Effect of sprint and strength training on glucoregulatory hormones: Effect of advanced age

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Abstract

The aim of this study was to examine the effect of high-intensity sprint and strength training (HISST) on glucoregulatory hormones in young (20 years) and middle-aged (40 years) men. Thirty-six moderately trained men participated as volunteers in this study. After medical examination, eligible subjects were randomly assigned to one of four groups according to their age: a young training group (21.3 ± 1.3 yrs, YT, $n = 9$), a young control group (21.4 ± 1.7 yrs, YC, $n = 9$), a middle-aged training group (40.7 ± 1.8 yrs, AT, $n = 9$), and a middle-aged control group (40.5 ± 1.8 yrs, AC, $n = 9$). YT and AT participated in HISST for 13 weeks. Before and after HISST, all participants performed the Wingate Anaerobic Test (WAnT). Blood samples were collected at rest, after warm-up (50% $\text{VO}_{2\text{max}}$), immediately post-WAnT, and 10 min post-WAnT. Before HISST, we observed significantly higher ($P < 0.05$) glucose concentrations in AT (5.86 ± 0.32 mmol.L⁻¹) compared to YT (4.24 ± 0.79 mmol.L⁻¹) at rest, and in response to WAnT (6.56 ± 0.63 mmol.L⁻¹ vs. 5.33 ± 0.81 mmol.L⁻¹). Cortisol levels were significantly higher ($P < 0.05$) in AT than in YT in response to WAnT (468 ± 99.50 ng.mL⁻¹ vs. 382 ± 64.34 ng.mL⁻¹). Catecholamine levels measured at rest and in response to WAnT rose in a similar fashion. After HISST, this “age effect” disappeared at rest and in response to exercise in the trained groups (YT and AT). Changes in hormone concentrations with intense training are due to adaptive changes in various tissues, especially in the skeletal muscle and liver in trained subjects. HISST may, at least in part, counteract the negative “age effect” on glucose metabolism.

Keywords: Hypothalamic–pituitary–adrenal axis, catecholamine, glucose metabolism, cortisol, Wingate-test, aging

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Introduction

Catecholamines are known to stimulate the regulation of glucose metabolism at rest and in response to exercise.^{1,2} However, changes in sympatho-adrenal activity and glucoregulatory hormone response are altered at rest and in response to severe stress by aging. In fact, Zouhal *et al.*³ showed that advancing age was accompanied by an alteration in hypothalamic–pituitary–adrenal (HPA) function in response to supramaximal exercise in endurance runners. This decline in catecholamine response was observed as soon as the fourth decade in national elite runners, whereas Zouhal *et al.*⁴ have shown that 35-year-old runners exhibited lower plasma adrenaline concentrations than 20-year-old runners, while noradrenaline levels were slightly higher. Furthermore, the “maximum Adrenaline/maximum Noradrenaline” ratio was significantly lower in the middle-aged group compared with the younger group.

Hence, it was suggested that a single decade in age may decrease the capacity of the medulla to secrete adrenaline.

Cox *et al.*⁵ have reported a sharp decline in resting insulin sensitivity in healthy elderly (60 yrs) compared with younger (20 yrs) subjects. Moreover, according to Zhao *et al.*⁶ the impaired response of HPA axis which appears with aging is associated with higher cortisol levels in older men. Colman *et al.*⁷ observed that resting glucose levels increased by 7% per decade. However, this increase was not significant in men (≥ 47 yrs),^{8,9} and appears more related to physical training status than to age. In fact, elderly subjects usually reduce carbohydrate intake and decrease their physical activity, which may alter glucose metabolism.¹⁰ Training level and type appears to be a key factor affecting the hormonal changes with aging.

Endurance training (bicycle ergometer training: 45 min exercise at 70% $\text{VO}_{2\text{max}}$) has been shown to decrease resting

catecholamine levels as well as systemic cortisol levels pre- and post-incremental exercise tests in sedentary males,¹¹ while no effect was seen in other studies.¹² In addition, nine months of vigorous endurance training (80% of maximal heart rate) or strength training has been shown to increase insulin sensitivity from 13% to 36% in older men (60 to 70 yrs) in response to a maximal graded test using the Bruce protocol.¹³ However, Houmard *et al.*¹⁴ found higher insulin sensitivity with high-intensity training (running at 65% to 80% $\text{VO}_{2\text{max}}$) compared to moderate-intensity training (running at 40 to 55% $\text{VO}_{2\text{max}}$) in 50-year-old men and women. Strobel *et al.*¹⁵ observed higher catecholamine responses during exercise in male sprinters compared to endurance athletes. For Ohkuwa *et al.*,¹⁶ the adrenaline levels measured in response to a 400-m race were two-times higher in sprinters compared with endurance-trained or untrained men. Zouhal *et al.*¹⁷ suggested also that most previous studies found that anaerobic training “can increase the capacity to secrete adrenaline more than aerobic training”. However, most of the transversal and the few longitudinal studies used either strength or sprint training programs, while, when combined with strength training, sprint training leads to a greater improvement in sprint performance.^{18–21} In addition, the training effect on glucoregulatory hormone response at rest and during exercise was usually studied with elderly subjects aged over 50 years, while the aging process begins to act at an early age (30–40 years). To assess the effect of training and advanced age on the glucoregulatory system, we compared the fluctuations in circulating levels of glucoregulatory hormones in young (20 years old) and middle-aged men (40 years old). We assumed that the intense sprint and strength training would reduce and/or counteract the negative effect of advancing age on glucoregulatory hormones.

