REVIEW

Exercise-induced homeostatic perturbations provoked by singles tennis match play with reference to development of fatigue

Alberto Mendez-Villanueva, Jaime Fernandez-Fernandez, David Bishop

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This review addresses metabolic, neural, mechanical and thermal alterations during tennis match play with special focus on associations with fatigue. Several studies have provided a link between fatigue and the impairment of tennis skills proficiency. A tennis player's ability to maintain skilled on-court performance and/or optimal muscle function during a demanding match can be compromised as a result of several homeostatic perturbations, for example hypoglycaemia, muscle damage and hyperthermia. Accordingly, an important physiological requirement to succeed at competitive level might be the player's ability to resist fatigue. However, research evidence on this topic is limited and it is unclear to what extent players experience fatigue during high-level tennis match play and what the physiological mechanisms are that are likely to contribute to the deterioration in performance.

The physical and physiological demands of singles tennis match play have been described in detail, mainly in male participants. The duration of a tennis match is often more than an hour and in some cases can be more than 5 h,[1 2] with an effective playing time of 20–30% on clay courts and 10–15% on fast court surfaces.[2] The type of exercise performed in tennis is intermittent (ie, short bouts of exercise interrupted by several periods of longer duration and lower intensity activity).[2 3] Typically, matches consist of work periods of 5–10 s intercalated with rest intervals of 10–20 s, interrupted by several periods of longer duration (60–90 s). During each point, players hit the ball an average of 2 to 3 times and make four directional changes per rally.[4 5] About 80% of all strokes are played within 2.5 m of the player's ready position. About 10% of strokes are made with 2.5–4.5 m of movement with primarily a sliding type movement pattern, and fewer than 5% of strokes are made with more than 4.5 m of movement and a running type movement pattern.[5] Overall, tennis players run an average of 3 m per shot and a total of 8–12 m in the course of a point,[6] completing 300–500 high-intensity efforts during a best of three sets match.[4] On average, the exercise intensity during the game is about 50–60% of maximal oxygen uptake or 60–80% of maximum heart rate.[1]

Owing to the intermittent nature of the game and the relatively long periods of rest between rallies, mean values are unlikely to represent the full demands of tennis. In this regard, the demands imposed during periods of high-intensity, intermittent exercise are probably more relevant to understand the specific requirements of tennis.[7] It is likely that during these high-intensity periods matches will be won or lost, and hence a player's physical condition could be an important influencing factor.[1 2]

This review discusses fatigue during the game and the mechanisms that might impair on-court tennis performance. Fatigue, for the purpose of this review, is defined as the sensations of tiredness and associated decrements in muscular performance and function.[8] Understanding the mechanisms underlying fatigue in tennis match play are important as they might help in devising training strategies to avoid/delay the onset of fatigue during competition and training. These interventions will eventually provide tennis players with a greater ability to maintain the desired performance level over time.

EXERCISE-INDUCED IMPAIRMENTS IN TENNIS-SPECIFIC SKILLS

Performance in tennis is complex, but coaches, athletic trainers and players themselves often attribute the final outcome of a match to decreases in hitting accuracy subsequent to mental mistakes and/or to decreases in physical performance as players tire. Although it is unclear to what extent players experience fatigue during a competitive game, several investigations have observed impaired performance through effects on stroke production and court movement during a match. Fatigue could lead to a decrease in stroke quality and efficiency, a reduced serve velocity, an increase in the percentage of errors on first serves and defensive groundstrokes, and an increase in errors in successful strokes (''winners'').[9–12] Court movement is also affected by fatigue, as the number of balls that could not be reached and the time to complete pattern shuttle-runs increases throughout a match.[10 11] These results support the notion that success at the competitive level might be in part determined by a player's ability to resist fatigue.[13]

See end of article for authors' affiliations

Correspondence to: Alberto Mendez-Villanueva, Facultad de Ciencias de la Actividad Física y el Deporte, Universidad de Alcalá, Campus Universitário, Ctra. Madrid-Barcelona Km. 3,600, 28871 Alcalá de Henares (Madrid), Spain; amendezvillanueva@yahoo.com

