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

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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 remains, 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 ≥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

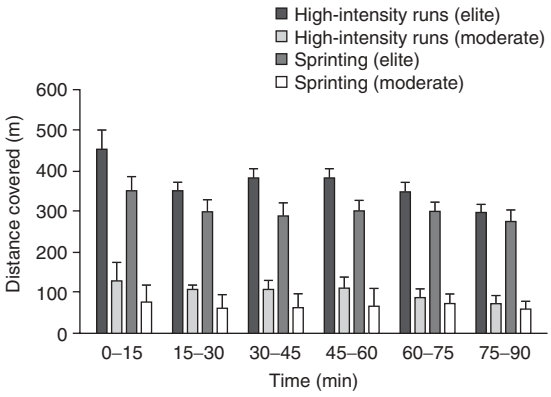

Fig. 1. A comparison of high-intensity running and sprinting during match-play among elite- and moderate-level soccer players. The high-intensity running diminishes to protect the integrity of defended variables (such as core temperature, plasma osmolality, blood pH) from reaching critical levels (data 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]

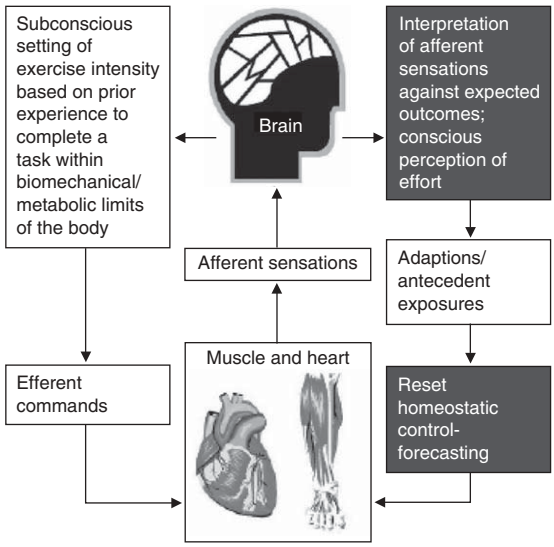

Fig. 2. Relationship between the teleoanticipatory governor centre in the brain and perceived exertion during exercise. Exercise intensity is set at a subconscious level by the CNS, and the perceived effort is the interpretation of afferent sensations against expected outcomes set by the subconscious 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 downregulation 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

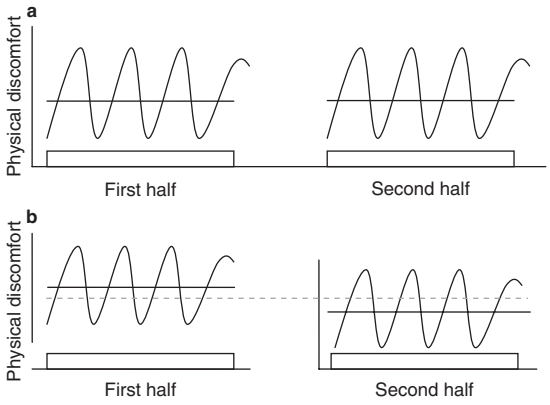

Fig. 3. A multi-level model of pacing in elite soccer. (**a**) represents a schematic view of the 'meta' pacing strategy pre-set by each individual player at the commencement of the match. The horizontal line represents exercise-homeostasis (subconscious level of tolerable physical discomfort) the player 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

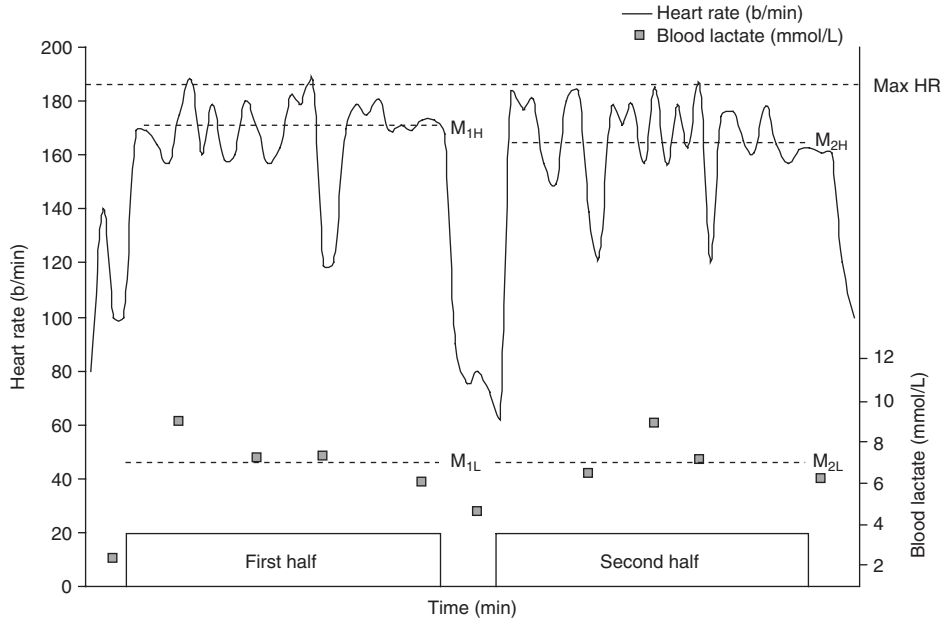

Fig. 4. An example of soccer match-play pacing strategy in operation. M_{1H} and M_{2H} = mean of first- and second-half heart rates, respectively. The difference between M_{1H} and M_{2H} lines demonstrate the downregulation of heart rate in the second half of match-play. M_{1L} and M_{2L} = mean of 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). $\mathbf{HR_{max}}$ = 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 I. 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 I.

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.

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Constraints on the Complete Optimization of Human Motion

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Abstract

In sport and exercise biomechanics, forward dynamics analyses or simulations have frequently been used in attempts to establish optimal techniques for performance of a wide range of motor activities. However, the accuracy and validity of these simulations is largely dependent on the complexity of the mathematical model used to represent the neuromusculoskeletal system. It could be argued that complex mathematical models are superior to simple mathematical models as they enable basic mechanical insights to be made *and* individual-specific optimal movement solutions to be identified. Contrary to some claims in the literature, however, we suggest that it is currently not possible to identify the complete optimal solution for a given motor activity. For a complete optimization of human motion, dynamical systems theory implies that mathematical models must incorporate a much wider range of organismic, environmental and task constraints. These ideas encapsulate why sports medicine specialists need to adopt more individualized clinical assessment procedures in interpreting why performers' movement patterns may differ.

A compelling challenge facing biomechanists working in sports medicine and the sports and exercise sciences is that of identifying optimal techniques for the performance of a wide range of motor activities. In this way, their work with movement scientists could lead to improvements in sport performance while preventing the occurrence of injuries through dysfunctional movement patterning. In tackling this challenge, biomechanists have typically resorted to forward dynamics analyses or simulations, in which sets of ordinary differential equations derived from Newtonian and Lagrangian mechanics are used to calculate and propose optimal movement solutions.[1] In a forward dynamics analysis, input variables are typically composed of the applied forces or net joint torques acting on the neuromusculoskeletal system and the calculated output variables are kinematic data describing the motion of the component parts (i.e. torso and limb segments) of the neuromusculoskeletal system. The accuracy and validity of these output variables are largely dependent on the complexity of the mathematical model used to represent the neuromusculoskeletal system.

There is a dichotomy of opinion in the literature as to the requisite complexity of mathematical models required for meaningful forward dynamics analyses. On the one hand, Hatze[2] suggested that, to identify the complete optimal

movement solution for a given motor activity, it is necessary to use a highly sophisticated mathematical model. In contrast, Alexander[3] argued that attempting to reproduce the complexity of the neuromusculoskeletal system in a mathematical model is a futile exercise and the generation of simpler (i.e. more abstract) models should be given preference. The general consensus of opinion, however, is that the complexity of the mathematical model used to represent the neuromusculoskeletal system should be governed by the research question to be answered and the analytical goals of the investigator.[1,4,5]

In this article, we critically analyse mathematical models used in forward dynamics analyses or simulations and discuss how applications of dynamical systems theory in the movement sciences might influence how the process of optimization may be conceptualized by biomechanists working in sports medicine and the sports and exercise sciences. We begin by providing a brief overview of mathematical models currently used to represent the neuromusculoskeletal system and we outline the relative merits of simple and complex mathematical models. Next, we introduce some key concepts of dynamical systems theory, applied to the human movement sciences, with specific reference to Newell's model of constraints.[6] We then discuss the limitations of current mathematical models of the neuromusculoskeletal system from a dynamical systems perspective and highlight the potential of more sophisticated mathematical models for optimizing movement.

Contrary to some extant views in sports and exercise biomechanics, we discuss why completely optimal movement solutions for specific motor activities, generalizable to all performers, may currently be inaccessible. We evaluate the argument that a complete optimal solution for a given motor activity requires mathematical models that incorporate a much wider range of organismic, environmental and task constraints. We then outline a research strategy for identifying the constraints most influential in shaping and guiding emergent patterns of movement coordination and control. To conclude, we discuss the potential implications of a dynamical systems approach for practitioners in science and medicine and the assessment of functional or dysfunctional movement patterns.

1. Mathematical Models of the Neuromusculoskeletal System

Current mathematical models used to represent the neuromusculoskeletal system range from the very simple to the highly complex. Simple mathematical models are generally characterized by a limited number of rigid body segments, which are of a standardized length and have uniform mass distribution. These body segments are typically interconnected by frictionless pin joints and are operated by no or very few muscles. An example of a simple mathematical model and its application was proposed by Alexander[3] in descriptions of walking and jumping. The model for walking (figure 1) consisted of a massless leg and a point mass at the hip and the model for jumping (figure 2) consisted of a massless thigh and shank and a point mass at the hip. The model for jumping also included a knee extensor muscle that was assigned realistic force-velocity properties. Despite the abstract nature of these models, they were instructive in providing basic insights such as why normal

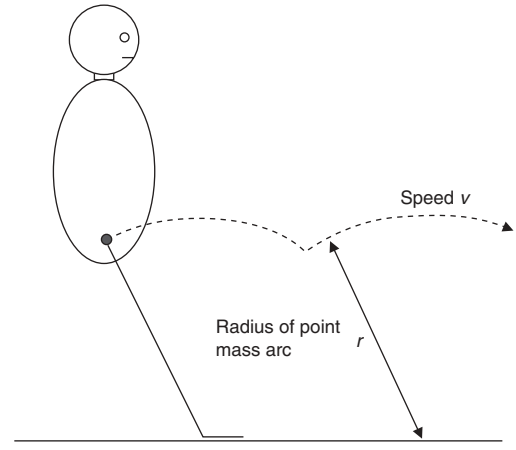

Fig. 1. The simple 2-segment model of walking (reprinted from Alexander,[3] with permission from Elsevier © 1992).

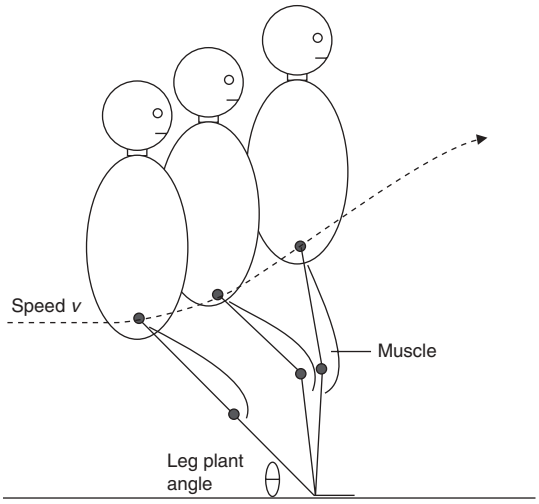

Fig. 2. The simple 3-segment model of jumping (reprinted from Alexander,[3] with permission from Elsevier © 1992).

walking speeds are limited to approximately 3 m/s, and why long jumpers run up faster and take off using larger leg plant angles than high jumpers.

Complex mathematical models, in contrast, generally include many rigid or deformable body segments and muscle groups, which have their own unique individual-specific anthropometric (geometrical and inertial) and strength characteristics, respectively. For example, Hatze[2,7-10] described a 17-segment mathematical model of the neuromusculoskeletal system complete with 42 mechanical degrees of freedom and 46 individual muscle groups. The 17 body segments were as follows: the abdomino-thoracic segment, the head-neck segment, left and right shoulders, upper arms, forearms and hands, the abdomino-pelvic segment, and the left and right thighs, lower legs and feet. Figure 3 shows the 42 generalized coordinates ($q_1, \ldots, q_{42}$), which defined the configuration of the mathematical model in space, and the 17 local, segment-fixed coordinate systems (x_1, y_1, z_1) … (x_{17}, y_{17}, z_{17}), which were located at the centre of mass of each segment. The 46 individual muscle groups required neural control time histories as input variables that could be adjusted until the required limb and torso movements were produced (see Hatze[11] for a comprehensive review). To personalize the equations of motion, a battery of 242 individual-specific anthropometric measurements, and a variety of isometric and isotonic strength measurements, were required.

This sophisticated mathematical model of the neuromusculoskeletal system took account of gender differences and even the specific morphological characteristics of obesity and pregnancy in humans. Hatze[7] claimed that this mathematical model was adequate for investigating gross human movement, as in his weighted-boot study.[12] Although it has often been reported[13] that Hatze used the full neuromusculoskeletal model in his simulation of the

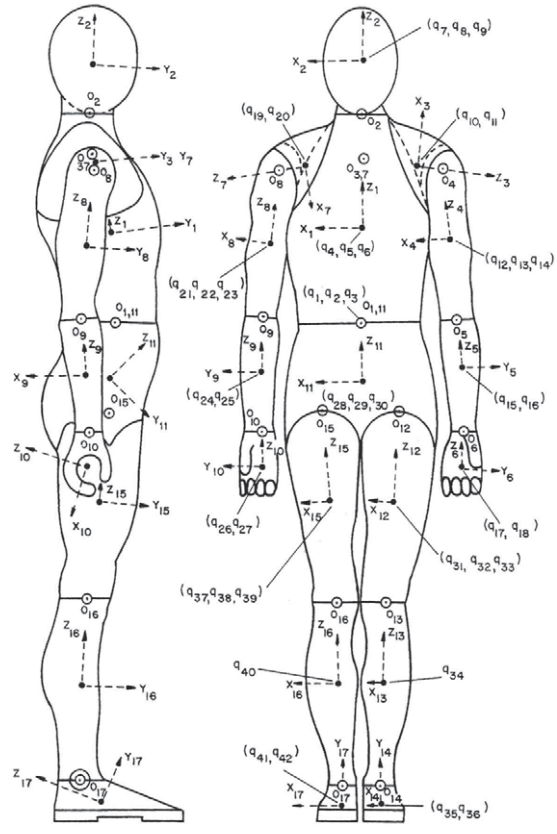

Fig. 3. Lateral and anterior view of the 17-segment mathematical model (reprinted from Hatze,[7] with permission from Elsevier © 1980).

long jump,[14] joint torques were optimized in that study without using the full muscle model.[15]

1.1 Model Sophistication

Exactly how sophisticated a mathematical model has to be is a contentious issue for sport and exercise biomechanists. In the past, simple 2-dimensional (2-D) or planar models have been popular because of their mathematical convenience and computational simplicity.[16] Indeed, due to the comparatively small number of outcome scenarios with simple mathematical models, it is easier to locate global optima with these models than it is with more complex mathematical models. The latter typically require the use of more advanced optimization techniques, such as simulated annealing,[17] to decipher global optima from local optima. Another advantage of using simple mathematical models is that they provide results that are more easily interpretable by clinical practitioners, coaches and athletes.[15] Simple mathematical models, such as those outlined by Alexander[18] for walking, running, jumping and throwing, have been useful for providing basic mechanical insights and developing fundamental principles of human movement. Indeed, it has been argued that the most fundamental understanding often comes from the simplest of models[19] and that establishing cause and effect is often easier with simpler models.[3]

More recently, however, more complex 3-dimensional (3-D) or spatial models have gained popularity as a result of advances in computer technology and the advent of automated equation-generating programs such as AUTOLEV, NEWEUL, MACSYM, DADS, ADAMS and SYMBA.[20,21] Since most human movements are not planar, 3-D models are more realistic than 2-D models.[10] Complex mathematical models can be considered superior to simple mathematical models as they putatively enable individual-specific optimal movement solutions to be identified *and*, with the use of appropriate constraints during the modelling process, can also provide basic mechanical insights for developing fundamental principles of human movement.[13] Due to the wide range of constraints on movement behaviour, and contrary to some reports in sport and exercise biomechanics,[12] it is argued that the identification of the complete optimal movement solution for a given motor activity is currently not possible.

In section 2, we elucidate these arguments. We provide an overview of some key concepts of dynamical systems theory, such as 'self-organizing optimality', and elaborate why it is currently not possible to identify the complete optimal movement solution for a given motor activity. To support these arguments, we outline a constraints framework[6] that could, in principle, enable the complete optimal movement solution for a given motor activity, at any given time, and for any given individual, to be identified.

2. The Role of Pleiotropy and Degeneracy in Complex, Dynamical Neurobiological Systems

Traditionally, the human neuromusculoskeletal system has been conceptualized as a deterministic, information-driven machine finitely controlled via integrated sensory feedback loops by a capacity-limited microcomputer acting as the brain.[22] In traditional deterministic experimental paradigms, a major aim is the reduction of uncertainty for examining movement behaviour of such 'closed systems'. From this philosophical standpoint, legitimate scientific enterprises include the empirical identification of 'optimization mechanisms' of behaviour[23] (e.g. how some individuals achieve reliable movement performances over repeated trials). This perspective, popularized by scientists in the physical sciences, engineering and robotics, has been questioned in the human movement sciences by a biophysical theoretical orientation in favour of conceptualizing neurobiological systems as complex, nonlinear dynamical systems. From this relatively new perspective, the human neuromusculoskeletal system can be described as an integrated network of co-dependent subsystems that are composed of many interacting component parts, or degrees of freedom, operating over multiple scales of space and time.

It has become apparent that complex neurobiological systems are *pleiotropic* and *degenerate* with their multitudinous degrees of freedom having the capacity to adopt different roles in satisfying the different constraints on their behaviour. That is, such systems are versatile and not highly specialized. Their inherent *pleiotropy* is based on their capacity to use their many degrees of freedom in different roles. Pleitropy instills inherent back up in metastable complex systems (systems that switch continuously between states of stability and instability) by providing a number of different options to support the search for alternative behavioural solutions. Conceptualizing neurobiological systems as complex suggests that inherent pleitropy could support a range of alternative outcomes from the dynamical interactions of system components (e.g. muscles or joints in a human movement system or the players in a sports team). For example, characteristics such as complexity and pleitropy make it challenging to identify 'the' most appropriate solution for a performer in sport, requiring a re-evaluation of decision-making behavior in dynamic performance contexts. Characterizing attacker-defender interactions in team games as a complex system implies that inherent pleitropy could make it hard to predict decision making of players, since there could be a range of alternative outcomes from the dynamical interpersonal interactions of system components.

Degeneracy refers to the capacity of structurally different components of complex neurobiological systems to achieve different outcomes in varying contexts, and is exemplified by the networks existing at different levels of human movement systems including molecular, genetic, neural and musculoskeletal.[22] Degeneracy in complex biological systems provides the neurophysiological basis for the diversity of actions required to instantaneously negotiate information-rich, dynamic environments, as well as providing a huge evolutionary fitness advantage.[24] In recent years, questions have arisen over the efficacy of descriptions of human movement systems as complex systems with many redundant degrees of freedom, inspired by the insights of Bernstein.[25] The term 'redundancy' is a more relevant descriptor for physical and engineering control systems, generally used in relation to the strategy of duplicating system components as a back-up in case of system failure.[24] Redundant systems provide similar outputs from different components. Biological systems are not structured in the same way as mechanical or electronic systems and there are different solutions for ensuring robustness and adaptivity in animate systems. In this respect, degeneracy is a better descriptor of biological systems than the term redundancy.

A good example of degeneracy in biological systems is during sensory deficits, where the use of brain imaging techniques has revealed that the visual cortex in blind humans can be activated during tasks requiring attention to auditory and haptic information sources. These findings have demonstrated how compensatory adjustment in degenerate nervous systems can result in the pickup of novel information sources for guiding movements.[26] Additionally, it is now well established that motor equivalence, or the ability of different patterns of neuromuscular activity to achieve specific movement outcomes, can provide the degenerate human movement system with a distinct advantage through the contextual adjustment of actions to information-rich environments, typically needed in many performance environments. Degeneracy of human movement systems provides the capacity to trade-off specificity and diversity of actions under changing task constraints, influencing the emergence of decision making and action.

Characteristics such as complexity, pleitropy and degeneracy make it challenging to identify a 'common optimal movement solution' for all performers, requiring a re-modelling of movement behaviour in dynamic performance contexts. In complex neurobiological systems, spontaneous pattern formation among degrees of freedom emerges, not through the intervention of an external regulating agent, but through generic processes of physical self-organization.[27-29] As neuromusculoskeletal systems are 'open' systems – that is, they are stable yet far from thermodynamic equilibrium – they constantly engage in energy transactions with the environment,

encouraging orderly and stable relations to form between their component parts.