Materials and methods

Participants

Thirty-six moderately trained men participated in this study. Informed consent was obtained from all individual participants included in the study. All procedures performed in this study were approved by the local Ethics Committee on Human Research (ECHR) of the University of Manouba (Tunisia) and in accordance with the ethical standards of the 1964 Helsinki Declaration. A medical examination was carried out before the start of training; intended to detect health problems. It included a medical evaluation with a history, a treadmill test to exhaustion with ECG and blood pressure monitoring, blood glucose exam (during fasting and after standard breakfast) and chest radiograph. Eligible subjects were non-smokers and did not use any specific medication. Subjects with body mass index $> 25 \text{ kg} \cdot \text{m}^{-2}$, diabetes mellitus, asthma, or cardiovascular disorders were excluded. In addition, no one had undergone surgery or blood donation during the previous six months. To assess the physical condition level of participants, we chose the Baecke questionnaire²² with the maximal oxygen uptake ($\text{VO}_{2\text{max}}$) as criterion measures.

All subjects were randomly assigned to one of four groups according to their age: a young trained group (YT,

$n=9$, 21 yr), a young control group (YC, $n=9$, 21 yr), a middle-aged trained group (AT, $n=9$, 40 yr) and a middle-aged control group (AC, $n=9$, 40 yr).

Participants who did not meet the inclusion criteria or become sick during test/training program were excluded from the study. In fact, during first testing period (P1), two participants dropped out the study (AT: $n=1$ and YC: $n=1$) due to vasovagal syncope during blood sampling. In addition, two others participants (AC: $n=1$ and YT: $n=1$) were excluded because of flu or injuries (foot and ankle). Consequently, the study only included 36 subjects.

Training protocol

The YT and AT groups participated in a specific high-intensity sprint and strength training (HISST) program four days a week for 13 weeks (Table 1). It consisted of two sessions of high-intensity sprint running and cycling, a strength session, and a low-intensity endurance session. This latter session represented 25% of total program and was recommended to facilitate body recovery and to avoid the risk of overtraining syndrome due to strenuous workout. In addition, it was adjusted at maximum 50% of $\text{VO}_{2\text{max}}$ to eliminate the “interference effect” of aerobic training on developing strength hormone response.^{23,24} The three intense training sessions, which represented 75% of total training, were performed in the morning and were separated by 48 h of rest. All sessions started with a standard warm-up (jogging, walking, and stretching for about 15 min) and finished with a period of rest (walking and stretching for about 15 min).

The training week began with a sprint running session on Mondays. The trained group performed 3–5 sets of 3–5 short bouts of sprint running distances (30–60 m) at maximum speed. The passive recovery was held every 2–3 min between each sprint and included walking and static stretching for all the participants. Forty-eight hours later, YT and AT performed the strength training session which included 5–6 exercises targeting all major muscle groups (Table 1). Strength training exercises were carefully selected in order to improve strength endurance and to enhance the transfer of strength gains to sprint performance. The load used during exercise (% of 1 RM) was progressively increased from 40% to 65% of 1-RM, and increased by 5% of 1-RM per week according to the American college of Sports medicine Guidelines.^{25,26} The number of repetition was maintained at between 10 and 15 reps per sets and the number of sets increased from 3 to 4 during the training period. Hence, the training volume was increased progressively during HISST. A rest period of 3–5 min was permitted between sets (recommended by American College of Sports Medicine).²⁶ In order to adjust load during strength training session, we determined muscle strength using a one repetition maximum (1-RM) squat exercise in a Smith machine. After 48 h of rest, the trained groups performed sprint cycling sessions on a cycle ergometer. Each series comprised 3–5 repetition of 10–30 s. Each trial lasted 10–30 s (dependent on week of intervention) of all out exercise. Subjects recovered actively (at a power output corresponding to 50% $\text{VO}_{2\text{max}}$) for 3–5 min between each sprint.

Table 1 Sprint and strength training program.

	Week 1–3	Week 4–6	Week 7–9	Week 10–12	Week 13
Sprint running training					
Running distance (m)	25–30	30–40	40–50	50–60	50
Reps per set	3–5	3–5	3–5	3–5	3–5
Sets per exercise	3	4	4	4	4
Rest between reps (min)	2–3	2–3	2–3	2–3	2–3
Rest between sets (min)	6–8	6–8	6–8	6–8	6–8
Strength training					
Exercises: Squat with Smith machine (%1RM), (2) leg extension with leg extension machine, (3) leg curl, (4) calf raises over step, (5) push down with cable machine, (6) preacher with loads and bench press					
Load (% of 1 RM)	40	45–50	50–60	55–65	50
Reps per set	10–15	10–15	10–15	10–15	15
Blocks (number of sets)	3	3	4	4	3
Rest between sets (min)	3–5	3–5	3–5	3–5	3
Rest between blocks (min)	6–8	6–8	6–8	6–8	6
Sprint cycling training					
Cycling time (s)	10–15	15–20	20–25	20–25	20–25
Intensity (% of P_{peak})	80	80–90	90–95	100	90–95
Reps per set	3–5	3–5	3–5	3–5	3–5
Sets per exercise	3	4	5	5	4
Rest between reps (min)	3–5	3–5	3–5	3–5	3–5
Rest between sets (min)	6–8	6–8	6–8	10	6–8

Reps: repetitions, P_{peak} : maximal power determined during the force/velocity test.

Duration and intensity of sets, and recovery time, was adjusted individually with respect to the age and physical fitness of the subject.

