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Influence of Training-induced Testosterone and Cortisol Changes on Skeletal Muscle and Performance in Elite Junior Athletes

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Abstract This study aimed to investigate the influence of testosterone (T) and cortisol (C) on Skeletal Muscle Markers (SMM) at each phase of a track and field macrocycle. On a secondary basis, we also sought to determine whether C or T moderates the relationship between SMM and performance. Twenty-eighth (28) elite junior sprint athletes (15.48 ±1.89 years), and 13 non-athletic (16.15 ±1.51 years), age and gender-matched controls participated in this study. Isometric muscle strength (MS) and muscle thickness (MTH) were considered SMM. Salivary C and T levels, MS and MTH were collected between 2:30 and 3:00 p.m. before training sessions, twice during the preparatory phase [baseline (T1) and midway into the specific preparation period (T2)] and twice in competition phase [midway point of pre-competition (T3) and midway of the major competition period (T4)]. Performance data were collected during the competition phase only. No significant improvements in SMM were observed. T and T/C ratio significantly increased ($p<0.05$) across the season, while C levels increased relative to baseline only. While T levels did not significantly predict SMM at any phase, C levels significantly explained ($p<0.05$) a 60% and 74% variance in MTH and performance respectively. However, neither C nor T significantly moderated the relationship between SMM and performance. These findings suggest that raising T levels across a season may be more indicative of a response to training load, rather than a reflection of skeletal muscle adaptation. While training-induced C, which was demonstrated to have a greater influence on SMM is more sensitive to muscle function changes and performance compared to testosterone.

Keywords: *testosterone, cortisol, skeletal muscle, hormones, performance, macrocycle, junior athletes, sprint, muscle adaptation*

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1. Introduction

Skeletal muscle markers (SMM), such as muscle thickness (MTH) and strength (MS) are important determinants of skeletal muscle adaptation and are known to have a direct impact on sprint performance among junior athletes. Together, changes in these markers have been suspected to be directly influenced by training-induced steroidal hormones such as testosterone (T) and cortisol (C) [1,2]. T which is secreted by the testes, ovaries, and adrenals is an anabolic hormone [3], while C, which is secreted from the adrenal cortex is a catabolic hormone [4]. Together, these hormones act as a measure of the physiological status of skeletal muscles, regulating the balance between protein synthesis and breakdown, which ultimately affects skeletal muscle adaptation [5].

T is known to increase linearly in response to exercise within a specific intensity threshold [6]. The hormone plays a key role in the growth and maintenance of skeletal muscles, as an increase in T usually leads to increased mitochondrial biogenesis and angiogenesis, thereby increasing protein synthesis, and enhancing muscle growth [7]. Though the mechanism by which this occurs is still not fully understood, it has been proposed that T augments the expression of the insulin-like growth factor-I gene (IGF-1), which is responsible for the proliferation of muscle stem cells [8,9]. While at the cellular level, its anabolic effect has been linked to its ability to: 1) cause an increase in the cross-sectional area of type I and II muscle fibers and 2) increase myonuclear quantity [10]. Thereby, suggesting that T exerts a greater hypertrophic than a hyperplasic effect on skeletal muscle growth [10,11]. Indeed, previous studies have established strong positive correlations between resistance training-induced T, and muscle cross-sectional area [1,12]. For example, Ahtiainen, Pakarinen,

Alen, Kraemer, and Häkkinen [12] demonstrated significant correlations between basal T and isometric strength and found that acute T response to resistance training is significantly correlated to muscle cross-sectional area. On the other hand, studies have reported a lack of significance between muscle hypertrophy and T levels, while suggesting that T may not be a useful indicator of tissue anabolism [13].

C, on the other hand, exerts an opposite effect to that of T. It is catabolic and causes decomposition of muscle tissues which in some cases leads to sarcopenia [7]. It is, however, a glucocorticoid and as such exerts many beneficial effects in humans, such as increasing metabolic substrate availability, maintaining normal vascular integrity, and protecting the body against exaggerated responses of the immunological system to exercise-induced muscle damage [14]. However, its role in protein degradation, although necessary to provide amino acid for gluconeogenesis, may affect multiple tissues including skeletal muscle integrity during the training process [6]. This is because glucocorticoids influence intracellular signaling pathways (p38 MAPK and PI3K/Akt/mTOR) involved in training adaptation [15], consequently impairing muscle function through diminished MS. Moreover, according to Grandys, Majerczak, Kulpa, Duda, Rychlik, and Zoladz [16] the diminished C level after a training program seems to create a favorable condition for intracellular pathways that augment muscle protein synthesis and muscle performance.

Indeed, both T and C seem to have optimal levels, at which a favorable hormonal milieu can be established for optimal muscle performance and recovery. However, studies assessing the implications of changes in this hormonal milieu [12,13,17,18] on muscle function are limited. Further, the implication of training-induced hormonal changes on muscle function and athletic performance is grossly under-researched. Studies do, however, show that higher levels of the T/C ratio represent a favorable hormonal milieu for muscle hypertrophy [19,20]. Literature is, however, limited as to whether, this is due to increases in T levels or a decrease in C and the catabolic response, as chronically high levels of C are associated with decreases in muscle mass [21,22]. Additionally, it is speculated that an increase in C occurs concurrently with decreases in T in response to prolonged training [23]. The mechanism underlying this inverse relationship between C and T in response to physical stress may be due to the fact that physical stress shunts the precursors (cholesterol and progesterone) of both C and T towards the direction, favorable for C production, thereby tipping the scales towards protein break down. Some studies have also suggested that the catabolic effect of C is mediated through glucocorticoid receptors [24]. As androgenic hormones are also capable of binding to these receptors, resulting in competitive binding of C and T for the glucocorticoid receptor [25]. Therefore, when T levels are increased due to athletic conditioning, this would to some extent limit the catabolic action by inhibiting the binding of C to these receptors, a step that is necessary for protein breakdown to occur [24]. This then explains why prolonged physical stress may result in an imbalance in the anabolic/catabolic state of skeletal muscles. An imbalance, where, if the catabolic state predominates is unfavorable for skeletal muscle adaptation [23].

