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TITLE PAGE

Title: Is salivary cortisol moderating the relationship between salivary testosterone and hand-grip strength in healthy men?

Running head: Moderating effect of cortisol

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Abstract

This study examined the moderating effect of cortisol (C) on the relationship between testosterone (T) and hand-grip strength (HGS) in healthy young men. Sixty-five males were monitored for salivary T, C and HGS before and 15 minutes after a short bout (5 × 6-second trials) of sprint cycling exercise. Sprint exercise promoted ($p<0.05$) positive changes in T (6.1±24.9%) and HGS (3.4±7.5%), but a negative C response (-14.4±33.1%). The T and C measures did not independently predict HGS, but a significant T × C interaction was found in relation to these outcomes. Further testing revealed that pre-test T and HGS were negatively associated ($p<0.05$), but only in men with high C levels. The exercise changes in T and HGS were also negatively related in men with low C levels ($p<0.05$), but no relationship was seen in men with high C levels. In summary, complex relationships between T and HGS emerged when considering C as a moderating variable. The pre-test combination of high C and low T levels favoured absolute HGS, whereas low pre-test C levels and a smaller T change were linked to larger HGS changes. These associations suggest that, in the current format, T is not necessarily anabolic to muscle strength in healthy young men. These complexities could explain some of the inconsistent T relationships with physical performance in lesser-trained male populations.

Key words

Testing; Stress; Endocrinology; Performance

Introduction

Often considered the primary androgen, testosterone (T) is known to exert both anabolic (i.e., muscle and bone growth) and androgenic (i.e., development of sex characteristics) effects (Wood & Stanton, 2012), although physiological elevations in T do not appear to be necessary for muscle growth to occur (West et al., 2010). Other physiological and psychological functions (e.g., behaviour, mood, neural activity, motor system outputs, cognition, cellular signalling) might also be supported by T and its active metabolites (Crewther et al., 2011a; Wood & Stanton, 2012). Subsequently, T could potentially contribute to physical performance and long-term adaptation via multiple mechanisms spanning a wide timeframe.

The reported associations between T and physical performance tend to be stronger and more consistent among elite-trained than sub-elite and untrained men (Crewther et al., 2011a); perhaps reflecting trainable features such as sport-specific experience (Ahtiainen et al., 2003) and baseline strength (Crewther et al., 2012). For example, a strong correlation ($r=0.92$) was found between T and squatting strength in very strong men (squatting >2 times their body mass [BM]), but in less strong men this relationship was weak ($r=0.35$) (Crewther et al., 2012). Alternatively, these results might be less about physical ability and more about coping and performing under stress. Crucially, untrained individuals can exhibit a larger neuroendocrine stress (e.g., cortisol [C]) response than trained individuals when exercising at the same workloads (Hackney, 2006).

One less explored perspective in sport and exercise is the moderating role of C, whereby C can influence T activity or T release via the motivational circuitry, psychological processing and feedback inhibition (Mehta & Prasad, 2016). In a behavioural domain, it has been demonstrated that T is positively related to dominance or aggression outcomes in men with low C levels, whereas no (or negative) relationships were found in high C men (Mehta & Josephs, 2010; Mehta & Prasad, 2016). These findings are applicable to the exercise testing of healthy men, not only because muscle performance and dominance are linked (Gallup et al., 2007), but any exercise protocol deemed to be stressful would likely induce large changes and subject differences in C availability. To our knowledge, no research has investigated this interplay between T and C in a healthy cohort of men performing high-intensity exercise.

This study examined the moderating effect of C on the T relationship with hand-grip strength (HGS) in healthy young men. To create a hormonal stress response, the men were assessed around a short bout of sprint cycling exercise (Goto et al., 2007; Obmiński et al., 1998). The following hypotheses were developed based on the literature presented; first, sprint exercise would acutely elevate T and C levels; second, the T and HGS measures taken (i.e., pre-test, changes) around the exercise stimulus would be unrelated; third, significant T and HGS associations will be identified once low C and high C men are considered separately.