At the level of muscular-articular links,[30,31] the number of biomechanical degrees of freedom to be regulated can effectively be reduced by the spontaneous formation of functional muscle synergies[25,32] or coordinative structures.[33,34] Tuller et al.[35] defined a coordinative structure as "… a group of muscles often spanning several joints that is constrained to act as a single functional unit." A characteristic of a coordinative structure in pleiotropic and degenerate neurobiological systems is that, if one of the component parts introduces an error into the common output, the other component parts automatically make compensatory adjustments to minimize the effect of the original error.[36,37] Furthermore, the 'soft assembly' of coordinative structures is a feature of neurobiological complexity, affording great flexibility and adaptability as individual muscles can participate in different coordinative structures on different occasions.[38,39] These task-specific structural units can be modulated or tuned by perceptual information to accommodate sudden, unforeseen changes in task demands.[40,41]

2.1 Constraints on Neurobiological Complexity

The formation of coordinative structures or functional muscle synergies is dependent not only on processes of self-organization, but also the constraints imposed on specific neuromusculoskeletal systems.[6,25,42-44] The constraints concept has a rich tradition in theoretical physics, evolutionary and theoretical biology, and mathematics. In general, constraints are internal or external features that limit the number of possible configurations that complex systems, such neurobiological systems, can adopt. Constraints define the boundaries within which human neuromusculoskeletal systems must operate and, therefore, shape the emergence of patterns of coordination and control. The problem for neurobiological systems with a vast multitude of degrees of freedom is to constrain the number of micro-components involved in system behaviour, and in the movement sciences this has been addressed as 'Bernstein's problem'.[36] According to the influential framework of Newell,[6] there are three categories of performance constraint – organismic, environmental and task – that coalesce to channel and guide patterns of coordination and control produced by the neuromusculoskeletal system (see figure 4). It is important to note, however, that these categories of constraint identify the source, rather than the actual nature, of the constraint.[45]

Newell[6] considered organismic constraints to be those constraints that are endogenous to individual neuromusculoskeletal systems. Organismic constraints can be subdivided into structural and functional constraints. Structural organismic constraints tend to be physical constraints that remain relatively constant over time and include: height, body mass and composition; genetic make-up; the anthropometric and inertial characteristics of the torso and limbs; the number of mechanical degrees of freedom and ranges of motion of articulating structures; the fast- and slow-twitch fibre composition; angle of pennation, cross-sectional area, and the activation and fatigue characteristics of skeletal muscle; and so on.[46] Functional organismic constraints that have a relatively faster rate of change, on the other hand, tend to vary quite considerably over time and can either be physical or psychological. Important functional organismic constraints include intentions, emotions, perception, decision-making and memory. Perhaps the most prominent and influential organismic constraint that can shape movement coordination is the intentions of the specific individual under scrutiny.[29]

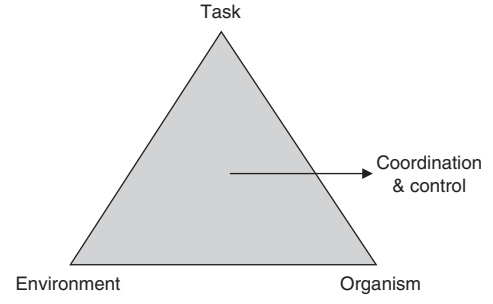

Fig. 4. Newell's[6] theoretical model of interacting constraints.

Environmental constraints were defined as exogenous to the neuromusculoskeletal system. They tend to be constraints that pertain to the spatial and temporal layout of the surrounding world or the field of external forces that are continually acting on the neuromusculoskeletal system. Environmental constraints are typically more challenging to manipulate during experimentation. Examples of environmental constraints include ambient light and temperature, altitude, acoustic information, ubiquitous gravitational forces and the reaction forces exerted by *terra firma* and other contact surfaces and apparatus. More recently, Newell and Jordan[47] proposed that environmental constraints encompassed any physical constraint beyond the boundaries of the organism. Implements, tools or apparatus, originally categorized by Newell[6] as being tasks constraints, were classified under the category of environmental constraints under the revised framework.

Task constraints, as the name implies, are specific to the task being performed and are related to task goals and rules governing performance contexts as well as boundaries and instructional constraints imposed on performers. The constraints of the task operate as an umbrella over all other constraints in influencing what patterns of coordination and control are produced.[6,42,44] The relative impact of task constraints on the neuromusculoskeletal system is largely dependent on the motor activity being performed. For example, in many ontogenetic motor activities, task constraints set certain limits or boundaries to patterns of coordination and control. In many phylogenetic motor activities, by contrast, task constraints do not inhibit or restrict patterns of coordination and control to any great extent.

One of the most profound conceptual implications of Newell's model of constraints[6] is that optimal patterns of coordination and control emerge from the unique confluence of constraints impinging on individual neuromusculoskeletal systems through a process referred to as 'self-organizing optimality'. The concept of self-organizing optimality is tantamount to the 'constrained optimization' concept advanced in the theoretical and evolutionary biology literatures by, amongst others, Maynard Smith[48] and Staddon and Hinson.[23] Constrained optimization states that the behaviour of a biological system at any time will always be optimal for the specific confluence of constraints acting on the system, or as Mazur[49] stated, the system will "always do the best it can." An important point for sport scientists and clinicians to note, however, is that even though the pattern of coordination and control produced by the neuromusculoskeletal system might be optimal in relation to the immediately imposed constraints, the performance outcome could still be suboptimal or unsuccessful.

It is important to note that models of 'constrained optimization' are harmonious with concepts from non-linear dynamics, such as emergence and self-organization under constraints, and capture the adaptability and compensatory variability of human actions required in dynamic performance contexts such as sport and exercise. Importantly, this modelling trend fits well with data from recent studies revealing the inherent degeneracy of biological movement systems.[24,50] From this constraints-led perspective, optimal motor performance, therefore, could be better defined as an individual attempting to satisfy the unique combination of interacting constraints impinging on him/her at any given point in his/her development. From this theoretical standpoint, optimization may be better considered from the perspective of the individual under scrutiny and the confluence of constraints impinging on that individual, not some abstract, external reference or independent criterion.

Since the constraints imposed on an individual dynamical movement system can fluctuate continuously over time, the optimal pattern of coordination and control for any given motor activity can change accordingly. Furthermore, as the conscious and sub-conscious interpretation of these constraints is dependent on the intrinsic dynamics (i.e. preferred states of coordination and control based on neuromusculoskeletal system architecture, genetic composition, previous task experience, emotions, etc.) of each individual under scrutiny, optimal patterns of coordination

and control for any given motor activity will always be individual-specific.[6,45] Inter-individual variations in movement patterning may, therefore, be interpreted as adaptive behaviour on the part of dynamical neurobiological systems as they exploit surrounding constraints to shape the functional, self-sustaining patterns of behaviour that emerge in specific performance or rehabilitation contexts. Clearly, these theoretical insights from neurobiology have important implications for sport and exercise biomechanists working towards the complete optimization of human movement.

3. Modelling the Neuromusculoskeletal System: A Dynamical Systems Approach

It could be argued that, from a dynamical systems perspective, a major reason why sport and exercise biomechanists have been unable to identify the complete optimal solution for a given motor activity is that mathematical models of the neuromusculoskeletal system currently do not take into account the full range and uniqueness of the constraints impacting on each individual. Indeed, as Newell[51] pointed out, "…optimization modelling has been largely confined to a consideration of mechanical constraints. However, mechanical constraints are clearly not sufficient criteria for optimization in biological systems, although they represent an important beginning to this effort." Newell's[51] analysis suggests that sport and exercise biomechanists working towards the complete optimization of human motion need to include a much wider range of organismic, environmental and task constraints in their mathematical models of the neuromusculoskeletal system because "… in principle, these constraints will determine the optimal coordination and control for a given individual in a given activity." These ideas are reminiscent of criticisms of the 'adaptationist' stance in evolutionary biology which surmises that the constraints of natural selection are so powerful that a biological organism can be 'atomized' into traits or structures, each of which can be adapted independently of the whole system. This theoretical perspective has been criticized for being too reductionist. That is, it has inappropriately reduced complex systems in nature to a series of discrete objects each operating under a narrow range of constraints, when actually a wide range of constraints are considered to interact in shaping the evolutionary development of the whole organism.[52]

In recent times, because of advances in technology and increased computer processing power, the number of constraints (or parameters in biomechanical modelling parlance) that can be incorporated into mathematical models of the neuromusculoskeletal system has grown significantly. Organismic constraints have begun to be included in some models, for example in the form of individual-specific anthropometric (geometrical and inertial) parameters,[53,54] strength parameters,[55,56] soft tissue movement (so-called 'wobbling' masses),[57,58] and limits to joint ranges of motion.[59,60] In the highly sophisticated mathematical model of the neuromusculoskeletal system described by Hatze,[2,7-10] a wide range of structural and functional organismic constraints has been incorporated into the mathematical model of the skeletal, muscular and neural subsystems. The most advanced of these was the model of the muscular subsystem[7,9,61,62] designed to closely mimic the behaviour of real muscle tissue. Indeed, according to Hatze[11] this muscle model fully accounted for the dynamics of sarcoplasmic calcium release upon stimulation, the non-linear active state dynamics, the non-linear dynamics of motor unit recruitment, and the highly non-linear contraction dynamics (i.e. force-velocity and length-tension relationships).

Environmental constraints have also been included in the form of aerodynamic forces,[63] contact surfaces[64] and apparatus.[65] An important addition to the literature has been the mathematical model for the control of interceptive actions introduced by Beek et al.[66] Previously, Beek and Beek,[67] acknowledging the work of Newell,[6] argued that: "In many instances, the first requirements for successful actions are not exclusively in a sufficiently large delivery of either force or energy, but also, and foremost, in an optimal guidance of force and/or energy on the basis of perceptual information." However, attempts to mathematically model the

integration of perceptual information and movement have been scarce and they have generally been limited to 'short route' models that do not address how perception-action patterns might be constrained by the dynamical properties of the neuromusculoskeletal system. Beek et al.[66] reportedly overcame the phenomenology of existing 'short route' models by developing a 'long route' model where the dynamics of the sensory, neural and musculoskeletal subsystems were integrated to reproduce interceptive actions (figure 5). Their model consisted of neural network architecture for the online generation of motor outflow commands, based on time-to-contact information and information about the relative position and velocities of hand and ball. Beek et al.[66] showed their mathematical model to be consistent with both behavioural and neurophysiological data.

Although not included in the actual mathematical model of the neuromusculoskeletal system, task constraints have been included during the simulation process, generally in the form of an optimality criterion or a specific cost function that must be maximized or minimized. These objective measures describe either the task goal or an aspect of performance that is strongly related to the task goal. Whereas other optimality criteria or cost functions, such as smoothness, accuracy, speed, minimum fatigue and minimum sense of effort have been used,[68-70] energy consumption or, more precisely, energetic efficiency, has typically been the chief optimality criterion or cost function in the biomechanical modelling of human motion.[71] While the optimality criterion or cost function can be viewed as the main, overarching task constraint, there are other nested task constraints that need to be incorporated. However, one of the problems with contemporary simulation approaches is that they are inflexible and generally rely on only a single optimality criterion or cost function for each simulation. If the simulation of human movement is unable to take into account a wider range of interacting task constraints, it runs the risk of becoming merely an academic exercise with limited practical relevance to performance in dynamic environments.

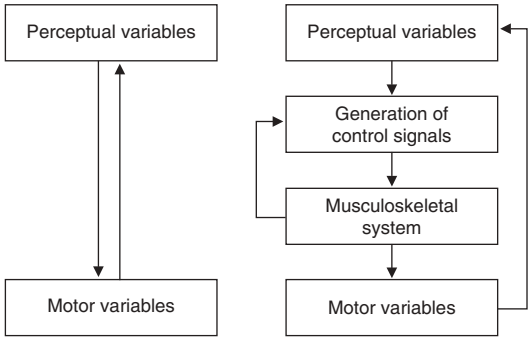

Fig. 5. 'Short route' and 'long route' models of the control of interceptive actions (reproduced from Beek et al.,[66] with permission from The Royal Society).

Another task constraint that has featured in the modelling literature has been that of consistency of technique or, more specifically, robustness to perturbation. For example, Wilson et al.[60] undertook an optimization of a running jump for height that, along with angular momentum at take-off and joint range of motion, included robustness to perturbation of activation timings as a constraint on performance. It was shown that the optimization that included all three of these constraints produced a simulated jump performance (1.99 m) that was similar to a high jumping performance (2.01 m) recorded from the athlete. However, when the robustness to perturbation constraint was removed, the simulated jump performance increased to 2.14 m, suggesting that stability of performance is an important consideration even in motor activities that require maximal effort. Wilson et al.[60] acknowledged the work of Newell,[6,51] recognizing the ubiquity and interplay of a multitude of constraints in the self-optimization of emergent patterns of coordination and control across the lifespan.

In describing performance robustness, along with global and local optima, Yeadon[72] invoked a landscape metaphor analogous to the 'attractor' landscape that features prominently in the dynamical systems literature on motor control, learning and development.[73] Yeadon[72] stated that: ''The search for an optimum can be likened to the search for the highest mountain peak in a given terrain: an optimization routine may find a

local rather than a global optimum – the top of a foothill rather than the summit of the highest mountain. On the other hand, the routine may be successful in finding the top of a singular pinnacle that stands on a narrow base high above the surrounding terrain. Even if this is the global optimum it is a summit that should not be attempted, since any small location error will land on a low terrain. In other words, if there is an optimum technique in javelin that is surrounded by poor performances, it is a poor strategy that strives for this in the distant hope that everything will come right on one of the attempts. The likelihood is that all performances will be poor. A better strategy may be to find a high hilltop with a large plateau so that points even some distance away are high. There is much to be said for consistency when competing.''

3.1 Identifying Key Constraints: An Opportunity for Interdisciplinary Research

Having established the need to include a wider range of constraints into mathematical models of the neuromusculoskeletal system for the purpose of identifying the complete optimal movement solution for a given motor activity, a key question is: which constraints are most influential in shaping and guiding patterns of coordination and control?

Although, as we have discussed, it is the confluence of organismic, environmental and task constraints that ultimately dictates the optimal pattern of coordination and control for specific individuals, clearly some constraints will be more influential than others depending on the motor activity being performed and the specific requirements of the performance or rehabilitation context. To establish the relative impact of different constraints, an experimental research strategy is required that systematically manipulates to their extremes, either singularly or in combination, a broad range of organismic, environmental and task constraints, in a variety of different motor activities.[6,22,45] Such an approach is necessary because constraints on neurobiological systems are not always identifiable *a priori* nor are they particularly amenable to theoretical analysis. Furthermore, it is important to manipulate constraints through their entire range because small-scale changes in a particular constraint can lead to large-scale changes in patterns of coordination and control.[74]

Once constraint manipulations have been undertaken, it is necessary to monitor qualitative and quantitative changes in patterns of coordination and control. McGinnis and Newell[75] outlined a framework based on topological dynamics that uses biomechanical measurements – normally, but not limited to, kinematic time series data[47] – and control spaces for mapping movement to constraints. Each control space frame of reference (i.e. configuration space, event space, state space and state-time space) describes different spatio-temporal properties of movement and provides a useful insight into the restrictive nature of the constraints impinging on the system. The value of this topological approach is that it describes both movement and the imposing constraints in common terms – an issue that has been longstanding in the motor learning and control literature.[74] This framework also provides yet another opportunity for biomechanists and motor control to integrate their respective skills in genuine interdisciplinary collaborations (see Davids et al.[76] and Glazier et al.[77,78] for similar recommendations).

4. Conclusions and Implications for Sports Medicine

In this article, we have discussed the relative merits of simple and complex mathematical models of the neuromusculoskeletal system using pertinent examples from the literature. We have suggested that complex mathematical models might be considered superior to simple mathematical models since they enable basic mechanical insights to be made *and* individual-specific optimal movement solutions to be identified. However, to identify a complete optimal movement solution for any given motor skill, at any given instant in time, it is necessary to incorporate more of the essential constraints that collectively shape and guide the neuromusculoskeletal system through its preferred states of

coordination and control. Some constraints are clearly more influential than others so a priority for biomechanists and motor control experts is to identify a hierarchy of nested constraints through the implementation of systematic experimental research programmes. Although the computer technology and methodological procedures are currently not available to implement such a framework, we believe that the constraints model offered by Newell[6] could, in principle, be used by sport and exercise biomechanists working towards the complete optimization of human movement. Adopting such a model of human movement organization involves re-conceptualizing the human neuromusculoskeletal system, not as a linear, deterministic, mechanical system, but rather as a non-linear, stochastic, biological system.

The notion of individualized adaptation to specific constraints has major implications for scientists working in the related fields of ergonomics, robotics, cybernetics, and more pertinently to the field of sports medicine. Traditionally, practitioners in sports medicine, rehabilitation and therapy have been strongly influenced by the 'medical model', in which variations in movement patterns are often viewed as deviations from a hypothetical 'norm' rather than as adaptive behaviours on the part of individuals who are seeking to satisfy the unique range of interacting constraints on them.[79] Viewing physical, perceptual or cognitive differences in a positive or negative light is too judgmental of individual variations in movement solutions. From this viewpoint, a physical, perceptual or cognitive disability should be viewed as a constraint on the structure or function of the individual neuromusculoskeletal system. Indeed, the use of the term 'disability' to refer to an individual's personal constraint, such as a lower-limb amputation, implies a value judgement on the part of the observer, and in the constraints-led approach there are no negative connotations attached to this system state. Unique structural or functional constraints merely describe different states of neurobiological system organization and would be likely to add to the existing variations observed in the diversity of human behaviour.

For example, the misconception of 'common optimal movement patterns' exists in the study of human movement behaviour and has infiltrated the study of neuro-perceptual-motor disorders. Many clinicians have derived a unitary, biologically determined perspective of health and movement behaviour in which variability is viewed as deviation from 'accepted' norms. In medicine, individual variability is often seen as dysfunctional and an index of abnormality. In this traditional medical model, health and performance behaviours are identified as 'problems' for the individual if they deviate from what are perceived as accepted population norms. The idea that current clinical and medical practice does not recognize individual variation enough has begun to emerge in science. For example, West[80] proposed that science and medicine has tended to overemphasize the significance of an average value in observations of phenomena related to individual health. The science of complexity has questioned traditional assumptions in physiology, supported by innovative thinking in fractal physiology, which pre-supposes that understanding variability provides more insights into an individual's health than does measuring average values in system behavior. According to West,[80] it is important to understand that variability in system behaviours such as heart rate, breathing and walking are much more susceptible to the early influence of disease than are averages. The over-use of central tendency and dispersion statistics in medicine has been implicated in this perspective.[80]

An alternative view prompted by dynamical systems theory is that variability in movement behaviour may be viewed as individual adaptations to unique structural or functional constraints. Variability has a functional role in helping individuals adapt to ever-changing constraints imposed on them by environmental, anatomical and physiological changes due to performance, disease, injury and aging.[81] An implication of this view for sports clinicians is that movement behaviours exist on a spectrum characterizing the boundaries of naturally occurring variability. From this standpoint, the terms 'impaired' and 'elite', used in relation to performance

in sport and exercise, need to be understood as relative and interpreted in relation to the constraints on each individual because the precise location of a neuromusculoskeletal system in the performance spectrum emerges from the multitude of constraints acting upon it at specific points during the lifespan. Since the constraints on each individual are many and unique, it follows that movement solutions will differ within and between individuals in order to optimize functionality. As Latash and Anson[82] noted, the ''phenomena of variability of voluntary movements by themselves indicate that 'correct' peripheral motor patterns may form a rather wide spectrum''. This fundamental insight applies to healthy individuals across the lifespan as well as those with injuries, diseases and perceptual-motor disorders. In line with the arguments of Latash and Anson,[82] it is clear that adaptations to constraints should not necessarily be perceived as pathological since motor patterns may be optimal for the conditions affecting an individual's motor system at any point in time. Motor patterns in individuals with cognitive, perceptual and motor deficits have been labelled by some sports clinicians as 'abnormal' compared with 'common optimal motor patterns' idealized in a 'medical model' approach. However, we have argued that they may be better viewed as a functional and emergent response under the confluence of constraints that each individual needs to satisfy in different performance environments. Therefore, treatment interventions in sports medicine should not be directed towards the achievement and maintenance of an 'ideal' motor pattern during therapy or rehabilitation (a sort of 'one-size-fits-all' approach). The overarching aim of sports clinicians should be to help individuals satisfy the multitude of unique constraints acting on them, improving their functionality in the performance environment.

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Accumulated versus Continuous Exercise for Health Benefit

A Review of Empirical Studies

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Abstract

Current physical activity guidelines endorse the notion that the recommended amount of daily physical activity can be accumulated in short bouts performed over the course of a day. Although intuitively appealing, the evidence for the efficacy of accumulated exercise is not plentiful. The purpose of this review was to compare the effects of similar amounts of exercise performed in either one continuous or two or more accumulated bouts on a range of health outcomes.

Sixteen studies met the selection criteria for inclusion in the review, in which at least one outcome known to affect health was measured before and after continuous and accumulated exercise training interventions. Where improvements in cardiovascular fitness were noted, most studies reported no difference in the alterations between accumulated and continuous patterns of exercise. In the few studies where a normalization of blood pressure was observed from baseline to post-intervention, there appear to be no differences between accumulated and continuous exercise in the magnitude of this effect. For other health outcomes such as adiposity, blood lipids and psychological well-being, there is insufficient evidence to determine whether accumulated exercise is as effective as the more traditional continuous approach.

Seven short-term studies in which at least one health-related outcome was measured during the 0- to 48-hour period after a single continuous bout of exercise and a number of short bouts of equivalent total duration were included in the review. Many of the studies of such short-term effects considered the plasma triglyceride response to a meal following either accumulated short or continuous bouts of exercise. Collectively, these studies suggest that accumulated exercise may be as effective at reducing postprandial lipaemia. Further research is required to determine if even shorter bouts of accumulated exercise (<10 minutes) confer a health benefit and whether an accumulated approach to physical activity increases adherence among the sedentary population at whom this pattern of exercise is targeted.

Since 1995, physical activity guidelines have embraced the notion that for gaining health benefits, exercise or physical activity may be accumulated in bouts spread over the course of the day.[1] Given that a lack of time is a frequently cited barrier to physical activity,[2] a recommendation that allows individuals to perform short bouts of activity throughout the day rather than having to put aside a continuous time-slot in a busy schedule, is intuitively appealing. Theoretically, such a recommendation should make it easier for individuals to adhere to recommended amounts of physical activity. At the time of publication of the 1995 guidelines,[1] only two empirical studies had compared the effects of performing short bouts of exercise over the course of a day with completing a similar amount of activity in one continuous bout.[3,4] Although both studies indicated that accumulating short bouts of exercise was effective in improving fitness and favourably altering a number of health outcomes, the endorsement of accumulated exercise was advanced primarily on the basis of epidemiological evidence suggesting a dose-response relationship between physical activity volume and health. A review by Hardman[5] identified a further three studies that made direct comparisons of equal amounts of physical activity performed in a single continuous bout or accumulated in shorter bouts over the course of the day.[6-8] Since that review, several authors have conducted intervention studies to investigate the effects of bouts of physical activity accumulated over the course of a day.

It is well established that at least some of the health benefits of regular physical activity are likely to be due, in part, to the short-term changes that occur in the hours and days following a bout of activity.[9] Benefits such as reduced resting blood pressure, improved glucose control, favourable alterations in lipids[10] and enhancement of mood[11] have all been demonstrated in the post-exercise period. Whether such immediate beneficial effects of exercise are retained when a single continuous bout is split into shorter bouts accumulated over the course of the day has been the focus of number of recent studies.

The purpose of this article is to review the empirical studies that have compared the effects of physical activity on a range of health outcomes for activity performed in a single continuous bout or short accumulated bouts of the same total duration. The article includes two categories of studies: (i) studies that compared the long-term response to training using accumulated versus continuous exercise; and (ii) short-term studies that compared the effects of accumulated and continuous exercise during the 48-hour period following the last exercise bout.