Training-induced skeletal muscle adaptation occurs along a continuum [26,27,28]. At one end of the continuum, athletes are exposed to minimal training stimuli, resulting in a very minute hormonal response. At this point athletes are undertrained, resulting in a low anabolic response which would limit mitochondrial biogenesis and angiogenesis, thereby limiting hypertrophic changes. Should training be increased beyond this point to an optimal level, and appropriate recovery is practiced, a super compensatory effect where muscle adaptation and improvements relative to baseline would be observed [5]. At this point of the continuum, the physiological responses such as enhanced muscle growth and hormonal increases would compensate for the training-induced stress [29]. Consistent training would then eventually result in a downregulation of glucocorticoid receptors, which would reduce the catabolic response of C [30]. Ideally, given that the athlete is exposed to adequate recovery, the hormonal milieu at this point would result in positive skeletal muscle response to the training process. According to Meeusen, Duclos, Foster, Fry, Gleeson, Nieman, Raglin, Rietjens, Steinacker, and Urhausen [31], this phase is necessary for performance improvement to occur. Athletes exposed to training stress which surpasses this optimal training point along the continuum, often present hormonal levels consistent with overtraining or the risk of overtraining [12,16,17]. Overtraining is characterized by underperformance and non-functional adaptations such as strength loss [31] and has been found to affect over 64% of junior-level athletes [5]. Among elite athletes, T and C are considered useful markers for the detection of overtraining [32]. The hormonal response of overtraining is often detrimental to skeletal muscle adaptation as studies have shown that athletes at risk of overtraining, usually demonstrate relatively high C levels [12,16,17,33,34].

According to Meeusen, Duclos, Foster, Fry, Gleeson, Nieman, Raglin, Rietjens, Steinacker and Urhausen [31], maladaptation is key in the identification of overtraining. Furthermore, a T/C milieu where C is elevated indicates overtraining, as such the modulation of this ratio can be key in identifying those athletes who are susceptible to overtraining [32]. It is established both T and C levels directly influence muscle performance [30]. Yet, many studies have disputed the individual uses of chronic C, T, and/or T/C ratio as diagnostic markers of skeletal muscle adaptation [31,35]. Possibly because, the roles of these steroidal hormones on skeletal muscle adaptation are still not fully understood. Indeed, the T/C hormonal milieu has been previously explored relative to changes in skeletal muscle markers [36], specifically, increases in MTH and MS have been reported in athletes who exhibited a significant decrease in C concentration and a significant increase in T/C ratio [16,37]. However, some studies suggest that this is merely a hormonal response to training and these changes may have no direct influence on skeletal muscle adaptation [38,39,40]. Thereby suggesting that equivocality exists regarding the relationship of SMM and hormonal changes within the literature. Furthermore, while an increase in T and a decrease in C may be ideal for intrinsic muscle growth and strength development [6].

Such findings have been disputed, particularly in competitive environs. As studies have found that the combination of low T and high C levels was associated with greater hand-grip strength before stressful exercise [1]. Moreover, there have been reports of rising C levels being necessary for optimizing MS under competitive stress more so than T [41,42]. With such contrasting data in the literature, clarity is needed as it pertains to the association between muscle function and training-induced hormonal changes. This study seeks to increase the understanding of this association by longitudinally monitoring both muscle function and hormonal changes in response to training. As such the overall aim was to determine changes in SMM (MTH and MS), T, and C and to investigate the influence of the measured hormonal changes on the SMM at each phase of the track and field macrocycle. We hypothesized that a greater increase in T and decrease in C across the macrocycle will result in greater improvement in SMM. Furthermore, given that the SMM has a direct impact on sprint performance [43,44] this study also sought to determine whether C or T moderates the relationship between SMM and performance during the preparatory and the competition phases. Concerning this aim, and in accordance with the literature, we predicted that both T and C independently moderate the relationship between SMM and sprint performance.

2. Method

2.1. Participants

Descriptive characteristics of participants can be seen in Table 1. Forty-one participants volunteered for the study, this included 28 elite track athletes (14 males and 14 females) and 13 non-athletes (6 females, 7 males). Among the participants, 49% were found to be in the late pubertal stage of pubertal development while 27% were at the mid pubertal, 22.0% in the post-pubertal, and 2% in the early pubertal stage. All athletes participated exclusively in athletics in sprint events (100m-400m). Athletes were invited from five Jamaican high school track and field teams. Non-athletes were age and gender-matched individuals within a healthy body mass index (BMI: 18.5-25 kg/m^2) and did not participate in any organized sport. Ethical approval was obtained from the UWI/UHWI Faculty of Medical Sciences Ethics Committee, and the study was carried out in adherence to international ethical standards (Declaration of Helsinki). Relevant approvals from the local Ministries of Health and Education were also obtained. Before the start of the study, parents and coaches were informed about the purpose and design of the study. Assent and consent forms were obtained from participants <18 years and their parents/legal guardians respectively. Participants ≥18 years consented to themselves. All participants were healthy and devoid of any form of acute musculoskeletal, neurological, orthopedic disorders and any disease or illness that may have confounded study results. None of the participants suffered any injury during the season that would have caused them to miss more than two weeks of training.

