HOW DO WE GET THERE?

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As we modify our teaching methods and incorporate active learning strategies to make our classes more student-centered, one of the biggest challenges we face is how to get buy-in from the students so they will engage in the learning process. This new learning environment requires students to take on new roles and responsibilities that are likely quite different from those associated with the traditional college class or what they may have experienced in high school. So how do we motivate students to take ownership of their own learning? How do students learn what their role will be? Why do students resist learner-centered teaching? What factors contribute to a student's poor performance? Why are students poor judges of effectiveness of study strategies? How does a student know when they have mastered the material? And what about us? What is our role in this process? How do we provide students opportunities to practice taking on their roles and evaluate whether they are

performing their roles satisfactorily? How do we implement evidence-based teaching strategies while at the same time making students realize the value of desirable difficulties? How do we evaluate our own teaching? How do we ensure our evaluation of students is consistent with the roles they have been required to take on in the course? In this workshop, we will discuss questions and challenges such as these and explore strategies and approaches you can implement and integrate in your classes to help your students become lifelong learners. References: Brown, P. C., Roediger, H. L., and McDaniel, M. A. (2014) *Make It Stick*. Doyle, T., and Zakrajsek, T. (2013) *The New Science of Learning*. Doyle, T (2008) *Helping Students Learn in a Learner-Centered Environment*.

16:0 WORKSHOP III*

16.1
EDUCATIONAL SCHOLARSHIP: A STEP-BY-STEP GUIDE TO IMPLEMENT AND PUBLISH YOUR CLASSROOM RESEARCH

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Cross and Steadman (1996) defined *classroom research* as a continual, progressive intellectual inquiry by a teacher about the teaching and learning within one's classroom. As one assesses the effectiveness of an instructional method or educational intervention, the findings not only help improve teaching and learning, but also may lead to further questions about how our students learn. Classroom research is a subset of educational research, which may examine teaching and learning beyond the scope of a single classroom. Educational scholarship refers to the practice of making educational research public, by presenting your findings to peers for review. While most scientists are familiar with the process of publishing in our specific bench-research areas, few of us have training in developing a classroom research project and publishing our findings (i.e., performing educational scholarship). In this interactive session, participants will learn the basics about developing a clear classroom (or educational) research hypothesis, how to carefully review the educational research literature, obtaining Institutional Review Board (human subjects) approval (as our students are human subjects!), and selecting appropriate methods of assessment for your hypothesis. We will discuss potential presentation and publication venues for your classroom research. Come prepared with a potential question you want to examine in your classroom, and by the end of the session you will leave with a refined educational research hypothesis and potential methods of assessment for that question. References: Cross, K. P., and Steadman, M. I. (1996) *Classroom Research: Implementing the Scholarship of Teaching*. San Francisco, CA: Jossey-Bass, and O'Loughlin, V. D. (2006) A 'how to' guide for developing a publishable Scholarship of Teaching project. *Adv Physiol Educ* 30:83-88.

17:0 PLENARY LECTURE IVa*

17.1
THE CHANGING USMLE AND NBME MEDICAL SCHOOL SERVICES

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The United States Medical Licensing Examination (USMLE) Composite Committee (CC), the governing body of USMLE, requested an in-depth review of the USMLE program in terms of purpose, design, and format. The USMLE underwent a comprehensive review between 2006 and 2008 by the Committee to Evaluate the USMLE Program (CEUP). The CEUP Committee made six recommendations, five of which were accepted by the CC. The five recommendations were: 1) the examination program should support two decisions, entry into supervised practice and entry into independent practice; 2) the design, development, and scoring of the of the examinations should be driven by a general competencies schema; 3) foundational science should be assessed throughout USMLE; 4) clinical skills assessment should remain a component of USMLE and enhancement of testing methods assessing clinical skills should be considered; and 5) USMLE should introduce a format to assess an examinee's ability recognize and define a clinical program, access appropriate references to address the problem, and to interpret and apply that information in an effective manner. Since 2008, a great deal of progress has been made. Extensive practice analyses were conducted to inform changes, and more recently, a new Step 3 was introduced and is now two examinations, Step 3A, Foundations of Independent Practice and Step 3B, Advanced Clinical Medicine. The major changes in Step 3 examinations include an increased emphasis on foundational science and evidenced-based medicine. Two new formats, pharmaceutical advertisements and scientific abstracts, have been introduced. While many changes have occurred in Step 1 and Step 2, in the near future, more significant changes will occur. Some of the potential changes include new formats assessing communications skills, additional emphasis on patient safety, enhancing the assessment of evidence-based medicine skills, and use of clinical decision making tools during an examination, such as a metabolic map when answering biochemistry/metabolism questions. The purpose of the USMLE section of the pre-

sentation is three-fold: 1) information sharing, 2) getting feedback on the proposed new formats, and 3) brainstorming regarding other possible competencies or sub-competencies to assess. The NBME presentation provides an overview of the NBME services to medical schools and students and an update on the changes and initiatives that are underway with the Subject Test Program, Customized Assessment Services, NBME Self-Assessment Program, and NBME-U. The NBME section will also give participants an opportunity to provide recommendations on how to improve services and what the NBME can do to better meet the needs of medical schools, medical educators, and students.

19.0: WORKSHOP IV*

19.1
USING SOCIAL MEDIA TO ENHANCE STUDENT LEARNING

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Social media has become ubiquitous and Twitter is one of the most popular sites used with 500 million tweets per day. While many would assume this use of social media is for disseminating information only, it can also be used as part of a college science course. Twitter is used to hold weekly asynchronous discussions on specific topics related to the assigned course material. By requiring brevity, 140 character limit, students need to choose wisely about what they will tweet. Credible sources (The Scientist, Nature, Science, Smithsonian) can be followed on Twitter and used as sources of articles for class discussions. Posting articles from these credible sources and asking the class a question about the article enhances class discussion. Students become accustomed to the high quality and high quantity of science information that is posted daily on Twitter and appreciate the choices of articles they can use to tweet. In this workshop you will sign up for a Twitter account, and learn how to participate in a robust class discussion on Twitter. The uses of: hashtags, *following*, URL shortener, messaging and retweeting will be explored. During the workshop we will use all these tools to hold our own robust discussion on a current science topic. If time permits, the use of a closed group in Facebook to promote informal learning will also be demonstrated. Reference: Using Twitter in a Non-science major science class increases students' use of reputable science sources in class discussions. Journal of College Science Teaching [In Press].

20:0 WORKSHOP V*

20.1
USING CONCEPTUAL FRAMEWORKS IN TEACHING AND LEARNING PHYSIOLOGY

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Learning of physiology involves the development of an enduring understanding and application of the core concepts of our discipline. Core concepts in physiology have been identified (Michael & McFarland 2011) and our group has also developed and vetted conceptual frameworks for three core concepts: Flow Down Gradients (Michael & McFarland 2011), Homeostasis (McFarland et al. 2016) and Cell-Cell Communications. A conceptual framework is a hierarchical "unpacking" of the ideas underlying a core concept that are important for understanding. Each core concept is made up of "critical components", essential aspects of an accurate mental model of the core concept. "Constituent ideas" are necessary for working understanding of the critical components. Conceptual frameworks serve many functions in teaching and learning. They help students organize their growing understanding and develop sound explanations of phenomenon. They allow learners to fit factual knowledge into a logical structure that relates these facts to the core concept. Thus, students can more easily assimilate new information and prior knowledge as they progress thru a physiology course or courses. Frameworks give faculty a lens to view the student learning outcomes, and guide courses, curriculum and programs toward helping students gain meaningful understanding of physiology core concepts, as well as mastering content knowledge. Frameworks can provide a progression of outcomes across a series of courses, with introductory courses introducing specific core concepts and critical components. Faculty in upper division courses might expect students to appropriately apply and explain constituent ideas. For faculty using backwards design, the frameworks can direct formative and summative assessment and make these assessments more meaningful for students. In this workshop, participants will use one of three conceptual frameworks (homeostasis, cell-cell communications and flow down gradients or flux) to explore how these might be used in course design, student assessment and learning. There will be time for questions and discussion and applications in physiology teaching and learning. (NSF DUE-1043443) References: Michael, J. and McFarland, J. (2011) The core principles ("big ideas") of physiology: results of faculty surveys. *Adv Physiol Educ*. 25:336-341. McFarland et al. (in press 2016). A conceptual framework for homeostasis: development and validation. *Adv Physiol Educ*.

22.0: INNOVATIVE CURRICULA THROUGHOUT EDUCATION LEVELS

22.1
UNPACKING THE CORE CONCEPT OF CELL-CELL COMMUNICATIONS FOR UNDERGRADUATE OR PROFESSIONAL LEVEL PHYSIOLOGY COURSES

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A conceptual framework (CF) is an "unpacking" of a core concept into its constituent ideas (1). We have published a validated CF for homeostasis (1). More recently we unpacked the core concept of cell-cell communications and validated the CF that resulted (2). The cell-cell communications conceptual framework (CC-CF) is made up of seven critical components and 44 constituent ideas arranged in a hierarchy with as many as three levels. It is large, complex, and encompasses many physiological mechanisms. The CC-CF is more comprehensive, contains more ideas, than students in a typical **undergraduate introductory physiology** course are expected to learn. On the other hand, students in a **professional level physiology course** (medical, dental) might well be expected to learn it all. This thus raises the question of how to edit ("trim") the CC-CF (or any CF) to meet the needs of a particular course. In the process of preparing and then validating the homeostasis CF (1) we applied a simple rule: if fewer than 30% of our respondents rated the item as Essential it was dropped from the framework. The same rule applied to the CC-CF results in 17 items being eliminated, reducing the size and complexity of the CF. However, some of the items eliminated may be deemed important by a particular physiology teacher. An alternative rule would call for elimination of all items at the 4th level in our hierarchy (four items would be removed). While algorithmic approaches are appropriate for preparing CFs for dissemination to a wide audience, they may not serve the individual teacher well. A CF is, by its very nature, highly structured, and removing items according to some arbitrary rule may create a distorted representation of the core concept. CFs are meant as teaching and learning tools, and such tools must be adapted to the particular needs of the students. Nevertheless, the cell-cell communications conceptual framework offers teachers of physiology at any level a tool that can be customized to meet their needs. References: (1) McFarland et al., (in press). A conceptual framework for homeostasis: development and validation. Adv Physiol Educ. (2) Michael et al. (2016). A conceptual framework for the core concept of cell-cell communication. Abstract 553.20, Experimental Biology 2016, San Diego, CA. (3) McFarland et al (in preparation). (4) Modell et al. (2015). A physiologist's view of homeostasis. Adv Physiol Educ 39: 259-266.

22.2
LEARNING TO WORK TOGETHER: IN-CLASS TRAINING TO ENHANCE STUDENT COLLABORATION AND INCREASE LEARNING OUTCOMES

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Skill sets associated with team-based productivity are increasingly sought after by employers. As such, it is necessary for students to have opportunities to develop, evaluate, and hone these skills during their undergraduate programs. Anecdotally, students report poor experiences working in teams even though, individually, all students in the team are intelligent and capable of completing the task. This suggests that there are certain skills that must be learned in order to work effectively in a team. Therefore, it is necessary to develop tools that aid in training students to collaborate effectively. To begin addressing these issues, we sought to determine whether effective teams were capable of improving learning outcomes for students in those groups. In our undergraduate core natural sciences course, Scientific Literacy for the 21st Century, students work together in teams of four for the duration of the semester. In an effort to aid students in identifying what an effective team is, students in one section of our course were shown YouTube videos about teams working together with varying degrees of success. This led to an in-class discussion about factors that characterize "good groups" or "bad groups". Afterward, each team was tasked with developing their own rules that they would abide by in order to be an effective group. Each team member signed the document upon agreement of the rules. Teams were provided time every two to three weeks to reflect and discuss whether they were meeting the established rules for their team. This section constituted the experimental group, and each student was given an anonymous group effectiveness assessment at the end of the semester to evaluate qualities of their team and the impact their team had on their learning in the course. These results were compared against graded assessments in

the course, as well as pre- and post-course assessments. A different section underwent a similar group effectiveness assessment with comparison to graded tasks in the course, as well as the pre- and post-course assessment, however, this section was not given the extra tools to identify factors of an effective team throughout the semester. Preliminary analysis suggests that students exposed to tools that aid in the identification of factors that may contribute to an effecttive team typically have greater learning outcomes and higher grades.

22.3
EXERCISE PHYSIOLOGY MOBLAB-BASED PROTOCOL PROMOTES LEARNING AND STUDENT ENGAGEMENT

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The MobLabs (mobile learning laboratories) are smartphone applications (Apps) or small portable devices that facilitates teaching and learning process by acquiring scientific data. This pedagogical tool can alternatively optimize physiology practical lessons and meet the necessity for innovative and engaging didactic approaches by digital natives students. We aimed to create a practical lesson protocol to teach exercise physiology by using Apps in classes for higher education and K-12 students. Specific exercise physiology Apps were not available on virtual stores. However we identified, selected and applied several Apps, developed for different purposes, into one protocol of exercise physiology hands-on activity. To explore their potential for providing physiological status before and after physical activities, we tested a practical lesson protocol that includes endurance jogging, house cleaning, meditation and a control group. Six Apps were identified as useful to provide reliable and statistically significant results about heart and respiratory rate, forced expiration, vasodilation, muscular tonus and reaction time. Additionally, the protocol was effecttive to promote learning of exercise physiology concepts and student engagement. Thus, exercise physiology is an appropriate topic to use smartphone as MobLab, which allows student to experiment data collection similar to conventional laboratory methods. Funding sources: OCRC-FAPESP.

22.4
BIG BROTHER IS WATCHING

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Several summative assessments are often required before instructors can identify students at high risk for course failure. Since it is common in large lecture courses to have only a few hourly exams per term, this realization may not come until late in the semester. The widespread incorporation of online learning and course management platforms such as BlackBoard, Canvas and Moodle in universities across the country affords instructors access to a trove of student online behavioral data. In today's world in which government and commercial entities utilize data mining and analysis software with success, student activity report spreadsheets represent valuable sources of untapped data. Is it possible to discern online behavioral patterns that correlate with student performance on high-stakes, summative assessments? Might the student who has never downloaded the lecture notes fall into a predictive category of risk? Or, might the student who completes online assignments the same day they are posted be in some way predisposed for better assessment outcomes than a student who completes assignments an hour before they are due? Asking questions like these and others, we identified online behavior parameters that correlate with summative assessment performance in a cohort of undergraduate nursing students of Anatomy and Physiology. We then algorithmically integrated these parameters and assessed the algorithm for predictability post-hoc. Herein we report on our algorithmic approach to analysis of student online behavioral data for early-semester identification of students at high-risk of scoring poorly on lecture exams. Deploying similar models an early-warning system, instructors may be able to preemptively align students with learning resources of which they may be unaware such as supplemental instruction, free on-campus tutoring, office hours, and the like.

22.5
MOVING FROM MEMORIZATION TO CURIOSITY: ENCOURAGING STUDENT-DIRECTED LEARNING IN AN ADVANCED PHYSIOLOGY CLASS

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The college experience is ideally designed to develop students' creative, critical, and independent thinking skills. This goal is seemingly at odds with a traditional introductory Anatomy and Physiology course that emphasizes the memorization of nomenclature and the acquisition of facts. At Curry College, a small liberal arts college primarily serving a population of students who score low on standardized tests or are otherwise underprepared, we find that some of our students are more comfortable with memorization as a mechanism of learning and prefer it to more

inquiry-based methods. To support student-directed learning in this population of students, Advanced Physiology (a 3000-level course with the prerequisites of Cell Biology and Genetics) was designed to encourage curiosity and creative learning within a Human Physiology framework. Eleven third- and fourth-year students are enrolled in the inaugural semester of this course in Spring 2016. Three student-directed projects are integrated into this course, the first being an inquiry-based lab where students design, apply for IRB approval, and carry out a simple physiology experiment using the iWorx IX-TA teaching lab system that has been used throughout the semester. The second is a student-led book club, in which each student is responsible for integrating a chapter of Robert M. Sapolsky's "Why Zebras Don't Get Ulcers" with the physiological concepts presented in class and leading a group discussion with their peers. The third student-directed project is a midterm assignment in which students write four physiology questions related to topics covered in the first half of the semester, and then answer them. Students are encouraged to ask questions that really drive their curiosity, and are given a rubric that supports inquiry rather than finding a definitive answer or writing a case study. Researching "curious questions" is also used during the second half of the semester to encourage the students to observe physiology in their daily lives and bring that lived experience into the classroom. These methodologies will be assessed quantitatively by student performance on the final exam, and qualitatively by student self-assessment and a class survey.

22.6
PHYSIOLOGY UNDERSTANDING WEEK (PHUNWEEK): K-12 OUTREACH PROGRAM PROMOTES BOTH ENGAGEMENT AND IMPACT

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In 2005, the American Physiological Society (APS) established a member-driven outreach program, Physiology Understanding Week (PhUn Week) with five objectives: 1.) Increase student interest in & understanding of physiology in their lives. 2.) Introduce students to physiology as a possible career. 3.) Increase teacher recognition of physiology in the curriculum. 4.) Involve more physiologists in outreach to students & teachers in their communities. 5.) Reach a broad base of students (grades, gender, race/ethnicity). This study evaluates # 2, 4 and 5 and describes plans to evaluate # 1 and 3. PhUn Week was held each November, 2006-2016, engaging more than 96,500 students in grades pre-K to 12. APS members worked with local schools and teachers to plan and implement meaningful interactions between physiologists, trainees, and students. APS provided resources (hands-on lessons, career presentations, and follow-up materials). Reaching diverse students: Initially, PhUn Week involved primarily high school classroom visits. However, 2015 data show a balanced mix of primary (24%), elementary (21%), middle (21%), and high school (31%) events. PhUn Week has consistently garnered participation of both female (52%) and male (48%) students, and the percentage of PhUn Week students from minority groups underrepresented in science increased from 34% to 47% in eight years. Involving physiologists: Participation of physiologists and graduate/postdoctoral trainees increased more than 400% in the past ten years, providing students with direct contact with science role models. In addition, PhUn Week has expanded both across US and to 4 countries over the past decade. Although PhUn Week started as individual classroom visits, it has evolved into a variety of event types in museums, on university campuses, and at outdoor venues. These models provide flexibility in planning events and engaging both physiologists and students in different community types and countries. Career education: Career trading cards were developed to encourage students to explore careers at the APS career web following PhUn Week. Demographics are collected on number and length of visits. Data suggest that students not only keep the cards but use the links to access career information. Student interest: The poster will discuss possible evaluation methods for gathering student impact data from an international, volunteer-based program.

22.7
EDUCATORS RATE LIFE SCIENCE TEACHING RESOURCE COMMUNITY AS BOTH USEFUL AND USABLE

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The Life Science Teaching Resource Community (LifeSciTRC, www.lifescitrc.org) is a collaborative digital library and online educator community involving nine life science societies in areas including physiology, anatomy, developmental biology, genetics, plant biology, and science policy. In 2015, we conducted the first survey of registered LifeSciTRC users in order to gauge both the functionality and user benefits of the community site. The survey measured: (1) ease of using search, resource sharing, and community tools; (2) usefulness of resource metadata; (3) helpfulness of LifeSciTRC resources in teaching; and (4) satisfaction with the LifeSciTRC. Responses were collected from 316 educators. Overall, responses to all sections of the survey were positive, indicating that respondents found the LifeSciTRC easy to use and beneficial to their teaching. Analyses also will be presented by institutional level, par-

ticipation in the LifeSciTRC Scholars Program, and user demographics (gender and race/ethnicity). Future directions for new community tools and programs will be discussed.

22.8
EXPANSION OF UNDERGRADUATE PROGRAMS DURING THE VISION AND CHANGE ERA: PHYSIOLOGY AND OTHER LIFE SCIENCE FIELDS

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The *Vision and Change in Undergraduate Biology Education* reports cite the critical role of professional societies in undergraduate life science education and, since 2008, have called for their increased involvement in support of undergraduate education. A 2014 survey by the Professional Society Alliance for Life Science Education (PSALSE) explored the level of support being provided by societies for undergraduate education and documented changes in support during the *Vision and Change* era. Society representatives responded to a survey on programs, awards, meetings, membership, teaching resources, publications, staffing, finances, evaluation, and collaborations that address undergraduate faculty and students. A Comparison Group of societies responded to similar surveys in both 2008 and 2014. Results indicate that life science professsionnal societies are extensively engaged in undergraduate education in their fields, setting standards for their discipline, providing vetted education resources, engaging students in both research and education, and enhancing professional development and recognition/status for educators. Comparison Group responses indicate that there has been a significant expansion of undergraduate efforts in several areas since 2008. This poster will also document changes in undergraduate programs by the American Physiological Society during this period, describing increased offerings of undergraduate research experiences, faculty development, and teaching resources. Directions for future undergraduate programs and resources will be discussed. References: AAAS. (2009a). *Vision and Change in Undergraduate Biology Education: Preliminary Reports of Conversations.* Washington, DC: AAAS; AAAS. (2009b). *Vision and Change: A Call to Action.* Washington, DC: AAAS; AAAS. (2011). *Vision and Change in Undergraduate Biology Education: A Call to Action*. Washington, DC: AAAS; AAAS. (2015). *Vision and Change in Undergraduate Biology Education: Chronicling Change, Inspiring the Future*. Washington, DC: AAAS.

22.9
AGENTS OF CHANGE - MOVING FROM SURVEY TO EXPERIENTIAL LEARNING: REVAMPING UNIVERSITY OF ALASKA ANCHORAGE'S INTRODUCTORY BIOLOGY COURSE ALONG VISION AND CHANGE GUIDELINES.

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The University of Alaska Anchorage (UAA) department of Biological sciences became "agents" of change and revitalized their curriculum to start in academic year 2015/2016. The cornerstone of the transformation was to take a two semester introductory biology survey course (8 total credits) and create a one semester lab intensive introductory biology course (6 credits; BIOL A108). Experiential learning is essential for many professions, including scientific fields of study. UAA biology students were immersed in the scientific method to explore biological questions based on current knowledge and to experience the challenges of experimental design and data collection. Pre/post tests were given to track student progress and quantify student retention of relevant material. One of the major goals of AAAS Vision and Change is to help higher education biology courses retain more students thereby improving student continuation in STEM related fields. In this we were successful: historically the 2 semester survey introductory course reported withdrawal rate of 16%, however when including failure rates this percentage consistently increased to over 20% for each semester analyzed (2012 to 2014). In contrast, 96% of students passed (defined as a C or better grade) with a withdrawal rate of 2% in the first semester (Fall 2015) of our experiential learning introductory biology course (BIOL A108). Data from the current semester (Spring 2016) suggest similar trends. A second goal of the program revision was to increase student learning and engagement in the process of science; in this our data suggest we were successful. Within one month of BIOL A108, students have improved their use of the scientific method to tackle challenging biological questions and core concepts. Preliminary assessment data show 96% of BIOL A108 students can create and use hypotheses statements correctly. Additionally, BIOL A108 student pre/post data indicate a 25% improvement in their comprehension of Mendel's principles. Since UAA draws students from the local area which has a highly diverse population, the increased student retention and learning in UAA's new introductory biology course may lead to a greater proportion of non-traditional and minority students continue in biology and other STEM related fields.

22.10
REINFORCING AND TRACKING PREREQUISITE CONCEPT MASTERY IN PHYSIOLOGY USING TWO-STAGE COLLABORATIVE TESTING

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Instructors and administrators frequently establish prerequisite requirements to ensure that entering students possess the background knowledge to succeed in their course. However, prerequisite courses can only benefit students if they have mastered the course concepts, and can only benefit instructors if they can accurately measure student mastery. In this study, we used two-stage collaborative testing to assess and reinforce student understanding of key prerequisite concepts across a two-course physiology sequence. The final exam of the first course included ten questions addressing concepts particularly relevant for the second course. During the first week of the second course, students were given a paper quiz consisting of the same ten questions, and were asked to provide their individual answer and confidence level for each. Students provided their name for tracking purposes, but were assured that the activity was not for marks. Students subsequently worked in small groups to come up with a first group consensus answer, and then used IF-AT scratch cards to discover the correct answer. The activity was followed with an extensive instructor debriefing, and students recorded when they first understood the concept and their perceptions of the activity. Individual scores were used to identify students that might potentially benefit from additional remediation, and the pooled scores for each question revealed which topics needed to be revisited in greater detail. The average individual quiz scores were 63 +/- 2.8%, vs. 88 +/- 2.4% % for the first group answer. When the data from all questions and students were pooled, students reported that they understood the concept addressed by the question prior to the activity 52% of the time. Of the concepts that were not understood prior to the activity, 56% were understood after the group discussion and 41% after the instructor explanation; only 3% were not understood after the activity. Student perception data revealed that they found the activity useful and enjoyable. In conclusion, no-stakes collaborative testing can be an effective introductory activity for courses with prerequisite requirements, enabling students to activate previous knowledge and to prepare for future collaborative learning activities. This activity also enables instructtors to identify prerequisite concepts requiring remediation prior to covering new material, and to identify students needing extra help.

22.11
TEAM-BASED ASSESSMENT OF NEPHRON STRUCTURE AND URINE FORMATION USED AS A FORCED RETRIEVAL ACTIVITY TO IMPROVE STUDENT LEARNING

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An underlying topic in A&P is the complementarity of structure and function. In the urinary system it is crucial for students to understand that structure will determine function. The relationship of the structure to the function of the nephron is a perfect example of this theme. Students tend to overlook this relationship. Additionally, the kidneys play a major role in many homeostatic control mechanisms. Students' success in subsequent courses and programs depends upon a good understanding of this learning outcome. Moreover, the nature of urine formation runs contrary to most physiological processes and is a source of confusion for the students. Feedback from students on the pre-quiz, addressing the Unit Learning Outcome: Describe the structure of the nephron and relates its structure to the functional processes of urine formation, indicates students have difficulty with this topic. The goal of mastery and more durable learning of the unit learning outcome prompted the development of a new in-class forced retrieval activity and new exam question. As with all topics in the course, students are initially exposed to the material via a pre-class reading assignment followed by a pre-quiz that is to be completed before coming to class. In-class exposure to the material is via concept questions and peer discussion/instruction. Both the concept questions and peer instruction force students to retrieve material they have previously read. Forced retrieval leads to more permanent and durable learning. Another activity was developed to assist and assess student learning of the learning outcome stated above in addition to other forced activities currently used in this course. The activity included a collaborative learning session as part of a 'team' peer instruction opportunity where students were required to come to consensus regarding the answers and in an attempt to make the learning more authentic students were allowed to use class notes and textbooks. This team-based, forced retrieval activity was designed to be a formative assessment that provided low risk/low stress feedback to students and instructor alike and was followed up with a new question for the unit exam to assess student learning of the learning outcome stated above. The answers for the activity were discussed in the following class meeting. Distribution of student responses for each question was examined for both the individual and team-based assessment to identify misconceptions and weaknesses in understanding. This information was used to write the unit test question. Student

responses to the unit test question demonstrated improved understanding of the topic and better than acceptable achievement of the learning outcome.

24:0 PLENARY LECTURE V

24.1
SUPPORTING FACULTY: RESOURCES FROM PROFESSIONAL SOCIETIES AND ONLINE COMMUNITIES

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For higher education faculty at all levels and in all science fields teaching is changing rapidly. Incorporation of student-centered teaching methods such as flipped classrooms and authentic research experiences, use of blended and online-only formats for course delivery, expansion of assessment methods to gather richer student data, and online/on demand formats such as MOOCs have compelled faculty to venture away from the tradition and familiarity of the lecture model into new arenas. For many faculty members, especially those at smaller institutions with limited science faculty, these forays into the unknown are done single-handedly, without collaboration or support from colleagues. Fortunately, changing technology also provides opportunities for professional societies and online communities to play an increasing role in supporting faculty development and science teaching. Although working in isolation, the lone science faculty member at a rural college can find resources tested by colleagues teaching the same type of course, join a multi-institutional collaboration to evaluate a new lesson, or have her students team with students from another country to do a research project. More importantly, she can have direct conversations with colleagues about the teaching issues that are most problematic or most rewarding. This presentation will explore sources of teaching resources, professional development, collaboration, and recognition by professional societies and other organizations. Data sources include surveys of life science professional societies and online community users. Opportunities for community involvement, including methods evaluation and research collaborations, will be discussed and ideas for additional society programs and activities will be solicited.

25:0 WORKSHOP I*

25.1
TEACHING PHYSIOLOGICAL CONCEPTS BY ENHANCING STUDENT VISUAL LITERACY

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Students learn by building on a framework of prior knowledge. As educators, how do we provide experiences for students to recognize and enhance this process? This workshop will engage participants in specific activities for the classroom that prompt student discussions of how assumptions informed by prior knowledge, social identity, diversity, and team dynamics affect learning, data interpretation, decision-making, critical thinking, and creativity in the sciences. As a means to focus on these specific ideas, visual interpretation of patterns and information, often known as visual literacy, provide a platform for discussion that allows for simple implementation and diverse interpretations. Workshop participants will brainstorm and discuss how these activities and discussions benefit their respective student population, including how experience and practice of these reflective exercises will enhance and promote student learning, ability to apply information, and creativity to better understand physiology. References: Jones, M.G. & Brader-Araje, L. 2002.The impact of constructivism on education: Language, discourse, and meaning. *American Communication Journal*, 5(3), for discussion of constructivism of learning through connecting new information with prior knowledge. Williams, T.L. 2007. "Reading" the painting: Exploring visual literacy in the primary grades. *The Reading Teacher*, 60(7):636-42, for a basic explanation of visual literacy. Schönborn, K.J. & Anderson, T.R. 2006. The importance of visual literacy in the education of biochemists. *Biochemistry and Molecular Biology Education*, 34(2):94-102, for specific guidelines for teaching and learning visual literacy in the sciences.

26:0 WORKSHOP II*

26.1
STATISTICS IN EDUCATIONAL SCHOLARSHIP

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When I teach and write about statistics, I want to engage my audience. To do this I use simulations as thought experiments my audience can see. This workshop provides an opportunity to do just that: we will investigate basic concepts in statistics using simulations that will mimic hands-on exploration. Workshop participants will explore how these concepts are critical to the conduct, analysis, and interpretation of educational research. References: Curran-Everett D. Explorations in statistics: hypothesis tests and *P* values. *Adv Physiol Educ* 33: 81–86, 2009. Curran-Everett D. Explorations in statistics: confidence intervals. *Adv Physiol Educ* 33: 87–90, 2009. Curran-Everett, D. Explo-

rations in statistics: permutation methods. *Adv Physiol Educ* 36: 181–187, 2012.

27:0 WORKSHOP III*

27.1
INSTRUCTORS' UNDERSTANDING OF STUDENT'S MISCONCEPTIONS CAN IMPROVE MEANINGFUL LEARNING

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Students' knowledge of physiology derives from previous learning experiences. Bransford, Brown, and Cocking (1999) state that students shape new knowledge and understandings grounded on what they already know. Oftentimes, what students know and believe doesn't match what is physiologically correct, and that can become a barrier to learning. Identifying students' misconceptions and helping the students directly confront them is essential for effective teaching. As instructors of physiology, we need to recognize that students have to construct their own conceptual understanding. Students use new information and their current knowledge to make accurate connections with instructtion and not merely absorb the knowledge (Wiliam, 1994). Learning complex material, such as concepts in physiology, can occur differently for every student. For example, a student may have some related knowledge of the to-be-learned concepts but no prior knowledge. Students' existing knowledge and the process they use to link it with new knowledge influence the learning outcomes achieved. Therefore, both need to be taken into account through teaching. By taking into account students' thinking, physiology instructors can respond to and interact with the students to provide the opportunity for the student to improve their conceptual understanding. Therefore, the student is able to make a conceptual change and progress from novice to expert. As a physiology instructor, I am astonished when I learn that despite using the best instructional strategies, students do not grasp the concepts covered in class. Some of the top students give the correct answers with correctly memorized words, but when asked for more information, these students reveal their failure to fully understand the underlying concepts. Crouch and Mazur (2001) described students who had high algorithmic ability but had trouble answering parallel conceptual questions. Therefore, the learner has to recognize a feature they had not realized before, either instinctively or by having it pointed out during instruction. It is important for the learner to notice the differences, but they also must be able to accurately remodel their framework. With appropriate instruction the learner's new framework will more closely match a physiology expert's framework. The learning goals of the workshop are: 1.) Descriptions of the types of misconceptions with examples types of some common homeostatic misconceptions. 2.) Methods to identify misconceptions. 3.) Methods to break down misconceptions. 4.) The approach students take to confront and overcome their misconceptions. 5.) The role of misconceptions in the learning process. (Supported by NSF grant DUE-1043443). References: Crouch, C.H. & Eric Mazur, E. (2001) Peer instructtion: ten years of experience and results. Am. J. Phys.69 (9) pg. 970-977. National Research Council (NRC) (1999) How people learn: brain, mind, experience, and school, expanded edition. Bransford J.D., Brown A.L., Cocking R.R., editors. National Academies Press; Washington, DC. Wiliam, D. (1994). Reconceptualising validity, dependability and reliability for national curriculum assessment. pg.11-34. In D. Hutchison & I. Schagen (eds.), How reliable is national curriculum assessment? Slough, UK: National Foundation for Education Research.

28:0 PLENARY LECTURE VIa*

28.1
CENTRAL ROLE OF PHYSIOLOGY IN THE PROFESSIONAL CURRICULUM

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Too often, physiology instructors ask "How many hours are needed for Physiology instruction?". This reflects a view that footprint in the curriculum calendar is a measure of worth. It is exactly the wrong question, as it leads to a contest with other instructors, mistakenly interpreting curricular hours as a measure of student attention. Physiology is an essential foundational science, and as physiologists, we have to stop fighting a battle that we have already won. Medical students realize this, and appropriately recognize that a foundation in physiology is essential to their progression through their clinical training. Each spring, the Association of American Medical Colleges (AAMC) administers a survey to the allopathic medical graduating classes in the USA. Analysis of these results shows the exceptional strength of the discipline of physiology regardless of the instructional format. The survey also shows opportunities for further enhancement, and it is these opportunities that provide a vehicle for further strengthening the discipline of physiology. When the class graduating in 2015 was asked "How well did your study of the following sciences basic to medicine prepare you for the clinical clerkships and electives?" (Scale 1 = Poor and 4 = Excellent), Physiology scored 3.4, and ranked 3rd of the 14 basis sciences. The two areas that scored higher than Physiology were "Introduction to Clinical Medicine/Clinical Skills" (3.5) and "Patho-

physiology of Disease" (also 3.5). Physiology and Pathophysiology rank in the top 3 disciplines, and are valued because of their relationship to clinical study. Moreover, these rankings have remained constant for the past 4 years. In contrast, these same students rated "Basic science coursework had sufficient illustrations of clinical relevance" as 3.9 of 5.0, and "Required clinical experiences integrated basic science content" as 4.0 of 5.0. These are two areas that physiology instructors must address in order to further strengthen physiology instruction in the medical curriculum. Physiology as a discipline is strengthened when we incorporate select clinical applications to illustrate physiological control mechanisms. In addition, physiology instructors must better collaborate with our clinical colleagues to strengthen the mechanistic emphasis on disease processes in the clinical years.

29:0 PLENARY LECTURE VIb*

29.1
THE PIPELINE OF PHYSIOLOGY COURSES IN COMMUNITY COLLEGES

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Community colleges are significant in the landscape of undergraduate STEM education (NRC 2012) and are particularly important for biology and pre-professional students. A third of medical school applicants in 2012 had attended a community college (CC): while in high school, in their first 2 years of college and post-baccalaureate. The CC pathway is particularly important for the education and service of traditionally under-represented groups. Pre-medical students who first attend CC are more likely to practice in underserved communities as physicians. (AACC 2012) There is great diversity amongst CCs. Some 2-year colleges focus on technical degrees, certificates and job training, but the role of CCs has changed over the decades and now emphasizes associates degrees and transfer programs to 4-year institutions. CCs have significant advantages over 4-universities for many students. Pragmatically, they are more affordable, local and offer flexibility to accommodate students' work and family commitments. Academically, community colleges offer teaching faculty, smaller class sizes and accessible learning support systems. CC student populations are more diverse that their 4-year counterparts. So CCs are fertile ground for 4-year colleges and medical schools to recruit students and lend support. Challenges do exist for CC students (Labov 2012). There are often limited interactions between 2 and 4-years institutions and the ease and types of implementation of articulation agreements vary. There is a need for additional mechanisms to broaden participation of CC students in research, internships, volunteer opportunities and mentoring. Many existing mechanisms do not understand the constraints that limit CC-students' access to opportunities, including place-bound and financial constraints, language, age and culture. With the growing demand for health care professionals and there is an increased demand for introductory biology, physiology, and anatomy & physiology (A&P) courses. In this talk we will focus on the introductory biology series and A&P courses for life science majors and pre-professional students at CCs. References: NRC & NAE. (2012) *Community Colleges in the Evolving STEM Education Landscape*. NAP. Labov, J.B. (2012). Changing and Evolving Relationships between two-and four-year colleges and universities. CBE LSE 11:121-128. AACC (2012). http://www.aacc.nche.edu/Publications/datapoints/Documents/MedicalPipeline.pdf

30:0 WORKSHOP IV*

30.1
PHYSIOLOGY MAJORS INTEREST GROUP

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There has been a recent growth and evolution of physiology as a stand-alone undergraduate major (not just departments that offer a course on physiology). While this major has existed in a few isolated cases with great success (e.g, University of Arizona and Michigan State University), the last decade has seen the conversion of several programs from Kinesiology to Physiology, Human Physiology, Integrative Physiology, etc. These programs are typically seeing 3 to 5-fold increases in enrollment over the last five years, and are becoming the primary pathways for many students heading into medicine, physical therapy, and other allied health professions. From an APS perspective, if we want a strong pipeline of future researchers, teachers, faculty, this may be the best place to foster development. Most of these B.S. programs function in isolation and are being created without a broader conversation, each is inventing its own wheel, based on local faculty interests and visions. The symposium will explore: 1) State of Undergraduate Programs in Physiology, 2) National Trends in Undergraduate Physiology Curriculum, 3) NSF funding of Physiology BS programmatic assessment, 4) Professional Skills Development for physiology undergraduates, 5) Career Trends for BS Physiology Students. The goal of this interactive session is to start a national conversation, share ideas among peer programs, and form an interest group. The target audience is members of APS who are currently involved in these programs or may be interested in developing them.