Training data

A medical team composed of one coach, two sports doctors, and a nutritionist supervised every training session. The control groups were asked to maintain their typical physical activity (walking and jogging), which was less than 180 min per week. The nutritionist of the Military Health Center of Tunis prescribed a varied and balanced diet providing the necessary calories daily for both the 20- and 40-year-old men. All of the participants were asked to avoid high glycemic loads, saturated and trans fatty acids, caffeine, alcohol and a low fiber diet.

Training adherence has been determined from the rate of the participation (numbers included divided by the total number of eligible subjects); the rate of attendance (numbers completed all sessions divided by the numbers included); and the exercise intensity adherence (numbers completed the exercises at prescribed intensity divided by the numbers included). Adherence was expressed as proportion (%) in Table 2. Good adherence was defined as reaching at least an 80% adherence rate for each group. To guarantee adherence to the prescribed exercise intensity, heart rate (S810, Polar Instruments Inc., Oulu, Finland) was monitored continuously during exercise. In each session, the rate of perceived exertion was determined by Borg's 6–20 scale. Peak heart rate (PHR) obtained during sprint exercises should reach at least 90% of their predicted maximum age-related heart rate.

Table 2 Training adherence data by group

	YT	AT
N eligible	10	10
N dropout	1	1
N included	9	9
N completed all session	9	9
N completed all exercises at prescribed intensity	9	8
% participation rate	90	90
% attendance rate	100	100
% exercise adherence	100	89

N: the number of subjects, YT: young trained group, AT: middle-aged trained group.

Anthropometric measurements

Anthropometric parameters were measured on the morning of the first day. Measurements of body weight (kg) and height were taken from all participants. Body mass was measured to the nearest 0.1 kg, with the subject in light clothing and without shoes, using an electronic scale (Kern, MFB 150K100). Height was determined to the nearest 0.5 cm with a measuring tape fixed to the wall. Then, skin-folds were measured using a Harpenden caliper (Harpenden skinfold caliper, Sweden). The percentage of body fat was determined by the four skin-folds method.²⁷ Fat free mass (FFM) was calculated by subtracting the fat mass from the body mass. The standard error of estimate of the skin-fold method was set at 0.34.

Testing procedure

Before training, all subjects were familiarized with the experimental procedures in the laboratory. The testing

period was divided into two phases: before (P1) and after (P2) training. Each period lasted seven days. The second phase (P2) was performed 48 h after training cessation and finished seven days later. All tests were performed after a standard breakfast (2 h. postprandial). The breakfast comprised of 10 kcal·kg⁻¹, 55% carbohydrate, 33% lipids, and 12% protein.

On the first laboratory visit, all subjects performed a maximal graded test on a cycle ergometer Monark (Ergomeca, Bessenay, France) to determine the maximal oxygen uptake (VO_{2max}) using the same protocol as the study of Zouhal *et al.*³ Forty-eight hours later, all subjects performed a force-velocity test on a cycle ergometer (Ergomedic 894 E, Monark Exercise), by applying the method of Vandewalle *et al.*²⁸ The test consists of a series of short 6 s maximum sprint of against loads increased by 2 kg after each trial, and a 5-min delay was inserted between each trial. The speed was recorded on a computer connected to the support of a photocell previously set on the bike wheel. The trial series continued until the velocity began to decrease. The maximum speed was used for calculating the peak load used later in the Wingate-test. 48 h later, all of the subjects performed the Wingate Anaerobic Test (WAnT) as described in the Zouhal *et al.*'s study.³ This test requires the subject to pedal a mechanically braked bicycle ergometer (Ergomedic 894 E, Monark Exercise) as fast as possible for 30 s against the previously determined load in the force/velocity test. The peak power (W_{peak}) was then determined from the load curve of the recording speed on the curve displayed on the computer. The average power of all values measured during WAnT was taken as the average power (W_{mean}). The heart rate was measured continuously during all of the tests using a heart rate monitor (S810, Polar Instruments Inc., Oulu, Finland).

Blood analysis

Upon arriving at the laboratory on the third day, a heparinized catheter (Insyte-W, 1.1 mm outer diameter [o.d.] × 30 mm) was inserted into an antecubital vein (sitting position). The heart rate and blood pressure measurements were taken using an automatic blood pressure monitor (Omron Model HEM-737AC [Omron Healthcare, Inc., Vernon Hills, IL]) as recommended by the European Society of Hypertension.

Prior to breakfast, the first venous blood samples (5 mL) were drawn to measure fasting glucose and insulin levels. The results were used to determine the "Homeostatic model assessment for insulin resistance" (HOMA-IR) index as described by Matthews *et al.*²⁹

$$\text{HOMA} - \text{IR} = (\text{fasting glucose [mmol.L}^{-1}\text{]}) \\ \times (\text{fasting insulin [mU.L}^{-1}\text{]})/22.5$$

Subsequently, participants consumed their standardized breakfast. Two hours later (between 8:00 and 9:00), four venous blood samples were drawn at four different times: at rest (after 20 min of sitting position on the bike), after the warm-up (15 min at 50% VO_{2max}), immediately after the WAnT, and after 10 min of recovery. During each sampling,

10 mL of blood was collected in tubes containing ethylenediaminetetraacetic acid (EDTA) to determine concentrations of plasma adrenaline [A], noradrenaline [NA], cortisol [C], insulin [Ins], and glucose [Glc]. Samples were centrifuged immediately (at 3000 rpm for 15 min at 4 °C) and were stored at -80 °C for later analysis.

