The Neuromuscular, Biochemical, and Endocrine Responses to a Single-Session Vs. Double-Session Training Day in Elite Athletes

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Abstract

Johnston, MJ, Cook, CJ, Drake, D, Costley, L, Johnston, JP, and Kilduff, LP. The neuromuscular, biochemical, and endocrine responses to a single-session vs. double-session training day in elite athletes. *J Strength Cond Res* 30(11): 3098–3106, 2016—The aim of this study was to compare the acute neuromuscular, biochemical, and endocrine responses of a training day consisting of a speed session only with performing a speed-and-weights training session on the same day. Fifteen men who were academy-level rugby players completed 2 protocols in a randomized order. The speed-only protocol involved performing 6 maximal effort repetitions of 50-m running sprints with 5 minutes of recovery between each sprint, whereas the speed-and-weights protocol involved the same sprinting session but was followed 2 hours later by a lower-body weights session consisting of 4 sets of 5 backsquats and Romanian deadlift at 85% one repetition maximum. Testosterone, cortisol, creatine kinase, lactate, and perceived muscle soreness were determined immediately before, immediately after, 2 hours after, and 24 hours after both the protocols. Peak power, relative peak power, jump height, and average rate of force development were determined from a countermovement jump (CMJ) at the same time points. After 24-hours, muscle soreness was significantly higher after the speed-and-weights protocol compared with the speed-only protocol (effect size $\eta^2 = 0.253$, $F = 4.750$, $p \leq 0.05$). There was no significant difference between any of the CMJ variables at any of the posttraining time points. Likewise, creatine kinase, testosterone, and cortisol were unaffected by the addition of a weight-training session. These data indicate that the addition of a weight-training session 2 hours after a speed session, whereas increasing the perception of fatigue the next day does not result in a difference in endocrine response or in neuromuscular capability.

Key Words testosterone, cortisol, creatine kinase, neuromuscular fatigue, speed

Introduction

Elite athletes are usually required to undertake a block of training that involves multiple high-intensity training sessions per day repeated over the course of 4–6 weeks to stimulate the adaptations required to improve performance (12). Neural adaptations in particular are reported to be sensitive to training intensity (42), and it is therefore important that sessions aimed at inducing neural adaptations are performed when athletes are in an optimal state.

A recent study into the pattern of neuromuscular recovery after speed training found countermovement jump (CMJ) performance, although depressed immediately after, to be significantly increased 2 hours after maximal speed training consisting of 6 × 50-m sprints when compared with 24 hours later (24). This finding would suggest that this 2-hour post-window might be an appropriate time point to perform a second intensive neuromuscular training session. Indeed, a number of studies have used multiple training sessions on the same day to optimize neural adaptation (11,18,21,23), and there is evidence of improvements in the isometric peak force of the knee extensors in both women (4.8 ± 5.0%) (18) and men (5.1 ± 10.2%) weightlifters (21) using this approach.

However, intensive dynamic training sessions result in inflammatory processes, which, in turn, can affect performance on subsequent training days (5). For example, isometric rate of force development has been shown to be depressed 24 hours after a strength session consisting of 10 sets of 3 repetitions at 90% of one repetition maximum

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(1RM) with 5 minutes of recovery between sets (27), whereas squat jump and CMJ performance were reported to be depressed 24 hours after a plyometric session consisting of 50 hurdle and 50 drop jumps (8).

To date, very few studies have examined the effects of multiple training sessions on neuromuscular performance and recovery (9,17,38,39). Of these, only 2 performed any sort of follow-up in the days posttraining (38,39). In both studies, the loss of performance evident after the second bout of exercise was no greater compared with the loss after the first (38,39). This lead the authors to conclude that this was because of the initial bout damaging the weak fibers and the stimulus from the second session being insufficient to produce any additional damage. However, it is unclear from these studies as to how neuromuscular performance was affected 24 hours later, and if any changes in neuromuscular performance at these time points would be different from those resulting from a single session. Having this information would allow the coach to make informed decisions about the use of twice-daily training and the placement and type of sessions they wish to have the athlete perform during the rest of the training week.

