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Endocrine CONNECTIONS

HIIT produces increases in muscle power and free testosterone in male masters athletes

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Abstract

High-intensity interval training (HIIT) improves peak power output (PPO) in sedentary aging men but has not been examined in masters endurance athletes. Therefore, we investigated whether a six-week program of low-volume HIIT would (i) improve PPO in masters athletes and (ii) whether any change in PPO would be associated with steroid hormone perturbations. Seventeen male masters athletes (60 ± 5 years) completed the intervention, which comprised nine HIIT sessions over six weeks. HIIT sessions involved six 30-s sprints at 40% PPO, interspersed with 3 min active recovery. Absolute PPO (799 ± 205 W and 865 ± 211 W) and relative PPO (10.2 ± 2.0 W/kg and 11.0 ± 2.2 W/kg) increased from pre- to post-HIIT respectively ($P < 0.001$, Cohen's $d = 0.32–0.38$). No significant change was observed for total testosterone (15.2 ± 4.2 nmol/L to 16.4 ± 3.3 nmol/L ($P = 0.061$, Cohen's $d = 0.32$)), while a small increase in free testosterone occurred following HIIT (7.0 ± 1.2 ng/dL to 7.5 ± 1.1 ng/dL pre- to post-HIIT ($P = 0.050$, Cohen's $d = 0.40$)). Six weeks' HIIT improves PPO in masters athletes and increases free testosterone. Taken together, these data indicate there is a place for carefully timed HIIT epochs in regimes of masters athletes.

Key Words

- cortisol
- HIIT
- power
- steroid
- testosterone

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Introduction

Peak muscle power is an important determinant of athletic performance across the lifespan that declines with age (1) and is accompanied by a precipitous decline in serum testosterone (2). Both present a noteworthy impediment to the competitive masters athlete and negotiating this physiological decline requires a training program tailored for the older athlete. However, in contrast to the abundant evidence base for optimal training and performance for younger athletes, there is a paucity of comparable literature for the masters athlete, and a broad assumption that recovery profiles are analogous across the aging continuum. In the absence of age-specific exercise training guidelines, older athletes typically adhere to training routines comparable with their younger counterparts.

High-intensity interval training (HIIT) is a time-efficient strategy to achieve health (3) and performance (4) benefits in younger cohorts, which contradicts the recommended minimum physical activity threshold guidelines (150 min/week) (5). Six HIIT sessions has over a two- to three-week period improved muscle force in physically active young men and women (6), and six-week low-frequency HIIT improved peak oxygen uptake (VO_{2peak}) and quality of life in both sedentary and athletic aging men (7). There is some evidence that older persons take longer to recover from strenuous exercise than their younger counterparts (8), which can exceed five days (9). More recently, our research group identified that aging men take longer to recover from a single HIIT session

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than younger counterparts, highlighting HIIT programs that employ traditional (three sessions/week) regimens are likely to be overly strenuous for the aging athlete (10) and support the suggestion that athletes of different age groups may require differing recovery profiles. This may partly explain why endurance-focused masters athletes partake in high-volume aerobic training (11), and substituting their normal training regimens with comparatively minuscule volumes of HIIT would appear counterintuitive.

The relationship between testosterone and exercise in older males is a topic of ongoing debate and equivocal research findings (12, 13, 14). Conclusions have primarily relied on associative data from epidemiological studies (15, 16, 17). More recently, our group reported increased total testosterone (TT), but not free testosterone (free-T), in sedentary older men following six-week moderate aerobic training (18). Subsequently, we observed increased free-T following the addition of HIIT to this group (13). Conversely, Sylta and coworkers (19) reported a decrease in TT and free-T following four-week HIIT in well-trained young cyclists. These authors also reported that PPO increased concomitantly, which would appear counterintuitive given the pervasive belief that muscle power is positively influenced by testosterone. While Sylta and coworkers (19) observed decreased testosterone following HIIT in young athletes, the influence of HIIT on androgens of the master athlete remains unknown.

