

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/23674684>

Dehydration Cause of Fatigue or Sign of Pacing in Elite Soccer?

Article in *Sports Medicine* · February 2009

Source: PubMed

CITATIONS

133

READS

2,438

2 authors, including:



[Andrew M Edwards](#)

Canterbury Christ Church University

72 PUBLICATIONS 2,047 CITATIONS

[SEE PROFILE](#)

Some of the authors of this publication are also working on these related projects:



Stress [View project](#)

Dehydration

Cause of Fatigue or Sign of Pacing in Elite Soccer?

Andrew M. Edwards^{1,2} and Timothy D. Noakes³

1 UCOL Institute of Technology, Faculty of Health Sciences, Palmerston North, New Zealand

2 Leeds Metropolitan University, Carnegie Research Institute, Leeds, UK

3 Department of Human Biology, University of Cape Town, Cape Town, South Africa

Abstract

Numerous studies have suggested that dehydration is a causal factor to fatigue across a range of sports such as soccer; however, empirical evidence is equivocal on this point. It is also possible that exercise-induced moderate dehydration is purely an outcome of significant metabolic activity during a game. The diverse yet sustained physical activities in soccer undoubtedly threaten homeostasis, but research suggests that under most environmental conditions, match-play fluid loss is minimal (~1–2% loss of body mass), metabolite accumulation remains fairly constant, and core temperatures do not reach levels considered sufficiently critical to require the immediate cessation of exercise. A complex (central) metabolic control system which ensures that no one (peripheral) physiological system is maximally utilized may explain the diversity of research findings concerning the impact of individual factors such as dehydration on elite soccer performance. In consideration of the existing literature, we propose a new interpretative pacing model to explain the self-regulation of elite soccer performance and, in which, players behaviourally modulate efforts according to a subconscious strategy. This strategy is based on both pre-match (intrinsic and extrinsic factors) and dynamic considerations during the game (such as skin temperature, thirst, accumulation of metabolites in the muscles, plasma osmolality and substrate availability), which enables players to avoid total failure of any single peripheral physiological system either prematurely or at the conclusion of a match. In summary, we suggest that dehydration is only an outcome of complex physiological control (operating a pacing plan) and no single metabolic factor is causal of fatigue in elite soccer.

Dehydration has often been suggested as a factor responsible for the development of fatigue in the later stages of a soccer game.^[1,2] This is a credible observation as even moderate dehydration is known to be associated with negative endurance-based exercise responses both in a controlled laboratory environment^[3,4] and also in more soccer-specific field conditions.^[5] It re-

mains, however, unclear whether game-induced dehydration is a cause of fatigue, or merely a single characteristic outcome of exercise controlled by a complex metabolic system.^[6-9]

Previous laboratory studies have demonstrated that failure to ingest fluid during prolonged exercise causes plasma sodium (Na⁺) concentration, osmolality and anti-diuretic hormone activity to

increase.^[3] Consequently, there is little doubt that game-induced dehydration contributes to the challenge of maintaining homeostasis during a game. For example, it has been proposed that these factors may be a mechanism for reductions in skin blood flow at advanced stages of dehydration.^[10] However, this has rarely been observed in outdoor conditions where opportunities for convective and evaporative cooling are enhanced when compared with a laboratory.^[4] Nevertheless, several studies have been conducted in realistic field conditions that appear to confirm that physiological performance is impaired when exercising even in a moderately dehydrated (~2% loss of body mass) condition.^[5,11] However, it is less clear whether it is water loss *per se* that leads to performance decrements, or whether the concurrent negative psychological associations attributable to thirst act as a signalling mechanism to promote a greater conscious perception of effort and so invokes a behavioural change to reduce physical effort.^[7,8,12,13]

The overall intensity of exercise performed in elite (professional) soccer matches broadly corresponds to a sustained oxygen consumption of ~75%^[14] of maximum interspersed by varied physical challenges that change every 4–6 seconds during a 90-minute match.^[15–17] These diverse but sustained physical activities undoubtedly threaten homeostasis. However, match-play core temperatures in thermoneutral environments have rarely been reported to reach levels considered sufficiently critical to require the immediate cessation of exercise. In addition, fluid loss is usually minimal (~1–2% of body mass),^[18–20] which suggests that in normal circumstances players are able to pace themselves in order to complete a match in a reasonable physical condition.

Numerous studies have demonstrated that the amount of sprinting, high-intensity running and distances covered drop from the first to the second half of a match.^[15–17,21] Some of this inter-half disparity is probably due to tactical changes towards the end of a game, but there is currently no evidence to support that conclusion, nor the extent to which the development of fatigue influences tactics. Limited data suggest^[16,17] that the majority of the downregulation of physical

activity in the second half of a match is attributable to the effects of fatigue. This suggests that either players are subject to the effects of accumulative fatigue (towards total physiological system failure, requiring the immediate cessation of exercise) or that each player employs a subconscious pacing strategy to ensure they reach the end of the game. Examination of time-motion video analysis from international match-play supports the presence of non-random physical activity patterns during a game, whereby the more physically demanding phases are followed by prolonged periods of minimal activity.^[22] As such, players appear to temporarily experience symptoms of fatigue during a game,^[23] probably to subconsciously defend homeostasis and as a consequence of a personalized pacing plan. This explains the ability of all players to complete the full duration of a match, regardless of differences in fitness levels, and remain in a reasonable physical condition.