1. Methods

1.1 Literature Search Strategy

A search was made through MEDLINE using the following terms: 'accumulated AND exercise', 'accumulated AND physical activity', 'short bouts AND exercise', 'short bouts AND physical activity'. The search incorporated any article published in English that included any of these combinations in the title, keywords or abstract. This search was cross-checked and supplemented using the authors' personal libraries. Over 340 articles were identified through this search method. In several instances, the publications identified referred to short intermittent bouts of repeated very high-intensity activity performed with short recovery within a single training session. This type of intermittent exercise is normally undertaken by individuals training for multiple sprint sports and was therefore outside the scope of this review. The search also identified several studies of 'lifestyle physical activity' where individuals are encouraged to accumulate additional physical activity over the course of the day by incorporating planned or opportunistic activities of daily living into their routine. These studies were not designed to provide a direct comparison between accumulated and continuous exercise. Moreover, this 'lifestyle' approach has previously been reviewed and therefore is not included in this review.[12-14]

1.2 Selection Criteria

The following criteria were used to select studies included in the review:

1. Studies were designed to compare the effects of 'equal' amounts of exercise performed in either one continuous or two or more accumulated bouts.
2. The duration of physical activity bouts was prescribed by the researcher with no opportunity for the participant to self-select bout duration.
3. The continuous or accumulated exercise was the primary intervention or where an additional intervention (e.g. weight loss programme) was included, this was applied equally to both exercise conditions.

In addition, studies selected had to meet one of the following criteria depending on which category of study was being considered:

4. For the training studies, at least one outcome known to affect health was measured before and after continuous and accumulated exercise interventions of at least 4 weeks' duration.
5. For the short-term studies, at least one health-related outcome was measured during the 0- to 48-hour period after a single continuous bout of exercise and a number of short bouts of equivalent total duration.

These criteria were independently applied to all studies by two authors (MM and EM), with any discrepancies resolved by consensus.

2. Findings

2.1 Characteristics of the Training Studies

Using the search strategy and inclusion criteria described above, 16 training studies were identified. Walking was the predominant mode of exercise in 12 studies with two studies using jogging or running,[3,4] and one study employing aerobic dance as the exercise modality.[15] Collectively, these studies involved 836 subjects, predominantly females (n = 630). Although the physical activity status of subjects varied between studies, no subjects were meeting the current physical activity guidelines at the start of the study. The majority of interventions were short, ranging in duration from 4 to 20 weeks and involved total daily exercise durations of between 20 and 40 minutes on 3–5 days per week. The intensity of exercise prescribed in the studies ranged from 50% to 80% of maximum oxygen uptake ($\dot{V}O_{2max}$). For subjects assigned to the accumulated exercise groups, this was prescribed in 2, 3 or 4 bouts of between 10 and 15 minutes, with half of the studies (n = 8) requiring participants to separate the accumulated exercise bouts by at least 2 hours.[4,7,15-20] More than half of the studies (n = 9) employed a non-exercise control group.[7,15-17,19,21-23] Table I provides a summary of the design and findings of the intervention studies included in the review.

2.2 Long-Term Training Responses

2.2.1 Fitness

Most of the selected intervention studies reported measures of fitness before and after the exercise interventions. With the exception of two studies,[15,29] all others reported significant improvements in at least one measure of cardiovascular fitness following training.

Only three studies[4,18,27] reported a significant difference between accumulated and continuous exercise groups for improvements in $\dot{V}O_{2max}$. De Busk and colleagues[4] noted greater improvements in $\dot{V}O_{2max}$ in the continuous group compared with the control group. Murphy and co-workers[27] reported greater increase in $\dot{V}O_{2max}$ in the accumulated group compared with the continuous group. Quinn et al.[18] also noted an increase in $\dot{V}O_{2max}$ in the accumulated group with no change in $\dot{V}O_{2max}$ among the continuous exercisers.

2.2.2 Body Composition

Body Mass

Of the nine studies that reported body mass measurements before and after the exercise intervention, five noted significant reductions in body mass following accumulated or continuous exercise training compared with controls.[7,16,22,24,25] However, three of these studies included caloric restriction as part of the intervention.[22,24,25] Four of the studies reporting weight loss noted similar reductions following accumulated and continuous exercise training,[16,22,24,25] while Murphy and Hardman[7] noted a significant

Table I. Training studies comparing the effects of accumulated and continuous exercise on a range of health outcomes

Study	Subjects; age	Intervention	Patterns	Control	Duration	Outcome measures	Changes over time
Ebisu[3]	53 M; 21 y	3- to 6 mile (4.8–9.6 km) run 80% HR_{max} 3 d/wk	a. 1 bout b. 2 bouts c. 3 bouts	Yes	10 wk	$\dot{V}O_{2max}$ 1.5-mile (2.4-km) run time HDL TC/TG/LDL/VLDL	a. +6.9%[a] b. +9.8%[a] c. +8.4%[a] (nsd between groups) a. −10.2%[a] b. −10.8%[a] c. −12.7%[a] (nsd between groups) a. +3.4% b. +4.7% c. +9.6%[a] nsd
DeBusk et al.[4]	36 M; 40–60 y	Jogging 65–75% HR_{peak} 5 d/wk	a. 1 × 30 min b. 3 × 10 min	No	8 wk	$\dot{V}O_2$ Exercise test duration HR during submax test Body mass	a. +13.9%[a] b. +7.6%[a] (a sig > b) a. +12.4%[a] b. 10.4%[a] a. −6.6%[a] b. −6.3%[a] nsd
Jakicic et al.[24]	56 F OW; 25–50 y	20–40 min walking 70% HRR 5 d/wk Caloric restriction	a. 1 bout b. multiple 10-min bouts	No	20 wk	Body mass Time to 80% HRR $\dot{V}O_2$ at HR_{peak} $\dot{V}O_2$ at HR = 110 bpm $\dot{V}O_2$ at HR = 125 bpm SBP DBP Resting HR	a. −7.2%[a] b. −9.7%[a] a. +7.2%[a] b. +7.8%[a] (nsd between groups) a.+ 5.6%[a] b. +5.5%[a] (nsd between groups) a. +8.9%[a] b. +21.2%[a] (nsd between groups) a. +7.6%[a] b. +14.5%[a] (nsd between groups) a. −3.3%[a] b. −2.3%[a] (nsd between groups) a. −5.1%[a] b. −6.5%[a] (nsd between group) a. −6.2%[a] b. −6.7%[a] (nsd between groups)
Murphy and Hardman[7]	47 F; 44.4 ± 6.2 y (mean ± SD)	Walking 70–80% HR_{max} 5 d/wk	a. 1 × 30 min b. 3 × 10 min	Yes	10 wk	$\dot{V}O_{2max}$ $\dot{V}O_2$ at 2 mmol/L blood La Body mass Skinfolds Waist circumference SBP	a. +8.5%[a] b. +8.3%[a] (nsd between groups) a. +18.1%[a] b. +13.8%[a] (nsd between groups) a. −1.3% b. −2.6%[a] (nsd between groups) a. −4.2%[a] b. −4.6%[a] (nsd between groups) a. −2.3% b. −3.8%[a] (nsd between groups) nsd
Woolf-May et al.[19]	49 (21 F); 40–71 y	20–40 min walking 73% HR_{max} Progressive from 60 to 200 min/wk	a. 1 bout b. 10–15 min bouts	Yes	18 wk	HR_{peak} during submax test Recovery HR Blood lactate TC/LDL/HDL Apolipoproteins	a. −4.3%[a] b. −4.9%[a] (nsd between groups) nsd nsd nsd increase in control group only
Jakicic et al.[25]	115 F OW; 36.7 ± 5.6 y (mean ± SD)	20–40 min exercise 5 d/wk Weight-loss programme	a. 1 × 20–40 min b. 2–4 × 10 min c. 2–4 × 10 min + treadmill	No	18 mo	Body mass Body fat percentage Waist : hip Bone mineral content Predicted $\dot{V}O_2$ peak	a. −11.4%[a] b. −10.3%[a] c. −11.7%[a] (nsd between groups) a. −6.5%[a] b. −3.6%[a] c. −9.4%[a] (c sig >b) nsd nsd a. +9.9%[a] b. +6.3%[a] c. +11.5%[a] (nsd between groups)
Coleman et al.[26]	21 (18 F); 18–55 y	30 min walking 68–70% HR_{max} 6 d/wk	a. 1 × 30 min b. 3 × 10 min[b]	No	16 wk	Predicted $\dot{V}O_{2max}$ Body fat percentage Body mass SBP DBP	a. +7.8%[a] b. +6.4%[a] (nsd between groups) nsd nsd a. −4.7%[a] b. −2.1%[a] (nsd between groups) nsd

Continued next page

Table I. Contd

Study	Subjects; age	Intervention	Patterns	Control	Duration	Outcome measures	Changes over time
Woolf-May et al.[20]	56 (37 F) 50.1 ± 6.3 y (mean ± SD)	60–200 min walking/wk 70–75% $\dot{V}O_{2max}$	a. 20–40 min bout b. 10–15 min bouts c. 5–10 min bouts	Yes	18 wk	Predicted $\dot{V}O_{2max}$ HR_{peak} during submax test Lactate during submax test LDL TC/TG Apolipoprotein A-II	nsd nsd a. –45.5%[a] b. –33.3%[a] c. –46.2% (nsd between groups) a. –5.9%[a] b. –8.3%[a] c. –2.6% nsd a. –10.9%[a] b. –4.4%[a] c. +2.5%
Schmidt et al.[22]	38F OW; 19–21 y	30 min walking 75% HRR 3–5 d/wk Caloric restriction	a. 1 × 30 min b. 2 × 15 min c. 3 × 10 min	Yes	12 wk	$\dot{V}O_{2max}$ Skinfolds Body mass BMI Sum of circumferences	sig increase all groups (nsd between groups) sig decrease all groups (nsd between groups) a. –3.3%[a] b. –3.5%[a] c. –5.0%[a] (nsd between groups) a. –3.5%[a] b. –3.6%[a] c. –4.9%[a] (nsd between groups) sig decrease in all groups (nsd between groups)
Murphy et al.[27]	21 (14 F); 44.5 ± 6.1 y (mean ± SD)	30 min walking 70–80% HR_{max} 5 d/wk	a. 1 × 30 min b. 3 × 10 min	No	2 × 6 wk crossover	Predicted $\dot{V}O_{2max}$ Waist circumference Hip circumference Sum of 4 skinfolds Body mass SBP DBP TC HDL TG Mood State POMS Self-efficacy Barriers to exercise	a. +3.8%[a] b. +14.2%[a] (b sig > a) a. –1.2%[a] b. –0.3%[a] (nsd between groups) a. –0.6%[a] b. –0.9%[a] (nsd between groups) a. –3.3%[a] b. –1.6%[a] (nsd between groups) nsd nsd a. –1.8%[a] b. –1.7%[a] (nsd between groups) a. –7.1%[a] b. –5.1%[a] (nsd between groups) a. +9.2%[a] b. +5.4%[a] (nsd between groups) a. –10.8%[a] b. –9.3%[a] (nsd between groups) Similar decreases in tension/anxiety in both groups nsd for walking, b. increased efficacy for other activities[a] a. decreased health barrier[a] b. decreased effort barrier[a]
Asikainen[28]	130 F; 48–63 y	Walking 1500 kcal/wk 65% $\dot{V}O_{2max}$ 5 d/wk	a. 1 bout b. 2 bouts	Yes	15 wk	$\dot{V}O_{2max}$ Submax HR response Body mass Body fat percentage	a. +12.5%[a] b. +12.7%[a] (nsd between groups) nsd a. –1.8%[a] b. –1.8%[a] (nsd between groups) a. –6.8%[a] b. –5.6%[a] (nsd between groups)
Schachter et al.[15]	143 F FM; 20–55 y	Progressive to 30 min 'aerobic dance' 65–75% HRR 3–5 d/wk	a. 1 bout b. 2 bouts	Yes	16 wk	Peak $\dot{V}O_2$ Treadmill test duration Physical function Symptoms/severity/pain Self-efficacy Well-being	nsd nsd nsd b. decreased disease severity[a] b. increased[a] nsd

Continued next page

Table I. Contd

Study	Subjects; age	Intervention	Patterns	Control	Duration	Outcome measures	Changes over time
Murtagh et al.[21]	48 (31 F); 45.7 ± 9.4 (mean ± SD)	20 min walking ~73% HR_{max} 3 d/wk	a. 1 × 20 min b. 2 × 10 min	Yes	12 wk	$\dot{V}O_2$ during submax test HR during submax test RPE during submax test Body mass Body fat percentage Waist and hip circumference SBP DBP TC/TG/HDL/LDL	nsd decrease in both groups (a sig > b) a. −15%[a] b. −11.2%[a] (nsd between groups) nsd nsd nsd nsd nsd nsd
Osei-Tutu and Campagna[17]	40 (21 F); 20–40 y	30 min walking 60–79% HR_{max}	a. 1 × 30 min b. 3 × 10 min	Yes	8 wk	$\dot{V}O_{2max}$ Body fat percentage POMS	a. +6.7%[a] b. +7.2% (nsd between groups) a. −6.7%[a] (a sig > b) Increase in vigour activity[a] and decrease in total mood disturbance[a] in both groups a. decreased tension-anxiety[a] and decreased depression-dejection[a]
Quinn et al.[18]	37 (20 F); 48.8 ± 9.0 y (mean ± SD)	30 min aerobic exercise (various modalities) 70–80% HRR 4 d/wk	a. 1 × 30 min b. 2 × 15 min	No	2 × 12 wk crossover	$\dot{V}O_{2max}$ HR during submax test $\dot{V}O_2$ during submax test Treadmill test duration Treadmill test SBP Treadmill test DBP HDL TC/LDL/TG	a. +4.5% b. +8.7%[a] a. −4.7% b. −9.0%[a] a. −4.1% b. −14.7%[a] a. +12.0%[a] b. +21.2%[a] a. −9.3% b. −11.6%[a] a. −13.7%[a] b. −8.4%[a] a. nsd b. +6.4%[a] nsd
Altena et al.[29]	18 (11 F); 18–45 y	30 min jogging 60% $\dot{V}O_{2max}$ 5 d/wk	a. 1 × 30 min b. 3 × 10 min	No	4 wk	$\dot{V}O_{2max}$ Waist and hip circumference Sum of 3 skinfolds TC HDL IDL TG AUC TG peak	nsd nsd nsd a. −4.7%[a] b. −11.3%[a] (nsd between groups) nsd nsd nsd nsd

a Significant change from pre- to post-intervention.

b Study also included a third group who could self-select bout duration (these subjects are not included in the table).

AUC = area under the concentration-time curve; **BMI** = body mass index; **bpm** = beats/min; **DBP** = diastolic blood pressure; **F** = female; **FM** = subjects with fibromyalgia; **HDL** = high density lipoprotein cholesterol; **HR** = heart rate; $\mathbf{HR_{max}}$ = maximum heart rate; $\mathbf{HR_{peak}}$ = peak heart rate; **HRR** = heart rate reserve; **LDL** = low density lipoprotein cholesterol; **M** = male; **nsd** = no significant difference; **OW** = overweight; **POMS** = profile of mood states; **RPE** = rate of perceived exertion; **SBP** = systolic blood pressure; **sig** = significantly; **sig** >= significantly greater than; **TC** = total cholesterol; **TG** = triacylglycerol; **VLDL** = very low density lipoprotein cholesterol; $\mathbf{\dot{V}O_2}$ = oxygen uptake; $\mathbf{\dot{V}O_{2max}}$ = maximal oxygen uptake.

weight loss from pre- to post-intervention in the accumulated group compared with controls, but not the continuous exercise group.

Adiposity

Six studies reported percentage body fat.[3,17,21,25,26,28] Three of these[3,21,26] reported no changes in any exercise group. Another two studies[25,28] reported significantly reduced body fat in all exercise groups, but no significant differences between the continuous and accumulated conditions. Osei-Tutu and Campagna[17] noted a decrease in percentage body fat in the continuous exercise group only.

Four authors recorded sum of skinfolds at pre- and post-intervention.[7,22,27,29] Three of these studies[7,22,27] noted a reduction in skinfold thickness in all exercise groups; however, there was no significant difference between groups. Altena and colleagues[29] noted no alteration in skinfolds following either exercise pattern.

Waist and Hip Circumference

Five studies reported waist and hip circumferences before and after the exercise intervention. Murtagh et al.[21] and Quinn et al.[18] found no changes in these measures. A significant reduction in waist circumference was recorded by the accumulated exercise group, but not the continuous group in the study by Murphy and Hardman,[7] while Murphy et al.[27] noted significant reductions in waist and hip circumferences for both exercise conditions. Similarly, Jakicic and colleagues[25] recorded changes over time for waist girth and waist-to-hip ratio. No study noted a significant difference for changes in waist and hip circumferences between continuous and accumulated exercise interventions.

Blood Pressure

Six of the selected studies reported resting systolic and diastolic blood pressure before and after the exercise interventions.[7,18,24,26,27,30] Two of these studies found a significant decrease in both systolic and diastolic blood pressure following training with no differences between the two patterns of exercise.[24,26]

Quinn and colleagues[18] also measured ambulatory blood pressure during treadmill walking before and after training. During exercise, diastolic blood pressure decreased following 12 weeks of accumulated or continuous exercise and systolic blood pressure, during exercise, decreased following the accumulated exercise training.

Blood Lipids

Seven studies included assessment of fasting blood lipid profiles before and after the intervention.[3,18-21,27,29] Three studies reported an increase in high-density lipoprotein (HDL) cholesterol.[3,18,27] One found an increase following accumulated[18] exercise training and two found an increase in both exercise groups[3,27] from pre- to post-intervention. There were no consistent observations of alterations in total cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides or other lipid parameters measured in the studies. One study that compared the postprandial response to a high-fat meal before and after 4 weeks of training found no differences between continuous and accumulated conditions.[29]

2.2.3 Other Health Outcomes

One study compared the effects of accumulated and continuous sessions of low impact aerobics on signs and symptoms of fibromyalgia.[15] Small improvements in self-efficacy and disease severity following both exercise conditions were observed with no difference between the two exercise patterns.

2.2.4 Psychological Outcomes

Three studies reported measures of psychological health before and after training. Murphy and colleagues[27] noted that both accumulated and continuous exercise conditions resulted in similar decreases in tension and anxiety. However, Osei-Tutu and Campagna[17] found a positive effect on mood in the continuous exercise group only, although they note that a definite trend in the direction of positive mood was evident for the accumulated exercise group. Schachter and co-workers[15] observed that improvements in psychological well-being were

evident in the continuous exercise group at mid-test, but not after 16 weeks of training.

2.3 Characteristics of Short-Term Studies

Seven studies meeting the selection criteria compared the effects of continuous and accumulated exercise in the 48 hours following physical activity.[31-37] Study designs included measurement of lipids, insulin, glucose, substrate oxidation, metabolic rate and/or enzyme activity at baseline. Several studies considered responses to a standardized high-fat meal,[31,34] mixed meals[36,37] and an oral glucose load[32] following accumulated and continuous exercise bouts. Two studies considered fasting lipid responses at baseline, 24 and 48 hours following the two patterns of exercise.[33,35] Six studies employed a no-exercise control condition.[31,32,34-37] Subject numbers were consistently low in these studies (n = 10–18) with a total of 96 participants (56 males, 40 females). Exercise modality consisted of running or walking with total duration varying from 30 to 90 minutes. Table II summarizes the design and findings of the short-term studies included in this review.

2.4 Short-Term Responses

2.4.1 Fasting Blood Lipids

Fasting blood lipids in the 48-hour period following accumulated and continuous exercise bouts were measured in five studies.[31,33-36] No differences were observed in triglyceride or total cholesterol levels between exercise and control or between accumulated and continuous exercise conditions. Two studies reported an increase in fasting HDL cholesterol in the 48 hours after exercise compared with baseline.[33,35] Campbell et al.[33] reported no difference between accumulated and continuous conditions, whereas Mestek et al.[35] report greater increases in HDL cholesterol following accumulated compared with continuous exercise of equivalent total energy expenditure. Only one study determined the activity of enzymes involved in lipid metabolism in response to different patterns of exercise. Campbell and colleagues[33] noted an increase in the activity of the HDL cholesterol-forming enzyme lecithin-cholesterol acyl transferase (LCATa) in the 24–48 hours following continuous and accumulated exercise.

2.4.2 Postprandial Lipaemia

All four studies that compared the effects of exercise on the lipid responses to high-fat or mixed meal ingestion reported a decrease in postprandial triglycerides compared with controls.[31,34,36,37] Three studies reported no difference in the pattern of this response between accumulated and continuous exercise conditions[34,36,37] with one study noting a lower triglyceride response compared to controls following the accumulated, but not the continuous exercise.[31]

2.4.3 Fasting Glucose and Insulin

Three studies determined fasting glucose and insulin levels the morning after accumulated and continuous exercise compared with controls.[32,34,36] Two of these studies noted no differences in fasting glucose between exercise and control conditions nor between the accumulated and continuous exercise trials.[32,34] The study by Miyashita and colleagues[36] showed lower fasting glucose on the morning following accumulated exercise compared with controls with no difference between continuous and control trials. None of the three studies noted any alterations in fasting insulin following exercise trials.