Furthermore, the majority of research conducted to date have used similar exercises and loadings in both training sessions (19). A multiple daily resistance session approach is commonly used by weightlifters (21), whereas the weekly training of an elite games player and a sprinter often requires them to undertake both lifting and running sessions on the same day (12,27). To date, no studies have investigated the effect of a training day containing speed-and-weights training sessions. Given that it has been suggested that changes in the contraction type (10) and variations in stimulus (38) are factors that exacerbate the inflammatory response, it is possible that a second session containing a significant change in stimulus may result in more muscle damage and a greater loss in neuromuscular performance.

Highly intensive sessions have also been shown to result in changes in serum testosterone (19) and salivary (12) and serum (8) cortisol release on subsequent training days. Acute changes in testosterone and cortisol have been not only linked to chronic adaptation (1) also been strongly linked to changes in acute neuromuscular function (11). Therefore, changes in both testosterone and cortisol levels in the days that follow intensive training may, in turn, influence the athlete's readiness to undertake further intensive training at these time points. To date, only Johnston et al. (24) have examined the endocrine response to maximal speed training over a 24-hour period. Johnston et al. reported no change in either plasma testosterone or cortisol in response to a session consisting of 6 × 50-m sprints with 5 minutes of recovery between repetitions 24 hours after completion. However, serum testosterone levels have been reported to be depressed 24 hours after a weight-training protocol (19), but elevated 24 hours after a plyometric protocol (8), whereas serum cortisol levels have been found to be elevated 24 hours after weight training (44), but depressed 24 hours after a concurrent strength and endurance session (37). It is also unclear as to what effect the addition of a second session would have on the endocrine response the next day.

Therefore, it is important to consider the combined effect of 2 sessions on both the neuromuscular and endocrine profiles, to determine if the second training session results will have an impact on the athlete's readiness to train in the hours or days that follow. If this were found to be the case, it would have important implications for the subsequent training days and competition preparation.

Given this, the aim of the current study was to investigate the effect of a 2-session training day (speed-and-weights) vs. a 1-session training day (speed only) on neuromuscular performance, markers of muscle damage, and hormone response.

Methods

Experimental Approach to the Problem

This study profiled a training day consisting of a single maximal speed training session and compared it with a training day consisting of a maximal speed training session followed 2 hours later by a heavy-weight training session. The decision to perform the second session 2 hours after the maximal speed training session was based on the findings of a previous study, which showed neuromuscular performance to have recovered by this time point after this type of session (23). The study was designed as a randomized crossover trial, and each experimental protocol was completed over 2 days. On the speed-only training day, baseline measurement (Pre) of lactate, perceived muscle soreness, creatine kinase, testosterone, cortisol, and CMJ performance preceded the maximal speed training session. These measurements were then recollected immediately (IPS), 2 hours and 24 hours after the completion of the training session.

During the speed-and-weights protocol, the same measurements were also collected Pre, IPS, and 2 hours after the maximal speed training. However, after the 2-hour postcollection, subjects completed a heavy-weight training session. Immediately after the completion of the weights session, subjects were retested for lactate, muscle soreness, and CMJ performance. Twenty-four hours after completion of the maximal speed training session, lactate, perceived muscle soreness, creatine kinase, testosterone, cortisol, and CMJ performance were all assessed for a final time. Countermovement jumps were processed for peak power (absolute and relative), jump height, and average rate of force development. Blood samples were analyzed for lactate, creatine kinase, testosterone, and cortisol.

Subjects

Fifteen academy-level rugby players from a professional rugby team were recruited for this study (mean ± *SD*: age 21 ± 1 year; age range: 19–22 years; 100.5 ± 10.5 kg; height 185.7 ± 6.6 cm). Each player had been involved in the professional academy system for a minimum of

Table 1. Mean 10 and 50-m times from the maximal speed training session performed during the speed-only and speed-and-weights protocols. Data are presented as mean ± *SD*.

