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The 10-20-30 training concept improves performance and health profile in moderately trained runners

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Gunnarsson TP, Bangsbo J. The 10-20-30 training concept improves performance and health profile in moderately trained runners. *J Appl Physiol* 113: 16–24, 2012. First published May 3, 2012; doi:10.1152/japplphysiol.00334.2012.—The effect of an alteration from regular endurance to interval (10-20-30) training on the health profile, muscular adaptations, maximum oxygen uptake ($\dot{V}O_{2max}$), and performance of runners was examined. Eighteen moderately trained individuals (6 females and 12 males; $\dot{V}O_{2max}$: 52.2 ± 1.5 ml·kg^{-1}·min^{-1}) (means ± SE) were divided into a high-intensity training (10-20-30; 3 women and 7 men) and a control (CON; 3 women and 5 men) group. For a 7-wk intervention period the 10-20-30 replaced all training sessions with 10-20-30 training consisting of low-, moderate-, and high-speed running (<30%, <60%, and >90% of maximal intensity) for 30, 20, and 10 s, respectively, in three or four 5-min intervals interspersed by 2 min of recovery, reducing training volume by 54% (14.0 ± 0.9 vs. 30.4 ± 2.3 km/wk) while CON continued the normal training. After the intervention period $\dot{V}O_{2max}$ in 10-20-30 was 4% higher, and performance in a 1,500-m and a 5-km run improved ($P < 0.05$) by 21 and 48 s, respectively. In 10-20-30, systolic blood pressure was reduced ($P < 0.05$) by 5 ± 2 mmHg, and total and low-density lipoprotein (LDL) cholesterol was lowered ($P < 0.05$) by 0.5 ± 0.2 and 0.4 ± 0.1 mmol/l, respectively. No alterations were observed in CON. Muscle membrane proteins and enzyme activity did not change in either of the groups. The present study shows that interval training with short 10-s near-maximal bouts can improve performance and $\dot{V}O_{2max}$ despite a ~50% reduction in training volume. In addition, the 10-20-30 training regime lowers resting systolic blood pressure and blood cholesterol, suggesting a beneficial effect on the health profile of already trained individuals.

high-intensity training; maximal oxygen uptake; blood pressure; plasma lipid profile; muscular adaptations

IT IS WELL ESTABLISHED that untrained individuals have major muscle adaptations and increase in maximum oxygen uptake ($\dot{V}O_{2max}$) and performance after a period of endurance training (10, 15, 24, 29, 31, 33, 35, 38, 39). On the other hand, for already trained individuals it appears necessary to intensify the training and include exercise bouts at an intensity close to or slightly above the intensity corresponding to $\dot{V}O_{2max}$, to obtain improvements in $\dot{V}O_{2max}$ and performance (11, 18, 25, 45, 48). Training at maximal and near-maximal exercise intensities seems also to be effective in creating muscular adaptations, such as increases in the activity of oxidative enzymes, and expression of Na^+-K^+ pump subunits and lactate and H^+ transporters, and endurance performance improvement in untrained individuals (5, 8, 33). Even well-trained individuals improved short-term performance after having carried out training with 30-s maximal running bouts for a 4-wk period, despite a 64% reduction in training volume (20). When combining training with 30-s sprints and, on separate days, aerobic high-intensity training consisting of 4 × 4 min with a heat rate of 90–100% of maximal heart rate (HRmax), long-term performance was also improved although the training volume was lowered by 25% (4). In these studies the improvements in performance were associated with a better running economy and an increased amount of Na^+-K^+ pump subunits α1 and α2. In accordance, running economy has been shown to be better after a period of interval (6, 14, 18, 43), plyometric (36, 42, 44, 46), and strength (32) training. Furthermore, studies on well-trained subjects, who either performed strength training (30) or increased their training intensity (13, 29), have reported increased Na^+-K^+ pump concentrations as determined by the [^{3}H]ouabain-binding technique. In contrast, Aughey et al. (3) did not find changes in the abundance of any of the Na^+-K^+ pump α isoforms when already trained subjects performed a period of intensified training. The lack of effect in the latter study may have been a result of the exercise intensity being below the one corresponding to $\dot{V}O_{2max}$. Nevertheless, the changes in the expression of Na^+-K^+ pump may affect performance, since Nielsen et al. (34) observed that elevated levels of Na^+-K^+ pump α_1- and α_2-subunits after 8 wk of knee-extensor training at supramaximal exercise intensities were associated with a reduced muscle interstitial K^+ concentration during exercise as well as better performance during intense exercise (34). In addition, other muscle ion transport proteins, such as the Na^+/H^+ exchanger isoform 1 (NHE1) and monocarboxylate transporters 1 and 4 (MCT1 and MCT4), facilitating lactate and H^+ exchange across the muscle membrane, have been shown to be changed with intense training and may have contributed to the improved short-term performance (7, 20–22, 33). It is, however, unclear whether training using 10-s near-maximal sprints has the same effect as 30-s intervals and whether combining aerobic and anaerobic training (19), i.e., maintaining a relatively high HR during training, can affect $\dot{V}O_{2max}$ and performance and lead to adaptations in the trained muscles. Such type of training is performed in the 10-20-30 concept where the participant in a 5-min period is alternating between low speed for 30 s, moderate speed for 20 s, and high-speed running (>90% of maximal speed) for 10 s.