To determine the peak blood lactate ([La]_{peak}), arterialized capillary blood was collected by pricking a fingertip at the third minute of recovery, since a delay is necessary for the transport of lactate from the muscle to the blood stream.

Biochemical assays

Blood lactate was determined by an enzymatic method using a lactate analyzer (Microzym, Cetrix, France). Plasma glucose concentrations were measured by the glucose oxidase method using an automated glucose analyzer (Beckman Instruments). Plasma insulin concentrations were determined by the radioimmunoassay (RIA) method using an Insulin-CT Kit (CIS-Bio International) and according to the rules specified by the SCPRI. Plasma catecholamine concentrations were measured by high performance liquid chromatography (Chromosystems, Thermofinnigan, France). Cortisol was analyzed using a RIA kit (Gamma Coat [125I] Diasorin, Stillwater, MN).

Statistical analysis

The results were expressed as mean values ± standard deviation (SD). SPSS for Windows (version 16.0, SPSS Inc, Chicago) was used for statistical analyses. On the basis of power analysis (expected standard deviation of residuals = 0.9 nmol.L⁻¹, desired power = 0.80 and an alpha error = 0.05) we determined that a sample size of $n=9$ per group would be sufficient to detect a 2.0 nmol.L⁻¹ increase in adrenaline and noradrenaline. A medium effect size (partial eta squared = 0.066) was determined. After testing for normal distribution (Kolmogorov Smirnov test), the differences within and between the groups were calculated using a two-way analysis of variance for repeated measurements. After confirmation of significant differences between groups and over time, a Newman-Keul's post hoc test was performed. Linear regression analyses were used to assess the independent contribution of anthropometric characteristics, contraction stress test performances and lactate to incident adrenaline and noradrenaline, cortisol, glucose, and insulin concentrations. The power of correlation analyses was calculated using the Pearson test (for parametric data). A value of $P < 0.05$ was accepted as the minimal level of statistical significance.

Results

Adherence

As shown in Table 2, all participants completed all training sessions (100% attendance rate). The YT group attended the greatest number of exercises completed at the prescribed intensity (100%), whereas AT had a lower exercise adherence (89%). In fact, the same protocol was used for all participants except for one middle-aged man ($n=1$, AT group)

who was allowed to perform two repetitions per set during the sprint running session (week 1 and week 2).

Anthropometric and physiological parameters

The results of the anthropometric and physiological parameters are presented in Table 3. HISST decreased ($P < 0.05$) body mass, with a slight increase in FFM in both YT and AT groups. VO_{2max} was significantly lower in the middle-aged group compared to the younger group at pre- (P1) and post-training (P2). VO_{2max} increased ($P < 0.05$) after HISST in both trained groups, with significantly ($P < 0.05$) higher levels compared with the control groups.

Results of WAnT are displayed in Table 4. Absolute W_{peak} and W_{peak} related to body mass increased significantly ($P < 0.05$) after training in both YT and AT groups. The significant ($P < 0.05$) effect of age for W_{peak} (W) at P1, was ameliorated following HISST ($P > 0.05$) when comparing trained groups (YT vs. AT).

After HISST, $[La]_{peak}$ change (Δ) was higher ($P < 0.05$) in YT compared to the AT group (Δ : 2.1 ± 2.1 vs. 1.8 ± 0.9). Although $[La]_{peak}$ was significantly ($P < 0.05$) higher in young participants compared to middle-aged participants at P1, the effect of age was absent post-training (P2) in both YT and AT groups ($P > 0.05$).

Plasma glucose concentration measured before (P1) and after (P2) HISST for all groups

Table 5 shows the plasma glucose concentration (Glc) determined before (P1) and after (P2) HISST at rest (Glc_0), after warm-up (Glc_w), immediately post- (Glc_{end}), and 10 min post-WAnT (Glc_{10}). Glc_0 , Glc_w , Glc_{end} , and Glc_{10} measured at P1 were lower ($P < 0.05$) in young participants (YT and YC) compared to middle-aged participants (AT and AC). After HISST (P2), no differences were observed ($P > 0.05$) between AT and YT groups. In addition, Glc_{end} and Glc_{10}

were lower at P2 ($P < 0.05$) in the trained compared to the untrained groups.

Plasma insulin concentration measured before (P1) and after (P2) HISST for all groups

Table 6 shows the plasma insulin concentrations (Ins) determined before (P1) and after (P2) HISST at rest (Ins_0), after warm-up (Ins_w), immediately post- (Ins_{end}), and 10 min post-WAnT (Ins_{10}). Ins_0 , Ins_w , Ins_{end} , and Ins_{10} measured at P1 were higher ($P < 0.05$) in young participants (YT and YC) than in middle-aged participants (AT and AC). After HISST, there were no differences ($P > 0.05$) between AT and YT. In addition, at P2, insulin levels were lower ($P < 0.05$) in the trained compared to the untrained groups.

Homeostatic model assessment for insulin resistance (HOMA-IR)

HOMA-IR results are represented in Table 7. After HISST, HOMA-IR was lowered ($P < 0.05$) in both trained groups (YT and AT).

Plasma cortisol concentration measured before (P1) and after (P2) HISST for all groups

Table 8 shows the plasma cortisol concentrations (C) determined before (P1) and after (P2) HISST at rest (C_0), after warm-up (C_w), immediately post- (C_{end}), and 10 min post-WAnT (C_{10}). C_0 , C_w , C_{end} , and C_{10} measured at P1 were lower ($P < 0.05$) in young participants (YT and YC) compared to middle-aged participants (AT and AC). In addition, HISST induced an increase ($P < 0.05$) in C_{end} in YT and AT at P2. Interestingly, this main effect was not present ($P > 0.05$) between YT and AT post-training (P2).