Table 1. Descriptive characteristics of participants (Data are mean [Standard Deviation])

Variable	Athletes (*n*=28)	Non-athletes (*n*=13)
Age/ years	15.48 [1.89]	16.15 [1.51]
Weight/ kg	62.69 [11.68]	57.20 10.38]
Height/cm	170.83 [9.13]	168.33 [9.64]
BMI/ Kg/m^2	21.22 [2.61]	20.86 [2.57]
BF%	13.75 [4.46]	16.58 [5.17]
FFM/kg	54.21 [11.54]	46.03 [10.80]

2.2. Study Design

An illustration of the study design can be seen below in Figure 1. In this study, a longitudinal approach was adapted to systematically monitor athletes across the 2019/2020 junior athletic season. The macrocycle lasted 7 months and consisted of one competitive phase. Data were collected once during each period [i.e, the general preparation period (T1), specific preparation period (T2), pre-competition period (T3), and major competition period (T4)]. T1-T2 is collectively referred to as the preparatory phase, with T1 representing baseline data, which was collected during the first week of training, while T3-T4 is collectively considered the competition phase. Data collections were carried out at the midway point of T1, T2, and T3 at the athletes' training grounds before the beginning of a training session and following class hours (2:30 p.m-3:30 pm). During each data collection session, athletes were given sterilized 4 oz sample containers for saliva sampling. Ultrasound measures of MTH and isometric assessment of MS were carried out. Additionally, questionnaires were distributed to each athlete, regarding their dietary practices, injury history, and general health. Athletes were thoroughly instructed on how to accurately complete the questionnaire and how to go about giving samples.

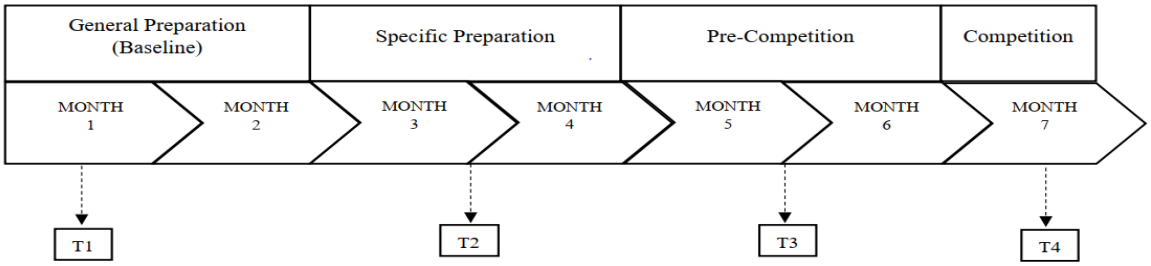

Figure 1. Illustration of study design: Illustration of the mesocycles and the points of data collection, across the junior athletic season

2.3. Description of Training

The general preparation period (T1) training consisted primarily of endurance and resistance sessions. As fitness, MS and muscle endurance capacity are usually emphasized by coaches at this mesocycle. During the specific preparation period (T2), training exercises became less diversified, and athletes focused on high mechanical specificity aimed at developing muscular strength and hypertrophy for later speed and power work. While during the pre-competition phase (T3) focus was placed on maintaining fitness, muscle size and strength while enhancing technical consistency. For this phase, athletes were exposed to 3-4 resistance and sprint sessions weekly and ~2 endurance training sessions weekly. During the major competition phase (T4) athletes performed minimal resistance exercise sessions weekly, with majority of training focused on sprint exercise sessions and 1-2 endurance sessions weekly. It is during this phase that athletes reach peak performance. However, typically during the last few days before important competitions intensity is reduced to encourage adequate recovery. This latter portion of a peaking phase (typically 8–14 days), when volume is markedly reduced, is referred to as a "taper" [45]. Each mesocycle lasted approximately 8 weeks except for the major competition period which lasted a month. The athletes followed the stipulated training protocols administered by their coach during each of the described training phase, while the non-athletes were not exposed to any formal training, and were relatively sedentary individuals

2.4. Procedure

2.4.1. Anthropometry and Body Composition

Height and weight were determined using a portable stadiometer and the Weight Gurus Bluetooth Body Composition Scale (model:0382) respectively. Both measures were carried out with the participant being barefoot. The BodyMetrix™ BX2000 (IntelaMetrix, Inc., Livermore, CA) 2.5 MHz, A-mode ultrasound transducer in conjunction with the associated Body View Professional Software (IntelaMetrix, Inc., Livermore, CA) as described by Melvin, Smith-Ryan, Wingfield, Ryan, Trexler, and Roelofs [46] were used to determine body composition. The manufacturer's recommendations for making single-point ultrasound measurements at skinfold sites; triceps, waist, hip, and chest) were followed. This technique allowed measurement of body fat percentage (BF%), fat-free mass (FFM), and BMI which were displayed on the Body View Professional software.

2.4.2. Ultrasound MTH Scan

Muscle thickness (MTH) was defined as the distance between the adipose tissue-muscle interface and the muscle-bone interface [47]. Thigh MTH was assessed using panoramic scans of knee extensor muscles (rectus femoris, vastus intermedius) and knee flexor muscles (hamstring group muscles) [19]. The anatomical locations of interest were identified before the measurement took place. The landmarks for the knee flexors were identified along with the longitudinal distance over the femur at 50% of the length of the muscle, while the knee flexors were taken longitudinally from the mid posterior thigh to the popliteal fossa. Cumulative values of MTH for the knee extensor and knee flexor muscles were considered MTH. All measures required the participant to lay supine. For each region, three consecutive images were collected. The actual MTH of each muscle was determined by ultrasonography using on-screen digital calipers provided by the Body View Professional software (IntelaMetrix, Inc., Livermore, CA).