3. Discussion

The notion that the recommended daily amounts of physical activity required for good health can be accumulated through several separate episodes of activity interspersed throughout the day is persistent in recent public health guidelines.[38,39] Given the multiple demands upon individuals' time, the flexibility afforded by an accumulated approach may help to increase the proportion of the population meeting physical activity recommendations. However, the empirical evidence to support splitting a continuous bout of activity into several shorter bouts is not extensive or unequivocal. If accumulated

Table II. Studies comparing the acute effects (0–48 h) of accumulated and continuous exercise on a range of health outcomes

Study	n	Interventions	Patterns	Control	Timing of measures	Outcome measure	Reported change
Gill et al.[34]	18 M 30.6 ± 9.0 y (mean ± SD)	90-min run at 60% $\dot{V}O_{2max}$	a. 90 min continuous b. 3 × 30 min run >3 h between bouts	Yes	12 h post-exercise 2, 3, 6 h post-high-fat meal	Triglycerides	No difference in fasting levels pp response 18.1% lower in continuous 17.7% lower in accumulated compared with control (nsd between exercise pattern)
						Non-esterified fatty acids	Fasting levels 30.3 % higher in continuous compared with control No difference in pp response
						Insulin	No difference in fasting levels pp response 22.4% lower in accumulated compared to control
						Glucose	No difference in fasting levels No difference in pp response
Murphy et al.[37]	10 (7 F) 34–66 y	30-min walk at 60% $\dot{V}O_{2max}$	a. 30-min continuous b. 3 × 10 min >3 h between bouts	Yes	Baseline 1, 2, 3 h After breakfast, lunch and dinner	Metabolic rate	No difference in fasting or pp response
						Triglycerides	No difference in fasting levels pp response lower in both exercise patterns compared to control (nsd between exercise pattern)
						Insulin	No difference in fasting or pp response
						Non-esterified fatty acids	No difference in fasting or pp response
						Glucose	No difference in fasting or pp response
Altena et al.[31]	18 (11 F) 25 ± 1.8 y	30-min run at 60% $\dot{V}O^{2max}$	a. 30-min continuous b. 3 × 10 min 20 min between bouts	Yes	Baseline and 2, 4, 6, 8 h After high-fat meal	Triglycerides	pp response 26.9% lower in accumulated pattern compared with control
						Total cholesterol	No difference in fasting or pp response
						HDL	No difference fasting or pp response

Continued next page

Table II. Contd

Study	n	Interventions	Patterns	Control	Timing of measures	Outcome measure	Reported change
Baynard et al.[32]	15 F[a] 42–60 y	30-min walk at 60–65% $\dot{V}O_{2max}$	a. 30 min continuous b. 3×10 min >3.5 h between bouts	Yes	Baseline + every 30 min for 4 h	Glucose	No difference
					After 75 g oral glucose load	Insulin	No difference
Miyashita et al.[36]	10 M 21–32 y	30-min run At 70% $\dot{V}O_{2max}$	a. 30 min continuous b. 10×3 min 30 min between bouts	Yes	Baseline and 1, 2 and 3 h	Triglycerides	pp response 24 % lower in continuous 22% lower in accumulated compared with control (nsd between exercise pattern)
					After breakfast and lunch	Insulin	pp response lower in accumulated compared to control
						Glucose	No difference
Mestek et al.[35]	9 M 25±4 y (mean±SD)	500 kcal walk/jog at 70% $\dot{V}O_{2max}$	a. 500 kcal in single bout b. 3 bouts of 167 kcal 4 h between bouts	No	24 h before	Triglycerides	No difference
					Baseline, 24 and 48 h post-exercise	Total cholesterol	No difference 10.2% increase at 24 h post and 14.3% increase at 48 h post-accumulated
						HDL	4% increase at 48 h post-continuous accumulated significantly higher than continuous at 48 h post-exercise
Campbell et al.[33]	16 M 22±2 y (mean±SD)	450 kcal Treadmill running at 65% $\dot{V}O_{2max}$	a. 450 kcal in single run b. 2×225 kcal >4 h between bouts c. 3×150 kcal >4 h between bouts	No	Baseline, Post-exercise: 0, 24 and 48 h	Triglycerides Cholesterol Enzymes	No difference Increase in HDL subfraction cholesterol 13–15% in all 3 exercise trials (nsd between exercise pattern) Increase in lecithin cholesterol acyl transferase activity for all three exercise trials (nsd between exercise groups)

a Nine subjects with type 2 diabetes mellitus.

F = females; **HDL** = high-density lipoprotein cholesterol; **M** = males; **nsd** = no significant difference; **pp** = postprandial; $\dot{V}O_2$ = oxygen consumption; $\dot{V}O_{2max}$ = maximum oxygen consumption.

exercise is to be recommended at a population level, there are two major issues to be considered. The first of these is whether or not continuous and intermittent exercise of comparable doses will produce similar health benefits. A second important issue is to determine if the flexibility and convenience of splitting an exercise dose into several bouts increases adoption and maintenance of regular physical activity for previously sedentary persons. This article focuses on the first of these questions, and considers both short- and long-term effects of the two approaches to on a range of fitness and health parameters.

3.1 Long-Term Effects

Cardiovascular fitness is recognized as an independent risk factor for cardiovascular disease.[40] The majority of studies included in this article demonstrate increases in fitness following exercise interventions ranging in volume from as little as 60 minutes per week to those that meet or surpass current physical activity guidelines. The magnitude of the increase in $\dot{V}O_{2max}$ ranged from 3.8%[27] to 13.9%.[4] Where no measurable alterations in fitness were noted, this can be attributed to the very short duration of the exercise intervention[29] or the use of cardiovascular fitness measures that did not reflect the mode of activity used in the intervention.[15] Among the studies that identified improvements in fitness following the exercise intervention, the majority of studies noted no difference between accumulated and continuous approaches to exercise in the magnitude of this improvement. Where differential responses were noted, there appears to be no clear pattern in the findings with a few studies suggesting that continuous exercise results in greater fitness enhancement,[4,25] while others report greater improvements using the accumulated approach.[18,21,37] Most studies used multiple indices of cardiovascular fitness including $\dot{V}O_{2max}$, heart rate and blood lactate response to exercise and field fitness tests. In studies that found differences between continuous and accumulated exercise, these were often not consistently observed in all of the fitness measures employed. The available evidence suggests that there is no difference between accumulated and continuous exercise of the same total duration on improvements in cardiovascular fitness.

The majority of the intervention studies included in the review measured body mass, body composition or fat distribution before and after training. Although eight studies noted positive alterations in body composition[4,16-18,22,24,27,28] following training, there is little consistency in the comparisons between the two exercise patterns. Four studies noted similar favourable alterations in body composition in both patterns,[4,22,24,27] whereas three studies noted greater improvements following the accumulated exercise[16,18,24] and one study reported greater improvement following the continuous approach.[17]

Despite the increased energy expenditure, which is likely to have occurred with the exercise intervention, four studies noted no alteration in body composition.[3,21,26,29] Such equivocal findings reflect the difficulty in using body composition measures in exercise intervention studies with free-living individuals. Aside from the weight-loss intervention, which was applied to subjects in three of the training studies,[22,24,25] deliberate alterations in exercise habits may result in conscious or unconscious alterations in energy intake. These alterations may contribute to changes in body composition noted following an exercise intervention. Theoretically, at least, it seems likely that a given quantity and quality of exercise would induce similar changes whether it is performed in single daily bouts or accumulated in shorter bouts of equal total duration. Where increased weight loss has been noted following accumulated exercise, this has been attributed to reduced energy intake, increased total exercise volume or even the small increases in metabolism that occur following each exercise bout; however, none of these explanations have been fully empirically tested.

Normalization of resting blood pressure following exercise training is well documented,[41] although the benefit is not always observed.[42] Only six of the training studies included in this review used resting blood pressure as an outcome measure.[7,18,21,24,26,27] Where reductions in resting systolic or diastolic blood pressure were noted, these tended to be among subjects who

had elevated blood pressure levels at baseline. Although there are insufficient data to allow a comparison of the effects of accumulated and continuous patterns of exercise on blood pressure, all but one of the reviewed studies[18] that found decreases in blood pressure following exercise reported that these reductions were similar following accumulated or continuous patterns of exercise.[24,26,27] The proposed mechanisms for exercise-induced blood pressure reduction include decreased peripheral resistance resulting from increased lumen diameter, decreased vasoconstriction or greater vasodilation.[43] It is perhaps not surprising, therefore, that exercise of a given mode, intensity and total duration produces similar long-term effects irrespective of whether it is accumulated or performed in a continuous bout.

Only seven of the intervention studies in the review considered the effect of continuous and accumulated exercise on fasting blood lipid profiles.[3,18-21,27,29] Collectively, these studies suggest that 60–200 minutes of moderate-intensity exercise expending approximately 300–1000 kcal per week whether performed in a continuous or intermittent manner is insufficient to alter blood lipids in normolipidaemic individuals. This is consistent with suggestions that such favourable blood lipid alterations require a training threshold of 1200–2200 kcal per week.[44] In the studies that noted an alteration in one or more lipid parameters, this may be attributable to pre-intervention lipid profile.[18,20,27]

There is an insufficient number of studies in the review (n = 3) that have compared the effects of the two approaches to exercise on psychological parameters to allow reliable conclusions to be advanced.[15,17,27] Although no alterations in self-efficacy or well-being were noted in a patient population following 16 weeks of accumulated or continuous aerobic exercise,[15] in normal populations, similar improvements in mood state were observed following continuous and accumulated exercise interventions.[17,27] Given the importance of such enhancements in psychological attributes to exercise adherence, this may represent an important area for future comparisons of accumulated and continuous exercise.[11]

The data included in the current review derive from studies where subjects were predominantly middle-aged females with relatively low baseline levels of cardiovascular fitness. Whether our conclusions hold true for males or females with average or above-average fitness levels and indeed other populations, such as ethnic minorities, children and older people, cannot be determined at this stage.

3.2 Short-Term Effects

In addition to the effect of regular exercise on fitness and a range of health outcomes, it is now accepted that a single episode of exercise results in a number of short-term physiological alterations that may benefit health. These short-term effects are attributable to the last bout of exercise. Such last bout effects are thought to contribute to the health-enhancing effects of near daily exercise. The second element to this review focuses upon the small number of studies that have compared the short-term responses to accumulated and continuous exercise. Two of these studies compared the effects of continuous and accumulated exercise on fasting blood lipids 24 and 48 hours after the exercise was completed.[33,35] Both studies report modest increases in HDL cholesterol or a HDL subfraction following exercise, which is consistent with previous reports from continuous exercise studies.[44] Although Mestek and colleagues[35] demonstrate more favourable alterations following accumulated exercise, there is insufficient evidence at this time to support the conclusion that accumulated exercise is superior in this regard.

In recent years, how an individual metabolizes dietary fat has emerged as a risk factor for cardiovascular disease and it has been established that a single exercise session can result in a reduction in postprandial lipaemia.[45] Four of the seven studies included in this review focused on whether splitting the session of exercise into several smaller bouts alters this beneficial effect of exercise.[31,34,36,37] The range of study designs, including duration of exercise, meal content and timing, and post-exercise blood sampling schedule make it difficult to draw sound conclusions.

While three of these studies suggest that splitting exercise into shorter accumulated bouts has no effect on its capacity to alter postprandial lipaemia,[34,36,37] one study suggests that accumulated bouts of exercise may provide greater reductions in plasma triglycerides than a single exercise session.[31]

Collectively, the short-term studies included in this review provide tentative support for the notion that splitting a bout of continuous exercise into shorter bouts accumulated over the course of day does not alter its ability to elicit modest reduction in fasting or postprandial lipaemia. Although a small number of studies have reported greater short-term benefits following accumulated exercise, at this stage such suggestions should be regarded as speculative. There is currently a dearth of research comparing the short-term effects of moderate-intensity exercise performed in a continuous and accumulated pattern.

4. Recommendations for Future Research

Most of the training studies included in this review used accumulated bouts of 10 minutes or longer in duration. In a real-life setting, however, individuals attempting to accumulate ≥30 minutes of physical activity in line with current recommendations may opt for even shorter bouts performed more frequently. Jakicic et al.[24] have shown that subjects attempting to meet a daily 30-minute exercise target selected bouts that were <10 minutes in duration. Furthermore, Miyashita and colleagues[36] observed similar reductions in postprandial lipaemia on the day after subjects performed either one 30-minute run or ten 3-minute runs with 30 minutes between bouts. From a practical research perspective, it may be difficult to investigate the efficacy of very short bouts (<5 minutes) of moderate-intensity exercise in free-living individuals as the boundaries between 'accumulated bouts' of activity and 'lifestyle' physical activities such as walking for personal transport become blurred.[14] However, the use of objective monitoring devices such as accelerometers may make such studies feasible, and it would be useful to know if accumulating the exercise in very short bouts is beneficial.

Accumulating exercise in such very short bouts has, however, been investigated using the vigorous-intensity exercise of stair-climbing. Recent studies have demonstrated that five to eight 2-minute bouts of stair-climbing accumulated over the course of a day conferred health benefits including increases in cardiovascular fitness compared with non-exercising controls.[46-48] Of the short-term studies included in this review, only one compared very short bouts of <10 minutes in duration.[36] The alterations in lipid clearance observed the day after 30 minutes of running or three 10-minute runs are likely to be due to increased activity of key enzymes involved in lipid metabolism and increased energy expenditure.

Given the high levels of inactivity in most developed countries, an important consideration is whether an accumulated exercise is more palatable and easier to maintain. Future studies should examine whether the continuous or accumulated pattern of activity results in increased adoption and maintenance of regular exercise for previously sedentary persons. Such information is crucial to shaping future public health campaigns aimed at the sedentary majority.

A number of limitations of the studies included in this review have been identified, which should be considered in future study design by researchers. Seven of the training studies included in this review did not have a control group limiting the degree to which alterations in health outcomes can be attributed entirely to the exercise intervention.[4,18,24-27,29] The majority of the training studies rely on self-report of exercise bouts. More objective measures of exercise duration, intensity and timing may be warranted to ensure reliable comparisons.

In general, the studies included in this review involved low subject numbers. This probably reflects the practical difficulties encountered by researchers attempting to recruit sedentary participants and encourage them to undertake exercise that is somewhat rigidly prescribed in terms of intensity, duration and bout length. With a few exceptions, the studies included in this review

involved fewer than 60 subjects assigned to two or more intervention groups. Inevitably, therefore, some of these studies may have been insufficiently powered to detect alterations in some of the outcome measures selected.

5. Conclusion

The available evidence suggests that at least for fitness, accumulated and continuous patterns of exercise training of the same total duration confer similar benefits. For the effects of continuous and accumulated training on the other health outcomes identified and the short-term effects of continuous and accumulated exercise, firm conclusions are difficult to draw.

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Exercise, Vascular Wall and Cardiovascular Diseases

An Update (Part 2)

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Contents

Abstract

There is much evidence extolling the virtues of physical activity on cardiovascular disease (CVD). The evidence derives from different population groups where leisure time physical activity reduced the risk of coronary heart disease and cardiovascular mortality in both men and women. Recent meta-analyses have shown that large risk reductions for both ischaemic and haemorrhagic stroke can be achieved by moderate or intense physical activity. There are many data from human and animal studies confirming a beneficial role for exercise in the prevention and treatment of CVD. Physical inactivity and obesity/overweight are not only associated with a number of health-related risk factors, but are considered to be independent risk factors for CVD, type 2 diabetes mellitus and hypertension. Clinical trials confirm that lifestyle interventions (dietary modification and increased physical activity) reduce the risk of progressing from impaired glucose tolerance to type 2 diabetes. Moreover, epidemiological studies indicate that the risk of hypertension

increases by being overweight. Modest increases in exercise intensity and frequency have hypotensive effects in sedentary hypertensive patients. Long-term training improves endothelium-dependent dilatation in the aorta and resistance arteries of the heart, whereas short-term training increases endothelial function in coronary conduit arteries. Overall, more scientific evidence will undoubtedly encourage the widespread advocacy of the clinical benefits of exercise therapy in the prevention and treatment of CVD.

Epidemiological data have long suggested that exercise has a decidedly beneficial role in the prevention and treatment of cardiovascular diseases (CVD).[1-3] Exercise leads to a number of favourable alterations in systemic function, including reductions in blood pressure, adiposity, improvement of endothelial function and insulin sensitivity. These effects of exercise combine to lower the incidence of stroke, diabetes mellitus and coronary heart disease.[4] Early clinical findings already demonstrated reduced morbidity and mortality in physically active individuals compared with their sedentary counterparts.[5] The preventative effect of exercise on CVD is demonstrated by a noticeably lower mortality rate among nonsmoking retired men who walk daily for 3.2 km (2 miles)[6] and in postmenopausal women who walk for 2.5 hours every week.[7] It is important to note that the reduction of cardiovascular morbidity and mortality achievable with exercise as primary and secondary prevention strategies is comparable with the effects of inhibitors of the renin-angiotensin-aldosterone system or statins.[8]

It is likely that the positive association between exercise and reduced cardiovascular mortality results from a reduction of one or more CVD risk factors. In general, exercise training improves endothelial function by upregulating endothelial nitric oxide synthase (eNOS) protein expression and phosphorylation, although improved endothelial function seldom occurs in healthy subjects.[9] Exercise favourably modifies several cardiovascular and related diseases, including coronary artery disease, metabolic syndrome, stroke, diabetes, aging, hypertension and menopause (see figure 1). The combined effects of exercise and obesity on CVD (diabetes and hypertension) are discussed later in section 1.8. The possible dose-response relationship between exercise training on cardiovascular benefits is be reviewed.

The first part of this review highlights recently published literature concerning the effects of increased physical activity in the treatment of CVD/risk factors such as coronary heart disease, metabolic syndrome, stroke, diabetes mellitus, aging, hypertension and menopause. The second part of this review highlights the benefits of exercise in mitigating obesity-related CVD (particularly diabetes and hypertension). The underlying mechanisms mediating the beneficial effects of exercise training (e.g. improved endothelial function, regression of coronary atherosclerosis, formation of collaterals and development of new vessels by vasculogenesis) are discussed. Finally, the dose dependency (duration, frequency and intensity) of physical activity and CVD in the context of ameliorating CVD is explored.

1. Exercise and Cardiovascular Diseases (CVD)/Risk Factors

1.1 Exercise and Coronary Artery Disease

A sedentary lifestyle is one of the five main risk factors for coronary artery disease (CAD), with the others being hypertension, abnormal blood lipid profiles, smoking and obesity.[10] After an acute myocardial infarction, the effects of these risk factors combine, leading to the clinical progression and prognosis of CAD. Data from meta-analyses reveal that regular physical exercise decreases the risk of developing coronary events in patients with CAD.[11,12] In view of the clinical benefits gained and its well documented cardioprotective

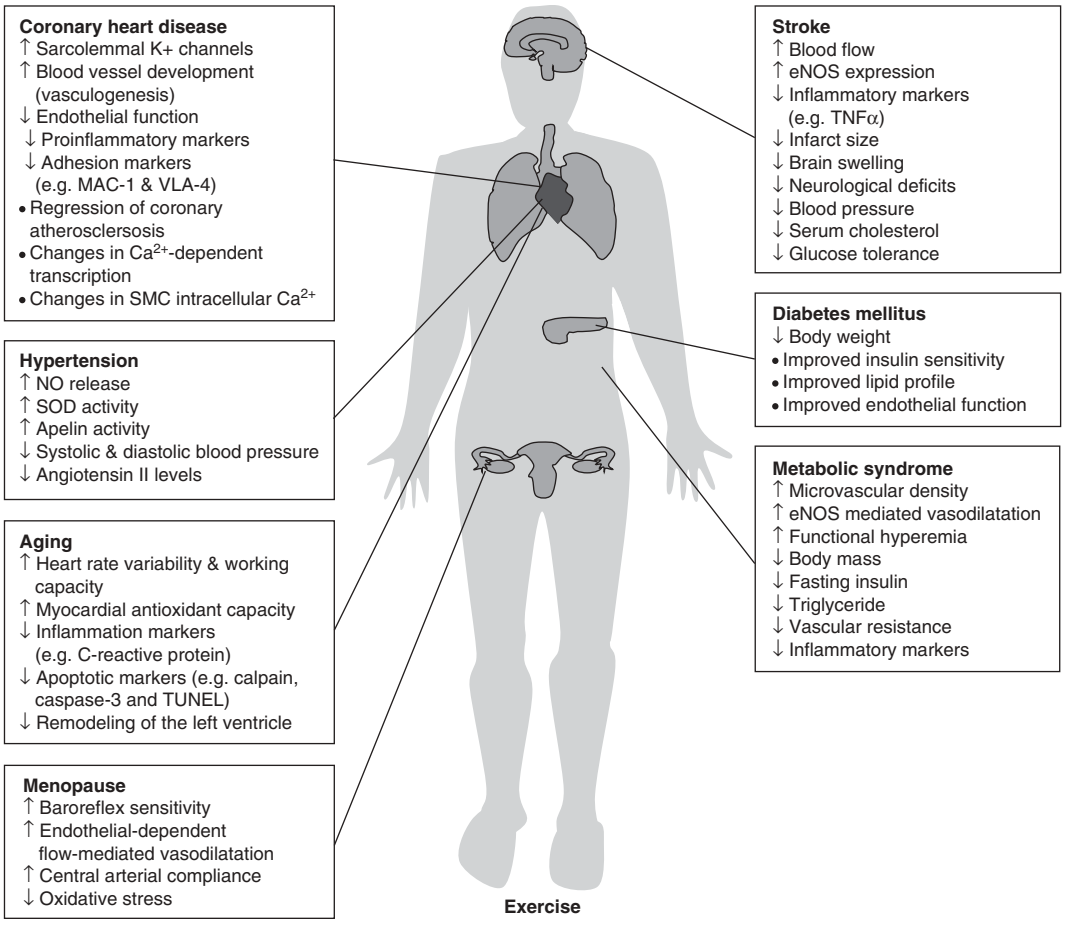

Fig. 1. The effect of exercise on cardiovascular disease risk factors including coronary artery disease, metabolic syndrome, stroke, diabetes mellitus, aging, hypertension and menopause. **eNOS** = endothelial nitric oxide synthase; **IL-6** = interleukin-6; **NO** = nitric oxide; **SOD** = superoxide dismutase; **SMC** = smooth muscle cell; **TNFα** = tumour necrosis factor-α; **TUNEL** = Terminal deoxynucleotidyl Transferase Biotin-dUTP Nick End Labeling; ↓ indicates decrease; ↑ indicates increase.

mechanisms, regular physical activity should be regarded by general practitioners and cardiologists alike as an effective part of the therapy provided to patients with CAD).[13] Surprisingly though, despite the wealth of clinical and epidemiological data indicating benefits of exercise,[14] the physiological or mechanistic basis of the cardioprotection provided by exercise remains largely unknown. Current theories include changes in cardiac excitation-contraction pathways such as reductions in intracellular Ca^{2+}-mediated regulation of the contractile apparatus in coronary smooth muscle, alterations in Ca^{2+}-dependent gene transcription and augmented activation of sarcolemmal K^+ channels (see figure 1). On the other hand, a recent review by Linke et al.[15] suggests that endothelial dysfunction is a likely predictor of cardiovascular events. They proposed the following four underlying mechanisms mediating the positive effects of exercise training: (i) improved endothelial function; (ii) regression of coronary atherosclerosis; (iii) formation of collaterals; (iv) development of new vessels by vasculogenesis (see figure 1).

CAD, as well as other CVD, is associated with increased levels of pro-inflammatory markers, and in keeping with this are recent studies

suggesting that exercise promotes cardioprotection through anti-inflammatory effects.[16] Also, Schumacher et al.[17] demonstrated that physical performance is inversely correlated with levels of pro-inflammatory markers in CAD patients, so possibly retarding atherosclerosis. Furthermore, Peschel et al.[18] examined the benefits of a multifactorial intervention programme on atherogenic adhesion molecules on the surface of monocytes in patients with CAD. They concluded that a 4-week, high-frequency, long-duration exercise training regimen diminished the expression of atherogenic adhesion molecules MAC-1 und VLA-4 in patients treated with statins. However, after 5 months of home-based exercise training of moderate frequency and duration, these effects were blunted, suggesting that patients in a cardiac rehabilitation programme might enjoy additional antiatherogenic benefits if offered a high-intensity, prolonged exercise programme (see figure 1). A recent study by Wisløff et al.[19] confirmed that high-intensity interval training has a greater beneficial effect than moderate-intensity training in patients with heart failure.