	Speed-only	Speed-and-weights
10 m time (s)	1.80 ± 0.90	1.80 ± 1.10
50 m time (s)	6.57 ± 0.32	6.55 ± 0.34

2 years, during which time they were exposed to regular strength, power, and speed training and testing (mean ± *SD*: squat 1RM 170 ± 20 kg, bench 1RM 135 ± 10 kg, 10-m sprint time 1.75 ± 0.1 seconds). The study was undertaken at the end of the regular playing season, and subjects were performing physical training programs that consisted of speed, strength, and conditioning sessions 4 days per week. Subjects provided written informed consent, and a university research ethics committee provided ethical approval for the study.

Procedures

Before arriving at the indoor track on day 1 of each protocol, subjects were given 2 days off training. Each subject was given an arrival and start time, which was maintained throughout the study to account for circadian variation in hormones and body temperature (16). Upon arrival, subjects filled out a questionnaire on perceived muscle soreness and a blood sample was collected for subsequent analysis. Subjects then performed a 10-minute standardized warm-up before reporting to the testing area.

During each protocol, the first day's breakfast, lunch, snacks, and dinner along with the next day's breakfast were provided (Soulmate Food, Lancashire, United Kingdom). Both calorie intake and food choice were kept the same throughout both the speed-only and speed-and-weights protocols to ensure that the participant's nutritional intake was standardized throughout the study. Consumption of water was also allowed throughout the testing and training periods.

Maximal Speed Training. After running a specific warm-up consisting of 4 submaximal 50-m sprints interspaced with 2 minutes of recovery, subjects proceeded to an indoor track. After a 5-minute passive recovery period, a session consisting of 6 maximal 50-m sprints with 5-minute recovery between each run was completed. Each sprint started 30 cm behind the start line and was timed at 10 and 50 m using electronic timing gates (Brower timing system, Salt Lake City, UT, USA). The training parameters used reflected the subjects' normal speed training sessions and are in line with the volume of maximal speed running per session suggested by elite track and field coaches (15).

Weight Training. In the speed-and-weights protocol subjects undertook a typical lower-body strength training session, consisting of 5 sets of 4 repetitions of the backsquat and 5 sets of 4 repetitions of the Romanian deadlift all at 85% 1RM and with 4 minutes of recovery between sets. Each exercise was preceded by 2 sets of 4 at 50% and 70% 1RM by way of a warm-up. Subjects were regularly tested on their 1RM, and the percentages were calculated from tests performed within 3 weeks of the data collection. This session was performed 2 hours after the completion of the maximal speed training session.

Biochemical Testing. After lying in the supine position for 10 minutes, blood samples were collected from the antecubital vein to determine the acute responses of testosterone, cortisol, and creatine kinase, and each sample was taken by trained practitioners via venipuncture. After collection, the samples were centrifuged at 3,000 rpm for 10 minutes at room temperature. Plasma was analyzed for testosterone, cortisol, and creatine kinase activity using commercially available kits (Roche Diagnostic Limited, Charles Avenue, Burgess Hill, United Kingdom) on a Cobas C8000 analyzer (Roche

Table 2. Testosterone and cortisol response to speed-only and speed-and-weights protocols. Data are presented as mean ± *SD*.

	Before	Immediately after	After 2 h	After 24 h
Speed-only protocol				
Testosterone (nmol·L^{-1})	16.91 ± 4.16	19.51 ± 4.02*	16.52 ± 4.53	18.02 ± 4.59
Cortisol (nmol·L^{-1})	526 ± 94	404 ± 154	307 ± 83*	530 ± 96
Speed-and-weights protocol				
Testosterone (nmol·L^{-1})	16.31 ± 3.66	18.65 ± 3.97*	15.15 ± 5.06	17.38 ± 3.96
Cortisol (nmol·L^{-1})	491 ± 103	357 ± 114*	297 ± 73*	520 ± 106

*Significant at 0.05 when compared with immediately before.