31:0 WORKSHOP V*

31.1
FACILITATING SMALL GROUP DISCUSSION (BASIC)

Sydella Blatch[1], and Carol Schmidhauser[1]
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Discussion is a powerful tool for active learning. It allows the participants to probe their understanding and misconceptions and to synthesize new ideas while acknowledging and valuing the contributions of others. The key to facilitating small group discussions involves the use of different perspectives and different techniques to create an inclusive environment. In addition, it is important to consider the characteristics of effective discussions and conditions that promote small group interaction and engagement. In this workshop, we will discuss parameters to consider when deciding which types of activities lend themselves well to small group discussions, determining the structure and composition of small groups, strategies for fueling group discussions, and how to assess group work. By working in small groups, by the end of this workshop participants will have created an action plan for using group activities in a course or setting of their choosing.

32:0 WORKSHOP VI*

32.1
FACILIATATING SMALL GROUP DISCUSSION: ADVANCED

Betsy Jones[1]
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Our current post-lecture landscape for millennial learners in higher education challenges us to deliver teaching content that is interactive and that engages students in developing skills they will need as scientists and clinicians—among them teamwork, problem solving, and preparing to be lifelong learners. Developing, implementing and facilitating small group activities is an important means for teachers to allow their students to participate in active—rather than passive—learning that is collaborative and self-directed. This workshop will outline characteristics of effective small group learning activities and discuss means to reshape traditional lecture content to take advantage of small group settings. It will address techniques for writing learning objectives, developing materials, and training faculty to participate and facilitate learners' engagement. Employing those same techniques, participants will have the opportunity to work together to brainstorm, outline, and share small group session ideas. References: Gavriel J. Teaching tips for small-group facilitation. Education For Primary Care: 2015;26(2), 102-104. Kilgour J.M., Grundy L., Monrouxe L.V. A Rapid Review of the Factors Affecting Healthcare Students' Satisfaction with Small- Group, Active Learning Methods, Teaching and Learning in Medicine, 2016;28:1, 15-25.

33:0 BEST PRACTICES IN UNDERGRADUATE PHYSIOLOGY

33.1
ORIGINAL COURSE-BASED UNDERGRADUATE RESEARCH EXPERIENCE TO DETECT PARASITIC WORMS IN LOCAL PARKS IN TWO UNDERGRADUATE COURSES

Carol Schmidhauser[1], and Sydella Blatch[1]
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Traditional "cookbook" labs are a missed opportunity for students to participate in novel research and may not strongly reinforce skills needed for or interest in laboratory research. Studies have shown that course-based undergraduate research experiences (CUREs), where students conduct original research as part of a traditional course, can increase student persistence in science and understanding of the scientific process. Many course-based research projects either focus on experimental questions to which the answers are already known, or rely on methods that the instructor has already successfully applied to very similar experimental questions. This course-based research experience differed in that experimental outcomes were not known in advance, and the instructors had little experience with significant portions of the methods used. Thus, students were almost entirely responsible for researching and developing the methods they needed to try. The students were given only their experimental aim and a brief overview of the methods that could be used. The overall aim of the experiment was to identify the presence of *Toxocara spp.* and *Toxoplasma gondii* worms in the Baltimore MD area. Parasitology students were tasked with collecting soil samples from a number of local parks, and isolating and identifying the parasite eggs from soil samples using floatation methods. Animal Physiology students were tasked with detecting parasite genes in DNA isolated from soil using PCR. It was the responsibility of the students to locate relevant primary literature, use the literature to propose specific methods, develop experimental controls, determine the materials and equipment needed, perform the methods, analyze the outcomes, and return to the literature to refine unsuccessful methods. Surveys of the students show that their perceptions and thoughts about the experi-

ence were diverse. However, a majority of students felt the experience was frustrating, but improved their research ability, ability to analyze data, understand primary literature and work independently. We provide considerations for the implementation of such experiences. Future studies will examine effects of the experience on specific, measured outcomes of laboratory and quantitative skills, and attitudes towards science. Use of this kind of laboratory research question could easily be adapted to a CURE in biology courses of many types, as the presence of any kind of organism can be detected from a host of environmental samples.

33.2
EFFECT OF COLLABORATIVE EXAMS ON RETENTION IN AN UPPER DIVISION PHYSIOLOGY CLASS

James Cooke[1]
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In an attempt to help our upper division physiology students increase their retention of key course concepts, we have been using two-stage group exams. During these exams, students write their exams, submit them, and then get into groups to re-write all/some of the original exam. The idea is that the feedback they receive from peers help clarify any difficult concepts and improve retention of course material (Gilley and Clarkston, 2014). Similar attempts have been made to use collaborative exams in physiology classes (Cortright et al., 2003), but these studies had methodological problems controlling for the 'testing effect' (Roediger and Karpicke, 2006), in which multiple exposures to material in a test setting increases retention. The model employed by Gilley and Clarkston (2014) used a pseudo-randomized cross-over design in which each student re-writes a question from the exam individually and in a group setting, and they found that collaborative exams increased retention of course material 3 days after the exam itself. Using their cross-over design to control for the test effect, we wondered whether we would see an increase in retention of material in our physiology class at longer time points (3 and 6 weeks), using open-ended questions instead of multiple choice questions used by Gilley and Clarkston (2014). We followed our initial design with a modification that allowed students to re-write answers to questions individually after discussing the question in a group. We will discuss the results of our study and the implications for whether collaborative exams might be a useful tool to increase retention of key physiology concepts.

33.3
AN INTERRUPTED CASE STUDY APPROACH FOR A DIABETES LAB EXERCISE

Nancy Aguilar-Roca[1]
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Students are interested in understanding Type 2 Diabetes, but it can be difficult to set-up a relevant teaching lab activity because of safety issues related to using human urine and blood. Rats can be problematic because of the expense, IACUC protocols and the technical difficulties of working with live animals. The approach I used was to substitute a traditional wet lab activity with a dry lab using stuffed animals as part of an interrupted case study. Students were given a population of "rats" and their goal was to determine which rats had diabetes, which rats had diabetes but had been part of an exercise study, which rats had uncontrolled diabetes and which rats were non-diabetic controls. Students were given simulated data in stages, starting with basic information about the rats such as diet, eating/drinking habits and urine output. They had to weigh the rats to receive information about weight gain/loss. Students then made predictions about which rat was which and researched what clinical tests would be the most useful. I created simulated urine strips (Multistix 10) and metabolic panel data for students to read and decide if they should revise their predictions. After students had identified which rats were controls or diabetic, they had to look more carefully at the diabetic rats that appeared to be part of an exercise study. They were told that half of the rats were in a high intensity interval training (HIIT) study and half were from an aerobic exercise study. Students develop hypotheses about which group would be more insulin sensitive, select relevant cellular markers (e.g. protein or mRNA expression), and decide what data they wanted to collect to test their hypotheses. The final set of simulated data was from a VO2-max test, a hyperinsulinemic euglycemic clamp, and assays for GLUT4 and PGC-1alpha. The data may or may not have supported their hypotheses, leading to discussions about lab protocols and cellular mechanisms of diabetes. Overall, students felt that although the stuffed animals were silly, they appreciated having something physical and found it challenging to think critically about all of the test results. This approach could be modified for use in a classroom.

33.4
USE OF ANIMAL GASTROINTESTINAL TRACTS IN UNDERGRADUATE PHYSIOLOGY COURSES

Cristina Caldari[1]
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Non-preserved animal gastrointestinal (GI) tracts are a cost-effective sustainable resource around which low-preparation, student-satisfying, active learning laboratory exercises can be built for undergraduate physiology courses. Porcid

GI tracts, which are virtually available for free all over the US, are best suited for teaching human digestive anatomy and physiology. Tracts from small ruminants (deer, sheep, goats) can be easily obtained, usually at no cost, and used to emphasize the GI adaptations of herbivorous animals. Use of animal organs (offal) is a sustainable and inexpensive way of introducing active learning into the undergraduate physiology classroom. Animal tracts are routinely used as teaching tools in animal science courses, both at the undergraduate and high school level. Slight modifications in the methodology related to their preparation and use can allow for their introduction into non-agricultural programs, also. I will discuss the methodology involved in obtaining, transporting, and processing the GI tracts, as well as some of the safety mechanisms that need to be in place in order to avoid student or instructor exposure to zoonotic diseases. Additionally, I will detail several learning objectives that can be associated with this exercise, which should include, but are not limited to, anatomical and histological identifications, observation of luminal contents throughout the different parts of the tract, and comparing and contrasting the anatomy of omnivore and ruminant tracts if both are available. Some major anatomical differences that should be emphasized include the size of the cecum in the pig compared to the human and the chambers of the stomach in ruminants compared to omnivores, both of which can be used to introduce or expand the discussion of the role of symbiotic microbes in forage digestion. Additionally, the lack of a gallbladder in herbivores, if a tract is available, can lead to a discussion of the function of bile in lipid digestion. Longitudinal data on student satisfaction will be presented, and plans for improvement of this laboratory exercise based on the collected data will follow. I will conclude with a discussion on the use of abattoir offal as teaching tools for other physiological systems (reproductive, respiratory, excretory, and cardiovascular).

33.5
PHYS-MAPS: AN ASSESSMENT TOOL TO MEASURE STUDENT LEARNING IN UNDERGRADUATE PHYSIOLOGY PROGRAMS

Katharine Semsar[1], Michelle Smith[2], Sara Brownell[3], Brian Couch[4], Alison Crowe[5], Scott Freeman[5], Mindi Summers[2], Christian Wright[3], and Jennifer Knight[1]

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Setting educational goals and assessing student learning of those goals are fundamental steps to improving both teaching and learning. The recent *Vision and Change* report provides faculty and institutions with a set of core concepts and competencies in biology to use as a guide to assess students' understanding as they progress through an undergraduate biology curriculum. To meet this assessment goal, we have established an NSF-funded, multi-institution collaboration to develop a suite of assessment tools (Biology Measuring Achievement and Progression in Science: Bio-MAPS) that will measure student learning across a curriculum. By using these tools to measure students' conceptual understanding at multiple points throughout a program, data will be available to faculty, department chairs, and deans to help understand and gauge student learning, and could also be provided to accreditation agencies as evidence of programmatic success. Here we present the development of one of the Bio-MAPS assessments, the Physiology-MAPS (Phys-MAPS). The Phys-MAPS is designed to address two frameworks of key concepts in physiology: 1) the BioCore Guide, which elaborates on the Vision and Change core concepts, and 2) the top seven Core Principles of Physiology outlined by Michael and McFarland. We iteratively developed questions using faculty feedback (n>32 faculty) and student interviews (n>79 students) to help collect evidence of face and content validity. We conducted a national pilot in Fall 2015, collecting data from 14 undergraduate institutions, 21 courses, and over 2600 students ranging from sophomores to seniors. Using a 3PL item response model, we eliminated poorly discriminating items, and refined others using additional student interviews. The final assessment includes 13 question stems, each with 6 associated statements (78 total items); students select each statement as likely/unlikely to be true. Student performance on the pilot demonstrated that the Phys-MAPS discriminates well, with the top quarter of students scoring an average of 75% correct and the lower quarter of students scoring 49% correct. The assessment also includes items with a range of difficulties in each of the concept categories, allowing the Phys-MAPS to reflect both easier and more difficult sub-concepts within the larger concept categories. Together these data suggest the assessment will be useful for tracking student progress through a physiology program. We are piloting the revised Phys-MAPS nationally in April 2016 and will present these results.

33.6
REVISITING THE PHYSIOLOGICAL COST OF NEGATIVE WORK: A TEAM-BASED ACTIVITY FOR UNDERGRADUATE EXERCISE PHYSIOLOGY STUDENTS

Steven Elmer[1], and Matthew Kilgas[1]

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We implemented a team-based activity in our undergraduate exercise physiology laboratory that was inspired from Abbott's classic *Journal of Physiology* (1952) paper titled "Physiological Cost of Negative Work". These investigators connected two bicycles via one chain. One person cycled forward (active muscle shortening, positive work) while the other resisted the reverse moving pedals (active muscle lengthening, negative work) and the cost of positive and negative work was compared. This study was the first to link human whole-body energetics with isolated muscle force-velocity characteristics. Our students (n = 35) were asked to not only review this paper but re-enact the original experiment. The goals of the activity were to: 1) integrate previously learned concepts and techniques into a comprehensive end of the semester experiment, 2) preserve the history of Abbott's work, and 3) connect negative work to sport training and rehabilitation. For each laboratory section, students (~12/section) split into two teams (positive work vs. negative work). One student from each team volunteered to cycle against the other for 10 min. Remaining students in each team were tasked with collecting: 1) oxygen consumption, 2) blood lactate, 3) quadriceps muscle activity, and 4) perceptual responses. Students discovered that oxygen consumption during negative work was ~1/2 that of positive work and quadriceps muscle activity was also substantially lower. Majority of students (75%) strongly agreed that they stayed engaged during the activity and that it improved their understanding of exercise physiology. All students recommended that this activity be performed again. This laboratory activity was highly engaging, emphasized teamwork, yielded robust results with a good signal to noise ratio, was well received by students, and highlighted classic physiology work.

33.7
TEACHING UNDERGRADUATE ONLINE SCIENCE COURSES WITH LABORATORY COMPONENTS

Joan Lafuze[1]

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I am a systems medical physiologist who has taught undergraduate physiology for 29 years. I attended summer workshops three consecutive summers at Indiana University Bloomington that were held jointly by the Department of Education and the Department of Information Technology to learn how to develop and present courses employing "Distance Education Strategies" (Interactive video, hybrid and all online classes). Twenty years ago I launched my first all online class. A decade ago I was challenged by the then Vice-Chancellor of Academic Affairs to design an all online physiology course including laboratory components. Teaching that course has been very rewarding. In this workshop I plan to present what I believe to be critical elements in developing such a course. I will also address how I have advanced in helping learners achieve success, discovering advantages and disadvantages of using such strategies and meeting resistance both from learners and many others related to teaching laboratory science courses online.

33.8
TECHNOLOGY AND PUBLISHERS' MATERIALS IN TEACHING PHYSIOLOGY TO UNDERGRADUATE LEARNERS

Joan Lafuze[1]

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In this workshop I am hoping to become part of a community of persons who are in conversation about active learning strategies. I have been part of such conversations by participating in Indiana University Workshops, The Institute for Emerging Leadership in Online Learning (IELOL) and Quality Matters. Finding such communities where the conversation has addressed such strategies with undergraduate learners in physiology has been difficult. When it appeared as a topic for this workshop, I made a special effort to attend. I propose to bring my efforts to integrate technology with existing publishers' materials to address coordinating learning outcomes with text information, using clickers to assess learner progress and to manage employing available voice-over strategies for creating "flipped classroom-like" endeavors. I will share my experiences with choosing technologies, published instructor resources and course delivery. I am also interested in sharing conversations about using active learning strategies to improve student retention and student success in moving from the classes I teach to their future needs for what they are expected to learn in an introductory physiology class.

33.9
A GRADING SCHEME TO INSPIRE HOPE AND FOSTER GRIT IN A LARGE UNDERGRADUATE PHYSIOLOGY COURSE

Kevin Strang[1]

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Traditional grading schemes commonly used in science courses have the potential to inhibit the motivation and learning of at-risk students, and thus contribute to the achievement gap. A substantial literature suggests that curricular efforts to increase the "hope" that students feel can increase expectations for success, coping strategies, achievement of goals, and graduation rates, and that the amount of grit students demonstrate is a positive predictor

of college success. The present work represents a multi-year effort to optimize the grading scheme in a large undergraduate physiology class to inspire hope and foster grit among students. Methods employed have included the development of a mathematical method for rewarding improvement in an objective way, using discovery-based presentation to explain the grading scheme, and educating learners about "Fixed" and "Growth" mindsets. Preliminary results suggest that while these efforts have not had a major impact on grades of the class as a whole, individual students who might otherwise have lost hope and given up have benefited significantly. This presentation will address the role of hope, grit, and mindset in student success, and report the results of this grading scheme as it has evolved over the past 4 years.

33.10
INVESTIGATING THE EFFECT OF QUESTION ORDER ON STUDENT UNDERSTANDING OF STRUCTURE AND FUNCTION

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Written assessments provide insight into student understanding by allowing students to assemble their ideas. Surface features, such as the order in which questions are presented, may influence student explanations by acting as a knowledge retrieval cue. Yet, it is critical that physiology students have a robust understanding of concepts like structure and function that they can consistently apply regardless of context. We investigated how changing question order reveals students' level of physiological understanding. We hypothesized that asking students to give examples provides a retrieval cue to help them formulate definitions. We collected data from 131 students in a junior level general physiology course at a large southeastern public university. The class was randomly split in half and each half received two short answer questions in different orders; 1) (Order DX) **D**efine the principle of complementarity *followed by* Give an e**X**ample of the principle of complementarity from the human body, and 2) (Order XD) Give an e**X**ample of the principle of complementarity from the human body *followed by* **D**efine the principle of complementarity. We coded student responses for the presence or absence of the concepts structure and function, and whether students linked structure and function in their responses with an interrater reliability of the human coding above 0.70 kappa. We then used text analysis to extract words and phrases relevant to structure and function from student responses. Human coding showed that when asked to define the principle of complementarity, only 2% of students with order DX and 17% of students with order XD were able to link structure and function. When asked to provide examples, students with order DX (41%) performed better than students with order XD (26%). Using text analysis we created 25 categories of words and phrases, and identified levels of organization of the structures within student responses. For both question orders, students used multiple levels of organization when providing examples. However, DX students gave more organ examples, while XD students gave more cellular component and tissue examples. Results from our study indicate that students struggle with linking structure and function regardless of question order. In future studies we will investigate how additional question features, such as cognitive level and guiding context, influence student responses and reveal their physiological understanding.

33.11
ASSESSING THE EFFECTIVENESS OF STUDENT LEARNING AND ENGAGEMENT IN A CONTENT HEAVY FLIPPED CLASS

Joost Monen[1]
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Studies have shown that greater student learning gains are achieved during active learning, when students must apply their understanding solving problems, in application exercises, in discussion-based activities, and during team-based learning. Because these active learning approaches can take up valuable class time, leaving less time for content, it is often difficult to integrate them into a content heavy lecture course. Using a "flipped classroom" approach, student-centered learning activities can be routinely integrated into the classroom while not having to sacrifice content. To test the effectiveness of the flipped classroom approach, this study assesses a 300 level Genetics class by comparing outcomes of students in a flipped class to outcomes of students in a more traditional lecture class. To control for student population differences and to ensure that student populations between the classes were comparable, portions of the flipped class experimental group (*flipped group*) were taught utilizing approaches similar to those in the *traditional groups* [Units 1 & 3 (Control Units)], whereas other portions of the course were taught utilizing the flipped classroom approach [Unit 2 (Experimental Unit)]. Exam scores during the Control Units showed no statistical difference between *traditional groups* and the *flipped group* (Unit 1, $p=0.266$ & Unit 3, $p=0.698$); whereas the *flipped group* performed significantly better than the *traditional groups* during the Experimental Unit (Unit 2, $p < 0.005$). Additionally, student surveys indicated that students were more engaged in the class, came to class better prepared, dedicated more time working out problems, felt more confident with the material, better paced themselves, and overall preferred the

flipped class over the traditional class. This study suggests that flipping a content heavy course can improve student engagement and result in greater learning gains.

33.12
USING CONFLUENT CLASSROOM DESIGN TO TEACH UNDERGRADUATE ANATOMY AND PHYSIOLOGY

Andrew Petzold[1], and Robert Dunbar[1]
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In an effort to deliver an undergraduate curriculum that advances an outcome-driven, learner-centered, technology-enhanced pedagogy in the health sciences, we have developed a blended teaching model that provides best environment for all students to learn. To accomplish this, we have utilized multimodal classrooms that are based on recent pedagogical innovations including backward course design and active and semi-flipped classroom engagement to create a collaborative learning space that allows for a student-centered approach to learning. By combining these approaches with a variety of active-learning implementation strategies, we are able to facilitate an environment that is optimized for learning that can be utilized for educational research. This multimodal approach to learning, entitled the "confluent classroom" provides faculty with the opportunity to focus on specific institutional learning and developmental outcomes while able to modularize the curriculum at all levels of the curriculum. The confluent classroom strategy is implemented as of a flipped/student-driven punctuated lecture for introduction to a topic followed by reinforcement through practical application in a collaborative learning situation and finally exploration or further application in a hands-on laboratory experience. This prescribed approach to instruction allows for the "plug and play" of differing modules, or portions without much disruption of the entire curriculum of at the course level. The confluent classroom encourages classroom engagement by the student while encouraging group engagement while still maintaining individual accountability.

35:0 PLENARY VII

35.1
EDUCATIONAL LEADERSHIP: BENEFITS OF STEPPING OUTSIDE THE CLASSROOM

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Although most educators have their greatest impact in the classroom, the increasing need for diverse learning activities has created new opportunities for leadership. Moreover, many Tenure and Promotion Committees are finding that it is no longer sufficient to consider only lecture hours when evaluating a faculty member's contributions to the teaching mission of a school. Accordingly, the career path for an educator in a college or professional school is evolving. A newly-recruited faculty member may start out with classroom responsibilities, but activities other than lecture such as flipped classrooms, online resources, and peer-to-peer teaching are quickly added to the mix. As faculty members gain experience, they often progress to positions of curriculum design or program review within an institution. Similarly, there is a need for administrators who have participated in a variety of learning activities, and schools frequently recruit for these positions from faculty with such exposure. Many senior faculty members leverage this expertise to regional or national levels by authoring textbooks and online materials or serving on advisory boards, review committees, and governance in professional societies and funding agencies. Excelling in these leadership opportunities can have a profound effect on the success of promotion and tenure applications, and they reward a skill set that extends beyond the teaching and organization needed in the classroom.

36:0 PLENARY VIII

36.1
IMPLEMENTING AND MANAGING CHANGE

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This closing session will be a wrap-up discussion of the experiences of the week and a challenge for continuing as the Physiology Education Community of Practice. In addition to feedback on the 2016 Institute on Teaching and Learning, planning for future grant opportunities and the 2018 ITL will begin. An organizer and an organizing committee for the 2018 institute will be formed. Brainstorming will occur for topics to include and individuals to invite for the 2018 institute. Participants and facilitators for the 2016 ITL will be acknowledged and potential collaborations will be identified and encouraged. Activities to occur between 2016 and 2018 will be discussed.

Thank you to the generous support of this workshop from

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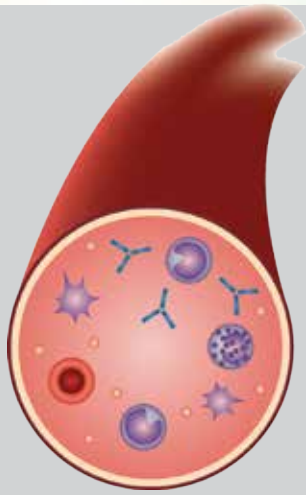

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2016 APS Conference: Inflammation, Immunity and Cardiovascular Disease
Week-at-a-Glance Schedule

Wednesday, August 24, 2016	Thursday, August 25, 2016	Friday, August 26, 2016	Saturday, August 27, 2016
3:00-8:00 PM: **Registration**	7:00 AM-7:30 PM: **Registration**	7:30 AM-7:30 PM: **Registration**	7:30 AM-4:30 PM: **Registration**
6:00-8:00 PM: **Welcome and Opening Reception**	8:00-10:00 AM: Symposia I: **Basic Aspects of Innate Immune Cells** Chairs: Cornelia Weyand and Tomasz Guzik	8:00-10:00 AM: Symposia V: **CV Disease in Inflammatory and Autoimmune Disease** Chair: Mike Ryan	8:00-10:00 AM: Symposia VIII: **Inflammation, Hypertension, and End-Organ Damage** Chairs: David Mattson and Jens Titze
8:00-8:15 PM: **Opening Comments** Speaker: David G. Harrison	10:00-10:30 AM: **Break**	10:00-10:30 AM: **Break**	10:00-10:30 AM: **Break**
8:15-9:15 PM: Plenary Lecture: **The Endothelium and Vascular Immunity** Speaker: Jordan Pober	10:30 AM-12:30 PM: Symposia I: **Basic Aspects of Innate Immune Cells (Continued)** Chairs: Cornelia Weyand, and Tomasz Guzik	10:30 AM-12:45 PM: Symposia VI: **Inflammation and Hypertension During Pregnancy and Gender Differences** (with Selected Oral Presentations) Chair: Kathryn Sandberg	10:30 AM-12:30 PM: Symposia VIII: **Inflammation, Hypertension, and End-Organ Damage (Continued)** Chairs: David Mattson and Jens Titze
	12:30-2:00 PM: **Poster Session I and Lunch**	12:45-2:30 PM: **Poster Session II and Lunch**	12:30-1:30 PM: **Lunch**
	2:00-4:00 PM: Symposia II: **Basic Aspects of T Cells** Chair: Jorg Goronzy	2:30-3:30 PM: Career Workshop: **Get a Job: Build the Skills that Employers Want!**	1:30-2:45 PM: **Selected Oral Presentations:** Chairs: David Mattson and Jens Titze
	4:00-7:00 PM: **Free Time**	3:30-7:00 PM: **Free Time**	2:30-4:30 PM: Symposia IX: **Inflammation and Atherosclerosis** Chair: Elena Galkina
	7:00-8:30 PM Symposia III: **Basic Aspects of B Cells** Chair: Amy Major	7:00-8:30 PM: Symposia VII: **Inflammation, Immunity, Intestinal Flora and the Metabolic Syndrome** Chair: Sean Davies	4:30-7:00 PM: **Free Time**
	8:45-10:30 PM Symposia IV: **Basic Aspects of Vascular Cells with Immune Function** Chair: Meena Madhur	8:45-10:00 PM: Symposia VII: **Inflammation, Immunity, Intestinal Flora and the Metabolic Syndrome (Continued and with Selected Oral Presentations)** Chair: Sean Davies	7:00-9:30 PM: **Closing Banquet and Awards Ceremony**

Location:

The 2016 APS Conference: Inflammation, Immunity, and Cardiovascular Disease will be held August 24—27, 2016 at the Westin Westminster Hotel located at: 10600 Westminster Blvd., Westminster, CO 80020, telephone (303) 410-5000.

Onsite Registration Hours:

Wednesday, August 24…….....3:00—8:00 PM
Thursday, August 25……..7:00 AM—4:00 PM
……6:30—7:30 PM
Friday, August 26……...7:30 AM—4:00 PM
……6:30—7:30 PM
Saturday, August 27……...7:30 AM—4:30 PM

On-Site Registration Fees:

APS Member	$850
APS Retired Member	$550
Nonmember	$1,050
APS Postdoctoral Member	$650
Postdoctoral Nonmember	$750
APS Student Member	$550
Student Nonmember	$650

Payment Information:

Registrants may pay by institutional or personal check, traveler's check, MasterCard, VISA or American Express or in United States Dollars. 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 onsite 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 four 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.**

Included in your Registration:

Your registration to this conference includes entry into all scientific sessions, program book, opening reception, poster sessions, networking socials, and the closing banquet meal. **There are no substitutions or refunds.** You must pay the registration fee regardless of whether you are not able to stay for the entire conference or partake in any of the meals during the conference program. Guests of attendees are not permitted in the scientific sessions.

Press Registration:

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.

Photograph/Video Recording:

The photographing and/or the video recording of any the conference sessions for personal or private use is strictly prohibited.

Code of Conduct:

APS is committed to providing a friendly, safe, and welcoming environment for all, regardless of gender, sexual orientation, disability, race, ethnicity, religion, national origin, or other protected characteristics. We expect all attendees, media, speakers, volunteers, organizers, venue staff, guests, and exhibitors to help us ensure a safe and positive workshop experience for everyone. Alert the APS Registration Desk if you notice a dangerous situation, someone in distress, or violations of this Code of Conduct.

Program Objective:

The purpose of this conference is to review fundamentals of innate immunity and how various cardiovascular diseases affect these functions. Particular areas of interest include monocyte/macrophages, complement and reactive oxygen species. As well as review 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 cell subtypes, such as CD4+, CD8+, T regulatory cells and TH17 cells will be highlighted. To review role of B cells and humoral immunity in cardiovascular diseases and to discuss gender differences in cardiovascular inflammation. We will discuss the propensity for cardiovascular disease among individuals with autoimmune disease as well as encourage presentation of preliminary data from young investigators regarding inflammation and cardiovascular disease.

Target Audience:

This conference is intended for all professionals involved in teaching, research and clinical fields related to inflammation, immunity and cardio-vascular diseases.

Did you register early?

The drawing for the iPad Pro will be held on Saturday, August 27 during the Closing Banquet and Awards Ceremony

Winner must be in attendance to receive prize

WEDNESDAY, AUGUST 24, 2016

Welcome

1.0 WELCOME AND OPENING COMENTS

Wednes., 8:00—8:15 PM, Westminster III.

8:00 PM **1.1** Welcome and Opening Comments. **David Harrison,** *Vanderbilt Univ.*, and **David Mattson,** *Med. Coll. of Wisconsin, Milwaukee.*

Plenary Lecture

2.0 PLENARY LECTURE

Wednes., 8:15—9:15 PM, Westminster III.

8:15 PM **2.1** The Endothelium and Vascular Immunity. **Jordan Pober.** *Yale Univ.*

THURSDAY, AUGUST 25, 2016

Symposia I

3.0 BASIC ASPECTS OF INNATE IMMUNE CELLS

Thurs., 8:00 AM—12:30 PM, Westminster III.

Chairs: **Cornelia Weyand**, *Stanford Univ.*
Tomasz Guzik, *Univ. of Glasgow, UK.*

8:00 AM **3.1** Tissue-resident Intimal Macrophages Contribute to Atherosclerotic Lesion Initiation and Plaque Progression. **Jesse Williams.** *Washington Univ., St. Louis.*

8:30 AM **3.2** IL-13 Signaling in Tissue Repair and Fibrosis. **Thomas A. Wynn.** *NIH, NIAID.*

9:00 AM **3.3** The Regulation of Macrophage Activation by Endogenous Secretory Products. **David M. Mosser.** *Univ. of Maryland, College Park.*

9:30 AM **3.4** Immunogenetics of Natural Killer Cells. **Peter Parham.** *Stanford Univ.*

10:00 AM Break

10:30 AM **3.5** DAMP'S & PAMP'S. **Karen Newell-Rogers.** *Texas A&M Univ.*

11:00 AM **3.6** Interleukins and Innate Immunity. **Charles Dinarello.** *Univ. of Colorado, Denver.*

11:30 AM **3.7** MHC and Antigen Presentation. **Elizabeth Mellins.** *Stanford Univ.*

12:00 Noon **3.8** Neuro-immune Reflex Arc. **Kevin J. Tracey.** *Hofstra Univ., North Shore-LIJ.*

Poster Session I

4.0 POSTER SESSION I

Thurs., 12:30—2:00 PM, Westminster IV.

Poster Board

1 **4.1** KLF6-BCL6 Signaling Axis Regulates Macrophage-mediated Inflammation. **G. Mahabaleshwar.** *Case Western Res. Univ.*

2 **4.2** Aortic and Microvascular Endothelial Cells Differentially Influence Inflammatory and Vascular Responses to Engineered Nanomaterial. **V. Minarchick, J. Shannahan, E. Sabolsky, and J. Brown.** *Univ. of Colorado Denver, and West Virginia Univ.*

3 **4.3** Lymphatics—The Key Component of the Immunovascular System. **D. Zawieja, W. Wang, W. Cromer, A. Narayanan, A. Gashev, and M. Muthuchamy.** *Texas A&M Univ.* **(7.5)**

4 **4.4** Plasma Levels of Angiopoietin-1, Angiopoietin-2 and Vascular Endothelial Growth Factor in Sickle Cell Anaemia Patients With or Without Complications in Ghana: A Case-control Study. **C. Antwi-Boasiako, E. Frimpong, B. Gyan, E. Kyei-Baafour, F. Sey, D. A. Antwi, B. Dzudzor, M. Abdul-Rahman, W. Agbozo, K. Antwi-Boasiako, B. G. Dankwah, T. A. Ndanu, A. D. Campbell, and I. Ekem.** *Univ. of Ghana, Accra, Korle-Bu Teaching Hosp., Accra, Ghana, and Univ. of Michigan.*

5 **4.5** Mechanisms of Increased Complement Activation in Placental Ischemia-induced Hypertension in the Rat. **J. Regal, C. Wing, N. F. Nieto, J. Gilbert, and S. Fleming.** *Univ. of Minnesota Med. School, and Kansas State Univ.* **(9.4)**

6 **4.6** Platelet-P2Y12 Receptor is Important for the Cellular Immune Response in Erosive Arthritis. **C. A. Barrero, A. E. Garcia, W. Shao, M. Amin, M. F. Barbe, O. M. Perez-Leal, and M. C. Rico.** *Temple Univ.* **(7.3)**

7 **4.7** Interleukin-19: A Novel Sexually Dimorphic Cardiac Cytokine. **D. Bruns, S. Thoemmes, A. Ghincea, C. Ghincea, and L. Walker.** *Univ. of Colorado, Denver.* **(9.5)**

8 **4.8** Prior Preeclampsia Results in Persistent Immune Activation Following Pregnancy in the Dahl Salt Sensitive Rat. **E. B. Taylor, M. R. Garrett, M. J. Ryan, and J. M. Sasser.** *Univ. of Mississippi Med. Ctr.*

Poster Board

9 **4.9** Increased Pro-inflammatory T cells and HMGB1 are Associated with Vascular Dysfunction in Male SHR. **E. Gillis, J. Musall, and J. Sullivan.** *Augusta Univ.* **(9.6)**

10 **4.10** Mitochondrial ROS Mediates PKM2 Kinase Activity to Promote Proinflammatory Activity of Human Macrophages in Coronary Artery Disease. **R. Nazarewicz, T. Shirai, J. Young, B. B. Wallis, R. Yanes, D. G. Harrison, J. C. Giacomini, T. L. Assimes, J. J. Goronzy, and C. M. Weyand.** *Stanford Univ., and Vanderbilt Univ.*

11 **4.11** Identification of an Additional Role for apoC-III in Atherogenesis: Overexpression of apoC-III Modulates Basal T Cell Profile in Mice. **C. Rodia, and A. Kohan.** *Univ. of Connecticut, Storrs.*

12 **4.12** Role of Smooth Muscle Cell Specific iPLA2β in Vascular Inflammation and Neointimal Formation in a Murine Femoral Artery Wire Injury Model. **S. Liu, G. Zhao, M. Gong, and Z. Guo.** *Univ. of Kentucky, Lexington, and Lexington VA Med. Ctr.* **(7.4)**

13 **4.13** Role of Neuregulin-1β in the Acute Inflammatory Response to Coronary Artery Bypass Grafting (CABG). **S. Peterson, S. Ryzhov, M. Robich, D. Roberts, R. Quinn, J. Burgess, R. Rath, S. Bosworth-Farrell, R. Kramer, and D. B. Sawyer.** *Maine Med. Ctr. Res. Inst., Scarborough, and Maine Med. Ctr., Portland.*

14 **4.14** The Role of PSGL-1-Ecpressing CD4 T Cells in the Atherosclerotic Development and Plaque Instability. **K. Sato, K. Kitamura, K. Fukushima, and N. Hagiwara.** *Tokyo Women's Med. Univ., Japan.*

15 **4.15** Influence of HIV-1 on Circulating Vascular-Related microRNAs. **G. Lincenberg, J. Hijmans, P. Kavlich, J. Greiner, B. Stauffer, E. Connick, and C. DeSouza.** *Univ. of Colorado, Boulder, Univ. of Arizona, Tucson, and Univ. of Colorado Denver.*

16 **4.16** HIV-1, Inflammation and Endothelial Dysfunction. **R. Fay, B. Weil, J. Greiner, B. Stauffer, E. Connick, and C. DeSouza.** *Univ. of Colorado, Boulder, Univ. of Arizona, Tucson, and Univ. of Colorado, Denver.* **(15.4)**

Poster Board

17 **4.17** The alpha7 Nicotinic Acetylcholine Receptor Agonist AZ6983 Reduces Atherosclerosis in ApoE-/- Mice and TNF Production in Human Cells. **L. J. Yang, M. A. Ulleryd, D. Panagaki, S. Hua, E. Michaëlsson, and M. E. Johansson.** *Univ. of Gothenburg, Sweden, and AstraZeneca R&D, Mölndal, Sweden.*

18 **4.18** The Anti-inflammatory Role of α7nAChR and α4β2nAChR in Human Blood Samples. **D. Panagaki, M. A. Ulleryd, and M. E. Johansson.** *Univ. of Gothenburg, Sweden.*

19 **4.19** The Association Between Autonomic Dysfunction, Inflammation and Prevalent Cardiovascular Disease in Subjects with Atherosclerosis. **M. A. Ulleryd, U. Prahl, J. Börsbo, C. Schmidt, S. Nilsson, G. Bergström, and M. E. Johansson.** *Univ. of Gothenburg, Sweden, and Chalmers Univ. of Tech., Gothenburg, Sweden.*

20 **4.20** Activation of Nonclassical Monocytes During Atherogenesis is Mediated by the Scavenger Receptor CD36. **P. Marcovecchio, E. Ehinger, K. Mueller, G. Thomas, Z. Mikulski, K. Ley, and C. Hedrick.** *La Jolla Inst., Univ. of California, San Diego, and Medizinische Klinik, Tübingen, Germany.*

21 **4.21** Targeted UPLC-MS/MS Analysis of Oxylipins: From Profiling to Quantification for Translational Research Studies. **B. Molloy, and A. Peck.** *Waters Corp., Wilmslow, UK, and Waters Corp., Milford, MA.*

22 **4.22** Ly6CHI Monocytic Clearance of Dying Cardiomyocytes by CD36 Activates NR4a1 and is Required for Transition to Reparative Inflammation Following Myocardial Infarction. **S. Dehn, and E. Thorp.** *Northwestern Univ.* **(15.5)**

23 **4.23** Interaction of IL-6 and TNF-α Contributes to Endothelial Dysfunction in Type 2 Diabetic Mice Heart. **J. Lee, S. Lee, C. Zhang, and Y. Park.** *Univ. of Houston, and Univ. of Missouri, Columbia.*

24 **4.24** Infiltrating Macrophages Promote Adverse Cardiac Remodeling During Acute and Chronic Pressure-Overload. **B. Patel, S. Bansal, A. Ismahil, T. Hamid, and S. D. Prabhu.** *Univ. of Alabama at Birmingham.*

Poster Board

25 **4.25** The Alarmin Complex S100A8/9 as a Bioimaging and Therapeutic Target After Myocardial Infarction. **G. Marinković, A. Schiopu, and J. Nilsson.** *Lund Univ., Malmö, Sweden.*

26 **4.26** Oxidative and Nitrosative Stress in Valve Tissue of Patients with Aortic Valve Disease. **S. Ben-Aicha, K. Casós, and M. Galiñanes.** *Univ. Autònoma de Barcelona, Spain.*

27 **4.27** Inhibition of Inflammatory Serine Proteases Potentiates B cell Development and Function in Diabetic Cardiomyopathy. **A. Sarkar, W. Liu, A. Abbott, K. Sikder, S. K. Shukla, A. Alqatawni, C. Barbery, C. Pham, and K. Rafiq.** *Thomas Jefferson Univ., and Washington Univ. Sch. of Med.*

28 **4.28** Inflammatory Serine Protease Inhibition Attenuates Myocyte Apoptosis and Cardiac Dysfunction via Intervention of PPAR gamma-Induced Lipotoxicity and Inflammation in High Fat Diet-Induced Diabetic Cardiomyopathy. **K. Sikder, A. Sarkar, S. Shukla, A. Abbott, D. Carrier, C. Barbery, L. Tian, R. Pestell, C. Pham, and K. Rafiq.** *Thomas Jefferson Univ., and Washington Univ., Sch. of Med.*

29 **4.29** Porphyromonas Gingivalis Lipopolysaccharide Enhances the Rupture of Experimental Cerebral Aneurysms by Inducing Vascular Inflammation. **T. Miyamoto, K. T. Kitazato, H. Maekawa, K. Yagi, K. Shimada, Y. Tada, M. Korai, T. Yamaguchi, T. Kinouchi, Y. Kanematsu, J. Satomi, and S. Nagahiro.** *Tokushima Univ. Grad. Sch., Japan.*

30 **4.30** Loss of Lymphocyte Adaptor Protein LNK Promotes Aortic Dissection. **F. Laroumanie, M. A. Saleh, W. McMaster, A. Kirabo, and M. S. Madhur.** *Vanderbilt Univ. Med. Ctr.*

31 **4.31** Effect of Inflammatory Serine Protease Inhibition on CXCR4 Expression During the Development of Diabetic Cardiomyopathy. **S. K. Shukla, K. Sikder, W. Liu, A. Sarkar, A. Abbott, C. Barbery, C. Pham, and K. Rafiq.** *Thomas Jefferson Univ., and Washington Univ. Sch. of Med.*

32 **4.32** Neutrophil Functional Changes are Associated with Metabolic Alterations Following Ten Weeks of High Interval Exercise Training in Men and Women with Prediabetes. **D. B. Bartlett, C. A. Slentz, L. H. Willis, K. M. Huffman, J. M. Lord, and W. E. Kraus.** *Duke Univ. Sch. of Med., and Univ. of Birmingham Sch. of Med., UK.*

Poster Board

33 **4.33** High Salt Activates Dendritic Cells to Promote Hypertension. **J. D. Foss, N. R. Barbaro, L. Xiao, S. Dikalov, D. G. Harrison, and A. Kirabo.** *Vanderbilt Univ. Med. Ctr.*

Symposia II

5.0 BASIC ASPECTS OF T CELLS

Thurs., 2:00—4:00 PM, Westminster III.