It is clear that physical activity has a significant impact on the health profile in untrained individuals. Thus a typical response for a sedentary individuals to a period of endurance training is a reduction in blood pressure (BP) and lowering of the blood cholesterol levels (37). However, less is known about the effect of intense intermittent training. In a recent study Nybo et al. (35) found in untrained individuals a lowering in systolic BP after 12 wk of interval training (40 min/wk at an

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intensity corresponding to 95% of HRmax), but no change in diastolic BP and resting HR was observed. In contrast all variables were lowered in a group performing endurance training for 150 min at 80% of HRmax per week. The blood lipid profile, expressed as a ratio between total- and high-density lipoprotein (HDL) cholesterol, did not change in the interval group whereas there was a 15% reduction in the endurance training group. The difference may be related to the shorter training duration in the interval group. In a study by Kraus et al. (23), 111 sedentary overweight men and women with mild to moderate dyslipidemia were randomly assigned to either a control group or training group for 8 mo. In two of the training groups (moderate intensity) participants either jogged for 19 (low amount) or 32 (high amount) km/wk at 65–80% of $\dot{V}O_{2max}$, and in a third group participants walked for 19 km/wk at 40–55% of $\dot{V}O_{2max}$ (low amount; low intensity). Only the high amount, moderate-intensity training group lowered the concentration of low-density lipoprotein (LDL) and raised the concentration of HDL, suggesting that moderate-, but not low-, intensity training can have beneficial effects on the lipoprotein profile. In a recent study Williams (49) showed that exercise intensity was inversely associated with the prevalence of elevated BP and blood cholesterols independent of cardiorespiratory fitness and amount of exercise, suggesting that the higher the exercise intensity the greater the health benefits. However, it is unclear whether training at near-maximal intensity can affect the health profile of already trained subjects.

Thus the aim of the present study was to test the hypothesis that 7 wk of 10-20-30 training can improve endurance performance, cardiovascular fitness, and health profile as well as induce muscular adaptations in already trained subjects.

METHODS

Subjects

Eighteen moderately trained subjects (12 men and 6 women) with an age, height, weight, and $\dot{V}O_{2max}$ of 33.8 ± 1.6 yr, 178.8 ± 2.1 cm, 75.2 ± 3.5 kg, and 52.2 ± 1.5 ml·kg^{-1}·min^{-1}, respectively, participated in the study. The subjects were divided into a group training after the 10-20-30 concept (10-20-30; $n = 10$) (see below) and a control group (CON; $n = 8$). Groups were matched by $\dot{V}O_{2max}$ (52.2 ± 2.4 and 52.3 ± 2.0 ml·kg^{-1}·min^{-1}, respectively) and performance in a 5-km run (23.03 ± 1.06 and 23.03 ± 1.25 min, respectively). Furthermore, groups did not differ in age, weight, and body mass index, and there were 3 female runners in each group. All participants were fully informed of experimental procedures and any discomforts associated with participating in the study before signing a written informed consent. This study conformed to the code of Ethics of the World Medical Association (Declaration of Helsinki) and the Title 45, U.S. Code of Federal Regulations, Part 46, Protection of Human Subjects, Revised November 13, 2001, and was approved by the Ethics Committee of Copenhagen and Frederiksberg communities.

Experimental Design

In a 7-wk intervention period the 10-20-30 training group trained by the 10-20-30 training concept replacing all regular training sessions with three weekly 10-20-30 training sessions and CON continued with their regular endurance training (see *Training*). Four weeks prior to as well as before and after the intervention period the subjects underwent a series of tests: *1*) a treadmill test to determine $\dot{V}O_{2max}$ and maximal aerobic speed (MAS), *2*) a 1,500-m run, and *3*) a 5-km run (see *Testing*). In addition, on a separate day before and after the intervention period, subjects reported to the laboratory after an overnight fast and had a blood sample taken and BP measured. Furthermore, before, during (*week 4*), and after the intervention period, a biopsy from the vastus lateralis muscle was taken.

Training

Prior to the intervention period subjects had two to four weekly training sessions with a training volume of 27.3 ± 2.8 km lasting 137.5 ± 13.4 min with no difference ($P > 0.05$) between 10-20-30 and CON with regard to weekly training volume (30.4 ± 4.3 and 24.1 ± 3.6 km) or weekly duration of training (155.9 ± 19.9 and 119.2 ± 16.4 min), respectively.

The 10-20-30 training concept consisted of a standardized ~1.2 km warm-up at a low intensity followed by 3–4 × 5 min running interspersed by 2 min of rest. Each 5-min running period consisted of five consecutive 1 min intervals divided into 30, 20, and 10 s at an intensity corresponding to <30%, <60%, and 90–100% of maximal intensity (determined from 5-Hz GPS data), respectively. During the intervention period 10-20-30 had 3 weekly training sessions with a volume of 14.0 ± 0.6 km/wk (including warm-up). In the first 4 wk, 10-20-30 conducted three 5-min intervals and, in the remaining 3 wk, four 5-min intervals per training session. The total high-speed running amounted to 8.6 ± 0.5 min/wk during the intervention period. In CON the weekly training volume (24.8 ± 3.4 and 24.1 ± 3.6 km) and time spent (132.4 ± 16.6 and 119.2 ± 16.4 min) during the intervention period was the same as before the intervention period.

Testing

Prior to all testing subjects refrained from severe physical activity for at least 48 h and all testing was at least 3 h after ingestion of a meal. The subjects performed *1*) a 1,500-m run, *2*) a 5-km run, and *3*) an incremental test to exhaustion on a motorized treadmill (see below). The subjects were familiarized to all testing protocols on at least one separate occasion, and all tests were preceded by a thorough and standardized 15-min warm-up program. Calculation of the individual running speed (60% and 75% of MAS) was based on a $\dot{V}O_{2max}$ test performed within the last 2 wk prior to the study.

1,500-m run. The 1,500-m test consisted of 3.75 laps on a 400-m synthetic track. Subjects were wearing a HR monitor (Polar team system, Polar, Electro Oy) but did not wear watches during the 1,500-m and thus were not aware of running time. The running time for the first 400 m (1 lap) was given. Time to complete the 1,500 m was used as the test result.

5-km run. The 5-km test consisted of 12.5 laps on a 400-m synthetic track. Subjects were wearing a HR monitor (Polar team system, Polar, Electro Oy, Kempele, Finland) but did not wear watches during the 5-km run and thus were not aware of running time. The time for the first 1,000 m (2.5 laps) was given. The time to complete the 5-km was used as the test result.