Table 3 Anthropometric characteristics and maximal oxygen uptake (VO_{2max}) of young and middle-aged male before and after training

		YT (n = 9)	YC (n = 9)	AT (n = 9)	AC (n = 9)
Age (year)	P1	21.3 ± 1.3 ^d	21.4 ± 1.7 ^e	40.7 ± 1.8	40.5 ± 1.8
	P2	21.6 ± 1.8 ^d	21.7 ± 1.5 ^e	40.8 ± 1.8	40.7 ± 1.0
Height (cm)	P1	179.6 ± 3.5	179.7 ± 6.4	178.6 ± 5.7	177.3 ± 4.4
	P2	179.7 ± 3.6	179.8 ± 6.5	178.9 ± 5.8	177.3 ± 4.8
Body mass (kg)	P1	70.8 ± 5.8	69.5 ± 7.3 ^e	72.1 ± 5.6	76.6 ± 3.9
	P2	68.5 ± 5.5 ^a	68.8 ± 8.7	71.6 ± 5.9 ^a	75.5 ± 4.8
Body fat (%)	P1	11.9 ± 1.7	11.3 ± 1.8	13.2 ± 1.1	12.4 ± 2.2
	P2	10.1 ± 1.8 ^{a,c}	11.7 ± 1.7	11.1 ± 1.3 ^{a,b}	12.4 ± 2.1
FFM (kg)	P1	63.4 ± 4.3 ^d	64.1 ± 4.5 ^e	60.3 ± 5.3	60.4 ± 3.2
	P2	64.6 ± 3.7 ^{a,c,d}	64.6 ± 5.7 ^e	62.1 ± 4.2 ^{a,b}	61.4 ± 4.4
VO_{2max} (mL.min ⁻¹ .kg ⁻¹)	P1	42.8 ± 5.2 ^d	43.8 ± 5.1 ^e	38.4 ± 8.8	38.5 ± 3.2
	P2	46.6 ± 5.4 ^{a,c,d}	42.1 ± 3.2 ^e	43.8 ± 9.5 ^{a,b}	40.1 ± 3.8

Data are means (±SD). FFM: fat free mass (kg), VO_{2max} : maximal oxygen consumption (mL.min⁻¹.kg⁻¹), YT: young trained group, YC: young control group, AT: middle-aged trained group, AC: middle-aged control group, P1: before training, P2: after training.

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 4 Physical performances and maximal lactate concentration of young and middle-aged male before and after training

		YT (n = 9)	YC (n = 9)	AT (n = 9)	AC (n = 9)
W_{peak} (W)	P1	1007.2 ± 126.6 ^d	1000.4 ± 311.9 ^e	887.2 ± 162	886.8 ± 102.2
	P2	1050 ± 123.6 ^{a,c}	943.8 ± 246 ^e	998 ± 135 ^{a,b}	823.5 ± 112.8
W_{peak} (W.kg ⁻¹)	P1	14.2 ± 2.1 ^b	14.4 ± 4.2 ^e	12.3 ± 2.1	11.5 ± 1.1
	P2	15.3 ± 1.8 ^{a,c}	13.8 ± 3.8 ^e	13.9 ± 1.3 ^{a,b}	11.1 ± 1.3
W_{mean} (W)	P1	573.9 ± 58.6 ^d	500 ± 92.5 ^e	430.4 ± 86.6	444.5 ± 37.1
	P2	599.8 ± 71.7	473 ± 80.6 ^e	566.8 ± 67.1 ^{a,b}	402.1 ± 80.4
W_{mean} (W.kg ⁻¹)	P1	8.1 ± 0.9 ^d	7.2 ± 1.2 ^e	5.9 ± 1.1	5.8 ± 0.4
	P2	8.7 ± 0.8	6.8 ± 0.9 ^e	7.9 ± 0.9 ^{a,b}	5.3 ± 2.4
[La] _{peak} (mmol.L ⁻¹)	P1	14.6 ± 2.1 ^d	13.9 ± 3.4 ^e	13.9 ± 2.7	13.2 ± 3.2
	P2	16.7 ± 2.3 ^{a,c}	14.3 ± 3.3 ^e	15.7 ± 2.6 ^a	13.3 ± 3.1
	Δ	2.1 ± 2.1 ^{c,d}	0.4 ± 1.7 ^e	1.8 ± 0.9 ^b	0.1 ± 1.9

Data are means (±SD) maximal power (W_{peak}), maximal power related to body mass (W_{peak} [W.kg⁻¹]), mean power (W_{mean}) in absolute values (W), mean power related to body mass (W_{mean} [W.kg⁻¹]), peak lactate concentration ([La]_{peak} [mmol.L⁻¹]), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2), the average change of [La]_{peak} per mmol.L⁻¹ (= P2 - P1).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 5 Plasma glucose concentrations (mmol.L⁻¹) of young and middle-aged male before and after training