Chair: **Jorg J. Goronzy,** *Stanford Univ.*

2:00 PM **5.1** T Cell Subsets and Immune Responses. **John O' Shea.** *NIH, NIAMS.*

2:30 PM **5.2** T Cell Trafficking and Memory. **Jorg J. Goronzy.** *Stanford Univ.*

3:00 PM **5.3** T Cell Trafficking and Memory. **Eric Clambey.** *Univ. of Colorado, Denver.*

3:30 PM **5.4** T Regulatory Cell (Treg) Polarization and Function. **Karen Newell-Rogers.** *Texas A&M Univ.*

Symposia III

6.0 BASIC ASPECTS OF B CELLS

Thurs., 7:00—8:30 PM, Westminster III.

Chair: **Amy Major,** *Vanderbilt Univ. Med. Ctr.*

7:00 PM **6.1** B Cell Subsets and Immune Responses. **Amy Major.** *Vanderbilt Univ. Med. Ctr.*

7:30 PM **6.2** Genetic Risk Factors and Loss of B Cell Anergy in Development of Autoimmunity. **John Cambier.** *Univ. of Colorado, Denver.*

8:00 PM **6.3** B Cells and Cardiovascular Disease. **Myra Lipes.** *Harvard Univ. Med. Sch.*

Symposia IV

7.0 BASIC ASPECTS OF VASCULAR CELLS WITH IMMUNE FUNCTION

Thurs., 8:45—10:30 PM, Westminster III.

Chair: **Meena Madhur,** *Vanderbilt Univ. Med. Ctr.*

8:45 PM **7.1** CD4+ T cells in atherosclerosis. **Dennis Wolf.** *La Jolla Inst. for Allegy and Immunology.*

9:15 PM **7.2** IL1β Promotes Athero-Protective Changes in Late Stage Atherosclerotic Lesions. **Gary Owens.** *Univ. of Virginia Sch. of Med.*

9:45 PM **7.3** Platelet-P2Y12 Receptor is Important for the Cellular Immune Response in Erosive Arthritis. **Mario C. Rico.** *Temple Univ.* **(4.6)**

10:00 PM **7.4** Role of Smooth Muscle Cell Specific iPLA2β in Vascular Inflammation and Neointimal Formation in a Murine Femoral Artery Wire Injury Model. **Shu Liu.** *Univ. of Kentucky.* **(4.12)**

10:15 PM **7.5** Lymphatics—The Key Component of the Immunovascular System. **David Zawieja.** *Texas A&M Univ.* **(4.3)**

FRIDAY, AUGUST 26, 2016

Symposia V

8.0 CV DISEASE IN INFLAMMATORY AND AUTOIMMUNE DISEASE

Fri., 8:00—10:00 AM, Westminster III.

Chair: **Mike Ryan,** *Univ. of Mississippi Med. Ctr.*

8:00 AM **8.1** Utilizing A Human Model of Disease to Study Inflammatory Atherogenesis. **Nehal Mehta.** *NIH, NHLBI.*

8:25 AM **8.2** Periodontal Inflammation and CV Disease. **Tomasz Guzik.** *Univ. of Glasgow, UK.*

8:45 AM **8.3** Autoimmunity: A Causal Factor in the Pathogenesis of Hypertension. **Mike Ryan.** *Univ. of Mississippi Med. Ctr.*

9:05 AM **8.4** Defective Inflammation Resolution in Atherosclerosis: Mechanisms and Therapeutic Opportunities. **Ira Tabas.** *Columbia Univ. Med. Ctr.*

9:30 AM **8.5** Vascular Inflammation/Arteritis. **Cornelia Weyand.** *Stanford Univ.*

10:00 AM Break.

Join your colleagues for the Opening Reception

Wednesday, August 24, 2016
6:00—8:00 PM

South Courtyard

Symposia VI

9.0 INFLAMMATION AND HYPERTENSION DURING PREGNANCY AND GENDER DIFFERENCES

Fri., 10:30 AM—12:45 PM, Westminster III.

Chair: **Kathryn Sandberg,** *Georgetown Univ.*

10:30 AM **9.1** Adaptive Immunity and Gestational Hypertension During Pregnancy. **Babette LaMarca.** *Univ. of Mississippi Med. Ctr.*

11:00 AM **9.2** Innate Immunity and Gestational Hypertension. **Brett Mitchell.** *Texas A&M Univ.*

11:30 AM **9.3** Gender Differences in Inflammation and Hypertension. **Kathryn Sandberg.** *Georgetown Univ.*

12:00 Noon **9.4** Mechanisms of Increased Complement Activation in Placental Ischemia-induced Hypertension in the Rat. **Jean Regal.** *Univ. of Minnesota Med. Sch., Duluth.* **(4.5)**

12:15 PM **9.5** Interleukin-19: A Novel Sexually Dimorphic Cardiac Cytokine. **Danielle Bruns.** *Univ. of Colorado, Denver.* **(4.7)**

12:30 AM **9.6** Increased Pro-inflammatory T Cells and HMGB1 are Associated with Vascular Dysfunction in Male SHR. **Ellen Gillis.** *Augusta Univ.* **(4.9)**

Poster Session II

10.0 POSTER SESSION II

Fri.,12:45—2:45 PM, Westminster IV.

Poster Board

1 **10.1** The Impact of Exercise Intensity and Cytomegalovirus Infection on Immune Cell Redeployment by Exercise. **E. LaVoy, M. Hussain, J. Reed, H. Kunz, M. Pistillo, and R. Simpson.** *Univ. of Houston.*

2 **10.2** Obesity and Circulating Inflammation-Related microRNAs. **J. Hijmans, T. Bammert, P. Kavlich, G. Lincenberg, K. Diehl, J. Greiner, B. Stauffer, and C. DeSouza.** *Univ. of Colorado, Boulder, and Univ. of Colorado, Denver.* **(12.5)**

3 **10.3** Characterization of Factors that Predispose to the Metabolic Syndrome and Adipose Tissue Inflammation in Aged Male and Female Mice. **H. Ahnstedt, A. Chauhan, M. Roy-O'Reilly, and L. D. McCullough.** *Univ. of Texas Hlth. Sci. Ctr., Houston.* **(12.6)**

Poster Board

4 **10.4** Effects of an 8-Week Paleo Dietary Intervention on Inflammatory Cytokines. **C. Dolan, A. Carrillo, N. Davies, and M. Markofski.** *Univ. of Houston, and Chatham Univ., Pittsburgh.*

5 **10.5** Perivascular Adipose Tissue Macrophages are Responsible for Endothelial Dysfunction in the Obese Microvasculature. **J. Candela, and C. White.** *Rosalind Franklin Univ., Chicago.* **(12.7)**

6 **10.6** Cullin3 Regulates Endothelial Function by Modulating eNOS Activity. **J. Wu, K. Lu, L. Agbor, X. Liu, M. Mukohda, A. Nair, and C. Sigmund.** *Univ. of Iowa, Iowa City.*

7 **10.7** Renal and Splenic Cytokines are Altered in Early Life Stressed (ELS) Adult Male Rats. **I. Obi, C. De Miguel, D. Ho, A. Loria, and J. Pollock.** *Univ. of Alabama at Birmingham, Tripler Army Med. Ctr., Honolulu, HI, and Univ. of Kentucky.*

8 **10.8** γ/δ T Cells Mediate Angiotensin II-Induced Hypertension and Vascular Injury. **A. Caillon, M. O. R. Mian, J. C. Fraulob-Aquino, K. Huo, T. Barhoumi, P. Paradis, and E. L. Schiffrin.** *Lady Davis Inst. for Med. Res., Montreal, Canada.* **(14.1)**

9 **10.9** Macrophages Regulate the Expression of Stromal Cell-derived Factor 1 via Indoleamine 2,3-dioxygenase After the Renal Acute Ischemia-reperfusion Injury. **X. Wan, F. Zhang, and C. Cao.** *Nanjing Med. Univ., People's Rep. of China.*

10 **10.10** Model Establishing and Factor Analysis: An Insight into the Prediction of Acute Kidney Injury After Heart Surgery in China. **X. Wan, J. Li, and C. Cao.** *Nanjing Med. Univ., People's Rep. of China.*

11 **10.11** Extracellular Histones in Relation to Organ Dysfunction and Inflammation during Kidney-Lung Crosstalk. **X. Wan, Y. Gendoo, and C. Cao.** *Nanjing Med. Univ., People's Rep. of China.*

12 **10.12** Risk Factors of In-hospital Mortality in AKI Associated with Cardiopulmonary Bypass: A Retrospective Cohort Study. **X. Wan, X. Ji, and C. Cao.** *Nanjing Med. Univ., People's Rep. of China.*

13 **10.13** Infiltrating Macrophages Promote Vascular Sympathetic Hyperinnervation During Experimental Hypertension. **A. Wang, C. Chan, Y. Ling, Z. Wei, G. Drummond, R. Widdop, R. Haddock, and A. Vinh.** *Monash Univ., Clayton, Australia.*

Poster Board

14 **10.14** Impaired Right Coronary Vasodilative Function in Pulmonary Hypertensive Rat Assessed by Synchrotron Microangiography. **T. Inagaki, H. Tsuchimochi, J. Pearson, D. Schwenke, K. Umetani, Y. Nakaoka, and M. Shirai.** *Natl. Cerebral and Cardiovascular Ctr., Osaka, Japan, Univ. of Otago, Dunedin, New Zealand, and Japan Synchrotron Radiation Res. Inst., Hyogo, Japan.*

15 **10.15** CARD9 Knockout Ameliorates Fibrosis and Hypertrophy in a TAC Pressure-overload Model. **M. Peterson, S. Haller, K. Wilson, D. P. Thomas, and G. He.** *Univ. of Wyoming, Laramie.* **(14.2)**

16 **10.16** Cytotoxic $CD8^+$ T-cells Play a Role in Hypertension-Associated Inflammatory Responses in Female Dahl Rats. **A. V. Pai, A. Souza, C. A. West, and K. Sandberg.** *Georgetown Univ.*

17 **10.17** Arterial Stiffness Due to Carotid Calcification Disrupts Neurovascular Coupling and Leads to Cognitive Deficits. **M. F. Iulita, G. Muhire, D. Vallerand, M. Gratuze, F. R. Petry, G. Ferland, E. Planel, and H. Girouard.** *Univ. de Montréal, Canada, and Univ. de Laval, Quebec, Canada.*

18 **10.18** Dynamic Vascular T Cell-antigen Presenting Cell Interactions During Hypertension. **A. Vinh, Z. Wei, A. Wang, I. Spizzo, M. Hickey, and R. Widdop.** *Monash Univ., Clayton, Australia.* **(14.3)**

19 **10.19** Loss of ET_B Receptor Function Activates NOD-like Receptor and Inflammasome Signaling Pathways in Renal Outer Medulla During Type 1 Diabetes Through an ER Stress-independent Mechanism. **C. De Miguel, W. C. Hamrick, D. M. Pollock, and J. S. Pollock.** *Univ. of Alabama at Birmingham.*

20 **10.20** A High Fat Diet Increases Blood Pressure and Leads to a Proinflammatory Immune Cell and Cytokine Profile in the Aortae of Female Dahl Salt-Sensitive Rats (DSS). **L. Taylor, B. Baban, and J. Sullivan.** *Augusta Univ.*

Poster Board

21 **10.21** Induction of AP-1, NF-κB, miR-21 Expression, and Angiogenesis by Tungsten Carbide-Cobalt Nanoparticles Involves ROS-Mediated MAPK Pathways and Results in Transformation of JB6 and BEAS-2B Cells. **T. Barber, J. Aldinger, L. Bowman, T. Meighan, and M. Ding.** *CDC, NIOSH, Morgantown, WV.*

22 **10.**22 Sympathetic Nerves Promote Hypertension-specific Effector Memory T Cell Homing and Proliferation in the Bone Marrow of Mice with Angiotensin II Infusion. **L. Xiao, H. Itani, J. Foss, and D. G. Harrison.** *Vanderbilt Univ. Med. Ctr.*

23 **10.23** Induction of Ap-1 Signaling and DNA Damage by Copper Oxide Nanoparticles Involve ROS-mediated MAPK Pathways. **T. Barber, L. Bowman, J. Aldinger, and M. Ding.** *CDC, NIOSH, Morgantown, WV.*

24 **10.24** A New Role of Sox6 in Blood Pressure Control Through Renin Regulation. **J. A. Gomez, C. P. Hodgkinson, A. Payne, D. G. Harrison, and V. J. Dzau.** *Vanderbilt Univ. Med. Ctr., and Duke Univ.*

25 **10.25** Pulmonary Hypertension-in-duced Right Ventricular Pressure Overload Triggers Acute Epigenetic Reprogramming of Pro-inflammatory Cardiac Fibroblasts. **D. Bruns, S. Thoemmes, K. Stenmark, P. Buttrick, and L. Walker.** *Univ. of Colorado, Denver.*

26 **10.26** miR-762 Inhibition Prevents and Reverses Angiotensin II Induced Aortic Fibrosis and Stiffening. **K. R. C. Montaniel, J. Wu, M. R. Bersi, L. Xiao, H. A. Itani, K. C. Vickers, J. D. Humphrey, and D. G. Harrison.** *Vanderbilt Univ. Med. Ctr., and Yale Univ.*

27 **10.27** The R213G Polymorphism in EC-SOD Protects Early Bleomycin-induced Pulmonary Inflammation and Attenuates Induction of Genes Involved in Leukocyte Extravasation. **K. Swain, G. Mouradian, R. Gaurav, S. Pugliese, A. Hernandez-Lagunas, K. El-Kasmi, A. Karimpour-Fard, R. Bowler, C. Sucharov, and E. Nozik-Grayck.** *Univ. of Colorado, Denver, and Natl. Jewish Hlth., Denver, CO.*

28 **10.28** Interleukin 21 Promotes Hypertension and End-Organ Dysfunction. **B. L. Dale, F. Laroumanie, M. A. Saleh, H. M. Scott Algood, and M. S. Madhur.** *Vanderbilt Univ. Med. Ctr.*

Poster Board

29 **10.29** Effect of Beta Blockers on Post-exercise Cytokine Response. **M. Markofski, C. Dolan, H. Kunz, R. Graff, N. Agha, R. Azadan, F. Baker, P. Mylabathula, and R. Simpson.** *Univ. of Houston.*

30 **10.30** Investigating the Mechanisms by Which IgG Antibodies Contribute to Angiotensin II-dependant Hypertension. **M. Lieu, C. T. Chan, C. G. Sobey, A. Vinh, and G. R. Drummond.** *Monash Univ., Clayton, Australia.* **(14.4)**

31 **10.31** Macrophage-derived IGF-1 Contributes to Aortic Fibrosis and Stiffening in Hypertensive Mice. **G. R. Drummond, C. T. Chan, A. Ilinykh, A. Vinh, S. M. Krishnan, M. Lieu, C. V. Lewis, H. Diep, A. Pinto, and C. G. Sobey.** *Monash Univ., Clayton, Australia.*

32 **10.32** Opposing Effects of C3a and C5a on Kidney Injury in Angiotensin II Induced Hypertension. **U. Wenzel, S. Weiss, A. Rosendahl, C. Meyer-Schwesinger, J. Koehl, and H. Ehmke.** *Univ. of Hamburg, Germany, and Univ. of Lübeck Germany.*

33 **10.33** Intrarenal Angiotensinogen Formation in Response to Chronic High Salt Intake and Angiotensin Administration is Augmented in TNF-α Receptor Type 1 Knockout Mice. **E. Mahaffey, A. Castillo, L. G. Navar, and D. Majid.** *Tulane Univ. Sch. of Med.*

34 **10.34** NLRP3 Inflammasome-mediated Immunity in Dahl Salt-Sensitive Hypertension. **J. Abais-Battad, H. Lund, and D. Mattson.** *Med. Coll. of Wisconsin, Milwaukee.*

Don't forget to view the posters during the daily coffee breaks and lunch

Westminster IV

Career Workshop

11.0 CAREER WORKSHOP

Fri., 2:30—3:30 PM, Westminster III.

2:30 PM **11.1** Get a Job: Build the Skills Employers Want! **Jennifer Sasser.** *Univ. of Mississippi Med. Ctr.*

Symposia VII

12.0 INFLAMMATION AND HYPERTENSION DURING PREGNANCY AND GENDER DIFFERENCES

Fri., 7.00—8:30 PM, Westminster III.

Chair: **Sean Davies,** *Vanderbilt Univ. Med. Sch.*

7:00 PM **12.1** TMAO as a Mediator and Therapeutic Target in Cardiovascular Disease. **Stan Hazen.** *Cleveland Clinic.*

7:30 PM **12.2** Systemic Olfactory Receptors and the Gut Microbiota. **Jennifer Pluznick.** *Johns Hopkins Univ. Sch. of Med.*

8:00 PM **12.3** Metabolic Syndrome. **Kamal Rahmouni.** *Univ. of Iowa, Iowa City.*

8:30 PM **12.4** Altering the Microbiota for Weight Control. **Sean Davies.** *Vanderbilt Univ. Med. Ctr.*

9:00 PM Break.

9:15 PM **12.5** Obesity and Circulating Inflammation-related microRNAs. **Jamie Hijmans.** *Univ. of Colorado, Boulder.* **(10.2)**

9:30 PM **12.6** Characterization of Factors that Predispose to the Metabolic Syndrome and Adipose Tissue Inflammation in Aged Male and Female Mice. **Hilda Ahnstedt.** *Univ. of Texas Hlth. Sci. Ctr.* **(10.3)**

9:45 PM **12.7** Perivascular Adipose Tissue Macrophages are Responsible for Endothelial Dysfunction in the Obese Microvasculature. **Carl White.** *Rosalind Franklin Univ.* **(10.5)**

iPad Pro Drawing will be held at the Closing Banquet and Award Ceremony on Saturday, August 27. You must be in attendance to win!

SATURDAY, AUGUST 27, 2016

Symposia VIII

13.0 INFLAMMATION, HYPERTENSION, AND END-ORGAN DAMAGE

Sat., 8:00 AM—12:30 PM, Westminster III.

Chairs: **David Mattson,** *Med. Coll. of Wisconsin, Milwaukee.*
Jens Titze, *Vanderbilt Univ. Med. Ctr.*

8:00 AM **13.1** Inflammation, Adaptive Immunity and Hypertension. **David G. Harrison.** *Vanderbilt Univ. Med. Ctr.*

8:30 AM **13.2** T Regulatory Lymphocytes in Hypertension. **Ernesto Schiffrin.** *McGill Univ., Montreal, Canada.*

9:00 AM **13.3** Renal Immune Cells and Hypertension. **David Mattson.** *Med. Coll. of Wisconsin, Milwaukee.*

9:30 AM **13.4** Inflammation and Renal Fibrosis. **Steve Crowley.** *Duke Univ. Med. Ctr.*

10:00 AM Break.

10:30 AM **13.5** Salt and Cardiovascular Risk. **Larry Appel.** *Johns Hopkins Bloomberg Sch. of Public Hlth.*

11:00 AM **13.6** High Salt, Gut Microbiome and Blood Pressure. **Dominik Mueller.** *Max Delbruck Ctr. for Molecular Med., Berlin, Germany.*

11:30 AM **13.7** T Cell Serum and Glucocorticoid-regulated Kinase 1 (SGK1) and Hypertension. **Meena Madhur.** *Vanderbilt Univ. Med. Ctr.*

12:00 Noon **13.8** Salt, Skin, and Macrophages. **Jens Titze.** *Vanderbilt Univ. Med. Ctr.*

Selected Oral Presentations

14.0 SELECTED ORAL PRESENTATIONS

Sat., 1:30-2:30 PM, Westminster III.

Chairs: **David Mattson,** *Med. Coll. of Wisconsin, Milwaukee.*
Jens Titze, *Vanderbilt Univ. Med. Ctr.*

1:30 PM **14.1** γ/δ T Cells Mediate Angiotensin II-induced Hypertension and Vascular Injury. **Antoine Caillon.** *Lady Davis Inst. for Med. Res., Montreal, Canada.* **(10.8)**

1:45 PM **14.2** CARD9 Knockout Ameliorates Fibrosis and Hypertrophy in a TAC Pressure-

overload Model. **Matt Peterson.** *Univ. of Wyoming.* **(10.15)**

2:00 PM **14.3** Dynamic Vascular T Cell-antigen Presenting Cell Interactions During Hypertension. **Antony Vinh.** *Monash Univ., Australia.* **(10.18)**

2:15 PM **14.4** Investigating the Mechanisms by Which IgG Antibodies Contribute to Angiotensin II-dependant Hypertension. **Maggie Lieu.** *Monash Univ., Australia.* **(10.30)**

Symposia IX

15.0 INFLAMMATION AND ATHEROSCLEROSIS

Sat., 2:30—4:30 PM, Wesminster III.

Chair: **Elena Galkina,** *Eastern Virginia Med. Sch., Norfolk.*

2:30 PM **15.1** T Cell Plasticity in Atherosclerosis. **Elena Galkina.** *Eastern Virginia Med. Sch., Norfolk.*

3:00 PM **15.2** B Cell Subsets in Atherosclerosis. **Coleen McNamara.** *Univ. of Virginia, Charlottesville.*

3:30 PM **15.3** Cytomegalovirus, Inflammation, and the Microvasculate. **Karen Stokes.** *Louisiana State Univ. Hlth. Sci. Ctr.*

4:00 PM **15.4** HIV-1, Inflammation, and Endothelial Dysfunction. **Jared Greiner.** *Univ. of Colorado, Boulder.* **(4.16)**

4:15 PM **15.5** Ly6CHI Monocytic Clearance of Dying Cardiomyocytes by CD36 Activities NR4a1 and is Required for Transition to Reparative Inflammation Following Myocardial Infarction. **Shirley Dehn.** *Northwestern Univ.* **(4.22)**

NOTES

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Abstracts of Invited and Contributed Presentations

2.0: PLENARY LECTURE

2.1
THE ENDOTHELIUM AND VASCULAR IMMUNITY

Jordan S. Pober[1]
[1]Immunobiology, Yale Sch. of Med., 10 Amistad St., Rm. 401D, New Haven, CT, 06519.

Endothelial cells (EC) line the entire vascular system but display characteristics that vary among tissues and among vascular segments within tissues. Post-capillary venular EC typically regulate the recruitment and activation of circulating immune cells at sites of inflammation. EC are typically activated to recruit leukocytes by cytokines of innate immunity (TNF or IL-1.) that increase expression of proteins that capture and activate the migration of myeloid and lymphoid cells. EC additionally respond to cytokines of adaptive immunity (IFN-g and IL-4) by expressing proteins that selectively recruit specialized immune effector cells. Surprisingly, EC are unresponsive to IL-17, which instead acts on pericytes. Human microvascular EC basally express both class I and class II MHC molecules (signal 1) and a key costimulator for memory T cells (CD58, signal 2.) and inducibly express additional costimulators as well as cytokines (signal 3) that enable effective antigen presentation to circulating effector memory T cells. Antigen presentation induces effector memory T cells to undergo diapedesis by a process that differs from chemokine-based recruitment in that T cells round up instead of spreading and push their MTOC in front of their nuclei instead of trailing be-hind their nuclei in a uropod. CD4+ T cells recruited by antigen use the lateral border recycling compartment of EC to transmigrate and partly degranulate during this process, releasing granzyme A, whereas CD8+ T cells do neither. Antigen-recruited T cells shut off expression of i-NOS, proliferate and produce effector cytokines whereas chemokine-recruited T cells express i-NOS but not cytokines. IFN-g further increases EC expression of immune-genic signals (MHC molecules and cytokines) but also induces inhibitory signals (PD-L1 and PD-L2). In some settings, e.g. transplantation, graft MHC molecules not only activate host T cells but also trigger B cells to produce anti-donor antibodies that can activate complement, and through novel signaling pathways activated by membrane attack complex, further enhance immune functions of EC. These immune functions of EC present new targets for therapeutic intervention. [Grants from NIH (HL36003, HL51034, and HL109455), AbbVie and Alexion]. Refs: Pober and Tellides, Trends Immunol 2011; 33:49-57; Pober and Sessa, Cold Spring Harb Perspec Biol. 2104; 7 pii:a016345; Abrahimi, Liu and Pober Am J Transplant 2015; 15:1748-1754.

3.0: BASIC ASPECTS OF INNATE IMMUNE CELLS

3.1
TISSUE-RESIDENT INTIMAL MACROPHAGES CONTRIBUTE TO ATHEROSCLEROTIC LESION INITIATION AND PLAQUE PROGRESSION

Jesse Williams[1], Ki-wook Kim[1], Stoyan Ivanov[1], Slava Epelman[2], Kory Lavine[3], Bernd Zinselmeyer[1], and Gwendalyn Randolph.[1]
[1]Pathology/Immunology, Washington Univ. in St. Louis, BJCIH 8307/Randolph Lab, 425 S. Euclid Ave., St. Louis, MO, 63110, [2]Div. of Cardiology, Univ. Hlth. Network, Univ. of Toronto, Toronto Med. Discover Tower, 3rd Fl. Rm. 3-903, 101 College St., Toronto, ON, M5G 1L7, Canada, [3]Cardiovascular Div., Washington Univ. in St. Louis, Campus Box 8086, 660 S. Euclid Ave., Saint Louis, MO, 63110.

Atherosclerosis is an underlying cause of cardiovascular disease (CVD) and a leading cause of morbidity and mortality worldwide. Macrophage accumulation in plaques, their uptake of cholesterol, and subsequent local death drive disease progression. A population of so-called vascular dendritic cells has also been described that take up lipid early in plaques, but it is unclear if they persist and contribute to disease as it progresses. Here, we thoroughly characterize the presumed dendritic cells and find that they are instead macrophages and we go on to show they persist in plaques through disease progression, contributing to the heterogeneity of plaque macrophages. Using en face whole-mount confocal microscopy of aortas, we identify a uniform resident CD64+ CD11c+ CX3CR1+ MHCII+ macrophage population, which is present in C57/BL6 mice resistant to atherosclerosis. However, they did not express dendritic cell restricted genes zBTB46 or L-myc. Aortic macrophages required M-CSF and Flt3 signaling for survival, but were independent of CCR2, CCR7, and GM-CSF receptor signaling, making them a distinct myeloid population. Lineage-tracing and parabiosis approaches revealed that these cells derive from definitive hematopoiesis and are then self-maintained independent of blood-progenitors. Using these characterization data, we developed a labeling strategy to identify resident from recruited macrophages during kinetic studies of lesion progression. We find that resident aortic macrophages are the first cells to take up lipid following high fat diet exposure and proliferate within the arterial wall to form the initial lesion bed. In the absence of resident

macrophages early lipid deposition in the aortic arch is ablated. They remain a component of plaque macrophages at all stages we examined and seem to localize particularly to plaque borders. Finally, we have developed isolation approaches to characterization gene expression profiles of resident aortic macrophages before and following high fat diet exposure relative to monocyte-derived macrophages. Future studies will determine whether these macrophages have a distinct role and regulation in atherosclerosis compared with macrophages derived from other origins.

3.2
IL-13 SIGNALING IN TISSUE REPAIR AND FIBROSIS

Richard L. Gieseck III[1,2], Thirumalai R. Ramalingam[1], Kevin M. Hart[1], Kevin M. Vannella[1], David A. Cantu[1], Wei-Yu Lu[3], Sofia Ferreira-González[3], Stuart J. Forbes[3], Ludovic Vallier[2,4], and Thomas A. Wynn[1]

[1]Immunopathogenesis Section, Lab. of Parasitic Dis., NIAID, NIH. Bethesda, Maryland, 20852, [2]Wellcome Trust–Medical Res. Council Stem Cell Inst., Anne McLaren Lab., Dept. of Surgery, Univ. of Cambridge, Cambridge, UK, [3]Med. Res. Council Ctr. for Regenerative Med., Univ. of Edinburgh, Edinburgh, UK, [4]Wellcome Trust Sanger Inst., Hinxton, UK.

Fibroproliferative diseases are a major cause of morbidity and mortality and affect nearly every organ system in the body. Recent studies have suggested that type-2 cytokine responses (Interleukin-4/13) are critically involved in tissue repair; however, the mechanisms that regulate tissue repair versus pathological fibrosis are not well understood. Here, we show that the type-2 effector cytokine interleukin-13 simultaneously, yet independently, directs hepatic fibrosis and the compensatory proliferation of hepatocytes and biliary cells. Using conditional knockout mice with interleukin-13 signaling disrupted in hepatocytes, biliary cells, or resident tissue fibroblasts, we reveal direct and distinct roles for interleukin-13 in fibrosis, steatosis, cholestasis, and hepatobiliary proliferation. Together, these studies show that these mechanisms are simultaneously controlled but distinctly regulated by interleukin-13 signaling. Thus, IL-13 could be exploited in the clinic to promote tissue repair without generating pathological fibrosis.

3.3
THE REGULATION OF MACROPHAGE ACTIVATION BY ENDOGENOUS SECRETORY PRODUCTS

Kajal Hamidzadeh[1], Elizabeth Dalby[1], Stephen Christensen[1], and David Mosser[1]

[1]Cell Biology and Molecular Genetics, Univ. of Maryland, Rm. 3102 BRB, College Park, MD, 20742.

Genome-wide transcriptional profiling was performed to examine the resolution of the macrophage activation response over time following stimulation with the TLR ligand, LPS. As expected, the initial response to this stimulus included the upregulation of transcripts encoding inflammatory cytokines, chemokines, and mediators. TLR stimulation also rapidly induced transcripts encoding receptors for purinergic nucleotides and prostaglandins. Over time many of the cytokines and chemokines that were initially produced were turned off, and replaced by an upregulation in transcripts encoding growth and angiogenic factors and anti-inflammatory cytokines. The induction of these new regulatory transcripts depended on the initial stimulus-induced upregulation of receptors for purinergic nucleotides and prostaglandins. We hypothesize that human macrophages maintain homeostasis by the stimulus-dependent induction of receptors for adenosine and prostaglandin. The upregulation of receptor expression increases their sensitivity to the endogenous production of purinergic nucleotides and prostaglandin E2. We propose that this feedback loop is necessary for macrophages to actively terminate inflammatory responses and initiate the process of wound healing. This work was supported by NIH R01 GM 102589 to DMM.

3.4
IMMUNOGENETICS OF NATURAL KILLER CELLS

Peter Parham[1]

[1]Structural Biology, Stanford Univ., D157 Fairchild Bldg., 299 Campus Dr. W., Stanford, CA, 94305.

Natural killer (NK) cells are lymphocytes of innate immunity that have many of the same effector functions as the CD4 and CD8 T cells of adaptive immunity. Notably, they include cellular cytotoxicity and cytokine production. The development, also called education, and immune response of NK cells are regulated by interactions between NK cell receptors and major histocompatibility complex (MHC) class I ligands. Whereas T- cell receptor diversity is created by rearranging genes encoding its component polypeptides, NK cell receptor diversity is the result of a variety of receptors, each encoded by a conventional non-rearranging gene. The MHC class I receptors of human NK cells are membrane glycoproteins of two structural very different types: the CD94:NKG2A/C receptors with binding sites similar to those of C-type lections and the killer cell immunoglobulin-like receptors (KIR) that have binding sites made from two or three immunoglobulin-like domains. The ligand for CD94:NKG2A/C receptors is the complex of HLA-E, a conserved MHC class I mol-

ecule, and a nonamer peptide that is cleaved from the leader sequences of HLA-A, HLA-B and HLA-C, the highly polymorphic MHC class I molecules. The ligands for the KIR receptors are a set of mutually exclusive epitopes of the HLA-A, -B and -C molecules. Although structurally different the lectin-like receptors and the immuneglobulin-like receptors are functionally analogous and work together in complementary fashion. A major difference is that the interactions between and HLA-A,-B and -C are highly diverse within human individuals and populations, whereas the interaction of HLA-E and CD94:NKG2A/C is conserved.

3.5
DAMP'S AND PAMP'S

M. Karen Newell Rogers[1]
[1]Surgery, Texas A&M Hlth. Sci. Ctr. Coll. of Med., 702 H. K. Dodgen Loop, MRB 114, Temple, TX, 76504.

The innate immune response is the "front line" of defense against infection, damage, and injury, culminating in acute inflammation. An evolutionarily conserved protective strategy of the innate response is the host response to **pathogen-associated molecular patterns** (**PAMP**s), molecules with relatively conserved molecular patterns that are the products of pathogens, or **damage/danger associated molecular patterns (DAMPs)**, host-derived molecules that induce a noninfectious inflammatory response. PAMPs are recognized by Toll-like receptors (TLR) in both plants and animals. PAMPs protect the host from infection by inducing an acute inflammatory response. Lipopolysaccharides (LPS) are released from bacteria, and as prototypical PAMPs are recognized by TLR4, while other PAMPs including CpG motifs are recognized by TLR9. Bacterial flagellin are recognized by TLR5, and virally associated nucleic acids, including double- or single-stranded RNA are recognized by TLR3. When PAMPS bind to the cellular TLR, adapter proteins within the cell are recruited to the complex and are then responsible for the activation of protein kinases that can lead to inflammation, the production of cytokines, proliferation, and survival. Proteins of the NOD family of receptors (NLR) detect a variety of stimuli and are central to an IL-1 response. TLR engagement can also facilitate the transition to an adaptive immune response. DAMPs are the products of damage or injury to the host and, like PAMPs, bind to host receptors that initiate and propagate signals that lead to inflammation, cytokine production, and survival signals. Following tissue damage, DNA can be released outside the cell where it serves as a danger signal. The characteristics of DAMPs vary between injured tissues. In addition to nucleic acids as DAMPs, protein DAMPs include heat-shock proteins, high-mobility group box 1 (HMGB1) proteins, extracellular matrix proteins, ATP, uric acid, heparin sulfate, and S100 proteins, a family of calcium modulate proteins. Receptors for HMGB1 include TLR2 and TLR4. HMGB1 can induce increased expression of costimulatory molecules and CD11c on dendritic cells, can induce production of IL-1, TNF-α, IL-6, and IL-8, and can upregulate expression of cell adhesion molecules on endothelial cells. Extracellular ATP can trigger mast cell degranulation via P2X7 receptor signaling and adenosine via P1 receptor signals. Thus, a system of either microbial or host derived molecules, the products of infection or injury, respectively, are centrally important in initiating the protection afforded by an innate immune response to the infection or the danger resulting from host injury. REFERENCES: Han J, Brown T, Beutler B. Endotoxin-responsive sequences control cachectin/TNF biosynthesis at the translational level. J Exp Med. 1990;171:465–475. Janeway C (September 1989). "Immunogenicity signals 1,2,3 ... and 0". *Immunol. Today*. **10** (9): 283–6. doi:10.1016/0167-5699(89)90081-9. PMID 2590379. Matzinger P (1994). "Tolerance, danger, and the extended family". *Annu. Rev. Immunol.* **12**:991–1045. doi:10.1146/annurev.iy.12.0-40194.005015. PMID 8011301.

3.7
MHC AND ANTIGEN PRESENTATION

Elizabeth Mellins
Dept. of Pediatrics and Program in Immunology, Stanford Univ., Stanford, CA 94305.