2.4.3. Muscle Strength (MS)

Muscle Strength (MS) was assessed as maximal isometric voluntary contraction and was measured with a hand-held dynamometer (HydraulicPush-Pull Dynamometer, Baselines Evaluation Instruments, White Plains, New York, USA) similar to Krause, Schlagel, Stember, Zoetewey, and Hollman [48]. Measurements were taken three times with one-minute rest intervals between contractions. Isometric muscle force from knee flexion and extension were measured. The measure of isometric muscle force required one contraction for every individual muscle starting with knee extension and then a knee flexion. This sequence was performed three times on the participant's dominant leg. Subjects were asked to gradually increase their muscle force to a maximum effort which would need to be sustained for three seconds. The 'break technique' was employed whereby the examiner overpowers the maximum effort of the participant, thereby producing a measurement of eccentric muscle force [49]. The average muscle force of three repetitions was calculated to compensate for and minimize measurement errors.

The measurements were all taken by the same co-investigator, using the break method, the co-investigator broke through the subject's muscle force by countering the force employing a continuous, slow movement. This position was maintained throughout the entire test.

Standardized encouragement was given to each participant. If the observer was unable to break through the patient's generated force, this was recorded in the data collection form, and that result was omitted from the data analysis. Measurements were taken in a standardized and gravity-neutral body position. Cumulative values of knee extensor and knee flexor contraction were considered lower body MS.

2.4.4. Testosterone and Cortisol Sampling and Assay

For T and C assays, whole saliva samples were used. Participants were instructed to passively drool into the provided sterile 4 oz containers. Saliva samples (~1ml) were collected from each participant within the same time frame (2:30 pm-3:30 pm) to account for diurnal variation [50]. Following which samples were immediately placed on ice for transport to the laboratory where they were then kept at -20°C for storage. Standard assay kits (Salimetrics, State College, PA, USA) employing the enzyme-linked immunosorbent assay (ELISA) technique were used to determine the concentration of C and T in each sample. Both assays employed the use of the competitive ELISA, which utilizes two specific antibodies, an enzyme-conjugated antibody and another antibody that is present in the test sample. Combining the two antibodies into the wells allows for a competition for binding to antigen. The intra-assay coefficient of variation and inter-assay reproducibility for saliva T were 5.63±10.63 % and 14.60

±1.36 %, respectively. The intra-assay coefficient of variation and inter-assay reproducibility for saliva C were 5.84±13.83% and 11.30±0.23% respectively.

2.4.5. Performance

Performance was assessed by determining each athlete's relative change in performance (ΔP) throughout the competition period using the formula:

Relative Performance Change:

$$\Delta P = CP / (CP + SB)\ X\ 100 \quad (1)$$

Season-best (SB) times and championship (CP) times were obtained from the Jamaica Athletics Administrative Association database. The athletes' season's best times from development meets during the pre- competition phase were used along with the fastest time posed at the major championship games (Annual Boy's and Girl's Championship). To calculate relative performance change, times were converted to points using the 2017 revised edition of the IAAF scoring table as use of this system allows for comparison of performance across events. When ΔP≥50 this was considered improvement or maintenance of championship performance relative to the SB. When ΔP < 50 this was considered as a decline in championship performance relative to SB time.

2.5. Statistical Analyses

All analyses were performed using IBM SPSS statistics version 23 software. The Shapiro-Wilk test was used to determine normality of data distributions. T, C, and T/C failed the normality test and as such were log-transformed to meet diagnostic assumptions. To aid interpretations, the raw hormonal data are presented. Analysis of Covariance (ANCOVA) with repeated measures (participant type = athlete/non-athletes vs mesocycle= T1-T4) was used to determine possible differences in testosterone, cortisol, MS, and MTH between the preparatory and competition phase (T1-T4). In assessing changes in MTH covariates: height, body mass, age, pubertal age, and gender were entered in the model, while only age and gender were controlled for in assessing MS change. For analyses of hormonal changes; age, gender, and pubertal age were entered as covariates. Geisser-Greenhouse correction was used when the sphericity assumption in repeated measures ANCOVA was violated (Mauchly's test). Post-hoc tests were performed using Bonferroni post hoc analyses. Partial eta squared (ηp2) was used to describe effect size as small (ηp2 = 0.01), medium (ηp2 = 0.06) and large (ηp2 =0.14) effect for all RM-ANCOVAs. Separate linear Pearson correlations (r) were performed to determine the relationships between SMM and hormonal changes. The association between SMM, hormonal changes, and performance were also assessed using Pearson correlation. To further analyze the relationship between SMM with testosterone and cortisol, hierarchical regression analyses were carried out at the preparatory and competition phases. MTH was entered as the dependent variable and age, body mass, height, and pubertal age were entered as control variables in step 1, while step two included T, C, and T/C. This test was then repeated with MS entered as the dependent variable, with the same covariates and independent variables used. Another hierarchical regression analysis was then carried out with performance entered as the dependent variable. Age and gender were entered as control variables, SMM was entered in step two, and T, C, and T/C were entered in step 3. To determine whether T or C is moderating the relationship between SMM and performance, interaction variables were entered in subsequent steps. Step 5 included MTH*C and MTH*T, while step six included MS*C and MS*T. Prior to running the hierarchical regression, all continuous variables of interest for the model were standardized (i.e., coefficients expressed in SDs) to reduce multicollinearity and aid interpretation. The interaction terms were calculated by multiplying the standardized scores. The significance level was set at $p<0.05$.