This article critically evaluates the existing research evidence surrounding the influence of dehydration on elite soccer performance. In addition, a complex system control mechanism^[6,7,9] and a new multi-level pacing strategy are proposed as contemporary interpretations of a long-standing issue. These factors may explain how players regulate effort during match-play, thus avoiding debilitating fatigue and the requirement for a premature cessation of exercise prior to the conclusion of a game.^[7,24]

1. Thermoregulatory Responses to Soccer: Laboratory and Field Applications

Due to practical difficulties in obtaining reproducible and sufficiently frequent data during match-play,^[25] the thermoregulatory stresses associated with soccer have generally been estimated by focusing on the responses to match simulations in either laboratories or controlled indoor environments.^[26,27] For example, Drust et al.^[26] devised an intermittent laboratory-based protocol for a motorized treadmill based on observed movement patterns during soccer.^[17] Nicholas et al.^[27,28] developed the Loughborough

Intermittent Shuttle Test (LIST) to mimic the demands of soccer match play using a 20-m shuttle-running model, of similar construction to the original progressive shuttle-running test devised by Leger and Lambert.^[29] In the case of the LIST, the intensities of repeated efforts are manipulated to reflect the different types of activities experienced during a game (i.e. high- and low-intensity running, sprinting and walking). A well controlled test-retest reliability study^[28] confirmed that variables such as heart rate are highly reproducible in the LIST protocol. Consequently, this protocol is now commonly used by investigators seeking to indirectly examine the effects of different nutritional intervention strategies on soccer performance and, to some extent, as a sports-specific fitness test to identify physiological characteristic differences between groups of players.^[30]

Although several soccer-simulation protocols such as those briefly discussed here are sensibly constructed, their direct validity to match play is obviously limited by factors such as the absence of a ball, direct competition, straight-line running, and minimal opportunities to self-regulate physical effort.^[25] It must also be considered that most studies utilizing these procedures are invariably conducted in indoor, windstill conditions^[26–28,30,31] imposing a different physical stress to outdoor match-play. For example, during outdoor match-play, soccer players are exposed to environmental conditions that increase the efficiency of evaporative heat loss and consequently minimize elevations in thermal stress when compared with indoor situations.

1.1 Sweat Rates and Fluid Intake

Mustafa and Mahmoud^[32] reported mean sweat rates ranging from 0.6 L/h in an outdoor match in cool conditions to 2.9 L/h in hot, humid weather. More recently, similar mean sweat rates have been reported during outdoor soccer training in cool conditions (1.1 L/h; 5°C; 81% relative humidity [RH])^[33] and also in the heat (1.5 L/h; 32°C; 20% RH).^[34] These results are slightly higher than those we recently reported^[5] in response to a protocol combining 45 minutes of

indoor cycling exercise (90% at individual ventilatory threshold) followed by 45 minutes of an outdoor soccer match (0.8–0.9 L/h). However, fluid deficits between 1–2% losses of body mass seem typical in competitive soccer matches across the majority of environmental conditions. Such fluid losses are also largely inevitable due to the restricted opportunities to drink during a game.^[5,18,35,36] Assuming typical sweat rates during match-play range between 0.8–1.5 L/h across most environmental conditions, this would correspond with a maximal sweat loss of 2.3 L/h and a mean of 1.7 L/h over the course of a full game.^[35,36] The similarity of sweat rates across non-extreme environmental conditions indicates that sweat production is unlikely to be crucial to soccer performance, as these remain well below levels reported in endurance events such as the marathon (~3 L/h).^[20,37]

Adequate fluid intake before a match is important for all players and well considered guidelines for match-play fluid intake have been previously reported on this issue.^[38] However, it remains unclear whether relatively modest game-induced dehydration impairs soccer performance in thermoneutral conditions. It is plausible that the moderate water loss liberated by carbohydrate utilization is simply a feature of diminishing glycogen stores and not the debilitating effects of water loss *per se*.^[39,40] However, diminishing stores of muscle and liver glycogen probably act as a signalling mechanism^[41] to identify a diminishing supply of high-energy adenosine triphosphate (ATP) and so may cause a behavioural change to reduce work intensity, thus saving stored glycogen to ensure that exercise terminates without total muscle glycogen depletion.^[42]

With an adequate supply of sweat and in the absence of any impairment to convective cooling, it seems unlikely that core temperatures in an outdoor game reach critical levels in well conditioned players. One study^[43] reported individual values above 40°C; however, more recently, mean core temperatures reported at the end of professional (38.8°C) and recreational (39.1°C) matches^[18] have been shown to be beneath those typical of fatigue (~41.5°C).^[20,37] As no differences have been found in core temperatures

between the end of the first and the second halves of elite match-play,^[18,19] it is probable that a measure of homeostatic control is maintained over the game among experienced and well trained players.^[5] This seems in contrast to sub-elite and less well conditioned players who have been observed to increase core temperature progressively over the course of a match,^[18] presumably as a consequence of less match experience, under-developed heat dissipation mechanisms, and poorly developed pacing strategies.

1.2 The Impact of Dehydration on Soccer Performance

The random nature of game play inevitably means it is difficult to measure the impact of intervention strategies in competitive soccer match-play. Consequently, direct performance implications are usually inferred from either soccer simulation models such as those discussed in section 1,^[26,27] or from other exercise modalities.^[44,45] This indirect evidence has often been used to suggest that relatively modest fluid losses incurred during a soccer game cause decrements in both psychological and physiological factors of importance to match performance.^[1,2,23,31] However, research evidence does not necessarily support this observation, or at the very least, definitively identify water loss as *the* factor determining impaired performance.

One research group found that a diuretically induced 2% loss of body mass significantly impaired physiological endurance running performance.^[44] That study is commonly cited to support the view that even moderate water loss impairs physiological performance in a range of sports. However, it was poorly controlled because there was no control for the specific effects of the diuretic itself on exercise performance, independent of its effects in inducing dehydration. It is also well known that diuretics induce dehydration through the uncomfortable symptoms of polyuria and also promote excess electrolyte loss. It is unlikely that similar proportions of electrolyte loss would occur in exercise-induced dehydration during a soccer match. Nevertheless, several other studies have also demonstrated

that moderate water loss across a range of exercise modalities is associated with impaired exercise performances.^[5,11,31,45] One such study^[45] tested heat-acclimatized subjects with fluid losses corresponding to 1%, 2%, 3%, and 4% of body mass and reported that deficits of $\geq 2\%$ were associated with significant and progressive reductions in the performance of several visumotor and psychological tests compared with a euhydrated condition. In contrast, Serwah and Marino^[46] examined the effects of hydration and exercise heat stress on choice reaction time following 90 minutes of cycling (~70% of peak power) and found that performance was not compromised in the presence of either dehydration or heat stress.