T cells require that protein antigen be processed into peptides, typically inside antigen presenting cells (APC), such as dendritic cells or macrophages, and the peptides are then bound to major histocompatibility complex (MHC) molecules and these MHC/peptide complexes are presented on the APC surface. This paradigm applies to both CD4+ T and CD8+ T cells, with CD4+ T cells recognizing MHCII/peptide complexes and CD8+ T cells recognizing MHCI/peptide complexes. Peptide loading of MHCI and MHCII molecules occurs in primarily in different intracellular compartments, with the involvement of distinct accessory molecules. Both canonical and alternative peptide-loading pathways have been described, presumably broadening self and foreign peptide presentation for tolerance and host defense, respectively. These pathways also contribute to immune consequences associated with the atherosclerotic lesion. Reference: Mellins, E.D., 2016. Ligand Selection and Trafficking for MHC II. In: Ratcliffe, M.J.H. (Editor in Chief), Encyclopedia of Immunobiology, Vol. 2, pp. 247–254. Oxford: Academic Press.

4.0: POSTER SESSION I

4.1
KLF6-BCL6 SIGNALING AXIS REGULATES MACROPHAGE-MEDIATED INFLAMMATION

Ganapati Mahabaleshwar[1]
[1]Dept. of Med., Case Western Res. Univ., 2103 Cornell Rd., Cleveland, OH, 44106.

The macrophages are the predominant innate immune cells recruited to tissues following injury or infection. These early-responding pro-inflammatory macrophages play an essential role in the amplification of inflammation. However, macrophage pro-inflammatory gene expression should be tightly regulated to avert host tissue damage. In this study, we identify KLF6-BCL6 signaling axis as a novel regulator of macrophage inflammatory gene expression and function. Utilizing complementary gain- and loss-of-function studies, we observed KLF6 is essential for macrophage motility under *ex vivo* and *in vivo* conditions. Concordant with these observations, myeloid specific deficiency of KLF6 significantly attenuate macrophage pro-inflammatory gene expression, recruitment and progression of inflammation. At the molecular level, KLF6 suppress Bcl6 expression by elevating PRDM1 levels in macrophages. Interestingly, pharmacological or genetic inhibition of BCL6 in KLF6-deficient macrophages completely abrogated attenuation of pro-inflammatory cytokine/chemokine expression and cellular motility. Collectively, our observations reveal that KLF6 repress BCL6 to enhance macrophage inflammatory gene expression and function.

4.2
AORTIC AND MICROVASCULAR ENDOTHELIAL CELLS DIFFERENTIALLY INFLUENCE INFLAMMATORY AND VASCULAR RESPONSES TO ENGINEERED NANOMATERIALS

Valerie Minarchick[1], Jonathan Shannahan[1], Edward Sabolsky[2], and Jared Brown[1]
[1]Pharmaceutical Sci., Univ. of Colorado Denver, 12850 E. Montview Blvd., MS C238, Aurora, CO, 80045, [2]Mechanical and Aerospace Engineering, West Virginia Univ., Benjamin M. Statler Coll. of Engineering and Mineral Resources, PO Box 6106, Morgantown, WV, 26506.

Exposure to cerium dioxide nanoparticles (CeO_2) has been shown to cause vascular dysfunction in animal models. However, the mechanisms and target cell types of vascular dysfunction are unknown. We hypothesized that phenotypic differences in aortic and microvascular endothelial cells will influence responses to CeO_2 exposure and ultimately impact vascular dysfunction. Rat aortic endothelial cells (RAEC) or rat microvascular endothelial cells (RMEC) were exposed to CeO_2 (0-100 μg/ml). Cytotoxicity, alterations in inflammatory (IL-6) and surface adhesion marker (ICAM and VCAM) mRNA expression, and reactive oxygen species (ROS) generation were assessed following exposure. To assess potential influences on vascular function, isolated naive arterioles were intraluminally exposed to the supernatant from CeO_2 exposed RAEC and RMEC. Endothelium-dependent and –independent reactivity was assessed with acetylcholine (ACh, 10^{-9}-10^{-4} M), and spermine NONOate (10^{-9}-10^{-4} M). Finally, enhanced darkfield microscopy and flow cytometry was used to determine CeO_2 cellular uptake in vitro. Following exposure to 25 μg/ml CeO_2, RAEC had an increase in nitric oxide synthase (NOS, 2.5-fold) and VCAM (3.3-fold) mRNA expression. RMEC also had an increase VCAM (2.8-fold) which was similar to the RAEC; however, NOS mRNA expression (35.7-fold) significantly higher than the RAEC. Lastly, CeO_2 exposure did not elicit ROS production in RAEC but was increased 23±1.3% in RMECs as compared to control following 25 μg/ml treatment. Taken together, these results indicate that the aortic and microvascular endothelium respond uniquely to CeO_2. Furthermore, these distinct responses may have differential impacts on vascular function. R01-ES019311 (JMB), K99-ES024392 (JHS).

4.3
LYMPHATICS-THE KEY COMPONENT OF THE IMMUNOVASCULAR SYSTEM

David Zawieja[1], Wei Wang[1], Walter Cromer[1], Anand Narayanan[1], Anatoliy Gashev[1], and Mariappan Muthuchamy.[1]
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The lymphatic vascular system is part of the cardiovascular system responsible for the return of fluid and macromolecules extravasated from the exchange blood vessels into the interstitium back to the venous blood. In this role it accounts for the removal of ~ 4 liters of fluid from the interstitium daily in the form of lymph. Lymphatics are known to play key roles in the development and resolution of inflammation in many body tissues. Afferent lymph also carries macromolecules, lipids, antigens, cytokines, parenchymal cell degradation products, infectious and inflammatory agents, immune cells and other immune relevant signals from the interstitium to the node. Efferent lymph then carries the unique response elements elicited from the nodal cells into the lymph inside the node back to the blood. Understanding the role of the lymphatics in the regulation and integration of fluid and immune homeostasis if of critical importance towards a

full understanding of edema, inflammation and immunity. Over the last decade we have integrated our studies of the role of lymphatics in body fluid homeostasis with their roles in inflammation, tolerance and immune response. This work has lead us to identify and characterize the intimate interactions of the muscularized prenodal lymphatics with a number of immune cells that exist in and around these lymphatics and are responsive to inflammatory and/or tolerogenic signals from the parenchyma. These interactions lead to the modulation of lymph transport as a means to alter immune signal delivery to the lymph node and to changes in immune cell function. We identified a unique resident population of $MHCII^+$ cells that live on and within the wall of the muscularized lymphatics that can rapidly sense, capture and process antigens carried in lymph. These cells are variably attracted to the lymphatics under different conditions, can then modulate lymphatic function and be quickly transported to the node to integrate rapid remote immune responses. These and other local and resident immune cells have been shown to interact with each other, and to local and distant parenchymal inflammatory/immune signals carried in lymph to modulate lymphatic transport and immune functions. Thus the lymphatics form the critical information path of the immunovascular system responsible for linking the fluid and immune status of the parenchymal environment to inflammation, immune response and cardiovascular function.

4.4
PLASMA LEVELS OF ANGIOPOIETIN-1, ANGIOPOIETIN-2 AND VASCULAR ENDOTHELIAL GROWTH FACTOR IN SICKLE CELL ANAEMIA PATIENTS WITH OR WITHOUT COMPLICATIONS IN GHANA: A CASE-CONTROL STUDY

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Sickle Cell Disease (SCD) results in ongoing vasculopathy and end organ damage. Angiogenesis have been implicated as a key contributing factor to ongoing vascular mediated tissue injury in SCD. The relative plasma levels of angiopoietin-1 (Ang-1), angiopoietin-2 (Ang-2) and vascular endothelial growth factor (VEGF) greatly influence angiogenesis. Dysregulation of these growth factors leading to a pro-angiogenic state have been found in blood samples of Non- Ghanaian SCD. Ghana has one of the highest prevalence of SCD where 2% of all births are burdened with this disease. To date, there is no data on the levels of angiogenic factors in Ghanaian SCD patients. The aim of this study was to assess plasma levels of Ang-1, Ang-2 and VEGF in homozygous (HbSS) SCD patients with or without complications and healthy controls (HbAA) in Ghana. The study was a case- control study involving 544 participants conducted at the Sickle Cell Clinic Accra, Ghana. The plasma levels of Ang-1, Ang- 2 and VEGF were measured with a double sandwich enzyme-linked immunosorbent assay (ELISA) technique and Complete Blood Count with an autoanalyser. The mean plasma Ang-1, Ang-2 and VEGF were significantly higher in HbSS SCD patient with or without complications than healthy controls ($p<0.001$). The Ang-1/Ang-2 ratio was significantly higher in the controls than the HbSS patients ($p <0.001$). Ang-2/Ang-1 ratio was higher in the HbSS patients with complications especially, leg ulcer patients than healthy controls ($p<0.001$). There were higher plasma levels of Ang-1, Ang-2 and VEGF in HbSS patients with complications than those without complications and healthy controls. In conclusion, there was an overall evidence of angiogenesis, which was highest in HbSS patients with complications, especially those with leg ulcer. The study provides first report on plasma levels of Ang-1, Ang-2 and VEGF HbSS SCD patients in Ghana. Funded by ORID University of Ghana and University of Ghana-Carnegie Next Generation of Academics in Africa.

4.5
MECHANISMS OF INCREASED COMPLEMENT ACTIVATION IN PLACENTAL ISCHEMIA-INDUCED HYPERTENSION IN THE RAT

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Preeclampsia is characterized by newly diagnosed hypertension and reduced placental perfusion. Previous studies in a rat model of placental ischemia-induced hypertension demonstrated that inhibiting complement activation attenuated increased maternal blood pressure, indicating that complement is a promising therapeutic target. Given that both natural IgM and antigen antibody complexes can initiate complement activation, we hypothesized that placental ischemia exposes neoepitopes recognized by IgM to cause complement activation and hypertension. Alternatively, we considered that autoantibody to angiotensin II Type 1 receptor (AT1-AA) interacts with AT1 receptors to cause complement activation. Since complement activation occurs in kidney and placenta in preeclampsia, we used immunohistochemistry to determine IgM deposition and local complement activation in each organ (C3 deposition), and qRTPCR to quantitate mRNA for endogenous regulators of complement activation CD55, CD59 and Crry. On gestation day (GD)14, timed pregnant Sprague Dawley rats underwent Sham surgery or placement of clips on inferior abdominal aorta and ovarian arteries to create placental ischemia (RUPP). As previously reported, RUPP increased mean arterial pressure and circulating C3a on GD19. In placenta, IgM and C3 deposition increased in RUPP vs Sham animals ($p<0.05$), whereas mRNA for complement regulators Crry and CD59 decreased in RUPP compared to Sham (Crry, 0.85±0.03; CD59, 0.79±0.10 fold; $p<0.05$). In kidney, IgM deposition increased in RUPP vs Sham ($p<0.05$) without a significant change in C3 deposition. Kidney mRNA for complement regulators increased in RUPP compared to Sham (CD55, 1.21±0.09, $p=0.07$; CD59, 1.19±0.04 fold, $p<0.05$). The AT1 receptor antagonist losartan is known to prevent AT1-AA interaction with AT1 receptors. Losartan treatment prevented placental ischemia-induced hypertension without affecting complement activation as measured by circulating C3a or placental C3 deposition. Overall, these data suggest that increased complement regulators limit local complement activation in kidney, and decreased complement regulators in placenta are consistent with increased C3 deposition with placental ischemia. Importantly, our studies indicate that complement activation following placental ischemia is not due to AT1-AA but is associated with IgM deposition, suggesting a role for natural antibodies interacting with neoepitopes following placental ischemia. Funding: NIH HL109843.

4.6
PLATELET-P2Y12 RECEPTOR IS IMPORTANT FOR THE CELLULAR IMMUNE RESPONSE IN EROSIVE ARTHRITIS

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Rheumatoid arthritis (RA) is an immune-mediated inflammatory disease with a complex pathogenesis, which comprises activation of humoral and cellular immune responses. There is increasing evidence of the role of platelets in RA. Platelet-derived microparticles, platelet-leukocyte interaction and activated platelets are related to RA development. Therefore, inhibiting platelet activity might benefit RA patients. Adenosine diphosphate activates platelets through the Gq-couple receptor P2Y1 and the Gi-couple receptor P2Y12. P2Y12 receptor has been targeted to inhibit platelet activity using thienopyridine pro-drugs such as clopidogrel and prasugrel. Our previous studies using a rat model demonstrated aggravation of arthritis after administration of thienopyridine compounds. Therefore, we evaluate the role of the P2Y12 receptor in arthritis by using a P2Y12 null mice. In contrast to the results obtained with P2Y12 pharmacological inhibition, the induction of arthritis by administration of collagen antibodies in the P2Y12 KO mice was mild. We found decrease in synoviocyte hyperplasia, articular leukocyte infiltration, pannus formation, and pro-inflammatory cytokine production including I-L6 and TNFα when compared to WT. This conundrum in results could be explained by the difference of the mouse and rat arthritis models. The rat model generates a cellular mediated immune response against the joints of the rat while the mouse model generates an arthritogenic effect due to the humoral immune response of auto-antibodies against the collagen epitope. These suggest that the P2Y12 receptor is a selective regulator of the platelet-mediated cellular immune response in RA, and might have potential for the development of new therapies against RA.

4.7
INTERLEUKIN-19: A NOVEL SEXUALLY DISMORPHIC CARDIAC CYTOKINE

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Heart failure (HF), the leading cause of mortality within the United States, is a sexually dimorphic disease. Al-

though the prevalence of HF is lower in women compared to men, treatment and survival outcomes for female patients are poorer with females having disproportionally higher morbidity and mortality. The deleterious effects of inflammatory cytokines in the context of HF are well documented as elevated circulating levels of cytokines predict adverse outcomes. Few studies, however, have examined sex-specific differences in cytokine expression, though sparse data indicate inflammatory profiles may be sexually dimorphic. Interleukin-19 (IL-19) is a recently described member of the anti-inflammatory IL-10 cytokine family. There is significant controversy surrounding the role of IL-19 in cellular signaling as it has been shown to be both pro- and anti-inflammatory depending on cellular context. To date, the role of IL-19 in the heart has not been characterized. We now show that IL-19 is expressed in the rodent and human heart, with increased expression in a female-dominant model of HF, the dominant-negative CREB (dnCREB) transgenic mouse. Further, we show that the relative expression of the two IL-19 receptor isoforms IL-20Rα/β, known to have divergent influences on cell phenotype, manifest differently in the heart by sex and by disease in both our murine HF model and in cardiac tissue from human HF patients. To test the hypothesis that IL-19 promotes cardiac dysfunction in dnCREB female mice we generated a double transgenic (DTG) mouse of IL-19 knockout and dnCREB. Surprisingly, survival analyses demonstrate that female DTG were not protected and that male DTG mice had accelerated, mortality, as their corresponding dnCREB cohort. To understand the mechanism(s) of IL-19 signaling in the male and female heart, we treated mouse cardiac myocytes and fibroblasts with recombinant IL-19 and show that IL-19 treatment activates STAT3, a canonical downstream IL-19 signaling cascade. Together, these data suggest IL-19 is an important cytokine in sex-specific cardiac dysfunction. Ongoing investigations will elucidate the mechanism(s) of sex-specific IL-19 mediated cardiac remodeling.

4.8
PRIOR PREECLAMPSIA RESULTS IN PERSISTENT IMMUNE ACTIVATION FOLLOWING PREGNANCY IN THE DAHL SALT SENSITIVE RAT

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Preeclampsia is a risk factor for later cardiovascular disease resulting in an increased risk of stroke, heart attack, hypertension, and chronic kidney disease 5-15 years postpartum. While it is known that immune activation is involved in the pathogenesis of preeclampsia and that T cells contribute to hypertension and end organ damage in male experimental animals, the role of T cells in the progression of cardiorenal disease following preeclampsia has not been examined. We hypothesized that prior preeclamptic pregnancy would result in postpartum activation of T cells and production of the inflammatory cytokine tumor necrosis factor (TNF)-α. Dahl salt-sensitive rats on a 0.3% salt diet (previously characterized by our lab as a model of superimposed preeclampsia) who experienced 2 pregnancies (at 12 and 17 weeks of age) and virgin littermate controls were allowed to age to 6 months. Rats were implanted with telemetry transmitters (DSI), mean arterial pressure (MAP) was recorded, and rats were placed in metabolic cages for 24 hour urine collection prior to tissue harvest. Prior pregnancy did not result in a further increase in MAP at 6 months (virgin: 185±6.9 mm Hg, prior pregnancy: 184.6±6.6 mm Hg, n=8-10); however, despite similar blood pressure, rats who experienced prior preeclamptic pregnancy had significantly greater renal injury compared to virgin littermates. Urinary excretion of protein (96±20 vs 195±45 mg/day), nephrin (0.6±0.4 vs 3.1±1.2 μg/day), and podocalyxin (4.9±1.0 vs 21.0±7.6 μg/day) was higher in the postpartum group ($p<0.05$, Bradford assay and Exocell ELISA). These measures of renal injury were corroborated by histological examination as kidneys from rats that experienced preeclampsia (2.9±0.3) demonstrated greater glomerular sclerosis compared to virgin littermates (2.5±0.3, $p=0.05$). Flow cytometry analysis revealed that $CD3^+$ T cells (10±3 vs 19±3%, $p<0.05$) and specifically $CD3^+/CD4^+$ T helper cells (10±4 vs 17±2%, $p=0.06$) are greater in the kidney following preeclamptic pregnancy. Furthermore, we observed greater plasma levels of the pro-inflammatory cytokine TNF-α (0.7±0.3 vs 1.5±0.1 pg/ml) and lower levels of the anti-inflammatory cytokine interleukin-10 (12.0±1.0 vs 8.8±1.4 pg/ml, $p<0.05$), suggesting that there may be an imbalance in effector T cells vs regulatory T cells. These data support the hypothesis that activation of immune cells could link the maternal syndrome of preeclampsia to the increased postpartum risk of cardiovascular and renal disease.

4.9
INCREASED PRO-INFLAMMATORY T CELLS AND HMGB1 ARE ASSOCIATED

WITH VASCULAR DYSFUCTION IN MALE SHR

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Dysfunctional inflammatory responses have been implicated in the development of hypertension, however, the mechanisms that trigger these inflammatory processes are not yet well understood. High mobility group box 1 protein (HMGB1) is a damage-associated molecular pattern released by dying cells that can then trigger an inflammatory response and cause vascular dysfunction. Recently, our lab studied vascular reactivity in the aortas of male and female Spontaneously Hypertensive Rats (SHR) and normotensive Wistar Kyoto (WKY) rats. Male rats had enhanced contractile and diminished vascular relaxation, compared to female rats, in both strains. Furthermore, SHR males had the greatest contractile response and most diminished vasorelaxation compared to all other groups. We hypothesized that hypertension in the SHR increases HMGB1 to promote a pro-inflammatory T cell profile in the aorta. Male and female SHR and WKY rats (n=6 per group) were sacrificed at 13 weeks of age, and aortas were collected for flow cytometric analysis. Anti-inflammatory Tregs were lower in both male ($p<0.001$) and female ($p=0.001$) SHR compared to the normotensive WKY controls, with male SHRs having the lowest Treg population (Table). Pro-inflammatory $TH17^+$ cells and $HMGB1^+$ cells were greater in the SHRs relative to the WKY rats (Table). No sex difference was observed in $TH17^+$ cells in the WKY rats, but male SHRs did have significantly greater $TH17^+$ cells compared to the female SHR ($p=0.02$). Interestingly, a sex difference in HMGB1 levels was observed in both strains, with male SHR ($p<0.001$) and WKY ($P=0.02$) having greater expression of HMGB1 compared to their female controls. These data suggest that enhanced aortic vascular dysfunction in male SHR is associated with greater increases in pro-inflammatory T cells and HMGB1 compared to all other groups. Future studies will address the ability of HMGB1 to activate T cells and induce vascular dysfunction.

	Tregs	$TH17^+$	HMGB1
Female SHR	$4.3\pm0.4^{*\#}$	$5.0\pm0.8^{*\#}$	$4.0\pm0.6^{*\#}$
Male SHR	$2.8\pm0.5^{\#}$	$8.8\pm1.1^{\#}$	$8.8\pm0.7^{\#}$
Female WKY	7.5 ± 0.6	1.5 ± 0.3	$1.3\pm0.2^{*}$
Male WKY	6.2 ± 0.5	2.3 ± 0.3	2.5 ± 0.3
$^{*}p<0.05$ vs. male, same strain, $^{\#}p<0.05$ vs. WKY, same sex			

4.10
MITOCHONDRIAL ROS MEDIATES PKM2 KINASE ACTIVITY TO PROMOTE PRO-INFLAMMATORY ACTIVITY OF HUMAN MACROPHAGES IN CORONARY ARTERY DISEASE

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Inflammatory macrophages are key drivers of the tissue inflammatory response typical for coronary artery disease (CAD). Reactive oxygen species (ROS) are produced in macrophages by cytoplasmic and mitochondrial sources and regulate macrophage function through a multitude of pathways. Processes underlying the production of pro-inflammatory cytokines by macrophages in CAD are insufficiently understood. Here, we have examined how metabolic activity of such macrophages is mechanistically connected to their cytokine release. We found that LPS stimulated macrophages from coronary artery disease (CAD) patients overexpress the glycolytic master regulators, c-Myc and HIF-1α as well as genes involved in glucose transport and its glycolytic breakdown (PKM2, GLUT3, PDK1, PFK FB3, HK2, PGK1, PFK1, and GLUT1). To understand the contribution of the glycolytic machinery to cytokine production, we quantified and manipulated the metabolic activity of these macrophages. Compared to healthy macrophages, CAD macrophage had over 20% higher rate of glucose absorption and utilization. Their mitochondrial respiration and membrane potential indicated higher mitochondrial activity. Using [U-$^{13}C_5$] glutamine isotope tracing we analyzed glutamine utilization as an indicator of mitochondrial activity and confirmed intensified mitochondrial metabolism. ^{13}C enrichments of the Krebs cycle-associated metabolites malate, aspartate, and glutamate increased by approximately two-fold in CAD macrophages. We found that increased metabolic activity augmented mitochondrial superoxide and hydrogen peroxide production. These mitochondrial ROS targeted the glycolytic enzyme pyruvate kinase M2 (PKM2) and induced its dimerization. Dimerized PKM2 was translocated to the nucleus where it gained protein kinase activity and phosphorylated the transcription factor STAT3. Increased STAT3 phosphorylation was sufficient to accelerate production of both IL-6 and IL-1β. Treatments that reduced glucose metabolism (2DG), promoted

PKM2 tetramerization (ML265) or limited ROS production (mitoTempo, Tempol) prevented PKM2 dimerization, abrogated translocation to the nucleus, suppressed STAT3 phosphorylation and corrected the proinflammatory CAD macrophage phenotype. Taken together, our study shows that the production of the key pro-inflammatory cytokines IL-6 and IL-1β in CAD is driven by metabolic reprogramming and depends on a pathway involving oxidative modification of a cytoplasmic enzyme to sustain nuclear STAT3 activation.

4.11
IDENTIFICATION OF AN ADDITIONAL ROLE FOR APOC-III MODULATES BASAL T CELL PROFILE IN MICE

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Apolipoprotein C-III (apoC-III) is a component protein of triglyceride-rich lipoproteins and an independent predictor of cardiovascular disease (CVD) in humans. ApoC-III functions in the blood and liver to increase plasma triglyceride levels, but in the intestine it functions to delay dietary fat absorption. Therefore, apoC-III may have differing roles in promoting CVD, depending upon the circumstances of its expression and secretion. Since the immune system plays such a large role in atherogenesis, we asked whether apoC-III is a potential regulator of inflammation and immune profile, which has not yet been fully elucidated. We characterized the circulating $CD3^+CD4^+$T cell subsets and $CD14^+CD68^+$ monocyte subsets in mice that overexpress the human apoC-III gene (apoC-III Tg) compared with their wild-type C57Bl/6J littermates. Whole blood was isolated from the tail of each group and prepared for flow cytometric analysis. Samples were lysed, stained for surface antigens, fixed in 2% paraformaldehyde, and permeabilized for intracellular staining. Flow cytometric analysis was performed using BD LSR II-B (Becton-Dickinson) after appropriate fluorescence compensation, and subsets were gated using FlowJo software (Treestar). Interestingly, total $CD19^+$ B cell count did not vary between the groups but total $CD3^+$ T cells, $CD4^+$ T cells, $CD4^+CD25^+FoxP3^+$ regulatory T cells, and $CD4^+CXCR4^+$ helper T cells decreased significantly in the apoC-III transgenic mice. Overall, significant increases were seen in the $CD8a^+$ cytotoxic T cell and $CD4^+CD44^{high}CD62L^{high}$ central memory T cell populations. In the myeloid cell fraction, significant increases were seen in the Ly-$6C^{High}$ M1-like macrophage population while $CD68^-$Ly-$6C^+$ monocytes significantly decreased in apoC-III Tg. We also characterized the cytokine profile in these mice to confirm the phenotype of these cells. Interestingly, these studies were completed in the absence of high fat diet feeding or LPS stimulation, suggesting that apoC-III itself, or its causative role in hypertriglyceridemia, results in changes to the circulating immune profile in mice. These results show a significant trend towards a pro-inflammatory profile of apoC-III Tg at baseline and suggest that apoC-III may play a previously unknown role in stimulating $CD8a^+$ T-cells to modulate atherogenesis.

4.12
ROLE OF SMOOTH MUSCLE CELL SPECIFIC IPLA2β IN VASCULAR INFLAMMATION AND NEOINTIMAL FORMATION IN A MURINE FEMORAL ARTERY WIRE INJURY MODEL

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Coronary heart disease is the single leading cause of death in the United States. Coronary revascularization, including coronary artery bypass graft and percutaneous coronary intervention (PCI) is the most common modality in patients with coronary diseases. However, it is also among the most costly and is often associated with a high incidence of restenosis. Thus, there is an urgent need to identify new therapeutic targets for coronary heart disease. Calcium independent phospholipase A_2β (iPLA_2β) is a member of the phospholipase A_2 superfamily that acts on phospholipids to produce a free fatty acid and a lysophospholipid. iPLA_2β is ubiquitously expressed and is implicated in many human diseases. To investigate whether targeting smooth muscle-specific iPLA_2β is sufficient to reduce vascular inflammation and neointima formation, we established a femoral artery injury model that better mimics PCI and developed a novel tamoxifen-inducible smooth muscle-specific iPLA_2β knockout mouse model (SM-iPLA_2β-iKO). By using genomic PCR with smooth muscle-specific Cre-mediated genomic DNA recombination, we demonstrated that iPLA_2β was specifically deleted in smooth muscle tissues such as aorta, carotid artery, mesenteric artery, and bladder, but not that in others, including heart, kidney, lung, brain, liver, and skeletal muscle. Smooth muscle cell specific deletion of iPLA_2β was verified by real-time PCR, Western blot, and iPLA_2 enzymatic assay. Interestingly, SM-iPLA_2β-iKO mice, unlike global iPLA_2β knockout mice, did not have

iPLA$_2\gamma$, iPLA$_2\zeta$, and cPLA$_2\alpha$ genetic compensatory up-regulation. iPLA$_2\beta$ protein was upregulated by wire injury and was predominantly detected in neointima area in a time dependent manner. Using a femoral artery injury model, we illustrated that wire-injury-induced neointima formation was markedly decreased in SM-iPLA$_2\beta$-iKO mice, which was temporally correlated with a remarkable reduction of neutrophil infiltration into neointima in SM-iPLA$_2\beta$-iKO mice. These data demonstrate a critical role of smooth muscle cell iPLA$_2\beta$ in neointima formation and inflammation in a femoral artery injury model, suggest that smooth muscle cell iPLA$_2\beta$ participates in the initiation and early progression of vascular inflammation and neointima formation, and indicate that iPLA$_2\beta$ may represent a novel therapeutic target for treatment of coronary heart disease. This work was supported by VA Merit Award I01BX002141, NIH grants HL088389, HL106843, and HL125228.

4.13
ROLE OF NEUREGULIN-1β IN THE ACUTE INFLAMMATORY RESPONSE TO CORONARY ARTERY BYPASS GRAFTING (CABG)

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Neuregulin-1β (NRG) is a cardioactive growth factor required for cardiac morphogenesis and maintenance. NRG interacts with tyrosine kinase receptors ERBB2, ERBB3, and ERBB4 to activate intracellular signaling cascades regulating proliferation, apoptosis, protein synthesis, cell motility and survival. Our previous work has demonstrated a cardioprotective effect of recombinant NRG therapy in heart failure, associated with a decrease in production of the inflammatory cytokine tumor necrosis factor alpha (TNFα). This led us to hypothesize that NRG may be beneficial in attenuating the inflammatory response to CABG, a common surgical intervention for coronary artery blockage. Following CABG, some patients develop an unrestrained systemic inflammatory response syndrome (SIRS), which can ultimately lead to multiple organ dysfunction and death. It is difficult to predict which patients are at highest risk for SIRS, and there are no previous studies connecting NRG and the acute inflammatory response in cardiovascular disease. For this study, we are examining whether individual variability in circulating NRG correlates with risk for SIRS, as measured by production of inflammatory cytokines in the post-operative period. Our patient cohort is comprised of adults undergoing CABG at Maine Medical Center, with a target enrollment of 150 patients. Peripheral blood specimens are obtained preoperatively, during surgery, at 4-8 hours, at 24 hours and at 4 days following surgery. Blood specimens are then tested for circulating NRG levels, and levels of the acute inflammatory cytokines TNFα, interleukin (IL)-6, and IL-8 by enzyme-linked immunosorbent assay (ELISA). As expected, our preliminary data indicate an acute increase in inflammatory cytokine production following CABG. IL-6 and IL-8 production peak at 4-8 hours post surgery. A modest but non-significant increase in TNFα is also detected in some patients at 4-8 hours. Surprisingly, our preliminary data show the full range of endogenous NRG response to CABG, ranging from increasing detection of NRG, decreasing detection of NRG, to no change in detection levels over the surgical and post-operative period. Upon completion of this study, we will be able to determine whether circulating NRG status correlates with the inflammatory response to CABG. These studies are of high translational potential given that recombinant NRG therapy has already shown promise as a safe and effective therapy for heart failure. Funded by the Osher Research Fund at Maine Medical Ctr.

4.14
THE ROLE OF PSGL-1-EXPRESSING CD4 T CELLS IN THE ATHEROSCLEROTIC DEVELOPMENT AND PLAQUE INSTABILITY

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Background: Many inflammatory infiltrates, such as leukocytes are observed in the atherosclerotic plaque. Adhesion of circulating leukocytes to the endothelial cells and subsequent trans-endothelial migration are important steps in the development of atherosclerosis. Recently, we found PSGL-1$^+$CD4 T cells contribute the pathogenesis of acute coronary syndrome. **Methods and Results:** In this study, we examined the role of PSGL-1$^+$CD4 T cells for the atherosclerotic development and plaque instability. We constructed PSGL-1$^{-/-}$ApoE$^{-/-}$mouse fed with high-fat diet (PSGL-1$^{-/-}$ApoE$^{-/-}$) and compared to apolipoprotein E$^{-/-}$ mice (ApoE$^{-/-}$) fed with high-fat diet (HFD). Immunohistochemistry of aortic sinus showed many integrinβ_2^+ CD4 T cells, IFNγ^+CD4 T cells (Th1), and IL17$^+$CD4 T cells (Th17) were infiltrated into the atherosclerotic

plaque in $ApoE^{-/-}$, however, these inflammatory infiltrates were less in $PSGL\text{-}1^{-/-}ApoE^{-/-}$. Furthermore, we examined the expression of PSGL-1 ligands, P-selectin and E-selectin in atherosclerotic plaque. P-selectin and E-selectin expression on endothelial cells and vascular smooth muscle cells were strongly increased in $ApoE^{-/-}$. In contrast, those in $PSGL\text{-}1^{-/-}ApoE^{-/-}$ were inhibited. Finally, TUNEL positive apoptotic cells in atherosclerotic plaque were strongly suppressed in $PSGL\text{-}1^{-/-}ApoE^{-/-}$, contributing more stabilized morphologic features. **Conclusion:** We concluded that PSGL-1 expressing $CD4^{+}$T cells participate directly in the development of atherosclerosis and plaque instability.

4.15
INFLUENCE OF HIV-1 ON CIRCULATING VASCULAR-RELATED $_{MICRO}$RNAS

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Over 1.2 million adults in the United States are living with human immunodeficiency virus (HIV)-1. Alarmingly, cardiovascular disease (CVD) is the leading cause of morbidity and mortality in adults with HIV-1 in the United States. MicroRNAs (miRs) are short (~22 nt), non-coding RNAs that regulate gene expression post-transcriptionally. miRs are transported in the circulation by argonaute2 complexes, lipoproteins and microvesicles. Extracellular miRs are abundant and stable in blood and have been linked with vascular disease and numerous CVD risk factors such as dyslipidemia and hypertension. As a result, it has been suggested that circulating miRs may be a useful biomarker for vascular risk and disease. The aim of this study was to determine whether the expression of specific circulating endovascular-related miRs (miR-17, miR-92a, and miR-126) are dysregulated in HIV-1 seropositive adults. Thirty-eight adults were studied: 18 HIV-1 seronegative (12M/6F; age 40±3 yr) and 20 HIV-1 seropositive (13M/7F; 38±2 yr) on stable efavirenz-based antiretroviral therapy. All subjects were non-obese, normotensive, normolipidemic and free of overt cardiometabolic disease. Circulating expression of miRs was determined in plasma using standard RT-PCR techniques with miR primers of interest. Expression was normalized to exogenous C. *elegans* miR-39 and reported as relative expression in arbitrary units (AU). There was no significant difference in circulating expression of miR-17 in the HIV-1 seronegative (0.10±0.03 AU) and seropositive (0.11±0.04 AU) groups. In contrast, circulating expression of miR-92a was ~70% higher (2.96±0.47 vs 1.71±0.35 AU; $P<0.05$) and miR-126 was ~90% higher (0.58±0.11 vs 0.30±0.04 AU) in HIV-1 seropositive compared with seronegative adults. Differential expression of miR-92a may be involved with various pathologies associated with HIV-1. For example, overexpression of miR-92a leads to downregulation of endothelial nitric oxide-synthase and, in turn, nitric oxide bioavailability. In contrast, miR-126 is highly enriched in endothelial cells and considered to be vasculoprotective as it regulates endothelial cell angiogenic activity, cell adhesion and, in turn, vascular permeability. Upregulation of miR-126 with HIV-1 may represent an endothelial protective mechanism. Expression profile of circulating vascular-related miRs may provide mechanistic insight into the increased CVD risk associated with HIV-1 infection and deserves further study.

4.16
HIV-1, INFLAMMATION AND ENDOTHELIAL DYSFUNCTION

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Cardiovascular disease morbidity and mortality is a burgeoning problem in human immunodeficiency virus (HIV)-1-infected adults. Mechanisms underlying the increased cardiovascular risk are not completely understood. Local and systemic inflammation and oxidative stress play an important role in the initiation and progression of atherosclerotic vascular disease. HIV-1 infection and various types of antiretroviral therapy have the potential to adversely influence oxidative stress and inflammatory burden. In an ongoing study, we are testing the hypothesis that increased inflammatory burden and oxidative stress may contribute to endothelial dysfunction in HIV-1-infected adults. To date, 10 HIV-1-seronegative (9M/1F; age 36±3 yr; BMI: 24.0±0.7 kg/m^2) and 10 HIV-1-seropositive adults on stable antiretroviral therapy (9M/1F; 41±2 yr; 25.1±1.1 kg/m^2) have been studied. All subjects were sedentary, normotonesive, normolipidemic and free of overt cardiovascular disease. Forearm blood flow (FBF) responses to intrabrachial infusions of acetylcholine (Ach: 8.0-32.0 μg/min) and sodium nitroprusside (NTP: 2.0-8.0 μg/min) were measured by venous occlusion plethysmography. Plasma concentrations of C-reac-

tive protein (CRP), tumor necrosis factor (TNF)- 1α, interleukin (IL)-6, IL-18, myeloperoxidase (MPO), and oxidized (ox)-LDL were quantified by enzyme immunoassay. FBF responses to Ach were ~40% lower in HIV-1-seropositive (from 4.6±0.4 to 11.9±1.3 mL/100 mL tissue/min) compared with HIV-1-seronegative (4.7±0.3 to 18.7±1.0 mL/100 mL tissue/min) adults. FBF responses to NTP were not different between the groups. Plasma concentrations of CRP (1.1±0.2 vs. 0.5±0.1 mg/L), IL-6 (1.7±0.2 vs. 0.9±0.1 pg/mL), and IL-18 (304.6±30.4 vs. 194.2±14.1 pg/mL) were significantly higher (~50%-100%) in the HIV-1-seropositive than HIV-1-seronegative adults. There were no significant differences in plasma concentrations of ox-LDL (73.1±11.8 vs 49.9±6.6 U/L), MPO (114.7±22.7 vs 104.4±21.2 mg/L), and TNF-α (1.7±0.4 vs. 1.6 ±0.3 pg/mL) between the groups. Plasma concentrations of CRP ($r=-0.68$, $P<0.05$) and IL-18 ($r=-0.56$, $P<0.05$) were inversely related to peak vasodilation to Ach. Increased inflammation may contribute to the impairment in endothelium-dependent vasodilation in HIV-1-infected adults and, in turn, the increased risk of cardiovascular disease in this population.

4.17
THE ALPHA7 NICOTINE ACETYLCHOLINE RECEPTOR AGONIST AZ6983 REDUCES ATHEROSCLEROSIS IN APOE-/- MICE AND TNF PRODUCTION IN HUMAN CELLS

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Background and objective: Autonomic nerve signaling via the alpha 7 nicotinic acetylcholine receptor (α7nAChR) is known to modulate inflammation. We have previously shown that ablation of hematopoietic α7nAChR is associated with increased atherosclerosis, thus, linking α7nAChR to chronic cardiovascular disease. In the current study we investigated the effect of the α7nAChR agonist AZ6983 on the progression of atherosclerosis and potential effect in human cells. **Methods and results:** Atherosclerosis-prone ApoE-/- mice on a high fat diet were treated with AZ6983 or vehicle admixed in food for 12 weeks, or infused by osmotic minipumps (50 μmol/kg/day) for 8 weeks. Quantification of atherosclerotic lesions in thoracic aorta and aortic root revealed that AZ6983 reduced atherosclerosis by 49% after 12 weeks of oral treatment and 37% after 8 weeks of minipump treatment. AZ6983 treatment did not influence cholesterol levels, nor CD3+ T cells or CD68+ macrophages in the aortic root, quantified by immunostaining. There was no difference in immune cell markers in the aorta, investigated by real-time PCR. In addition, AZ6983 treatment in LPS-stimulated human and murine whole blood, reduced pro-inflammatory cytokine TNF in a dose-dependent manner *in vitro* ($p<0.01$ and $p<0.001$, respecttively). **Conclusion:** Treatment with AZ6983 decreases the progression of atherosclerosis in ApoE-/- mice, and additionally inhibits pro-inflammatory cytokine production in human whole blood, thus suggesting that stimulation of the α7nAChR may be a novel anti-inflammatory treatment strategy for atherosclerosis. Funding sources: The Swedish Heart and Lung Association, The Åke Wiberg Foundation and The Wilhelm and Martina Lundgren Foundation.