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Abbreviations: ATP, adenosine triphosphate; CHO, carbohydrate; PCr, phosphocreatine; RFD, rate of force development; SSC, stretch–shortening cycle

POTENTIAL CAUSES OF FATIGUE DURING SINGLES TENNIS MATCH PLAY

Although the exact causes of muscular fatigue development during high-intensity intermittent exercise, such as tennis, have yet to be determined, in all likelihood the aetiology of these fatigue-induced decrements in muscle performance is multifactorial and diverse.[14] Power output loss with fatigue can originate from several sites from the motor cortex through to contractile elements. The collective results of high-intensity intermittent exercise protocols suggest that the performance impairment and the associated mechanisms are largely influenced by the task performed.[15 16] In particular, the number of repetitions (eg, sprints), contraction duration as well as the rest periods provided between the consecutive efforts (ie, work to rest ratio) appear to be key factors in the task-dependent nature of muscle fatigue during intermittent exercise. According to the available literature it seems clear that the loss of muscular power output is exacerbated by increasing the duration of each exercise period, by reducing the available rest time between efforts, or by increasing the number of efforts.[15–19] A large majority of the potential factors suggested as causes of intermittent exercise fatigue could be placed into three general categories: metabolic, neuromechanical and thermal.

Metabolic factors

Limitations in energy supply (eg, phosphocreatine) and intramuscular accumulation of metabolic by-products (eg, lactate, H^+, inorganic phosphate) have often been implicated as the main cause of muscular fatigue during intense, intermittent exercise.[20] While the exact mechanisms are unclear, the available evidence suggests that a dependence on non-oxidative pathways of adenosine triphosphate (ATP) resynthesis impairs muscle force production after relatively few contractions.[21–23]

Brief sprints and explosive movements, as required in tennis, demand high rates of ATP turnover to the contracting muscles. It is now generally accepted that the ATP turnover to fuel sprint exercise lasting 6 s or less is almost exclusively provided by anaerobic metabolism, namely phosphocreatine (PCr) degradation and anaerobic glycolysis.[24–27] Tennis points last on average between 4 and 10 s.[1 2] Thus, to fuel on-court movements tennis players are likely to rely on anaerobic pathways of ATP resynthesis. PCr is a powerful energy buffer used to replenish ATP during intensive, anaerobic exercise.[24] During a single short (5–6 s) maximal sprint, PCr degradation is reported to account for approximately 50% of the total anaerobic ATP provision.[24 27] Accordingly, it has been suggested that PCr resynthesis, and thus PCr availability, is critical for maintenance of muscle performance during repeated, short duration all-out efforts.[24] The importance of PCr availability for intense muscle contractions is supported by the observation that restoration of peak power output after high-intensity cycling and the isometric force after a static contraction follows a similar time course to that of PCr resynthesis in human muscle.[28 29] Furthermore, a strong correlation has been reported between power recovery in the first 10 s of a second sprint of 30 s and the resynthesis of PCr.[30]

Following such intense exercise, complete PCr recovery might require 3–5 min.[31] However in tennis, breaks between two consecutive efforts are normally shorter.[1–3] In repeated exercise bouts, if the subsequent recovery interval is less than a few minutes long, as in tennis, the PCr stores might be only partially restored before the onset of subsequent exercise, resulting in compromised performance in successive bouts. As PCr stores are progressively depleted with subsequent high-intensity work bouts separated by incomplete recovery periods, there will be increased reliance on aerobic metabolism but at the expense of a truly maximal performance.[20 24 26] In this regard, Ferrauti *et al*[18] showed that running speed and stroke quality during intermittent tennis drills are highly dependent on recovery time; when recovery was too short, running speed for stroke preparation and stroke speed decreased. Albeit speculative, these reported impairments in on-court movements (ie, running velocity) and sport-specific skills (ie, stroke speed) might have been due to the incomplete restoration of anaerobic muscle reserves (eg, PCr). Taken together, these findings suggest that repeated tennis movements might reduce muscle PCr content enough to impair power output during on-court play. Moreover, increased levels of the products of PCr hydrolysis (ie, inorganic phosphate in particular) have also been correlated with muscle fatigue,[23 29] although its role during in vivo muscle function remains unclear.[32]