Research evidence on the effects of dehydration remains equivocal over a range of experimental models. Using the LIST exercise protocol in a wind-still indoor gymnasium, McGregor et al.^[31] reported that the subsequent performance of a repeated sprint-dribbling test was compromised in a moderately dehydrated condition ($p < 0.05$). However, that study also noted that post-protocol performance in a mental concentration test was unaffected by fluid losses of 2.4% of body mass. Additionally, Hoffman et al.^[47] identified that basketball players with fluid losses of 2% of body mass did not experience post-match performance impairments in selected tests for anaerobic power, vertical jumping height or goal shooting ability.

In soccer-specific circumstances, we replicated the mental concentration test used by McGregor et al.^[31] following a soccer match and confirmed that mental concentration was unaffected by moderate water loss.^[5] However, our study also found that the denial of fluid over 90 minutes of exercise (45-minute cycling at 90% of ventilatory threshold and 45 minutes of outdoor soccer) significantly impaired the immediate post-match physiological performance of a sport-specific fitness test (Yo-Yo intermittent recovery test) compared with a fluid-intake condition ($p < 0.05$). Participants also reported significantly greater ratings of perceived exertion and elevated sensations of thirst when access to fluid was denied throughout the 90-minute exercise protocol ($p < 0.05$). This suggests that consciously

perceived (negative) factors such as thirst may be responsible for the observed limitations to performance.

From the limited available evidence, it is reasonable to conclude that performance during a soccer game is probably compromised by even modest losses of body water. This effect has been less evident in indirect and simple psychological tests, although it may occur where task complexity increases, thus requiring greater attention or effort. For example, the soccer skill test utilized by McGregor et al.^[31] required the performance of repeated sprint dribbling and so the performance limitation in that case is likely to have been related to a physical cause. In those circumstances, effort was probably reduced through less muscle recruitment as a precautionary action to defend homeostasis in the presence of intracellular disturbances.^[6,24,48] Therefore, in terms of soccer performance, increased perceptions of effort and greater sensations of thirst in a dehydrated condition may invoke behavioural changes^[48] such as reduction in maximal effort,^[5] or the frequency of high-intensity activities. Others have observed this effect in a dehydrated state^[49] and so it is plausible that conscious feelings such as thirst rather than the water loss *per se* may be causally linked to performance impairment.

1.3 Fluid Intake and the Thirst Mechanism

In relatively short-duration activities such as soccer, the greatest effect of fluid ingestion is probably to reduce the perception of effort during prolonged exercise, especially in the heat.^[49] For example, numerous studies have reported negative associations between moderate dehydration and perceived discomfort during exercise when fluid intake is either denied or restricted.^[5,48] The conscious perception of a developing cellular homeostatic disturbance induces the following two behavioural changes: (i) an increased desire to drink; and (ii) a temporary reduction in the exercise intensity.^[12,50] According to this model, a developing thirst acts as the primary mechanism to trigger the two behavioural changes.^[6,8,12,50] For example, the tongue provides the first internal analysis of potentially

ingestible stimuli and the sensory mechanisms embedded in the lingual epithelium are used to extract information about the fluid for central processing in the brain. The level of an individual's dehydration is also detected as a change in plasma osmolality, which is a key homeostatically defended variable.^[6] Physiological mechanisms are subsequently invoked, which affect water intake (via augmenting the desire to drink) and water reabsorption (via anti-diuretic hormone secretion).

Several studies have demonstrated that exercise performance is maximized by drinking according to the dictates of thirst (*ad libitum*).^[13,49] *Ad libitum* drinking does not produce identical levels of dehydration in all humans during exercise and has previously been shown to result in widely different percentage alterations (+6% to -11%) in body mass.^[49] Therefore, the conclusion that a specific level of dehydration such as the 2% common in soccer matches will always cause an impaired exercise performance cannot be correct. It is more likely that any impaired exercise performance in a dehydrated condition is causally linked to the thirst mechanism^[13] and not the absolute level of dehydration.^[38] Fluid intake by humans is therefore influenced by a complex interaction of physiological and behavioural mechanisms.^[6,8]

2. Match Observations and the Development of Fatigue

The distances covered by players and the intensity of their physical work are well known to decline from the first to the second half of a soccer match across all outfield positions^[16,17,21] (figure 1), which, when considered in isolation, could suggest that either accumulative fatigue (driving towards total physiological system failure) is a feature of elite match-play or that there is a defined pacing strategy in place (to defend a regulated level of exercise homeostasis).

Bangsbo et al.^[21] reported a typical overall second half drop of ~5% in the total distance covered among elite male players, while the inter-half disparity appears to be greater at the

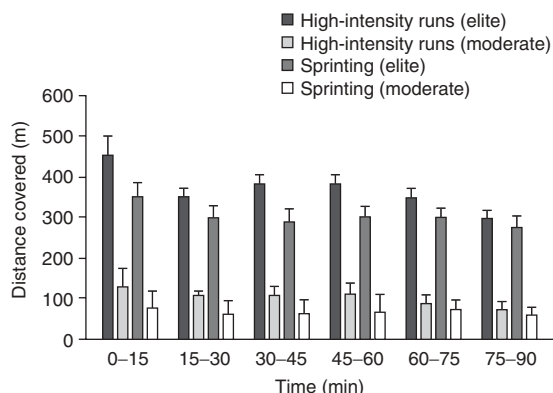


Fig. 1. A comparison of high-intensity running and sprinting during match-play among elite- and moderate-level soccer players. The high-intensity running diminishes to protect the integrity of defended variables (such as core temperature, plasma osmolality, blood pH) from reaching critical levels (data reproduced from Mohr et al.,^[16] with permission from the publisher, Taylor & Francis Ltd, <http://www.tandf.co.uk/journals>).

sub-elite level (~9–10% decrement).^[16,21,51] A recent study^[22] also identified fluctuations in high-intensity exercise, running speeds and recovery time from sprints during several top-class soccer matches, and particularly observed that longer sprints (30 m) demanded markedly longer recovery than the average sprints (10–15 m) during a game. These findings identified that fatigue occurred both acutely during the game (transient fatigue) and also as a developing feature of 90 minutes of prolonged intermittent exercise. Nevertheless, despite numerous attempts, no researchers have identified a precise cause of fatigue in soccer.^[2,23] This is presumably due to a number of factors such as the complexities of players' movements during a match, the random characteristics of playing a game, and also a strong prevalence among researchers to infer reductionism theory as the primary interpretative model for evaluating research findings.