Incremental test to exhaustion. The participants reported to the laboratory ~1 h before the $\dot{V}O_{2max}$ test. After 20 min of rest in the supine position, a muscle biopsy from the vastus lateralis muscle was collected through an incision made in the skin under local anesthesia (20 mg/ml lidocaine without norepinephrine) and a catheter (18 gauge, 32 mm) was placed in an antecubital vein. In addition, a HR monitor (Polar team system, Polar, Electro Oy) was placed on the subject and HR was recorded in 5-s intervals to determine peak HR. The treadmill test protocol consisted of 2 × 6 min running at 60 and 75% of MAS interspersed with 2 min of rest. After the two submaximal running bouts an incremental test to exhaustion was performed starting with 3 min at 75% of MAS. Hereafter running speed was increased by 1 km/h every minute until volitional fatigue. $\dot{V}O_{2max}$ was measured throughout the protocol with a breath-by-breath gas analyzing system (Oxycon Pro, Viasys Healthcare, Hoechberg, Germany) that was calibrated before each test. $\dot{V}O_{2max}$ was determined as the highest value achieved during a 30-s period. Criteria used for achievement of $\dot{V}O_{2max}$ were a plateau in $\dot{V}O_2$ despite an increased running

speed and a respiratory exchange ratio above 1.15. Blood samples during the test were collected in heparinized 2-ml syringes before and immediately after each of the running bouts and at exhaustion as well as 1, 3, and 5 min in recovery of the incremental test to exhaustion. Immediately after being taken, the blood sample was stored on ice and analyzed for blood lactate using an ABL 800 Flex (Radiometer, Copenhagen, Denmark).

Health Profile

Subjects reported to the laboratory between 6 and 10 A.M. on a separate day after an overnight fasting. After resting for at least 15 min in the supine position, BP was measured six consecutive times by an automatic upper arm BP monitor (M7, OMRON, Vernon Hills, IL) and fasting blood and plasma lipoproteins, hemoglobin, iron, glucose, myoglobin, creatine kinase, cortisol, insulin, and triglycerides were determined under standardized conditions.

Muscle Analysis

The muscle sample was immediately frozen in liquid N_2 and stored at −80°C. The frozen muscle tissue samples were weighed before and after freeze drying to determine the water content. After freeze drying, connective tissue, visible fat, and blood were carefully dissected away in the samples. Dissecting was done under a stereomicroscope with an ambient temperature of ~18°C and a relative humidity below 30%.

Muscle ion transport proteins. A part of the muscle sample taken at rest (~4–5 mg dry wt) was homogenized on ice in a fresh batch of buffer (10% glycerol, 20 mM Na-pyrophosphate, 150 mM NaCl, 50 mM HEPES, 1% Nonidet P-40, 20 mM β-glycerophosphate, 10 mM NaF, 2 mM PMSF, 1 mM each of EDTA and EGTA and 10 μg/ml each aprotinin and leupeptin and 3 mM benzamidine) with a Polytron 3100 (Kinematica) for not more than 30 s. After rotation end over end for ~1 h, the samples were centrifuged for 30 min at 17,500 *g* at 4°C, and lysates were collected as the supernatant. Protein concentrations were determined in the lysates using BSA standards (Pierce Reagents). The lysates were diluted to appropriate protein concentrations in a 6 × sample buffer (0.5 M Tris-base, DTT, SDS, glycerol, and bromphenol blue), and equal amount of total protein (5–15 μg in accordance with the antibody optimization) were loaded for each sample in different wells on 10% precasted Tris·HCl gels (Bio-Rad Laboratories, Hercules, CA). For comparisons, samples from the same subject were always loaded on the same gel. The gel electrophoresis ran for ~80–100 min with 55 mA and a maximum of 150 V per gel. Afterward proteins were blotted to a polyvinylidene difluoride membrane using 70 mA and a maximum of 25 V per gel in ~2 h. The membranes were incubated overnight with 20–30 ml of primary antibody diluted in either 2% nonfat milk [monoclonal Na^+-K^+ pump α1-subunit (~100 kDa), 1:500 dilution (C464.6, no. 05–369, Millipore); polyclonal α2-subunit (~100 kDa), 1:500 dilution (no. 07–674, Millipore); and monoclonal β1-subunit (~50 kDa), 1:1,000 dilution (MA3–930, Affinity BioReagents)] or 3% BSA [monoclonal NHE1 (~100 kDa), 1:500 dilution; polyclonal MCT1 (~43 kDa), 1:1,000 dilution; and polyclonal MCT4 (~43 kDa), 1:1,000 dilution (MAB3140, AB3538P, and AB3316P, Millipore)]. After being washed briefly in a Tris-buffered saline-Tween, membranes were incubated with secondary antibody for ~1 h at room temperature. The secondary horseradish peroxidase-conjugated antibodies used were diluted 1:5,000 in 2% nonfat milk or 3% BSA depending on the primary antibody (P-0447, P-0448, and P-0449, DakoCytomation). The membrane staining was visualized by incubation with a chemiluminescent horseradish peroxidase substrate (Millipore) immediately before the image was digitalized on a Chemi Doc MP (Bio-Rad Laboratories). Net band intensities were quantified using Image Lab (Image Lab v. 4.0, Bio-Rad Laboratories).

Data treatment. Double determinations were made for the muscle samples, i.e., the biopsies were divided and kept in two parts before freeze drying, resulting in two results for the same time point. The mean signal intensity of the two samples was used as the result for the individual time point. The intensity of the individual time points were divided with the mean intensity of the pre values within the group, to show the variation in the pre-biopsies.

Muscle enzymes. A part of the muscle sample (~2 mg of dry weight) was homogenized (1:400) in a 0.3 M phosphate BSA buffer adjusted to pH 7.7 and phosphofructokinase (PFK), hydroxyacyl-CoA dehydrogenase (HAD), and citrate synthase (CS) muscle enzyme activity was determined fluorometrically as described by Lowry and Passonneau. (27).

