Neuromuscular adjustments of the knee extensors and plantar flexors following match-play tennis in the heat

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ABSTRACT

Objectives This study tested the hypothesis that impairments in lower limb maximal strength and voluntary activation (VA) are exacerbated following match-play tennis in hot compared with cool conditions.
Methods Torque and VA were evaluated during brief (5 s) and sustained (20 s) maximal voluntary isometric contractions of the knee extensors (KE) and plantar flexors (PF) in 12 male tennis players before (pre) and after (post, 24 h and 48 h) ~115 min of play in hot (~37°C) and cool (~22°C) conditions.
Results Rectal temperature was higher following play in hot than in cool (~39.2 vs ~38.5°C; p<0.05). Torque production decreased from prematch to postmatch during the brief and sustained contractions in hot (KE: ~22%; PF: ~13%) and cool (KE: ~9%, PF: ~7%) (p<0.05). KE strength losses in hot were greater than in cool (p<0.05) and persisted for 24 h (p<0.05). Postmatch brief and sustained KE VA was lower in hot than in cool (p<0.05), in which VA was maintained. PF VA was maintained throughout the protocol. Peak twitch torque and maximum rates of torque development and relaxation in the KE and PF were equally reduced postmatch relative to prematch in hot and cool conditions (p<0.05), and were restored near baseline within 24 h.
Conclusions Neuromuscular system integrity of the lower limbs is compromised immediately following match-play tennis in hot and cool conditions due to the development of peripheral fatigue. The larger and persistent KE strength losses observed under heat stress are associated with greater levels of central fatigue especially during sustained contractions.

INTRODUCTION

Tennis is an intermittent activity that interposes short bouts (3–10 s) of high-intensity exercise interspersed with light activity or rest (20–25 s) for a protracted period of time (1–5 h).[1–3] The cumulative effect of repeating these high-intensity efforts is manifested by the development of fatigue and suboptimal performances.[4 5] Owing to the multifactorial nature of the game, fatigue can originate from a variety of sources (eg, cardiovascular, metabolic, neural or psychological) and/or the attainment of various states (eg, hyperthermia, dehydration or hypoglycaemia).

From a neuromuscular perspective, fatigue is defined as an exercise-induced decrement in the capacity to produce/maintain maximal force or power.[6] It is purported that sources proximal (ie, central fatigue) and distal (ie, peripheral fatigue) to the neuromuscular junction modulate this decrement. Correspondingly, it has been reported that neuromuscular function deteriorates during and following prolonged (~3 h) match-play tennis in temperate conditions.[7–10] More specifically, it was shown that maximal strength of the knee extensors (KE) and plantar flexors (PF) decreases during brief maximal voluntary isometric contractions (MVCs) in association with a reduction in voluntary muscle activation (VA), adjustments in contractile function and/or the development of low-frequency fatigue, depending on the muscle group.[7–10] This latter form of fatigue is characterised by a proportionally greater loss of force at low frequencies of muscle stimulation and a slow recovery over the course of several hours or even days.[11] Alternatively, reductions in VA measured via twitch interpolation are indicative of a loss in neural drive to active muscles during an effort. Accordingly, these central and peripheral alterations have the potential to negatively influence the efficiency of on-court movements and stroke proficiency,[9] especially during multiple-match tournament play.

Interestingly, the recovery kinetics of the KE and PF following match-play tennis remain relatively unknown, having only been studied 30 min after play.[8–10] Although a recent study has shown that bilateral leg press force is reduced immediately after the first match of a 3-day tournament and remains depressed prior to the start of play in the following matches (ie, days 2 and 3),[12] measures of central and peripheral fatigue were not conducted. As such, the origin of fatigue, along with the extent and timeframe of these potential alterations, as well as the contribution of different muscle groups were not elucidated.

Moreover, there is strong evidence to suggest that central fatigue is exacerbated by hyperthermia per se (ie, passive heating).[13–18] However, the precise influence of exercise-induced hyperthermia on central activation deficit remains contentious. For example, the ability to generate force during a sustained MVC was shown to decline after constant rate exercise in hot relative to cool conditions, due to a hyperthermia-induced reduction in VA.[19] Conversely, force production was similarly impaired following self-paced and incremental exercise in hot and cool conditions, with a level of central fatigue that was equivalent.[20 21] After a soccer match, a high-intensity intermittent activity more closely comparable to tennis, decrements in torque production and VA were similar between matches played in hot and cool conditions, and were restored within 24 h.[22]

Therefore, the aim of this study was to test the hypothesis that impairments in lower limb strength

and VA are exacerbated following play in hot conditions compared with cool conditions. A secondary aim was to test hypothesis that the time course of recovery in neuromuscular function is impaired over a 48 h period. From a tennis performance perspective, acute (ie, immediately postmatch) and delayed (ie, 24–48 h into recovery) lower-limb strength losses, along with central activation deficits following match-play in the heat, could have important implications for competition preparation and recovery.