It has previously been suggested that the development of fatigue in soccer may be casually linked with a range of physiological factors including dehydration, hydrogen ion accumulation, potassium imbalance and substrate depletion.^[2,23] However, laboratory-based investigations have found no single factor or combination of factors

that could definitively explain fatigue during a match.^[15] This is unsurprising since exercise performance is a complex phenomenon seldom regulated by a single variable.

2.1 Complex Metabolic System Control

Metabolic control has often been described in terms of the limiting capacity of a physiological system, where either metabolite accumulation or substrate depletion causes catastrophic failure of a particular physiological system.^[52] The central premise of this cardiovascular/anaerobic model^[53] is that it is the provision of a substrate (e.g. oxygen) to muscle that limits exercise performance so that fatigue is a direct consequence of a failure of oxygen delivery to the exercising muscles. This model remains the most popular for explaining why fatigue develops during exercise, how the body adapts to training, and how these adaptations enhance performance.

According to this physiological model, progressive dehydration over the course of a soccer game would contribute to this fatiguing process by increasing the viscosity of blood, thus diminishing heat dissipation (also increasing heart rate) and as the blood flow falls behind demand, metabolites of anaerobic metabolism (in particular blood lactate and hydrogen ions) would then inhibit muscle contraction and relaxation, inducing fatigue.^[52] Recently, however, this model of physiological control of exercise has been suggested to be flawed.^[6-9] For example, Fitts^[54] identified that even in circumstances where muscles are forced to contract under ischaemic conditions, ATP concentrations do not drop below 60% of resting values, which indicates that muscle ATP concentrations are in some way 'defended'^[55] in order to prevent the development of skeletal muscle rigour. In addition, there is a wide range of inter-individual variation in muscle pH concentrations reported at exhaustion,^[56] questioning whether metabolites such as hydrogen ions can induce skeletal muscle fatigue. One possible explanation is that exercise is terminated (or effort reduced) by a central (neural) governor responding to a range of factors and continually acting to regulate performance (by manipulating muscle

recruitment) in order to ensure that these systems are never maximally utilized.^[6-9,53]

Ulmer^[9] described a teleo-anticipation model of the human body representing a complex system in which there are a vast number of regulated physiological responses centrally controlled by a brain algorithm (central governor). According to this model, feedback mechanisms from a range of peripheral physiological variables (e.g. skin temperature, plasma osmolality, blood pH) induce symptoms of fatigue^[48] to influence the conscious behaviour of an individual to reduce physical effort (figure 2), thus avoiding depletion of any single physiological variable.^[6,8,12,24] The essential component of this model is that the brain induces behaviour change through the relationship with perceived exertion (e.g. increased desire to drink through greater thirst and altered exercise intensity in the absence of fluid) before a total failure of homeostasis develops^[6-8] (figure 2). A full commentary of this model has been provided elsewhere.^[6]

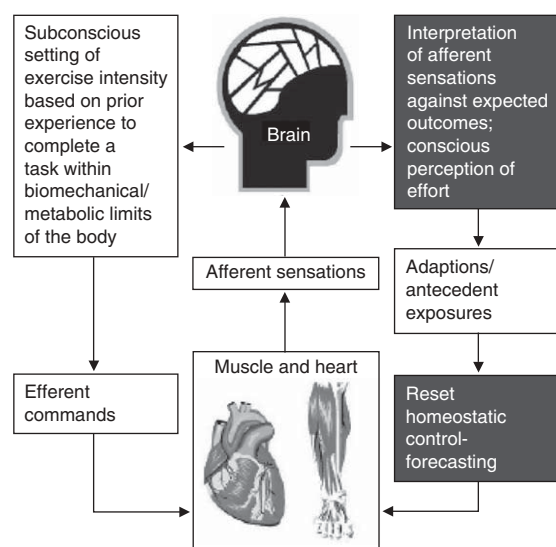


Fig. 2. Relationship between the teleoanticipatory governor centre in the brain and perceived exertion during exercise. Exercise intensity is set at a subconscious level by the CNS, and the perceived effort is the interpretation of afferent sensations against expected outcomes set by the subconscious teleoanticipatory governor centre (reproduced from Lambert et al.,^[6] with permission).

In constant-effort endurance exercise, it has recently been demonstrated that physiological mechanisms operate dynamically to continually modulate physical effort on the basis of feedback and feedforward regulatory systems.^[57] We propose that a complex control system is also highly applicable to the successful performance of team games such as soccer in which players pace themselves to complete a match on the basis of continuous physiological information from numerous regulatory mechanisms.

2.2 A Pacing Model for Elite Soccer Performance

Well conditioned soccer players rarely reach core temperatures considered critical^[18,20,37,43] during a game, lose relatively modest amounts of body fluids across most match-play conditions^[32-35] and regulate metabolite concentration (such as blood lactate) at sustainable levels^[18,43,51] for the match duration. The avoidance of critical (fatigue-inducing) change in any single physiological system is commonly achieved through a down-regulation of high-intensity effort in the second half of a match compared with the first half across all outfield positions.^[16,21,51] The greatest disparity between halves of the match occurs in less well conditioned players^[16,21,51] (e.g. figure 1) and, as such, players do not simply respond to the demands of match-play, but operate an individualized self-pacing plan within the context of the game. Aspects of each individual's pacing plan may vary according to positional requirements in the team (i.e. greater number of total sprints in strikers and wing backs), but all outfield positions in elite soccer matches require players to respond to a variety of dynamic challenges and consequent downregulation of high-intensity effort has been observed in the second half of match-play independent of outfield position.^[16,21]

While a large amount of research has focused on the limits to human performance and the development of fatigue during exercise, relatively few studies have examined the influence of pacing on sports performance.^[12,58,59] St Clair Gibson et al.^[12] recently identified that, whether or not

the endpoint of an exercise bout is known, athletes invariably apply a pacing strategy to complete the task. Within the context of continuous endurance exercise, four main pacing strategies (all-out, slow-start, even and variable pace) have previously been described,^[58] but currently no studies have proposed a model for pacing in intermittent activity team games, presumably due to the difficulties in identifying an appropriate model that adequately represents the complex movement patterns and the unpredictable energetic demands of the game. We propose that the physical responses of elite soccer players in a match conform to an individualized homeostatic set-point theory, yet within the context of a generic (across all outfield positions) pacing strategy operating at three connected levels.