HIIT has been shown to improve maximal aerobic power in masters athletes (7), and PPO and free-T in untrained older participants (13, 20). Moreover, HIIT increased PPO, but decreased TT and free-T in young trained participants (19). However, the effect of HIIT on PPO, TT and free-T in masters athletes is currently unknown. One of the most important articles on HIIT exercise (20) highlighted the lack of HIIT studies in aging cohorts. Surprisingly, until a recent study of HIIT in sedentary aging men (21), there were no data on the impact of HIIT exercise on muscle power in aging men. With these aspects in mind, the present study set out to examine the influence of substituting normal exercise training with a six-week (nine sessions) low-volume HIIT program on PPO, TT, free-T, cortisol and the TT:cortisol ratio, in male masters athletes. We hypothesized that (i) six weeks of low-frequency HIIT would improve PPO compared with normal exercise training and (ii) systemic steroid hormones would be unchanged following low-frequency HIIT in masters athletes.

Materials and methods

Participants

Following familiarization with experimental procedures and approval to exercise by their general practitioner, participants provided written informed consent prior to enrolment to the study, which was approved by the University of the West of Scotland Ethics Committee. Experiments were performed in accordance with the ethical standards of the Helsinki Declaration (2013). Seventeen male masters' athletes (60 ± 5 years, with a stature of 173 ± 6 cm, body mass of 78 ± 12 kg, and peak oxygen uptake of 41 ± 6 mL kg/min (as previously determined {7})) completed the investigation. Participants were highly active exercisers and had been so for the previous >30 years. They consisted of masters competitors in water-polo, triathlon, track cycling, road cycling and distance running. Participants underwent two familiarization sessions before initial testing and arrived at the laboratory in the morning, following an overnight fast.

Phase A-B: capturing habitual exercise training

To allow for the comparison of HIIT with participants' normal training regimens, the study necessitated three distinct assessment phases (phase A, B and C), each lasting one week, which were separated by six weeks. Between assessment phases A and B, participants were instructed to maintain their habitual training practices, which were recorded by heart rate telemetry and training diaries. This included type, frequency, duration and intensity of exercise. Participant weekly average time spent <65% heart rate reserve (HRR), and ≥65% HRR totaled 214 ± 131 min/week and 67 ± 52 min/week respectively.

Phase B-C: high-intensity interval training (HIIT)

From phase B to C, participants underwent a supervised HIIT program. HIIT sessions were performed once every five days, for six weeks (nine sessions in total). Rationale for this program is provided by our previous work which identified five days was required for recovery of PPO following HIIT among older males (10). Each session consisted of 6 × 30-s sprints at 40% PPO (determined during familiarization) interspersed with 3 min active recovery on a cycle ergometer (Wattbike Ltd., Nottingham, UK). Sessions were conducted in groups of four to six participants and were the sole exercise performed during this period. To allow for comparison with existing

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literature, training intensities were compared with power achieved at VO_{2peak}. In the majority of cases, 40% PPO was greater than power at VO_{2peak}. In three cases, it exceeded 90% of power at VO_{2peak} (92; 96; 98%). Mean training intensity equated to 126±22% of power output at VO_{2peak}.

Peak power output assessment

The Herbert 6s cycling test (22) consisted of a 6s maximal sprint against constant resistance on an air-braked cycle ergometer (Wattbike Ltd., Nottingham, UK). For each subject, the damper resistance was set at 10. Participants completed a standardized 3min warm-up involving pedaling at 60rpm interspersed with three ~2s sprints. The test commenced from a standing start (i.e. not pedaling). Participants were verbally encouraged throughout the test to promote maximal effort. A recovery period of 5min was permitted between the warm-up and the test. Power output was calculated each second for the duration of the test and peak power over 1s was recorded.