3. Results

3.1. Change in Variables Across the Macrocycle

Table 2 shows changes in T, C, T/C, MTH, and MS across the four phases of the athletic season. Using repeated measures ANCOVA, changes in T and C were assessed, with age, gender, body mass, and pubertal age entered as covariates. While no significant change in C was observed across the season, significant changes were however observed for T (F (3, 53.60)=5.58, p=0.01, ηp2=0.17), and T/C (F (2.24, 34)=3.54, p=0.02, ηp2= 0.18). There was also a significant group*time interaction (F (2.26, 77.14) = 3.13, p=0.04 ηp2= 0.09) observed for T.

Table 2. Testosterone, cortisol, T/C, muscle thickness and muscle strength according training phase

		Preparatory Phase		Competition		Total between-group effects ATH-NATH (95% CI)
	Group	T1 (Baseline)	T2	T3	T4	
T (pmol/L)	ATH	441.46±249.61[b,c,d]	483.70±265.51[a,c,d]	406.73±249.29[a, b,d]	485.81±274.60[a,b,c]	-331.43 (-555.95, -106.92)
	NATH*	887.16±560.85[b,c,d]	658.21± 447.82[a,c,d]	915.37±543.12[a,b,d]	889.90±490.17[a, b,c]	
C (nmol/L)	ATH	1.97±1.25	4.15±3.15 [a]	2.89±1.62 [a]	2.84±1.47 [a]	-2.04 (-3.40, -0.68)
	NATH	4.84±3.69	4.34±3.12	5.81±4.18	5.88±3.57	
T/C	ATH	0.41±0.83[b,c,d]	0.24±0.32[a,c,d]	0.17±0.14[a,b,d]	0.24±0.25[a,b,c]	0.03 (-0.06, -0.22)
	NATH	0.29±0.28[b,c,d]	0.34±0.55[a,c,d]	0.20±0.12[a,b,d]	0.21±0.15[a,b,c]	
MTH (mm)	ATH	73.87±13.39	72.18±9.14	69.76±8.87	72.24±11.12	18.24 (11.95, 24.53)
	NATH*	54.13±8.17	53.36±8.51	53.75±7.16	54.00±8.16	
MS (N)	ATH	331.27±97.96	321.28±107.28	319.20±81.47	328.16±74.08	52.55 (1.20, 103.90)
	NATH*	286.53±69.57	264.49±64.96	274.59±62.97	270.59±62.97	

(a) Different from T1, (b) Different from T2, (c) Different from T3, (d) Different from T4, (*) Different from ATH

Additionally, both C and T/C levels increased significantly relative to baseline (baseline: C- 3.27± 2.64 nmol/L, T/C: 0.35±0.71 nmol/L; T2-C: 4.17± 3.68 nmol/L p=0.007. T/C-0.27± 0.40, p=0.01; T3-C: 3.84±3.01nmol/L p=0.04, T/C: 0.18± 0.13, p=0.002; T4-C: 3.80± 2.71nmol/L p=0.02, T/C:0.23±0.22 p=0.001). Repeated measures ANCOVA revealed that changes in MTH and MS across the season were not significant for the groups. However, significant group effects were observed for both variables (MTH: (F (1,35)=49.90, P=0.001, ηp2=0.58), MS: (F (1,32) =6.72, P=0.01, ηp2= 0.17), where MTH and MS among athletes (MTH: 71.99±10.06mm, MS:328.16 ± 74.08N) was significantly higher compared to non-athletes (MTH:53.75±7.16mm, MS:275.60±75.66N).

Table 3 and Table 4 represents the correlational matrices for SMM and hormones during the preparatory and competition periods respectively.

Table 3. Correlation between SMM and hormonal changes during preparatory phase

Variables	1	2	3	4	5	6
1. T	-					
2. C	0.20	-				
3. T/C	0.57**	-0.36	-			
4. MTH	0.37*	0.37	0.47**	-		
5. MS	0.36*	0.31	0.41**	0.55**	-	
6. Performance	0.004	-0.11	0.04	0.69	0.29	-

** significant at < 0.001 level, * significant at the < 0.05 level.

Table 4. Correlation between SMM and hormonal changes during competition phase

Variables	1	2	3	4	5	6
1. T	-					
2. C	0.09	-				
3. T/C	0.62**	-0.29	-			
4. MTH	0.14	-0.30*	0.48**	-		
5. MS	0.45**	-0.04	0.48	0.45**	-	
6. Performance	0.11	-0.32*	0.06	0.71**	0.26	-

** significant at < 0.001 level, * significant at the < 0.05 level.

Pearson correlation coefficient showed that during the preparatory phase MTH was significantly positively correlated to T (r=0.37, p=0.01) and T/C (r=0.47, p=0.003) while during the competition phase, MTH displayed significant relationships with C (r=-0.30, p=0.05) and T/C (r=0.48, p= 0.002). MS was found to display significant correlations with MTH (r=0.55, r=0.001), T (r=0.36, p=0.03), and T/C (r=0.4, r=0.02) during the competition phase but only showed significant correlations with MTH (r=0.45, r=0.004), and T (r=0.42, r=0.009), during the competition phase. Regarding performance, significant correlations were observed with C (r=0.55, r=0.001), and MTH (r=-0.32 r=0.04) during the competition phase only.