Soccer players are well known to self-regulate match-play efforts according to numerous intrinsic and extrinsic factors such as their current fitness levels, the importance of the game, and positional and tactical considerations.^[2,25] The level of each player's sustained cardiovascular stress during a match has also been shown to be positively related to pre-match aerobic fitness,^[18] with better conditioned players more able to sustain a higher level of physical work throughout the full duration of the match and regulating core body temperature more effectively at a sustainable level.^[15,16,18] This suggests that players modulate effort according to a subconscious strategy based on both pre-match (e.g. prior experience in similar circumstances, fitness levels, match importance) and dynamic considerations during the game (skin temperature, accumulation of metabolites in the muscles, plasma osmolality and substrate availability). Each player's perception of a developing cellular homeostatic disturbance consequently induces behavioural changes across all outfield positions (such as covering an opponent's movement rather than intercepting, walking rather than jogging, or passing rather than dribbling) to limit physical efforts so as to avoid unsustainable elevations in physical discomfort at a premature stage of the match. As such, subconscious physiological factors influence conscious behavioural decisions to regulate effort.

Pacing strategies require continual regulation by the brain during an exercise bout.^[12,58,59] For example, during a game, each 'all-out' sprint is likely to be paced in relation to an overall 90-minute pacing plan with graduated muscle recruitment occurring throughout the sprint.^[50] This inevitably means that each match-play sprint is slower than non-match sprint performances, with fewer (or slower) sprints performed, while there is a sustained threat to homeostasis. This results in muscle power output being continuously modified in relation to an overall pacing strategy. Consequently, feedback to the brain algorithm invokes a behavioural decision either to employ a temporary reduction from vigorous work, or to maintain a continuation at a similar level if the peripheral physiological information does not suggest that the effort had compromised the integrity of the pacing strategy. Practically, this might mean players in positions requiring sporadic rather than constant movement would retain a capacity for higher intensity efforts, while facing a lower sustained stress to homeostasis. Further studies of the dynamic responses to match-play may confirm this observation.

It has previously been noted that players experience both a gradual decline in high-intensity work and also temporary periods of fatigue during a game.^[23] These phenomena can be explained by the presence of three complementary pacing strategies (figure 3).

We propose that the main (meta) pacing strategy of each soccer player is to reach the conclusion of a match having physically worked at a vigorous, yet sustainable level of performance. This level corresponds to the maintenance of tolerable physical discomfort (exercise-homeostasis) that the player is prepared to endure for the match. Homeostatic processes subsequently serve the teleological protective function of preventing damage to peripheral physiological systems whose metabolic activity is increased during exercise.^[7,8]

According to our pacing model, a brain algorithm initiates a particular pacing strategy at the start of the match, based on both the knowledge of the duration of the game and prior knowledge of previous similar activities.^[59] Prior experience

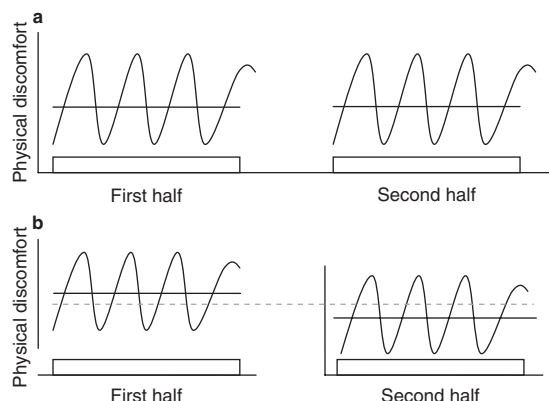


Fig. 3. A multi-level model of pacing in elite soccer. (a) represents a schematic view of the 'meta' pacing strategy pre-set by each individual player at the commencement of the match. The horizontal line represents exercise-homeostasis (subconscious level of tolerable physical discomfort) the player anticipates experiencing during the game. The oscillations represent the dynamic (micro) pacing strategy to release energy and sustainable effort in relation to the long-term objectives. (b) represents the meso-pacing plans (first and second halves of the match) with differential levels of subconscious regulatory points between halves of the match. The second-half level is subconsciously downregulated from the first half, although the mean of the two (dotted line) broadly equates to the pre-match expectations of the player.

explains the soccer-specific fitness observations of both greater total and higher intensity work by experienced players,^[21] despite similarities in aerobic capabilities with less experienced players.^[21,30] Other factors considered within the overall (meta) pacing decision include variables such as current environmental conditions, health status, and metabolic fuel reserves.^[41] These all mediate changes in pre-match decisions over the precise regulatory level of subconscious physical discomfort (exercise-homeostasis) each player is willing to endure.

A regulatory set-point of exercise-homeostasis within the meta-pacing strategy would also be affected by extrinsic factors such as the importance of the occasion, forthcoming game commitments, tactical strategies, and formations of team play. However, the dynamics of activity during the game inevitably mean that elements of the player's overall pacing strategy need to be free to vary. Consequently, while the regulatory set-point represents a behaviourally 'defended' level of homeostasis, factors such as the quality of the

opposition, crowd support, match score, tactical and positional considerations mean that players may or may not reach this pre-set level. These factors require the presence of additional intra-match pacing strategies to support the main plan of reaching the end of the match without experiencing total system failure and within the pre-set expectations of the individual player.