Statistics

Student's unpaired *t*-tests were used before the intervention period to compare subject characteristics ($\dot{V}O_{2max}$, 5-km performance, age, weight, and body mass index) as well as before and during the intervention to compare group differences in training volume and time. Changes in performance (5 km and 1,500 m), BP, resting HR, pulmonary $\dot{V}O_2$, fasting blood, and plasma samples (total cholesterol, LDL- and HDL-lipoproteins, hemoglobin, iron, glucose, myoglobin, creatine kinase, cortisol, insulin, and triglycerides) and enzyme activities were evaluated using a two-way ANOVA for repeated measures,

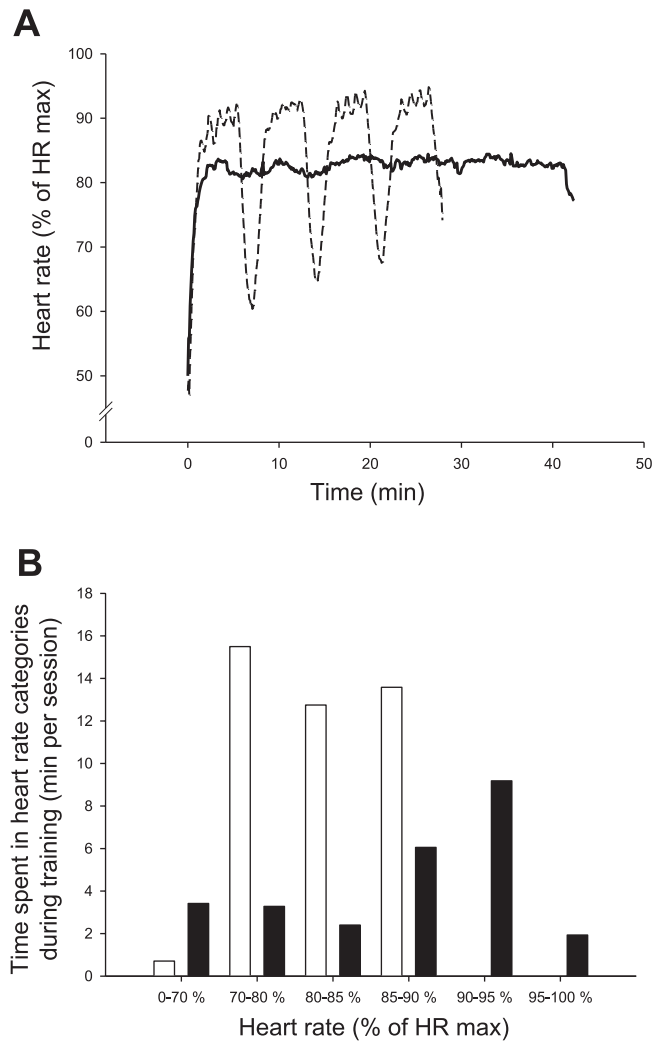

Fig. 1. Average heart rate during a representative training session for the 10-20-30 (dashed line) and control (CON; solid line) group during the intervention period (*A*) and time spent in various heart rate zones during a training session in the 10-20-30 (filled bars) and CON (open bars) group (*B*). HRmax, maximal heart rate. See *Training* for description of 10-20-30 group.

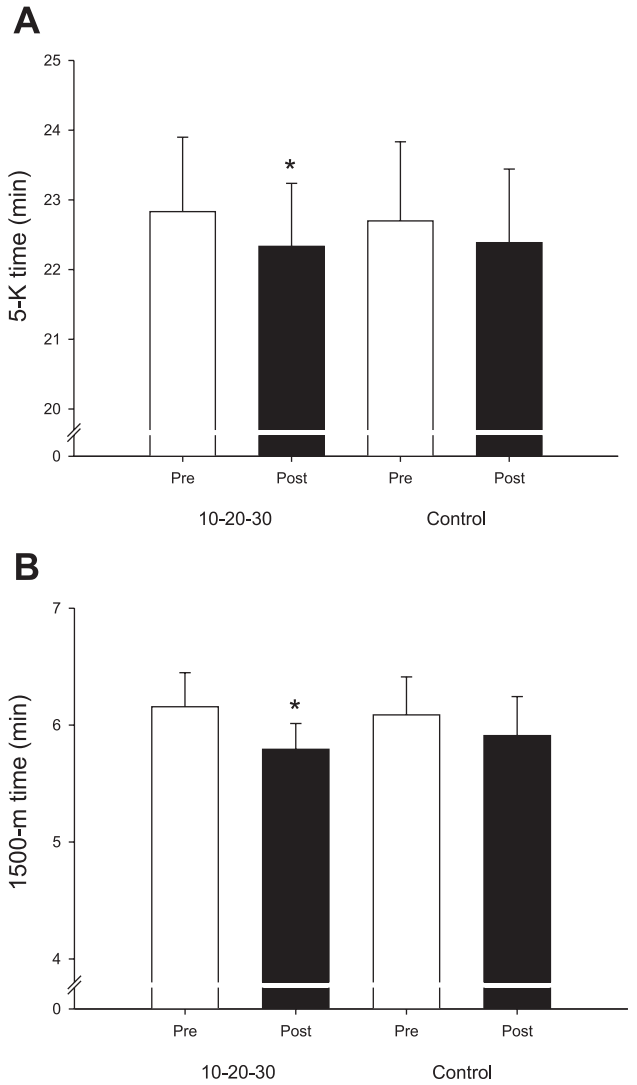

Fig. 2. Performance during a 5-km (*A*) and 1,500-m (*B*) before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and control (CON) group. *Different ($P < 0.001$) from Pre.

with time as one factor and group as the other factor. When a significant interaction was detected, data were subsequently analyzed using a Student-Newman-Keuls post hoc test. Changes in blood lactate during treadmill running before and after the intervention were evaluated using a two-way ANOVA for repeated measures with sample time as one factor and time (pre vs. post) as the other factor within each group. Group differences in blood lactate response within pre and post were evaluated using a two-way ANOVA with group as one factor and sample time as the other factor. Changes in muscle membrane transport proteins were evaluated using a one-way ANOVA for repeated measures with time (before and after 4 and 7 wk) as the factor. A significance level of $P < 0.05$ was chosen. Data are presented as means ± standard error of the mean (SE) unless stated otherwise.