3.2. Regression Analyses

Multiple hierarchical regression analyses were conducted to examine the extent to which T, C, and T/C predict MS and MTH. When MS was entered as the dependent variable, the controlling variables entered in step 1 did not significantly predict MS at either phase. When T, C, and TC ratio were entered at Step 2, despite the model being significant at both the preparatory (F (7,23) =3.50, p=0.01, ΔR2= 0.51) and competition (F (7,27)=4.45, p=0.002, ΔR2= 0.53) phase, neither T, C, nor T/C was able to significantly predict MS. When MTH was entered as the dependent variable, the controlling variables entered in step 1 did not significantly predict MTH at either phase. While in step 2 the model was found to be significant (F (7,31) =8.57, p=0.02, ΔR2=0.14) with C significantly explaining a 60% variance in MTH (β=-1.64. t (31) =-2.36p=0.02) during the competition phase only. Within this step T and T/C were not observed to be significant predictors of MTH.

Another Hierarchical regression analysis was carried out at both phases to determine whether T or C is moderating the relationship between SMM and performance. Control variables age and gender were entered in step 1, for both the preparatory and competition phase where age contributed significantly to the model accounting for a 18.7% (β= -2.36, t(29)=-2.81, p= 0.01) and 14% (β=-0.52, t(32)= -2.74, p=0.01) variance in performance at the preparatory (F(3,29)=3.45, p=0.02, ΔR2=0.26,) and competition phases (F(3,32)=3.92 p=0.0, ΔR2=0.21,) respectively. In step 2, MTH and MS were found to significantly improve the model fit, accounting for a 70% variance in performance for the preparatory phase (F(5,27)=15.96, ΔR2=0.48, p=0.01,) and a 71% variance in the competition phase (F(5,30)=18.06, ΔR2=0.53, p=0.01,). MTH was observed to be a significant predictor at the preparatory phase: (β= 0.057, t(27)= 6.751, p=0.01) , and competition phase: (β= 0.06, t(30)=7.26, p=0.01). When T, C, and T/C were added in step 3, the efficiency of the model declined by 2% at the preparatory phase (F(7,25)=10.81, ΔR2=0.004 p=0.001) while an improvement in the model by 3% was observed at the competition phase (F(7,28)=15.15, ΔR2=0.04, p=0.01) following the addition of the same variables. Additionally, at this step, C was found to be a significant predictor (β= -0.07, t(26)=-2.13, p=0.04) of performance, during the competition phase only. To determine whether T or C is moderating the relationship between SMM and performance, interaction variables were entered into steps 4 and 5 of the regression models. Interaction variables MTH*T and MTH*C were included in step four. The model was found to be significant at both preparatory (F(9,23)=8.66, ΔR2=0.03, p=0.001) and competition (F(9,26)=12.41, ΔR2=0.02, p=0.001) phase, however, while the model did not improve for the preparatory phase, a 1% increase was observed during the competition phase. The interaction variables were not observed to be significant predictors at either phase, indicating that the relationship between MTH and performance, is not dependent on T or C. MS*T and MS*C were entered at step 5, and the model was found to be significant at both the preparatory (F(11,21)=6.60, ΔR2=0.003, p=0.001) and competition phase (F(11,24)=10.21, ΔR2=0.01, p=0.001), however, the model did not improve for the preparatory phase, declining by 3%, while no changes were observed during the competition phase. The interaction variables were not observed to be significant predictors at either phase, indicating that the relationship between MTH and performance and that of MS and performance, is not dependent on T or C.

4. Discussion

In accordance with the literature [51], it was predicted that an increase in MTH and MS would occur in response to training, and this would be accompanied by an increased anabolic state and a decreased catabolic state [40]. Results obtained, however, show that athletes displayed no significant increases in MS or MTH, despite the significant increases observed in both T (p=0.01) and T/C (p=0.02) across the season. There was a 2.8% decrease in MTH and a 0.8% decrease in MS between the preparatory and competition phase respectively. Thereby indicating that, despite favorable anabolic changes (i.e., increase in T and T/C), skeletal muscle adaptation was not facilitated. This is possible, because the observed increase in cortisol levels relative to baseline, induced a predominantly catabolic state within the muscles, thus limiting recovery and functional muscle adaptation. Moreover, T levels were possibly too low to ease the training-induced catabolic state. Thereby suggesting that the C level during the competition phase to some extent inhibited the anabolic response induced by the training regime.

T levels in this study were similar to those reported by Guilhem, Hanon, Gendreau, Bonneau, Guével, and Chennaoui [52]; (Preparatory: 404.5 pmol/L, Competition: 409.6 pmol/L) across the season. Like our findings, these authors reported an increase in T in the competition phase, relative to the preparatory phase. It can therefore be inferred that the training regime, to which the athletes in this study were exposed, was successful in elucidating a favorable anabolic response. The C levels observed were between 1.24 nmol/L and 8.52 nmol/L and are as such in the lower range of that observed for athletes (1.8-19.99 nmol/L; Guile, Hanon, Gendreau, Bonneau, Guével and Chennaoui [52]). In response to training, however, C levels in T2-T4 became significantly higher than baseline (T1) values. Studies have suggested that during long-term exercise, hypercortisolemia occurs to maintain adequate energy levels during training [9]. Therefore, the C response may have been a result of the prolonged, high-volume training endured during the preparatory phase.

Further, an increased catabolic state may also be induced during competition, in response to the competition being a psychological stressor. As the body induces a C response, to adapt to high-stress conditions [42]. This may then explain the higher levels of C observed during competition relative to the baseline values. It is, therefore, possible that this increase in C, may have also contributed to the lack of skeletal muscle adaptation as, the anabolic response, while moderated through T may be lowered by high C levels [6]. Moreover, in contrast to our findings, studies that have reported skeletal muscle adaptation have also reported significant increases in T/C and decreases in C [16,37]. These studies, however, included participants who were only exposed to a single exercise modality, such as endurance only or resistance only, and not a complete training program as was the case in this study.