		Glc ₀	Glc _w	Glc _{end}	Glc ₁₀
YT (n = 9)	P1	4.24 ± 0.79 ^d	5.56 ± 0.63 ^d	5.33 ± 0.81 ^d	4.22 ± 0.89 ^d
	P2	4.95 ± 0.64	5.66 ± 0.87	5.17 ± 0.49 ^c	5.01 ± 0.88 ^c
YC (n = 9)	P1	4.20 ± 0.77 ^e	5.34 ± 0.99 ^e	5.63 ± 1.06 ^e	4.18 ± 0.23 ^e
	P2	4.45 ± 0.37 ^e	5.43 ± 0.35 ^e	5.48 ± 0.35 ^e	5.84 ± 0.56
AT (n = 9)	P1	5.86 ± 0.32	6.22 ± 0.72	6.56 ± 0.63	5.50 ± 0.65
	P2	5.13 ± 0.66	5.85 ± 0.81 ^b	5.92 ± 1.00 ^b	4.97 ± 0.64 ^b
AC (n = 9)	P1	5.11 ± 0.33	6.08 ± 0.66	6.53 ± 0.58	5.23 ± 1.10
	P2	5.12 ± 0.54	6.88 ± 0.21	6.51 ± 0.68	5.80 ± 0.31

Data are means (±SD). Plasma glucose concentration at rest (Glc₀), after warm-up (Glc_w), at the end of Wingate test (Glc_{end}) and during recovery (Glc₁₀), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 6 Plasma insulin concentrations (mU.L⁻¹) of young and middle-aged male before and after training

		Ins ₀	Ins _w	Ins _{end}	Ins ₁₀
YT (n = 9)	P1	16.62 ± 8.49 ^d	14.10 ± 2.35 ^d	14.54 ± 5.17 ^d	10.09 ± 4.42 ^d
	P2	16.20 ± 3.12 ^c	13.72 ± 2.76 ^c	14.32 ± 7.80 ^c	10.15 ± 2.01 ^c
YC (n = 9)	P1	17.95 ± 7.10 ^e	13.83 ± 9.34	15.38 ± 7.19 ^e	11.60 ± 3.70 ^e
	P2	17.87 ± 9.19 ^e	14.28 ± 5.55	15.41 ± 8.30	11.06 ± 6.62 ^e
AT (n = 9)	P1	15.17 ± 8.99	13.09 ± 5.00	13.56 ± 6.69	10.68 ± 5.64
	P2	15.72 ± 6.82	13.56 ± 4.38 ^b	13.49 ± 8.11 ^b	10.70 ± 4.97
AC (n = 9)	P1	15.34 ± 1.01	13.99 ± 2.46	14.57 ± 0.71	10.10 ± 2.33
	P2	15.66 ± 0.76	14.50 ± 4.23	15.03 ± 0.88	10.19 ± 2.48

Data are means (±SD). Plasma insulin concentration at rest (Ins₀), after warm-up (Ins_w), at the end of Wingate test (Ins_{end}) and during recovery (Ins₁₀), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 7 Homeostatic model assessment for insulin resistance (HOMA-IR) of young and middle-aged male before and after training

		YT (n = 9)	YC (n = 9)	AT (n = 9)	AC (n = 9)
HOMA-IR	P1	2.26 ± 0.20	2.46 ± 1.80	2.32 ± 0.40	2.54 ± 0.30
	P2	2.02 ± 1.21 ^a	2.38 ± 2.20	2.10 ± 1.01 ^a	2.48 ± 1.40

Data are means (±SD). Homeostatic model assessment for insulin resistance (HOMA-IR) determined at rest (fasting) in young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 8 Plasma cortisol concentrations (ng.mL⁻¹) of young and middle-aged male before and after training

		C ₀	C _w	C _{end}	C ₁₀
YT (n = 9)	P1	256 ± 21.73 ^d	294 ± 80.05 ^d	382 ± 64.34 ^d	348 ± 80.79 ^d
	P2	266 ± 23.78 ^d	379 ± 86.29 ^{a,c,d}	479 ± 112.52 ^{a,c}	359 ± 101.01
YC (n = 9)	P1	222 ± 20.50 ^e	264 ± 33.39 ^e	322 ± 70.04 ^e	255 ± 25.92 ^e
	P2	233 ± 21.70 ^e	265 ± 32.72 ^e	326 ± 69.04 ^e	265 ± 65.11 ^e
AT (n = 9)	P1	360 ± 52.30	481 ± 113.47	468 ± 99.50	459 ± 133.01
	P2	367 ± 53.70 ^b	521 ± 69.69 ^{a,b}	543 ± 92.01 ^a	468 ± 57.28
AC (n = 9)	P1	352 ± 52.20	474 ± 101.55	514 ± 109.89	424 ± 145.46
	P2	353 ± 81.33	479 ± 90.65	494 ± 91.43	433 ± 103.74

Data are means (±SD). Plasma cortisol concentration at rest (C₀), after warm-up (C_w), at the end of exercise (C_{end}), and during recovery (C₁₀), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 9 Plasma noradrenaline concentrations (nmol.L⁻¹) of young and middle-aged male before and after training

		NA ₀	NA _w	NA _{end}	NA ₁₀
YT (n = 9)	P1	1.07 ± 0.35 ^d	2.68 ± 0.50	3.43 ± 0.55	2.22 ± 0.63
	P2	1.80 ± 0.20	2.47 ± 0.41	3.56 ± 0.62 ^c	2.01 ± 0.62
YC (n = 9)	P1	1.79 ± 0.49 ^e	2.35 ± 0.81 ^e	2.87 ± 1.00 ^e	2.13 ± 0.51 ^e
	P2	1.64 ± 0.37 ^e	2.36 ± 0.79 ^e	2.74 ± 0.60 ^e	2.18 ± 0.50 ^e
AT (n = 9)	P1	2.14 ± 0.45	2.79 ± 0.70	2.98 ± 0.35	2.59 ± 0.80
	P2	1.96 ± 0.37	2.44 ± 0.72	3.38 ± 0.66 ^{a,b}	2.16 ± 0.67
AC (n = 9)	P1	2.50 ± 0.89	3.37 ± 1.49	3.40 ± 0.79	3.29 ± 1.45
	P2	2.52 ± 0.90	3.81 ± 1.20	3.21 ± 1.14	2.84 ± 1.06