RESULTS

HR Response to Training

Average and peak HR for 10-20-30 and CON were 85 ± 1 vs. 82 ± 2 and 96 ± 1 vs. 87 ± 2% of HRmax, respectively. The largest difference in the HR response to training in 10-20-30 and CON was time spent above 90% of HRmax, which amounted to 11.1 and 0 min corresponding to 43 and 0% of weekly training time, respectively (Fig. 1).

Performance

In 10-20-30, performance improved ($P < 0.01$) by 6% in the 1,500-m run (5.79 ± 0.22 vs. 6.16 ± 0.29 min) and 4% in the 5-km run (22.26 ± 0.90 vs. 23.07 ± 1.07 min) during the 7-wk intervention period whereas performance was not changed in CON (Fig. 2).

Pulmonary $\dot{V}O_2$

In 10-20-30 $\dot{V}O_{2max}$ was 4% higher ($P < 0.05$) after the intervention period (53.8 ± 2.3 vs. 51.6 ± 1.9 ml·kg^{-1}·min^{-1}), whereas no change was observed in CON (Table 1). $\dot{V}O_2$ at running speeds of 9.9 and 12.4 km/h before and after the intervention period was not different in either of the groups (Table 1).

Fasting Blood and Plasma Values

After the intervention period total cholesterol (4.3 ± 0.3 vs. 4.8 ± 0.4 mmol/l) and LDL cholesterol (2.7 ± 0.3 vs. 2.3 ± 0.3 mmol/l) was lower ($P < 0.05$) in 10-20-30, whereas no changes were observed in CON (Fig. 3). No changes were found in blood hemoglobin and plasma iron, glucose, myoglobin, creatine kinase, cortisol, insulin, and triglycerides during the intervention period in either of the groups (Table 2)

Resting BP and HR

In 10-20-30, systolic BP at rest was lower ($P < 0.05$) after the intervention period (122 ± 3 vs. 127 ± 4 mmHg), whereas no change was observed in CON (Fig. 4). Diastolic BP was the same before and after the intervention period in both 10-20-30 (76 ± 3 vs. 75 ± 3 mmHg) and CON (67 ± 4 vs. 65 ± 3 mmHg). Also resting HR was unaltered in 10-20-30 (55 ± 3 vs. 53 ± 3 beats/min) and CON (52 ± 2 vs. 49 ± 3 beats/min).

Muscular Adaptations

The Na^+-K^+ pump subunits α1, α2, and β1 as well as NHE1, MCT1, and MCT4 were not changed during the intervention period in either of the groups (Fig. 5). Likewise, no changes were observed in the CS, HAD, or PFK activity during the intervention period (Table 3).

Table 1. *$\dot{V}O_{2max}$ and $\dot{V}O_2$ during two submaximal running bouts before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and the control group*

	10-20-30		CON	
	Pre	Post	Pre	Post
$\dot{V}O_{2max}$				
l/min	3.98 ± 0.29	4.16 ± 0.31*	3.84 ± 0.22	3.91 ± 0.23
ml·kg^{-1}·min^{-1}	51.6 ± 1.9	53.8 ± 2.3†	52.3 ± 1.6	53.5 ± 1.6
$\dot{V}O_2$, ml·kg^{-1}·km^{-1}				
9.9 km/h	214 ± 7	214 ± 5	214 ± 8	213 ± 7
12.4 km/h	210 ± 5	213 ± 4	206 ± 7	210 ± 6

Values are means ± SE. $\dot{V}O_2$, oxygen consumption; $\dot{V}O_{2max}$, maximal oxygen consumption; CON, control. See *Training* for description of 10-20-30 protocol. *Different ($P < 0.05$) from Pre. †Different ($P < 0.01$) from Pre.

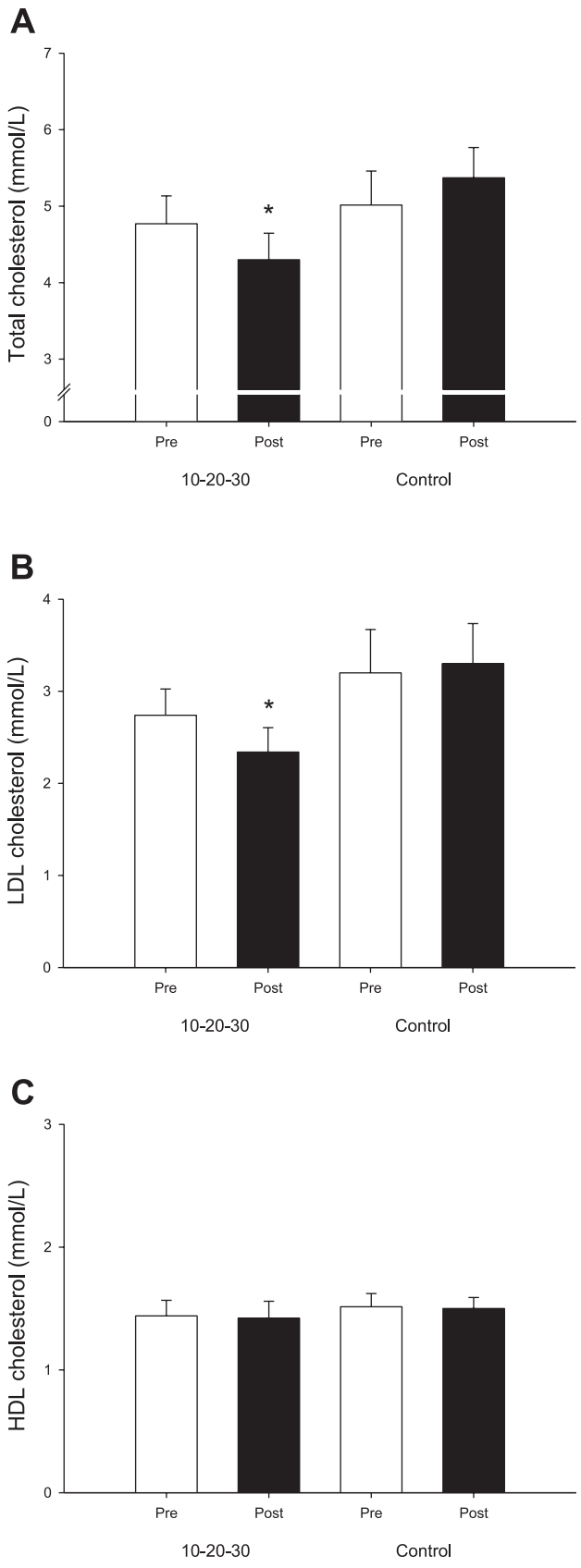

Fig. 3. Total cholesterol (*A*), low-density lipoprotein (LDL; *B*), and high-density lipoprotein (HDL; *C*) before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and control (CON) group. *Different ($P < 0.01$) from Pre.