4.18
THE ANTI-INFLAMMATORY ROLE OF α7NACHR AND α4β2NACHR IN HUMAN BLOOD SAMPLES

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Background and Aim: Autonomic nerve signaling via the alpha 7 nicotinic acetylcholine receptor (α7nAChR) is known to modulate inflammation. We have previously shown that the α7nAChR is expressed by $CD68^+$ macrophages and $CD3^+$ T cells in human atherosclerotic plaques, however, little is known about the functional role of the α7nAChR in human cells. The aim of the current study is to investigate the functional role of the α7nAChR in human cells and to compare the effects of the α7nAChR with other nicotinic receptors expressed by human blood cells. **Methods:** To evaluate the role of the α7nAChR in patients with atherosclerosis, blood samples from patients and healthy donors were stimulated with lipopolysaccharide (LPS, 10ng/ml) and subsequently treated with nicotine (1000 μmol/L). After 4 h of incubition, samples were centrifuged and supernatant was saved for analysis. Further, to characterize the specific effects of α7nAChR stimulation, human blood samples from healthy donors were incubated with LPS and treated with different concentrations (0.001-1000 μmol/L) of selective agonists for the a7nAChR (GTS-21 or PHA 568487) and α4β2nAChR (RJR-2403 or A85380), as described above. In part of the experiments, blocking with alpha-bungarotoxin, a specific α7nAChR antagonist, was included. From the collected supernatant, TNF-a production was measured using ELISA. **Results:** In both blood samples

from patients with atherosclerosis and healthy donors, nicotine reduced the TNF-a, IL-6 and IL1- β levels. When further characterizing the α7nAChR-specific effects in whole blood stimulations from healthy donors, both α7nAChR agonists decreased TNF-a cytokine production. After blocking the a7nAChR with alpha-bungarotoxin, only the PHA 568487 agonist lost its ability to decrease the TNF-a levels. Further, both α4β2nAChR agonists were also capable of reducing the TNF-a production in the highest concentration. **Conclusions:** This study shows that stimulation of nicotinic acetylcholine receptors decreases pro-inflammatory TNF-a production, partly via the α7nAChR. However, alpha-bungarotoxin did not abolish the TNF-a response when using GTS-21, raising the possibility of GTS-21 acting on other nicotinic receptors. Interestingly, α4β2nAChR agonists also reduced the TNF-a response. Thus, both α7nAChR and α4β2nAChR may have an immune modulating role. Funding source: Swedish Heart-Lung foundation.

4.19
THE ASSOCIATION BETWEEN AUTONOMIC DYSFUNCTION, INFLAMMATION AND PREVALENT CARDIOVASCULAR DISEASE IN SUBJECTS WITH ATHEROSCLEROSIS

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Background: Cardiovascular diseases, including atherosclerosis, are associated with autonomic dysfunction. However, the mechanism linking autonomic dysfunction to cardiovascular disease is not known. We investigated the role of inflammation as the link between autonomic dysfunction and presence of atherosclerosis, and how it is associated with prevalent cardiovascular disease, defined as stroke or myocardial infarction. **Methods:** We examined 226 subjects with carotid atherosclerosis, determined by duplex ultrasound imaging. Autonomic function was assessed by measuring heart rate variability (HRV SDNN) and baroreflex sensitivity (BRS Slope), and inflammatory markers were determined (white blood cell count; WBCC, C-reactive protein; CRP). Risk factors including age, gender, smoking, body mass index (BMI), hypertension, lipids and diabetes were assessed. **Results:** Subjects with prevalent CVDs showed a larger carotid plaque area ($p<0.01$), higher WBCC ($p<0.05$), and reduced BRS Slope ($p<0.01$) compared to subjects with subclinical atherosclerosis. Further, BRS Slope was inversely associated with carotid plaque area ($p<0.05$). In a mediator analysis, adjusting for WBCC, the association between BRS Slope and carotid plaque area was decreased with 29%, indicating that WBCC may link autonomic tone to atherosclerosis. In subsequent analysis, BRS Slope and HRV SDNN were both inversely associated with WBCC ($p<0.01$), but not with CRP. Further, WBCC was independently associated with carotid plaque area ($p<0.05$), after adjusting and controlling for confounders including gender, age, BMI, medication and cardiovascular risk factors. **Conclusions:** Reduced BRS is associated with increased carotid plaque area, at least partly mediated via WBCC. This suggests that autonomic dysfunction is associated with the extent of atherosclerosis, possibly mediated via increased systemic inflammation. Funding source: The Swedish Heart-Lung Foundation.

4.20
ACTIVATION OF NONCLASSICAL MONOCYTES DURING ATHEROGENESIS IS MEDIATED BY THE SCAVENGER RECEPTOR CD36

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Nonclassical monocytes in both humans and mice have previously been implicated in endothelial repair and vascular homeostasis. Our lab has investigated the role of nonclassical monocytes in atherosclerosis and we have found that the deletion of these cells leads to an increase in plaque formation and presence of M1 macrophages. A major function of nonclassical monocytes is to "patrol" the blood vessel wall to survey the vasculature for pathogens, damaged endothelium, and possibly DAMPS (damage associated molecular patterns). We found that ApoE$^{-/-}$ and C57Bl6/J mice fed an atherogenic diet showed increased nonclassical monocyte patrolling activity compared to chow fed mice, and that this increase in activity occurs rapidly within 24 hours of diet change. When various forms of low density lipoproteins were in-

jected in vivo, oxidized LDL specifically induced nonclassical monocyte localization to the endothelial wall and was preferentially taken up via the scavenger receptor CD36. By live imaging of blood vessels, we found that nonclassical monocytes from CD36$^{-/-}$ mice showed reduced uptake of oxLDL and failed to patrol. Nonclassical monocytes produced CCL5 in response to atherogenic diet feeding. In summary, nonclassical monocytes are activated early in atherogenesis to patrol the vasculature in response to oxidized LDL signaling and this is dependent on CD36. Thus, targeting this monocyte subset is important therapeutically for reducing the inflammation associated with cardiovascular disease.

4.21
TARGETED UPLC-MS/MS ANALYSIS OF OXYLIPINS: FROM PROFILING TO QUANTIFICATION FOR TRANSLATIONAL RESEARCH STUDIES

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Oxylipins are signaling molecules that play a role in the regulation of many key biological processes, most notably inflammation. Here, we describe targeted, quantitative ultra performance liquid chromatography - tandem mass spectrometry (UPLC-MS/MS) assays for the analysis of two subsets of oxylipins. These subsets represent downstream products from particular precursor lipids and metabolic pathways. Developing separate analytical methods for subsets of analytes is a more specific approach, giving superior results overall when compared to generic, low-fidelity profiling methods that compromise on performance. Matrix samples were prepared using mixed mode OASIS MAX µElution SPE and analyzed using an ACQUITY UPLC I-Class system interfaced to a Xevo TQS Micro tandem quadrupole mass spectrometer. We demonstrate these methods to be sensitive, selective, linear and precise and therefore suitable for use in quantitative translational research studies.

4.22
LY6CHI MONOCYTIC CLEARANCE OF DYING CARDIOMYOCYTES BY CD36 ACTIVATES NR4A1 AND IS REQUIRED FOR TRANSITION TO REPARATIVE INFLAMMATION FOLLOWING MYOCARDIAL INFARCTION

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Rationale. Efficient phagocytosis of dying cells is a prerequisite for inflammation resolution and tissue repair after myocardial infarction (MI). MI triggers the early recruitment of Ly6CHI monocytes, which subsequently require *Nr4a1* to differentiate into Ly6CLO macrophages. Separately, Ly6CLO macrophages require MERTK for the clearance of myocardial apoptotic cells. The phagocytic contributions of Ly6CHI monocyte receptors to heart repair, which are first to encounter the necrotic myocardium, are unclear. **Objective.** To test causal requirements and consequences of Ly6CHI phagocytic receptors during cardiac wound repair and macrophage differentiation post MI. **Results.** Scavenger receptor CD36 was found to be heightened on Ly6cHI cardiac monocytes post experimental MI. Bone marrow-derived *Cd36* was required for both necrotic cardiomyocyte clearance and containment of early infarct size, specifically before the emergence of Ly6CLO cardiac macrophages. Decreased engulfment was directly associated with suppressed monocytic Nr4a1 expression and an increased ratio of Ly6CHI:Ly6CLO myeloid cells. Chromatin immunoprecipitation revealed direct *Cd36*-dependent binding of NR4A1 to MERTK, sequentially linking the two critical phagocytic receptors. Deficiency of both *Cd36* and *Mertk* (i.e., double knockout mice) led to catastrophic defects in phagocytic clearance and an increased incidence of myocardial rupture. **Conclusions**. Taken together, these data identify a CD36 phagocytosis-initiated *Nr4a1-Mertk* axis during inflammatory repair of the infarcted heart. **Funding.** National Heart, Lung, and Blood Institute.

4.23
INTERACTION OF IL-6 AND TNF-α CONTRIBUTES TO ENDOTHELIAL DYSFUNCTION IN TYPE 2 DIABETIC MICE HEART

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Background/Aims: Although inflammatory cytokines, such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), are known as the important contributors to endo-

thelial dysfunction in obesity and type 2 diabetes (T2D), their interactions established in coronary endothelial function remain unclear. The aim of this study is to elucidate the interactive effect of TNF-α and IL-6 in impairment of the coronary endothelial function in T2D. ***Methods***: We used wild type (WT), diabetic db/db, db/db null for TNF (db^{TNF-}/db^{TNF-}), and db/db mice treated with neutralizing antibody to IL-6 (anti-IL-6). Endothelium-dependent (acetylcholine, ACh, or flow) vasodilation of isolated and pressurized coronary arterioles was measured by a concentration-dependent manner using ACh and sodium nitroprusside (SNP). Real-time PCR, Western blot, and immunofluorescence staining were utilized for mechanistic studies. ***Results:*** Dilation to ACh or flow was blunted in db/db mice and db^{TNF-}/db^{TNF-} + IL-6, whereas it was greater in db^{TNF-}/db^{TNF-} and db/db mice treated with anti-IL-6 compared with db/db mice. Immunofluorescence staining illustrated co-localization of IL-6 with endothelial cells. Protein and mRNA expression of IL-6 and superoxide (O_2^-) production were higher in db/db mice but they were reduced by anti-IL-6 treatment. Increased protein and mRNA expression of TNF-α in db/db mice were reduced by anti-IL-6 treatment and genetic depletion of TNF-α in db/db mice reduced the mRNA and protein expression of IL-6 suggesting that TNF-α and IL-6 regulated their expression in parallel and interacted with each other. Superoxide dismutase 2 (SOD2) and phosphorylated eNOS (p-eNOS) were lower in db/db mice coronary arterioles and they were restored in db/db + Anti-IL-6 and db^{TNF-}/db^{TNF-} mice. ***Conclusion:*** Our findings suggest that the endothelial dysfunction of coronary circulation in T2D mice results from the interactive effects of TNF-α and IL-6 through enhanced oxidative stress and reduced phosphorylation of eNOS.

4.24
INFILTRATING MACROPHAGES PROMOTE ADVERSE CARDIAC REMODELING DURING ACUTE AND CHRONIC PRESSURE-OVERLOAD

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Recent evidence indicates that tissue-resident and infiltrating macrophages (Mϕ) have different reparative and pro-inflammatory capacities in the heart. During the early stages of cardiac pressure overload (PO), cardiac Mϕ may serve as important drivers of both subsequent left ventricular (LV) hypertrophy and the activation of T-cells that contribute importantly to the transition to heart failure. We hypothesized that cardiac Mϕ are expanded during the early stages of PO, primarily due to an increase in infiltrating Mϕ, and contribute to long-term T-cell activation in the chronically pressure-overloaded heart. Cardiac Mϕ were characterized by flow cytometry as $MerTK^+CD64^+F480^+MHCII^+$ cells, with infiltrating Mϕ populations identified as expressing C-C chemokine receptor 2 (CCR2). One week after the imposition of PO by transverse aortic constriction (TAC), C57Bl/6 mice exhibited significant ($p < 0.05$) increases in cardiac macrophages (3.15 ± 0.64 vs 1.09 ± 0.24%), specifically in the infiltrating $CCR2^+$ population (2.8 ± 0.6 vs 0.9 ± 0.2%). TAC mice also exhibited higher circulating levels of $Gr1^-CD11b^+Ly6C^{hi}$ monocytes (2.6 ± 0.5 vs 1.3 ± 0.2%) that are recruited to tissue via CCR2. During acute PO, treatment with a CCR2 antagonist in TAC mice significantly blunted LV hypertrophy when compared to vehicle-treated TAC animals (heart weight/tibia length 8.9 ± 0.3 vs 10.53 ± 0.3 mg/mm; LV Wall thickness 0.70 ± 0.01 vs 0.77 ± 0.02 mm) and suppressed cardiac infiltration of macrophages. To determine activation of the adaptive immune response during acute PO, we analyzed the T-cell population in mediastinal heart draining lymph nodes (MLNs). Expansion of $CD3^+$ T-cells in MLNs was blunted in CCR2 antagonist-treated TAC mice when compared to vehicle controls (1.3×10^5 vs 2.6×10^5 cells/MLN). To determine the effects of acute CCR2 blockade on chronic LV remodeling, we treated mice with a CCR2 antagonist for 3 days during the first week of PO, and assessed cardiac function 4 weeks post-TAC. Chronic TAC mice treated with the antagonist had significantly improved ejection fraction when compared to vehicle-treated TAC mice (42.0 ± 2.5 vs 33.5 ± 2.9%). We conclude that infiltrating $CCR2^+$ Mϕ are major contributors to PO-induced adverse cardiac remodeling and help drive systemic T-cell activation. Moreover, these results suggest targeting infiltrating Mϕ during PO hypertrophy may have therapeutic potential for delaying or preventing the transition to heart failure.

4.25
THE ALARMIN COMPLEX S100A8/9 AS A BIOIMAGING AND THERAPEUTIC TARGET AFTER MYOCARDIAL INFARCTION

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Objective: Myocardial infarction (MI) is an ischemic injury of the myocardium. Excessive myocardial inflammation after infarction increases the risk of recurrent coro-

nary events and heart failure. One of the biological mediators involved in post-MI inflammatory processes, the alarmin complex S100A8/9, is potently upregulated after MI in the heart tissue mainly due to a strong influx of neutrophils an subsequently monocytes. In turn, S100A8/9 amplifies local inflammation by supporting further myeloid cell recruitment. We hypothesize that i*n-vivo* visualization of this protein complex could provide important information on MI severity. Additionally, we aimed to test whether S100A8/A9 inhibition with the specific blocker ABR-238901 can reduce inflammation and lead to improved physiological performance of the heart. **Methods and Results:** We used coronary artery ligation to induce MI in C57Bl/6 WT mice. Using PET/CT and radiolabelled Cu^{64} anti-S100A9 Ab, the complex was visualized and strongly upregulated after MI compared to the sham operated animals. The S100A8/A9 positive signal was present in the walls of the left ventricle, reflecting the infarcted areas and the size of the respective injury. The PET/CT signal was still detectable in *ex-vivo* PET/CT imaged hearts, collected 8 days after MI. In a parallel experiment mice undergoing permanent coronary artery ligation were treated with 30mg/Kg ABR-238901administered i.p. daily, and the performance of the heart was monitored using ultrasound. Two days after MI, ABR-238901-treated mice showed significantly higher ejection fraction (EF=55%) in a comparison with animals treated with buffer (EF=43%). Moreover, EF was higher both in the AMI and in the sham groups treated with ABR, suggesting beneficial effects of the compound. **Conclusion:** *In-vivo* PET/CT bioimaging of S100A8/9 after MI provides useful information about the size and the location of the injury. Our data indicates that S100A8/A9 blockage using ABR-238901 has beneficial effects on reducing myocardial damage, reflected by improved ejection fraction in the immediate period after an MI.

4.26
OXIDATIVE AND NITROSATIVE STRESS IN VALVE TISSUE OF PATIENTS WITH AORTIC VALVE DISEASE

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Background: Calcific aortic valve disease is becoming the most frequent heart illness in aged people of developed countries. Although several lines of evidence suggest that the mechanism of aortic valve degeneration is similar to that of atherosclerosis, the true mechanism remains undefined. Here we have investigated the levels of oxidative and nitrosative stress in aortic valve tissue of patient with aortic valve disease. **Methods**: The explanted aortic valve leaflets of 56 consecutive patients aged between 18-90 years, with a LV ejection fraction>30%, with normal sinus rhythm and without coronary artery disease were taken at the time of surgery for aortic valve replacement. Those suffering from renal or liver disease were excluded. The leaflets were put in physiological serum and kept at 4˚C, then the calcium deposits were removed and the remaining tissues were kept at -80˚C until analysis. The tissues were homogenated and processed to determine the content of nitric oxide (Nitrate/NitriteColorimetricAssayKit), superoxide anion (by luminescence using coelenterezine) and nitrotyrosine (OxiSelect Nitrotyrosine ELISA Kit). Patients were divided by their valve pathology: (i) calcific aortic valve stenosis (transvalvular gradient≥30mmHg, n=30), (ii) non-calcific aortic valve regurgitation (regurgitation≥grade II, n=14) and (iii) mix aortic valve disease (transvalvular≥30mmHg and regurgitation≥grade II, n=12). Characteristic demographics and cardiovascular risk factors were recorded and the results expressed as median [IQR]. **Results**: Tissue valve contents of superoxide anion were significantly elevated by the increase with age ($p<0.05$). The valve tissues from patients with aortic valve stenosis and mixed disease (calcific valves) had also greater content in nitrotyrosine than in patients with pure aortic valve regurgitation (non calcific valves) (67.96[52.25-86.94] and 89.18[45.86-110.88] vs. 42.96[22.05-63.79], respectively; $p<0.05$). The presence of cardiovascular risk factors did not result in significant changes. **Conclusion**: Oxidative and nitrosative stress is an important component of calcific aortic valve disease and may play an important role in the degeneration of the valve, particularly in elderly people. The results support the use of antioxidants as a useful therapeutic strategy to stop or reduce the development of calcific aortic valve disease. **Support:**VHIR, Hospital Vall d'HebrónBarcelona,Proposal Nº: 603049 TRANSLINK Project.

4.27
INHIBITION OF INFLAMMATORY SERINE PROTEASES POTENTIATES B CELL DEVELOPMENT AND FUNCTION IN DIABETIC CARDIOMYOPATHY

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Objective: To investigate the novel role of serine proteases on B cell development and growth and their reciprocal humoral response which may contribute to pathological cardiac remodeling during the development of diabetic cardiomyopathy (DCM). **Methods and Results:** 10 weeks old male mice with deletion of dipeptidyl peptidase-I (DPPI), an enzyme involved in maturation of inflammatory serine proteases (ISPs), and wild type (WT) mice were injected with streptozotocin (50 mg/kg for 5 days, intraperitonealy) and studied after 4 and 8 weeks after induction of diabetes mellitus (DM). Mice elevated glucose levels (>300mg/dL) were classified as DM. Induction of diabetes in WT mice resulted in an increase in DPPI expression and activity in hearts compared to shams that correlated with an increase in the activity of several ISPs (cathepsin G (CG), elastase, chymase). DPPI deletion had no effect on hyperglycemic state of the animals. However, DPPI deficiency reduced ISP activity, increased B cell expression and their anti-inflammatory cytokines as well as toll like receptors, attenuated cell apoptosis and improved cardiac function after STZ treatment compared to WT mice. To delineate the mechanisms involved, we found that diabetes increases IL-21 which mediates B cell apoptosis through down-regulation of BCl2 and BCL-xL. Interestingly, DPPI deletion significantly decreased IL-21 expression and up-regulated the two anti-apoptotic members of the BCl2 family compared to WT-diabetic mice. **Conclusions:** These findings emphasize the implication of inflammatory serine proteases in B cell development and growth alteration, myocyte loss and adverse cardiac remodeling observed in DCM. These insights might have useful implications on future studies utilizing neutrophil serine protease blockers to treat human DCM.

4.28
INFLAMMATORY SERINE PROTEASE INHIBITION ATTENUATES MYOCYTE APOPTOSIS AND CARDIAC DYSFUNCTION VIA INTERVENTION OF PPAR GAMMA-INDUCED LIPOTOXICITY AND INFLAMMATION IN HIGH FAT DIET-INDUCED DIABETIC CARDIOMYOPATHY

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Background: A diet consisting of high percentage of fat induces lipotoxicity, leading to type II diabetes (T2D) and diabetic cardiomyopathy (DCM). Although high fat diet (HFD)-mediated lipotoxicity has been previously associated with hyperglycemia and myocyte apoptosis, the underlying mechanisms leading to myocardial death is not well understood. An emerging concept is that neutrophil inflammatory serine proteases (ISPs) released as a result of lipotoxicity might be linked to the alteration of nuclear receptor peroxisome proliferator-activated receptor gamma (PPAR-γ) signaling. Altered PPAR-γ signaling then induces the maladaptive structural and functional cardiac pathology during the development of DCM via its downstream mitochondrial enzymes 3-hydroxy3-methylglutaryl-CoA synthase2 (HMGCS2) and pyruvate dehydrogenase kinase 4 (PDK4). This study aimed to investigate the effect of ISP on PPAR-γ activation and its downstream effects on HFD-induced inflammation and apoptosis that occur in DCM. **Methods and Results:** Hearts from wild type (WT) diabetic mice (16 weeks HFD-fed) showed increased neutrophil infiltration, Cathepsin G activity and myocyte death. Immunoblot analysis and qPCR showed an increase in the expression of PPAR-γ. The latter was associated with an enhancement in mitochondrial lipogenic and ketogenic enzymes, such as HMGCS2 and PDK4, indicating an increase in lipid metabolism and potential lipotoxicity. qPCR also revealed that PPAR-γ activation is involved in mTOR/AKT signaling down-regulation and myocyte apoptosis in HFD-diabetic mice. Interestingly, ISP deletion *in vivo* using DiPeptidyl Peptidase I (DPPI) KO mice (mice that lacking major ISPs), diminished the expression of PPAR-γ, HMGCS2 and PDK4. Additionally, apoptotic markers (Bax, Bcl2, Bad and cleaved Caspase3) were also significantly attenuated in HFD-fed DPPI-KO group in comparison to WT-HFD. Gene expressions of mTOR and AKT were increased in DPPI-KO group compared to WT group fed with HFD. Interestingly DPPI deletion did not affect high levels of lipids in diabetic mice, suggesting that ISPs independently affect myocyte survival. **Conclusions:** The findings from our study emphasize that DPPI inactivation enhances myocyte survival and improve cardiac remodeling through neutralization of major ISPs that upregulate the PPAR-γ mediated inflammatory cascade. Our data highlight DPPI and PPAR-γ as potential therapeutic targets for the preservation of cardiac structure and function in DCM.

4.29
PORPHYROMONAS GINGIVALIS LIPOPOLYSACCARIDE ENHANCES THE RUPTURE OF EXPERIMENTAL CEREBRAL ANEURYSMS BY INDUCING VASCULAR INFLAMMATION

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Introduction. Subarachnoid hemorrhage (SAH) is a catastrophic event that results in high morbidity and a poor prognosis. To prevent SAH its pathogenesis must be understood. Bacterial infections have been suggested to have a role in the etiology of cardiovascular disease. Recently the evidence that dental infection could be a part of pathophysiology in intracranial aneurysm was reported. We hypothesized that periodontal pathogen enhances the formation and rupture of experimental cerebral aneurysms and studied it with our aneurysmal rat model. **Methods.** Ten-week-old female Sprague-Dawley rats were subjected to aneurysmal induction surgery, which consisted of estrogen deficiency, renal hypertension and hemodynamic stress. Two weeks later, they were divided into 2 groups; rats treated with Porphyromonas gingivalis lipopolysaccharide (LPS) or a saline control (VC). The VC- (n=17) or LPS (n=13) rats received intraperitoneal injection weekly. We observed death or abnormal behavior in the course of 90 days after last operation and confirmed ruptured aneurysm. **Results.** 7 LPS rats (54%) and 6 VC rats (35%) suffered aneurysmal rupture during 90 days. Especially LPS rats (38%) had higher incidence of aneurysmal rupture than VC rats (6%) within 60 days. LPS promoted experimental aneurysmal rupture in our rat model. The administration of LPS increased the plasma level of IL-1β and MMP9 and gene expression of TLR2, pro-inflammatory cytokines and matrix degradation molecules in vascular wall in rat aneurysm model. **Conclusion.** Our findings suggest that Porphyromonas gingivalis enhances the rupture of intracranial aneurysms through promotion of inflammatory response.

4.30
LOSS OF LYMPHOCYTE ADAPTOR PROTEIN LNK PROMOTES AORTIC DISSECTION

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Objective: Although inflammation likely contributes to aortic dissection and rupture, the role of the lymphocyte adaptor protein LNK in aortic dissection is unknown. Genome-wide association studies revealed that a polymorphism in the gene *SH2B3* encoding LNK is associated with several autoimmune and cardiovascular diseases in humans. LNK is an intracellular adaptor protein that negatively regulates cytokine signaling. We studied the effects of LNK deletion on the development of aortic dissection and rupture in angiotensin II infused mice. **Approach and Results:** WT and $LNK^{-/-}$ mice were infused with angiotensin II (Ang II; 1000 ng/kg/min) for 14 days in the absence or presence of an initial intraperitoneal injection of saline equal to 10% of the body weight. LNK deficiency predisposes to aortic dissection (50% survival in $LNK^{-/-}$ mice versus 100% in WT mice) and saline injection prior to Ang II infusion, accelerates the rate of aortic dissection or rupture compared to WT mice. Histological studies showed an increase in macrophages and T lymphocytes in the aorta of $LNK^{-/-}$ mice compared to WT mice. Aortic expression of the Th1 markers (T-bet and IFNγ) and MMP9 were increased, while the expression of the procollagens type I and III were reduced in $LNK^{-/-}$ mice. Furthermore, analyses by flow cytometry of the aortic draining lymph nodes reveals that, in the absence of LNK, the early phase of the disease (3 days after Ang II infusion) is characterized by an increase in the number of IFNγ producing $CD4^+$ and $CD8^+$ T cells compared to WT mice. In addition, when naïve splenic $CD4^+$ T cells isolated from $LNK^{-/-}$ mice are cultured for 72 hours in the presence of Th1 polarizing cytokines, they express more T-bet and produce more IFNγ compared to naïve $CD4^+$ T cells isolated from WT mice. In contrast, Th17 polarization is not affected by loss of LNK. **Conclusion:** Deletion of LNK enhances Th1 polarization and promotes adverse aortic remodeling leading to the development of aortic dissection and rupture.

4.31
EFFECT OF INFLAMMATORY SERINE PROTEASE INHIBITION ON CXCR4 EXPRESSION DURING THE DEVELOPMENT OF DIABETIC CARDIOMYOPATHY

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Objective: Diabetic cardiomyopathy (DCM) is characterized by ventricular remodeling and dysfunction independent of atherosclerosis, coronary heart disease, or hypertension. DCM is characterized by intramyocardial inflammation, cardiomyocytes apoptosis and cardiac fibrosis. The molecular mechanism that links inflammation to DCM is incompletely understood. An emerging concept is that inflammation might be linked to alteration in CXC chemokine receptor (CXCR) 4 signaling, thus leading to the maladaptive cardiac structural and functional consequences of DCM. Therefore we hypothesized that increased inflammatory proteases may contribute to CXCR4 downregulation that lead to the pathophysiological changes observed in the heart during the development of diabetic cardiomyopathy. **Methods and Results:** 10 weeks old mice with deletion of dipeptidyl peptidase I (DPPI), an enzyme involved in the maturation of major ISPs, and wild type (WT) mice controls were injected with streptozotocin (50 mg/kg for 5 days, intraperitoneally) and studied 4 weeks after induction of diabetes. Induction of diabetes in WT mice resulted in an increase in DPPI expression and activity in hearts compared to shams that correlated with an increase in the activity of several ISPs (cathepsin G (CG), elastase, chymase). DPPI deletion had no effect on hyperglycemic state of the animals. However, DPPI deficiency reduced ISPs activity, attenuated CXCR4 signaling downregulation, reduced cell apoptosis and improved cardiac function after STZ treatment compared to WT mice. DPPI inhibition also enhanced insulin sensitivity and increased glucose uptake after STZ treatment compared to WT mice, suggesting that ISPs negatively regulate CXCR4 signaling. To delineate the mechanisms involved, we found that treatment of cardiomyocytes with neutrophil-derived protease CG impaired CXCR4 signaling and led to myocyte apoptosis. Incubation of myocytes in hyperglycemic conditions enhanced CXCR4 signaling deterioration and myocyte apoptosis induced by CG. **Conclusion**: These data suggest that ISPs are important regulators of CXCR4 signaling and myocyte death and may play an important role in regulating glucose metabolism during the development of DCM.

4.32
NEUTROPHIL FUNCTIONAL CHANGES ARE ASSOCIATED WITH METABOLIC ALTERATIONS FOLLOWING TEN WEEKS OF HIGH INTENSITY INTERVAL EXERCISE TRAINING IN MEN AND WOMEN WITH PREDIABETES

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Intro: With advanced age, dysfunctional neutrophils are a hallmark of immunesenescence and increased risk of infection. Further, neutrophil dysfunction is associated with increased systemic inflammation and insulin resistance. Improving neutrophil function in those at risk for diabetes may reduce disease risk. We have recently observed that neutrophil function in older individuals without disease is maintained better by engaging in regular physical activity. The aim of this study was to determine if neutrophil functions were improved by structured exercise in those at risk of diabetes. **Method**: Ten older (71±5 years) sedentary participants with prediabetes (HbA1c = 6.1±0.3%) completed 10-weeks of a high intensity interval training program with ten ≥60 second intervals of low intensity (50-60% heart rate reserve (HRR)) separated with bouts of high intensity intervals (80-90% HRR). Neutrophils were analyzed for chemotaxis, phagocytosis, oxidative killing and cellular metabolism before and after training. **Results**: Significant improvements were observed for relative peak VO_2 (p=0.004) and absolute peak VO_2 (p=0.01) with no change in body mass index (p=0.196) or body fat percent (p=0.574). Using live video-microscopy, compared to baseline, post training neutrophils migrated towards the chemokine IL-8 at a similar speed (p=0.857) but with improved velocity (p=0.01) and accuracy (p=0.008). Phagocytosis of opsonized *E.Coli* was enhanced on a per-cell basis (p=0.007), while oxidative killing capacity was slightly increased (p=0.05). Improved functions were not related to changes in the cell surface receptors CXCR2 or TLR-4 (both p>0.05). Using Seahorse technology, mitochondrial function was improved following training. Specifically, proton leak across the mitochondrial membrane was reduced (p<0.05), while basal and maximal respiration and ATP production were all improved (all p<0.05). **Conclusion:** Ten weeks of interval training in older individuals at risk for diabetes improved aerobic fitness with no change in body composition. Improved neutrophil functions were associated with mitochondrial improvements on a per cell basis. Ten weeks of interval training in persons with prediabetes alters neutrophil metabolism which is associated with improved primary functions, potentially reducing the risk of infection and inflammatory insult which can enhance diabetes risk. *This work was funded by an EU Marie Curie Outgoing Fellowship Grant (PIOF-GA-2013-629981).*

4.33
HIGH SALT ACTIVATES DENDRITIC CELLS TO PROMOTE HYPERTENSION

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Inflammation and dietary salt intake have been implicated in the pathogenesis of hypertension. We have previously shown that NADPH oxidase-dependent formation of immunogenic isoketal-protein adducts in dendritic cells (DCs) contributes to the development of experimental hypertension. Recently, it has been shown that salt can accumulate in the interstitial space and promote inflammation. In the current study, we tested the hypothesis that exposure to high salt activates DCs, leading to an NADPH oxidase-dependent production of immunogenic isoketals and the promotion of hypertension. To test this hypothesis, mouse splenic DCs were cultured in media with either normal salt (NS, 150 mM NaCl) or high salt (HS, 190 mM NaCl) for 24 hours. Exposure to HS caused a 2-fold increase in superoxide production in DCs compared to NS. This was NADPH oxidase-dependent since incubition with the gp91ds-tat peptide prevented the increase. Western blots revealed that all NADPH oxidase subunits (p47phox, p22phox, gp91phox and p67phox) were increased by exposure of cells to HS. Moreover, phosphorylation of the regulatory subunit, p47phox and assembly of NADPH oxidase were increased by HS. Exposure to HS also led to an increase in the activation markers CD80 and CD86, and doubled the number of DCs containing isoketal-protein adducts. These effects were prevented by inhibition of the serum-and-glucocorticoid-inducible kinase-1 (SGK1). In additional experiments, mice received adoptive transfer of splenic DCs that were cultured for 24 hours in either NS (n = 6); HS (n = 5); HS plus an SGK1 inhibitor (n = 5) or HS plus an isoketal scavenger (n=6). Mice were implanted with radiotelemeters to measure mean arterial pressure (MAP). Following recovery and two days of baseline, subcutaneous osmotic minipumps were implanted for administration of a generally sub-pressor dose of angiotensin II (140 ng/kg/min). This caused no increase in blood pressure in mice that received NS DCs, whereas MAP increased significantly (14 ± 4 mmHg, $p<0.05$) by one week of angiotensinogen II infusion in mice that received salt-activated DCs. The pro-hypertensive effect of salt on DCs was completely blocked by inhibition of SGK1 and scavenging of isoketals during salt exposure. These data indicate that DCs can be activated by exposure to a HS environment which can exist *in vivo*, and that this likely involves increased NADPH oxidase-mediated superoxide production and formation of isoketal-protein adducts. Moreover, high salt exposure can cause DCs to become pro-hypertensive. These studies define a new pathway linking salt to immune activation and identify a previously undefined role of SGK1 in this process.

5.0: BASIC ASPECTS OF T CELLS

5.3
T CELL TRAFFICKING AND MEMORY

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As cells of the adaptive immune system, T cells are characterized by exquisite antigen specificity. Naïve CD4 and CD8 T cells primarily circulate between blood, lymphatics and secondary lymphoid organs. In contrast, activated T cells acquire multiple properties including enhanced effector functions (e.g. cytokine production, cytolytic function) and altered trafficking patterns, to enter into non-lymphoid tissues where they are both influenced by the local microenvironment and further shape inflammation and immunity. Notably, a subset of activated T cells further achieves long-term survival to become memory T cells, which have altered sensitivity to stimulation and the capacity to respond to inflammatory cytokines in a bystander manner. In this talk, I will discuss how T cell trafficking and memory T cell responses can profoundly alter the local composition of lymphocytes in a tissue, in turn influencing the baseline responsiveness of these tissues to subsequent inflammatory cues and tissue damage. (Support: AHA #13SDG14510023 and CCFA #311295). Reference: Clambey, E.T., E.N. McNamee, J.A. Westrich, L.E. Glover, E.L. Campbell, P. Jedlicka, E.F. de Zoeten, J. Cambier, K.R. Stenmark, S.P. Colgan and H.K. Eltzschig. "Hypoxia-inducible factor-1 alpha dependent induction of FoxP3 drives regulatory T cell abundance and function during inflammatory hypoxia." *Proc Natl Acad Sci USA*. 2012 Oct 9;109(41):E2784-93.

5.4
T REGULATORY CELL (TREG) POLARIZATION AND FUNCTION

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Antigen specific T cell activation, as well as antigen specific B cell activation, are the hallmarks of, and define, an adaptive, specific immune response. As such, effective T cell and B cell activation are centrally important for maintaining protection from infectious pathogens and injury. However, maintaining homeostasis, while avoiding self-reactivity, and dampening the anti-pathogen response

once the pathogen has been contained, necessitate tight regulation of antigen specific T cell functions. This type of control is accomplished, at least in part, via the activation and expansion of T regulatory cells (Tregs). The purpose of this overview is to review Tregs in terms of development (including naïve versus memory Tregs), function, subset characterization, and distribution. We will characterize their contributions to homeostatic control and maintenance of self-tolerance, and we will also explore the capacity of some Treg, or their absence, to contribute to pathologies ranging from cancer to autoimmune disease, respectively. We will review existing information on the phenotype, function, cytokines, and growth factors involved in Treg activation and function, discussing the best-characterized of the Treg sub-populations, including natural Tregs (nTreg) and inducible costimulator (ICOS)(+) Tregs, inducible/adaptive Tregs (iTreg), interleukin (IL)-10-producing type 1 Tregs (Tr1 cells), CD8(+) Tregs, and IL-17-producing Tregs. These cells share some common features, including expression of the transcription factor Foxp3 (except for Tr1 cells), and secretion of inhibitory cytokines, including IL-10 and/or TGF-β. As to their distribution, Foxp3(+)CD4(+) regulatory T cells circulate throughout lymphoid organs and appear to have counterparts that also function to suppress immune responses in parenchymal tissues. In this overview, we will explore proposed basic and molecular mechanisms by which the various Treg subsets exert their immune suppressive activities and we will discuss the implications for both the beneficial and deleterious consequences of their activation and expansion. References: Abbas AK, Benoist C, Bluestone JA, Campbell DJ, Ghosh S, Hori S, Jiang S, Kuchroo. VK, Mathis D, Roncarolo MG, Rudensky A, Sakaguchi S, Shevach EM, Vignali. DAA*, Ziegler SF. Regulatory T cells: Recommendations to simplify the nomenclature. *Nature Immunology* 2013 14 (4):307-8. Ito T, Hanabuchi S, Wang YH, Park WR, Arima K, Bover L, Qin FX, Gilliet M, Liu YJ: Two functional subsets of FOXP3+ regulatory T cells in human thymus and periphery. Immunity. 2008, 28 (6): 870-880. 10.1016/j.immuni.2008.03.018. Noble A, Giorgini A, Leggat JA: Cytokine-induced IL-10-secreting CD8 T cells represent a phenotypically distinct suppressor T-cell lineage. Blood. 2006, 107 (11): 4475-4483. 10.1182/blood-2005-10-3994. Voo KS, Wang YH, Santori FR, Boggiano C, Wang YH, Arima K, Bover L, Hanabuchi S, Khalili J, Marinova E, Zheng B, Littman DR, Liu YJ: Identification of IL-17-producing FOXP3+ regulatory T cells in humans. Proc Natl Acad Sci U S A. 2009, 106 (12): 4793-4798. 10.1073/pnas.0900408106. Wu K, Bi Y, Sun K, Wang C: IL-10-producing type 1 regulatory T cells and allergy. Cell Mol Immunol. 2007, 4 (4): 269-275.