Self-pacing is known to occur in soccer^[2,25] and it is also clear that, within the context of their designated positional responsibilities in a team, players decide when and how to respond to the diverse challenges posed in a game. However, this has not been discussed in relation to an overall strategy, or as an explanation for the regulation of physiological responses to match-play and the avoidance of metabolic system failure. For example, Ekblom^[43] first illustrated the oscillatory dynamics of match-play heart rates during a game (figure 4), yet closer inspection of the cardiovascular responses reported demonstrate that fluctuations in match-play heart rates occur in a non-random pattern whereby each heart rate peak is rapidly followed by a sudden and substantial drop beneath a sustained mean rate (figure 4). This is consistent with match-play video analysis^[16,22] in which longer sprints and greater periods of sustained effort require prolonged (yet temporary) periods of recovery.^[23] Consistent evidence^[18,19,43] supports the observation that exercise heart rates are regulated at a higher level in the first half, compared with the second half of a match and the pattern of these heart rate oscillations within each half of the game is both non-random and somewhat predictable. This carries obvious performance implications in so far as players consciously seek temporary tactical alternatives to maximal work when exercise-homeostasis is threatened. The regulation of conscious effort enables the player to regulate important metabolic factors such as blood lactate accumulation at a relatively constant level^[43] (figure 4) and thus avoid an unsustainable (and unwanted) change in any one physiological system.^[6,12]

A single subconscious pre-match regulatory level of homeostasis does not provide players with an intra-match opportunity to re-regulate effort. According to our model, it is therefore probable that a regulatory set-point is only initially fixed for

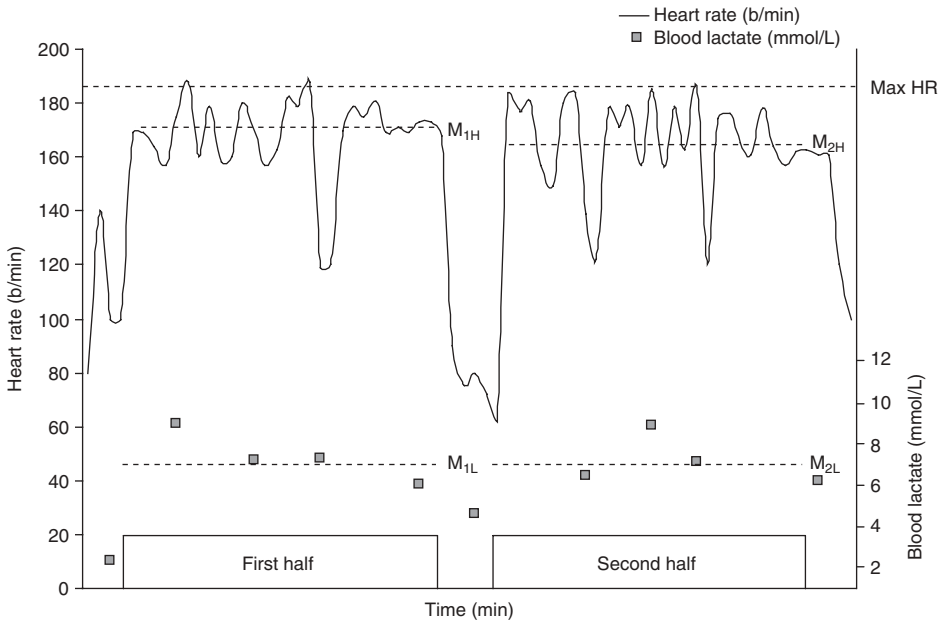


Fig. 4. An example of soccer match-play pacing strategy in operation. M_{1H} and M_{2H} = mean of first- and second-half heart rates, respectively. The difference between M_{1H} and M_{2H} lines demonstrate the downregulation of heart rate in the second half of match-play. M_{1L} and M_{2L} = mean of first and second half blood lactate concentrations, respectively. Blood pH is a defended variable and consequently blood lactate does not demonstrate substantial change between halves of the match (reproduced from Ekblom,^[43] with permission). HR_{max} = maximum heart rate.

the first half of a game (meso-pacing). At the half-time break, set-point regulation is subconsciously re-evaluated and reset on the basis of updated intrinsic and extrinsic factors. It is inevitable that the work accomplished in the second half will be reduced (as is) from the first half due to diminishing fuel stores, enhanced thirst, and accumulation of intra-muscular metabolites (table I).

The disparity between first and second half physical match-play performances demonstrates a clear trend for reduced high-intensity work in the second half of a game (figures 1 and 4). However, despite high-intensity efforts being substantially reduced in the second half,^[16,22] players still retain sufficient energy reserves to respond to match demands until the end of the game, which refutes the presence of progressive fatigue towards a situation that would induce the immediate cessation of exercise.

The presence of a subconscious, yet dynamic pacing plan protects the integrity of the longer term aim of the player to finish the match in a

reasonable condition. Therefore, although first and second half regulatory set-points will be different (although perception of effort would remain the same throughout), the mean of the two plans broadly approximate the pre-match objective (meta-plan) of the player, i.e. to finish the game and be able to respond to match demands as necessary. In consideration of this, the most important pacing strategy in soccer is the dynamic (micro) plan connecting each level of pacing strategy and which exists throughout the duration of the game. This strategy explains the occurrence of non-random^[57] temporary fatigue during a game.^[23] For example, a temporary threat to intracellular homeostasis induces behavioural change to ensure that the longer term meso- and macro-pacing strategies are not compromised by continued exercise at an unsustainable level. This will result in a temporary reduction in work (figure 3), noticeable by a short-term decline in heart rate and distances covered until the intracellular disturbance has diminished.

Table 1. A summary of characteristics within the multi-level pacing model in elite soccer

Pacing strategies	Purpose and characteristics	Set-point	Dynamic
Macro-pacing	Overall pacing plan for the match. A subconsciously pre-conceived strategy based around a set-point of tolerable physical discomfort expected over the duration of the game	✓	x
Meso-pacing	Inter-half pacing plan. This variation from the macro-plan enables up- and downregulation of effort during a match based on tactical and specific match considerations	✓	x
Micro-pacing	Dynamic pacing plan. This strategy enables the player to evaluate the impact of acute periods of intense exercise on the set-point strategies. For example, a prolonged sprint late in a game may require an extended recovery to protect homeostasis	x	✓

set-point=pacing strategies based on defending exercise homeostasis at an individualized set-point of tolerable physical discomfort in accordance with each player's fitness and experience; **dynamic**=pacing strategy based on the principles of negative feedback where temporary fluctuations from the set-point require longer or shorter recovery, depending on whether exercise homeostasis is threatened.