Table 3. Neuromuscular responses to the speed-only and speed-and-weights protocols. Data are presented as mean ± *SD*.*

	Before	Immediately after	After 2 h	Post weights	After 24 h
Speed-only protocol					
CMJ peak power (W)	5,345 ± 477	5,066 ± 467†	5,439 ± 437		5,202 ± 458†
CMJ jump height (m)	0.39 ± 0.06	0.35 ± 0.07†	0.39 ± 0.06		0.37 ± 0.06†
CMJ aRFD ($n \cdot s^{-1}$)	4,688 ± 1,570	4,591 ± 1,004	4,838 ± 1,535		4,528 ± 1,497
CMJ relative peak power ($W \cdot kg^{-1}$)	54.80 ± 6.76	52.03 ± 6.76†	55.70 ± 6.95		53.37 ± 7.23†
Speed-and-weights protocol					
CMJ peak power (W)	5,371 ± 452	5,109 ± 474†	5,408 ± 429	5,037 ± 429†‡	5,174 ± 415†
CMJ jump height (m)	0.40 ± 0.05	0.37 ± 0.06†	0.39 ± 0.06	0.36 ± 0.05†‡	0.37 ± 0.06†
CMJ aRFD (total) ($N \cdot s^{-1}$)	4,973 ± 1,504	4,742 ± 944	4,913 ± 1,218	4,492 ± 1,194	4,342 ± 1,102†
CMJ relative peak power ($W \cdot kg^{-1}$)	55.42 ± 6.15	52.54 ± 6.95†	55.47 ± 6.78	51.55 ± 5.40†‡	53.32 ± 6.63

aRFD = average rate of force development.
*CMJ = countermovement jump.
†Significant difference from immediately before (0.05).
‡Significant difference from 2 h post (0.05).

Diagnostics, Risch-Rotkreuz, Switzerland). Lactate was analyzed from blood taken from a capillary using a lactate analyzer (Lactate pro; Arkray, Tokyo, Japan).

Neuromuscular Performance. The CMJ tests were performed on a force platform (type 9287CA; Kistler Instruments Ltd., Franbourgh, United Kingdom). It has previously been reported that CMJ correlates well with dynamic performance (45), making it a relevant marker for the assessment of neuromuscular function. A sample rate of 1,000 Hz, and a vertical force range of 20 kN were used for all trials, in accordance with previous research (32). After collection, the vertical component of the ground reaction force (GRF) time history was exported to a custom-built excel sheet for analysis. Body mass, jump start time, and takeoff were calculated using methods previously described (41).

To calculate jump height, maximal vertical displacement was first calculated using the impulse momentum method. Jump height (m) was then defined as the difference between vertical displacement at takeoff and maximal vertical displacement. Data collected from a pilot study found the test-retest reliability of jump height using this method to be high (intraclass correlation [ICC] 0.93). To calculate peak power, instantaneous power was first calculated by multiplying vertical GRF by the vertical velocity of the center of gravity. Relative peak power was calculated by dividing the peak power by the body weight, in kilograms. Peak power and relative peak power were both found to have test-retest ICCs of 0.96 during a pilot study.

Countermovement jump average rate of force development was calculated using a published method (43) and was defined as the change of force during the eccentric deceleration phase divided by the time of the eccentric deceleration phase. The eccentric deceleration phase was defined as the point at which the force passed through body weight

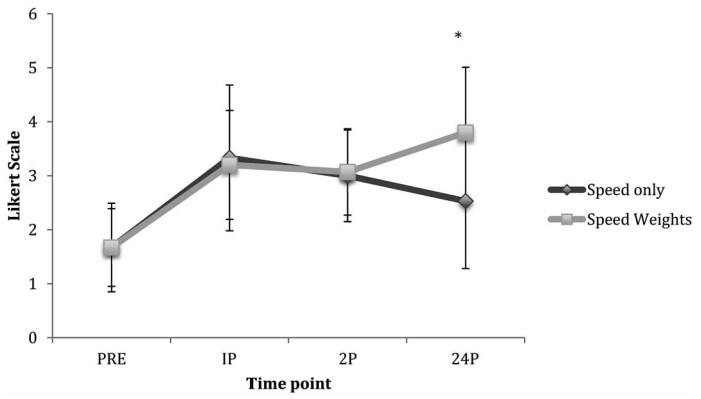

Figure 1. Muscle soreness before, immediately after (IP), 2 hours later (2P), and 24 after (24P) the speed-only and speed-and-weights protocols. *Significant difference between protocols.