Epidemiological studies have long supported the view that exercise reduces morbidity and mortality from CAD. Some mechanistic insight was provided by Yamashita et al.[20] who demonstrated that exercise significantly reduced the magnitude of a myocardial infarction in a biphasic manner. There was an initial acute increase in manganese-superoxide dismutase (Mn-SOD) activity (30 minutes after exercise in rats), which was then followed by a rapid inactivation (3 hours after exercise). Such a reversible phenomenon, similar to phosphorylation and dephosphorylation mechanisms of kinases, confers biphasic cardioprotection.[19] Administration of an antisense oligodeoxyribonucleotide to Mn-SOD abolished the expected exercise-related decrease in infarct size.[20] Thus, it was proposed that the production of reactive oxygen species (ROS) and endogenous tumour necrosis factor-α (TNFα) and interleukin-1β induced by exercise-activated Mn-SOD, which in turn orchestrated the biphasic cardioprotection against ischaemia/reperfusion injury.[20]

Of general importance is that the quantity and intensity of physical activity required for the primary prevention of CAD remains unclear. The Harvard Alumni Health Study suggests that total physical activity, especially if vigorous, reduced CAD risk. Moderate and light activities produced non-significant inverse associations between physical activity and CAD risk reduction.[21] Physical activity also reduces CAD risk in male patients with multiple coronary risk factors.[21]

Regular exercise training increases intraluminal shear stress, which is a powerful stimulus for improved endothelial function in coronary and peripheral arteries.[22] What remains unresolved is the effect of different exercise intensities on endothelial function. Recently, Farsidfar et al.[23] examined the interactions of anaerobic threshold and peak oxygen uptake levels of acute exercise on flow-mediated dilatation in patients with stable CAD. In such patients, endothelium-independent vasoreactivity increased with threshold levels of exercise; however, it was significantly decreased at peak levels of exercise.[23] Moderate levels of exercise (at near anaerobic threshold level) can be considered as both therapeutic and preventative in CAD patients enrolled in cardiac rehabilitation programmes.[23] There is also an important study needed of the underappreciated comorbidity of coronary heart disease and mental illnesses such as schizophrenia. One proposal is that in this population, the confluence of a diet rich in fat and poor in fibre, coupled with a lack of exercise and increased smoking, are likely causes of coronary heart disease, irrespective of medication and socio-economic deprivation.[24] Clearly, regular exercise may offer considerable benefits in the context of a more comprehensive preventative strategy for CAD patients with mental illness.[24]

1.2 Exercise and the Metabolic Syndrome

The metabolic syndrome refers to a combination of abdominal obesity, insulin resistance and atherogenic dyslipidaemia that is accompanied by prothrombotic and proinflammatory states. The metabolic syndrome is not only of global concern, but is also gender neutral as shown by

recent studies underlining the high prevalence of the metabolic syndrome in women, and those aged 60 years or more.[25]

Two definitions in widespread use are those put forward by the WHO[26] and the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III).[27] The WHO[26] definition includes impaired glucose tolerance, diabetes and/or insulin resistance together with two or more of the following: (i) arterial blood pressure ≥140/90 mmHg; (ii) dyslipidaemia, defined as plasma triglyceride concentration ≥1.7 mmol/L (150 mg/dL) and/or high-density lipoprotein cholesterol (HDL-C) <0.9 mmol/L (35 mg/dL) in men, <1.0 mmol/L (39 mg/dL) in women; (iii) central obesity, defined as a waist-to-hip ratio of >0.90 in men, >0.85 in women and/or a body mass index (BMI) of >30 kg/m^2; and (iv) microalbuminuria, defined as urinary albumin excretion rate ≥20 mg/min or a albumin : creatinine ratio ≥30 mg/g.

The NCEP ATP III[27] definition requires the presence of any three of the following: (i) abdominal obesity, defined as a waist circumference of >102 cm (>40 inches) in men, >88 cm (>35 inches) in women; (ii) plasma triglycerides ≥1.69 mmol/L (150 mg/dL); (iii) HDL-C <1.03 mmol/L (40 mg/dL) in men, <1.29 mmol/L (50 mg/dL) in women; (iv) blood pressure ≥130/85 mmHg; (v) fasting glucose ≥6.1 mmol/L (110 mg/dL). Other markers, such as chronic subclinical inflammation, hyperuricaemia and coagulation disorders, are also associated with metabolic syndrome,[28-30] and the definition is likely to continue to evolve.

Patients with metabolic syndrome are at an increased risk for developing type 2 diabetes and CVD.[31,32] Epidemiological studies reveal a prevalence of metabolic syndrome that increases with age and obesity, and patients with metabolic syndrome should be recognized as being at a higher risk for cardiovascular complications such as atherosclerosis, diabetes and CAD.[33-35] The many beneficial effects of increased physical activity and exercise on metabolic syndrome have recently been extensively reviewed.[36] Ford et al.[37] examined the roles of sedentary behaviours such as watching television (or videos) or even prolonged used of a computer as potential determinants of prevalence of the Metabolic syndrome in US adults. Not surprisingly, they confirmed that reducing the time that adults spent engaged in such activities (watching television/videos, computer usage etc), especially if coupled with increased physical activity, could result in substantial decreases in the prevalence of metabolic syndrome.[37] Cross-sectional relationships between moderate and vigorous physical activity and metabolic syndrome were examined in the Whitehall II study of civil servants (aged 45–68 years).[38] Moderate and vigorous physical leisure-time activities were each associated with a reduced risk of being classified with metabolic syndrome independently of age, smoking and high alcohol intake.[38] Both vigorous and moderate activities reduce the impact of the metabolic syndrome cluster of risk factors in middle-aged populations.[38] Reduced BMI and increased cardiovascular fitness are key elements in reducing the impact of known risk factors for metabolic syndrome.[38] In another cross-sectional survey of 60-year-old men and women undertaken recently, there was a robust inverse dose-response relationship between the extent of leisure-time physical activity and the incidence of metabolic syndrome.[39] There is, therefore, uniform agreement, as exemplified by the findings of Rennie et al.[38] and Halldin et al.,[39] that physical activity is pivotal in the prevention and treatment of metabolic syndrome.

The obese Zucker rat (OZR) is an animal model of the metabolic syndrome, which is based on chronic hyperphagia. This animal model rapidly becomes obese and develops profound insulin resistance and hypertriglyceridaemia with accompanying hypertension and the emergence of prothrombotic and proinflammatory states. A recent study by Yan et al.[40] reports that (i) rats on a high-fat diet showed the typical characteristics of metabolic syndrome, including obesity and hypertension; (ii) endurance exercise mitigated these signs of metabolic syndrome, while also reducing cannabinoid receptor type 1 (CB1) and activated peroxisome proliferator-activated receptor-δ (PPAR-δ) expression in adipose tissue. These findings imply that both the endocannabinoid system and its regulation by PPAR-δ are

involved in therapeutic interventions in the treatment of the metabolic syndrome, including the use of endurance exercise.[40] There are promising new results reported in a recently conducted phase III study where rimonabant, a new class of selective CB1 receptor blocker, was used to produce weight loss in overweight/obese patients with untreated dyslipidaemia; there were also concomitant improvements of their glucose and lipid profiles.[41] Among patients who completed a full 1-year course of treatment at 20 mg/day, ~73% of patients lost ≥5% bodyweight, compared with ~28% for placebo.[41]

The progressive reduction (~25%) in skeletal muscle microvessel density that develops during the metabolic syndrome in the OZR is most likely due to a chronic reduction in nitric oxide (NO) bioavailability as part of insulin resistance[42] (see figure 1). Low vascular NO bioavailability impairs angiogenesis through stimulation of matrix metalloproteinase (MMP)-2 and MMP-9 activities. A 10-week regimen of treadmill exercise (1 hour/day, 5 days/week, 22 m/min) causes reduced body mass, fasting insulin and triglyceride levels in OZR rats (compared with their sedentary counterparts[42] (see figure 1). Exercise also produces a nearly 20% increase in microvascular density and an improved endothelial function in both large and small arteries of the gastrocnemius muscle.[42] Functional hyperaemia is also improved when OZR undergo regular exercise, and there are reductions in the minimum vascular resistance in the gastrocnemius muscle with no changes in arteriolar stiffness[42] (see figure 1). It is possible that exercise-related improvements in microvascular density in the OZR are better related to improve NO bioavailability and reduced inflammatory states rather than to augmented MMP function[42] (see figure 1).

1.3 Exercise and Stroke

Stroke is the third leading cause of death in the US, after coronary heart disease and cancer. Many stroke survivors are left with varying degrees of physical and mental disabilities. Because of extended hospitalizations and the resultant loss of productivity, stroke has a large negative economic and social impact. Due to the limited treatment options for stroke,[43] efforts should be directed towards its prevention, with an emphasis on modifiable risk factors. Regular exercise is associated with decreased cerebrovascular events, which may be caused by improved endothelial regulation of cerebrovascular tone. However, the manner of the protective effects of physical activity on stroke risk is less clear as this is compounded by some inconsistent findings. Several studies report an inverse association between regular physical activity and the risk of stroke. For example, there is an inverse association between physical activity and the risk of stroke in male physicians.[44] Leisure-time exercise also decreases the occurrence of ischaemic stroke in an urban, elderly, multiethnic population.[3]

Additional evidence that exercise is beneficial in modifying risk factors for different types of stroke (total, ischaemic or haemorrhagic) is provided by a recent meta-analysis showing that the largest risk reduction for both ischaemic and haemorrhagic stroke is achieved by moderate or intense physical activity compared with inactivity.[2] Related to this are findings that leisure time, occupational, or daily commuting involving physical activity are independently associated with a reduced risk of ischaemic stroke.[45] This is of importance because daily active commuting is a major source of total physical activity in some populations, is inexpensive and can be implemented virtually anywhere. For example, >90% of people in urban China walk or cycle to work daily[45] and >40% of the Finnish do likewise.[45] Modest levels of exercise decrease the risk of stroke likely through mechanisms involving enhanced eNOS activity,[46] deceased expression of TNFα,[47] and reductions in bodyweight, blood pressure, serum cholesterol and glucose tolerance[47] (see figure 1). In a large prospective cohort of apparently healthy women, a lifestyle consisting of abstinence from smoking, low BMI, moderate alcohol consumption, regular exercise and a healthy diet was associated with a significantly reduced risk of total and ischaemic stroke, but not of haemorrhagic stroke. These findings underscore the importance of healthy behaviors in the prevention of stroke.

It has been suggested that increased expression and activity of eNOS is an important component of exercise-induced amelioration of brain injury after acute focal cerebral ischaemia in an animal model of middle cerebral artery occlusion (MCAO)[48] (see figure 1). Exercise is thus beneficial in cerebral vascular injury by augmenting NO-dependent vasodilatation, increasing regional cerebral blood flow, reducing cerebral infarct size and brain swelling, and diminishing neurological deficits[46] (see figure 1). In contrast, these neuroprotective effects of exercise are absent in eNOS-deficient mice, supporting a central role of enhanced eNOS activity in exercise-induced protection against cerebral injury.[46] Exercise limits the extent of inflammatory injury during reperfusion by reducing the expression of inflammatory mediators (such as intercellular adhesion molecule [ICAM]-1), and the accumulation of leukocytes in the vascular lumen, intramural wall or in the brain parenchyma of rats. There is intriguing evidence reporting that compared with spontaneous recovery, early treadmill training (starting 24 hours after MCAO) accelerates the reduction in brain infarct volume, while at the same time improving neurological function.[48] These benefits also occurred when exercise was initiated later (1 week post-MCAO). However, the effect was not as prominent when compared with early treadmill training.[48]

1.4 Exercise and Diabetes Mellitus

Diabetes is a metabolic disease characterized by hyperglycaemia, insulin resistance and/or impaired insulin production. Type 2 diabetes is one of the fastest growing public health concerns in both developed and developing countries, with the estimated number of people with diabetes in the world increasing from 171 million in 2000 to 366 million in 2030.[49,50] Of particular concern is that diabetic people have a 2- to 4-fold greater risk of CVD,[51,52] which manifests with both micro- and macrovascular complications. Some reports suggest that heart disease may account for 75% of all deaths in diabetic patients,[53] while others suggest that atherosclerosis accounts for 80% of mortality in diabetic patients.[54] Whatever the absolute risk, it remains clear that the cardiovascular risk is profound in diabetes; in fact an increased risk occurs even prior to development of diabetes. For example, in the Nurses Health Study,[55] the relative risk for myocardial infarctions and stroke was 2.82 before diagnosis of diabetes, and 3.71 after diagnosis (compared with non-diabetic individuals). For participants who had diabetes at baseline, the relative risk was 5.02. Since macrovascular damage is present prior to the diagnosis of diabetes, early improvements in cardiovascular health are crucial, and form the guiding principle in recommending lifestyle modifications in the management of diabetes. Physical activity has many beneficial effects on diabetes, which likely occurs through several different mechanisms. Some of these exercise-related improvements include reduced bodyweight, improved insulin sensitivity, improved lipid profiles and enhanced endothelial function among others.[56] For most patients, by the time a diagnosis of diabetes is made, cardiovascular complications have already begun.[57] Approximately 70% of people with diabetes will die as a result of a vascular event.[58] In attempting to reduce this excessive risk, all coronary risk factors in the diabetic patient must be addressed and treated aggressively. The first priority should be a reduction in cardiovascular risk by vascular protection through a comprehensive approach that includes lifestyle modifications,[59] such as cessation of smoking, healthy eating habits, achieving and maintaining a healthy weight and importantly, engaging in regular physical activity.

Large, well designed randomized controlled trials have demonstrated that lifestyle intervention reduces the risk of type 2 diabetes in people with impaired glucose tolerance.[60,61] In both studies, a 5–7% bodyweight loss was achieved with supervised programmes that included dietary control and moderate exercise, and the relative risk of developing type 2 diabetes was reduced by 58% compared with usual care.[60,61] In one of these studies of 3234 patients,[61] metformin was approximately half as effective as lifestyle intervention in reducing diabetes risk. Metformin has several beneficial effects on cardiovascular risk factors and it is currently the

only oral antihyperglycaemic agent associated with decreased macrovascular outcomes in diabetic patients.[62] Of particular interest are the findings of the Diabetes Prevention Program[61] showing that exercise and diet were able to delay the onset of diabetes by 11 years, compared with a delay of ~3 years by metformin. In fact, there is a 'dose-response relationship' between increased bodyweight and risk of diabetes.[63] While lifestyle modifications are effective in early type 2 diabetes, metformin is often used in later stages.[64] Physical activity helps diabetic patients achieve several goals, including increased cardiopulmonary fitness, increased vigour, improved glycaemic control, decreased insulin resistance, improved lipid profile and maintenance of weight loss.[65-67] Large cohort studies confirmed that people with type 2 diabetes benefit from regular physical activity – reductions in cardiovascular and overall mortality ranged from 45% to 70% over 12–14 years.[58,68,69] Physical activity, independent of weight loss, has a beneficial effect on diabetic mortality.[70] In the Nurses Health Study, the health determinants and risk factors of a large group of females were tracked over 20 years.[68] The greatest reduction in relative risk for CVD occurred in the group reporting the highest levels of physical activity. Thus, those who reported ≥4 hours of moderate to vigorous activity a week showed a 40% reduction in cardiovascular events compared with those who reported <2 hours of physical activity. While moderate amounts of exercise are beneficial, vigorous exercise may be contraindicated in some diabetic individuals. High-intensity exercise is contraindicated since it may exacerbate diabetic cardiovascular complications such as overt peripheral neuropathy and retinopathy.[53] Clinical meta-trials such as the Diabetes Prevention Program make a convincing case for the implementation of lifestyle intervention programmes involving diet and/or exercise as they retard the progression of the impaired glucose tolerance to type 2 diabetes.[71] A prescription for aerobic exercise of mild to moderate intensity, including walking and jogging lasting 10–30 minutes a day for 3–5 days a week is recommended.[71] Prospective clinical trials show that moderate or high levels of physical activity or physical fitness accompanied by changes in lifestyle (dietary modification and increase in physical activity) can prevent type 2 diabetes. Based on a review of the scientific evidence, it was recommended that 30 min/day of moderate- or high-level physical activity be used as an effective and safe measure to prevent type 2 diabetes in all populations.[72] Of particular importance to diabetic patients unable to exert much physical exertion is a study demonstrating that type 2 diabetic patients can benefit from even modest muscular activity such as vibration exercise, which enhances glycaemic control in patients with type 2 diabetes.[73]

The etiological mechanisms underlying diabetes are uncertain, but it is clear that lifestyle modifications have definite yet modest beneficial effects on the diabetic CVD risk profile. Physical exercise in well controlled type 2 diabetic patients has endocrine and metabolic effects, including improved utilization of blood glucose and free fatty acids in muscles, while at the same time lowering blood glucose levels. Insulin mediates glucose uptake via the GLUT4 glucose transporters and subsequent suppression of lipolysis.[51] Beside its metabolic effects, insulin also directly causes NO-mediated vasodilatation[73] (see figure 1). These effects are reduced with insulin resistance, leading to the possibility that loss of insulin-mediated endothelial changes may be partly responsible for the increased cardiovascular risk in diabetes. Exercise increases glucose uptake in skeletal muscle by stimulating the GLUT4,[53,71] insulin receptor substrate 1,[71] and phosphoinositide-3 kinase protein[70] in skeletal muscle, while also improving insulin sensitivity (see figure 1). The role of the inflammatory response in atherosclerosis is well described.[54] Plasma levels of high-sensitivity C-reactive protein, a frequently used marker of inflammation and possibly as a predictor of cardiovascular risk and perhaps the development of diabetes,[54] are reduced by exercise[56] (see figure 1).

1.5 Exercise and Aging

Age-related impairment of endothelium-dependent vasodilation is likely related to the

increased occurrence of hypertension, atherosclerosis and CAD that emerges in the elderly.[62] Regular aerobic exercise can prevent the age-associated loss in endothelium-dependent vasodilation and restore vasodilator capacity in previously sedentary middle-aged and older healthy men.[74] Both the age-induced reduction and training-induced enhancement of endothelium-dependent dilatation in highly oxidative skeletal muscle occurs through NO signalling mechanisms.[74] Regular physical activity is able to prevent age-induced endothelial dysfunction in elderly athletes through restoration of NO bioavailability subsequent to a reduction in oxidative stress.[75] However, there are many unresolved issues when considering the relationship of aging and exercise.[76] Important questions include: how does aging alter exercise-induced intracellular and intercellular mechanisms that generate ROS? Can acute and chronic exercise retard the decline in gene expression of metabolic and antioxidant enzymes during old age? Does exercise prevent age-dependent muscle loss (sarcopenia) or the loss of muscle strength? What kinds of antioxidant supplementation, if any, should the physically active elderly use?[76]

Aging is accompanied by increased susceptibility to free radical mediated tissue damage, a process that results from a loss of antioxidant capacity and the associated accumulation of free radicals that characterizes oxidative stress. Schriner et al.[77] generated transgenic mice that overexpress human catalase localized to the peroxisome, the nucleus, or mitochondria (MCAT). Median and maximum life spans were maximally increased (averages of 5 months and 5.5 months, respectively) in MCAT animals. Thus, these important findings support the free radical theory of aging and reinforce the importance of mitochondria as a source of these reactive species.

Physical activity has many well established health benefits; however, strenuous exercise increases muscle oxygen flux and elicits intracellular events that can paradoxically lead to increased oxidative injury.[76] Aging increases muscle injury, and the inflammatory response exposes senescent muscle to further oxidative stress. At the same time, muscle repair and regenerative capacity is reduced in old age, a process that potentially facilitates the buildup of cellular oxidative damage. The physically active elderly are likely to benefit from exercise-induced adaptation of cellular antioxidant defense systems. A combination of improved muscle mechanics, strength and endurance makes them less vulnerable to the damaging effects of acute injury and chronic inflammation.[76]

It appears that despite increased ROS production, aging muscle has a decreased gene expression of antioxidant enzymes – possibly due to a diminished ability for cell signalling. A major benefit of non-exhaustive exercise is to induce a mild oxidative stress that stimulates the expression of some antioxidant enzymes, a process that is likely mediated by the activation of redox-sensitive signalling pathways.[78] For example, gene expression of muscle mitochondrial SOD is enhanced after an acute bout of exercise subsequent to elevations in the levels of nuclear factor-kappaB and activating protein-1 binding.[79] Aging does not abolish, but rather seems to attenuate, training adaptations of antioxidant enzymes. Thus, for senescent muscle, training should be assisted with supplementation of exogenous antioxidants in future efforts to determine the optimal levels of physiological antioxidant defense mechanisms.[78,79]

Regular physical activity is related to a better microvascular endothelial function in older athletes, probably due to increased antioxidant defenses.[80] One concept is that advancing age is characterized by an impairment of endothelium-dependent flow-mediated dilation of conduit arteries with parallel reductions of plasma antioxidant and increases in plasma oxidative stress (as measured by malondialdehyde). In contrast, in older subjects who regular perform aerobic endurance training, flow-mediated dilation is preserved.[81]

Several studies document augmented endothelin-1 (ET-1) release in patients with hypertension, heart failure, atherosclerosis and obesity.[82,83] Aging results in a greater arteriolar vasoconstriction in gastrocnemius muscle to ET-1, which is mediated by enhanced function of ET_A receptors (and not through ET_B) receptor mechanisms

that are associated with either the endothelium or vascular smooth muscle.[84] Although enhanced vascular ET-1 sensitivity plays a role in vascular dysfunction that is often associated with aging, exercise appears not to affect the responsiveness of skeletal muscle arterioles to ET-1.[84] Exercise training does, however, protect against age-induced remodelling of the left ventricle that includes the loss of cardiac myocytes, reactive hypertrophy of the remaining cells and increases of connective tissues in aging hearts.[85] Regular exercise downregulates apoptotic markers (Terminal deoxynucleotidyl Transferase Biotin-dUTP Nick End Labeling [TUNEL]-positive staining, DNA fragmentation, and cleaved caspase-3) and causes a reduction in procaspase-9 protein expression and the ratio of Bax/Bcl-2 levels (see figure 1). This intriguing evidence suggests that exercise-induced modulation of the mitochondrial Bcl-2 pathway could ameliorate aging-induced ventricular remodelling, and thus improve cardiac function.[85] Exercise also inhibits ischaemia/reperfusion-induced myocardial apoptosis in the hearts of both young and old animals. In this instance, cardioprotection is offered by exercise improving myocardial antioxidant capacity and attenuating activation of calpain and caspase-3[86] (see figure 1). Furthermore, aging is associated with reductions in the heart rate variability (HRV) and working capacity. Evaluation of a group of elite master athletes (males aged 68.5 ± 4.5 years who practiced endurance running for at least 40 years) demonstrates increased HRV and a higher exercise capacity, both of which are well established predictors of cardiovascular and overall mortality.[87]

There is increasing reliance on alternate therapies such as regular exercise to reduce the impact of age-related cardiovascular dysfunction. It is likely that appropriate levels of exercise offers protection against age-related cardiovascular dysfunction through a combination of the following mechanisms: (i) gene expression of muscle mitochondrial SOD is enhanced, which is preceded by an elevated level of nuclear factor-kappaB and activating protein-1 binding; (ii) enhancement of endothelial NO-dependent vasodilation; (iii) protection against aging-induced cardiac remodelling and cardiac muscle cell apoptosis; (iv) protection against ischaemia/reperfusion-induced myocardial apoptosis; and (v) reduction of heart rate variability and exercise capacity regardless of age (see figure 1).