Blood draws and analysis

Blood samples from each participant were collected at phase A, B and C, at 07:00–09:00h, 48–72h following the last exercise session as previously described (23, 24, 25, 26). Serum concentrations of TT, sex hormone-binding globulin (SHBG) and cortisol were determined by electrochemiluminescent immunoassay on the E601 module of the Roche Cobas 6000. Inter-assay CVs over a six-month period were 4.5, 2.4 and 4.2% for TT, SHBG and cortisol respectively. Analyses were carried out in a clinical pathology laboratory (Royal Glamorgan Hospital, Wales, UK). Free-T was calculated using the Vermeulen formula (27), which has been validated against equilibrium dialysis (28). The testosterone:cortisol ratio (T:C) was calculated by the following equation: $T{:}C = 100 \cdot (TT \div cortisol)$.

Statistical analysis

Following a Shapiro–Wilk test of normality and Levene's test for homogeneity of variance, a one-way analysis of variance (ANOVA) with *post hoc* Bonferroni correction was conducted to determine differences between phase A, B and C. Alpha level was set *a priori* at $P \leq 0.05$, and effect size (Cohen's *d*) was calculated for paired comparisons. Data are presented as mean±standard deviation (S.D.).

Results

Phase A to B: maintenance of high-volume aerobic training

Statistical power was confirmed as 0.994 for absolute PPO. There was no change to absolute PPO (766±163W and 799±205W; Cohen's $d=0.18$), relative PPO (9.7±1.8W/kg and 10.2±2.0W/kg; Cohen's $d=0.26$), TT (15.5±2.5nmol/L and 15.2±4.2nmol/L; Cohen's $d=0.09$), SHBG (45.3±12.5nmol/L and 48.5±16.9nmol/L; Cohen's $d=0.22$), free-T (7.2±1.1ng/dL and 7.0±1.2ng/dL; Cohen's $d=0.17$), and T:C (5.4±3.0 and 6.3±2.7 Cohen's $d=0.32$) from phase A to B respectively (all $P>0.05$). Cortisol decreased moderately from 345±138nmol/L to 278±114nmol/L ($P=0.038$; Cohen's $d=0.53$).

Phase B to C: substitution of high-volume aerobic training with HIIT

Absolute PPO (799±205W and 865±211W ($P<0.001$, Cohen's $d=0.32$)), and relative PPO (10.2±2.0W/kg and 11.0±2.2W/kg ($P<0.001$, Cohen's $d=0.38$)), were increased from pre- to post-HIIT respectively (Fig. 1).

Blood parameters are displayed in Figs 2 and 3. There was no change to TT (15.2±4.2nmol/L and 16.4±3.3nmol/L pre- and post-HIIT respectively ($P=0.061$, Cohen's $d=0.32$)) or SHBG (48.5±16.9nmol/L and 50.6±14.7nmol/L pre- and post-HIIT respectively ($P=0.204$, Cohen's $d=0.13$)) as a result of HIIT. However, there was a small increase in free-T (7.0±1.2ng/dL and 7.5±1.1ng/dL pre- and post-HIIT respectively ($P=0.050$, Cohen's $d=0.40$)), while a large increase in cortisol was observed (275±119nmol/L and 389±135nmol/L pre- and post-HIIT respectively ($P=0.01$ Cohen's $d=0.90$)). Therefore, T:C was moderately decreased following HIIT (6.3±2.7 and 4.7±1.9 pre- and post-HIIT respectively ($P=0.017$, Cohen's $d=0.69$)). No significant correlation existed between power profiles and any hormonal concentrations, at any phase, or delta change.

Discussion

The main finding of the present investigation was that replacing normal high-volume aerobic training with six-week low-frequency HIIT improved absolute and relative PPO in male masters athletes and increased free-T. These data provide preliminary evidence to inform optimization of training practices in masters athletes.