Methods

Participants

Sixty-five healthy young men were recruited from a University campus (means ± SD: age 22.6±4.9 years, height 180.1±5.84 cm, body mass [BM] 78.8±12.0 kg). The men were injury-free, with no medical or health conditions that would influence the study outcomes. Low to moderate levels of physical activity were reported (i.e., 2-5 days a week, low to moderate intensity) involving jogging, cycling, weight training and some team-sport activities. The men were also questioned about medication and drugs taken in the last 6 months, but none were reported. Informed consent was obtained before the study commenced and ethical approval was granted from the Swansea University Human Ethics Committee.

Experimental procedures

The experimental study was completed after a familiarisation session. Briefly, salivary T, C and HGS were assessed before and 15 minutes after a short bout of sprint exercise. Testing was conducted between 10am and 3pm, as we anticipated no diurnal variation in the measured variables over this period (Hayes et al., 2012; Patel et al., 2004). A control session with no warm-up or sprint exercise was completed by a sub-group of 15 men. This session was performed at a similar time of day (± 1 hr), as per the sprint exercise, and both sessions were randomised (>3 days separation) to reduce any order effect. Each participant was instructed to maintain the same food intake on each day of testing, and to refrain from eating or drinking hot fluids 2 hours beforehand (Crewther et al., 2014). No exercise was performed in the 24 hours preceding each session to eliminate the confounding effects of muscle fatigue.

Sprint cycling exercise

Testing began with a 15-minute rest period in a seated position. Following a 2-minute warm-up, the 5-minute sprint exercise protocol was performed on a Monark cycle ergometer (824E, Sweden) with a load equalling 7.5% of BM. In total, 5 × 6-second sprint trials were completed at the predetermined load with 54 seconds of slow pedalling (without load) between each sprint trial. The participants remained seated throughout testing and strong verbal encouragement was given by the lead investigator. These protocols were based on prior research to ensure a hormonal stress response (Crewther et al., 2011b; Goto et al., 2007; Obmiński et al., 1998). A 2-minute cool down was performed after the last sprint without load. Sprint testing on a cycle ergometer can produce similar salivary T responses in healthy men, independant of training experience (Crewther et al., 2014).

Salivary hormone testing

Salivary hormones are thought to reflect blood-free hormones (Crewther et al., 2012), thereby representing less than 10% of the total hormone fraction in blood. Saliva samples (~1ml) were taken by passive drool 5 minutes before and 15 minutes after exercise to coincide with expected hormonal changes in this fluid (Edwards & Casto, 2013). All samples were stored in a -30°C freezer. The samples were analysed in duplicate using an immunoassay kit (Salimetrics LLC, USA). The detection limit for the T and C assays were 6.1 $pg \cdot mL^{-1}$ and 0.12 $ng \cdot mL^{-1}$, respectively. The inter-assay coefficient of variation (CV) was <11% for T and <8% for C. The samples for each participant were analysed within the same assay run.

Hand-grip strength testing

The HGS assessment allowed testing of systemic hormonal changes induced by lower-body exercise, as well as being easy to implement and standardise across subjects. Strength was measured to an accuracy of 0.1 kg with a digital dynamometer (Camry, China) using similar procedures to published work (Gallup et al., 2007; Patel et al., 2004). Each person was seated throughout testing and, to eliminate any learning effect, only the dominant hand was assessed. Holding the dynamometer in a vertical position, the elbow was flexed to a 90-degree angle (keeping the upper arm in line with the torso) and maximal force was applied for 3-4 seconds before relaxing. Three trials were performed, each separated by a 40-second rest period, and the best effort was analysed. Pilot data (n=12) indicated excellent test-retest reliability (CV=2.0%) for this assessment.

Statistical analyses

Hormones were log-transformed before analysis to normalise data distribution and reduce non-uniformity bias, with the back-transformed data presented in their original units. Change scores were calculated for T, C and HGS (post – pre) across the sprint session and compared to a zero baseline using paired T-tests, with the control group results (i.e., no exercise, sprint exercise) tested in a similar manner. To aid interpretation, the raw values are shown and the change scores expressed as percent values. Effect sizes (ES) were computed using Cohen's d. Relationships between the T, C and HGS measures and demographic data (i.e., age [log-transformed], height, BM) were assessed using Pearson correlations. Hierarchical multiple linear regression was used to test the moderating effects of C, operationally defined as a significant T × C interaction (Mehta & Josephs, 2010). The independent variables were standardised by converting the raw scores to z-scores, with the interaction term calculated from the product of these variables. Simple slopes were used to interpret the significant interactions at high (i.e., 1 SD above the mean) and low (i.e., 1 SD below the mean) values. The level of significance was set at $p \leq 0.05$. All data are presented as means ± SD.