METHODS

Subjects

Twelve male players with an International Tennis Federation number of 1 to 3 (ie, nationally ranked players, or having professional tournament experience) participated in the study. Mean age, height and body mass were 22±4 years, 183.5±7.7 cm and 80.8±9.5 kg. The participants were informed of the study aims, requirements and risks before providing their written informed consent.

Study design

The participants played two counter-balanced simulated tennis matches on hard-court surfaces separated by 72 or 144 h. They were paired according to their level of play and competed against the same opponent in each match. One match was played indoors in temperate conditions (cool: 21.8±0.1°C, 72.3 ±3.2% relative humidity) and the other outside in hot conditions (hot: 36.8±1.5°C, 36.1±11.3% relative humidity). The matches consisted of 20 min (2×10 min) of effective play: the percentage of time spent within play and excluding the time between points and games. One to 3 days prior to the start of the study the participants visited the testing and playing venue where they were thoroughly familiarised with the testing procedures and neuromuscular function assessment protocol (see Motor nerve stimulation section) until they felt accustomed with the equipment (ie, coefficient of variation in three successive MVC trials lower than 5%).

Experimental protocol

Upon arrival on match days (09:00), participants voided and inserted a telemetric temperature pill (VitalSense, Mini Mitter, Respironics, Herrsching, Germany) in the rectum (the length of a gloved index finger beyond the anal sphincter) for the measurement of core temperature. A standardised warm-up of running at 9 km/h for 5 min on the indoor court was then performed. This was followed by the pre-exercise (pre) neuromuscular function assessment (see Neuromuscular function section). This assessment, along with the 24 and 48 h postmatch assessments, was preceded by a standardised warm-up consisting of 10 isometric contractions of 4 s in duration interspaced with 10–20 s of recovery. Contraction intensity was progressively self-adjusted by the participant to attain maximal torque in the last three contractions. The neuromuscular function assessment was repeated ∼20 min after play (post). Following the neuromuscular assessment, the participants performed a 10 min tennis-specific warm-up consisting of various exchanges and serves in the condition of play. Core temperature and body mass were recorded prematch and postmatch. During play, the participants consumed water and a commercially available sport drink (Gatorade, Chicago, Illinois, USA) ad libitum. On the days when the participants did not play they performed the running warm-up and the neuromuscular assessment (24 and 48 h) at the same time of day as prematch, and then followed a standardised training programme led by a tennis coach (∼60 min of drills and exchanges).

Neuromuscular function

The neuromuscular assessment protocol is presented in figure 1. The protocol was performed in a counter-balanced order between participants, once to evaluate the KE and once to evaluate the PF of the right leg. First, a brief (5 s) MVC was performed on which a paired stimulus (doublet, 100 Hz) was superimposed. This was followed by a doublet and three single pulses on relaxed muscle, each interspersed by 5 s. After repeating this initial part of the protocol and following a 60 s rest interval, a sustained (20 s) MVC was performed with a doublet superimposed at ∼2 and ∼18 s. In addition, a potentiated doublet was delivered 5 s after the sustained MVC. The sustained MVC was included in the assessment as it is suggested to better evaluate central activation failure.[23] The duration of the entire neuromuscular assessment (ie, for both muscle groups) was ∼10 min and was conducted in a temperate environment (∼22°C). During all MVCs, the participants were instructed to reach maximal torque as quickly as possible and maintain this level for the duration of the contraction. They were strongly encouraged with verbal feedback and a visual display of the torque production.

Torque measurements

For KE torque measurement, participants were seated upright on a custom-built adjustable chair with the hips and knees flexed at 90°. Restraining straps placed across the chest and hips secured the participants in the chair to prevent extraneous movement, while the dynamometer (Captels, St Mathieu de Treviers, France) was attached 3–5 cm above the tip of the lateral malleoli. PF torque was measured using a dynamometric pedal (Captels, St Mathieu de Treviers, France) with the participants seated upright with the hips, knee and ankle flexed at 90°, 100° and 90°, respectively. The foot of the leg performing the MVC was secured to the dynamometric pedal with two restraining straps. During all contractions, the torque signals were amplified, sent through an A/D board and sampled at 2000 Hz by commercially available hardware and software (MP35 and BSL Pro V.3.6.7, Biopac Systems Inc., Santa Barbara, California, USA).

