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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 6-week programme 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 of 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⁻¹ and 11.0 ± 2.2 W·kg⁻¹) 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⁻¹ to 16.4 ± 3.3 nmol·l⁻¹ [$P = 0.061$, Cohen's $d = 0.32$]), whilst a small increase in free testosterone occurred following HIIT (7.0 ± 1.2 ng·dl⁻¹ to 7.5 ± 1.1 ng·dl⁻¹ 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

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 programme 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 ($150 \text{ min}\cdot\text{wk}^{-1}$) (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 weeks' low frequency HIIT improved peak oxygen uptake ($\text{VO}_{2\text{peak}}$) 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 than younger counterparts, highlighting HIIT programmes that employ traditional ($3 \text{ session}\cdot\text{wk}^{-1}$) 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 (12-14). Conclusions have primarily relied on associative data from epidemiological studies (2,15-17). More recently, our group reported increased total testosterone (TT), but not free testosterone (free-T), in sedentary older men following six weeks' moderate aerobic training (18). Subsequently, we observed increased free-T following the addition of HIIT to this group (13). Conversely, Sylta et al. (19) reported a decrease in TT and free-T following four weeks' HIIT in well-trained young cyclists. These authors also reported that peak power output (PPO) increased concomitantly, which would appear counterintuitive given the pervasive belief that muscle power is positively influenced by testosterone. Whilst Sylta et al. (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 ageing cohorts. Surprisingly, until a recent study of HIIT in sedentary ageing men (21), there were no data on the impact of HIIT exercise on muscle power in ageing men.** With these aspects in mind, the present study set out to examine the influence of substituting normal exercise training with a six week (9 sessions) low-volume HIIT programme 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⁻¹ [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 phase 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 $\geq 65\%$ HRR totalled $214 \pm 131 \text{ min}\cdot\text{wk}^{-1}$ and $67 \pm 52 \text{ min}\cdot\text{wk}^{-1}$ respectively.

Phase B-C: High Intensity Interval Training (HIIT)

From phase B to C, participants underwent a supervised HIIT programme. HIIT sessions were performed once every five days, for six weeks (nine sessions in total). Rationale for this programme is provided by our previous work which identified five days was required for recovery of PPO following HIIT amongst older males (10). Each session consisted of 6 x 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 4-6 participants and were the sole exercise performed during this period. To allow for comparison with existing literature, training intensities were compared with power achieved at $\text{VO}_{2\text{peak}}$. In the majority of cases, 40% PPO was greater than power at $\text{VO}_{2\text{peak}}$. In three cases, it exceeded 90% of power at $\text{VO}_{2\text{peak}}$ (92; 96; 98%). Mean training intensity equated to $126 \pm 22\%$ of power output at $\text{VO}_{2\text{peak}}$.

Peak power output assessment

The Herbert 6 s cycling test (22) consisted of a 6 s 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 3 min

warm-up involving pedalling at 60 rpm interspersed with three ~2 s sprints. The test commenced from a standing start (i.e. not pedalling). Participants were verbally encouraged throughout the test to promote maximal effort. A recovery period of 5 min 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 1 s was recorded.

Blood draws and analysis

Blood samples from each participant were collected at phase A, B, and C, at 07:00-09:00 h, 48-72 hours following the last exercise session as previously described (23-26). 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 (Burgess Hill, West Sussex, U.K.). Inter-assay CV 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 Vermueulen formula (27), which has been validated against equilibrium dialysis (28). The testosterone:cortisol ratio (T:C) was calculated by the following equation: $T:C = 100 \cdot (TT \div \text{cortisol})$

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 \pm standard deviation (SD).

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 ± 163 W and 799 ± 205 W; Cohen's $d=0.18$), relative PPO (9.7 ± 1.8 W \cdot kg $^{-1}$ and 10.2 ± 2.0 W \cdot kg $^{-1}$; Cohen's $d=0.26$), TT (15.5 ± 2.5 nmol \cdot l $^{-1}$ and 15.2 ± 4.2 nmol \cdot l $^{-1}$; Cohen's $d=0.09$), SHBG (45.3 ± 12.5 nmol \cdot l $^{-1}$ and 48.5 ± 16.9 nmol \cdot l $^{-1}$; Cohen's $d=0.22$), free-T (7.2 ± 1.1 ng \cdot dl $^{-1}$ and 7.0 ± 1.2 ng \cdot dl $^{-1}$; 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 ± 138 nmol \cdot l $^{-1}$ to 278 ± 114 nmol \cdot l $^{-1}$ ($P=0.038$; Cohen's $d=0.53$).

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

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

INSERT FIGURE 1 NER HERE

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 \pm SD. *Denotes significantly greater than pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

Blood parameters are displayed in Figure 2 and Figure 3. There was no change to TT ($15.2 \pm 4.2 \text{ nmol}\cdot\text{l}^{-1}$ and $16.4 \pm 3.3 \text{ nmol}\cdot\text{l}^{-1}$ pre- and post-HIIT respectively [$P=0.061$, Cohen's $d=0.32$]) or SHBG ($48.5 \pm 16.9 \text{ nmol}\cdot\text{l}^{-1}$ and $50.6 \pm 14.7 \text{ nmol}\cdot\text{l}^{-1}$ 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 ($7.0 \pm 1.2 \text{ ng}\cdot\text{dl}^{-1}$ and $7.5 \pm 1.1 \text{ ng}\cdot\text{dl}^{-1}$ pre- and post-HIIT respectively [$P=0.050$, Cohen's $d=0.40$]), whilst a large increase in cortisol was observed ($275 \pm 119 \text{ nmol}\cdot\text{l}^{-1}$ and $389 \pm 135 \text{ nmol}\cdot\text{l}^{-1}$ 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.

INSERT FIGURE 2 NEAR HERE

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 \pm SD. *Denotes significant difference from pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

INSERT FIGURE 3 NEAR HERE

Figure 3: Cortisol, and the testosterone:cortisol (T:C) ratio in masters athletes pre and post six weeks' high intensity interval training (HIIT). Dashed lines represent individual participants and marker and error bars represent mean \pm SD. *Denotes significant difference from pre-HIIT ($P \leq 0.05$) as determined by Bonferroni correction.

Discussion

The main finding of the present investigation was that replacing normal high volume aerobic training with six weeks' 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.

In the present study, participants dramatically reduced their training volume from $\sim 281 \text{ min} \cdot \text{wk}^{-1}$ to $4.5 \text{ min} \cdot \text{wk}^{-1}$ (excluding active recovery) or $27 \text{ min} \cdot \text{wk}^{-1}$ (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 $\sim 8\%$ increase in relative PPO is in line with previous investigations reporting improved performance following HIIT in young athletic populations (29-31). For example, Sheykhlovand et al. (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 ($+4.4 \pm 2.8\%$) in PPO during an incremental test after HIIT, compared to high volume ($-1.5 \pm 4.9\%$), or threshold ($+1.8 \pm 4.8\%$), training in

endurance athletes. These results were achieved despite the HIIT group training for ~66 hrs over nine weeks, compared to ~102 hrs and ~84 hrs in the high volume, and threshold group respectively. Similarly, Naimo and colleagues (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 weeks' moderate aerobic training (18), and free-T following HIIT (13). Conversely, Lovell et al. (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 et al. (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 et al. (14) were an average of ~12 years older, this may provide another account for differences between studies.

Adlercreutz et al. (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 et al. (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 weeks' 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

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