Results

Sprint exercise effects (n=65)

The T change score from pre-test (187±68 pg·mL^{-1}) to post-test (199±74 pg·mL^{-1}) represented a relative increase of 6.1±24.9 % (t(64)=-2.14, p=0.036, ES=0.16). Conversely, the C response from pre-test (2.63±1.71 ng·mL^{-1}) to post-test (2.25±1.37 ng·mL^{-1}) corresponded to a relative decline of -14.4±33.1% (t(64)=4.34, p<0.001, ES=-0.27). A small relative increase of 3.4±7.5% emerged when the change in HGS (t(64)=-3.57, p=0.001, ES=0.13) was assessed from pre-test (45.6±9.9 kg) to post-test (46.9±9.6 kg).

Control and sprint exercise effects (n=15)

The following pre- and post-test results were noted for T (145±45 and 151±38 pg·mL^{-1}), C (1.43±0.63 and 1.49±0.63 ng·mL^{-1}) and HGS (56.2±7.7 and 56.8±7.7 kg) in the control session. Subsequent testing revealed no changes in T (5.9±30.0%, $t(14)=-0.84$, $p=0.412$, ES=0.19), C (3.2±23.1%, $t(14)=-0.59$, $p=0.566$, ES=0.07) or HGS (-0.9±3.0%, $t(14)=1.21$, $p=0.247$, ES=0.07). The exercise data were as follows for T (158±49 and 188±54 $pg \cdot mL^{-1}$), C (2.18±1.19 and 1.98±1.17 $ng \cdot mL^{-1}$) and HGS (55.7±7.6 and 57.2±7.3 kg). The analysed changes in T (19.1±28.8%, $t(14)=-2.68$, $p=0.018$, ES=0.55), C (-8.9±44.6%, $t(14)=0.97$, $p=0.346$, ES=0.19) and HGS (2.9±5.7%, $t(14)=-1.95$, $p=0.072$, ES=0.20) mirrored the population trends, but only the T and HGS data were, or verged on, significance. The between-session differences in T ($t(14)=1.40$, $p=0.184$) and C ($t(14)=-1.22$, $p=0.242$) were not significant, whilst the HGS results approached significance ($t(14)=2.06$, $p=0.058$). Pre-test comparisons revealed that C levels were 52% higher before the sprints than the control session ($p=0.011$), whereas the pre-test T and HGS were no different ($p>0.408$).

Correlations between age, BM, hormones and HGS

Participant age was positively correlated with BM, the T changes and pre-HGS, whilst pre-T was negatively related to age ($p \leq 0.05$, Table 1). Body mass was also positively related to height and pre-HGS ($p<0.01$). Hormonal comparisons revealed positive correlations between T and C before testing and their respective change scores ($p<0.001$). The pre- and post-test hormone values were strongly correlated ($r(63)>0.81$, $p<0.001$); thus, the post-test outcomes were not included to eliminate redundancy. Significant correlations determined which variables would be included in the regression models as covariates (Mehta & Josephs, 2010).

Insert Table 1.

Predicting pre-HGS

Pre-HGS was entered as the dependant variable and the following as predictors: BM and age in Step 1; pre-T and pre-C in Step 2; the pre-T × pre-C interaction in Step 3. In model 1 (Table 2), BM and age jointly explained 15.6% of the variance in pre-HGS (p=0.005), but adding pre-T and pre-C in model 2 did not improve this relationship (18.8%, p=0.311). In model 3, adding the pre-T × pre-C interaction increased the explained variance (24.8%, p=0.033). We tested this interaction (Figure 1A) and found a significant negative association between pre-T and pre-HGS at high pre-C levels (slope=-4.165, t=-2.529, p=0.014), but a non-significant relationship at low pre-C levels (slope=0.997, t=0.531, p=0.598).

Insert Table 2 and Figure 1A-1B.