Motor nerve stimulation

A high-voltage stimulator (Digitimer DS7AH, Digitimer, Hertfordshire, England) was used to deliver a square-wave stimulus of 0.2 ms duration with a maximal voltage of 400 V. The femoral nerve was stimulated by placing a cathode (5 mm diameter) in the inguinal crease and an anode (5×10 cm; Medicompex SA, Ecublens, Switzerland) in the gluteal fold. The
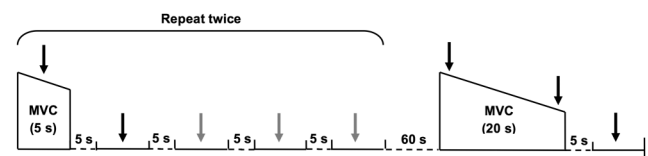

Figure 1 Neuromuscular function assessment protocol performed prior to and following 20 min of effective match-play tennis (2×10 min) in cool and hot conditions with the knee extensors and plantar flexors. A 5 s maximal voluntary isometric contraction (MVC) was performed and superimposed with motor nerve stimulation. This was followed by four stimulations at rest. After repeating this procedure, a 20 s MVC was performed with stimulations superimposed at 2 and 18 s. A final potentiated stimulation was evoked 5 s after the sustained MVC. Single (1 Hz: ↓) and paired (100 Hz: ↓) stimulations.

tibial nerve was stimulated using a cathode (9 mm diameter) placed in the popliteal cavity with compression supplied by a strap, and an anode (5×10 cm) positioned beneath the patella. During the familiarisation session, an isometric recruitment curve using motor nerve stimulation was drawn on relaxed KE and PF muscles to individualise the optimal stimulus intensities. Briefly, the current was progressively increased in 10 mA increments until plateau occurred in maximal twitch amplitude. Supramaximal stimulations were ensured by increasing the final intensity by 50%, and kept constant for each participant throughout all experimental trials.

Neuromuscular data analysis

All analyses were performed using Spike 2 Software (Cambridge Electronic Design, Cambridge, UK). During the MVCs, torque production was recorded during a 500 ms plateau prior to delivering the motor nerve stimulation. VA was calculated with the interpolated twitch technique using the potentiated doublet, as VA (%) =(1−(superimposed doublet/resting potentiated doublet))×100.[24] Contractile characteristics were assessed from the electrically evoked resting twitch as peak twitch torque, contraction time to peak torque, half-relaxation time and maximum rates of torque development and relaxation (ie, the steepest rates of torque production and decline, respectively). The better of the two brief MVCs was selected to assess brief maximal voluntary torque production and VA, whereas the mean of the six twitches was used to analyse contractile properties.

Statistical analysis

All statistical calculations were performed using PASW software V.21.0 (SPSS, Chicago, Illinois, USA). Repeated-measures analysis of variance were performed to test the significance between and within treatments. Outcome variables were tested using Mauchly's procedure for sphericity. Whenever the data violated the assumption of sphericity, p values and adjusted degrees of freedom based on Greenhouse-Geisser correction were reported instead. Where significant interaction effects were established, pairwise differences were identified using the Bonferroni post hoc analysis procedure adjusted for multiple comparisons. The significance level was set at $p < 0.05$. All values are expressed as means±SD.

RESULTS

Match-play responses

Match duration, the time to complete 20 min of effective play, was longer in the hot condition (124.2±9 min) than in the cool condition (107.1±19 min) ($p < 0.05$). The increase in core temperature from prematch to postmatch was greater in the hot condition (37.6±0.3 to 39.4±0.5°C) relative to the cool condition (37.5±0.3 to 38.7±0.2°C; $p < 0.05$). Core temperature during the neuromuscular assessment was also higher following play in the heat (39.2±0.5 vs 38.5±0.3°C; $p < 0.05$). The level of body mass deficit was similar between the hot (−0.5±1.3 kg) and cool (−0.3±0.3 kg) conditions, as the 0.7±0.4 L/h greater sweat rate observed in the hot condition was compensated for by a greater fluid consumption of 0.9±0.5 L/h ($p < 0.001$).
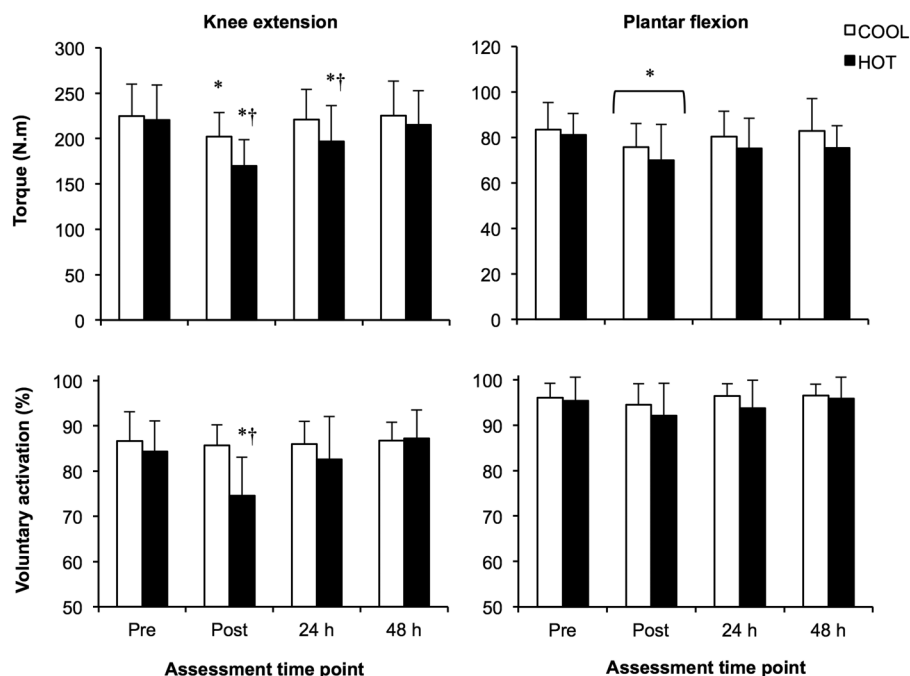
Maximal voluntary contractions