Table 4. Lactate, creatine kinase, and muscle soreness response to speed-only and speed-and-weights protocols. Data are presented as mean ± *SD*.*

	Before	Immediately after	After 2 h	Post weights	After 24 h
Speed-only					
Lactate (mmol·L^{-1})	1.33 ± 0.38	9.32 ± 1.65†	1.55 ± 1.05		1.05 ± 0.71
Creatine kinase (U/L)	498 ± 284	561 ± 301†	603 ± 302†		955 ± 876†
Muscle soreness (Likert)	1.67 ± 0.72	3.33 ± 1.35†	3.00 ± 0.85†		2.53 ± 1.25
Speed-and-weights					
Lactate (mmol·L^{-1})	1.50 ± 0.72	9.41 ± 1.38†	1.41 ± 0.64	2.45 ± 1.19†‡	0.89 ± 0.49
Creatine kinase (U/L)	485 ± 420	582 ± 454†	589 ± 423†	n/a	1,161 ± 816†
Muscle soreness (Likert)	1.67 ± 0.82	3.20 ± 1.01†	3.07 ± 0.80†	4.10 ± 1.95†	3.80 ± 1.21†§

*n/a = not applicable.
†Significant difference from pre-speed training.
‡Significant difference from 2P speed training.
§Significant difference from 24P speed training.

during the eccentric phase, through to the point when displacement became positive. The ICC for this method was found to be 0.92 during a pilot study.

Perceived Muscle Soreness. Perceived muscle soreness was recorded at each data collection point, using a 7-point Likert scale designed to measure soreness in the lower body. The scale ranged from very, very good (1) to very, very sore (7). The use of this Likert scale is supported by previous research in the area (2). The subjects were asked to base their scores on perceived soreness during normal movement and were alone when questioned to reduce the desire to provide favorable scores in front of their peers.

Statistical Analysis

Data are expressed in their recorded form as the mean ± *SD*. After tests for normal distribution, and before any further statistical analysis, creatine kinase recorded values were log transformed because of large interparticipant variability. Differences between and within protocols were assessed using a 2-way (time point and protocol) repeated-measures analysis of variance (ANOVA). If significant F values were observed ($p \leq 0.05$), a repeated-measure 1-way ANOVA was used in conjunction with Holm-Bonferroni method for control of type I error to determine where significant differences occurred. Effect size was determined using η^2 with an effect size of approximately 0.2 considered small, approximately 0.5 considered medium, and approximately 0.8 considered large. The level of significance was set at $p \leq 0.05$ for the present study, and all statistics were performed using SPSS 20.0 (SPSS, Inc., Chicago, IL, USA).

Results

Sprints

The mean 10- and 50-m times for the speed-only and speed-and-weights protocols can be seen in Table 1, and 10- and 50-m times were not found to differ between the protocols.

Endocrine Response

The endocrine data for the 2 protocols are presented in Table 2. Analysis revealed a significant time effect for both testosterone (effect size $\eta^2 = 0.530$, $F = 15.797$, $p \leq 0.05$) and cortisol (effect size $\eta^2 = 0.673$, $F = 28.824$, $p \leq 0.05$), with testosterone found to be significantly elevated immediately after the maximal speed training part of both protocols, whereas cortisol, in contrast, was significantly lower at the same time point during the speed-and-weights protocol but unchanged during the speed-only protocol.

After 2 hours, testosterone was no longer significantly different from baseline values, whereas cortisol had dropped significantly in both protocols. After 24 hours, testosterone and cortisol did not differ from pretraining levels in either protocol (Table 2).

No protocol-time interaction was found for either testosterone (effect size $\eta^2 = 0.025$, $F = 0.366$, $p > 0.05$) or cortisol (effect size $\eta^2 = 0.049$, $F = 0.722$, $p > 0.05$).

Neuromuscular Performance

The CMJ data are presented in Table 3. No protocol-time interaction was found for any of the CMJ variables. However, there was a time effect for peak power (effect size $\eta^2 = 0.733$, $F = 38.456$, $p \leq 0.05$), jump height (effect size $\eta^2 = 0.575$, $F = 18.966$, $p \leq 0.05$), average rate of force development (effect size $\eta^2 = 0.170$, $F = 2.860$, $p \leq 0.05$), and relative peak power (effect size $\eta^2 = 0.732$, $F = 38.216$, $p \leq 0.05$).