6.0: BASIC ASPECTS OF B CELLS

6.1
B CELL SUBSETS AND IMMUNE RESPONSES

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B cells are lymphocytes that function in adaptive humoral immunity primarily by producing antigen-specific antibody. Throughout the years, the diversity of B cell function and phenotype has grown and it is now known that, in addition to antibody production, B cells can present antigen to T cells, secrete cytokines and down regulate inflammation. Under normal circumstances, B cells protect against bacterial and viral infections by producing antibodies that inhibit infection or clear pathogens. Some of these responses are dependent on T cell help (T-dependent) whereas responses to polysaccharides or other pathogen components are T-independent. In general, the function and phenotype of B cells is associated by where they reside in secondary lymphoid tissues and by their environmental cues. In patients with atherosclerosis, antibody titers against oxidized-forms of LDL (oxLDL) have been associated with increased plaque burden. In animal models atherosclerosis, B cells have been demonstrated to be protective and harmful. Results of these studies appear to be dependent on experimental system, and specific type of B cell examined. Here, we offer an overview of basic aspects of B cell subsets and responses in health and disease, briefly discussing their relevance to cardiovascular disease.

6.2
GENETIC RISK FACTORS AND LOSS OF B CELL ANERGY IN DEVELOPMENT OF AUTOIMMUNITY

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Stochastic use of germline variable region genes inevitably leads to generation of B cells that are reactive with self-antigens and must be censored to prevent autoimmunity. Studies using primarily reductionist immunoglobulin transgenic models have defined mechanisms of autoreactive B cell silencing in the mouse but little is known regarding mechanisms operative in autoreactive B cell silencing in the human. Autoreactive B cells have been found in multiple phenotypically distinct B cell compart-

ments in human peripheral blood but the functional status of these cells is largely unknown. To examine and compare the *in vivo* lifestyles of autoreactive and naïve antigen-specific human B cells we devised nanoparticle adsorbent-based approaches to identify and isolate antigen reactive B cells, and study their status. This presentation will focus on results of analysis of cells reactive with insulin (autoantigen) and tetanus toxoid (exogenous antigen). Although it is well accepted that T cells function as the pancreatic beta cell executioners in Type 1 Diabetes (T1D), it is unclear what events precede and precipitate their activity in disease. A role for B cells is supported by the success of B cell depleting therapy (Rituximab). Results to be discussed demonstrate that in healthy subjects B cells bearing high affinity insulin-binding antigen receptors reside in the periphery but only in the, so-called B_{ND}, anergic compartment. Antigen receptors expressed by these cells are germline in sequence, and exhibit structural features consistent with autoreactivity and, interestingly, are polyreactive. Like anergic murine B cells, these cells are characterized by chronic activation of negatively regulatory BCR signaling pathways. With T1D development the lifestyle of these cells changes. B cells leave the B_{ND} compartment in a proportion of healthy but "at risk" first-degree relatives, and in all prediabetic and new onset diabetic patients. This loss appears correlated with localization of insulin-binding B cells in pancreatic islets. The apparent re-localization of high affinity insulin-reactive B cells in first-degree relatives is associated with particular disease risk allele genotypes. Results demonstrate escape of autoreactive human B cells from central tolerance, and residency in the B_{ND} compartment where they are silenced by anergy enforced by PI3-k-targeted regulatory signaling mechanisms. They are consistent with an essential role for loss of anergy in islet-reactive B cells early in T1D development. A role predicated at least in part on risk allele genotype. Funding sources: NIH R01 DK096492, R21 AI124488, R01 AI124487.

6.3
B CELLS AND CARDIOVASCULAR DISEASE

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The role of B cells, and antibodies in particular, in the pathogenesis of CVD is perhaps most widely recognized in rheumatic carditis, in which antibodies against streptococci cross-react with myocardial proteins, triggering inflammatory heart disease. However, the pathogenic role of antibodies in other forms of inflammatory cardiomyopathy is much less clear, with emerging data pointing to CD4 T cells as the critical mediators of myocarditis in both animal models and humans. Here, we will discuss how B cells also contribute to this disease process, independent of autoantibody (AAb) production, and how cardiac AAb can serve as biomarkers of heart disease. Starting with the unexpected discovery that humanized DQ8+NOD mice – a model of type 1 diabetes (T1D) – develop premature death due to spontaneous myocarditis, we found that the earliest antigenic target of IgG AAb responses, α-myosin heavy chain (α-MyHC), was also the major target of pathogenic CD4 T cells. We further observed that the spreading of humoral immune responses - from α- to β-MyHC, and then to other cardiac antigens - paralleled disease progression from myocarditis to dilated cardiomyopathy. With this knowledge, and modeling on the success of "biochemical" islet AAb assays for detecting individuals at high risk for developing T1D, we developed a panel of similarly designed radiobinding assays to measure cardiac AAb in humans. We then showed that patients with acute myocarditis tested positive for multiple cardiac AAb, especially α- and β-MyHC, similar to DQ8+NOD myocarditis mice. In another body of studies, we discovered experimental and clinical evidence for a chronic post-myocardial infarction autoimmune syndrome in T1D and furthermore, that cardiac AAb profiling can differentiate between ischemic heart disease in T1D and T2D. We hypothesize that unrecognized autoimmune heart disease contributes to the poor CVD outcomes in T1D and that cardiac AAb profiling might eventually be used to identify the patients most likely to benefit from antigen-specific therapeutic approaches. Support: NIH R01DK103609. References: Lv H et al.: Impaired thymic tolerance to α-myosin directs autoimmunity to the heart in mice and humans. *The Journal of Clinical Investigation,* 2011; 121:1561–1573. doi:10.1172/JCI44583. Gottumukkala R et al.: Myocardial infarction triggers chronic cardiac autoimmunity in type 1 diabetes. *Science Translational Medicine*, 2012; 4:138ra80. doi:10.1126/scitranslmed.3003551.

7.0: BASIC ASPECTS OF VASCULAR CELLS WITH IMMUNE FUNCTION

7.1
$CD4^+$ T CELLS IN ATHEROSCLEROSIS

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Autoreactive $CD4^+$ T cells drive atherosclerosis. They respond to auto-antigens including ApoB-100, the main protein in low-density lipoprotein (LDL), and accumulate

in atherosclerotic lesions. However, atherosclerosis-specific $CD4^+$ T cells have not been identified *in vivo*. In particular, their origin and differentiation in the plaque is unknown. Using a new multimer of the MHC-II-restricted ApoB-100 auto-epitope P6 (P6:I-A^b) that enabled detection of antigen-specific $CD4^+$ T cells, we identified a novel population of auto-reactive $CD4^+$ T cells in healthy, young C57BL/6 mice that is predominately differentiated into athero-protective T-regulatory cells (T_{regs}). This repertoire of T cells is detectable in the thymus at birth and expands within 28 days in peripheral lymph nodes. In atherosclerotic *Apoe*$^{-/-}$ mice, P6:I-A^{b+} $CD4^+$ T cells are initially expanded, but their numbers decline during the natural course of disease. The fraction of these ApoB-100-specific T cells in peripheral lymph nodes correlates negatively to the extent of advanced atherosclerotic lesions, suggesting that these $FoxP3^+$ auto-reactive T cells are protective. In contrast, we observed a distinct phenotype of T cells residing in the atherosclerotic plaque. In Apoe$^{-/-}$ mice with mature atherosclerotic lesions after 5 months of high fat diet, most aortic T cells express a unique combination of transcription factors ($FoxP3^+$ T-bet^+). These cells are exclusively found in the aorta and para-aortic lymph nodes. In contrast to classical T_{regs} in peripheral lymphoid tissue they do not suppress T-cell proliferation *in vitro* and are less potent in inhibiting cytokine secretion. Transcriptomic analysis shows that this subset of aortic T cells is closer to effector T cells than to T_{regs}. They secrete interferon-γ, interleukin-10, and tumor necrosis factor and increase atherosclerosis after an adoptive transfer. These findings indicate that atherosclerosis-specific $CD4^+$ T cells are protective $FoxP3^+$ T_{regs} before initiation of disease in lymphoid tissue, but acquire a pro-atherogenic phenotype in the aorta in later stages of disease. Indeed, in a T_{reg} lineage tracking mouse, T_{regs} specifically recognizing the ApoB-100 epitope P6 were more likely to lose FoxP3. In the plaque, many T cells can be identified as such previous T_{regs}, suggesting that atherosclerosis-specific $CD4^+$ T cells that homed to the aorta lost their regulatory capacity and instead further exacerbate atherosclerosis. Funding source: This work was supported by National Institutes of Health grants R01 HL115232 and HL121697 to K. Ley and from the Deutsche Forschungsgemeinschaft to D. Wolf.

7.2
IL 1β PROMOTES ATHERO-PROTECTIVE CHANGES IN LATE STAGE ATHEROSCLEROTIC LESIONS

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Despite decades of research, little is known regarding mechanisms and factors that control the pathogenesis of late stage atherosclerotic lesions and in particular the probability of plaque rupture that may lead to possible myocardial infarction or stroke. The widely accepted dogma is that plaques having a thin fibrous cap and a paucity of Acta2+ smooth muscle cells (SMC) relative to CD68+ macrophages have an increased risk of rupture. However, our recent rigorous lineage tracing studies have provided compelling evidence that SMC play a much more important role than has generally been appreciated with >80% of SMC derived cells failing to be detected using conventional SMC marker genes, and >30% of these expressing markers of macrophages or mesenchymal stem cells (Shankman et al. NMED 2015). Of even greater significance, we have shown the SMC specific conditional knockout of the pluripotency genes Klf4 or Oct4 (Cherepanova et al. NMED 2016) results in dramatic changes in lesion pathogenesis. However, contrary to dogma, *we showed that they can play either a beneficial or detrimental role depending on the nature of their phenotypic transitions*. Thus, there is a critical need to identify factors and mechanisms that promote beneficial changes in SMC phenotype including the ability to invest into the fibrous cap, to produce extracellular matrix (ECM). The pro-inflammatory Interleukin-1β (IL-1β) is believed to be a major driver of atherosclerotic plaque progression and neutralization of this key regulator of systemic inflammation is tested in an ongoing large-scale clinical trial (CANTOS). However, previous preclinical studies investigating the impact of genetic or pharmacological inhibition of the IL-1β pathway have provided ambiguous evidence of a beneficial effect of IL-1β neutralization. In this talk, I will present results of our recent studies in which we show that treatment of our SMC lineage tracing mice with the Novartis IL1β-neutralizing antibody after the establishment of advanced atherosclerosis (18 weeks of Western Diet feeding) resulted in multiple changes consistent with reduced plaque stability including marked reductions in the number of SMC-derived $eYFP^+$ cells

within the fibrous cap, enrichment in macrophages, as well as an inhibition of the beneficial outward remodeling of the brachiocephalic artery. Studies provide evidence that IL1β plays an unexpected protective role in maintenance of plaque stability in late stage atherosclerosis. Supported by NIH Grants R01 HL121008, HL087867, and HL057353 to GKO.

8.0: CV DISEASE IN INFLAMMATORY AND AUTOIMMUNE DISEASE

8.1
UTILIZING A HUMAN MODEL OF DISEASE TO STUDY INFLAMMATORY ATHEROGENESIS

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A growing body of evidence suggests that inflammation predisposes to atherosclerosis and cardiovascular events. The mechanisms by which inflammation predisposes to atherogenesis are not fully understood, and remain a topic of intense investigation with two large-scale, ongoing interventional studies (CIRT, CANTOS) testing the hypothesis that reduction in inflammation will reduce future CV events. While these trials will be highly informative to understand if reduction in inflammation by inflammasome or T-cell inhibition translates into clinical outcomes, they will not augment mechanistic understanding of *how* inflammation promotes atherogenesis. Our trans-disciplinary research program at the Intramural Research Program at the National Heart Lung and Blood Institute utilizes a human inflammatory skin disease, psoriasis, to understand pathways involved in modulation of inflammatory cardiometabolic dysfunction. The talk will focus on our deep phenotyping efforts utilizing imaging, cellular phenotyping and lipoprotein characterization in our ongoing cohort study (NCT01778569) which will serve as a growing resource to understand inflammatory atherogenesis.

8.3
AUTOIMMUNITY: A CASUAL FACTOR IN THE PATHOGENESIS OF HYPERTENSION

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Autoimmune diseases including systemic lupus erythematosus (SLE) and rheumatoid arthritis are associated with a marked risk for developing hypertension. In addition, several studies report that essential hypertension is associated with increased circulating levels of autoantibodies suggesting that a loss of immune tolerance and autoimmunity may be an important underlying mechanism in the pathogenesis of hypertension. Previous work from our laboratory demonstrated a role for renal inflammation, oxidative stress, and impaired renal hemodynamic function in SLE associated hypertension. More recently, we demonstrated that preventing autoimmunity in an experimental mouse model of SLE prevented the development of hypertension. Current work in the laboratory is related to testing the hypothesis that humoral immunity, specifically autoantibodies, mechanistically promote the development of hypertension during SLE. (*BX002604-01A2, P20GM104357, P01HL051971).*

8.4
DEFECTIVE INFLAMMATION RESOLUTION IN ATHEROSCLEROSIS: MECHANISMS AND THERAPEUTIC OPPORTUNITIES

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The inflammatory response to acute and reversible infection or tissue damage, mediated by PAMPs and DAMPs, respectively, triggers an essential resolution response that curtails inflammation and restores tissue homeostasis. This resolution response is mediated by a panoply of lipid and protein mediators that activate specific cell surface receptors and signal transduction pathways to trigger resolution endpoints. However, when the inflammatory response is indolent and persistent, the resolution phase is often not engaged, leading to a chronic, low-grade inflammatory response that causes clinically serious tissue damage. We have proposed that the maladaptive inflammatory response in atherosclerosis is, in essence, a classic example of failed resolution. The initial inflammatory response is triggered by the subendothelial retention of apoB-containing lipoproteins, and resolution fails because this trigger is not only persistent but is actually amplified. This scenario leads to a vicious cycle of failed resolution, tissue damage-mediated DAMP formation, and amplified DAMP-mediated inflammation. This talk will review the features of atherosclerosis progression that are consistent with failed resolution; discuss in detail how clearance of apoptotic cells (efferocytosis) is part of the resolution program and how it goes awry in advanced atherosclerosis; reveal new molecular-cellular mechanisms of how certain resolution mediators and apoptotic cells activate resolution signaling pathways in macrophages, a key inflammatory cell type in atherosclerosis; and discuss and show data supporting the contention that inflammation resolution mediator therapy may be an ideal way to pre-

vent atherosclerosis progression in a manner that would not compromise in host defense.

9.0: INFLAMMATION AND HYPERTENSION DURING PREGNANCY AND GENDER DIFFERENCES

9.1
ADAPTIVE IMMUNITY AND HYPERTENSION DURING PREGNANCY

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Preeclampsia (PE), new onset hypertension during pregnancy, is associated with a proinflammatory profile characterized by elevated CD4+ T cells, inflammatory cytokines and autoantibodies to the angiotensin II Type I receptor (AT1-AA), compared to women with normal pregnancy (NP). Previous studies from the Zenclussen lab showed that adoptive transfer of Th1-like splenocytes increased MAP, fetal rejection and inflammatory cytokines when transferred to NP mice, but the mechanisms causing the PE symptoms were not further investigated. We hypothesize that $CD4^+$ T cells play an important role to cause much of the pathophysiology associated with PE and illicit a memory response for production of the AT1-AA, where by it is produced chronically following a PE pregnancy. To determine if $CD4^+$ T cells isolated from PE patients cause PE symptoms during pregnancy compared to those collected from NP women, $CD4^+$ T cells were isolated from placentas of both PE and NP women using magnetic anti-CD4 antibodies, cultured and injected intraperitoneally into pregnant, nude-athymic rats on day 12 of gestation. On day 18, carotid catheters were implanted and on day 19 MAP was measured and blood and tissues were collected. MAP was 125±3 mmHg in rats injected with NP T cells and MAP significantly increased to 140±9 mmHg in rats injected with PE T cells. Circulating TNF-α, IL-6 and sFlt-1 were increased in rats receiving PE vs NP $CD4^+$ T cells (TNF-α- PE=142.4 pg/mL, NP=79.4 pg/mL; IL-6 - PE=311.6 pg/mL, NP=277.8 pg/mL; sFlt-1-PE=90.7 pg/mL, NP=58.2 pg/mL). ET-1 expression in the renal cortices was increased 4.5 fold in rats exposed to PE $CD4^+$ T cells vs those exposed to NP $CD4^+$ T cells ($p=0.06$). Importantly, AT_1-AA was significantly increased to 19.8 ± 0.9 bpm in recipients of PE T cells (n=6) compared to 1.3 ± 0.9 bpm seen in recipients of NP T cells (n=6, *$p<0.05$). To determine a role for the CD4+ T cell mediated AT1-AA to cause the PE symptoms, the study was repeated in the presence of AT1 receptor blocker, Losartan, given via drinking water. Blood pressure, cytokines and ET-1 were all decreased with AT1R blockade. These data suggest an important role for $CD4^+$ T cells, a player of adaptive long term immunological memory responses, to cause AT1-AA, inflammation, renal abnormalities and thus hypertension during pregnancy. These data also indicate that CD4+ T cells could contribute to cardiovascular disease later in life among previously PE women.

9.2
INNATE IMMUNITY AND GESTATIONAL HYPERTENSION

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Gestational hypertension (GH), a disease that affects ~10% of all pregnancies, may result from over-activation of the maternal immune system and is characterized by excessive innate immune cell activation, inflammation, and injury at the vascular, renal, and placental level. We and others have demonstrated that activation of the innate immune system as well as the adaptive immune system in female animals is able to produce a pregnancy-dependent phenotype that mimics GH in women. Ligands that activate the pattern recognition receptors on innate immune cells are able to induce GH in animals, and animals lacking these receptors are resistant to the development of GH. Therefore, exploring how the innate immune system is involved in the development of GH and how it signals to the adaptive immune system is important, and how we can specifically prevent this will be discussed in this symposium.

10.0: POSTER SESSION II

10.1
THE IMPACT OF EXERCISE INTENSITY AND CYTOMEGALOVIRUS INFECTION ON IMMUNE CELL REDEPLOYMENT BY EXERCISE

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Exercise results in a well characterized leukocytosis that is directly related to the intensity of the exercise, with

greater cell mobilizations observed as exercise intensity increases. High differentiated immune cells with effector functions are more exercise responsive. Factors driving the accumulation of the high differentiated cells, including infection with cytomegalovirus (CMV), significantly augment T cell mobilization by exercise. In contrast, CMV does not appear to impact the monocyte response to exercise, despite these cells being targeted by CMV infection. It is not known whether the impact of CMV on the exercise-induced redistribution of monocyte and T cell subsets varies with exercise intensity. Exercise is also reported to alter T cell cytokine expression. The relationship of these changes to exercise intensity has not yet been examined. **Objectives:** 1) Determine if CMV affects T cell and monocyte redeployment across multiple exercise intensities, and 2) determine cellular expression of type I and type II cytokines at varying exercise intensities. **Methods:** 17 cyclists completed three 30min cycling trials at -5, +5, and +15% of blood lactate threshold (LT). Monocyte and CD4+ and CD8+ T cell subsets (defined by CD14/CD16 and CD27/CD28 expression) and cytokine-expressing T cells (IL-2, IFN-γ, TNF-α, IL-6, IL-4, IL-10) present in the blood pre-, post-, and 1h post-exercise were characterized by flow cytometry. Effects of CMV and intensity on exercise-induced changes in cell subsets were analyzed with linear mixed models. Research conformed to the Declaration of Helsinki. **Results:** All exercise intensities increased monocyte and T cell subsets present in the blood post-exercise. CMV+ participants had greater numbers of high differentiated $CD4^+$ and $CD8^+$ T cells compared to CMV- at all time points, independently of exercise intensity. The ingress of T cells into the blood post exercise, and their egress 1h post exercise, increased as exercise intensity increased, independently of CMV. CMV did not impact the monocyte response to exercise at any intensity. Exercise-induced increases in T cell expression of type I cytokines increased with intensity. **Conclusions:** The mobilization of monocyte and T cell subsets by exercise was directly related to exercise intensity, as was exercise-induced changes in T cell cytokine expression. Although CMV+ mobilized more high differentiated T cells than CMV-, this occurred above and below LT. Therefore the augmenting effect of CMV on T cell mobilization is independent of exercise intensity.

10.2
OBESITY AND CIRCULATING INFLAMMATION-RELATED $_{MICRO}$RNAS

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microRNAs (miRs) are short single stranded noncoding RNAs that are involved in the regulation of a number of physiological and pathological processes. miRs down regulate target gene expression post-transcriptionally by degrading messenger RNA and/or by blocking translation. It is now recognized that miRs play a key role in regulating inflammation, vascular health and, in-turn, cardiovascular disease (CVD). For example, altered expression of specific miRs such as, miR-34a and miR-146a, have been linked with heightened vascular inflammation. Obesity is associated with increased inflammatory burden that is thought to contribute to greater CVD risk. The mechanisms underlying obesity-related inflammatory stress are not fully understood. It is currently unknown whether inflammation-related miRs are dysregulated with obesity. Accordingly, the aim of this study was to determine the influence obesity, independent of other risk factors, on circulating expression of miR-34a, miR -126, miR -146a and miR -150. To address this aim 30 sedentary, middle-aged (47-64 years) adults were studied: 15 normal weight (10M/5F; BMI: 22.9±0.5 kg/m^2) and 15 obese (10M/5F; 31.4±0.5 kg/m^2). All subjects were non-smokers, normotensive and free of overt cardiometabolic disease. Circulating expression of miRs was determined in plasma using standard RT-PCR techniques with miR primers of interest. Expression was normalized to exogenous C. *elegans* miR-39 and reported as relative expression in arbitrary units (AU). Circulating expression of miR-34a (0.024±0.015 vs 0.01±0.006 AU) was significantly higher (~240%); whereas miR-126 (0.182±0.037 vs 0. 315±0.048 AU) and miR-146a (0.031±0.007 vs 0.057±0.010 AU) expression was significantly lower (~40%) in the obese compared with normal weight adults. There was no significant group difference in miR-150 (0.088±0.120 vs 0.133±0.025 AU). Higher expression of miR-34a and lower expression of both miR-126 and miR-146a is consistent with a proinflammatory phenotype. In summary, these data suggest that obesity, independent of other cardiometabolic risk factors, adversely influences key inflammation-related miRs. Dysregulation of miRs may contribute mechanistically to the heightened inflammatory state associated with obesity.

10.3
CHARACTERIZATION OF FACTORS THAT PREDISPOSE TO THE METABOLIC SYNDROME AND ADIPOSE TISSUE INFLAM-

MATION IN AGED MALE AND FEMALE MICE

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The metabolic syndrome is defined by the presence of multiple risk factors for diabetes, heart disease and stroke. Younger and middle aged women are more likely to have increased triglycerides, low HDL cholesterol, an epidemiology that shifts after the age of 65, when risk factors become similar between the sexes. Stroke incidence is disproportionally climbing in middle aged women, and rates increase after menopause. Elderly women are more likely to die after a stroke compared to men, and have worse functional recovery. In pre-clinical studies, young female mice have less histological injury after stroke compared to males (27% vs 43%), while in middle aged and aged cohorts this scenario was reversed, and greater damage was seen in females (43% vs 23%) [1]. The underlying mechanism for the "switch" to an ischemia sensitive phenotype in middle-aged females is not known. We hypothesize that this change might originate from baseline differences in middle-aged mice that involve features of the metabolic syndrome. The current study characterized multiple factors involved in the metabolic syndrome in aged male and female mice including assessment of obesity, lipids and glucose. We also evaluated sex differences in white adipose tissue inflammation by flow cytometry. Body weights as well as abdominal white adipose tissue, liver and spleen weights were compared in 14-15 month old C57BL/6 mice (n=10 per group). Glucose and insulin tolerance tests were performed after 6 h fasting. Glucose, triglycerides and high density lipoprotein (HDL) and low density lipoprotein (LDL) concentrations were determined in plasma from fasted animals. Aged males had higher body weights than females (49±1g vs 35±1g), as well as higher liver (2.45±0.3g vs 1.43±0.07g) and abdominal white adipose tissue weights (2.19±0.15g vs 1.43±0.07), while no difference were seen in spleen weights. Plasma glucose concentrations were not significantly different in males and females (132±8 mg/dL vs 137±4 mg/dL). Male mice were less sensitive to insulin compared to females but only marginal differences in glucose sensitivity were seen. Lipid profiling showed a trend to higher triglycerides in females (1.28±0.19 mM vs 1.79±0.42), no difference in LDL cholesterol while lower concentrations of HDL were seen in females (0.84±0.07 μg/μl vs 1.35±0.06 μg/μl). Flow cytometry on adipocyte-derived immune cells indicate that aged males and females have different subsets of T lymphocytes. Sex differences in adipose tissue may disproportionally affect females and contribute to an enhanced pro-inflammatory milieu in aging females. References: 1. Manwani, B., et al. Exp Neurol, 2013. 249: p. 120-31.

10.4
EFFECTS OF AN 8-WEEK PALEO DIETARY INTERVENTION ON INFLAMMATORY CYTOKINES

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Introduction: The Paleolithic (Paleo) diet is promoted as a potential remedy to the supposed Western diet-induced "life-style" diseases of modern times. The purpose of this study was to determine the feasibility of transitioning from a traditional Western diet to a Paleo diet, and measure the effects of this diet on bio-markers of inflammation. **Methods:** Eight healthy individuals (age: 32.4 ± 12.0 years; body mass: 86.9 ± 34.3 kg; BMI: 29.9 ± 6.1 kg/m^2) were recruited for this study. Prior to the 8-week Paleo diet intervention all participants were consuming a typical Western diet. Dietary counseling and Paleo menus were provided to assist in this dietary modification. Pre- and post-intervention testing included resting serum concentrations of IL-4, IL-10, IFN-γ, and TNF-α. Data are reported as percent change from pre-intervention (%Δ). **Results:** Resting IL-10 increased 35.5% and resting IFN-γ increased 66.7% after the intervention. TNF-α and IL-4 changed minimally. Relationships between pre-intervention IL-4 and IL-10, IFN-γ, and TNF-α were found (p=0.02, R^2=0.88; p=0.02, R^2=0.92; $p\leq$0.001, R^2=0.97, respectively). Additionally, pre-intervention IL-10 and IFN-γ were found to have a relationship with pre-intervention TNF-α (p=0.03, R^2=0.85; p=0.01, R^2=0.95, respecttively). Similarly, post-intervention relationships between IL-4 and IL-10, IFN-γ, and TNF-α were found (p=0.08, R^2=0.88; $p\leq$*0.01,* R^2=0.97; p=≤0.001, R^2=0.96, respectively). Further, post-intervention IL-10 and IFN-γ were found to have a relationship with post-intervention TNF-α (p=0.07, R^2=0.78; p=0.01, R^2=0.95). **Conclusion:** The findings confirm that it is feasible to counsel research participants to transition from a typical Western diet to an ad-libitum Paleo diet when provided dietary counseling and a Paleo specific menu but not food. Furthermore, the Paleo diet induced weight loss (previously reported data) despite the ad-libitum intake and dietary counseling centric to individual levels of satiety not weight reduction. As a result of this preliminary feasibility project a research

study with a larger participant pool and longer duration is possible, and further examination of inflammation and the Paleo diet is warranted.

10.5
PERIVASCULAR ADIPOSE TISSUE MACROPHAGES ARE RESPONSIBLE FOR ENDOTHELIAL DYSFUNCTION IN THE OBESE MICROVASCULATURE

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The function of perivascular adipose tissue (PVAT) as an anti-contractile mediator is lost during obesity. Obesity results in inflammation of the PVAT that is paralleled by decreased levels of the vasorelaxant signaling molecule hydrogen sulfide (H_2S). The current objective was to assess the role of proinflammatory macrophages in determining vascular [H_2S] and defining how this impinged on vasodilation. Mesenteric resistance arterioles isolated from 20 week-old lean and obese mice were loaded with the fluorescent H_2S indicator SF7-AM, mounted in a pressure myography chamber, pressurized and imaged confocally to assess [H_2S] in the smooth muscle and endothelial layers. Steady-state [H_2S] was lower in both the smooth muscle and endothelium of obese compared with lean mouse vessels which correlated with impaired endothelial-dependent vasodilation. To assess the role of macrophages in driving these phenotypes, vessels from lean and obese mice were cocultured overnight in the presence of macrophages purified by immunomagnetic separation, or macrophage-conditioned media, from either lean or obese mice. In vessels from lean mice, [H_2S] in both smooth muscle and endothelium was decreased by exposure to macrophages from obese but not lean mice, which was observed in parallel with impaired vasodilation. These effects were mediated by low molecular weight species (<3.5 kDa) and dependent on macrophage inducible nitric oxide synthase (iNOS) activity. Collectively, these data support a model in which iNOS activity of PVAT-resident proinflammatory macrophages promotes microvascular endothelial dysfunction by reducing the bioavailability of H2S, and suggest that vascular H_2S depletion underpins the loss of PVAT anti-contractile function in obesity.

10.6
CULLIN3 REGULATES ENDOTHELIAL FUNCTION BY MODULATING $_E$NOS ACTIVITY

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Pseudohypoaldosteronism type II (PHAII) patients expressing dominant negative cullin3 mutations exhibit increased renal NaCl reabsorption and develop hyperkalaemia, metabolic acidosis and hypertension. It is unclear whether loss of cullin3 function in extra-renal tissues contributes to the hypertensive phenotype. In the vasculature, endothelial Nrf2 stability is tightly regulated by cullin3-based E3 ubiquitin ligase, which binds Nrf2 via the redox-sensitive adaptor Kelch-like ECH-associated protein 1(Keap1). In the present study, we found that 24-hour treatment with a pan cullin inhibitor MLN4924 (1 μM) caused a 3-fold increase of Nrf2 protein in mouse lung endothelial cells (MLECs), while tert-butyl hydroperoxide (tBHP, 240 μM) had no effect on Nrf2 level. However, both MLN4924 and tBHP triggered time-dependent accumulation of Nrf2 in the nuclei, which peaked at 40 minutes following treatment. As a result, both treatments induced marked upregulation of antioxidant genes including NAD(P)H quinone oxidoreductase 1, heme oxygenase 1, glutamate cysteine ligase (rate-limiting enzyme in glutathione synthesis), and catalase both in MLECs and primary mouse aortic endothelial cells (MAECs). Of note, MLN4924 and tBHP suppressed the expression of Nox1 and Nox4, but both markedly increased intracellular superoxide as determined by dihydroethidium (DHE) staining in cultured MLECs. This was associated with decreases in intracellular and extracellular nitric oxide (NO) bioavailability with no changes in endothelial nitric oxide synthase (eNOS) messenger RNA. To determine whether this redox imbalance was due to changes in eNOS protein or its activation, western blot analysis was performed. Inhibition of total cullin activity caused a 25% reduction in total eNOS and a 75% reduction in phosphorylated eNOS, while tBHP caused a 50% reduction in phosphorylated eNOS with no effect on total eNOS, suggesting that decreased eNOS activity contributed to the oxidative stress induced by these agents. To further verify whether cullin3 regulates endothelial redox machinery, MAECs isolated from cullin3$^{flox/flox}$ mice were treated with adenovirus expressing Cre recombinase for 48 hours to induce deletion of cullin3. This lead to an 80% reduction in cullin3 protein and significant decreases in total eNOS, phosphorylated eNOS and NO bioavailability, supporting a role of cullin3 in regulating eNOS expression and activity. These data imply that deletion or suppression of cullin3 in arterial endothelial cells may dampen endothelium-dependent vascular relaxation and contribute to the blood pres-

sure elevation observed in PHAII patients with global loss of cullin3 function. Although cullin3 also negatively regulates Nrf2-mediated antioxidant responses in vascular endothelial cells, this likely occurs as a compensatory mechanism.

10.7
RENAL AND SPLENIC CYTOKINES ARE ALTERED IN EARLY LIFE STRESSED (ELS) ADULT MALE RATS

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Substantial evidence indicates that inflammation contributes to the development of cardiovascular disease (CVD). Epidemiological observations showed that ELS is associated with elevated circulating markers of inflammation in adults. Thus, we hypothesized that ELS alters the inflammatory state of adult male rats. Maternal separation (MatSep), a rodent model of ELS, involves separating rat pups from the dam for 3hrs/day from postnatal day 2 (PD2) to PD14. Normally reared littermate rats were used as controls. Inflammation status was monitored by determinations of immune cell numbers and types as well as renal and splenic cytokine expression. Our results showed that renal mononuclear cells and T-cell numbers were similar in adults control and MatSep rats (control vs. MatSep; renal PBMC: 52.9 ± 9.2 vs. 56.5 ± 8.3 x 10^6 cells/kidney; renal T cells: 7.3 ± 1.2 vs. 6.7 ± 1.2 x 10^6 cells/kidney; $p < 0.05$). Renal expression of interferon gamma (IFNγ), interleukin 6 (IL-6), and interleukin 4 (IL-4) were similar in control and MatSep rats, while renal interleukin 1 beta (IL-1β) was significantly increased in MatSep rats compared to control (control: 4.4 ± 0.5 pg/mg protein, MatSep: 7.9 ± 1.0 pg/mg protein; $p<0.0186$). This increased IL-1β immunoreactivity was localized in the renal distal tubular epithelial cells of MatSep rats compared to control rats. In contrast, splenic IL-4 and IFNγ were significantly decreased in MatSep rats compared to control rats (IL-4; control: 0.7 ± 0.05 pg/mg protein, MatSep: 0.5 ± 0.08 pg/mg protein; IFNγ; control: 9.2 ± 0.5 pg/mg protein, MatSep: 7.6 ± 0.3 pg/mg protein $p<0.05$). No difference in IL-1β and IL-6 levels was observed in the spleen of control and MatSep rats. In response to an immune challenge with low dose LPS (2mg/kg), no differences were observed in renal and splenic IL-1β, IL-6, IL-4, IFNγ and TNFα protein expression (control LPS vs. MatSep LPS). These findings indicate that MatSep induces differential cytokine expression in the kidneys and spleen of control and MatSep rats at baseline. Future studies will determine: (1) which distal tubular epithelial cells have elevated IL-1β expression, (2) the effects of renal tubular IL-1β expression on renal transport function, and (3) whether IL-1β promotes renal dysfunction. (Funded by APS Porter Fellowship to IEO, T32DK007545 to CDM, K99/R00*HL111354* to ASL, F32HL116145 to DHH, and P01HL69999 to JSP).

10.8
γ/δ T CELLS MEDIATE ANGIOTENSIN II-INDUCED HYPERTENSION AND VASCULAR INJURY

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Objective: Both innate antigen-presenting cells and the adaptive immune system have been shown to play a role in the development of hypertension. Nevertheless, the T cell subsets involved in the pathophysiology of hypertension remains unclear. There is a small subset of "innate-like" T cells expressing the γ/δ T cell receptor (TCR) rather than the α/β TCR that could play a role bridging between the innate and adaptive immune systems. However, it is unknown whether γ/δ T cells contribute to the development of hypertension. We hypothesized that angiotensin (Ang) II-induced hypertension and vascular injury would be blunted in *Tcrδ*$^{-/-}$ mice, which are devoid of γ/δ T cells. **Design and method:** Thirteen to 15-week old male C57BL/6 wild-type and *Tcrδ*$^{-/-}$ mice were infused or not with Ang II (490 ng/kg/min, SC) for 7 or 14 days. Telemetric blood pressure (BP), mesenteric artery endothelial function and vascular remodeling by pressurized myography, and spleen T cell profile by flow cytometry were evaluated. **Results:** Fourteen days of Ang II increased systolic BP by 42 mmHg ($P<0.01$) in wild-type compared to control mice. The frequency of γ/δ T cells (2.3-fold, $P<0.05$) and activated (CD69+) γ/δ T cells (1.6-fold) was increased after 7 days of Ang II, and 7 days later remained increased or rose further (2.4-fold) in wild-type compared to control mice. Ang II decreased mesenteric artery relaxation responses to acetylcholine by 42% ($P<0.01$) and increased media/lumen by 45% ($P<0.01$) in wild-type mice compared to controls. BP rise and all the above mentioned Ang II effects were abrogated in *Tcrδ*$^{-/-}$

mice. **Conclusion:** These data suggest that γ/δ T cells mediate Ang II-induced BP elevation and vascular injury. γ/δ T cells could be key immune cells bridging innate and adaptive immune responses during the development of hypertension in mouse models and by extension in humans.

10.9
MACROPHAGES REGULATE THE EXPRESSION OF STROMAL CELL-DERIVED FACTOR 1 VIA INDOLEAMINE 2,3-DIOXYGENASE AFTER THE RENAL ACUTE ISCHEMIA-REPERFUSION INJURY

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Objective: To observe the expression of indoleamine 2,3-dioxygenase(IDO) and stromal cell-derived factor 1(SDF-1) in the kidney after ischemic reperfusion injury(IRI) and explore the relationship between IDO, SDF-1 and macrophage by depleting macrophages before the IRI. **Methods:** A total of 32 healthy C57BL/6 male mice were used to establish renal IRI model by clamping unilateral renal pedicle for 60 minutes followed by reperfusion. Kidney tissue samples were collected at indicated time points. Renal histological changes were estimated. The expression of SDF-1 and IDO were determined by immunohistochemistry, ELISA and real-time PCR. In LC group, after the liposomal clodronate was injected intra-peritoneally, the location of CD68 was observed by immunofluorescence. In 1-MT group, IDO was evaluated by immunofluorescence after injecting intraperitoneally with 1-methyl-tryptophan. Renalhistology and protein expression of SDF-1 and IDO were also detected. **Results:** Compared with sham-operated group, classical tubular damage was found in IRI group, accompanied by a lot of inflammatory cells infiltrate. The expression of total renal SDF-1 and IDO peaked on day 1 and decreased to normal levels after two weeks. IDO doesn't express in healthy kidneys, while SDF-1 in healthy kidney was localized at cortex and expand to the other area of the kidney during IRI. Compared with IRI groups elimination of macrophage by injection of liposomal clodronate alleviated renal IRI and down-regulated the expressions of CD68 while up-regulating SDF-1. In 1-MT group, which IDO was depleted by using 1-MT, the expression of CD68 was normal while SDF-1 was up-regulated. **Conclusions:** SDF-1 expression is up-regulated in IRI kidney and is associated with macrophages express with IDO. SDF-1 may play a role in the early phase of acute kidney injury and IDO inhibiter can be a new medicine in therapy of AKI. **Key Words:** Reperfusion injury; Macro-phages; Stromal cell-derived factor 1; Acutekidneyinjure; indoleamine 2,3-dioxygenase.