Knee extension

Compared with prematch values, larger reductions in brief KE torque production occurred postmatch in the hot condition (−22.0±10.9%) than in the cool condition (−9.5±6.9%; figure 2; $p < 0.05$). KE torque was also lower in the hot condition at 24 h relative to prematch (−10.6±7.3%) and cool (−11.2±9.1%) condition values ($p < 0.05$). VA during the brief KE MVC was maintained throughout the cool condition, whereas it was reduced postmatch in the hot condition compared with prematch (−11.4±9.1%) and cool (−12.9±9.1%) condition values ($p < 0.05$). Overall, KE torque production and VA were lower throughout the hot vs cool condition ($p < 0.05$).

Sustained KE torque production decreased similarly (−5% to 12%) throughout all MVCs (ie, from 2 to 18 s) (figure 3; $p < 0.01$). As with the brief MVCs, a decrease in mean sustained torque was observed postmatch relative to prematch in hot (−21.5±11.0%) and cool (−8.0±7.9%) conditions ($p < 0.05$). Mean KE-sustained torque production was also reduced 24 h after match completion in the hot condition, relative to prematch ($p < 0.01$). Torque production postmatch and at 24 h in

Figure 2 Torque production and voluntary activation during a brief (5 s) maximal voluntary isometric contraction of the knee extensors (left panels) and plantar flexors (right panels) performed prior to (pre) and following (post, 24 and 48 h) 20 min of effective match-play tennis in cool and hot conditions. *Significantly different from pre, $p < 0.05$. †Significantly different from cool, $p < 0.05$.
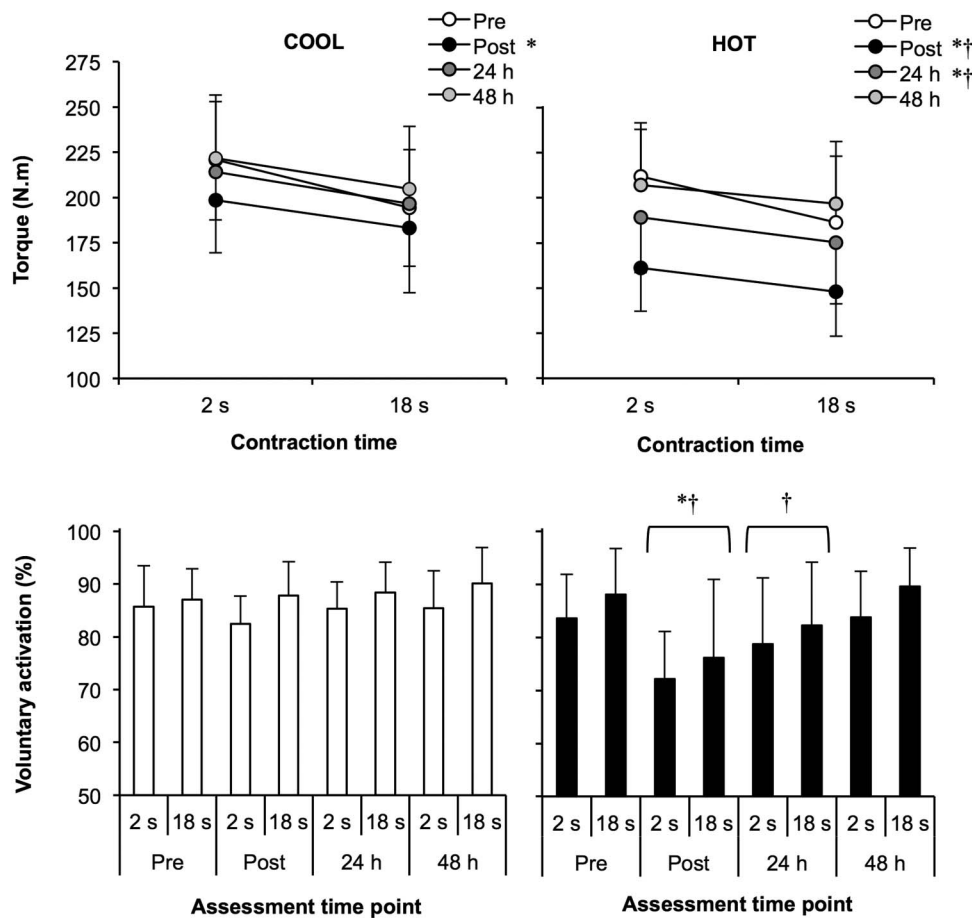
Figure 3 Knee extensor torque production and voluntary activation during a sustained (20 s) maximal voluntary isometric contraction (MVC) in cool (left panels) and hot (right panels) conditions with stimulations superimposed at 2 and 18 s. The MVCs were performed prior to (pre) and following (post, 24 and 48 h) 20 min of effective match-play tennis in each condition *Significantly different from pre, p<0.05. †Significantly different from cool, p<0.05.