Muscle glycogen depletion has repeatedly been shown to be associated with fatigue during prolonged submaximal exercise.[14] However, the potential role of carbohydrate (CHO) metabolism on performance during prolonged, intermittent exercise, such as tennis, has received limited attention. Recently, Krustrup *et al*[33] suggested an association between fatigue (ie, impaired repeated sprint ability) towards the end of a soccer match and reduced glycogen levels in individual muscle fibres. In this regard, it has been demonstrated that elevating muscle glycogen prior to exercise through a carbohydrate diet elevates performance during repeated sprint exercise.[34] Moreover, several studies have positively correlated carbohydrate electrolyte drink ingestion with prolonged intermittent endurance performance.[35–37] The mechanisms responsible for any impairment in high-intensity intermittent exercise due to any disruption in CHO utilisation are unclear, but include reducing muscle glycogen stores, decreasing circulating blood glucose, and altering neurotransmitter activity that could influence cognition, mood, motivation, and motor skill performance.[37 38] However, research on CHO supplementation and performance in tennis is conflicting.[39] To date, only one tennis-specific study has shown that CHO supplementation can benefit hitting accuracy and stroke quality in the latter stages of a 2 h tennis training session.[40] In addition, Ferrauti *et al*[41] found improvements in on-court running speed when players ingested a CHO drink at court changeovers during a tennis match of 4 h duration. Taken together with the results obtained in studies showing that CHO feedings during intermittent protocols similar to tennis match play can preserve physical and mental function,[36 37] maintaining appropriate muscle glycogen stores throughout practice or match conditions has been suggested to be important for maintaining tennis performance.[39]

Dynamic exercise is associated with an increase in glucose uptake by the active skeletal muscles.[42] When blood glucose levels drop, hepatic glucose production is stimulated to keep pace with the rate of glucose utilisation by skeletal muscles.[43] In the case of a constant high glucose uptake by the contracting muscles, episodes of hypoglycaemia can occur that result in compromised exercise performance.[44] Blood glucose concentrations in tennis players have been shown to remain stable during play and competition.[9 39 45] However, under tournament conditions, Ferrauti *et al*[46] showed frequent negative disturbance of glucose levels after the rest period between a first and second match. Thus, low blood glucose levels can be a factor underlying tennis-related fatigue when players have to compete twice in the same day. While this is unlikely to occur in Grand Slam Championships and Association of Tennis Professionals (ATP) tournaments, this can be of relevance to players contesting in lower entry-level tournaments, such as the Satellites and Futures circuits that represent the first level of opportunity for professional players to acquire computer ranking points[47] or when playing doubles and singles on the same day. Moreover,

caution should be taken as it has been reported that blood glucose levels remain elevated during a soccer game despite a significant decrease in muscle glycogen, with almost 50% of the individual muscle fibres being completely or almost empty of glycogen after the game.[33]

The decline in muscle force during the development of muscle fatigue during high-intensity exercise has been classically associated with accumulation of lactate and a concomitant lowering of pH in the muscle.[48] However, the direct effect of muscle acidosis on muscle contractile capabilities is now being challenged,[21 23 32 49] with recent reports in isolated muscle fibres showing that intracellular acidosis might be important for preserving muscle excitability.[49 50] Moreover, several studies in exercising humans have disassociated muscular fatigue and intracellular lactate accumulation/acidosis during intense exercise.[32 51] In addition, the reported low to moderate levels of blood lactate, indirect markers of glycolytic metabolism participation, found in various studies with tennis players suggest a modest degree of muscle acidosis during tennis match play.[1 3] While the role of muscle acidosis as a direct contributor to muscle fatigue during voluntary contractions can not be fully disregarded[52] the data obtained in tennis research,[1–3] together with results from several in vitro and in vivo studies,[21 23 32 33 49 51] does not support a relationship between impaired tennis performance and lactate and H^+ accumulation.