Predicting the HGS changes

The change in HGS was entered as the dependant variable with the following predictors: pre-T, pre-C, T change and C change in Step 1; the interactions between each pair of hormonal variables in Step 2. No demographic variables were correlated with the dependant variable; thus, regression was performed as a 2-step process. All possible combinations were tested, but only the model that included pre-C and T change as predictors produced a significant interaction (Table 3). Pre-C and T change did not jointly predict the HGS changes in model 1 (0.8%, p=0.780), but their interaction predicted 10.7% of the variance in model 2 (p=0.012). Probing this interaction (Figure 1B) revealed a significant negative association between the T and HGS changes at low pre-C levels (slope=-1.032, t=-2.189, p=0.032), but a non-significant relationship at high pre-C levels (slope=0.731, t=1.405, p=0.165).

Insert Table 3.

Discussion

This study is the first to document a moderating role for C with respect to the T association with HGS in healthy men. Sprint cycling exercise provided an effective stimulus for promoting rapid hormonal and HGS responses. Within this framework, the T and C measures did not predict pre-test HGS or the resultant HGS changes. A significant hormonal interaction was however identified, such that T predicted both strength outcomes when taking into account individual differences in pre-test C levels.

Consistent with prior studies (Crewther et al., 2011b; Goto et al., 2007; Obmiński et al., 1998), the sprint cycling protocol produced a small positive change in T (6.1%) in a short timeframe (<20 minutes). The negative C response (-14.4%) was somewhat unexpected, given the stressful nature of sprinting exercise involving the lower limbs. This finding might be due to T inhibition of the hypothalamic-pituitary axis during initial recovery (Viau, 2002), individual variation in C reactivity (Crewther et al., 2011a) and/or a delayed increase in C levels relative to the initial T response (Goto et al., 2007). It is noteworthy that subject C levels before the sprint exercise session were more than 50% higher than the control session, suggesting that a hormonal stress or anticipatory response occurred before exercise. This baseline difference may explain why we were unable to induce a subsequent rise in C with a relatively stressful stimulus. These hormonal responses were accompanied by a small (3.4%) increase in HGS, thereby supporting the possibility that acute T and/or C variation might also modify physical performance (Crewther et al., 2011b; Obmiński et al., 1998).

As hypothesised, the T measures were unrelated to the HGS outcomes, adding to the variable results among weaker or lesser-trained populations (Ahtiainen et al., 2003; Crewther et al., 2011a; Crewther et al., 2012). More complex hormonal interactions might be governing the expression of muscle strength, as we found. Specifically, pre-test T and HGS were negatively associated in men with high (not low) pre-test C levels. This implies that the pre-test combination of high C and low T levels favoured absolute HGS, which is partly supported by studies showing a link between high C and/or low T levels to greater maximal strength (Crewther et al., 2011c; Crewther et al., 2009; Passelergue et al., 1995). The T and HGS changes were negatively related in men with low (not high) pre-test C levels, indicating that low pre-test C levels and a smaller T change is linked to larger HGS changes. Speculatively, being less stressed (i.e., low C) might ensure that other potentiating mechanisms (e.g., myosin phosphorylation, motor unit recruitment) are activated by exercise (Tillin & Bishop, 2009), with a small or negative T response possibly indicating better tissue uptake (Crewther et al., 2011a) and/or metabolite conversion (Wood & Stanton, 2012) to augment this response.

The finding that C is moderating the T effect compliments behavioural studies (Edwards & Casto, 2013; Mehta & Josephs, 2010; Mehta & Prasad, 2016), although the reported T relationships are mostly positive at low C levels and negative at high C levels (Mehta & Prasad, 2016). This reversal could be attributed to population differences in circulating hormones, along with the exercise stimulus and assessment employed herein. Work in this and other domains has identified several possible mechanisms to explain the moderating role of C. For instance, C can regulate T coupling with brain activity (Denson et al., 2013), dominance behaviours (Mehta & Josephs, 2010) and the expression of androgen receptors (Burnstein et al., 1995). The adrenal and gonadal systems also interact at various levels to regulate T and C release (Viau, 2002), as evidenced by the correlations in this and other work (Edwards & Casto, 2013). Further research is needed to elucidate those mechanisms activated in a sport and exercise context, the hormonal responses accompanying these situations, and their combined role in supporting muscle and physical performance.