On the other hand, studies assessing hormonal changes across a competitive sports season have reported observations of elevation in both T and C resulting in skeletal muscle adaptation [53]. Yet other studies have reported that increases in MS performance are significantly associated with an increase in T/C ratio and a decrease in C concentration [37,54]. As can be seen, the physiological and biochemical relationship concerning skeletal muscle adaptability to athletic conditioning is indeed complex. For example, measurement of exercise-induced C often presents a conundrum based on its equivocal physiological role. In that, C is frequently elevated after resistance exercise protocols designed to elicit hypertrophy [30,39,55], and yet is generally considered to be catabolic, and as such, counteractive to hypertrophy [56,57]. However, it has been established in the literature that higher levels of T/C represent a favorable hormonal milieu for muscle hypertrophy and strength [19,20]. Literature is however limited as to whether, this is due to increases in T levels or a decrease in C and the catabolic response [21,22]. On the other hand, previous studies have suggested that basal changes in the anabolic-catabolic hormone balance during exercise training programs are above all, an outcome of the applied training load [38,39,40] and have rather little influence on skeletal muscle performance. Indeed, a significant group *time interaction and significant group differences for T and T/C respectively between athletes and non-athletes were observed in this study. Thereby, indicating that the observed anabolic changes were specific to athletes, and as such possibly occurred because of the training regime.

It can be speculated that the observed increase in C may have facilitated a predominantly catabolic environment, which inhibited any possible muscle development induced by the increased T. T increases in response to training [6], and promotes skeletal muscle adaptation via hypertrophic mechanisms, such as increased type I and II muscle fiber cross-sectional area and increased myonuclear quantity [10]. Consequently, C has an inversed relationship with T and increases in response to training stress also [6]. In contrast to T however, chronically high levels of C have been found to be associated with decreases in muscle mass and strength [21,22]. Further, according to Velloso [8], a catabolic state can lead to diminished isokinetic strength due to losses of contractile proteins or neural transmitters typically stimulated by T. Additionally, it is speculated that a rise in C level, directly affects T production in response to prolonged training [23]. As an increased catabolic state in athletes may arise from increased physical stress causing a shift in steroidal hormone production. When this occurs, precursors (cholesterol, progesterone) of both C and T are shunted towards the direction, favorable for C production. Thereby tipping the scales towards protein breakdown.

Another possible explanation is the competitive binding of T and C for the glucocorticoid receptors [24]. Since androgenic hormones are also capable of binding to these receptors, competitive binding of C and T for the glucocorticoid receptor arises [25]. As a result, when T levels are increased due to athletic conditioning, this would to some extent limit the catabolic action by inhibiting the binding of C to these receptors, a step which is necessary for protein breakdown to occur [24]. However, if C levels continuously elevate this would lead to increased binding with the glucocorticoid receptor. Thereby resulting in increased protein catabolism and

immune suppression, consequently resulting in concomitant losses in muscular force and functional performance [13]. Elevation of C levels to the point where a catabolic state predominates is often the result of the physical demands of practice, conditioning, and/or competition, being too high for the athlete. An elevation in T concentrations could possibly assist in balancing these catabolic effects. However, decreased, or low levels of T concentrations, would not be able to provide adequate or optimal anabolic stimulation to offset the catabolic effects. This was possibly the case in this study, where despite the significant increases in T and T/C, these changes were insufficient in offsetting the catabolic state induced by the C increase. This then begs the question; are physiological and functional skeletal muscle adaptation more influenced by a training-induced catabolic or anabolic response?

The literature surrounding the influence of training-induced T and C on SMM is still limited. While some studies have explored the relationship between hormonal changes and aspects of skeletal muscle adaptation, a consensus has yet to be established on the influence of changes in T and C on both MS and hypertrophy at different phases of an athletic season. In this study, both MTH and MS were negatively associated with C during competition. Similar findings have been previously reported by Crewther, Obmiński, and Cook [1], Grandys, Majerczak, Kulpa, Duda, Rychlik, and Zoladz [16]. In the former, the authors suggest that changes in muscle performance may be moderated through high C levels despite anabolic increases. While in the latter it was observed that a greater decrease in C concentration is accompanied by poorer improvement in skeletal muscle. While both studies corroborate our initial hypothesis, neither explored changes in both strength and muscle size relative to hormonal changes as was carried out in this study. In doing so, we were able to highlight that despite a strong positive relationship between MTH and strength, both variables are independently influenced by T and C and that these relationships vary with the training phase. For example, our results indicate that C and MTH are negatively associated, and that C explained 60% variance in MTH during the competition phase only. Indeed, it is known that high C levels cause impairment of skeletal muscle contractile functions which inhibits muscle hypertrophy and strength. Therefore, it is possible that the observed maladaptation was indeed influenced by C levels. Moreover, the lack of association between T and SMM has also been previously reported [36]. The authors ultimately concluded that despite T concentrations elevating after exercise, the lack of association to muscle adaptation was not surprising and is likely a result of the fact that exercise-induced T elevations were too small and transient to pose any potent effects on hypertrophy [58]. This was possibly the case observed in this study as resting saliva samples were collected when T was at a nadir point in the day and at a point where athletes were exposed to the most rest. However, saliva samples for T and C were collected at the same time and based on the diurnal variation, C levels were also at a nadir point at the time of data collection. It can then be inferred that training-induced C changes have a greater influence and are more sensitive to physiological and functional skeletal muscle adaptation, compared to training-induced T changes.