the hot condition was lower than in the cool condition at the same time-points (p<0.01). VA during the sustained MVC was lower postmatch in the hot condition compared with prematch (−13.8±9.5%) and cool (−12.9±12.4%) condition values (p<0.05). KE VA was also lower 24 h after play in the hot condition compared with the cool condition (p<0.05). Sustained KE torque and VA were also lower throughout the hot vs cool condition (p<0.05).

Plantar flexion
PF torque production during the brief MVCs decreased (−11.2 ±13.6%) postmatch relative to prematch, with no difference between conditions (figure 2; p<0.05). Brief PF torque production was higher throughout the cool vs cool condition (p<0.05). On the other hand, VA was maintained at a similar level throughout the protocol.

Sustained PF torque production decreased by −9% to 15% throughout all MVCs (ie, from 2 to 18 s) (figure 4; p<0.01). An overall reduction in mean torque was observed postmatch relative to prematch in the hot (−14.4±19.2%) and cool (−7.0 ±11.4%) conditions (p<0.05). Mean PF torque production was higher throughout the cool vs hot condition (p<0.05). Although VA was slightly lower postmatch compared with prematch (p=0.07) and throughout the hot condition relative to the cool condition (p=0.06), statistical significance was not attained. A significant difference in VA was observed between

the 2 and 18 s stimulation intervals, whereby the latter was lower (p<0.01).

Contractile properties
Peak twitch torque of the KE decreased significantly postmatch in both conditions compared with prematch values (table 1; p<0.05). Peak twitch torque and contraction time were higher throughout the hot vs cool condition (p<0.05). The maximum rates of torque development and relaxation were similarly reduced postmatch in the hot and cool conditions, relative to prematch (p<0.05).

Mean PF peak twitch torque decreased significantly from prematch to postmatch (table 2; p<0.05). Contraction time was lower throughout the cool vs hot condition (p<0.05). Mean PF maximum rates of torque development and relaxation were significantly reduced postmatch relative to prematch (p<0.05).