1.6 Exercise and Hypertension

Hypertension is a major health dilemma because of its silent nature, great prevalence and severe consequences if poorly managed. It is thought that one aspect of the genesis of hypertension may be the imbalance between endothelium-derived relaxing and constricting factors. Much evidence affirms the widely held view that regular physical activity reduces the risk of hypertension.[88-91] A meta-analysis of randomized controlled trials concluded that chronic dynamic aerobic endurance training lowers blood pressure, which was later confirmed in a study of chronic and moderate levels of exercise.[91] The mechanisms whereby exercise contributes to improved cardiovascular health remains poorly understood. A review by Hagberg et al.[92] critically addressed the effects of exercise on patients with hypertension and reported that women were better able to reduce blood pressure with exercise than men, and middle-aged people with hypertension obtained greater benefits than younger or older people. Low- to moderate-intensity exercise appears to be as beneficial as higher-intensity exercise for reducing blood pressure. Recently, Pinto et al.[93] evaluated the hypotensive effects of exercise in hypertensive patients using 24-hour ambulatory blood pressure monitoring and reported that exercise reduced the mean 24-hour systolic blood pressure from 143 to 136 mmHg and the mean 24-hour diastolic blood pressured from 91 to 85 mmHg. Similar results were obtained in animal studies, where exercise lowered systolic blood pressure (and angiotensin II levels) in spontaneously hypertensive rats (SHR)[94] (see figure 1).

Long-term exercise also positively impacts vascular function, as endothelium (NO)-dependent dilatation is significantly enhanced by physical activity in normotensive and hypertensive

patients.[95] Likewise, regular exercise attenuates adrenergic-induced vasoconstriction by increasing the production of endothelium-derived NO in both hypertensive and normotensive rats[96] (see figure 1). Exercise elevates total plasma nitrite concentration and SOD activity in the aorta and heart of SHR[14] (see figure 1). Interestingly, Boissiere et al.[97] studied the impaired vasodilation produced by the carbon monoxide-releasing molecule tricarbonyldichlororuthenium [$Ru(CO)_3Cl_2]_2$ in hypertensive rats (two-kidney, one-clip Goldblatt model of hypertension). Exercise training potentiated $[Ru(CO)_3Cl_2]_2$-induced relaxation in the thoracic aorta, possibly by modulating K^+ channel activity.[86] Moreover, exercise reversed the reduced cardiovascular expression of apelin and its receptor, APJ (endothelial G-protein-coupled receptor; thought to be a novel anti-hypertensive factor) and so attenuates both hypertension and cardiac hypertrophy in SHR.[97] Thus, enhancing the expression of cardioprotective factors such as the apelin/APJ system represents one mechanism by which exercise reverses hypertension and ameliorates its complications, at least in rodent models of hypertension[98] (see figure 1).

Previous meta-analyses have focused mainly on resting blood pressure and did not report on other outcomes, such as ambulatory blood pressure monitoring of the hypotensive effects of exercise. A study by Cornelissen and Fagard[99] reported that aerobic endurance training decreases blood pressure by reducing vascular resistance, in which the sympathetic nervous system and the renin-angiotensin system appear to be involved, while also reducing the impact of other cardiovascular risk factors such as body fat, waist circumference, blood lipids and glucose/insulin dynamics. Findings by Cox[100] suggest that exercise lowers resting blood pressure, and when combined with calorie restriction (reducing total energy intake by 1000–1500 kcals/day), there was a greater fall in blood pressure and this was sustained for at least 24 hours.[99] Moderate to intense exercise is well tolerated by older individuals and can be undertaken supervised or unsupervised, with obvious benefits in reducing blood pressure in the short and long term.[100] The intensity of exercise needs to be taken into account when recommending exercise programmes – for example, older individuals with hypertension should be monitored when enrolled in a swimming programme. Related to this are the findings of Cox[100] who found that swimming and not walking, increased supine systolic blood pressure by 4.4 mmHg after 6 months. The reasons for this are unclear, but it may be that there are additional factors that operate during swimming such as the different posture, the effects of hydrostatic pressure, facial immersion, breath holding, water temperature and the increased thermal conductivity of swimming-induced elevations in blood pressure may thus be a response to adaptation to the aquatic environment.[100]

1.7 Exercise and Menopause

Entering menopause significantly increases the risk of CVD, an effect that is largely related to the loss of estrogen-mediated cardiovascular protection. CVD is the leading cause of death among women and accounts for more than half of their deaths.[101,102] Reduced exercise capacity is associated with an increased risk of death, while increased physical activity leads to a 30–50% reduction in CVD in women.[103]

Increased central arterial stiffness, a risk factor for CVD, is significantly lowered by endurance training in postmenopausal female athletes compared with their sedentary peers.[104] Moreau et al.[105] reported that regular aerobic exercise at the upper end of the recommended physical activity level (defined as ~70% of maximal heart rate) lowered central arterial stiffness even in sedentary postmenopausal women who had previously used hormone replacement therapy. A recent cross-sectional study reported on the benefits of moderate and vigorous exertion on central arterial stiffness in postmenopausal women,[104] confirming similar benefits produced by activities such as walking, gardening or light sports[103] (see figure 1). More intense levels of physical exertion produce additional improvements in vascular reactivity and myocardial flow reserve.[103] A study by Manson et al.[7] demonstrated that both walking and vigorous exercise are associated with

substantial reductions in the incidence of cardiovascular events among postmenopausal women, irrespective of race, ethnic group, age or BMI. Moreover, exercise lowers plasma ET-1 concentrations and so retards ET-mediated hypertension and/or atherosclerosis. Related to this is that endothelial function, as assessed by brachial artery flow-mediated dilatation, is significantly impaired in sedentary postmenopausal women, but is markedly improved by both exercise and estrogen therapy[106] (see figure 1). However, the benefits of exercise and estrogen replacement are not additive, possibly because of a redundancy of the upregulation of NO signalling pathways in response to these two interventions[107] (see figure 1). More recently, a randomized, controlled study showed that previously sedentary, overweight or obese postmenopausal women experienced a graded dose-response increase in fitness across various levels of exercise training.[108] While the majority of post-menopausal women are not candidates for lipid-lowering therapy, they remain at high risk for coronary heart disease.[109] The WOMAN (the Women On the Move through Activity and Nutrition) study clearly shows that moderate exercise (150 minutes of brisk walking per week) coupled with a dietary modification successfully reduced markers of subclinical atherosclerosis.[109] In addition, exercise-induced improvements in plasma glucose, insulin and low-density lipoproteins persisted over an 18-month follow-up period.[110]

An 8-week treadmill exercise programme increased baroreflex sensitivity and reduced oxidative stress levels in ovariectomized rats[107] (see figure 1). There is also a direct cardioprotective effect of exercise on cardiac myofilament Ca^{2+} activation in ovariectomized rats.[110] Cardiac upregulation of β_1-adrenergic receptors in the left ventricular myocytes due to estrogen deficiency does not occur in exercise-trained ovariectomized rats.[111] Regular running with moderate intensity normalizes the changes in myofilament Ca^{2+} activation associated with ovariectomy, including suppression of Ca^{2+} hypersensitivity of the myofilaments and a shift in the myosin heavy chain (MHC) isoforms toward β-MHC. These cardioprotective effects of exercise training may be in response to the increased expression of β_1-adrenergic receptors (which would improve cardiac contraction and relaxation cycles) and HSP72 (a protective factor in molecular alterations in myofilament Ca^{2+} activation in ovariectomized hearts).[111]

What type of exercise is effective in combating CVD in postmenopausal women? Is walking as effective as vigorous exercise? The Women's Health Initiative Observational Study clearly shows that walking reduces the risk of CVD by about 12–40% over 3.2 years,[112] which is similar to the benefits achieved with more vigorous physical activity. These are encouraging findings, because sedentary postmenopausal women may find walking easier than starting a vigorous exercise programme.[112] A related finding suggests that even a 12-week aerobic exercise programme at low intensity improves central arterial compliance in postmenopausal women by levels that are similar to those obtained by moderate intensive training[113] (see figure 1). Thus, exercise is able to match estrogen replacement therapy in stimulating eNOS activity. This non-pharmacological intervention is devoid of obvious adverse effects and has the added benefit of reducing the damaging effects of ROS on cardiovascular function.

1.8 Joint Effects of Exercise and Obesity/Overweight on the Risk of CVD (Diabetes Mellitus and Hypertension)

Sedentary lifestyles and obesity are important global concerns and importantly represent modifiable cardiovascular risk factors. Physical inactivity and obesity/overweight are not only associated with a number of health-related risk factors, but are considered to be independent risk factors for CVD, type 2 diabetes and hypertension.[114,115]

It has been known for quite some time that obesity and weight gain are associated with an increased risk of type 2 diabetes.[114] Thus, it is not surprising that data from epidemiological studies confirm that higher levels of leisure time physical activity reduced the risk of developing type 2 diabetes.[115] Even moderate to intense

occupational physical activity, such as commuting to work by walking or cycling, significantly lowered risk of type 2 diabetes.[115] At least 30% of new cases of obesity and 43% of new cases of diabetes could be prevented by adopting a relatively active lifestyle (<10 hours/week of television watching and ≥30 min/day of brisk walking). It is well known that individuals with impaired glucose regulation are at a higher risk for developing type 2 diabetes.[116]

Recent clinical trials in China, Finland, and the US strengthen the belief that lifestyle intervention (dietary modification and increased physical activity) reduces the risk of progressing from impaired glucose tolerance to type 2 diabetes.[117,118] A prospective cohort study by Hu et al.[119] evaluated the association between physical activity, BMI, glucose levels and the risk of type 2 diabetes. They showed that during a mean follow-up period of 9.4 years, there were 120 cases of type 2 diabetes. After adjustment for confounding factors (age, education, gender, systolic blood pressure and smoking), reduced physical activity was a critical risk factor for type 2 diabetes. The association between a sedentary lifestyle and type 2 diabetes was particularly important in patients with (i) both obesity and impaired glucose regulation; (ii) either obesity or impaired glucose regulation; and (iii) a normal BMI and glucose regulation.

Data from clinical trials and cross-sectional studies[115,120,121] confirm the inverse relationship between the extent of physical activity or aerobic exercise and blood pressure, although this association is less clear in prospective studies where several studies indicate that regular physical activity reduces the risk of hypertension in men,[122-126] but not in women.[127-129] Epidemiological studies conclude that the risk of hypertension increases by being overweight, obese and gaining weight.[127,130,131] The association between physical activity and BMI as risk factors for hypertension was examined by Hu et al.[128] in 8302 Finnish men and 9139 women aged 25–64 years. They showed that regular physical activity and weight control lowered the risk of hypertension in both sexes, regardless of the level of obesity. Moreover, the Aerobic Center Longitudinal Study reported that low cardio-respiratory fitness is a strong and independent predictor of CVD mortality among men, independent of body composition and other CVD risk factors.[129] Importantly, however, was the observation that overweight or obese men with moderate to high levels of cardio-respiratory fitness had a greater risk reduction in CVD mortality than did normal-weight or overweight men with low levels of cardiorespiratory fitness.[129] Furthermore, the Lipid Research Clinics Study also evaluated the effect of fitness and body type on longevity using data from 2506 men and 2860 women with a mean age of 46 years.[132]

There was a greater risk of CVD mortality in subjects classified either as fit-fat, unfit-lean, and unfit-fat compared with people classified as fit-lean – stressing the importance of not having visceral obesity and undertaking regular exercise as preventative measures. The Nurses' Health Study and the Health Professionals' Follow-up Study also confirm a strong, graded inverse association between physical activity and the risk of CAD, which was present in both non-obese (BMI ≤29 kg/m^2) and obese (BMI >29 kg/m^2) nurses, with the findings also being applicable to lean (BMI <25 kg/m^2), and overweight (BMI 25–29.9 kg/m^2) men.[133,134] The first National Health and Nutrition Examination Survey assessed CVD mortality rates as a function of the level of physical activity and BMI confirms that CVD mortality rates are higher in those with sedentary lifestyles and obesity. On the other hand, increased exercise and a normal weight lowered CVD mortality rates.[135] Recent findings from the Nurses Health Study suggest even a modest weight gain (4–10 kg) in women significantly increased the risk of coronary artery disease.[136]

2. The Dose-Response Relationship of Exercise Training and Cardiovascular Benefits

Considering the cardiovascular benefits of exercise training and its underlying mechanisms, a more pragmatic question for many would be: just how much exercise is needed to confer such

benefits? To address this question, three parameters of physical activity should be considered: intensity, duration and frequency.[135] Intensity refers to the amount of time undertaking of an exercise activity; duration indicates the length of the exercise intervention; while frequency indicates the number of training sessions in a given period of time.

Relatively modest increases in exercise intensity and frequency have hypotensive effects in otherwise sedentary hypertensive patients.[136] The effect of exercise intensity appears to be dose-dependent since decreases in systolic and diastolic blood pressures were reduced (mean pressure reduction: ~5 mmHg) when exercising for 30–60 min/week and further reduced (mean pressure reduction; ~10 mmHg) if exercising for 61–90 min/week. However, there were no additional reductions in blood pressure when exercising for even greater periods.[136] On the other hand, the study by Ishikawa-Takata et al.[138] suggests no apparent association between exercise frequency and hypotensive effects, since systolic and diastolic blood pressures were unaffected in hypertensive patients regardless of the frequency of exercise intervention – mean blood pressures were similar when exercising either once or twice a week, or 3–4 times/week or even >5 times/week.[136] A final issue is the validity of the commonly held belief that the duration of exercise is a key to providing cardiovascular benefits. In general, there is no graded improvement in vascular function with increasing exercise duration, although there is enhanced endothelium-dependent dilatation in some arteries after endurance training intervention.[136]

Evaluating the benefits of exercise duration is complicated by the use of different vascular beds. While long-term training improves endothelium-dependent dilatation in the aorta and in resistance arteries of the heart,[137,138] short-term training increases endothelial function in coronary conduit arteries.[137]

It thus appears that the dose-response relationship between exercise training and cardiovascular benefits is likely to be sigmoidal. It is important to note that relatively modest amounts of exercise may not be sufficient to reduce the influence of cardiovascular risk factors.[121] Thus, the specific amounts of exercise required to induce cardiovascular health benefits depends on the various risk factors or CVD of interest. It is likely that it is the total amount of physical activity, rather than its specific characteristics, that may be of importance.[121] That being said, there remains other issues: will the cardiovascular benefits be sustained upon the cessation of exercise training? Or how long do cardiovascular benefits last with a given amount of exercise?

Finally, intensity of exercise training can also be interpreted in a relative manner, in terms of multiple of metabolisms at rest (metabolic equivalent [MET]). Typically, exercise of a vigorous intensity (≥6 MET) confers positive effects in lipid profiles, glucose control and cardiovascular risks.[139] However, the mechanisms whereby vigorous intensity induces cardiovascular protection remains unclear. Some postulate that there may be adaptations during exercise of vigorous intensity. Aerobic capacity is enhanced more effectively, when compared with exercise at a moderate intensity.[140] Another mechanism may be mediated through adaptations in autonomic control. As a consequence of aerobic training, sympathetic drive at rest is reduced and vagal tone is increased, with potential effects on blood pressure, thrombosis, and other factors associated with coronary risk.[141] During exercise, higher intensities elicit exponentially greater increases in sympathetic drive. Thus, one might hypothesize that vigorous-intensity training would result in greater autonomic adaptations than moderate-intensity exercise of equal energy expenditure.

3. Conclusions

In Part 1 of this review article, we highlighted some of the mechanisms by which exercise produced beneficial changes in cholesterol levels, antioxidant systems, blood pressure levels, inflammation, heat shock proteins and ion channel activity. Moreover, benefits of exercise-induced vascular remodelling and reactivity were also discussed in the context of some cardiovascular diseases including coronary artery diseases,

hypertension, heart failure, peripheral vascular diseases and pre-eclampsia.[142] In Part 2 of this review, we provide recent evidence the effects of increased physical activity in the treatment of CVD/risk factors such as coronary heart disease, metabolic syndrome, stroke, diabetes, aging, hypertension and menopause. It is reported that exercise is an important non-pharmacological approach in the current strategies for combating the rising incidence of childhood and adult obesity. There are many hospital and out-patient based rehabilitation programmes for cardiac patients that exploit the many beneficial effects of routine exercise. Epidemiological data show a promising primary preventive role in postmenopausal women, although it may be premature to suggest the use of exercise training as a cardiac preventive measure in postmenopausal women. Careful application of exercise training as a supplementary treatment, in addition to the regularly used preventive procedures, should do no harm. While clearly more experimental studies are needed to further elucidate the molecular basis of exercise-induced vascular benefits, the clinical evidence for its beneficial prognostic effects in both primary and secondary prevention is overwhelming. More enticing scientific evidence will undoubtedly encourage the widespread advocacy of the clinical benefits of exercise therapy as important adjuncts in the prevention and treatment of CVD.

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REVIEW ARTICLE

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Natural Turf Surfaces
The Case for Continued Research

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Contents

Abstract

It is well documented that health and social benefits can be attained through participation in sport and exercise. Participation, particularly in sports, benefits from appropriate surface provisions that are safe, affordable and high quality preferably across the recreational to elite continuum. Investment, construction and research into artificial sports surfaces have increased to meet this provision. However, not all sports (e.g. golf, rugby and cricket) are suited to training *and* match-play on artificial turf without compromising some playing characteristics of the games. Therefore, full sport surface provision cannot be met without the use of natural turf surfaces, which also have an important role as green spaces in the built environment. Furthermore, a significant number of people participate in outdoor sport on natural turf pitches, although this is a declining trend as the number of synthetic turf surfaces increases. Despite natural turf being a common playing surface for popular sports such as soccer, rugby and cricket, few biomechanical studies have been performed using natural turf conditions. It is proposed that if natural turf surfaces are to help meet the provision of sports surfaces, advancement in the construction and sustainability of natural turf surface design is required. The design of a natural turf surface should also be informed by knowledge of surface-related overuse injury risk factors.

This article reviews biomechanical, engineering, soil mechanics, turfgrass science, sports medicine and injury-related literature with a view to proposing a multidisciplinary approach to engineering a more sustainable natural turf sport surface. The present article concludes that an integrated approach incorporating an engineering and biomechanical analysis of the effects of variations in natural turf media on human movement and the effects of variations in human movement on natural turf is primarily required to address the longer-term development of sustainable natural turf playing surfaces. It also recommends that the use of 'natural turf' as a catch-all categorization in injury studies masks the spatial and temporal variation within and among such surfaces, which could be important.

The health benefits gained from participation in sport and exercise are well documented.[1,2] For example, in the UK, recognizing that 'sport matters', a detailed action plan was drawn up by the UK Department for Culture, Media and Sport[3] to modernize and integrate the aims of implementation groups (Sport in Education, Sport in the Community and Sporting Excellence), to promote and achieve physical activity for all.[3] However, the significant benefits for population health from increased, regular participation in sport require provision and access to community sports facilities in a number of different environments. In 2008, for the first time, global urban population will reach 50% and is predicted to reach 86% in 2050.[4] Consequently, facilities for sport will be subject to increased intensification as land-use pressure increases, reducing the area available for sports facilities.

It is recognized that participation in sporting activity can take place on almost any surface and within a limited space. However, at the elite/professional and recreational/community levels within the developed and to some extent the developing world, a reasonable quality, safe sports surface is desirable. The provision of quality surfaces is also likely to encourage participation.[5] The performance characteristics of a surface are sport specific and will vary with the level at which a sport is played. They should allow a participant to perform to the best of their ability, without an increased risk of surface-related injury. In addition, high-quality, high-performance sports surfaces have a direct impact on elite/professional athlete performance and the status and individual rewards this brings. Provision of such surfaces by the sports industry, sports governing bodies and national governments is a key requirement for realizing the benefit to individual sports and national morale and esteem from such successes.

The challenge in sports surface engineering is to meet the demand for such surfaces within the different client expectations and budgets (both construction and ongoing maintenance) of different sports facilities. Several studies have illustrated, however, the danger of engineering sports surfaces on the basis of surface performance and durability, without consideration of human interaction.[6-8]

The majority of studies in this area have been in relation to synthetic sports surfaces, with the assumption that natural turf surfaces are a benchmark standard for safety. However, development in the engineering of natural turf surfaces for more intensified use, and use within enclosed stadium environments, has resulted in significant changes in mechanical properties that should not be ignored. Furthermore, the availability of natural turf surfaces for a number of sports at elite level is necessary (e.g. golf and cricket) and the only option in many communities for the provision of sports facilities. In rugby union, new generation long-pile synthetic turf pitches can be used for international rugby if both teams agree, under Regulation 22 of the International Rugby

Board.[9] To date, this has not taken place and the current norm is for synthetic surfaces to be used for elements of training within elite-level rugby and for natural turf pitches to be used for both competition and training. The adoption in soccer is different, with a number of elite teams in Europe playing competitive soccer on certified long-pile synthetic turf and the surface has been used for international fixtures with approval from Fédération Internationale de Football Association (FIFA), the world governing body for football (soccer).[10] However, the majority of surfaces for both training and competition remain natural turf.

The engineering of sports surfaces and the understanding of surface-related injury requires an integrated understanding of both surface mechanical behaviour and player biomechanical behaviour. This integration is two-way: by improving knowledge of surface effects on players, the understanding of injury risk can be improved; likewise, through an improved analysis of human movement and loading on the surface, surface design and material selection is improved. This is critical in the development of natural turf surfaces that have properties that vary in space and time. This article considers the extent of our current understanding and highlights where significant contributions are required in the particular context of natural turf surfaces.