In addition to the observed relationships between SMM and T and C, one of the novel findings of this study was that the relationship between performance and SMM was not influenced/moderated by C or T. To our knowledge, no other study has attempted to determine whether T or C is moderating the relationship between SMM and performance. It is however possible that, while T and C may not be moderating the relationships between performance and SMM, they may be influencing both variables independently. For example, within this study, C was observed to have a negative relationship with MTH and explained a 60% variance in MTH. Additionally, C also displayed a negative relationship with performance and further explains a 74% unique variance in performance. Previous studies reported seemingly contradictory results concerning the impact of C on performance. Where C was found to correlate positively with performance in weightlifters [59]. However, in sports such as tennis [60] golf [61], and track and field [34], a negative association has been observed. This discrepancy might reflect the different requirements in different sports. The challenges a weightlifter faces in the competition are both physically and mentally very different from those that occur during a golf tournament or 400m run. Further, while MTH and MS are key components of sprint performance, these variables may not be the intermediary through which T and C regulate performance in sprint junior athletes. Some studies have suggested other possible mechanisms which could explain how the hormones act as possible intermediaries to physical performance. For example, C can interact with androgen receptors [62] to modify the actions of T at the target tissue. While on a functional level, C can moderate the T relationship to status-relevant behaviors (e.g., aggression, dominance) [59,60,62] and anger-induced brain activity [61]. It remains then, that the influence of hormonal changes on performance extends beyond that of SMM. Indeed, Crewther, Obmiński, and Cook [1] concluded that while C is not strictly anabolic and catabolic markers of performance, respectively, they work in tandem to regulate several physiological systems that may reflect physical performance.

In summary, this study demonstrated that despite favorable anabolic changes, improvement in MTH and MS did not occur. Given this, it can be ascertained that 1) the catabolic state predominated, thereby inhibiting skeletal muscle adaptation, and that 2) T levels were too low the alleviate the catabolic response of C. It can then be implied that the C level during the competition phase, to some extent inhibited the anabolic response induced by the training regime. A possible consequence of reciprocal signaling between the hypothalamic-pituitary-adrenal and hypothalamic-pituitary-gonadal axes jointly regulating C and T release, respectively [63]. Our findings thereby suggest that T and C are not strictly anabolic and catabolic markers of SMM respectively, but rather they exert complementary actions that could depend on the task and environmental needs of athletes. Moreover, novel to this study, it was found that C had a greater influence on the SMM compared to T. As T was not a significant predictor of the SMM at any phase, while at the competition phase, C explained a 60% variance in MTH. Despite this, neither T nor C was observed to be significantly moderating the

relationship between SMM and sprint performance. Our findings also suggest that the relationship between SMM, T, and C varied with each training phase and that salivary C may be more sensitive in detecting changes in the SMM, compared to T among junior athletes. It is possible that the use of other possible anabolic markers such as IGF, and growth hormone to determine changes in SMM would be best. In fact, West and Phillips [36] suggested that other previously measured acute intramuscular markers, such as p70S6K1 phosphorylation, microRNA expression, and satellite cell activation, can yield relatively robust associations with hypertrophy.

The results of this study should be taken with caution, as there were some limitations present. The first of which is that both C and T, may be influenced by the presence of other hormones e.g., growth hormone, catecholamines, and insulin growth factor 1. As such, future studies should account for any possible moderating effect they may have on the steroidal hormones. Additionally, psychological factors, such as anxiety and mood state are known to influence C response. This may be accounted for in the future using questionnaires such as profile mood state which may be used to account for any influence these may have over C changes.

5. Practical Implications

Hypertrophic and strength changes are important aspects of sprint performance as they directly influence sprint determinants such as power and acceleration. Throughout an athletic season, sprint athletes are exposed to a wide range of exercise modalities during training. Extending from high volume, low-intensity exercises during the preparation phase to high intensity, low volume, high-intensity training during the competition phase. Training-induced changes in steroidal hormones such as T and C directly influence skeletal muscle adaptation and by extension sprint performance. Yet the majority of these studies exploring the relationship between hormonal changes and skeletal muscle have been among endurance and resistance-trained athletes. This study demonstrated that C levels increased in response to sprint training, to levels where it induced negative implications on both performance and skeletal muscle adaptation during the competition season. Such a catabolic physiological status resulted in significant losses in muscular force and MTH and may have possibly led to decreases in muscle endurance, strength, and speed. Assessment of the salivary concentrations of T and C, therefore, represents a possible means of monitoring the skeletal muscle status of junior athletes. Furthermore, measurement of both hormones and their influence on SMM may indicate any possible moderating influence these hormones may have on each other and what this means for skeletal muscle adaptation across a season.

6. Conclusion

Results indicate that despite a favorable anabolic response to training, and testosterone displaying moderate positive significant associations with MS, there is an overall decline in muscle function, indicating skeletal muscle maladaptation to the training regime. This was likely due to the increase in cortisol levels observed during competition, as C was found to be negatively associated with TMTH at this phase. Despite this, neither cortisol nor testosterone was found to moderate the relationship between SMM and performance. Of particular interest, however, cortisol was found to independently predict both variables, explaining a 60% variance in TMTH and 74% variance in performance. As such, rising testosterone levels across a season may be more indicative of a response to training load, than it is of skeletal muscle adaptation. While training-induced C changes possibly have a greater influence and is more sensitive to physiological and functional skeletal muscle adaptation, compared to training-induced T changes. Therefore, while these findings may support the use of cortisol as a possible monitor of skeletal muscle adaptation, this may not be possible with training-induced testosterone levels.

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