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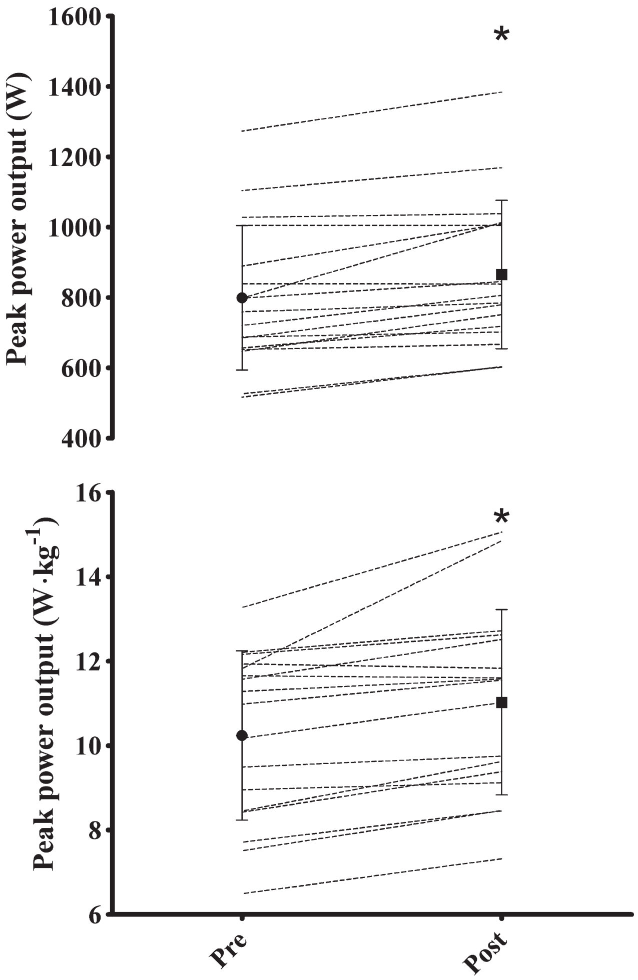

Figure 1
Absolute and relative peak power output in masters athletes pre and post six weeks of high-intensity interval training (HIIT). Dashed lines represent individual participants and marker and error bars represent mean ± s.d. *Denotes significantly greater than pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