Table 2. *Blood hemoglobin and plasma iron, glucose, myoglobin, creatine kinase, cortisol, insulin, and triglycerides after overnight fasting before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and the control group*

	10-20-30		CON	
	Pre	Post	Pre	Post
Hemoglobin, mmol/l	9.0 ± 0.1	8.9 ± 0.2	9.0 ± 0.3	9.0 ± 0.3
Iron, μmol/l	19.7 ± 2.1	20.9 ± 3.0	21.7 ± 1.8	21.3 ± 2.2
Glucose, mmol/l	5.1 ± 0.3	5.1 ± 0.2	4.9 ± 0.2	4.7 ± 0.1
Myoglobin, μg/l	51 ± 4.6	54 ± 3.2	52 ± 5	43 ± 4
CK, U/l	317 ± 147	140 ± 17	229 ± 49	122 ± 21
Cortisol, nmol/l	467 ± 63	466 ± 59	444 ± 23	463 ± 19
Insulin, pmol/l	35 ± 7	33 ± 4.3	31 ± 3	41 ± 4
Triglycerides, mmol/l	1.4 ± 0.4	1.2 ± 0.2	1.1 ± 0.3	0.9 ± 0.2

Values are means ± SE. CK, creatine kinase.

Blood Lactate Response to Treadmill Running

Before and after the intervention period, blood lactate at rest, after submaximal running, and after the exhaustive running was the same for both 10-20-30 and CON (Table 4). Likewise, no group differences within pre and post were observed.

DISCUSSION

The major findings of the present study were that after 7 wk of 10-20-30 training, with a ~50% reduction in training volume, $\dot{V}O_{2max}$ was elevated by 4% and performance in a 1,500-m and a 5-km run improved by 21 and 48 s, respectively. Furthermore, the 10-20-30 training led to a marked reduction in systolic BP as well as a lowering of total cholesterol and LDL-cholesterol.

The 7-wk period with 10-20-30 training led to an improvement in the 1,500-m and 5-km run of 6% and 4%, respectively, despite a 54% reduction in training volume. The major difference between the 10-20-30 training and the normal training was the speed during the 10-s intervals (>20 km/h), being much higher than the pace before the intervention period (10-14 km/h), which was similar to the speed during the 20-s and higher than the 30-s exercise periods in the 10-20-30

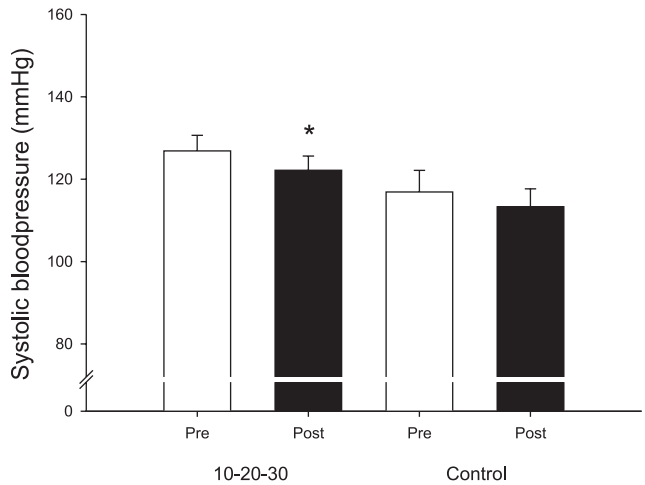

Fig. 4. Systolic blood pressure (mmHg) before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and the control (CON) group. *Different ($P < 0.05$) from Pre.