DISCUSSION
In agreement with our first hypothesis, impairments in lower limb strength and VA were exacerbated following match-play tennis in the hot condition compared with cool condition. More specifically, our results indicate that brief and sustained maximal torque production of the KE and PF was reduced immediately after play in hot and cool conditions. However, the reduction in torque was exacerbated under heat stress in the KE only, remaining depressed 24 h after match completion. The
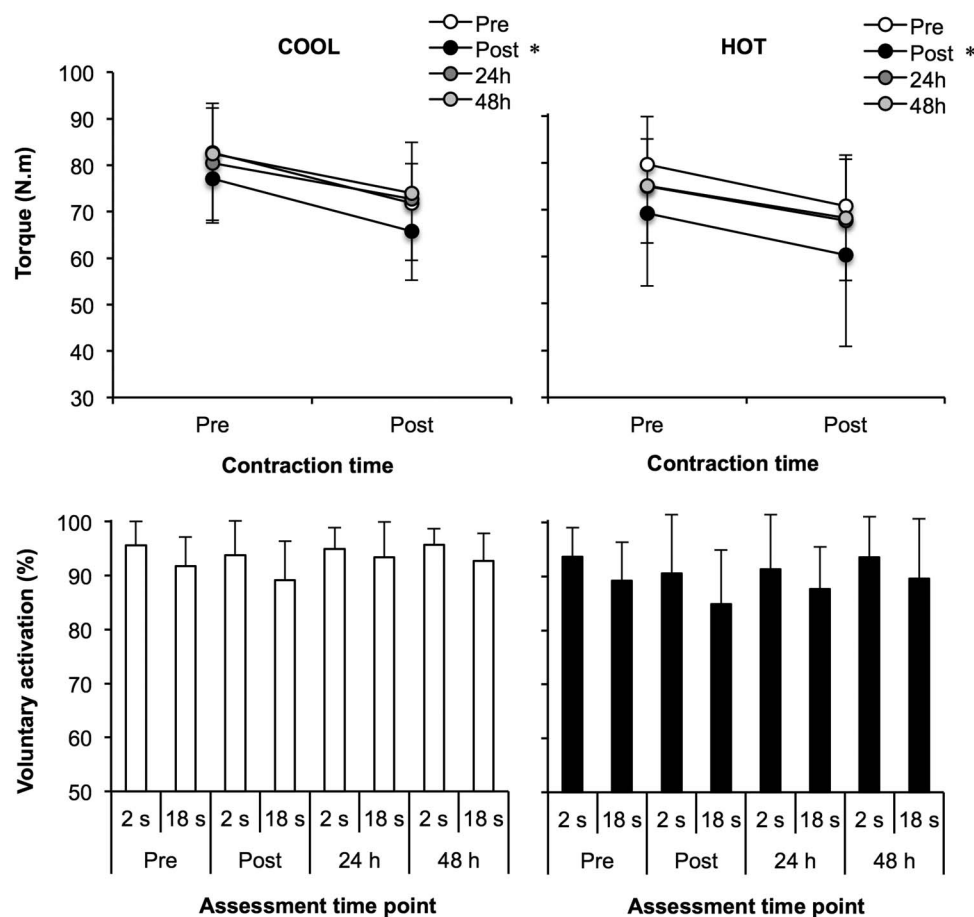
Figure 4 Plantar flexor torque production and voluntary activation during a sustained (20 s) maximal voluntary isometric contraction (MVC) in cool (left panels) and hot (right panels) conditions with stimulations superimposed at 2 and 18 s. The MVCs were performed prior to (pre) and following (post, 24 and 48 h) 20 min of effective match-play tennis in each condition. *Significantly different from pre, $p<0.05$.

loss of strength appears to have originated from a combination of central and peripheral fatigue factors in the KE and mainly from peripheral adjustments in the PF.

Acute postmatch responses
The KE strength losses noted in the brief (∼10%) and sustained (∼8%) MVCs performed after play in the cool condition are similar (∼9%) to those reported during brief contractions performed after 3 h of match-play in similar environmental conditions.[9 10] They are also similar to isometric bilateral leg press force losses (∼8%) shown immediately following the first 2 h match of a 3-day tournament in temperate conditions.[12] In the current study, the loss of KE strength postmatch was associated with a reduction in VA (figures 2 and 3) and adjustments in contractile function, as manifested by a reduction in peak twitch torque and slower rates of twitch torque development/relaxation (table 1). Hence, our data indicate that the adjustments in muscle contractility associated with peripheral fatigue are characterised

Table 1 Knee extensor peak twitch torque, contraction time, half-relaxation time, maximum rate of torque development and maximum rate of torque relaxation prior to (pre) and following (post, 24 and 48 h) 20 min of effective match-play tennis in cool and hot conditions

Contractile characteristic	Condition	Assessment time point			
		Pre	Post	24 h	48 h
Peak twitch torque (N m)	Cool	58.6±7.9	50.2±10.3*	58.2±6.3	61.5±9.9
	Hot	62.4±7.3	56.9±8.5 *	60.1±10.3	62.2±11.6
Contraction time (ms)	Cool	74.1±8.7	69.3±9.6	75.2±7.2	75.2±6.9
	Hot	75.6±8.0	74.3±5.9	77.5±7.3	82.8±9.1
Half-relaxation time (ms)	Cool	67.8±9.8	69.3±10.0	73.9±8.5	74.3±8.1
	Hot	70.4±10.0	73.0±9.8	77.1±14.8	76.4±12.9
Maximum rate of torque development (N m/s¹)	Cool	1634±363	1358±318*	1582±204	1663±310
	Hot	1658±271	1438±210*	1599±223	1551±302
Maximum rate of torque relaxation (N m/s)	Cool	−719±161	−567±148*	−671±120	−685±149
	Hot	−747±151	−607±132*	−704±143	−692±191
*Significantly different from pre, $p<0.05$.					