We acknowledge that sprint-type exercise can increase muscle temperature, physiological activity (e.g., catecholamines, lactate) and possibly induce muscle potentiation, as other mechanisms to explain the HGS results. These effects were partly addressed by the study design (e.g., choice of exercises, rest period). Since some men showed HGS gains and others no change under the same exercising conditions (Figure 1), the likely contribution from a temperature-related, or other peripheral, mechanism is reduced. Our sampling protocols (i.e., single post-exercise sample) also make it difficult to capture temporal hormone dynamics and the predictive models developed still have a large degree of unexplained variance. Nevertheless, we identified novel hormonal interactions that could be regulating muscle performance in a healthy male cohort, with broader applications for individualising workouts and identifying predispositions for absolute strength or exercise-induced strength changes.

To summarise, complex relationships between T and HGS emerged in healthy young men that were only identifiable when low C and high C individuals before exercise were considered separately. The direction of these relationships also suggested that T might be less important to muscle strength in healthy young men. This information could help to reconcile the inconsistent relationships seen in men with little or no training experience.

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Table 1. Correlations between the demographic, hormonal and performance variables.

	Height	BM	Pre-C	C change	Pre-T	T change	Pre-HGS	HGS change
Age	-0.22	0.24*	-0.23	0.23	-0.37#	0.24*	0.24*	0.01
Height		0.39#	0.02	0.04	-0.02	0.02	0.18	0.08
BM			-0.15	-0.01	-0.01	-0.03	0.36#	0.05
Pre-C				-0.23	0.41#	0.09	-0.14	-0.04
C change					-0.15	0.51#	0.15	-0.11
Pre-T						-0.19	-0.23	-0.03
T change							0.16	-0.08
Pre-HGS								-0.23

Note: BM = body mass, C = cortisol, T = testosterone, HGS = hand-grip strength. Significant correlation *$p \leq 0.05$. #$p < 0.01$.

Table 2. Multiple regression with pre-test cortisol and pre-test testosterone as predictors of pre-test hand-grip strength.

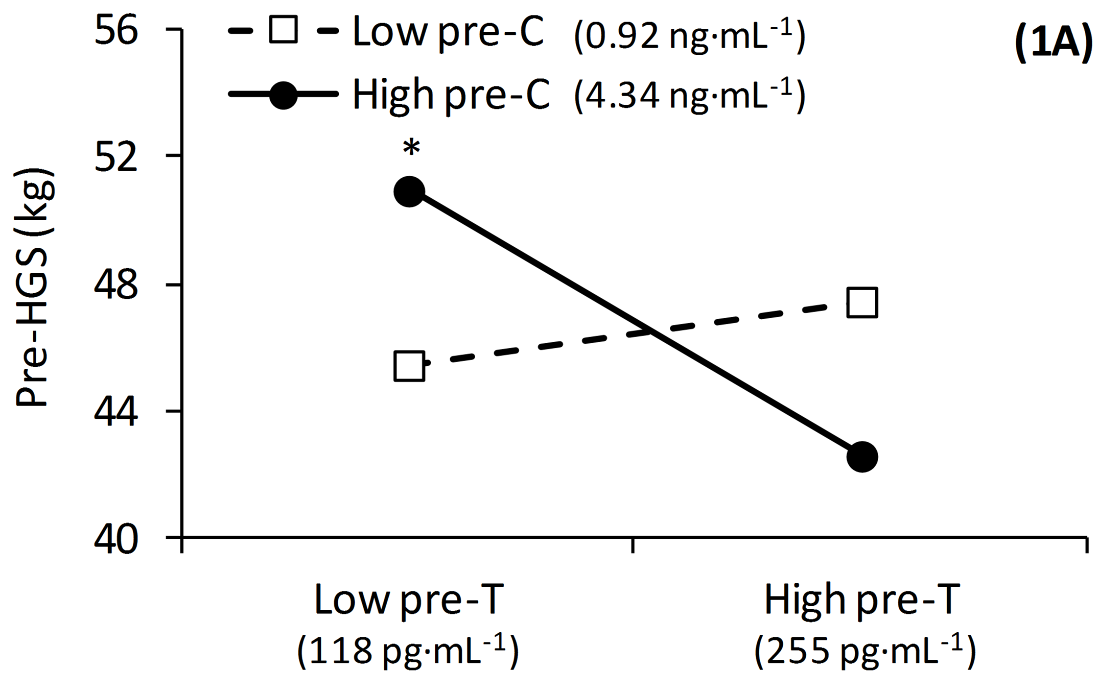
	Variable	β	t	*p*
Model 1	F(2,62)=5.715, p=0.005, R^2=0.156			
	BM	0.319	2.653	0.010
	Age	0.170	1.412	0.163
Model 2	F(2,60)=1.192, p=0.311, R^2=0.188			
	BM	0.336	2.769	0.007
	Age	0.094	0.725	0.470
	Pre-T	-0.198	-1.463	0.149
	Pre-C	0.011	0.088	0.930
Model 2	F(1,59)=4.748, p=0.033, R^2=0.248			
	BM	0.290	2.423	0.019
	Age	0.188	1.412	0.162
	Pre-T	-0.160	-1.208	0.232
	Pre-C	0.016	0.128	0.899
	Pre-T × Pre-C	-0.261	-2.179	0.033