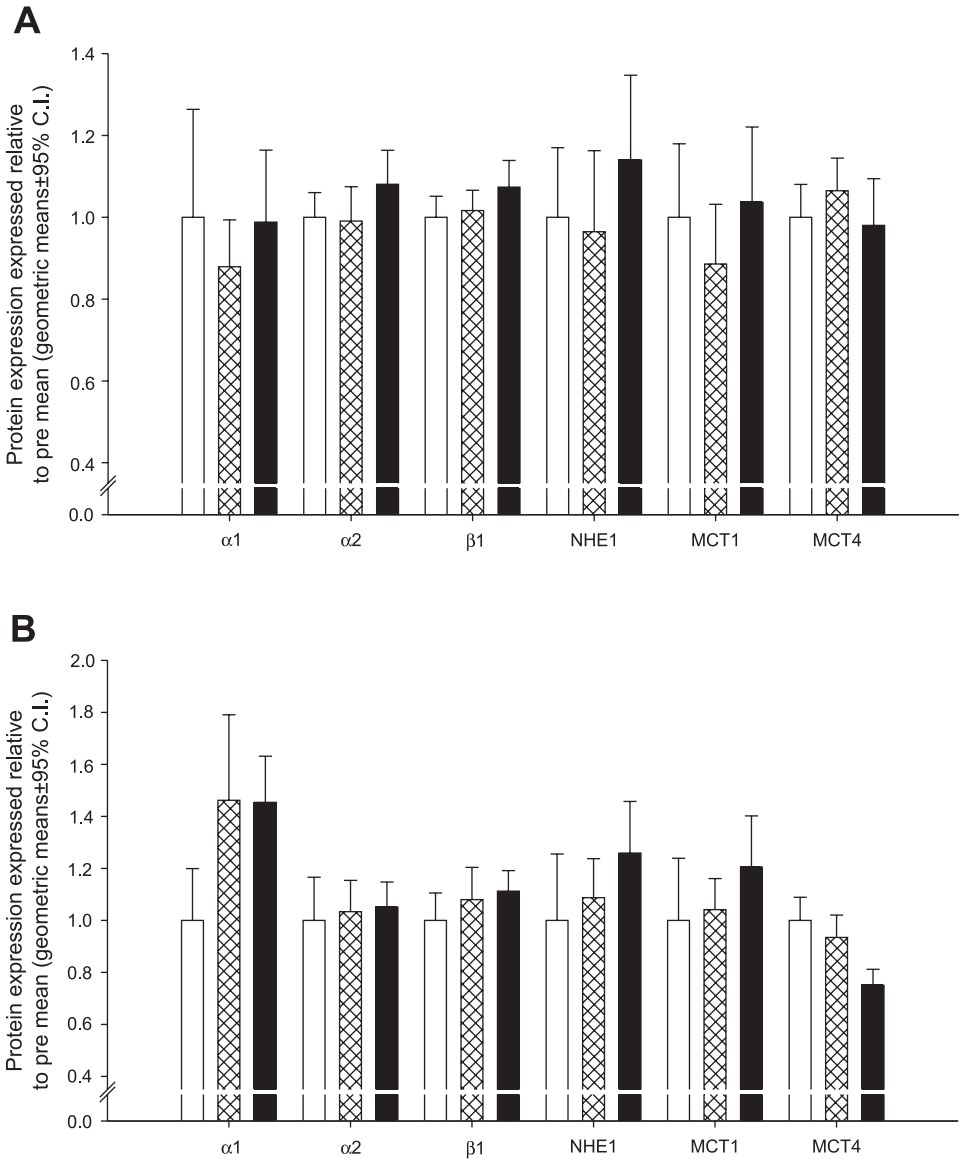

Fig. 5. Muscle Na^+-K^+ pump subunits (α1, α2, and β1), Na^+/H^+ exchanger 1 (NHE1), and monocarboxylate transporters 1 (MCT1) and 4 (MCT4) expression before (open bars), after 4 (hatched bars) and 7 (filled bars) wk of the intervention period for the 10-20-30 (*A*) and the control (*B*) group.

training. Iaia et al. (20) found an elevated short-term (0.5–2 min) performance, but no difference in the 10-km time when endurance-trained subjects for 4 wk replaced their normal training (~45 km/wk) with 30-s intervals at near-maximal speed (8–12 intervals per session) and reduced the amount of training by ~64%. In agreement with the present study, Bangsbo et al. (4) not only found improvement in short-term performance, but also in performance at a 10-km (37 vs. 36 min) after 6–9 wk with a reduced training volume of ~30% and adding repeated 30-s near-maximal running intervals as well as training sessions with four 4-min intervals at an intensity of 90–100% of HRmax. Other studies have shown 2–6% improvements in endurance performance in endurance-trained subjects when increasing the speed during training, but the speed has been around the one corresponding to the $\dot{V}O_{2max}$ and the amount of training has not been reduced (25, 26, 45, 47, 48). Taken together it appears that not only the 30-s near-maximal speed intervals are efficient in improving both

Table 3. *Citrate synthase, β-hydroxyacyl CoA dehydrogenase, and phosphofructokinase activity before (Pre) and after 4 wk (Mid), and 7 wk (Post) of the 7-wk intervention period for the 10-20-30 and the control group*

	10-20-30			CON		
	Pre	Mid	Post	Pre	Mid	Post
CS, μmol·g dry wt^{-1}·min^{-1}	34 ± 3	36 ± 4	32 ± 2	32 ± 4	31 ± 2	27 ± 2
HAD, μmol·g dry wt^{-1}·min^{-1}	18 ± 1	19 ± 1	17 ± 1	15 ± 2	16 ± 1	14 ± 1
PFK, μmol·g dry wt^{-1}·min^{-1}	193 ± 18	168 ± 17	182 ± 22	241 ± 32	201 ± 6	183 ± 49

Values are means ± SE. CS, citrate synthase; HAD, β-hydroxyacyl CoA dehydrogenase; PFK, phosphofructokinase.

Table 4. *Blood lactate at rest and after submaximal and exhaustive treadmill running before (Pre) and after (Post) the 7-wk intervention period for the 10-20-30 and the control group*

		Running Speed			Recovery		
	Rest	9.9 km/h	12.4 km/h	Exhaustion	1 min	3 min	5 min
10-20-30							
Pre	1.3 ± 0.2	1.8 ± 0.3	3.2 ± 0.7	10.3 ± 1.1	10.1 ± 1.1	10.2 ± 1.4	9.8 ± 1.2
Post	1.2 ± 0.1	2.0 ± 0.2	3.4 ± 0.5	10.7 ± 0.9	10.1 ± 0.7	10.5 ± 0.6	10.2 ± 0.6
CON							
Pre	1.0 ± 0.1	1.6 ± 0.2	3.1 ± 0.4	9.4 ± 0.9	8.9 ± 0.4	10.1 ± 0.7	9.9 ± 0.8
Post	1.4 ± 0.2	2.1 ± 0.3	3.3 ± 0.3	9.3 ± 0.5	9.5 ± 0.5	9.8 ± 0.6	10.0 ± 0.4

Values are means ± SE.

short- and long-term performance, but also, as demonstrated in the present study, that training with 10-s speed intervals have a major impact on performance.