Table 2 Plantar flexor peak twitch torque, contraction time, half-relaxation time, maximum rate of torque development and maximum rate of torque relaxation prior to (pre) and following (post, 24 and 48 h) 20 min of effective match-play tennis in cool and hot conditions

Contractile Characteristic	Condition	Assessment time point			
		Pre	Post	24 h	48 h
Peak twitch torque (N m)	Cool	16.0±3.2	14.1±3.1*	15.8±3.8	15.5±3.6
	Hot	15.3±4.3	13.8±4.2*	14.7±3.0	15.6±2.9
Contraction time (ms)	Cool	80.7±6.7	77.9±9.8	80.8±6.2	77.0±4.3
	Hot	80.3±9.3	83.3±8.0	83.3±8.8	80.5±8.1
Half-relaxation time (ms)	Cool	65.8±5.9	67.4±7.8	67.9±9.7	64.1±13.1
	Hot	66.3±7.5	63.8±9.0	68.9±11.8	65.7±8.4
Maximum rate of torque development (N m/s)	Cool	540±158	471±107*	530±141	538±143
	Hot	536±178	473±172*	495±120	513±107
Maximum rate of torque relaxation (N m/s)	Cool	−207±48	−173±33*	−192±51	−196±51
	Hot	−193±59	−162±51*	−180±48	−189±33

*Significantly different from pre, $p < 0.05$.

not only by decrements in peak twitch torque, but also by slowing of the muscle contractile speed. Interestingly, VA levels and contractile properties (peak twitch torque) in previous tennis studies were shown not to significantly differ in the KE from prematch to postmatch.[9] [10] However, the torque ratio of stimulations evoked at 20 and 80 Hz declined after play, as did the MVC-to-80 Hz stimulation ratio.[10] This led to the suggestion that peripheral and central adjustments occurred during fatigue,[10] the former being associated with low-frequency fatigue, which is a form of fatigue that stems from exercises that cause muscle damage and impairments in excitation–contraction coupling.[11 25 26]

In a previous study, impairments in PF brief torque production (∼15%) following match-play were similarly attributed to a reduction in voluntary drive, while a decrease in peak twitch torque further confirmed the presence of skeletal muscle fatigue.[8] Our results support part of these observations, in that the reduction in brief (∼11%) and sustained (∼7%) MVC torque in the PF was related to decrements in contractile function (table 2); however, central activation failure was not observed. Recently, Fabre et al[7] reported a similar decrease (∼9%) in brief MVC force after indoor match-play tennis, along with a reduction in peak twitch force and twitch contraction time. As with the current study, they observed that VA was unaffected, even after 45 min of effective play, concluding that the loss of strength occurred concomitantly with changes in muscle contractility. Our data thus confirm these tennis-specific observations in that KE and PF torque production is compromised following play in cool conditions, yet extends them to indicate that the decrement occurs to the same extent during brief and sustained MVCs. Furthermore, the reduction in KE torque appears related to central and peripheral alterations, whereas PF strength is primarily influenced by adjustments in contractile function.

Influence of heat stress

In the hot condition, the decrease in brief (∼22%) and sustained (∼22%) KE torque postmatch was greater than in the cool condition (figures 2 and 3). As with the cool condition, the decrement was associated with a reduction in VA and twitch contractility (table 1). However, during both contraction lengths, the level of central activation failure was greater in the hot condition. Interestingly, while several studies have shown that passive hyperthermia is associated with reductions in VA,[13 14 16–18] the role of exercise-induced hyperthermia in exacerbating central fatigue is unclear. Nybo and Nielsen[19] initially showed that force production and VA progressively decrease during a sustained (120 s) MVC of the KE following exhaustive cycling in the heat, whereas

the decrease in force production is significantly less pronounced and VA remains elevated after exercise in cool conditions. More recent studies have reported that VA and strength losses are equivalent during brief (5 s) and sustained (20 s) contractions conducted after intense self-paced and maximal incremental exercise in hot and cool conditions.[20 21] Part of the discrepancy may relate to the length of contraction. Indeed, after 30 s of maximal isometric effort, pain becomes increasingly severe, altering the perception of sensations originating from the active musculature, which leads to uncertainty as to the level of force being exerted.[27] Moreover, a lack of motivation during protracted MVCs might reduce central activation to the appropriate motor neurons, resulting in the loss of tension.[28] In the current study, the greater deficit in KE VA noted in the hot condition may have originated from a similar lack of motivation, decreasing the ability to voluntarily recruit (ie, brief MVC) and maintain (ie, sustained MVC) all available motor neurons, despite the feedback provided and the participants being constantly reminded to produce/sustain maximal torque. It may also have stemmed from a failure in descending drive (ie, insufficient motor unit firing rate) to compensate for a hyperthermia-induced increase in muscle relaxation rate.[15 17]