Neuromechanical factors

Human locomotor performance depends directly upon the forces that the musculoskeletal system can generate and transmit to the environment.[22] For example, runners modulate their speed primarily by altering the forces that they apply to the ground.[53] In order to maintain a maximal external mechanical output (ie, force or power), the nervous system would need to recruit all motor unit pools at their highest firing rate.[54 55] Accordingly, it has previously been demonstrated that neuromuscular activation and muscle mechanical properties are associated with performance in human movements that involve maximal or near maximal muscle force and power output.[56] Because on-court tennis movements (eg, rapid changes in direction) and stroke technique (eg, >210 km/h serves) are very high power output activities, any neural and/or mechanical factor that reduces the ability of the musculoskeletal system to generate explosive strength would likely impair tennis performance.

In tennis, sprints occur over short distances where athletes are, in many cases, unable to reach their maximum speed.[5 6 18] Therefore, the initial acceleration phase (~0–5 m) is likely to be of major importance to tennis players. Research on track sprinters starting in blocks and team sport players sprinting from a standing two-point start (straddle position) has identified that the first few ground-contact phases of a short sprint are dominated by propulsive forces when compared to braking forces,[57 58] and by concentric muscle actions.[58] The average vertical impulse of sprinters during the propulsive phase of the first ground contact has also shown significant correlation with initial running velocity.[57 58] It is therefore likely that tennis players with explosive first steps get into position quickly, set up well, and hit effective shots. In this regard, proper positioning on the court is essential as it has been reported that the more steps a player has to take to play a shot, the less likely he or she is to win the rally.[5 59]

Most performance-related tennis movements (ie, sprints and strokes) involve a combination of eccentric and concentric muscle actions, the so-called stretch–shortening cycle (SSC).[60] Research has examined the effects of repeated SSC exercise on the mechanical behaviour (ie, foot contact time and force) of the contracting muscles. For example, Nummela *et al*[61] found that the resultant ground reaction forces decreased by 13% in the braking phase and by 10% in the propulsion phase during maximal and submaximal 20 m runs performed at the end of 400 m runs, leading to a 19% decrement in running speed. Similar changes in the ground reaction forces have been observed after a marathon run.[62] These results support the link between fatigue-induced repeated stretch–shortening loads and alterations in the mechanical response of the active muscles. However, most of this research has utilised single maximal efforts to induce fatigue. Little is known about alterations in sprinting mechanics caused by fatigue induced by repeated dynamic sprint efforts, as occurs in tennis. Pinniger *et al*[63] found significant changes in the kinematics and EMG parameters of sprint running after fatigue induced by repeated maximal efforts, which were accompanied by a decrease in repeated sprint ability. However, the absence of ground reaction forces limited the ability to explain such performance decrements.

Leg stiffness regulation is believed to be another important determinant in the optimisation of human locomotion performance.[64–67] For instance, it has been reported that the ability to maintain high-level stiffness is related to maximal performance in explosive exercises[68–70] and more specifically during sprint running.[71–73] Moreover, Arampatzis *et al*[65] emphasised that the maximisation of mechanical power is attained through optimal leg stiffness. In addition, it has been suggested that increased muscular stiffness during landing would provide more joint stability and protection against joint injury.[74] In contrast, impairments in SSC performance are associated with a lowered tolerance to ground impact forces as a consequence of reduced joint stiffness.[75] Girard *et al*[60] showed a progressive reduction in leg stiffness throughout 3 h of tennis match play, which was accompanied by a parallel decline in the ability to generate maximum force. This is in line with previous research showing that repeated SSC contractions induced a reduced leg muscle stiffness.[76 77] Thus, it is possible that some of the observed fatigue-induced decrements in ''on-court'' tennis movements might be partially explained by alterations in the mechanocharacteristics of the muscle–tendon complex.