Data are means (±SD). Plasma noradrenaline concentration at rest (NA₀), at the end of the warm-up (NA_w) at the end of the Wingate-test (NA_{end}) and after 10 min recovery (NA₁₀), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Plasma catecholamine concentrations measured before (P1) and after (P2) HISST for all groups

Table 9 shows plasma noradrenaline concentrations (NA) determined before (P1) and after (P2) HISST at rest (NA₀), after warm-up (NA_w), immediately post-(NA_{end}), and 10 min post-WAnT (NA₁₀). NA₀ measured at P1 was

lower ($P < 0.05$) in young participants (YT and YC) compared to middle-aged participants (AT and AC). After HISST, no differences were observed ($P > 0.05$) between YT and AT, whereas the age effect remained statistically significant for the control groups ($P < 0.05$). Moreover, NA_{end} increased ($P < 0.05$) in AT at P2 and were significantly higher ($P < 0.05$) than in AC.

Table 10 Plasma adrenaline concentrations (nmol.L⁻¹) of young and middle-aged male before and after training

		A ₀	A _w	A _{end}	A ₁₀
YT (n = 9)	P1	1.07 ± 0.23 ^d	1.78 ± 0.42 ^d	2.72 ± 0.19 ^d	1.41 ± 0.28 ^d
	P2	1.61 ± 0.21	1.77 ± 0.41 ^d	3.09 ± 0.37 ^c	1.41 ± 0.27
YC (n = 9)	P1	1.29 ± 0.19 ^e	1.78 ± 0.31 ^e	2.67 ± 0.45 ^e	1.63 ± 0.33
	P2	1.31 ± 0.19 ^e	1.83 ± 0.34 ^e	2.70 ± 0.57 ^e	1.77 ± 0.60 ^e
AT (n = 9)	P1	1.53 ± 0.47	2.42 ± 0.80	3.21 ± 0.51	1.87 ± 0.62
	P2	1.65 ± 0.69	2.00 ± 0.61	4.38 ± 0.12 ^{a,b}	1.65 ± 0.46
AC (n = 9)	P1	1.51 ± 0.45	2.47 ± 0.74	3.59 ± 0.10	1.89 ± 0.53
	P2	1.70 ± 0.62	2.03 ± 0.59	3.19 ± 0.83	2.03 ± 1.31

Data are means (±SD). Plasma adrenaline concentration at rest (A₀), at the end of the warm-up (A_w) at the end of the Wingate-test (A_{end}) and after 10 min recovery (A₁₀), young trained group (YT), young control group (YC), middle-aged trained group (AT), middle-aged control group (AC), before training (P1), after training (P2).

^aSignificant differences from before and after training, $P < 0.05$.

^bSignificant differences between AT and AC, $P < 0.05$.

^cSignificant differences between YT and YC, $P < 0.05$.

^dSignificant differences between YT and AT, $P < 0.05$.

^eSignificant differences between YC and AC, $P < 0.05$.

Table 10 shows plasma adrenaline concentrations (A) before (P1) and after (P2) HISST at rest (A₀), after warm-up (A_w), immediately post-(A_{end}), and 10 min post-WAnT (A₁₀). A₀, A_w, A_{end}, and A₁₀ measured at P1 were ($P < 0.05$) lower in young participants (YT and YC) compared to middle-aged participants (AT and AC). For A₀, A_{end}, and A₁₀, no differences were found ($P > 0.05$) between YT and AT following HISST. In addition, HISST induced a significant increase ($P < 0.05$) in A_{end} in the AT group.

Discussion

The main result of this study is that 13 weeks of HISST induced a decrease in plasma glucose and insulin and an increase in catecholamine levels in response to WAnT in middle-aged and young trained men. Before HISST, the effect of age was observed in glucoregulatory hormones at rest and in response to exercise. However, HISST ameliorated the effect of aging as both training groups (AT and YT) had comparable adrenaline, glucose, and insulin levels at rest and at the end of exercise at P2.

Training effect on morphological characteristics, physiological characteristics, and physical performance

Researchers highlight the benefits of the combined strength and sprint training on body composition and sprint performance. In fact, Cadefau *et al.*³⁰ found decreased (3.3% and 4.1%) in sprint running time during 60-m and 300-m, respectively, in young trained men following combined sprint (30–500 m) and strength training (plyometric exercises). Sprint cycling performances were also increased in our study in young and middle-aged men following HISST. Previous studies^{31,32} found that combined sprint training is more efficient than sprint training performed alone in master athletes (≥45 yrs). Furthermore, we observed an increase in relative VO_{2max} following HISST, which is in line with previous investigators who reported an increase in cardiorespiratory fitness following high intensity interval training.³³ In this later study, it has been demonstrated that

two weeks of sprint interval training (6 sessions of repeated 30-s cycling efforts) increased the maximal activity of cytochrome C oxidase, buffering muscle capacity and muscle glycogen content in the trained group compared with endurance-trained athletes. Other evidence associated the aerobic benefit with sympathetic nervous system activation.³⁴

Training effect on plasma catecholamine levels

In recent study, intense training has been shown to increase plasma catecholamine levels at rest and during exercise in middle-aged men.³⁴ Chwalbinska-Moneta *et al.*¹¹ suggested that training at low and moderate intensity (≤70% VO_{2max}) leads to decline in basal levels of noradrenaline during incremental exercise test, while basal adrenaline levels did not change in young trained subjects. Similar results were also found in studies from our laboratory,^{3,4} in which the same exercise protocol was used, showing higher catecholamines in sprinters compared to endurance runners.