In contrast, the decrease in brief (∼11%) and sustained (∼14%) PF torque observed postmatch in the hot condition was associated mainly with a decline in twitch mechanical responses (table 2), as the ∼3% decrease in VA did not reach statistical significance (figure 2 and 4). Moreover, the decrement did not differ between hot and cool conditions. This is akin to soccer match data in which PF torque was reduced postmatch in hot and cool conditions, in association with a decrease in peak twitch torque (∼10%) and a modest decrement in VA (∼1.5%).[22] The difference in the extent of VA reductions between the KE and PF has previously been observed following ultra-marathon running in temperate conditions[29 30] and incremental exercise to exhaustion in hot and cool conditions,[21] suggesting that the reduction in central drive is not as severe in the PF as in the KE. This likely relates to muscle fibre composition, as the quadriceps have a higher percentage of type II fibres. The higher recruitment threshold of these fibres is suggested to result in a greater difficulty in fully activating this muscle group.[31 32] Of note, because of their role in producing powerful actions at high contraction velocities,[33] fatigue or damage to type II muscle fibres (ie, fast-twitch fatigue-sensitive fibres) may result in reducing on-court movement efficiency and stroke proficiency. Hence, performance may become compromised during

prolonged match-play tennis, especially under heat stress, owing to the progressive development of peripheral fatigue and a greater level of central fatigue in large muscle groups (eg, quadriceps).

Recovery kinetics

In temperate conditions, it has previously been shown that KE strength remains depressed 30 min in the recovery period following 3 h of match-play tennis.[9 10] It has also been shown that isometric bilateral leg-press force is reduced prior to the start of play on days two and three of a 3-day tournament, in parallel with increases in markers of muscle damage (ie, creatine kinase and muscle soreness).[12] Data from soccer indicate that quadriceps muscle strength, along with muscle soreness, creatine kinase and muscle glycogen levels, also remain compromised 24 h after match completion.[34 35] In contrast, PF torque production has been demonstrated to return to prematch level within 24 h of soccer play.[22] In the current study, voluntary torque production capacity, along with any postmatch adjustments in contractile function and central neural drive, was restored to prematch level within 24 h of playing in cool conditions for the KE and PF. Correspondingly, Kraemer *et al*[36] postulated that a 24 h recovery period allowed for neuromuscular performance characteristics to fully recover following successive days of indoor match-play tennis competition. Interestingly, however, the authors commented that mental and physical perceptions of fatigue could still persist.

In the hot condition, KE torque during the brief and sustained MVCs remained depressed 24 h into recovery (figures 2 and 3). The decrement was associated with a significant reduction in VA during the sustained, but not the brief, contraction. This decrease in VA and the accompanied reduction in torque may relate to the persistent perception of psychological and physical fatigue,[36 37] especially considering the return to baseline of contractile properties. Our findings, therefore, suggest that a certain level of mental fatigue may persist in the recovery period after play in the heat, whereby a sustained maximal isometric effort might be more difficult to reproduce with a large muscle mass high in fast-twitch fibres, such as the KE.[37] Alternatively, it appears that 24 h is sufficient for the PF to recover from match-play tennis in hot and cool conditions and return to prematch strength capacity.

CONCLUSIONS

This study has shown that knee-extensor and plantar-flexor muscle strength is compromised following match-play tennis in hot and cool conditions due to significant levels of peripheral fatigue. In the knee-extensors, the loss of strength is exacerbated following play in the heat in association with larger reductions in central neural drive, which persists 24 h into recovery when performing sustained contraction. These alterations in neuromuscular system integrity have the potential to negatively influence the efficiency of on-court movements and stroke proficiency during match-play tennis, as well as in the recovery period. Match-related physical performance may, therefore, be compromised during prolonged matches and tournament play, especially under heat stress, owing to the progressive development of fatigue in large muscle groups, such as the quadriceps.

PERSPECTIVES

Given the specific role of the knee-extensors (ie, rapid on-court movements and explosive stroke production) and plantar-flexors (ie, positioning to the ball) in performing tennis movements and actions, fatigue in these muscle groups could impact play acutely in one-off matches and persistently with potential exacerbation across rounds of play, due to the cumulative effect. Future research should focus on enhancing our understanding of the mechanisms of strength loss, especially those linked to central activation failure, identifying potential training models or countermeasures to minimise neuromuscular perturbations during play, and on strategies to accelerate the recovery process in the lower limbs.

What are the new findings?

► Brief and sustained maximal voluntary strength is reduced in the lower limbs (ie, knee extensors (KE) and plantar flexors) immediately following match-play tennis in hot and cool conditions due to the development of peripheral fatigue.

► The reduction in KE strength is exacerbated after play in the heat due to a greater level of central fatigue.

► The loss of strength in the KE persists in the recovery period (24 h) after play in the heat, owing to a reduction in voluntary activation during sustained contractions.

► In the plantar flexors, the impairment in strength following match-play is similar between the hot and cool conditions, recovering within 24 h, and appears mainly due to alterations in skeletal muscle mechanical responses.

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