In tennis, the ability to react to an opponent's action is vital for successful performance. For example, on the return of serve when the serve is hit at 200 km/h, the time available for action is approximately 500 ms.[78] Thus, the ability to generate very fast movements is crucial for tennis players. Explosive power depends, among other factors as mentioned above, on neural aspects such as high-frequency recruitment of the available motor unit pool and muscle fibres within the synergistic muscles.[14] The rate of force development (RFD), for instance, provides important information in relation to a muscle's ability to generate fast and forceful contractions–eg, within the initial 100–200 ms of contraction.[79] Consequently, any factor that reduces the RFD would contribute to fatigue during fast movements, such as a quick step to reach a ball, by decreasing the percent of force that can be achieved in the early phase of muscle contraction.[79]

RFD is influenced by neural and peripheral factors.[79] However, neural output to the motoneuron is likely to be a major contributor to the RFD magnitude.[80 81] During high-intensity intermittent exercise, as occurs in tennis, it has been reported that there is a decrease in neural drive to the motor unit.[82–85] This suboptimal efferent neural drive to muscle might reduce RFD. Training studies that have demonstrated concurrent increases in RFD and the efferent neuromuscular drive of skeletal muscle support this hypothesis.[79 86] Although the critical phases of play during tennis matches are likely to be dependent on repeated anaerobic efforts, these are superimposed on a background of largely aerobic submaximal activities.[7] As a result of high intensity intermittent exercise, punctual and transient episodes of muscular fatigue could

What is already known on this topic

Tennis players can experience fatigue during a competitive game.

What this study adds

- Specific homeostatic perturbations are identified and linked to deterioration of skilled on-court performance.
- More studies should be conducted to better determine the influence of fatigue on tennis performance.

occur after an intense exercise bout or after a set of repeated efforts.[87] Due to the prolonged pattern of exercise (tennis matches can last up to 5 h),[88] deterioration in performance similar to that occurring during long-term exercise might also be observed. Empirical observations of decreases in mental, technical skills and physical performance occurring towards the later stages of a match seem to provide some support to this assumption.[60] The occurrence of this type of fatigue late in a match might also be related with a reduction in neural drive to the motoneurons, as previously reported during long duration repetitive dynamic actions.[89–91] As tennis match play requires brief, fast limb movements, any fatigue-induced reduction in the RFD might limit muscular performance as it will be difficult to reach higher levels of muscle force within the initial phase of muscle contraction.

Muscle damage

Exercise-induced muscle damage is a common occurrence following activities with a high eccentric component, such as serves, ground strokes and intense shuttle runs in tennis match play. Many symptoms usually accompany muscle damage, which include muscle soreness, increased plasma levels of muscle proteins, swelling, inflammation, and impairment of muscle function.[92 93] Of pivotal importance for tennis performance is the reported negative effect of exercise-induced muscle damage on the ability to generate rapid force (ie, RFD).[94–96] A progressive reduction in maximal voluntary force and an increase in muscle soreness, indirect indicators of muscle damage, have been previously observed throughout 3 h of tennis match play.[59] Moreover, Hornery *et al*[97] reported a significant increase in circulating creatine kinase, which appears to be indicative of acute muscle damage. Thus, damage invoked by the numerous eccentric contractions associated with on-court tennis movements (ie, acceleration and decelerations, repetitive overhead motions and directional changes) might represent an important factor underlying the fatigue observed during tennis.[14]

Selective damage of type II fibres is a commonly proposed mechanism believed to mediate reduced power output following muscle contractions with an eccentric component.[96 98] Given that type II fibres are the fastest and stronger type of fibres in the human skeletal muscle,[99] their recruitment during explosive and forceful actions, such as accelerations or sprints, is crucial.[55] It seems logical to suggest that this selective fatigue of type II fibres, due to the muscular damage associated with eccentric loads during a tennis match, might reduce some player's abilities, such as acceleration and movement speed, resulting in impaired tennis performance.