1. Sports Facility Provision: The Role of Natural Turf

Affordable, safe and appropriate sports facilities are an important contribution to obtaining a healthy nation through sport and exercise participation.[1-3] A survey of households in England performed by 'Sport England' considered two questions: (i) where do people participate in sport for all reasons (e.g. recreation, fitness, competition); and (ii) where do people participate in sport for competition only? Natural turf surfaces were important for only three (soccer, golf and tennis) of the top ten sports when ranked by participation in the 4 weeks prior to the survey (table I).[11] This list is dominated by individual, indoor/home-based sports. In terms of competitive sport, however, natural turf was important in five of the top ten sports in the same survey.

1.1 Sports Participation

Synthetic turf sports surfaces have made an important contribution to increased opportunities for participation in organized competitive sports. Traditional sports such as hockey, soccer, rugby, tennis, golf and cricket require considerable investment (both financial and spatial) for surface provision. Traditionally, field hockey, soccer and tennis were played on natural turf before the development of artificial sports surfaces allowed year-round playing opportunity and in hockey, improved standards of play.[12] The continuing growth of artificial surfaces in school, community and club sport is important to provide a playing surface where performance is less influenced by adverse weather conditions, requires lower levels of maintenance[13-15] and provides the opportunity to make relatively smaller sports areas more cost effective due to a higher tolerance of regular multi-sport use. In comparison, a functional outdoor natural turf environment is heavily influenced by seasonal and day-to-day weather variations, intensive maintenance, drainage, its response to wear[14,16-18] and the provision of space to rotate pitch usage as a method of maintenance, restoration and damage prevention.

Comparative data for the costs of maintaining synthetic and natural turf are limited. However, a study by McLeod[19] indicated that costs were similar on an 'area of pitch' basis, but on a 'per hour of use' basis, the cost of maintaining natural turf was 3.6-fold greater than synthetic turf, including the cost of synthetic turf installation. Consequently, in some cases, traditional school playing fields have been downsized in order to raise capital for the rebuilding of sports facilities that utilize a synthetic pitch.[3] There is a danger, however, that this approach of selling portions of the school playing field in return for constructing a smaller (but full-sized for competitive sport) synthetic pitch reduces the area available for general play and unorganized sports activity in schools (National Playing Fields Association[20]),

Table I. The variation among surfaces used for different sports as indicated in a survey of English households as a percentage of respondents (adapted from Sport England[7])

Rank	Activity	Participation				Competition		
		indoor	home	outdoor	activity	indoor	home	outdoor
1	Swimming	45	10	45[a]	Rugby	0	0	100[b]
2	Keep-fit/yoga	45	40	15[c]	Bowls	53	0	47[b]
3	Snooker/pool/billiards	58	11	0	Hockey	0	0	100[d]
4	Cycling	0	3	68[c,e]	Cricket	0	0	100[b]
5	Weight training	45	47	0	Soccer	19	0	81[b,d]
6	Running (jogging)	0	0	82[c,e]	Netball	NR	NR	NR[f]
7	Soccer	19	0	81[b,d]	Golf	0	0	96[b]
8	Golf	0	0	96[b]	Motor sports	NR	NR	NR[e]
9	Ten-pin bowling	81	0	0	Athletics (T&F)	NR	NR	NR[b,g]
10	Tennis	0	0	100[b,f,h]	Volleyball	NR	NR	NR

a Water.

b Natural turf.

c Recreational open space.

d Synthetic turf.

e Road.

f Hard-court.

g Acrylic/polyurethane.

h Clay.

NR = not reported; **T&F** = track and field.

especially if the access to the new synthetic pitch is restricted during break and lunch times.

Within the UK, the maintenance of a natural turf sports environment has been highlighted as important for the protection of green spaces and playing fields for recreational sport in the community.[3] This is a priority for the UK and reflects the high urban population density and competition for land in that country and the urban ecosystems services that natural turf sports surfaces can provide. In other countries, priorities can be expected to differ depending on resources (both capital and land) and environmental and participation strategies.

1.2 Replication of Natural Turf: Playing Characteristics and Injury Patterns

The nature and properties of natural turf are also fundamental to the playing characteristics of soccer, rugby, golf and cricket. In cricket for example, pitch properties influence the range of shots played, ball speed after impact with the surface, the amount of seam movement and ball spin characteristics.[21] While these properties could be achieved with synthetic surfaces, controlled temporal variations in such properties are desirable and essential for the game; cricket is a sport that can end in a draw after 4–5 days of play; the likelihood of a positive result (win/loss) is increased when the deterioration of the pitch balances the game in favour of the bowler. Of course if this happens too soon, a 5-day game can finish prematurely, resulting in lost revenue from crowds and media coverage. In soccer, such temporal variation and spatial variation is undesirable and, as a consequence, there is an increasing use of sand materials to construct surfaces, with a resultant increase in maintenance costs for water, nutrients, etc. Another important characteristic is the natural temperature regulation and lubrication of the surface from transpiration in the grass plant. Grass plants can transpire at 110 kg $[H_2O]/m^2$/hour,[22] with

a latent heat of vaporization of 2.43 MJ/kg at 30°C.

Modification of hockey pitches that started in the 1970s from natural turf to artificial turf surfaces resulted in certain playing skill adaptations with a loss of some surface-related skills and an enhancement of other skills together with a faster-paced game.[12] The playing characteristics of hockey therefore changed as a result of the move from a natural turf pitch to artificial surfaces.

The development of modern synthetic turf has focused on reproducing the playing characteristics of natural turf. This development strategy has been motivated by experiences from the introduction of the first generation of synthetic turf into soccer in the early to mid 1980s. The early surfaces were characterized by higher stiffness, higher sliding friction and greater heat retention than natural surfaces. Consequently, players experienced higher ball bounce, faster ball roll, and significant skin damage and lower limb discomfort. 'First generation' describes the early nylon surfaces of the 1970s characterized by skin abrasions and excessive traction. 'Second generation' sand-filled surfaces with a typical pile length of 22–25 mm, are commonly used in recreational field hockey and multi-sport community facilities, but were deemed unsuitable for soccer when trialed in the 1980s. FIFA now permit and actively promote the use of a new generation of synthetic turf surfaces that meet new playing standards, commonly termed 'third generation' surfaces.[10] These surfaces have increased shock absorbency, longer pile length (typically 40–60 mm) and a rubber granular infill. However, variations in injury patterns derived from play on natural turf and a specific third-generation synthetic turf product (FieldTurf® – polyethylene/polypropylene fibre blend with silica sand and ground rubber infill) suggest that while the artificial turf has been designed to closely replicate natural turf characteristics, injury outcomes and therefore characteristics of play between the surfaces may not be comparable.[23]

A 5-year study of 240 Texas (USA) high-school American Football games found that the artificial and natural turf surfaces yielded unique patterns of injury incidence. While a greater incidence of muscle-tendon overload injuries, abrasions, non-contact, running and sprinting injuries (significant at $p < 0.05$) occurred on the artificial surface, lower incidences (non-significant) of concussion and ligament tears were reported on the third-generation artificial turf compared with natural grass.[23]

The observation that the majority of natural grass surfaces studied were an over-seeded Bermuda grass blend, existing with <46 cm (18 inches) of annual rainfall, <40% humidity in declining temperatures and in an overused multi-purpose environment highlights the challenges in providing natural turf surfaces in environments with a large range in temperature and where water resources are limited. The key point is that natural turf surfaces are geographically and temporally variable in both training and competitive use and so comparisons between synthetic and natural turf will be difficult to control.

The monitoring of injury incidence on natural turf and third-generation artificial surfaces during soccer match play has revealed a differing pattern to that occurring during training. A comparison of injury incidence sustained by men and women over a two-season period during soccer matches on natural turf and third-generation artificial surfaces in North America was performed by Fuller and colleagues.[24] While no major differences were observed between surfaces, it was observed that the most common season-ending injuries for men on artificial turf and grass were a hamstring tear and anterior cruciate ligament (ACL) tear, respectively. For women, a tear of the ACL was the most common season-ending injury on both surfaces. The incidence of ankle sprains in men remained similar on both surfaces, whereas for women there was a significant reduction on artificial turf compared with grass. The study concluded that the overall incidence of injury when playing matches on third-generation artificial turf was similar to that experienced on a natural turf surface.[24]

The comparison of injury incidence during soccer training by Fuller and colleagues[25] over the same 2-year period revealed that for men, mild (4–7 days of training missed) and moderate (8–28 days of training missed) injuries were significantly higher ($p < 0.05$) on artificial turf than on natural turf. For women, however, the incidence of mild injuries was significantly lower on artificial turf than on grass.[25] The most common season-ending injury for men during training was ankle ligament tear, which was significantly more common on artificial turf compared with natural turf. Foot injuries for men were also significantly higher ($p < 0.05$) on artificial turf than on natural turf. On natural turf, the most common injury sustained by men was knee ligament tear. Knee ligament tear was the most common season-ending injury for women on both artificial and natural turf.[25] Thus, whilst similar injury patterns appear to exist on natural turf and third-generation turf during soccer match play, injuries sustained during training differ for the two surface types. In both these studies, the specifications of different synthetic and natural turf surfaces were not specified.

A Union of European Football Associations (UEFA)-funded study by Ekstrand et al.[26] compared reported injury incidence for 290 soccer players at ten elite European clubs with third-generation synthetic turf pitches with 202 players from the Swedish Premier League playing home fixtures on natural turf. The authors concluded that the overall risk of injury on artificial turf was no higher than on grass. However, the study observed significantly higher incidences of ankle sprain during matches on synthetic turf and significantly lower incidences of lower extremity strain compared with natural turf. A reduced tendency towards severe training injuries on natural turf has been suggested to warrant further investigation by the authors.[26] This observation of different patterns for training injuries compared with match injuries is consistent with that of Fuller and colleagues.[24,25] Ekstrand et al.[26] also suggested that further research with larger sample sizes was required to confirm these findings. It was noted that not all third-generation surfaces included in the study met subsequent FIFA quality standards.

A study specifically assessing the injury risk of young (under 17 years) female soccer players (109 league teams; 2020 players) in Norway whilst training and playing on artificial turf (combination of second- and third-generation pitches) and natural turf reported that injury incidences, calculated as the number of injuries per thousand hours of training, and match-play exposure were similar for artificial and natural turf.[27] However, limitations of the study included a lack of control over the specification of both the synthetic and natural turf studied, the maintenance status of all surfaces and limited monitoring of weather conditions.

A comparison between artificial (undisclosed type) and natural turf properties for soccer by Martinez et al.[28] took into account anecdotal opinions from two sets of user groups followed by mechanical assessment of surface properties. User groups were found to prefer natural turf perceiving that impact reduction was higher (supported by mechanical impact test results), ball roll was slower, the surface was more comfortable (reduced heat retention and improved moisture retention) and leg and muscle problems were less frequent compared with artificial turf. Preference for soccer play on natural turf compared with artificial turf was also found by Dick et al.[29] Martinez and colleagues[28] suggested that in order for natural turf characteristics to be reproduced in artificial form, the following criteria needed to be achieved: increased force reduction (based on an artificial athlete test using a flat foot with studs) yielded from first, second and third consecutive impacts of 10% compared with force reduction magnitudes for each impact on existing synthetic surfaces; increased vertical deformation at impact by 5 mm and by 3 mm during second and third impacts, respectively (artificial athlete) and reduced ball bounce by 10% in both dry and damp climates.[28]

The comparison studies available highlight that the playing characteristics and injury patterns on artificial turf are compared against the

benchmark characteristics of natural turf. However, several authors have cited a lack of control over and reporting of natural turf maintenance status, temperature, humidity, soil moisture content and frequency of use.[23,27] The benchmark for these studies has thus been a variable one. The continued use of natural turf surfaces in both training and competition for a variety of sports requires not only guidance on optimum maintenance techniques, but primarily an understanding of what characteristics of a natural turf surface are desirable and how these can be engineered consistently and in a sustainable manner.

The new third-generation turf has gone a considerable way to providing an improved artificial playing surface for some sports traditionally played on natural turf and the numbers of these surfaces will increase globally. However, the unsatisfactory replication of some playing characteristics of artificial turf highlights the importance of maintaining the availability of natural turf playing surfaces. Evidence of variations in injury patterns (whilst not necessarily of a negative nature) when playing on artificial turf compared with natural turf highlights the need for continued and further research into the causal mechanisms that explain the injury on both types of surface, rather than just comparative studies.

1.3 Financial Considerations

The initial capital costs of constructing and maintaining an artificial turf pitch are considerable and where capital resources (and ongoing funds for maintenance) are not available, the importance of and reliance on natural soil and turf surfaces to provide a suitable area for recreational and club sports use within these communities is increased. Just as with synthetic turf, the modern natural turf pitch has been developed significantly in the last 20 years at both the elite and, to a lesser extent, the recreational level of the game. The principal aim in the development of natural turf surfaces has been to improve infiltration and drainage of the surface. Modern surfaces are constructed from high sand content rootzone materials, which are free draining with lower water retention[16] and reduced sensitivity of shear strength to increased moisture content. This increase in the sand content of construction materials is the largest difference between the 1970s and modern surfaces.[30] However, the consequences of using more freely draining, higher sand content materials are significant. In terms of environmental sustainability, there is an increased use of scarce water resources for irrigation and increased use of fertilizer due to lower nutrient retention. In the elite stadium context, such resources are available and necessary to produce the aesthetic qualities required for television in particular and even include the use of enhanced-growth light systems. It is for this reason, however, that such surface construction materials are not suitable for recreational facilities, where such resources are not available. Alternative approaches to providing sustainable turf pitches are therefore required, but not necessarily in an artificial form, depending upon the sport, resources and whether an ecosystem service is considered important.

Recreational surfaces in the UK have developed significantly following investment from lottery funding and professional sport (through organizations such as Sport England and the Football Foundation). For example, since 1995, Sport England has invested £38.3 million in natural turf-related projects and £64.6 million in synthetic turf pitches and multi-use games areas. Since 2000, the Football Foundation has funded 199 natural turf development projects and 163 synthetic turf projects. This is based on an aim to increase access to facilities and increase intensification of use of facilities. The demand for quality surfaces has risen with population increase, increased pressure on land in urban spaces and increased expectations of surface quality from participants through the television images of surfaces. The challenge for providers such as local authorities is to provide quality facilities that can sustain intensified use without increased risk of injury within budgets that are often limited or have competition for resources.

1.4 New Natural Surfaces

The demand for hard-wearing surfaces that do not increase injury risk has resulted in a significant change in mechanical properties, in particular increased stiffness (important in ball- and player-surface interactions) and shear strength (important in player-surface traction). This is reflected in the fact that whilst minimum values for traction are reported in the performance quality standards for natural turf of both the English Football Association and the Institute of Groundsmanship, maximum values are not reported. This is an historical anomaly; the concept of injury from high traction was not considered in the past because creating surfaces with sufficient traction for player stability was the principal challenge facing grounds staff.

Thus, over the past 20 years, there has been a significant change not only in the nature of synthetic turf surfaces, but also in the development of natural turf surfaces. New natural turf surfaces meet the requirements of the players for faster, higher traction surfaces, reflecting the increased fitness, strength and speed and more advanced technique developed over the same time period. With increased surface stiffness, player energy cost is reduced[31] and speed differentials between players are increased. Furthermore, increased uniformity of surface quality allows improved technique development, as ball/equipment behaviour becomes more predictable. In parallel with this, the fitness, speed of movement[32] and turning, and equipment of players have changed. To ignore these developments without assessing the increased risk of injury is foolhardy. The highest value players are playing on stiffer surfaces, and the majority of recreational players are playing on different surfaces with the same or different equipment. To study the change in injury risk requires an integrated investigation in order to understand the changing nature and properties of natural turf surfaces as player performance, movement and equipment also develop.

Advancement in natural turf pitch construction and engineering continues to be required to provide sustainable sports pitches for competitive and training purposes in sports such as rugby, cricket and golf where the characteristics of these games are generally not suitable for current artificial turf surfaces to be used. The initial capital costs of constructing and maintaining an artificial turf pitch are also too much for many providers. Thus, the importance and reliance on natural turf to provide a suitable area for recreational and club sports use within these communities is increased.

2. Relationships between Sports Surfaces, Biomechanics and Injury

A relatively large amount of research has been published on the mechanical properties of artificial sports surfaces and human interaction with these surfaces,[14,33-37] compared with research documenting assessment of natural turf.[38-41] In general, the available research does not point towards a preferential use of either natural or artificial surfaces with regard to their respective associations with injury prevalence.[23-29] Interpretation of findings is complicated by the varied properties of both natural and synthetic playing surfaces utilized in different studies. For example, early work studying synthetic surfaces was based on first-generation surfaces, which, as previously noted, have distinctly different properties to recent third-generation pitches. Results from earlier studies are still relevant to this review as many synthetic pitches currently used for multi-sports use are similar to second-generation synthetic turf sports pitches, certainly in the UK. For school and community artificial pitches it is also not always appropriate to install a third-generation surface because whilst suitable for soccer play, it is not possible to play tennis or field hockey, and consequently multi-sport use is restricted. Artificial turf pitches resembling the older second-generation turf designs are thus still in production, although with improved shock absorbent layers and a new generation of short piled, sand-dressed surfaces for field hockey.

Increased levels of impact,[42-47] altered joint movement patterns,[48,49] an increase in eccentric muscle activity[50] and differences in resistance to

sliding[46,51] are mechanisms that have been suggested to facilitate an observed increase in injury rates with the increased use of older generation artificial turf surfaces in sport.[46,52-54] However, a direct cause-effect relationship has not been established between increased artificial turf use and a particular type of injury.

2.1 Comparison of Surfaces

Evidence of natural turf injury analysis in the literature is relatively sparse. One of the first studies to consider the impact of artificial turf on injury[7] reported that studies have shown natural turf to yield a lower number of injuries compared with artificial turf, citing that deformation of the surface was the most variable and perhaps beneficial factor for natural turf over artificial turf. While perhaps not yielding as many injuries as artificial surfaces, injuries still occur on natural turf. A prospective study on the aetiology of soccer injuries reported that 24% of injuries were correlated with playing surfaces (Ekstrand[55] in Nigg and Yeadon[14]). It was assumed that features of a natural turf surface such as uneven playing ground, hardness and inappropriate friction characteristics were connected with injury prevalence.[55] A comparison between injury rates based on tennis surface type using elite male players revealed that competing on grass yielded a higher frequency of player medical treatments compared with hardcourt or clay.[56] Tactical and surface-enforced differences were suggested by the author to elevate injury risk when playing on grass (reduced sliding, lower ball bounce, variations in ball speed) compared with clay.

2.2 Traction

Hardness and traction on natural turf have been cited as the two main surface characteristics that may be related to injury incidence.[57] Accidental or acute injuries as a result of inappropriate magnitudes of traction on natural turf sports surfaces have received some attention in the literature. Efforts to reduce the amount of surface wear during sports use through soil material reinforcements, in the form of polyester nylon meshes near the soil surface, have been linked with an increased risk of ankle and knee joint injuries if feet become locked in the mesh.[58] The type of grass used to construct pitches also influences traction.[57,59] Traction on the soccer pitch is a function of soil type, soil density, grass root density, soil moisture content and shoe-surface interaction (influenced by the choice of stud pattern). For example, Bermuda (*Cynodon dactylon*) grass is suggested to result in greater shoe-surface traction compared with perennial ryegrass (*Lolium perenne*).[59] By the nature of its growth, Bermuda grass contains horizontally creeping stolons, which form a surface mesh that increases resistance to wear and also traction compared with perennial ryegrass, which is non-stoloniferous.[59]

Although shoe-surface traction is frequently cited in the literature as an important consideration in the cause and prevention of injury,[6,60-62] optimal recommendations of shoe-surface frictional characteristics have been difficult to determine.[46,51] According to Stucke et al.[51] variations in the amount of friction required depends on what type of movement is occurring between two contacting bodies (dynamic friction). Sports movements often require a static component of friction whereby movement suddenly ceases or begins from a position of rest. In these cases of static friction, the relative movement between two contacting surfaces is zero and this allows large horizontal ground reaction forces to be generated in either a stopping (decelerating) or accelerating movement. Typical vertical (Fz) and horizontal (Fy) ground reaction force data are presented in figure 1 from a participant accelerating from rest, on a natural turf surface while wearing studded footwear (soccer boots). The force-time history demonstrates the requirement of relatively large horizontal forces (approximately 0.6 bodyweights) in order to propel the body forward from a static standing position where the relative movement between the soccer boot and the turf surface was zero.

When the properties of the contacting surfaces are not sufficient to provide coefficients of friction that meet the requirements of the movement

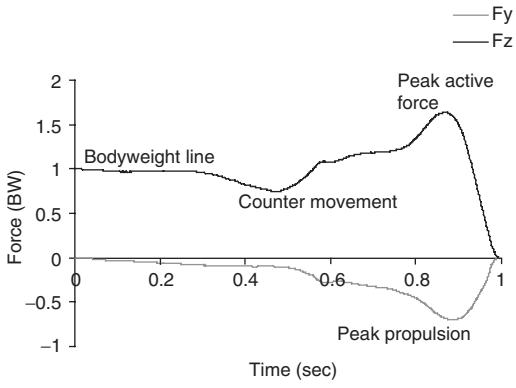

Fig. 1. Typical vertical (Fz) and horizontal (Fy) ground reaction force time histories for a subject accelerating from rest on a natural turf surface wearing studded footwear. **BW** = bodyweight.

(insufficient force-locking connection), spikes in the case of cricket and athletic track footwear or studs (cleats) in the case of rugby, soccer and field hockey provide a 'form-locking connection' between the shoe and surface.[51] These studs aim to improve traction between the boot and surface and also guard against too much traction, which has been cited as a potential cause of non-contact ACL injury of the knee and twisting injuries of the knee and ankle.[63] The configuration and length of these studs have received attention.[59,63,64] However, a definitive conclusion regarding appropriate stud length and configuration to minimize injury occurrence has not been reached. Study in this area is hindered by the ethical considerations of knowingly administering the use of a high traction boot to players during a season in order to test their response, which may be considered reckless.[57] Orchard[57] suggests that modification of the playing surface holds the key to providing players with a universal method of reducing shoe-surface traction and thus reducing the relative risk of injuries related to shoe-surface locking. Whether this is realistic is questionable due to the increased scale of engineering and variability when constructing surfaces in different environments – integrated surface-footwear studies remain critical for reducing injury risk, particularly given the commercial nature of footwear design.