The dynamic-pacing strategy therefore connects both meso- and macro-strategies by regulating intensity and frequency of short-term efforts in relation to both longer term strategies. It is with consideration to the longer term objectives that a player will subconsciously only expend maximal effort at any one time during the match if it consistent with the objectives of the meso- and macro-pacing strategies. Where the match-play requirements incur a prolonged threat to homeostasis, the player will require extended recovery to reduce the intracellular threat and so will consciously seek tactical alternatives to short-term high-intensity efforts. As such, players performing either a long sprint, or a rapid series of high-intensity activities should be expected to seek extended opportunities to minimize energy expenditure as has been observed in motion analysis of match play.^[16,21,22] The characteristics of our hypothetical pacing plan are summarized in table 1.

All three of the connected pacing strategies proposed within our hypothetical model demonstrate how players are able to complete the full duration of the game. The presence of a second half endpoint strategy (meso-pacing) ensures that the player can down-regulate effort to a new level for a prolonged period and the dynamic strategy (micro-strategy) ensures that no single factor such as dehydration causes the immediate cessation of exercise.

3. Conclusions

It is in consideration of the complex factors that control performance that a single variable such as dehydration is unlikely to be causally linked as the

limitation to physiological performance in soccer. In our opinion, a more probable scenario is that dehydration is one feature within a complex regulatory system and progressive fluid loss acts as a physiological (subconscious) stimulus to initiate conscious feelings of fatigue such as thirst. Consequently, symptoms of fatigue influence physical efforts and affect tactical decision-making across the outfield positions during match-play to prevent excessive threats to homeostasis.

In this current opinion article, we propose that a multi-level pacing plan ensures that players reach the conclusion of a game. Clearly, players must respond to the demands of the game, but if there were no pacing then no-one would reach the end of the match and no player would have reserves of energy for short-term sprints in the later stages of a game. This is not the case and, in practical terms, it can be observed in players' tactical decisions to cover an opponent's movement rather than to initiate an energy-consuming interception, or to pass the ball to a team mate rather than to perform a dribble. Further studies examining the dynamic characteristics of elite match-play may further support our pacing model. We therefore suggest that moderate dehydration is only a single sign that significant metabolic activity has occurred during exercise and it is not the cause of fatigue in elite soccer.

Acknowledgements

No sources of funding were received in the preparation of this article and the authors have no conflicts of interest directly relevant to its contents.

References

1. Maughan RJ, Leiper JB. Fluid replacement requirements in soccer. *J Sports Sci* 1994; 112: S29-34
2. Reilly T. Energetics of high intensity exercise (soccer) with particular reference to fatigue. *J Sports Sci* 1997; 15: 257-63
3. Montain SJ, Coyle EF. Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *J Appl Physiol* 1992; 73: 1340-50
4. Saunders AG, Dugas JP, Tucker R, et al. The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. *Acta Physiol Scand* 2005; 183: 241-55
5. Edwards AM, Mann ME, Marfell-Jones MA, et al. The influence of moderate dehydration on soccer performance: physiological responses to 45-min of outdoors match-play and the immediate subsequent performance of sport-specific and mental concentration tests. *Br J Sports Med* 2007; 41: 385-91
6. Lambert EV, St Clair Gibson A, Noakes TD. Complex systems model of fatigue: integrative homeostatic control of peripheral systems during exercise in humans. *Br J Sports Med* 2005; 39: 52-62
7. Noakes TD, St Clair Gibson A. Logical limitations to the 'catastrophe' models of fatigue during exercise in humans. *Br J Sports Med* 2004; 38: 648-9
8. Noakes TD, St Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans. *Br J Sports Med* 2004; 38: 511-4
9. Ulmer H-V. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia* 1996; 52: 416-20
10. Gisolfi CV, Copping JR. Thermal effects of prolonged treadmill exercise in heat. *Med Sci Sports Exerc* 1974; 6: 108-13
11. Walsh R, Noakes TD, Hawley J, et al. Impaired high-intensity cycling performance time at low levels of dehydration. *Int J Sports Med* 1994; 15: 392-8
12. St Clair Gibson A, Lambert EV, Rauch HG, et al. The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Med* 2006; 36: 705-22
13. Sawka MN, Noakes TD. Does dehydration impair exercise performance? Contrasting perspectives. *Med Sci Sports Exerc* 2007; 39: 1209-17
14. Reilly T, Secher N, Snell P, et al. *Physiology of sports*. London: E & FN Spon; 1990
15. Bangsbo J. The physiology of soccer: with special reference to intense intermittent exercise. *Acta Physiol Scand* 1994; 15: 1-156
16. Mohr M, Krstrup P, Bangsbo J. Match performance of high-standard soccer players with special reference to the development of fatigue. *J Sports Sci* 2003; 21: 519-28
17. Reilly T, Thomas V. A motion analysis of workrate in different positional roles in professional football match-play. *J Hum Mov Stud* 1976; 2: 87-97
18. Edwards AM, Clark N. Thermoregulatory observations in soccer match-play: professional and recreational level applications using an intestinal pill system to measure core temperature. *Br J Sports Med* 2006; 40: 133-8
19. Mohr M, Krstrup P, Nybo L, et al. Muscle temperature and sprint performance during soccer matches-beneficial effect of re-warm-up at half-time. *Scand J Med Sci Sports* 2004; 14: 156-62
20. González-Alonso J, Teller C, Andersen SL, et al. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 1999; 86: 1032-9
21. Bangsbo J, Norregaard L, Thorsoe F. Activity profile of competition soccer. *Can J Sports Sci* 1991; 16: 110-6
22. Bangsbo J, Mohr M. Variations in running speed and recovery time after a sprint during top-class soccer matches. *Med Sci Sports Exerc* 2005; 37: S87
23. Mohr M, Krstrup P, Bangsbo J. Fatigue in soccer: a brief review. *J Sports Sci* 2005; 23: 593-9
24. St Clair Gibson A, Noakes TD. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br J Sports Med* 2004; 38: 797-806
25. Drust B, Atkinson G, Reilly T. Future perspectives in the evaluation of the physiological demands of soccer. *Sports Med* 2007; 37: 783-805
26. Drust B, Reilly T, Cable NT. Physiological responses to laboratory-based soccer-specific intermittent and continuous exercise. *J Sports Sci* 2000; 18: 885-92
27. Nicholas CW, Lakomy HK, Phillips A, et al. Influence of ingesting a carbohydrate-electrolyte solution on endurance capacity during intermittent, high-intensity shuttle running. *J Sports Sci* 1995; 13: 283-90
28. Nicholas CW, Nuttall FE, Williams C. The Loughborough Intermittent Shuttle Test: a field test that simulates the activity pattern of soccer. *J Sports Sci* 2000; 18: 97-104
29. Leger LA, Lambert J. A maximal multistage 20-m shuttle run test to predict $\dot{V}O_{2\max}$. *Eur J Appl Physiol* 1982; 49: 1-12
30. Edwards AM, Clark N, Macfadyen AM. Test performance indicators from a single soccer specific fitness test differentiate between highly trained and recreationally active soccer players. *J Sports Med Phys Fit* 2003; 43: 14-20
31. McGregor SJ, Nicholas CW, Lakomy HK, et al. The influence of intermittent high intensity shuttle running and fluid ingestion on the performances of a soccer skill. *J Sports Sci* 1999; 17: 895-903
32. Mustafa KY, Mahmoud NE. Evaporative water loss in African soccer players. *J Sports Med Phys Fitness* 1979; 19: 181-3
33. Maughan RJ, Merson SJ, Broad N, et al. Fluid and electrolyte intake and losses in elite soccer players during training. *Int J Sports Nutr Ex Met* 2004; 14: 333-46
34. Shirreffs SM, Aragon-Vargas LF, Chamorro M, et al. The sweating response of elite professional soccer players to training in the heat. *Int J Sports Med* 2005; 26: 90-5
35. Burke LM. Fluid balance during team sports. *J Sports Sci* 1997; 15: 287-95
36. Broad EM, Burke LM, Cox GR, et al. Body weight and voluntary fluid intakes during training and competition sessions in team sports. *Int J Sports Nutr* 1996; 6: 307-20