Muscle damaging exercise can also impair muscle function subsequent to neural activation deficit.[77] Previous research has suggested that reduced force production during eccentric work is due to reduced neural drive to the muscle in response to peripheral inhibition of spinal motoneurons.[100] This mechanism is believed to reflexively arise from the muscle in response to fatigue, and to be mediated by small-diameter group III and IV muscle afferents.[101 102] As previously mentioned, a suboptimal neural drive to the muscle might impair factors (ie, RFD) believed to be important determinants of fast limb movements that are key aspects for success in competitive tennis.

Thermal stress

Competitive tennis is typically played in hot environments. It is well established that exercise is prematurely terminated in hot conditions.[103] Pivotal for this temperature-mediated performance impairment is the inability of the body to remove the accumulated heat.[104] During tennis competition, the increase in body heat load can be endogenous due to increases in player's metabolic rate[44 97] and/or exogenous (ie, ambient environment). For example, in many cases, the court surface can be warmer than the athlete's skin temperature, which causes heat transfer from the environment to the player and thus increases internal body temperature.[105] Although most research has been conducted using continuous prolonged exercise, some studies have found similar temperature-related impairments during intermittent exercise.[106–110] Thus, when tennis players play in hot conditions for extended periods of time there is an increased possibility they will experience symptoms of fatigue and subsequently also heat injury. While several mechanisms have been proposed to explain the negative impact of heat stress on exercise performance, the underlying causes are not completely understood.[111] It has been suggested that rather than dehydration or altered metabolic rate, a critical internal temperature will ultimately set the limit for voluntary exercise capacity.[104 111] It is likely that the attainment of a high internal temperature can impair central nervous system function, resulting in a reduced level of central cognitive or neural drive to the muscle, which might in turn decrease muscle function as previously discussed.[38] Thus, a hyperthermia-induced impairment of the ability to sustain high neuronal drive could be a factor underlying the observed decrements in tennis skills when tennis is played in the heat.

SPORT AND BIOMEDICAL RELEVANCE

Identification of the mechanisms that contribute to muscle fatigue during tennis match play has a number of practical applications. Clearly, such an understanding has implications in the field of sports science by helping to identify appropriate performance enhancement training, nutritional and other strategies/interventions to enhance performance. There are also implications for rehabilitation medicine through improved structuring of exercise tasks to improve or compensate for impairments of neuromuscular performance and to prevent or minimise injury.

CONCLUSIONS

Fatigue can develop as the duration and intensity of tennis match play increase, and it can be exacerbated by high environmental temperatures. The final outcome of many tennis tournaments is often decided by only few ''winning'' shots. Therefore, even small changes can greatly impact player's ability to succeed or fail. Further research is needed to understand the mechanisms limiting tennis performance. Further research is also needed to gain a greater understanding of the fatigue-induced mechanisms limiting tennis performance, as this will likely benefit players with better and more consistent performance, fewer injuries and longer careers.

Authors' affiliations

Alberto Mendez-Villanueva, Faculty of Physical Activity and Sport Sciences, University of Alcala, Madrid, Spain
Jaime Fernandez-Fernandez, Faculty of Physical Activity and Sport Sciences, University of Leon, Spain
David Bishop, Team Sport Research Group, Facoltà di Scienze Motorie, Università di Verona, Italy

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COMMENTARY

This paper neatly summarises some of the factors which may be critical in the development of fatigue during tennis. In longer matches, fatigue may play an important role in the decrease in performance that ultimately influences the final outcome. Coaches and trainers could assist their players by addressing some of the factors discussed.

Tim Wood
Glenferrie Private Hospital, Hawthorn, Victoria, Australia;
georgewood@bigpond.com