As can be seen in Table 3, several CMJ variables were found to have declined from their baseline values immediately after the maximal speed training session, before returning to baseline values after 2 hours. When observed 24 hours later, several jump variables were again found to be depressed vs. pretraining values, indicating a second decline in neuromuscular performance (Table 3).

During the speed-and-weights protocol, an additional measure of jump performance was taken immediately after the weights session. At this time point, peak power, jump

height, and relative peak power were significantly lower than both the pretraining and 2-hour postlevels (Table 3).

Creatine Kinase, Lactate, and Muscle Soreness

A protocol-time interaction was found for perceived muscle soreness (effect size $\eta^2 = 0.253$, $F = 4.750$, $p \leq 0.05$), but not for lactate or creatine kinase. Further analysis revealed that the speed-and-weights protocol resulted in significantly higher levels of perceived muscle soreness after 24 hours than in the speed-only protocol (Figure 1).

Significant time effects were found for lactate (effect size $\eta^2 = 0.975$, $F = 540.593$, $p \leq 0.05$), perceived muscle soreness (effect size $\eta^2 = 0.537$, $F = 16.205$, $p \leq 0.05$), and creatine kinase (effect size $\eta^2 = 0.503$, $F = 14.155$, $p \leq 0.05$) (Table 4).

Lactate levels were elevated immediately after the speed training sessions but not at either 2 or 24 hours later in either protocol (Table 4). Creatine kinase response was significantly elevated at immediately after, 2 hours later, and 24 hours after the maximal speed training session in both protocols.

Discussion

The present study is the first to compare the temporal responses of various neuromuscular, biochemical, and endocrine parameters from a training day consisting of a speed session performed in isolation with a training day containing 1 speed and 1 weight-training session separated by 2 hours. The main finding from the study was that, although the addition of a lower-weights session 2 hours after a speed training session did result in an increase in perceived muscle soreness after the 24-hour time point, it did not result in any additional changes in hormonal, biochemical, or neuromuscular response over the course of the 24-hour measurement period.

Immediately after the maximal speed training session in both protocols, several of the CMJ parameters had declined significantly when compared with pretraining levels. These initial depressions in neuromuscular performance were accompanied by significant elevations in plasma creatine kinase, blood lactate, and perceived muscle soreness.

When measured 2 hours after the maximal speed training session, blood lactate and jump performance had returned to pretraining levels in both protocols, whereas plasma creatine kinase and perceived muscle soreness continued to increase (Table 4). When considered alongside the recovery of neuromuscular performance, our finding that blood lactate had returned to baseline levels 2 hours after the maximal sprint session suggests that, at least in part, the decreased jump performance observed immediately after maximal speed training was because of decreased functioning of the contractile mechanisms of the muscle fiber (40) in the presence of the metabolites produced during exercise.

Several CMJ parameters were also found to be depressed after the weight-training session performed during the speed-and-weights protocol (Table 3). When these postweights session depressions in performance were compared with the drops experienced immediately after maximal speed training, no significant differences were found. This is consistent with the results of previous research (9,17,38,39) which, when using different measures of neuromuscular performance, also reported no significant difference in the losses experienced after each of the 2 training sessions performed on the same day. However, the current study is the first to report these findings after a training day consisting of a speed-and-weights training session, which is a common approach and is recommended by elite coaches (15).

To date, only Häkkinen et al. (20) have reported the blood lactate response to multiple daily sessions. In their study, they compared 2 strength sessions consisting of a mix of Olympic and strength lifts and found no difference between the postsession metabolic responses. This is in contrast to our findings, where a significant difference in the postsession blood lactate levels was observed. This observed difference in the metabolic response to the 2 sessions is an interesting finding, given that the recoveries were the same and the duration of the efforts were shorter during the sprint training. It appears that even though the duration of efforts performed during the maximal speed training would have been expected to primarily tax the adenosine triphosphate phosphocreatine system, and the between-effort recoveries of 5 minutes would have been expected to allow significant creatine phosphate replenishment, the high observed blood lactates of 9.31 ± 1.65 mmol (speed-only) and 9.41 ± 1.38 mmol (speed-and-weights) would suggest that replenishment did not occur. Research indicates that 3×100-m sprints produce greater blood lactate levels than 1 effort of 300 m (35). The authors suggest that this was a result of the 3×100-m protocol allowing their subjects to operate at higher speeds over the same total distance and that the repeated maximal efforts performed throughout the speed session most likely resulted in a significant posteffort energy demand. Given this, it seems likely that the fatigue observed after the maximal speed training was peripheral in origin.