In the present study $\dot{V}O_{2max}$ increased by 4% although the total volume was reduced by 54%. It may be explained by the HR being higher during the training than before the intervention despite the short intense intervals (~40 vs. ~0% of training time spent above 90% of HRmax; Fig. 1), suggesting that a high cardiac stress in combination with a reduction in training volume can elevate $\dot{V}O_{2max}$. A number of other studies have observed increase in $\dot{V}O_{2max}$ in trained subjects when performing intensified training but without a reduction in training volume (11, 18). In contrast, studies using 30-s near-maximal speed intervals separated by 3 min of recovery does not seem to lead to an increase in $\dot{V}O_{2max}$ (4, 20), suggesting that continuing the running after the high speed in the 10-20-30 training concept highly stimulates the cardiovascular system. On the other hand, the muscle oxidative system appears not to have been affected, since the activity of muscle CS and HAD was unchanged, which is in accordance with the findings in the study by Bangsbo et al. (4). This is in contrast to observed increases in oxidative enzymes with repeated short-term maximal exercise when performed with untrained individuals where most types of metabolic stress may lead to oxidative adaptations (8, 16, 28). The higher $\dot{V}O_{2max}$ may explain the better 5-km performance after the 10-20-30 intervention period. It was not due to a better running economy as it was unchanged at a speed close to the pace during the 5-km run (13.3 ± 0.4 km/h). Other studies have found a lower oxygen uptake during submaximal running after a period with 30-s near-maximal intervals (4, 20). Apparently, the longer duration of the intervals is important for the adaptations leading to a better running economy. Likewise, there was no change in the lactate response to submaximal exercise, suggesting that this is not of critical importance for the 5-km performance.

We observed no changes in muscle Na^+-K^+ pump subunits, NHE1, MCT1, and MCT4. In contrast, the studies using 30-s intervals for trained subjects have found increases in Na^+-K^+ pump subunits α1, α2, and β1, NHE1, and MCT1 (4, 20). It may be explained by the lower volume of high-speed running, since the weekly time in the 10-20-30 training with high-speed running was 150–200 s which is approximately two-thirds of that reported (>300 s/wk) in the other studies (4, 20). Another possibility is that greater metabolic stress and changes in ion homeostasis may be needed during training to obtain adaptations in the ion transport proteins. During the near-maximal repeated 30-s exercise intervals, muscle lactate rose to levels ~50 mmol/kg dry wt, muscle pH was lowered to ~6.98, and accumulation of potassium in the blood was ~6.2 mmol/l, likely reflecting concentrations above 10 mmol/l in the muscle interstitium (33). Such changes were probably significant less during the 10-s speed intervals used in the present study.

An interesting finding in the present study was that the 10-20-30 training period reduced the resting systolic BP in these already trained subjects. It is well established that a period of endurance and other types of training, such as soccer training, lowers systolic BP of untrained subjects (2, 24, 35, 40, 41), but to our knowledge this is the first study to show that intense training has this effect on systolic BP in trained subjects. In a recent study by Gosselin et al. (17), no difference in systolic and diastolic BP was found when comparing 20 min of normal endurance training (~70% of $\dot{V}O_{2max}$) with four different high-intensity training protocols. However, the intensities were significantly lower (<90% of $\dot{V}O_{2max}$) than in the present study (90–100% of maximal intensity). The underlying mechanism for the lowered BP is not clear but is likely multifactorial and involves modulation in the activity of the autonomic nervous system, neurohumoral and structural adaptations, as well as a reduction in systemic vascular resistance (9, 37). The lack of change in resting HR rate may suggest that the sympathetic outflow was not changed after the training period. Further studies are needed to elucidate the mechanism of the reduction in systolic BP. Nevertheless, the observed 5-mmHg decrease in systolic BP is of clinical relevance as a decrease of that magnitude is likely to reduce the risk of cardiovascular death by 10-15% (37).

A significant decrease in total cholesterol and LDL-cholesterol was also observed after the 10-20-30 intervention period. This finding suggests that the subjects obtained a better health profile, since high levels of total and LDL-cholesterol are associated with a higher risk of death and major adverse cardiovascular events. Thus a reduction in LDL of 1 mmol/l results in a 25% reduced cardiovascular risk, independent of baseline LDL levels (12). In accordance with the present study Randers et al. (41) also found a lowering of blood cholesterol when using soccer training as an intervention. On the other hand, in a number of studies the cholesterol levels were not changed, although the subjects were untrained (2, 24, 35). The diverging results may be related to differences in the training intensity. In the study by Krustrup et al. (24) the subjects performed moderate-speed running as the subjects in CON in the present study (~80% of HRmax). The subjects in the study by Nybo et al. (35) carried out repeated high-intensity running (2-min intervals), but at an intensity below the speed eliciting $\dot{V}O_{2max}$ ($\dot{V}O_{2max}$ ~95% of HRmax), and significantly lower than used in the 10-20-30 training (10 s at ~95% of maximal

speed). This could indicate that the improvement of the plasma lipid profile requires training at speeds above $\dot{V}O_{2max}$. However, further studies are needed to examine the cause of these changes in blood cholesterol.

In summary, the present study shows that the 10-20-30 training concept is efficient in increasing performance. Despite a ~50% reduction in training volume, $\dot{V}O_{2max}$ and performance were significantly elevated in moderately trained subjects without changes in running economy, muscle oxidative enzymes, and ion transport proteins. In addition, the 10-20-30 training led to reduced resting systolic BP and blood cholesterol, suggesting a better health profile for already trained subjects.

Perspectives

The 10-20-30 training concept is easy adapted in a busy daily schedule as it reduces time needed for training (~30 min including warm-up) and positively affects short- and long-term performance capacity. Furthermore, the present study is the first to show an improved cardiovascular health profile in trained subjects, which is in line with a prospective study by Albert et al. (1) suggesting that habitual vigorous exercise, as in the present study, diminishes the risk of death. The 10-20-30 concept is easy applicable for a variety of individuals ranging from the sedentary to the elite runner where the 10-20-30 concept may be used prior to a competition as the marked reduction in training volume in the present study (~50%) led to significant improvements in performance. Since the 10-20-30 concept deals with relative speeds and includes both low-speed running and 2-min rest periods, individuals with different fitness levels can train 10-20-30 together.

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS

Author contributions: T.P.G. and J.B. conception and design of research; T.P.G. and J.B. performed experiments; T.P.G. and J.B. analyzed data; T.P.G. and J.B. interpreted results of experiments; T.P.G. prepared figures; T.P.G. and J.B. drafted manuscript; T.P.G. and J.B. approved final version of manuscript.

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