10.10
MODEL ESTABLISHING AND FACTOR ANALYSIS: AN INSIGHT INTO THE PREDICTION OF ACUTE KIDNEY INJURY AFTER HEART SURGERY IN CHINA

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Objective: To explore the overall prediction of different stages of cardiac surgery associated-acute kidney injury (CSA-AKI) and the changes in renal function after cardiac surgery, try to make further efforts into factor analysis and give ideas to both clinical practice and predictive model establishing in China. **Methods:** Five years (2008-2012, n=2811) of retrospective data were collected in the Division of Thoracic and Cardiovascular Surgery, Nanjing First Hospital. The method of Logistic Regression was applied to establish three preliminary models for predicting different stages of CSA-AKI, and with both Logistic Regression and non-parametric statistical analysis were used to study the influences of different factors on renal function. **Results:** The three models for different stages of CSA-AKI showed differences in both the selected covariates and accuracy of prediction (the AUC of ROC curve for model of stage 1 to stage 3: 0.676 vs. 0.759 vs. 0.813). Covariates selected (with different methods) include male, age, emergency surgery, aortic aneurysm, hypertension, diabetes mellitus, insulin controlled diabetes, preoperative renal function, diagnosed chronic kidney diseases, preoperative hemoglobin, erythrocytes transfusion, duration of mechanical ventilation, ejection fraction (EF), body mass index (BMI) and surgical manner. Wherein, emergency surgery and aortic aneurysm showed statistical significance in non-parametric test ($P < 0.05$) while the result is the opposite in Logistic Regression. The rank sum of factors suggested that abnormal BMI, lower preoperative hemoglobin (<130g/L in male, <120g/L in female) and lower EF (<50%) may increase the risk of postoperative AKI and lower the postoperative renal benefits. **Conclusion:** Both Logistic regression models and scoring model may have some defects in predicting CSA-AKI; the previous classification for some factors may need adjustment; adjustment of BMI, EF and hemoglobin before elective surgery may lower the incidence of CSA-AKI and improve the prognosis.

10.11
EXTRACELLULAR HISTONES IN RELATION TO ORGAN DYSFUNCTION AND INFLAMMATION DURING KIDNEY-LUNG CROSSTALK

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Background: Mortality rates due to kidney-lung crosstalk have remained high despite advances in the management of AKI and ARDS. The inflammatory mediators believed to be responsible for this are yet unidentified. Extracellular histones are known to exacerbate inflammation and tissue injury. Here we investigate whether there is a clinical correlation between blood histone concentrations, kidney function, and lung function during AKI and ARDS. **Methods:** In a prospective cohort, blood samples were collected from 54 patients upon admission to our hospital who were diagnosed with AKI, ARDS, community acquired pneumonia, or a combination of these. Serum histone concentrations were measured by ELISA and plotted against PaO_2/FiO_2, eGFR, neutrophils, and CRP. Pearson correlation test was performed and $p < 0.05$ was considered significant. **Results:** The scattergraphs show histone concentrations against the four parameters. In the overall sample population, histone concentrations were significantly correlated with PaO_2/FiO_2 ($P=0.0017$, $R^2=0.1652$); change of eGFR from baseline ($P<0.0001$, $R^2=0.3107$); neutrophil counts ($P=0.0174$, $R^2=0.1021$) and CRP ($P=0.0004$, $R^2=0.2207$). However, when patients were divided according to diagnosis, there was no significant correlation between these same parameters. **Conclusions:** When viewed in light of the adverse effects of extracellular histones, the correlations reported in this study strongly suggest that extracellular histones may be potential mediators of kidney-lung crosstalk. Additional research is necessary to reveal the mechanisms of their involvement and whether they participate in or are merely a consequence of injury. Further endeavours to understand the pathophysiology of crosstalk will likely lead to improved treatment strategies and outcomes.

10.12
RISK FACTORS OF IN-HOSPITAL MORTALITY IN AKI ASSOCIATED WITH CARDIOPULMONARY BYPASS: A RETROSPECTIVE COHORT STUDY

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Background: CSA-AKI is a common and serious complication which increases morbidity and mortality and remains without effective prevention strategies. Studies in recent years have focused on identifying predictors of CSA-AKI incidence while few have explored the risks contributing to mortality. This study aims to identify the risk factors which can affect mortality and which can potentially be adjusted. **Study Design:** retrospetive cohort study. **Setting & Participants:** Consecutively, 2833 patients underwent first documented cardiac surgery with cardiopulmonary bypass (CPB) between January 2008 and December 2012 in Nanjing first hospital, China. Of these, 843 developed CSA-AKI and were thus included in the present study. **Risk Factors:** All possible perioperative variables were collected and analyzed to identify the factors involved in the mortality related to CSA-AKI. **Outcomes:** In-hospital mortality post CSA-AKI. **Results:** Logistic regression was used to analyze the independent influence of factors on outcomes. Cohort consisted of 2833 patients with a mean age of 55.9 years, 54% men, 36.5% (n=843) with AKI. The overall in-hospital mortality rate was 2.1% (18 of 842). In the multivariate analysis, sex (OR=0.074, 95% CI: 0.015-0.358, P=0.001), DIALYSIS (OR=216.008, 95% CI: 30.873-1511.352, P<0.001), ICU duration (OR=0.896, 95% CI: 0.811-0.990, P=0.031), Red blood cells (RBC) transfused (OR=4.715, 95% CI: 2.257-9.851, P<0.001) were independent predictors of mortality. **Limitation:** single center, retrospective cohort study. **Conclusions:** Multivariable logistic regression analysis of more than 20 variables showed that sex, CPB duration, dialysis and RBC transfused were independent risk factors of in-hospital mortality.

10.13
INFILTRATING MACROPHAGES PROMOTE VASCULAR SYMPATHETIC HYPERINNERVATION DURING EXPERIMENTAL HYPERTENSION

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The immune system has been implicated in the development of hypertension, although the pathophysiological contribution remains unclear. Hypertension is associated with elevated sympathetic innervation and activity that may promote vasoconstriction of blood vessels. Macrophages are known to produce nerve growth factor (NGF), a neurotrophin that induces sympathetic nerve growth, and may be an immune-mediated contribution to vascular

dysfunction and hypertension. The aim of this study was to determine the contribution of macrophages to vascular sympathetic nerve innervation during experimental hypertension. C57Bl/6J male mice were infused with either vehicle or angiotensin (Ang II; 490 ng/kg/day, s.c.) for 14 days. A subset of Ang II-infused mice was randomly assigned to receive liposomes containing PBS or clodronate (50 mg/kg, i.v.) every 3 days to deplete macrophages. To test the effect of lowering blood pressure alone, another group of Ang II-infused mice received hydralazine (25mg/L) via drinking water. Blood pressures were measured using tail-cuff plethysmography. Following treatment, wholemount mesenteric artery immunostaining of nerve bodies (synaptophysin), macrophages (F4/80), and NGF was conducted. Mesenteric vessels were imaged using confocal microscopy where Z-stack images were collected through the vessel. Ang II-infusion significantly increased sympathetic hyperinnervation of mesenteric arteries as shown by a 2-fold increase in synaptophysin staining (38±3% vs 19±2% positive staining; $P<0.001$, n=6). Macrophage infiltration into mesenteric arteries was also elevated in Ang II-infused mouse vessels compared to vehicle (F4/80+ cells: 26±8 vs 12±3; $P<0.01$, n=7), as were NGF-producing macrophages (5±2 vs 2±2 NGF+cells; $P<0.05$, n=6). In clodronate-liposome-treated mice, Ang II-induced pressor responses (135±8 vs 161±3 mmHg; $P<0.01$, n=8), vascular hyperinnervation (% synaptophysin area: 34±1 vs 40±2%; $P<0.01$, n=7) and macrophage infiltration (13±5 vs 20±7 cells; $P<0.05$, n=8) was significantly blunted compared to PBS-liposome-treated mice. Interestingly, despite reducing blood pressure to a similar degree as clodronate-liposome treatment, hydralazine had no effect on vascular hyperinnervation or macrophage infiltration. Ang II-induced hypertension is associated with elevated macrophage infiltration into resistance arteries, which induce local sympathetic hyperinnervation and represents a potential pathophysiological mechanism of immune-mediated hypertension.

10.14
IMPAIRED RIGHT CORONARY VASODILATIVE FUNCTION IN PULMONARY HYPERTENSIVE RAT ASSESSED BY SYNCHROTRON MICROANGIOGRAPHY

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Pulmonary hypertension (PH) causes cardiac hypertrophy in right ventricles (RV), and eventually leads to RV failure due to persistently elevated ventricular afterload. We hypothesized that the mechanical stress of RV associated with increased afterload impairs vasodilative function of the right coronary artery (RCA) in PH. Using microangiography, we compared the vascular function of RCA in the two different models of PH rats. All experiments were conducted in accordance with guidelines for experimental procedures as set forth in the Declaration of Helsinki and the APS "Guiding Principals in the care and Use of Animals." Rats were divided into 4 groups: (1) MCT group (5 wk after subcutaneous injection of monocrotaline, 60 mg/kg), (2) age-matched control group, (3) SuHx group (subcutaneous injection of Su5416, 20 mg/kg, with subsequent exposure to hypoxia (10%) for 3 wk followed by reexposure to normoxia for 5 wk), (4) age-matched control group. Coronary endothelial function was assessed using microangiography with synchrotron radiation in anesthetized rats. Imaging was performed at baseline and during acetylcholine (ACh, 5 μg/kg/min), sodium nitroprusside (SNP, 5 μg/kg/min), blockade of both NO synthases (NOS, L-NAME, 50 mg/kg) and cyclooxygenases (COX, meclofenamate, 3 mg/kg), and post blockade ACh. Since all rat of the SuHx group died immediately after NOS/COX blockade, we examined the effects of EDHF inhibition by charybdotoxin (0.15 μM/hr) and apamin (1.5 μM/hr) instead of NOS/COX blockade. In both PH rats, there was no difference in RCA vessel calibers at baseline compared to each control group. ACh and SNP mediated dilation was reduced in second and third order resistance arteries in both PH rats. MCT group displayed focal stenoses and segmental constrictions during NOS/COX blockade. On the other hand, SuHx group displayed focal stenoses and segmental constrictions during EDHF blockade. In conclusion, endothelium-dependent and independent vasodilative responses were significantly attenuated in the middle and small arteries in both severe PH rats and the presence of abnormal constriction mechanism was revealed in the right coronary circulation of severe PH rat. The observed impaired vasodilative function of RCA in these two PH models suggests that impaired RCA function might have causal relationship with RV failure in the patients with severe PH.

10.15
CARD9 KNOCKOUT AMELIORATES FIBROSIS AND HYPERTROPHY IN A TAC PRESSURE-OVERLOAD MODEL

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Nearly 1 in 3 adults aged over 20 in the US presents with hypertension, which can lead to left-ventricular hypertrophy, hypertensive heart disease, and heart failure. The progressions of each are tied closely to elevated systemic and local inflammation. CARD9, a cytosolic protein expressed in macrophages and neutrophils, mediates secretion of inflammatory cytokines by these cells. We recently reported rescued myocardial function by knockout of CARD9 (CARD9-\-) in high-fat diet overfeeding [1]. Macrophage deletion ameliorates the progression of TAC-induced left ventricular hypertrophy and fibrosis [2]. Therefore, the objective of this study is to investigate if CARD9 knockout rescues progression of TAC pressure-overload induced hypertrophy. Investigators adhered to standards of ethical animal treatment as stipulated by the IACUC. C57BL/6 wild-type (WT) and CARD9-\- mice were assigned to thoracic aortic constriction (TAC) or to sham (CONT). To assess heart function, fractional shortening (FS), was measured by echocardiogram 1, 2, and 3 months post-surgery. FS was compromised in WT-TAC at 2 (48.2, 36.1; $p<0.05$) and 3 months (47.5, 36; $p<.05$), but not significantly so in CARD9-\-TAC at any time point. At 3 months, mice were sacrificed. Heart weight/tibia length was elevated in WT-TAC compared to WT-CONT (6.7, 11.7$p<0.05$), but not in CARD9-\-TAC. Histological results of Masson's trichrome stain suggests concentric hypertrophy in both TAC groups, but revealed less fibrosis in CARD9-\-TAC than in WT-TAC. In conclusion, CARD9 is a novel target for TAC-induced dysfunction, possibly through reductions in fibrosis and hypertrophy. Further study is needed to investigate signaling transduction involved, as well as the extent to which CARD9-\- may be protective in order to determine if it is a potential target for intervention in hypertensive heart disease. *This study is funded by INBRE.* References: [1] L. Cao, X. Qin, M. R. Peterson, S. E. Haller, K. A. Wilson, N. Hu, X. Lin, S. Nair, J. Ren, and G. He, "CARD9 knockout ameliorates myocardial dysfunction associated with high fat diet-induced obesity," *J. Mol. Cell. Cardiol.*, vol. 92, pp. 185–195, 2016. [2] D. Kain, U. Amit, C. Yagil, N. Landa, N. Naftali-Shani, N. Molotski, V. Aviv, M. S. Feinberg, O. Goitein, T. Kushnir, E. Konen, F. H. Epstein, Y. Yagil, and J. Leor, "Macrophages dictate the progression and manifestation of hypertensive heart disease.," Int. J. Cardiol., vol. 203, pp. 381–395, 2016.

10.16
CYTOTOXIC CD8+ T-CELLS PLAY A ROLE IN HYPERTENSION-ASSOCIATED INFLAMMATORY RESPONSES IN FEMALE DAHL RATS

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Introduction: Oophorectomy is associated with increased body weight (BW) gain and incidence of hypertension (HT) compared with age-matched women. Increased BW gain and HT coincide with increased inflammatory biomarkers. To separate immune responses due to HT from those due to BW gain, we compared T-cell subpopulations in normotensive Dahl (DR) and hypertensive Dahl (DS) rats after ovariectomy. **Methods:** Six week old DS and DR rats were ovariectomized (Ovx) or sham operated (Sham). Mean arterial pressure (MAP) was measured acutely in anesthetized rats after 10 months (mo) by indwelling vascular catheter. Splenic T-cells were assessed by flow cytometry at 10 mo. **Results:** As expected, at 10 mo DS rats developed HT while DR rats did not. [MAP (mmHg): DS-Ovx, 190±12*; DS-Sham, 177±6*; DR-Ovx, 114±1; DR-Sham, 102±3; *$p<0.005$ vs DR, same surgical status; n=5-8/group]. Ovariectomy increased BW to a similar extent in both DS and DR rats [Ovx BW-Sham BW (g): DS, 179±13 vs DR, 202±14; ns]. This model allows us to study immune mechanism as involved in HT that are disassociated from the magnitude of BW gain. The hypertensive DS rats showed signs of ongoing kidney disease as compared to the normotensive DR rats. The frequency of $CD8^+$ interleukin (IL)-$17a^+$ T-cells were higher in DS compared to DR rats [Frequency at 10 mo (%): DS-Ovx, 9.6±2*; DS-Sham, 7.6±2*; DR-Ovx, 1.3±0.4; DR-Sham, 3.1±1.6; *$p<0.0001$ vs. DR, same surgical status; n=5-8/group]. Similarly, the frequency of $CD8^+$ $Foxp3^+$ T-cells were higher in DS compared to DR rats [Frequency at 10 mo (%): DS-Ovx, 26±6*; DS-Sham, 15±2*; DR-Ovx, 5±1.5; DR-Sham, 5.2±1; *$p<0.0001$ vs. DR, same surgical status; n=5-8/group]. **Conclusion:** Imbalance in the "pro" and "anti" inflammatory cytotoxic $CD8^+$ T-cell population may contribute to the hypertension-associated inflammatory response that is independent of body weight gain in female

DS rats. **Implications:** This work may lead to sex specific immune targets for the treatment of HT and kidney disease. **Funding:** This study was supported by NIH R01 HL119380 to KS and Georegtown University Medical Center.

10.17
ARTERIAL STIFFNESS DUE TO CAROTID CALCIFICATION DISRUPTS NEUROVASCULAR COUPLING AND LEADS TO COGNITIVE DEFICITS

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Arterial stiffness is a strong risk factor for cognitive decline and dementia. It refers to the diminished capacity of an artery to buffer pulsatile blood flow from ventricular ejection. Arterial stiffness increases with age and is associated to high systolic blood pressure; however its effects on the brain remain poorly understood. Therefore, this study aimed to examine the effects of arterial stiffness on learning and memory, neurovascular coupling, blood-brain barrier (BBB) permeability and features of Alzheimer's disease pathology, in a novel murine model based on carotid calcification. Arterial stiffness was induced by the application of a 0.3M $CaCl_2$ –soaked pad on the right carotid artery (20 min) of C57BL/6 male mice aged 10-12 weeks. Control mice received 0.9% NaCl, in identical conditions. Animals were sacrificed between 2 and 3 weeks after surgery. The application of $CaCl_2$ led to increased collagen deposition, elastin fragmentation and macrophage infiltration in the carotid artery. In the brain, carotid calcification attenuated the cerebral blood flow response to whisker stimulation and to the topical application of the endothelium-dependent vasodilator acetylcholine, monitored by laser-Doppler flowmetry *in vivo*. Mice with arterial stiffness also exhibited a slower spatial learning acquisition in the Morris water maze test and significant impairments in spatial reference memory. Although modest, arterial stiffness led to a shift in the Aβ40/Aβ42 ratio in the frontal cortex, without affecting tau protein phosphorylation. Analysis of cerebral autoregulation, vascular amyloidosis and BBB permeability are underway. These initial results show that arterial stiffness has a negative impact on cognitive and cerebrovascular functions, and should therefore be considered as a target to protect the brain in the elderly and in hypertensive individuals. *This research was supported by funding from the Heart and Stroke Foundation of Canada (HG), the Fonds de recherche du Québec-Santé (HG), the Canadian Foundation for Innovation (HG), the Natural Sciences and Engineering Research Council of Canada (EP) and the Canadian Institutes of Health Research (GF, EP, HG). MFI was the recipient of a postdoctoral fellowship from the Groupe de Recherche sur le Système Nerveux Central (GRSNC, and MG and FRP were recipients of a Biomedical Doctoral Award from the Alzheimer Society of Canada.*

10.18
DYNAMIC VASCULAR T CELL-ANTIGEN PRESENTING CELL INTERACTIONS DURING HYPERTENSION

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T cells contribute to the development of experimental hypertension, which is associated with significant accumulation of T cells into the perivascular fat surrounding the aorta and renal vasculature. While a hypertension-specific neoantigen has been implicated in T cell activation, whether vascular-infiltrating T cells recognize and are locally activated by an antigen within the vessel wall remains unclear. We have developed an explanted aorta model to examine live *dynamic* interactions between vascular T cells and antigen presenting cells (APCs) within healthy and hypertensive mouse vessels. Indicators of antigen presentation are slower T cell velocities, greater interaction time and a greater proportion of T cells interacting with APCs. The aim of this study was to identify whether hypertension-specific cognate antigens are presented to and recognised by T cells within the vessel wall of hypertensive mice. Splenic T cells were isolated from normotensive (*nT* cells) and hypertensive angiotensin II (Ang II)-infused (0.7mg/kg/day; 14 days; *hT* cells) C57BL6/J mice. Following anti-CD3/CD28 stimulation (48 hours), cells were fluorescently labelled and co-incubated simultaneously (16 hours) with explanted aorta from normotensive or hypertensive CD11c-YFP mice, where APCs expressed yellow fluorescent protein. We

detected a ~2-fold increase in CCR5 ligand (CCL3, CCL4 and CCL5) secretion from hypertensive mouse aorta compared to vehicle-treated mouse aorta (*P<0.05; n = 4). Using 2-photon microscopy, we observed a greater number (~2-fold) of *hT* cells compared to *nT* cells within Ang II-infused mouse aorta (390±113 Vs 198±49). Importantly, time-lapse recordings of hypertensive mouse aorta revealed *hT* cells exhibited significantly slower velocity (*hT* cells 2.6 μm/min Vs *nT* cells 4.4 μm/min; P<0.01, n=8-11), longer duration of interaction (*hT* cell 36.0±5.2 min Vs *nT* cells 25.7±5.1; P<0.01, n=8-11) and a greater proportion of interactions with APCs (*hT* cells 10.7±2.3 Vs *nT* cells 1.5±0.7%; P<0.01, n=8-11). Consistent with current evidence, chronic treatment of Ang II-infused CD11c-YFP mice with an isoketal scavenger, 2-hydroxybenzylamine (2-HOBA), blunted pressor responses to Ang II, but interestingly, also abolished antigen presentation to *hT* cells within the aorta. Collectively, these data are the first evidence that vascular-infiltrating T cells recognise cognate antigens that are presented by APCs within the diseased vessel wall during hypertension.

10.19
LOSS OF ET_B RECEPTOR FUNCTION ACTIVATES NOD-LIKE RECEPTOR AND INFLAMMASOME SIGNALING PATHWAYS IN RENAL OUT MEDULLA DURING TYPE 1 DIABETES THROUGH AN ER STRESS-INDEPENDENT MECHANISM

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Renal infiltration of immunocompetent cells and increased production of inflammatory markers are key in the pathogenesis of diabetic kidney disease. Endothelin-1 (ET-1), a potent vasoactive peptide that acts through two receptors (ET_A and ET_B), has been implicated in diabetes and is upregulated in patients with diabetic nephropathy (DN) and in animal models of diabetes-induced kidney injury. ET_B receptors are highly expressed in renal outer medulla (OM). ET-1 exerts pro-inflammatory actions in the kidney; however, the mechanism(s) by which ET-1 mediates these effects are unclear. The present studies were designed to determine the role of the ET_B receptor in the activation of inflammasome and NOD-like receptor signaling pathways in the renal OM during type 1 diabetes (T1D). ET_B deficient (ET_B def) and transgenic (TG) control rats were made diabetic by i.v. injection of streptozotocin (STZ). 10 wk later, OM were isolated and expression of inflammasome genes was assessed by RT-PCR array. Diabetes led to upregulation of NLRP5 (4-fold increase vs. TG controls; n=3/group; $p<0.05$) and IL-1β (~3-fold increase vs. TG controls; n=3/group; $p<0.05$) in OM of ET_B def rats. In addition, PSTPIP1, a negative regulator of the inflammasome, was decreased in OM of diabetic ET_B def rats compared to TG controls. Together, these results demonstrate an overactivation of the downstream signaling of NOD-like receptor and inflammasomes in the absence of functional ET_B receptor. Recently, endoplasmic reticulum (ER) stress has been identified as an inducer of inflammasome activation; thus, we tested if diabetic ET_B def rats had an exaggerated ER stress response in the OM. RNA expression of GRP78, ATF-4, ATF-6, s-XBP-1, CHOP and caspase-12 was not different in OM of diabetic TG control and ET_B def rats. Our data suggest that the activation of NOD-like receptor and inflammasome signaling pathways in this diabetic model is not mediated by ER stress. Funded by NIH T32 DK007545 to CDM and P01 HL95499 to DMP and JSP.

10.20
A HIGH FAT DIET INCREASES BLOOD PRESSURE AND LEADS TO A PROINFLAMMATORY IMMUNE CELL AND CYTOKINE PROFILE IN THE AORTAE OF FEMALE DAHL SALT-SENSITIVE RATS (DSS)

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A high fat (HF) diet has been linked to hypertension, which is often associated with an accumulation of macrophages and other leukocytes in the artery wall. Indeed, T lymphocytes were recently found to mediate HF-diet induced increases in blood pressure (BP) in male DSS. Women represent ~50% of hypertensive cases and are more likely than men to be obese; yet there is limited data on the impact of HF-diet on BP and immune cell activation in females. We hypothesized that a HF diet increases BP and proinflammatory immune cells and cytokines in the aortae of female DSS. Five-week old females were maintained on normal fat (NF; 7.2% fat n=4) or HF diet (35% fat, n=3) for 10 weeks. Systolic BP was measured by tail-cuff and aortic T cells, macrophages, and cytokines were measured by flow cytometric analysis. Total fat intake was significantly higher in the HF group after 10 wks (kcal/24hr: 127 ± 11 HF vs 80 ± 10 NF, P=0.03), and this likely contributed to the greater increase in body weight in HF-fed females after 10 wks (% increase: 196 ± 4 HF vs 160 ± 9 NF, P=0.01). A 10 wk HF diet increased BP (systolic BP in mmHg: 178 ± 7 HF vs 150 ± 8 NF, P=0.05). HF diet also led to an increase in total T cells (expressed as % total aortic cells, P=0.05) and

T cell activation (expressed as % total T cells, P<0.005). A 10 wk HF diet also resulted in greater numbers of pro-inflammatory Th17 cells (expressed as % $CD3^+CD4^+$ T cells: P=0.004), M1 macrophages (expressed as % total aortic cells, P=0.01), and $TNF\alpha^+$ cells (expressed as % total aortic cells, P=0.06). Although there was no change in anti-inflammatory T regulatory cells (expressed as % $CD3^+CD4^+$ T cells, P=0.67), M2 macrophages (expressed as % total aortic cells: P <0.0001), and $IL\text{-}10^+$ cells (expressed as % total aortic cells, P=0.01) were lower following a HF-diet. We conclude that a HF diet increases BP and the pro-inflammatory T cell, macrophage, and cytokine profile in the aortae of female DSS. Future studies will address the role of female sex hormones on BP and immune cell status in response to HF-diet, as well as the contribution of T cells and macrophages in HF-diet induced increases in BP.

Type of Immune Cell	Normal Fat	High Fat
Total T cells ($CD3^+$)	25 ± 0.3	30 ± 2
Activated T cell ($CD3^+$ $CD44^+$)	19 ± 3	42 ± 2
M1 macrophages ($CD11b/c^+$ $CD206^+$ $TNF\alpha^+$)	1 ± 0.01	3 ± 0.4
M2 macrophages ($CD11b/c^+$ $CD206^+$ $IL\text{-}10^+$)	1 ± 0.01	0.28 ± 0.05
Effector Th17 cells ($CD3^+$ $CD4^+$ IL-17)	5.3 ± 0.9	12.8 ± 1.1
T regulatory cells ($CD3^+CD4^+$ $FoxP3^+$)	1.3 ± 0.3	1.1 ± 0.3
$TNF\alpha^+$ cells	4 ± 0.9	8 ± 1
$IL\text{-}10^+$ cells	3 ± 0.5	6 ± 0.3

10.21
INDUCTION OF AP-1, NF-Kb, $_{MI}$R-21 EXPRESSION, AND ANGIOTENSIN BY TUNGSTEN CARBIDE-COLBALT NANOPARTICLES INVOLVES ROS-MEDIATED MAPK PATHWAYS AND RESULTS IN TRANSFORMATION OF JB6 AND BEAS-2b CELLS

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Tungsten carbide-cobalt (WC-Co) nanoparticle composites have wide applications because of their hardness and toughness. WC-Co has been classified as Group 2A (probably carcinogenic to humans) by the IARC. The present study examined the alterations of miR-21-PDCD4 signaling in JB6 cells after exposure to WC-Co nanoparticles. The results showed that (1) WC-Co induced AP-1and NF-κB activity as well as ERK 1/2, p38, AKT, and JNK signaling in JB6 and BEAS-2B cells. (2) WC-Co caused PDCD4 inhibition in JB6 cells; (3) exposure of cells to WC-Co caused a significant increase of miR-21 expression and decrease of PDCD4 expression; (4) inhibition of ERKs with U0126 reversed WC-Co-induced PDCD4 inhibition, but inhibition of p38 with SB203580 did not; and (5) ROS scavengers, N-acetyl-L-cysteine and catalase, reversed the inhibitory effect of WC-Co on PDCD4 expression, while superoxide dismutase promoted the inhibition. (6) Chronic exposure to WC-Co nanoparticles induces colony formation in JB6/AP-1 and BEAS-2B cells in the Soft Agar Transformation Assay. (7) Lastly, WC-Co nanoparticles also affected the cells to induce angiogenesis tested by chicken chorioallantoic membrane (CAM) assay. These findings demonstrate that WC-Co nanoparticles induce AP-1, NF-κB, miR-21 expression while inhibiting PDCD4, which may be mediated through ROS, especially by endogenous H_2O_2, and ERK pathways. Unraveling the complex mechanisms associated with these events may provide insights into the initiation and progression of WC-Co-induced carcinogenesis.

10.22
SYMPATHETIC NERVES PROMOTE HYPERTENSION-SPECIFIC EFFECTOR MEMORY T CELL HOMING AND PROLIFERATION IN THE BONE MARROW OF MICE WITH ANGIOTENSIN II INFUSION

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We have recently identified a critical role of hypertension-specific effector memory T lymphocytes (T_{EM} cells) in response to repeated surges of blood pressure. Formed during an initial hypertensive challenge, a part of T_{EM} cells reside in the bone marrow (BM) in a quiescent state for prolonged periods and can be reactivated upon re-exposure to the hypertensive stimulus. Hypertension is associated with increased sympathetic outflow, including the input to BM. We therefore hypothesized that sympathetic nerves regulate accumulation and reactivation of hypertension-specific T_{EM} cells in BM. To test this, we performed unilateral superior cervical ganglionectomy (SCGx) in wild-type C57BL/6 mice, which selectively sympathectomizes the forelimb on the surgical side. After recovery from surgery, the mice receive angiotensin II infusion (490ng/kg/min) for two weeks. The number of T_{EM} cells in the denervated BM following ang II was slightly but significantly less than observed in the innervated limb as measured by flow cytometry. To determine T cells in the BM that were specific to hypertension, 5×10^6 BM

cells were isolated from either the SCGx or control limbs, loaded with proliferation marker CFSE, and co-cultured with 0.5×10^6 splenic dendritic cells isolated from another ang II-infused mouse. We found 30% less $CD8^+$ T cell proliferation in the SCGx BM compared to control side (1.8 ± 0.1 vs. $2.6\pm0.3\times10^4$), but no difference in $CD4^+$ T cells. To further study the effect of sympathetic nerves on T cell homing in BM, 1×10^7 pan T cells were isolated from wild-type mice (w/CD45.2 allele) after angiotensin infusion, and adoptively transferred into CD45.1 mice that had previously received SCGx. As determined by flow cytometry 7 days after transfer, 25% less $CD8^+$ T_{EM} cells from hypertensive donors homed to the SCGx BM than the innervated BM of recipients (20.8 ± 2.5 vs. 27.8 ± 2.6 per 10^3 total BM cells). To study the response of these T_{EM} cells to a repeated hypertensive challenge, a subset of these SJL mice was also subjected to angiotensin infusion after adoptive transfer. We found respectively 50% and 36% less $CD4^+$ and $CD8^+$ T_{EM} cells from hypertensive donors were positive for the proliferation marker Ki-67. We conclude that sympathetic nerves promote T_{EM} cell homing to the BM after an initial blood pressure elevation, and also promote proliferation of these cells upon a repeated hypertensive challenge.

10.23
INDUCTION OF AP-1 SIGNALING AND DNA DAMAGE BY COPPER OXIDE NANOPARTICLES INVOLVE ROS-MEDIATED MAPK PATHWAYS

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Occupational exposures to copper dusts or fumes have been reported to be harmful to human health, with possible risk of cancer among copper smelter workers. Copper (II) oxide (CuO) nanoparticles have not, to our knowledge, been extensively examined for potential carcinogenic or genotoxic effects. To investigate the mechanisms of CuO-induced pathogenesis, the effect of CuO on AP-1-MAPKs and ROS generation were investigated. The results indicated that CuO caused a 2-fold increase in AP-1 activity in JB6 cells. The induction of AP-1 activity in cultured cell lines was time- and dose-dependent. The signal transduction pathways for AP-1 activation were also investigated. Western Blot analysis demonstrated that CuO stimulates phosphorylation of p38 MAPK and ERKs. CuO also generated ROS when incubated with the cells as measured by electron spin resonance (ESR). Span "mso-spacerun: yes;" Nano-sized CuO generated more ROS than the fine-sized particles when incubated with the cells. COMET assay suggests that exposure of the cells to CuO resulted in DNA damage. Soft agar transformation assays have concluded that there is a significant increase in colony formation in JB6/AP-1 cells treated with CuO particles as compared to the control. Unraveling the complex mechanisms associated with these events may provide insights into the initiation and progression of CuO-induced pathogenesis.

10.24
A NEW ROLE OF SOX6 IN BLOOD PRESSURE CONTROL THROUGH RENIN REGULATION

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Hypertension afflicts 33% of the U.S. adult population. Despite current treatments, approximately 50% of people with hypertension have uncontrolled blood pressure. Thus, there is a critical need to develop new therapies to treat this disease and its complications. The Renin Angiotensin Aldosterone System (RAAS) plays a key role in regulating blood pressure in humans. Renin controls the rate-limiting step in the conversion of angiotensinogen to angiotensin I. In adults, renin is produced and stored by juxtaglomerular (JG) cells in the kidney. However, the transcriptional mechanisms that govern the specification of renin expressing cells under normal or pathophysiological conditions and the contribution of the immune system remain poorly understood. During blood pressure changes the number of adult renal cells expressing renin increases through a process known as JG cell expansion. Mesenchymal stromal-like cells (MSCs) are pluripotent cells that can have immunomodulatory actions. We found that MSCs can differentiate to renin expressing cells *and sought to determine regulators of renin expression and blood pressure control*. Renin expression was induced in adult renal MSCs by treatment with 3-isobutyl-1-methylxanthine (IBMX) and Forskolin *in vitro*. Gene array experiments between renal MSC and JG cells identified a number of potential candidates that control MSC differentiation, including Sox 6. *In vitro* silencing of Sox6 by lentivirus-mediated shRNA decreased the differentiation of renal MSCs to renin producing cells (3.5 fold, n=4, P=

0.01). Our preliminary IHC data show that the transcriptt-ion factor Sox6 is expressed in renin producing cells in the adult kidney after stimulation with low sodium diet and furosemide, which promotes JG cell expansion. We used a novel transgenic mouse in which Sox6 is knocked out specifically in renin expressing cells (Ren1dCre/Sox6$^{fl/fl}$), to study the impact of Sox6 in renin expression *in vivo*. Importantly, our preliminary data show that plasma renin concentration (PRC - ug Ang I/mL/hr) highly increased during JG cell expansion in wild type mice (PRC increases 27-fold), and mice lacking Sox6 in JG cells (Ren1d Cre/Sox6$^{fl/fl}$) their PRC is as low as non-treated mice. These results support a novel role for Sox6 in renin expression and renal physiology, offering new approaches for understanding the physiological regulation of renin, immunity and hypertension.

10.25
PULMONARY HYPERTENSION-INDUCED RIGHT VENTRICULAR PRESSURE OVERLOAD TRIGGERS ACUTE EPIGENETIC REPROGRAMMING OF PRO-INFLAMMATORY CARDIAC FIBROBLASTS

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Right ventricular (RV) function is a strong predictor of survival in a variety of clinical contexts including pulmonary hypertension (PH), highlighting the importance of delineating signaling pathways that contribute to RV dysfunction. We have previously demonstrated increased inflammation in PH-induced RV dysfunction and hypothesized that PH stimulates pro-inflammatory changes in RV cardiac fibroblasts (Cfibs). Further, we hypothesized that this process may be mediated by epigenetic changes in the fibroblast such as DNA methylation, which allows rapid and dynamic regulation of gene expression. We explored this hypothesis using a large animal model with significant resonance with human disease- the neonatal calf exposed to hypobaric hypoxia (HH). We assessed DNA methylation, gene expression and inflammation in low (2, 3) and high (6, 7) passage control (CO) and HH RV Cfibs. Exposure to HH resulted in global DNA hypomethylation in low passage RV Cfibs while global DNA methylation was not different between CO and HH high passage Cfibs. Further, we quantified expression of regulators of global DNA methylation, DNA-methyltransferases (DNMT). DNMT3a expression was unchanged by HH in both high and low passage cells. However, DNMT1 and DNMT3b expression were significantly downregulated by HH in low passage Cfibs, correlating with DNA hypomethylation in these cells. In low passage cells, RNA-seq identified 2115 genes significantly changed in HH including 193 transcriptional regulators, 21 genes involved in DNA methylation, and 105 inflammatory genes. We verified enrichment of inflammatory signaling by qRT-PCR and found HH significantly upregulated Interleukin-1β and Interleukin-6 only in low passage cells. Experiments aimed at identifying differentially regulated pathways in high passage RV Cfibs by RNA-seq are ongoing. Together, these data suggest the combination of pressure overload and hypoxia is correlated with epigenetic reprogramming of Cfibs and increased inflammatory signaling. This unique pro-inflammatory Cfib phenotype is maintained for multiple passages but is eventually attenuated over time with removal of the PH insult. Future investigations will identify specific genes regulated by PH-induced fibroblast reprogramming. We propose that therapies which target this acute fibroblast-mediated inflammatory process have the potential to prevent RV dysfunction.