37. Byrne C, Lee JKW, Chew SAN, et al. Continuous thermoregulatory responses to mass-participation distance running in heat. *Med Sci Sports Exerc* 2006; 38: 803-10
38. Burke LM, Hawley JA. Fluid balance in team sports: guidelines for optimal practices. *Sports Med* 1997; 24: 38-54
39. Maughan RJ, Shirreffs SM, Leiper JB. Errors in the estimation of hydration status from changes in body mass. *J Sports Sci* 2007; 25: 797-804
40. Shepard R. Meeting carbohydrate and fluids needs in soccer. *Can J Sports Sci* 1990; 15: 165-71
41. Rauch HGL, St Clair Gibson A, Lambert EV. A signaling role for muscle glycogen in the regulation of pace during prolonged exercise. *Br J Sports Med* 2005; 39: 34-8
42. Baldwin J, Snow RJ, Gibala MJ, et al. Glycogen availability does not affect the TCA cycle or TAN pools during prolonged, fatiguing exercise. *J Appl Physiol* 2003; 94: 2181-7
43. Ekblom B. Applied physiology of soccer. *Sports Med* 1986; 3: 50-60
44. Armstrong LE, Costill DL, Fink WJ. Influence of diuretic-induced dehydration on competitive running performance. *Med Sci Sports Exerc* 1985; 17: 456-61
45. Gopinathan PM, Pichan G, Sharma VM. Role of dehydration in heat stress-induced variations in mental performance. *Arch Environ Health* 1988; 43: 15-7
46. Serwah N, Marino FE. The combined effects of hydration and exercise heat stress on choice reaction time. *J Sci Med Sport* 2006; 9: 157-64
47. Hoffman JR, Stavsky H, Falk B. The effect of water restriction on anaerobic power and vertical jumping height in basketball players. *Int J Sports Med* 1995; 16: 214-8
48. St Clair Gibson A, Baden DA, Lambert MI, et al. The conscious perception of the sensation of fatigue. *Sports Med* 2003; 33: 167-76
49. Robinson T, Hawley J, Palmer G, et al. Water ingestion does not improve 1-h cycling performance in moderate ambient temperatures. *Eur J Appl Physiol* 1995; 71: 153-60
50. St Clair Gibson A, Schabert EJ, Noakes TD. Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Regul Integr Comp Physiol* 2001; 281: R187-96
51. Smaros G. Energy usage during a football match. In: Vecchiet L, editor. *Proceedings of the 1st International Congress on Sports Medicine Applied Football*; 1979. Rome: Guanello; 1980: 801
52. Costill DL, Bennett A, Branam G, et al. Glucose ingestion at rest and during prolonged exercise. *J Appl Physiol* 1973; 34: 764-9
53. Noakes TD. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scan J Med Sci Sports* 2000; 10: 123-45
54. Fitts RH. Cellular mechanisms of muscle fatigue. *Physiol Rev* 1994; 74: 49-94
55. Hochachka PW. *Muscles as molecular and metabolic machines*. Boca Raton (FL): CRC Press, 1994
56. Mannion AF, Jakeman PM, Willan PLT. Skeletal muscle buffer value, fibre type distribution and high intensity exercise performance in man. *Exp Physiol* 1995; 80: 89-101
57. Tucker R, Bester A, Lambert EV, et al. Non-random fluctuations in power output during self-paced exercise. *Br J Sports Med* 2006; 40: 912-7
58. Foster C, Schrager M, Snyder AC, et al. Pacing strategy and athletic performance. *Sports Med* 1994; 17: 77-85
59. Paterson S, Marino FE. Effect of deception of distance on prolonged cycling performance. *Percept Mot Skills* 2004; 98: 1017-26
60. Noakes TD, Sharwood K, Speedy D, et al. Three independent biological mechanisms cause exercise-associated hyponatremia: evidence from 2135 weighed competitive athletic performances. *PNAS* 2005; 102: 18550-5

Correspondence: Dr *Andrew M. Edwards*, UCOL Institute of Technology, Faculty of Health Sciences, Palmerston North, New Zealand.
E-mail: a.m.edwards@ucol.ac.nz