In contrast, significantly lower blood lactates were observed immediately after the weight training. Research indicates that variations in the metabolic demand of exercise can result in different mechanisms of fatigue, even when the decreases in neuromuscular performance are similar (26). Furthermore, it is reported that central rather than peripheral mechanisms are the primary cause of the depressions in neuromuscular performance that occur as the result of high-intensity strength training (17,26). Therefore, although similar decreases in neuromuscular performance were observed after both sessions in our study, it is possible that different mechanisms may have contributed to these decreases.

In the current study, although several of the jump variables were depressed after the 24-hour time point in response to both protocols (Table 2), there was no significant difference between the protocols with regard to the degree

of depression experienced. Given this, the results from the CMJs suggest that the addition of a weight-training session 2 hours after maximal speed training does not result in a greater loss in neuromuscular performance after 24 hours. The finding that several jump variables underwent a secondary decline in response to the speed-only protocol confirms our previous findings (24) and suggests that maximal speed training induces a bimodal recovery pattern in this population. The depressions in performance after 24 hours were accompanied by elevations in both creatine kinase and perceived muscle soreness in both protocols, indicating significant muscle damage. It has been reported that it is the inflammatory response to muscle damage as opposed to the muscle damage itself that ultimately affects muscle performance (14). Although absent after 2 hours, this inflammatory response would be expected to be well underway after 24 hours (4) and, as such, represents the most likely explanation for the secondary decline in neuromuscular performance observed.

Interestingly, after 24 hours, there was a significant difference between the protocols in terms of perceived muscle soreness, but not plasma creatine kinase. Whereas perceived muscle soreness is often presented as a marker of muscle damage (30), previous research has reported perceived muscle soreness to provide a poor reflection of the degree of muscle damage and inflammation experienced (31), and as such, a distinction should be drawn between the 2. Given this, their roles in the development of fatigue may also be different. For example, a significant decrease (15%) in maximal voluntary torque has been demonstrated to correlate with elevated levels of perceived muscle soreness (34). However, the authors reported that this correlation was because of the subjects' reducing exercise intensity on a conscious and/or unconscious level, rather than via an acute exercise-related physiological or biochemical alteration. Nevertheless, in the current study, the majority of jump variables showed no difference in the degree of decline despite the difference in perceived muscle soreness. One possible explanation for this may be that trained athletes performance is less affected by perceived muscle soreness. This is supported by the findings of a study that tracked maximal voluntary isometric force and perceived muscle soreness in both trained and untrained subjects for 5 days after an eccentric protocol designed to induce muscle damage (29). The study reported that, although both groups reported similar levels of perceived muscle soreness, neuromuscular performance returned much quicker in the trained group.

It is important to highlight that, in the current study; the weight-training session followed the maximal speed training. Eccentric stress is reported to be one of the main mechanisms behind muscle damage/inflammation (8), and it is unclear if changing the exercise session order would have had an effect on the degree of muscle damage, perceived muscle soreness, and loss of performance experienced after 24 hours.

The current study also set out to compare how the 2 training protocols affected endocrine response. Plasma levels of testosterone significantly increased immediately after the speed training sessions, whereas plasma levels of cortisol did not increase (Figure 1). This finding is in contrast to previous work into the endocrine response to maximal speed training. Pullinen et al. (33) reported significant postsession increases in both serum testosterone and cortisol levels immediately after a training session consisting of 10 × 50-m sprints with 4 minutes of recovery between repetitions, whereas Johnston et al. (24) reported no change in plasma levels of either marker in response to a session consisting of 6 × 50-m sprints with 5 minutes of recovery between repetitions. However, an increase in salivary testosterone coupled with a decrease in cortisol has been reported after resistance training (6), and a similar response in serum levels has been reported after a repeated sprint training session consisting of 4 × 250-m sprints with 3 minutes of recovery between repetitions (28).