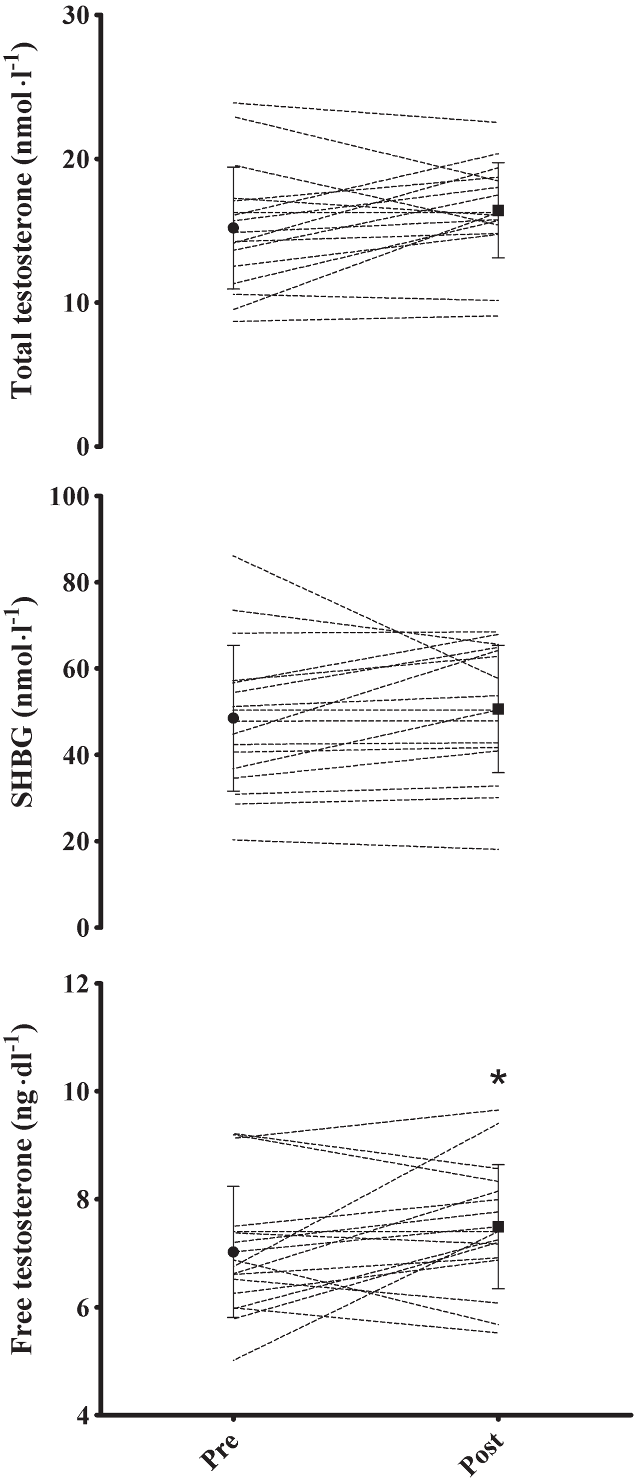

Figure 2
Total testosterone, sex hormone-binding globulin (SHBG), and free testosterone in masters athletes pre and post six weeks of high-intensity interval training (HIIT). Dashed lines represent individual participants and marker and error bars represent mean ± s.d. *Denotes significant difference from pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

In the present study, participants dramatically reduced their training volume from ~281 min/week to 4.5 min/week (excluding active recovery) or 27 min/week (including active recovery). To the authors' knowledge, this is the first study to investigate the impact of reduced volume HIIT on PPO in masters athletes. The ~8% increase in relative PPO is in line with previous investigations reporting improved performance following HIIT in young athletic populations (29, 30, 31). For example, Sheykhlouvand and coworkers (29) observed HIIT induced a 9.7–12.2% greater increase in PPO during the Wingate Anaerobic Test compared to a moderate-intensity training group in professional male canoe polo athletes. Moreover, Stoggl and Sperlich (32) noted a greater increase