Interestingly, the increase of catecholamine levels in response to WAnT measured following HISST permits comparable levels between YT and AT. To our knowledge, this is the first study to show a decrease in the “age difference” between trained groups using combined strength and sprint training. Thus, this type of training seems to be efficient in increasing neuroendocrine hormones in middle-aged men in response to exercise, since a higher catecholamine level was accompanied by higher performance in sprinters compared to endurance trained athletes after an intense workout.³ Our findings can mainly be explained by the increase in the secretory capacity of the adrenal medulla causing the “Sports Adrenal-Medulla” phenomenon described earlier.^{3,35}

Training effect on plasma cortisol, glucose, and insulin levels

Higher glucose levels with lower insulin levels were observed in several studies in elderly (≥50 yrs) men at rest^{10,36,37} and in response to WAnT in middle-aged (35 yrs) trained men.³⁸ Similar results were also found in our

study at P1. Interestingly, this main effect of age was not seen after HISST (i.e. between YT and AT). In addition, the middle-aged trained group decreased their circulating glucose levels at rest and in response to exercise. Such findings may be due to several physiological adaptations (i.e. insulin sensitivity and glucose transport) to short-term intensive training.

The lower glucose levels measured in AT could be explained either by decreased hepatic glucose production (HGP) or increased muscle glucose uptake (MGU) following HISST. However, during intense exercise, HGP is primarily controlled by adrenaline, cortisol, and growth hormones.^{39,40} At rest and in response to exercise, AT had higher adrenaline and cortisol levels at P2 compared with P1. Therefore, HISST is more likely to increase HGP than decrease it. Training intensity plays a major role in stimulating hyperglycemic hormones. In fact, it has been demonstrated that intense training (30 s maximal effort exercise) has been shown to increase cortisol levels at rest⁴¹ and during exercise.⁴² In addition, most studies have shown that endurance training did not affect cortisol levels during moderate-intensity exercise.⁴³

The second probable mechanism resulted to HISST is the greater MGU. This phenomenon can be explained by an increase in glucose transport, under-action of insulin and an increase in insulin sensitivity. Data from this investigation are in agreement with some studies in reporting lower systemic insulin levels at rest and in response to WAnT in endurance-trained men compared with untrained men (21 yrs).⁴⁴

We therefore conclude that the lower glucose levels observed in the trained group, especially AT, are probably the result of changes to the high-affinity transmembrane receptor for insulin with intense training. Because it is difficult to assess insulin resistance under non fasting conditions,^{45–47} we only measured the basal levels of fasting glucose and insulin (before the pre-exercise meal) to determine the insulin resistance index (HOMA-IR) for each participant. Interestingly, HOMA-IR results decreased slightly in the trained groups after HISST. According to some authors, a short-term intense physical training has been shown to enhance insulin sensitivity in insulin-dependent tissues⁴⁸ in type 2 diabetes patients aged 48–68 years. It appears that the training intensity had a direct effect on insulin levels. According to Seals *et al.*⁴⁹ insulin levels was 8% lower after low-intensity training and 23% lower in older men and women (63 yrs) after high-intensity training than before training. Sandvei *et al.*⁵⁰ also found that high-intensity cycling training may be more effective than continuous running at low/moderate intensity, because it increases not only oxidative capacity but also insulin sensitivity in young subjects.

MGU is shown to be dependent on glucose transporter (GLUT4) content. During exercise, the non insulin-dependent GLUT4 are the main glucose transporters involved in MGU. According to Holten *et al.*⁵¹ strength training (30 min/day, 3 times/week) increased the protein content of GLUT4, insulin receptor density, and insulin action in skeletal muscles.

Furthermore, it has been shown that adrenaline allows GLUT4 translocation and increases glucose transport in the

absence of insulin.⁵² The excessive increase in adrenaline release could stimulate GLUT4 translocation and allow the massive entry of glucose into the membrane space.⁵² Thus, reduced plasma glucose concentration with training probably resulted in a higher rate of its diffusion by GLUT4. This result suggests the important role that adrenaline release plays in regulating glucose metabolism.

In conclusion, our study shows that combined sprint (2 session/week) and strength training (1 session/week) improved $\text{VO}_{2\text{max}}$, peak power output, and body composition in young and middle-aged men. Before the training period, glucose, cortisol, and catecholamines levels were higher in the middle-aged groups compared to the younger groups. HISST decreased glucose and insulin levels in the middle-aged trained group following WAnT and increased catecholamines and cortisol levels in response to the exercise in both trained groups. Interestingly, the main effect of age was not seen after HISST in the trained groups. The present finding suggests that intense training can counteract the negative effects of aging in trained men. Further research is required to better understand the contribution of hormone-stimulated glycogenolysis in liver and muscle cells in response to this type of training.

Authors' contributions: MS: conducted the experiments, performed the analysis of data, and wrote the manuscript, ABA: supervised the project, revised and, approved the manuscript, WK: conducted statistical data and interpretation, revised, and approved the manuscript, MVDS: made analysis and interpretation of data provided feedback on paper and approved manuscript, HZ: designed and supervised the study, performed the analysis, and approved the manuscript. All authors had revised and approved the final version to be submitted.

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DECLARATION OF CONFLICTING INTERESTS

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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