The exact reason why cortisol did not respond immediately after the initial training session and testosterone did is unclear. Our initial baselines were taken immediately before the start of each protocol and, as such, it is possible they are unrepresentative of resting cortisol levels because of the subject's anticipation of the training sessions. However, it is unclear how much presession anticipation effects cortisol levels with a recent study into the endocrine response to a powerlifting competition reporting that preexercise anticipation did not cause elevations in salivary cortisol in all cases (25). Alternatively, it has been suggested that there is a training load "threshold" upon which the hypothalamic adrenal axis is activated (7), and it is possible that the low volume (6 × 50 m) in the current study was insufficient to activate it.

Testosterone has been reported to have several fast-acting nongenomic effects, including several related to muscle function (13). Given this, it is possible that instead of being a direct response to session volume, the post sprint training increases in testosterone occurred to support the effort to sustain neuromuscular performance throughout the session.

Considering the maximal speed training sessions performed in both the current study and in Johnston et al. (24) were identical, it is curious that they resulted in different testosterone responses. Testosterone response to a training stimulus is reported to be dependent on training background (1) and, in the current study, was considerably stronger with a 1RM squat and bench personal bests of 170 ± 20 kg and 135 ± 10 kg. In contrast, the subjects in Johnston et al. (24) had reported 1RM squat of 150 ± 22 kg and a reported 1RM bench of 121 ± 15 kg. Therefore, this difference in training level may have contributed to the differences in postexercise elevations observed.

Although the lack of a control group represented a limitation in the current study, previous research into the circadian pattern of testosterone suggests that declines in

levels would normally occur over the timeframes our data were collected (22). Given this, the lack of decrease observed after 2 hours in the current study may actually be viewed as an elevation vs. the levels that would have been expected without the sprint training session. Cortisol, conversely, appeared to follow the expected circadian pattern and was significantly depressed 2 hours after the maximal speed training in both protocols. Although the degree to which the sustained postexercise elevations in testosterone observed may or may not be directly involved in inducing muscle protein synthesis is subject to controversy (36), acute variations have been suggested to play a role in other adaptations relevant to strength/power athletes (3). For instance, it has been demonstrated that altering the normal circadian pattern of salivary testosterone with a morning weight-training session correlated with improved afternoon sprint performance in men rugby players (11). It cannot, therefore, be ruled out that the postexercise testosterone response observed in the current study may have resulted in a superior training or competitive environment. If so, this may have implications for training order and, potentially, precompetition preparation.

Previously, variations in testosterone and/or cortisol hormones in the days after training have been thought to give an indication of training stress (8). In the current study, the levels of both the hormones were not different from pretraining levels when assessed after 24 hours, and there was no difference in the response between the protocols. This would suggest that the addition of a second training session does not affect hormonal levels the next day. However, further research is required to see if this pattern continues in the long term or if continuously performing multiple training sessions per day does induce altered hormonal responses in the long term.

In conclusion, our primary finding is that the addition of a weight session 2 hours after an initial maximal speed session did result in an increase in perceived muscle soreness. However, this increase in muscle soreness did not result in any increased loss of neuromuscular performance or difference in the endocrine or biochemical responses. One possible explanation for this is that the weight training was less damaging than the maximal speed training and, as a result, any damage that was done during the speed-and-weights protocol had already been done before the weight-training session. However, further research is required to assess if indeed these findings were influenced by session order.

Practical Applications

Athletes are often required to undertake training sessions aimed at developing several different physical qualities in the same day and/or week. This study shows that 2 hours was sufficient for the neuromuscular system to recover from a maximal speed session. In addition, the performance of weight training 2 hours after speed training does not result in any difference in the biochemical or neuromuscular markers assessed 24 later when compared with a training day consisting only of maximal speed training. This has implications for the programming of the training day and week as compressing the weight and speed training into a single training day does not seem to result in additional fatigue or damage. Finally, given that neuromuscular performance was depressed the day after maximal speed training, this 2 a day training approach may actually promote superior adaptation.

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