2.3 Seasonal Variation

Climate and weather conditions can have a large influence on the playing conditions of natural turf. According to Australian Football League surveillance, a trend has been demonstrated for higher incidence of ACL injuries on harder natural turf ground compared with softer ground in Australian Rules football.[59,65,66] In a review of the influence of climatic conditions on the ground and the occurrence of lower-limb non-contact injuries in football (including all codes of football: soccer, rugby union, American football, Australian football league), Orchard[57] reported that the majority of studies found an early season bias towards a higher incidence of injury when games were played on natural turf over a typical autumn-winter season (harder ground in the autumn season). In contrast, where football (including all forms) was not played over an autumn-winter season or in subtropical climates, an early season bias was found to be non-existent. The variation in ground conditions (traction and hardness differences in autumn) during a typical autumn-winter season was suggested to be partially responsible for the early season injury bias across all codes of football.[57]

A comparison of injuries incurred during two old winter season fomats (typically August to April) of English rugby league, a new condensed winter league (August 2005 to January 2006) and the subsequent new summer season of rugby (March to August, 1996) has been performed.[67] This assessment of seasonal variation on injury incidence revealed an increased incidence of injuries per 1000 hours of match play over the four playing seasons studied. Specifically, the new summer season, which involved the least number of games played compared with previous seasons, incurred the highest number of injuries albeit ones that were less severe and required less surgical intervention compared with previous seasons. The researchers suggested that the significant increase in injury rates during the summer season compared with the previous condensed and continuous autumn-winter seasons were due to warmer weather where pitch drying conditions (evapotranspiration > precipitation) prevail, with resultant harder

ground conditions.[16,67] Unfortunately, measurement of surface parameters was not reported in this study. A greater integration of injury monitoring, mechanical characterization and biomechanical studies should improve our understanding of injury and weather and climate effects on natural turf pitches.

Approaches to managing the changes in natural turf as a result of climate and weather conditions have been suggested. Orchard[57] reported that shoe-surface traction on natural turf pitches was likely to be higher when the ground is hard, dry and the grass cover and root density are at their greatest. In the early part of an autumn-winter football season, pitch conditions are likely to be harder and drier. Orchard[57] concluded that measures taken to reduce shoe-surface traction should be employed including adequate watering and softening of the ground in the early part of the season, consideration of moving the season of play to use more of the winter months, increased use of natural turf (perennial ryegrass, *Lolium perenne* L.) as opposed to playing on artificial turf and the adoption of boots with shorter studs when playing on hard ground. Aeration and loosening of the soil (the process of reducing soil bulk density and increasing air-filled porosity by the action of inserting solid tines or rotary blades) are also proposed. This is resource dependent as materials, equipment and finances are required to manage surfaces in this way. For example, as competition for water increases, the ability to provide management of surfaces in this way could be limited, particularly at sub-elite levels of sport.

3. Quantifying Player-Surface Interaction

The player-surface interaction is a complex function of surface mechanical factors, human perception and human biomechanical response (both voluntary and involuntary). The interaction is two-way, the surface appearance and mechanical behaviour modifies the human biomechanical response, which in turn loads the surface, resulting in deformation that can change the surface behaviour and appearance. This interaction is variable in time (due to factors of player stamina, variable environmental conditions, cumulative use effects), in space (due to differential usage as part of the nature of the sport played), with the level of sport played and the age/size of the participants. To evaluate surface-related injury risk, it is necessary to quantify this interaction in terms of both surface mechanical and biomechanical parameters. Historically, the techniques to evaluate such parameters have been developed independently, but an integrated approach is proposed as necessary for understanding injury risk, surface performance and player performance simultaneously.

3.1 Surface Mechanical Testing

The development of methodologies for the testing of surface mechanical properties has been driven by: the need to benchmark synthetic turf surface performance against natural turf in the improvement of synthetic surfaces; and, the development of performance quality standards for the specification and improvement of natural turf surfaces.

Mechanical testing methodologies for natural turf can be divided into three principal groups: (i) ball-surface interaction; (ii) surface performance and aesthetics; and (iii) player-surface interaction. Ball-surface interaction tests comprise vertical and angled ball bounce behaviour from standard heights and initial velocities, and horizontal ball roll characteristics (speed, deceleration and deviation from straight line behaviour). These are important for the quality of the playing experience and are considered a priority for many players.[68] Aesthetics and surface durability will vary according to the nature of the sport played and the maintenance resources for the surfaces – again this is critical for the player and spectator experience. The majority of player-surface interaction tests are for friction/traction or hardness parameters designed to simulate loads applied by the human during sports movements. Traction is measured either by rotation or linear test devices. Linear sliding devices such as the sliding traction test measure the distance a weighted studded boot travels on a trolley when supported[69,70] or the force required to move a studded sled in a

single direction. Such linear tests are thought to be analogous to the type of traction required when moving and stopping in a straight line, but a single test assumes the surface is isotropic and tests should be performed in different orientations with respect to the surface being tested. Rotational tests typically comprise a studded plate, weighted to represent a static human that is rotated using a torque wrench that traditionally measured peak rotational force,[33,71] but have been adapted to log torque continuously as the device is rotated.[62] The continuous measurement of torque has allowed the rate at which torque is developed to be quantified – termed rotational stiffness – revealing differences between surfaces that were not evident when comparing peak torque alone.[62] A number of devices, such as the Pennfoot apparatus[72] and Strathclyde Sports Turf Testing Rig[73] incorporate the recording of both linear and rotational tests in one device.

The described mechanical tests are simplifications of the player-surface interaction. In 'real' player-surface interactions, the orientation of the principal stress axes is rotated during the movement, the loading-rates are variable and the magnitude of the stresses is variable. In most surface characterization tests, the surface is stressed in only the surface-normal and parallel directions using either constant velocities or dynamic loads applied from constant heights to maintain constant energy. Biomechanical subject-based research can characterize the stresses applied to a surface in both space and time and from this an understanding of injury development and risk for different player-surface interactions can be established. Subject testing of surfaces is difficult, however, as the stress paths are variable from subject to subject and within subjects over time, and therefore do not provide a suitable basis for surface characterization or for governing-body-led accreditation schemes such as those of international governing bodies for football (soccer) [FIFA[10]], field hockey (Federation Internationale de Hockey[74]) and lawn tennis (International Tennis Federation[75]).

Determination of the hardness and stiffness of surfaces is important for the understanding of impact-related injuries, whether to the leg or the head. Test devices include 'Artificial Athletes' such as the Berlin and Stuttgart Artificial Athletes; cylindrical missile drop-test devices of varying complexity such as the ASTM F1702 device; and dynamic plate tests, which include the lightweight deflectometer. In reviewing all these devices, Young and Fleming[76] identified that all have limitations in their replication of actual player loading. However, a key requirement was for future devices to be designed to incorporate a range of loads, contact areas and load durations to be able to measure the surface response to typical stress paths applied to surfaces of a particular type, particularly where surface materials are known to be non-linear. There are continued efforts to develop test devices that are more complex and realistic in order to test surfaces, for example the force-controlled traction device of Carré et al.[64]

There is a potential difference in the requirements of sport injury and sports surface engineering research and the requirements of sports governing bodies, manufacturers, etc. to characterize surfaces for player safety and quality of play (often termed 'playability') in the field. Complex test devices are small in number and prohibitively expensive for wide-scale testing of surfaces at current prices. Therefore, simplified test devices and methodologies are required; a key question is to what extent the simplifications are valid. The challenge for engineering is to develop test devices that balance the requirements for accurate, representative data to ensure data quality and validity, with affordability and portability to encourage extensive and frequent data collection.

3.2 Surface Biomechanical Testing

There is a scarcity of biomechanical research in the field or laboratory that has involved natural turf. The nature of analysis tools used in biomechanics are not always appropriate for sports-specific analysis in the field.[77] Integrating natural soil media and sustaining turf growth in

the laboratory environment complicates research into human interaction with natural surfaces, making natural turf-specific equipment difficult to assess and resulting in the assessment of equipment in an inappropriate environment. For example, previous assessment of studded soccer boots has taken place in the laboratory using artificial turf rather than natural turf.[40] Ideally, shoes should be assessed on the surface for which they are intended and vice versa.[78]

Biomechanical quantification of surface cushioning has included the use of force platforms, pressure insoles and accelerometers. Whilst there have been some attempts to site force platforms below natural turf in the field,[79,80] these have been focused on the provision of an appropriate environment for footwear testing, rather than the specific testing of natural turf surfaces. The recent application of pressure insoles placed within footwear to measure loads at the foot plantar surface has provided a more practical methodology for measurement of cushioning than the siting of a force platform within the turf surface. Patterns of plantar pressure distribution during soccer-specific movements have been assessed in the field on natural turf and red cinder surfaces.[41] The researchers highlighted a lack of soccer running pressure data in the literature and thus could only compare their soccer-specific data to running activity pressure data. A study comparing forefoot plantar pressure wearing three different soccer studded boot models while running on a treadmill and natural turf has highlighted the importance of testing footwear on appropriate playing surfaces.[39] This research found that treadmill running did not reflect the forefoot loading patterns derived when running on natural turf, as cleats were not able to penetrate into the surface. The researchers therefore suggested that analysis of cleated footwear should be undertaken on surface conditions for which the boots are intended. Peak pressures under metatarsals one, two and five were found to be related to foot landing characteristics and surface properties rather than the location of cleats below these aspects of the foot, thus potentially indicating a need for footwear design for different surfaces.[39]

Tillman et al.[81] used pressure insoles to compare resultant ground reaction force for asphalt, concrete, a running track and natural turf. These authors detected no difference in loading between the tested surfaces, concluding that surface stiffness was not directly linked to injury risk through loading. This study provided a useful demonstration of the potential of pressure insoles to allow comparison of surfaces in the field, but did not utilize the full potential of pressure insoles – to provide detail on the distribution of force over the foot plantar surface. More recently, Ford et al.[82] compared a synthetic turf surface with natural grass using in-shoe pressure distribution. These authors reported peak pressure and relative load at nine plantar regions of the foot during cutting movements. They observed differences in loading for the two surface conditions, with greater peak pressures at the central forefoot and lesser at the toes for the synthetic turf and greater relative load at the medial forefoot and lateral midfoot for the grass surface. This comprehensive assessment of pressure distribution highlights the potential of pressure data to detect different surface cushioning. To improve our understanding of natural turf, different turf surface type and conditions require testing. Work has commenced in this area with comparison of pressure distribution for different soil densities.[83] Further studies using this technology should contribute to our increased understanding of natural turf surfaces.

The measurement of horizontal forces has taken place on natural turf surfaces to indicate traction behaviour. An analysis of horizontal forces in soccer boot studs while performing sports-specific movements (accelerating from rest, inner and outer zigzag and turning movements) has been performed.[84] While not detailing what surface these movements were performed on, the researcher suggested that a diverse pattern of stud configuration was required based on the utilization (maximum loads and direction of force applied to studs) during the range of movements. However, an assessment of shoe-surface traction using artificial and natural soccer surfaces and cleated boots concluded that aggressively cleated boots were not

recommended due to their high resistance to rotation during cutting manoeuvres and subsequent risk of injury particularly of the ACL.[85]

There are few examples of analysis of movement patterns (kinematics) on natural surfaces. A study of cutting manoeuvres on artificial and natural turf has taken place in the field using movement speed as a measure of grip performance between shoe and surface and video analysis to provide information on boot-surface slip pattern.[38] An analysis of body and limb accelerations for a variety of surfaces in the field, including indoor and outdoor artificial turf and outdoor natural turf, has been performed to assess whether the characteristics of soccer-specific movement techniques are adapted for different surface conditions.[86] Maximum shank and pelvis acceleration were found to be similar between natural and artificial turf. The authors concluded that the findings were useful indicators of a comparable injury risk (assuming accelerations are correlated with injury prevalence) across all surface conditions.[86] Some initial kinematic findings have also been presented from participants running on a variety of natural turf surfaces in the biomechanics laboratory.[87] Initial and peak ankle and knee flexion angles during running appear to be maintained at similar levels even with distinct mechanical changes in turf type. Analysis of movement kinematics in addition to running, however, remains to be performed.[87]

3.3 Integrated Studies

The mechanical devices described in section 3.1 fail to incorporate the complexities of human movement and therefore do not consider the influence of a variety of human movements on the behaviour of shoe-surface materials and frictional coefficients during a repertoire of available movements in sport.[51] Biomechanical human assessment of surface frictional properties using stopping, starting and turning movements has been performed;[51] however, the inclusion of natural turf undergoing a repeated range of tests remains a rarity.

Some work has been performed that used biomechanically validated magnitudes of vertical, shear and torque loads[88] within a portable mechanical testing rig to characterize properties of a sports surface including linear and rotational traction, vertical impact and a combined vertical, shear and torque impact test.[73] This research found that traction coefficients and peak torque were lower on a 3G surface (third-generation artificial turf) than on a natural grass pitch.

Player surface interaction is two-way and studies that provide detail of the mechanism by which variations in human movement affect natural turf characteristics and performance are lacking. While the sports performance aspects of a surface can be assessed (ball bounce, ball roll, ball speed etc.) in relation to the composition and temporal characteristics of soil media, relatively little assessment of how the natural turf surface parameters affect the athlete have been performed, let alone the affect that variations in athlete movement can have on the surface. An integrated study investigated the effect of changing a soil surface from a soft to a hard condition by simultaneously measuring pressure distribution within the soil and the shoe when running.[83] An increase in the dry bulk density from 1460 to 1590 $kg\,m^{-3}$ resulted in an increase in peak G of 125 to 235 g measured using a 0.5 kg Clegg Hammer. Peak heel force was significantly lower for the lower density soil condition, but vertical stress distribution within the surface only varied significantly with depth (100 vs 200 mm depth), not between soil densities as predicted by a linear elastic model of soil behaviour. Similarly, Stiles et al.[89] and Stiles et al.[90] reported pilot data on the inclusion of turf surfaces of contrasting soil types (and therefore mechanical properties) into the traditional biomechanics laboratory environment and showed significant differences in rates of loading between different surface materials in both running and turning movements. In the study of Stiles et al.,[90] the turf conditions were carefully controlled and monitored. Such integrated studies and the use of novel laboratory environments allow the stresses on the human and the surface to be analysed simultaneously. Ideally the loads experienced at each level of the performer/shoe/surface system

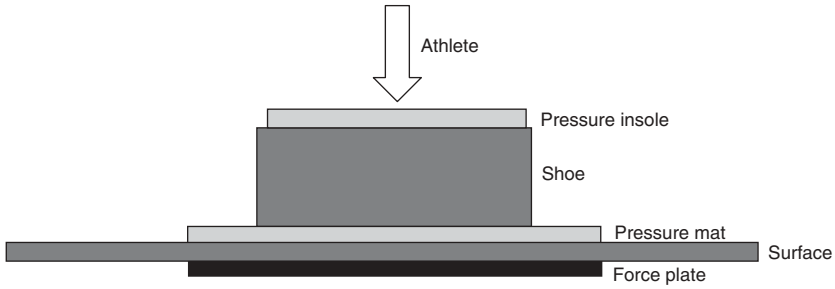

Fig. 2. A model to depict layers of the player-shoe-surface system between which loads should ideally be quantified.

(figure 2) should be quantified. Thus, better informed assessments of new or existing surfaces can then be made with regard to their effect on the player and surface performance (figure 3).

4. The Case for Further Research

Participation in sport and exercise activities yields important health and social benefits for the individual and reduced dependence on community primary care health provisions.[2] Promotion and attainment of a healthy nation can be aided by appropriate sports facilities that are affordable and safe. Benefits to health and society can be gained via participation in traditional, competitive sports such as hockey, football, tennis, rugby, cricket and lacrosse at school, club and elite level. Sports such as tennis, hockey and to some extent soccer have benefited from incorporating artificial surfaces into the game as they provide year-round playing opportunity. The influence of adverse weather conditions on the performance of a surface is also less when playing on artificial surfaces compared with natural turf. An artificial turf surface also requires a lower level of maintenance, can be constructed within a relatively small space and can tolerate regular multi-sport use compared with a natural turf surface.[13-15]

Artificial sports surfaces have made an important contribution to the provision of functional sports surfaces and increased sport participation. Currently, however, artificial turf surfaces do not adequately replicate the playing properties of all natural turf surfaces and thus are not suitable for every sport. There is therefore a need to continue to develop natural turf surfaces, the reasons for which are (i) the protection of green spaces and playing fields in the built environment is critical for urban ecosystem functioning; and (ii) the preservation of fundamental playing characteristics for sports such as soccer, rugby, golf, cricket and lacrosse, which are not well suited to play on any generation of artificial pitch, is paramount. There have been significant changes in natural turf properties over recent years in keeping with player requirements for a faster surface and higher traction component. The number of synthetic turf surfaces is increasing; however, a significant number of participants continue to play on natural turf at all ages and levels of sport. The sustainability of natural turf surfaces and the risk for injury they pose to players needs to be understood. Thus, further research is required.

Figure 4 illustrates a conceptual framework for further research. Soil physical properties vary in space and time;[91] the variability in natural sports surfaces needs to be understood and with the development of new construction techniques for natural turf surfaces using more frictional and reinforced soil media, the assumption that natural surfaces are a lower risk for participant safety needs to be revisited. It is hypothesized that there is a range of shear strength for minimum injury risk, which may or may not coincide with the optimum shear strength for surface performance. The relationship between stiffness and injury risk is different and is shown in grey in figure 4 to illustrate the lack of evidence as to whether stiff surfaces pose a greater or lower risk of injury than a compliant surface; the

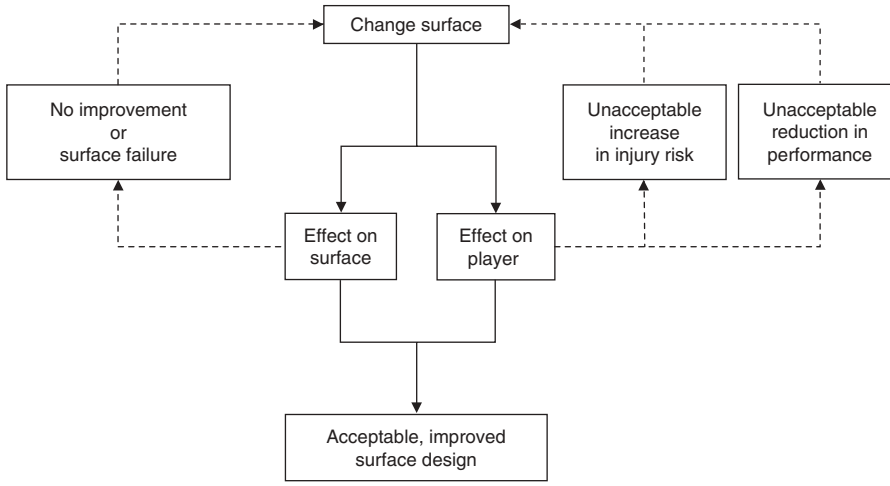

Fig. 3. A model for integrated development of surfaces. Any change in surface properties should be evaluated in terms of both the effect on the surface and the effect on the player (in terms of injury and performance). The model responds to negative feedback until an acceptable improved surface design can be determined – without detriment to the surface or player.

relationship for surface quality is more easily identified. There is also significant variation in compliance and shear strength properties among different sports surfaces.[21,92-94] The exact location of boundaries between areas in the diagram in figure 4 are not known, but are hypothesized to vary with construction material, type of sport, footwear, player characteristics and moisture content. Future research is required to identify boundary locations between areas on the diagram for different combinations of the above factors, e.g. pitch construction specification – a clay soil will have a narrower envelope of ideal traction compared with a sandy soil due to increased sensitivity of shear strength to moisture content, as discussed in section 1.3.

Initially, it will be important to obtain biomechanical data (e.g. ground reaction forces,

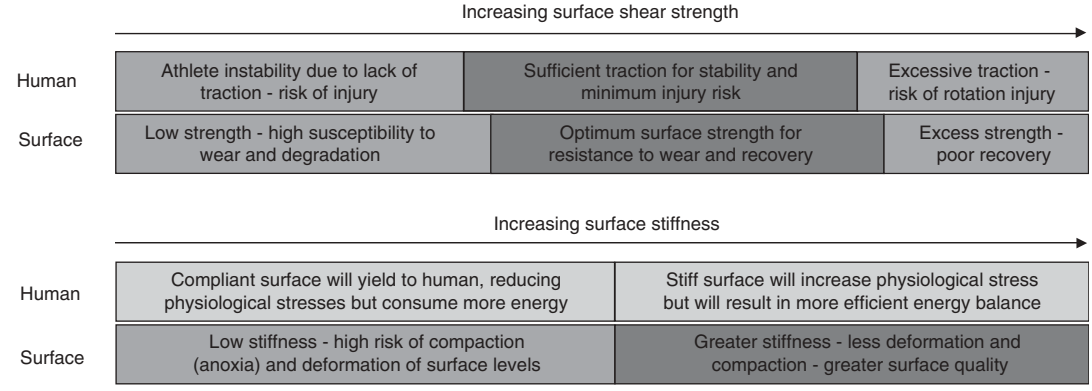

Fig. 4. A conceptual framework for natural turf research with the aim to quantify the boundaries to the shear strength and stiffness envelopes of optimum surface performance (in green) – two key mechanical properties governing player- (and ball-) surface interaction in natural turf. Red zones identify areas of risk to player or surface. Grey zones identify a particular uncertainty relating to surface stiffness and player interaction that requires further research. Note that performance is a third dimension and generally as shear strength and stiffness move towards extremes, ball-surface interactions will be adversely affected, although the same cannot be said for player performance, and both will vary with sport.

insole pressure data and kinematic data) that enable a number of sport-specific movements performed on a natural turf surface to be characterized. Running is an integral activity for soccer, rugby, lacrosse and cricket. Additional movements that occur during these games include accelerating from rest, stopping and turning manoeuvres. During a 90-minute game of professional soccer for example, it has been stated that each player performs approximately 50 turns.[95] This activity is therefore of relevance if the nature of turf wear and degradation, and its subsequent impact on surface mechanics and player interaction, is to be understood fully.

5. Conclusions

A 'natural turf surface' encompasses a range of soil and grass materials that combine in a complex interaction that varies in space and time; to characterize a surface as natural turf is as inappropriate as to label a surface as 'artificial turf' without specifying the length of pile, the plastic material used for the fibre and the type of granular infill used for shock absorbency. Future injury studies must characterize the nature of natural turf and its variation in space and time through the study.

Complementary future research should endeavour to utilize an integrated approach using engineering and biomechanical expertise that will permit greater understanding of factors that influence natural turf wear and degradation and factors that will influence how the athlete responds to changes in natural soil media and mechanisms behind injury patterns. This is a two-way interaction that is considered to hold the key to the future development of a more sustainable natural turf surface for training and competitive use for sports whose characteristics are not suited to play on artificial turf surfaces. Furthermore, such research would inform the continued development of synthetic turf.

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