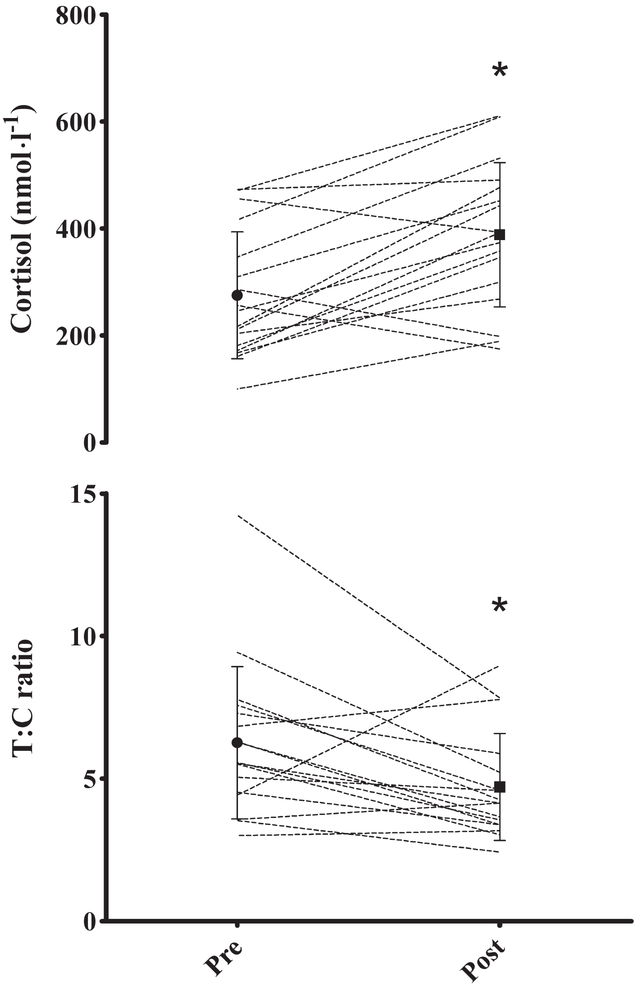

Figure 3
Cortisol, and the testosterone:cortisol (T:C) ratio, in masters athletes pre and post six-week high intensity interval training (HIIT). Dashed lines represent individual participants and marker and error bars represent mean ± s.d. *Denotes significant difference from pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

(+4.4 ± 2.8%) in PPO during an incremental test after HIIT, compared to high volume (−1.5 ± 4.9%), or threshold (+1.8 ± 4.8%), training in endurance athletes. These results were achieved despite the HIIT group training for ~66 h over nine weeks, compared to ~102 h and ~84 h in the high volume, and threshold group respectively. Similarly, Naimo and coworkers (33) observed an increase of ~12% PPO during the Wingate Anaerobic Test following HIIT compared to ~2% following continuous training in collegiate ice hockey players.

We recently demonstrated that sedentary older males increased TT (but not free-T) following six-week moderate aerobic training (18) and free-T following HIIT (13). Conversely, Lovell and coworkers (14) reported no increase in basal TT or free-T following 16 weeks of aerobic, resistance or combined, exercise training in a group of older men (~74 years). As both studies measured testosterone by immunoassay, and report similar CVs, the detection method is unlikely to explain differences in findings. We propose training intensity may mediate small differences in the testosterone response to exercise in older men, as we have now reported increased free-T in response to HIIT in both sedentary (13), and athletic older populations. We speculate that the training regimen employed by Lovell and coworkers (14) may not have achieved a threshold of exercise intensity to moderate the small changes in free-T demonstrated here. Moreover, as chronological age is known to dampen the physiological response to exercise (3) and participants in the study of Lovell and coworkers (14) were an average of ~12 years older, this may provide another account for differences between studies.

Adlercreutz and coworkers (34) previously suggested that a 30% reduction in the T:C ratio may be indicative of overtraining. In the present study, the T:C ratio was reduced by ~25%, possibly indicating greater stress and recovery time associated with HIIT. However, a reduction in training volume makes overtraining unlikely, and overtraining would typically be associated with a reduction in PPO, rather than an increase. Moreover, Fry and coworkers (35) observed increased strength and increased T:C ratio following overtraining in high-intensity resistance exercise, calling into question the predictive ability of this blood biomarker to detect overtraining.

One limitation of the present investigation is the single-arm prospective cohort design, which does not permit comparison of PPO or free-T improvements with a control group, or a comparative moderate intensity training group. However, the magnitude of improvement in PPO with reduced volume HIIT warrants further enquiry in masters athletes with implementation of a randomized control trial (RCT). Moreover, until HIIT-induced increases to free-T in the masters athlete are confirmed by equilibrium dialysis (the gold standard, but expensive and laborious), data in the present study remain preliminary.

The practical implication of the present study is that masters athletes can increase absolute and relative PPO, and free-T, by replacing high-volume aerobic training with low-volume HIIT. Our group has now demonstrated HIIT can improve PPO (as in the present study) and VO_{2peak} (7) in masters athletes. To progress this field, further research is required to confirm whether improvements in lab-based measures following HIIT translate to a performance advantage in masters competition.

In conclusion, six-week HIIT can induce large improvements in absolute and relative PPO, and small increases in free-T in male masters athletes. Taken together, this indicates there is a place for epochs of HIIT in training regimes of masters athletes, which may result in an improved anabolic environment. Given our previous work detailing that recovery of older adults takes five days to recover PPO following HIIT (10), carefully timed HIIT may be a pragmatic approach for maintaining athletic capability during periods of time restriction.

Declaration of interest

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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