10.26
MIR-762 INHIBITION PREVENTS AND REVERSES ANGIOTENSIN II INDUCED AORTIC FIBROSIS AND STIFFENING

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We and others have shown that hypertension (HTN) is associated with a striking deposition of collagen in the aortic adventitia. This leads to aortic stiffening and eventually end-organ damage. Through a screen of microRNAs (miRNAs) in the aorta, we found that miR-762 is the most upregulated miRNA in mice with angiotensin II (Ang II)-induced HTN. qRT-PCR confirmed that miR-762 is upregulated 6.35±1.22 ($p=0.03$) fold in Ang II-infused mice compared to controls. To study the role of miR-762 in HTN, we administered a locked nucleic acid inhibitor of miR-762. MiR-762 inhibition did not influence the hypertensive response to Ang II, yet it normalized stress-strain relationships and aortic systolic energy storage (ASE) (Table1). Moreover, miR-762 inhibition in the last 2 weeks of Ang II infusion reversed aortic stiffness in mice treated with Ang II for 4 weeks (ASE, 4 wk Ang II [51±5.18 kPa] vs 4wk Ang II + LNA-762 (last

two weeks) [20±1.76 kPa], p<0.0001). Further studies showed that miR-762 inhibition reduced mRNA for several collagens and fibronectin and dramatically upregulated collagenases MMP1a, 8 and 13 (Table1). We also found that miR-762 inhibition during Ang II infusion led to a 9.11±1.92 (p=0.007) fold upregulation of Sprouty1 mRNA, suggesting that miR-762 targets the degradation of Sprouty1 mRNA. Sprouty1 inhibits activation of p38-MAPK which is central in the pathogenesis of aortic stiffening. Hence, miR-762 modulates aortic stiffening and fibrosis through a Sprouty1-p38-MAPK dependent mechanism. Thus, miR-762 has a major role in modulating aortic stiffening and its inhibition dramatically inhibits pathological fibrosis, enhances matrix degradation, prevents and reverses aortic stiffness. miR-762 inhibition might represent a new approach to prevent aortic stiffening and its consequent end-organ damage. **Table 1:** The effect of miR-762 inhibition on aortic fibrosis, stiffening and matrix gene expression. All values are presented as mean±SE.

	Sham	Ang II	Ang II + LNA-762	P value (One way ANOVA)
Adventitial Collagen (mm^2)	75.8±10.0	283.9±15.6	79.9±7.1	<0.0001
Systolic energy storage (W_{sys}, kPa)	66±2.49	17±1.33	61±4.73	<0.0001
Collagen 1α1*	1.0±0.55	8.56±2.95	0.36±0.04	0.0149
Collagen 3α1*	1.0±0.35	6.93±1.37	0.00025±0.000018	0.0004
Collagen 5α1*	1.0±0.44	3.34±0.61	0.59±0.06	0.0033
Fibronectin 1*	1.0±0.36	2.10±0.18	0.00048±0.000055	0.0004
MMP1a*	1.0±0.21	1.84±1.12	5,010±600	<0.0001
MMP8*	1.0±0.34	5.27±2.83	126.74±20.09	<0.0001
MMP13*	1.0±0.21	3.76±1.55	39.35±3.22	<0.0001

*Note: All fold change values were normalized to the sham group.

10.27
THE R213G POLYMORPHISM IN EC-SOD PROTECTS EARLY BLEOMYCIN-INDUCED PULMONARY INFLAMMATION AND ATTENUATES INDUCTION OF GENES INVOLVED IN LEUKOCYTE EXTRAVASATION

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Extracellular superoxide dismutase (EC-SOD) is a key lung antioxidant enzyme, protecting against oxidative stress and inflammation in acute lung injury (ALI). A single nucleotide polymorphism (SNP) substituting arginine to glycine at 213 (R213G) in *SOD3* decreases tissue binding, thereby increasing EC-SOD in the extracellular fluids such as epithelial lining fluid and plasma. Higher EC-SOD in extracellular fluids confer protection against ALI in humans, and the R213G polymorphism protects against lipopolysaccharide induced ALI in mice. In this study, we analyzed how the R213G SNP modulates lung proinflammatory signaling pathways in an intratracheal bleomycin mouse model of ALI, characterized by early alveolar inflammation progressing to lung fibrosis. In wild type (WT) mice, bleomycin increased proinflammatory cytokines, measured with V-plex (mesoscale discovery), over 21 days. In contrast, R213G mice developed a more robust induction of inflammation 3 days post-bleomycin, including proinflammatory cytokines IL-1β, TNFα, IL-6, IFNγ, and anti-inflammatory cytokine, IL-10. However, the inflammation resolved quickly in R213G mice by day 7 (n=6-8). Unlike the WT mice, R213G mice did not progress to lung fibrosis, shown by pulmonary mechanics and collagen deposition. To identify molecular mechanisms by which the R213G SNP protects against ALI, we performed high throughput RNAseq in lung RNA and bioinformatics to evaluate gene pathways that were altered 7 days post-bleomycin in WT and R213G mice. The top differentially activated pathway, determined by ingenuity pathway analysis, was leukocyte extravasation (WT = 2.121, 9.55E-05; R213G = 0.59, 9.77E-08; z-score and q-value, respectively). This innate immune response has an important role in the recruitment, engagement/adhesive interactions and transendothelial migration of leukocytes. Genes involved in leukocyte diapedesis (ITGAM, ITGA4, ITGA1, ICAM1, and PECAM1), superoxide generation through NADPH oxidase (CYBs and NCFs) and hydrolyzation of extracellular matrix (MMP8, MMP12, MMP13), were differentially expressed between WT and R213G mice. Studies are ongoing to validate these findings. RNA sequencing and bioinformatic analysis identifies potential pathways responsible for the protective effects of the R213G SNP and provides a foundation to better understand the role of redox regulation in inflammatory lung diseases.

10.28
INTERLEUKIN 21 PROMOTES HYPERTENSION AND END-ORGAN DYSFUNCTION

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We have previously shown that T cell derived pro-inflammatory cytokines such as interleukin 17A (IL17A) and interferon gamma (IFNγ) are upregulated by and can promote angiotensin II-induced hypertension. IL21 is a pleiotropic cytokine produced primarily by T follicular helper (Tfh) cells and T helper 17 (Th17) cells. It was recently demonstrated that IL21 deficient mice have reduced gastric inflammation following Helicobacter pylori infection accompanied by a decrease in local production of IL17A and IFNg[1]. The role of IL21 in hypertension is unknown. We hypothesized that IL21 deficient mice would be protected from hypertension and hypertensive end-organ damage in response to angiotensin II (Ang II) infusion, in part through reduced production of IL17A and IFNγ. We found that indeed IL21$^{-/-}$ mice exhibited a 28 mmHg reduction in blood pressure in response to 4 weeks of Ang II infusion compared to age-matched wild type (WT) mice (p=0.0155). Preliminary data demonstrates that IL21 mRNA expression increases 1.5 fold in splenic T cells in response to Ang II (p=0.015). IL17A production from splenic CD4+ T cells and IFNγ production from splenic CD8+ T cells was reduced in IL21$^{-/-}$ mice compared to WT mice after 4 weeks of angiotensin II infusion. Finally, renal function was assessed by measuring albumin to creatinine ratio in spot urine following Ang II infusion. Wild type mice developed 3.6 times more albuminuria compared to IL21$^{-/-}$ mice (p=0.02). Taken together, these studies suggest that IL21 may play a key role in hypertension, in part through modulation of IL17A and IFNg production. Immunobiology of Blood and Vascular Systems Training Program, NIH 2 T32 HL069765-11A1. 1. Carbo A, Olivares-Villagomez D, Honetecillas R, Bassaganya-Riera J, Chaturvedi R, Piazuelo MB, Delgado A, Washington MK, Wilson KT, Algood HM. Systems modeling of the role of interleukin-21 in the maintenance of effector CD4+ T cell responses during chronic Helicobacter pylori infection. MBio. 2014; 5 (4).

10.29
EFFECT OF BETA BLOCKERS ON POST-EXERCISE CYTOKINE RESPONSE

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Aerobic exercise induces a stress response, including an increase in epinephrine and inflammatory cytokines. Based on results from cell culture models, monocyte inflammatory cytokine response is dependent on β_1 adrenergic receptors (AR). However, data from human in vivo studies is limited. The purpose of this study was to determine post-exercise cytokine changes when participants exercised after taking a non-selective or selective (β_1 AR) beta blocker. Methods: This is a preliminary report of an on-going study. To date, 12 healthy, young adults (30.9±6.0 yrs; body mass index 26.9±1.1) have completed the double-blind randomized cross-over design protocol. Three hours after ingestion of placebo, nadolol (non-selective beta blocker), or bisoprolol (selective β_1 AR beta blocker) a resting blood sample was collected. The subject exercised 30 minutes on a leg cycle ergometer at a workload 10% above lactate threshold. Immediately and one-hour post-exercise blood samples were collected. Serum cytokine analysis is underway (current analysis n=3). Results: Compared to pre-exercise, immediately post exercise serum TNF percent change was: placebo -6.7%, nadolol 44.0%, and bisoprolol -32.0%; and one-hour post exercise serum TNF percent change was: placebo -31.2%, nadolol 12.2%, and bisoprolol -68.5%. DIiscussion: Under the selective β_1 AR beta blocker (bisoprolol) condition TNF percent change from resting was lower immediately and one-hour post exercise, compared to placebo and non-selective beta blocker (nadolol). Similarly as to what is reported in cell culture models, the human in vivo post-exercise cytokine response may be dependent on β_1 AR. Although our subjects were healthy, there may be immune system considerations for those who exercise and are prescribed a beta blocker medication.

10.30
INVESTIGATING THE MECHANISMS BY WHICH IgG ANTIBODIES CONTRIBUTE TO ANGIOTENSIN II-DEPENDENT HYPERTENSION

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Recent studies by our laboratory have shown that angiotensin (Ang) II-induced hypertension in mice is associated with elevated serum and aortic IgG levels. Moreover, inhibition of B cell activation and IgG production protected mice against Ang II-induced increases in blood pressure

and vessel stiffening. However, the mechanism(s) by which IgG mediates these processes remains to be determined. Therefore, we investigated the potential role of two known effector mechanisms of IgG-mediated immunity in Ang II-induced hypertension, namely macrophage activation and upregulation of the complement system. Treatment of male C57BL6/J mice with Ang II (0.7 mg/kg/d, *s.c*) for 28 days increased tail cuff blood pressure by ~37 mmHg compared to saline-treated mice. Immunohistochemistry and flow cytometry revealed that Ang II-infusion was also associated with IgG deposition in the aortic adventitia as well as an increase in the number of CD206+ M2-like macrophages. Importantly, IgG was found to co-localise with these cells. As evidence that hypertension-specific antibodies can activate macrophages, stimulation of cultured RAW246.7 macrophages with purified IgG from Ang II but not saline-treated mice promoted the production of TGF-β. Finally, Ang II-dependent increases in aortic IgG were also associated with upregulation of C1q, C1r, C1s, C2, C3, C4 and C6, highlighting a possible role for the complement cascade. Taken together, these observations provide insights into the likely mechanisms by which B cell activation and IgG antibodies may promote hypertension and vascular remodeling, and suggest that targeting IgG-dependent macrophage and complement activation may represent novel therapeutic approaches to treating the condition.

10.31
MACROPHAGE-DERIVED IGF-1 CONTRIBUTES TO AORTIC FIBROSIS AND STIFFENING IN HYPERTENSIVE MICE

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M2 macrophages accumulate in the vessel wall during hypertension and are important mediators of vascular remodelling, fibrosis and stiffening. However, the mechanisms involved are yet to be defined. M2 macrophages are an important source of insulin-like growth factor-1 (IGF-1). In other disease settings, IGF-1 is known to contribute to fibrosis and tissue growth but its role in vascular remodelling during hypertension is unknown. Therefore, we examined whether macrophage-derived IGF-1 contributes to vascular fibrosis, aortic stiffening and elevated blood pressure (BP) in hypertensive mice. In male C57BL6/J mice, infusion of angiotensin II (Ang II, 0.7 mg/kg/d for 14 days, *s.c.*) elevated systolic BP by >50 mmHg ($P<0.05$) and increased collagen content and stiffness of the aorta, each by ~2-fold *(*$P<0.05$). These changes were accompanied by a 6.8-fold increase in M2 macrophage numbers ($CD45^+CD11b^+Ly6G^-F4/80^+CD206^+$ cells) in the aortic wall ($P<0.05$), and importantly expression of the M2 marker, CD206, was strongly positively correlated with that of IGF-1 ($r^2=0.57$; $P<0.001$). Depletion of monocytes (macrophage precursors) by treatment with clodronate-containing liposomes (50 mg/kg, every 3 days, *i.v.*) reduced the Ang II-dependent influx of M2 macrophages into the aorta by 75% ($P<0.05$) and simultaneously inhibited aortic IGF-1 expression by 32%, collagen deposition by 34%, aortic stiffening by 43% ($P<0.05$), and systolic BP by 25 mmHg (all $P<0.05$). Macrophage-specific IGF-1 deficient mice ($LysM^{Cre/+}$ x $IGF\text{-}1^{fl/fl}$) treated with Ang II also displayed reduced IGF-1 expression in the aorta (by 33%) compared to similarly treated control mice ($LysM^{+/+}$ x $IGF\text{-}1^{fl/fl}$; $P<0.05$). Moreover, IGF-1 deficient animals were protected from Ang II-induced increases in aortic collagen deposition, stiffening and systolic BP (all $P<0.05$). Finally, confirming that the effects of clodronate and macrophage-specific IGF-1 deficiency on vascular remodeling occurred upstream of their BP-lowering actions, an equivalent anti-hypertensive dose of the non-specific vasodilator, hydralazine (25 mg/L, *p.o.*), had no effect on Ang II-induced increases in aortic macrophages, IGF-1 expression or collagen deposition. In conclusion, M2 macrophage-derived IGF-1 plays a crucial role in the aortic fibrosis that contributes to vascular stiffening and elevated systolic BP during Ang II-induced hypertension in mice. Future studies aimed at unraveling the cellular targets and second messengers activated by IGF-1 in the aortic wall have the potential to reveal new targets for novel anti-hypertensive therapies.

10.32
OPPOSING EFFECTS OF $C3_A$ AND $C5_A$ ON KIDNEY INJURY IN ANGIOTENSIN II INDUCED HYPERTENSION

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Adaptive and innate immune responses participate in the pathogenesis of hypertension and hypertensive end-organ

damage. The complement system is an ancient part of innate immunity. Complement activation produces the cleavage fragments C3a and C5a. The anaphylatoxins C3a and C5a exert pleiotropic immunoregulatory functions that are mediated through cognate interaction with their G protein-coupled C3a receptor (C3aR) and C5a receptor 1 (C5aR1). Their role in hypertension is unknown. Using anaphylatoxin receptor reporter and knockout mice, we investigated the pathogenic role of the C5a and C3a in hypertensive renal injury. Out of the CD45+ cells isolated from the kidney of GFP-C5aR1 reporter knock-in mice 28% expressed C5aR1. The majority of CD45+ cells carrying C5aR1 were dendritic cells (89%) followed by neutrophils and macrophages. Using confocal microscopy, the C5aR1 was detected mainly on infiltrating cells, but a minor population of resident podocytes and parietal cells also expressed C5aR1. Since most mouse strains are resistant to hypertensive end-organ damage induced by Ang II, we used an aggravated model of hypertension by combining Ang II infusion with unilateral nephrectomy and salt in the drinking water. Deletion of C5aR1 ameliorated albuminuria compared to wildtype mice and reduced the expression of renal injury (NGAL) and the inflammation marker gene CCL2 in hypertensive mice. Additional deletion of C3aR eliminated the protective effects of C5aR1 deficiency, while deletion of C3aR alone aggravated albuminuria and renal injury (wildtype 18.7±4.1, $C3aR^{-/-}$ 35.7±8.3, $C3aR/C5aR1^{-/-}$ 6.0±1.6 mg albumin/mg creatinine). These findings suggest that the complement system regulates hypertensive kidney damage via opposing signaling pathways. While the C5a/C5aR1 axis promotes renal inflammation and albuminuria, the C3a/C3aR axis exerts protective effects.

10.33
INTRARENAL ANGIOTENSINOGEN FORMULATION IN RESPONSE TO CHRONIC HIGH SALT INTAKE AND ANGIOTENSIN ADMINISTRATION IS AUGMENTED IN TNF-α RECEPTOR KNOCKOUT MICE

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Background: Chronic angiotensin II (AngII) treatment enhances both angiotensinogen (AGT) and tumor necrosis factor- alpha (TNF-α) formation in the kidney. However, the link between such TNF-α increase and AGT formation remains unclear. We examined the hypothesis that AngII treatment with high salt (HS) intake reduces TNF-α receptor type 1 (TNFR1) activity and increases intrarenal AGT formation. Methods: Responses to AngII infusion (25 ng/min; implanted minipump) with HS (4% NaCl) diets for 4 weeks were evaluated in wild-type (WT; n=6) as well as knockout mice (KO) for TNFR1 (n=7) and TNFR2 (receptor type 2; n=6). Systemic blood pressure (SBP) was measured by tail-cuff plethysmography and 24-hour urine collections were done using metabolic cages. The urinary excretion rate of AGT (uAGT) was measured using ELISA. Results: AngII + HS induced increases in mean SBP was significantly greater in TNFR1KO (77±2 to 115±3 mmHg), but similar in TNFR2KO (78±2 to 99±5 mmHg) compared to WT (76±1 to 102±2 mmHg). Interestingly, the increase in uAGT was also significantly greater in TNFR1KO (6±2 to 167±75 ng/24hr) but not different in TNFR2KO (8±7 to 65±44 ng/24hr) compared to WT (6±3 to 46±16 ng/24hr). Conclusion: The results suggest that TNFR1 activity mitigates the hypertensive response to chronic AngII infusion with HS intake, likely by attenuating the increase in intrarenal AGT formation.

10.34
NLRP3 Inflammasome-mediated Immunity in Dahl Salt-Sensitive Hypertension

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Previous studies demonstrated the importance of infiltrating renal T lymphocytes in the pathogenesis of hypertension and renal damage in Dahl salt-sensitive (SS) rats. It remains unclear how this adaptive immune response becomes activated during the course of this sterile disease. The NLRP3 inflammasome identifies exogenous and endogenous danger signals and initiates innate immunity and host defenses. It is comprised of the sensory protein NLRP3, the adaptor protein ASC, and the effector procaspase-1, and when stimulated, allows for the processing of pro-inflammatory cytokines like IL-1β and IL-18. The following preliminary studies sought to characterize the expression of the NLRP3 inflammasome in SS kidneys during a low 0.4% NaCl and high 4.0% NaCl diet, and test whether pharmacological inhibition of NLRP3 inflammasome activation attenuates salt-induced hypertension and renal damage in SS rats. RNA sequencing data revealed a significant upregulation in RNA expression of NLRP3 (33.8%) and caspase-1 (26.4%), and a similar trend for IL-1β (25.5%), in the renal medulla of SS rats fed the high salt diet compared to low salt controls (n=4/group). Western blotting also showed increased protein expression of NLRP3 (1.5-fold) and ASC (3.2-fold) in the renal medulla of SS rats after 3 weeks of high salt, together demonstrating increased NLRP3 inflammasome

mRNA and protein expression in the renal outer medulla of SS rats during high salt. To assess the role of IL-1β, SS rats implanted with telemeters were administered IL-1 receptor antagonist anakinra (IL-1RA; 600μg/day i.p., n=4-6) daily during high salt challenge. Rats receiving IL-1RA tended to have lower mean arterial pressure (MAP) than those receiving saline, reaching significance on day 17 of treatment (159±6 mmHg saline vs. 141±5 mmHg IL-1RA) of high salt. An additional preliminary study using NLRP3 inflammasome inhibitor glyburide (Gly, 100mg/kg/day p.o., n=4-5) also demonstrated a trend for reduced MAP (156±8 mmHg vehicle vs. 145±9 mmHg Gly) and albuminuria (69±17 mg/day vehicle vs. 41±7 mg/day Gly). Since pharmacological NLRP3 and IL-1β inhibition appeared to mitigate the SS phenotypes, the NLRP3 inflammasome in SS kidneys may mediate the initiation of adaptive immunity and the subsequent infiltration of T lymphocytes that amplifies salt-induced hypertension and renal damage. (DK96859, HL116264, 5T32HL007792, 15SFRN2391002)

11.0: CAREER WORKSHOP

11.1
GET A JOB: BUILD THE SKILLS EMPLOYERS WANT!

Jennifer Sasser
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Data show that less than 8% of doctoral students will become tenure track faculty, and more and more graduates are in a wide array of non-academic positions. Therefore, it is crucial for trainees to develop additional "employability skills" during their PhD and postdoctoral training in order to be well qualified for the diverse job opportunities available to PhD's in the workforce. The overall objectives of the symposium are to provide information for trainees on the following: 1) PhD job market trends; 2) Employability Skills; 3) the APS/ACDP Professional Skills Document; and 4) the importance of an Individual Development Plan.

12.0: INFLAMMATION, IMMUNITY, INTESTINAL FLORA AND THE METABOLIC SYNDROME

12.2
SYSTEMIC OLFACTORY RECEPTORS AND THE GUT MICROBIOTA

Jennifer Pluznick[1], Niranjana Natarajan[1], Daijiro Hori[2], Sheila Flavahan[2], Jochen Steppan[2], Nicholas Flavahan[2], and Dan Berkowitz[2]
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SCFA metabolites (acetate, propionate and butyrate) are byproducts of gut microbial metabolism which have been shown to dilate blood vessels ex vivo. Recently, we reported that two SCFA receptors, Gpr41 and Olfr78, both play roles in blood pressure regulation. Here, we will highlight novel data showing the localization and function of Gpr41 – we find by RT-PCR that Gpr41 localizes to the vascular endothelium, where it plays a hypotensive role (Gpr41 KO mice have isolated systolic hypertension, as measured by telemetry). In agreement with a phenotype of systolic hypertension, KO mice also exhibited elevated pulse wave velocity in vivo, but surprisingly, no increase in ex-Â-vivo aorta stiffness (measured by tensile testing experiments). In sum, these studies demonstrate that endothelial Gpr41 lowers baseline BP, likely by decreasing vascular tone. Finally, we will explore the implications of these findings, and will integrate these new data with previously published work by ourselves and others regarding the role of the gut microbiota in the control of blood pressure.

12.3
METABOLIC SYNDROME

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The worldwide prevalence of obesity is associated with an escalating incidence of morbidity and mortality. This because the epidemic of obesity has been paralleled by an increase in the incidence of the metabolic syndrome, a cluster of risk factors including hypertension and that raises the risk for heart disease and other health problems. This has triggered a great interest in understanding the mechanisms underlying the adverse cardiovascular effects of obesity and metabolic syndrome which has led to significant progress in recent years. Defects in various biological processes ranging from genetic and humoral factors to basic cellular signaling pathways in different tissues have been involved in the pathogenesis of the obesity and metabolic syndrome. The diversity in the processes implicated is consistent with the polygenic and multifactorial nature of these conditions and their comorbidities. The new knowledge gained in recent years should be taken into account when seeking novel diagnostic and therapeutic approaches for the cardiovascular disorders caused by excess adiposity. Given the evidence pointing

to the significance of the neurogenic mechanisms in metabolic syndrome new strategies that disrupt these processes should be favored.

12.4
ALTERING THE MICROBIOTA FOR WEIGHT CONTROL

Sean Davies[1], Zhongyi Chen[1], Linda Zhang[1], Lilu Guo[1], Yongqin Zhang[1], Youmin Zhang[2], Lei Ding[2], Patricia Yancey[2], Arion Kennedy[3], Alyssa Hasty[3], MacRae Linton[2], and Kevin Niswender[4]

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Alterations in the gut microbiota have been implicated in the development of obesity-related diseases, establishing the gut microbiota as a therapeutic target. To alter the microbiota, we have focused on incorporating bacteria that sustainably biosynthesize metabolites with therapeutic effects. To assess the potential of this approach, we engineered *E. coli Nissle 1917* (EcN) to produce *N*-acylphosphatidylethanolamine (NAPE), by transformation of these bacteria with *A. thaliana* NAPE acyltransferase (pNAPE-EcN). When pNAPE-EcN are administered in drinking water concurrently with high fat diet (60% kcal), this pNAPE-EcN administration markedly inhibits gain of body weight and body fat compared to mice administered standard drinking water, vehicle (0.125% gelatin) or control bacteria (pEcN). Treatment of $LDLR^{-/-}$ mice receiving a Western diet with pNAPE-EcN resulted in reduced triglyceride accumulation in the liver, reduced expression of inflammatory genes such as *Tnfa* and *Ccl2*, and reduced expression of fibrotic markers such as *Timp1* and *Acta2*. These $LDLR^{-/-}$ mice also showed a modest trend toward reduced atherosclerotic lesions. The pharmacological effects of pNAPE-EcN persisted >4 weeks after cessation of administration. In mice fed a high fat diet (60% kcal) for four weeks or more prior to treatment with pNAPE-EcN, reductions in relative body weight are delayed by several weeks. One mechanism potentially underlying this delayed response is that pre-feeding high fat diet reduces activity of NAPE hydrolyzing phospholipase D (NAPE-PLD), the enzyme that converts NAPEs to N-acylethanolamides (NAEs). In support of this hypothesis, we found that administration of pNAPE-EcN failed to reduce weight gain or food intake in $Napepld^{-/-}$ mice, but the effects were restored if the administered bacteria heterologously co-expressed NAPE-PLD along with NAPE acyltransferase (pNAE-EcN). Together, these results demonstrate that incorporation of therapeutic bacteria into the gut microbiota can be used to treat obesity and that this treatment can potentially be tailored according to the specific phenotype of the individual. NIH R01 AT007830
Reference: Incorporation of Therapeutically Modified Bacteria into Gut Microbiota Inhibits Obesity. J. Clin Invest. 124:3391-3406 PMC4109548.

13.0: INFLAMMATION, HYPERTENSION, AND END-ORGAN DAMAGE

13.1
ALTERING THE MICROBIOTA FOR WEIGHT CONTROL

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Hypertension remains an enormous health care burden that affects 30% of Western populations. Despite its prevalence the cause of most cases of hypertension remain unknown. Our laboratory has defined a novel mechanism for hypertension involving adaptive immunity. We found that mice lacking lymphocytes ($RAG\text{-}1^{-/-}$ mice) develop blunted hypertensive responses to a variety of stimuli including chronic angiotensin II infusion, DOCA-salt challenge and norepinephrine infusion. Adoptive transfer of T cells, but not B cells, restores the hypertensive responses to these stimuli. Hypertension is associated with the infiltration of T cells into the kidney and vasculature, where they release cytokines, including IFN-γ, IL-17A, and TNFα, which promote sodium retention, vasoconstriction and oxidative injury. Recently, we have found that angiotensin II has striking effects on dendritic cells (DCs), promoting their propensity to activate T cells. Our data indicate that angiotensin II infusion increases DC superoxide production by 5-fold and causes a striking accumulation isoketals, oxidized products of arachidonic acid in these cells. These form covalent bonds to lysines of proteins and these modified proteins become immunogenic. Several isoketal scavengers, including 2-hydroxybenzylamine (2-HOBA) prevent DC activation, the ability of DCs to stimulate T cell proliferation and prevent hypertension. A major impetus for immune cell activation seems to be increased sympathetic outflow, stimulated by the central actions of angiotensin II. By lesioning the AV3V region of the forebrain of mice or inactivating the NADPH oxidase in the subfornical organ using Cre Lox technology, we have prevented the central actions of angiotensin II and found that this inhibits both T cell activetion and hypertension. Renal denervation likewise pre-

vents activation of DCs in the kidney and the accumulation of activated DCs in the spleen. Thus, the kidney seems to be a major site of DC activation in hypertension. In summary, we have identified a new mechanism underlying hypertension and a potential new therapy for this common and yet difficult to manage disease.

13.2
T REGULATORY LYMPHOCYTES IN HYPERTENSION

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In angiotensin II and aldosterone-infused mice, adoptive transfer of Treg blunted BP changes, vascular inflammatory cell infiltration, cytokine changes and oxidative stress increases induced by angiotensin II and by aldosterone. Endothelial dysfunction was also prevented when Treg were administered to angiotensin II or aldosterone-infused mice. Scurfy mice are deficient in Treg due to a mutation in the transcription factor forkhead box P3 gene. Angiotensin II induced endothelial dysfunction and oxidative stress in perivascular adipose tissue (PVAT) in mesenteric artery of wild-type T cell-injected *Rag1*$^{-/-}$, whereas these were exaggerated in Scurfy T cell-injected *Rag1*$^{-/-}$. Angiotensin II enhanced microvascular remodeling and stiffness in vehicle- and Scurfy T cell-injected *Rag1*$^{-/-}$. Angiotensin II increased monocyte chemotactic protein-1 expression in the vascular wall and PVAT, monocyte/macrophage infiltration and pro-inflammatory polarization in PVAT and the renal cortex, and T cell infiltration in the renal cortex only in Scurfy T cell-injected *Rag1*$^{-/-}$. Wild-type Treg co-injection with vehicle or Scurfy T cells prevented or reduced these effects of angiotensin II. In DOCA-salt rats, mineralocorticoid receptor blockade was associated with enhanced Treg action and reduced Th17. In conclusion, Treg counteract proinflammatory effects and microvascular injury by either angiotensin II or mineralocorticoids by modulating innate and adaptive immune responses. (CIHR Grants grants 82790 and 102606 and First Pilot CIHR Foundation Grant). Didion SP *et al*. Endogenous Interleukin-10 Inhibits Angiotensin II–Induced Vascular Dysfunction. Hypertension. 2009;54:619-624. Viel EC *et al*. Immune regulation and vascular inflammation in genetic hypertension. Amer J Physiol Heart Circ Physiol. 2010;298:H938-H944. Barhoumi T *et al*. T regulatory lymphocytes prevent angiotensin II-induced hypertension and vascular injury. Hypertension. 2011;57: 469-476. Kasal DAB *et al*. T regulatory lymphocytes prevent aldosterone-induced vascular injury. Hypertension 2012;59:324-330. Leibowitz A *et al*. Role of T regulatory lymphocytes in the pathogenesis of high-fructose diet-induced metabolic syndrome. Hypertension. 2013;61:1316-1321. Amador CA *et al*. Spironolactone Decreases DOCA–Salt–Induced Organ Damage by Blocking the Activation of T Helper 17 and the Downregulation of Regulatory T Lymphocytes. Hypertension. 2014;63:797-803.

13.3
RENAL IMMUNE CELLS AND HYPERTENSION

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Experiments in our laboratory have demonstrated that feeding a high NaCl diet to Dahl Salt-Sensitive (SS) rats results in a significant infiltration of T-lymphocytes into the kidney that is accompanied by the development of hypertension and renal disease. Since this disease phenotype closely resembles observations in patients, we performed studies to investigate the pathological role of immune cells in the kidney in hypertension and renal disease. Using zinc finger nuclease technology, we generated three mutant rat strains in the Dahl SS genetic background: one with a null mutation in recombination activating gene 1 (Rag1); a second with a null mutation in CD247 which encodes the CD3 zeta chain; and the third with an in-frame, 6 bp deletion in the Src Homology 2 domain of Sh2b3. As a result, we have rat strains with a full complement of T- and B-cells (the wild type Dahl SS), rats deficient in T cells with the Dahl SS genetic background (the CD247 mutant strain), rats deficient in T- and B-cells on the Dahl SS background (the Rag1 mutant rats), and rats with mutant Sh2b3 (a putative T-cell signaling molecule). Adding clinical significance to this work, mutations in CD247 and Sh2b3 are associated with hypertension and/or renal disease in patients. For example, studies on SS and Rag1 null rats fed a 4.0% NaCl diet for three weeks (n>5/group) demonstrated that infiltration of T-cells in the kidney following high salt was significantly blunted in Rag1 null rats ($1.7\pm0.6 \times 10^5$ cells/kidney) compared to the Dahl SS ($5.6\pm0.9 \times 10^5$ cells/kidney). Accompanying the reduction in T cells in the kidney, mean arterial blood pressure and urinary albumin excretion rat were significantly lower in Rag1 null mutants (158±3 mmHg and 60±16 mg/day, respectively) than in SS rats (180±11 mmHg and 251±37 mg/day). Finally, a histological analysis revealed that the glomerular and tubular damage in the kidneys of the SS rats fed high salt was also attenuated in the Rag1 mutants. Subsequent experiments demonstrated that mutation of CD247 and Sh2b3 also attenuates the infiltration of T-cells into

the kidney and the development of salt-sensitive hypertension and renal damage in Dahl SS rats. Together, these data demonstrate the pathological role of infiltrating T-cells in the development of salt-sensitive hypertension and renal disease and also provide potential mechanisms whereby genetic mutations in CD247 and Sh2b3 can affect blood pressure in humans. (NIH DK96859 and HL116264 & AHA 15SFRN2391002). Rudemiller NP, H Lund, JRC. Priestley, BT Endres, JW Prokop, HJ Jacob, AM Geurts, EP Cohen, and DL Mattson. Mutation of SH2B3 (LNK), a GWAS candidate for hypertension, attenuates Dahl SS hypertension via inflammatory modulation. Hypertension 65:111-1117, 2015.

13.4
INFLAMMATION AND RENAL FIBROSIS

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Interstitial fibrosis predicts organ failure in most tissues including the kidney. Macrophages have been implicated in the pathogenesis of kidney fibrosis, and type 1 angiotensin (AT_1) receptors are expressed on the macrophages that infiltrate the renal interstitium during the pathogenesis of hypertensive and normotensive kidney fibrosis. Recently, we examined the actions of AT_1 receptors on macrophages in progressive renal fibrosis. We found that deficiency of AT_1 receptors on macrophages exacerbates kidney fibrosis during angiotensin II-dependent hypertension or following unilateral ureteral obstruction (UUO). Macrophages isolated from obstructed kidneys of mice lacking AT_1 receptors solely on macrophages had heightened expression of pro-inflammatory M1 cytokines, including interleukin (IL-1), and kidney cross-transplant studies revealed that stimulation of IL-1 receptors in the kidney mediates the augmentation of renal fibrosis instigated by AT_1 receptor-deficient macrophages. In this session, we will report these findings and discuss signaling pathways through which the macrophage AT1 receptor may regulate macrophage differentiation during renal fibrogenesis. (VA Medical Research Service BX-000893). Reference: Zhang, J. D., Patel, M. B., Griffiths, R., Dolber, P. C., Ruiz, P., Sparks, M. A., Stegbauer, J., Jin, H., Gomez, J. A., Buckley. A.F., Lefler, W. S., Chen, D., and Crowley, S. D. (2014). Type 1 angiotensin receptors on macrophages ameliorate interleukin-1 receptor-mediated kidney fibrosis. Journal of Clinical Investigation 124,2198-2203.

13.6
HIGH SALT, GUT MICROBIOME AND BLOOD PRESSURE

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The presentation will cover the role of high salt on the gut microbiome, the gut immune system and its consequences on salt-sensitive hypertension.

13.7
T CELL SERUM AND GLUCOCORTICOID-REGULATED KINASE 1 (SGK1) AND HYPERTENSION

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T lymphocytes play a central role in the pathophysiology of hypertension by secreting cytokines that have a direct effect on the kidneys and vessel wall to both promote further increases in blood pressure and contribute to hypertensive end-organ damage. We previously showed that angiotensin II (Ang II) increases T cell production of the pro-inflammatory cytokine, interleukin 17A (IL-17A), and that mice deficient in IL-17A have blunted hypertension and reduced renal and vascular dysfunction. It was recently shown that salt enhances IL-17A production from CD4+ T helper cells via an SGK1 dependent pathway. SGK1 can be activated by multiple environmental stimuli including certain cytokines and Ang II. We hypothesized that SGK1 signaling in T cells is critical for T cells to promote hypertension and end-organ dysfunction. To test this hypothesis, we generated mice with T cell specific deletion of SGK1 ($SGK1^{fl/fl}$ x Tg^{CD4cre}). Loss of T cell SGK1 resulted in a 25 mmHg reduction in blood pressure in response to Ang II infusion. Importantly, renal and vascular inflammation was abrogated in these mice compared to SGK1 floxed control mice. Moreover, renal injury, assessed by albuminuria, was blunted in mice with T cell SGK1 deletion compared to control mice. Finally, vascular reactivity studies using isolated mesenteric vessels demonstrated that vessels from mice with T cell SGK1 deletion were protected from Ang II-induced endothelial dysfunction. Taken together, these studies demonstrate that T cell SGK1 is necessary for the full development of hypertension and hypertensive end-organ damage, and thus, this pathway may be a novel therapeutic target for hypertension. (NIH K08 HL121671, AHA Prevention SFRN, NIH F31 HL127986). References: Madhur et al. *Hypertension* 55: 500-507, 2010. Wu et al. *Nature* 496: 513-517, 2013. Kleinewietfeld et al. *Nature* 496: 518-522, 2013.

15.0: INFLAMMATION AND ATHEROSCLEROSIS

15.2
B CELL SUBSETS IN ATHEROSCLEROSIS

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B cells have emerged as important immune cells in murine atherosclerosis, regulating lesion development in a subset-dependent manner. B-2 B cells promote atherosclerosis through poorly defined mechanisms, and B-1 B cells exert atheroprotective effects largely through production of natural IgM antibodies (NAb). Natural IgM to oxidation-specific epitopes (OSE) that accumulate in atherosclerosis such as malondialdehyde (MDA) and phosphorylcholine (PC) present on oxidized low-density lipoprotein can antagonize oxLDL stimulation of macrophages limiting inflammation. B-1 cells are the major source of circulating IgM in mice. A human equivalent to the murine B-1 cell was recently identified through its ability to spontaneously produce IgM and data implicates this cell in producing IgM to OSE on LDL. Plasma levels of IgM to MDA-LDL are associated with less CAD and fewer CV events in humans. As such, unraveling the pathways that lead to B-1 cell production of IgM to OSE may enable targeted immune strategies to bolster production of IgM to OSE on LDL and protect from atherosclerosis in humans. Our work has identified the helix-loop-helix factor, Id3 as a key regulator of B-1 cells in mice and humans and implicated chemokine receptors as functionally important proteins on B-1 cells that are regulated by Id3. Using loss and gain of function studies in murine atherosclerosis models, we identified a key role for chemokine receptors on B-1 cells in promoting their atheroprotective function. Moreover, analyzing a human cohort with indices of coronary artery plaque volume and stability measured by intravascular ultrasound-virtual histology (IVUS-VH), we implicate chemokine receptors in B-1 cell atheroprotection in humans. Support: NIH R01 HL107490 and P01 HL055798. Reference: Rosenfeld SM, Perry HM, Gonen A, Prohaska TA, Srikakulapu P, Grewal S, Das D, McSkimming C, Taylor AM, Tsimikas S, Bender TP, Witztum JL, McNamara CA. B-1b Cells Secrete Atheroprotecive IgM and Attenuate Atherosclerosis Circ Res. 2015 Jul 17;117(3):e28-39.

NOTES

*Indicates Invited Speaker

*Indicates Invited Speaker

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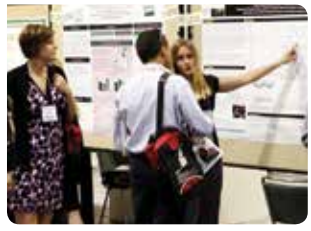

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