Note: BM = body mass, C = cortisol, T = testosterone.

Table 3. Multiple regression analyses with prê-test cortisol and the testosterone changes as predictors of the hand-grip strength changes.

	Variable	β	t	*p*
Model 1	F(2,62)=0.249, p=0.780, R^2=0.008			
	T change	-0.082	-0.645	0.521
	Pre-C	-0.029	-0.227	0.821
Model 2	F(1,61)=6.726, p=0.012, R^2=0.107			
	T change	-0.051	-0.416	0.679
	Pre-C	-0.139	-1.078	0.285
	T change × Pre-C	0.333	2.594	0.012

Note: C = cortisol, T = testosterone.



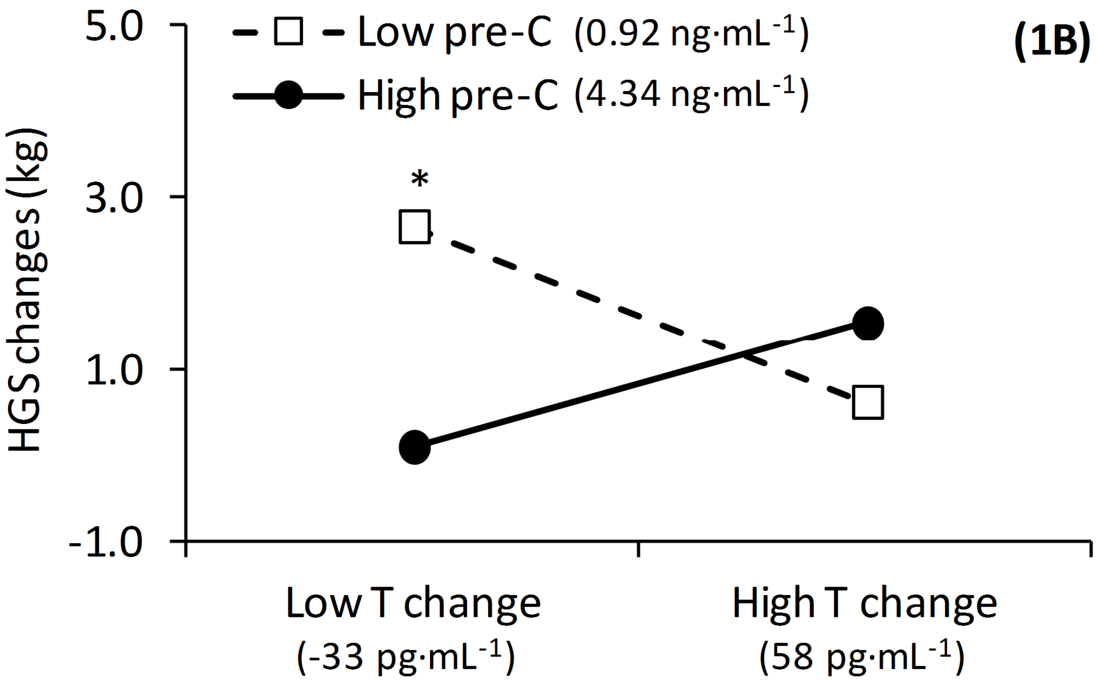


Figure 1. Interaction between pre-test cortisol (C) levels and the testosterone (T) measures in relation to the hand-grip strength (HGS) measures. Low hormone values = mean - 1SD, High hormone values = mean + 1SD. *Significant slope $p \leq 0.05$.