

# The physiological regulation of pacing strategy during exercise: a critical review

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## ABSTRACT

The regulation of the pacing strategy remains poorly understood, because much of classic physiology has focused on the factors that ultimately limit, rather than regulate, exercise performance. When exercise is self-paced and work rate is free to vary in response to external and internal physiological cues, then a complex system is proposed to be responsible for alterations in exercise intensity, possibly through altered activation of skeletal muscle motor units. The present review evaluates the evidence for such a complex system by investigating studies in which interventions such as elevated temperature, altered oxygen content of the air, reduced fuel availability and misinformation about distance covered have resulted in alterations to the pacing strategy. The review further investigates how such a pacing strategy might be regulated for optimal performance, while ensuring that irreversible physiological damage is not incurred.

Pacing strategy has been described as the efficient use of energetic resources during athletic competition, so that all available energy stores are “used before finishing a race, but not so far from the end of a race that a meaningful slowdown can occur”.<sup>1–3</sup> This description has recently been extended to include the regulation of other physiological variables, including the rate of heat storage and thus body temperature,<sup>4–7</sup> into the role of the appropriate pacing strategy during self-paced exercise.

This definition holds that during self-paced exercise, when the athlete is able to select an exercise work rate, performance is regulated to prevent changes in physiological systems that may be limiting or detrimental to performance. This regulation of work rate provides a challenge to classic models of fatigue and the factors that are considered to limit exercise performance.<sup>8–9</sup> For example, during endurance exercise in hot and humid environments, it has been established that fatigue and a resultant decrease in force production, running speed or power output occurs because the body temperature rises to reach critically high levels, above which volitional exercise is not possible.<sup>10–13</sup> The proposed mechanism for the failure to maintain force output is that skeletal muscle motor unit activation by the brain is reduced above this critical core temperature of approximately 40°C.

In contrast to that dogma, studies of self-paced exercise have found that motor unit activation and performance are reduced before the body temperature reaches critically high levels.<sup>4–6, 14</sup> For example, isometric force production and voluntary activation percentage decrease progressively during a

passive heating protocol, even at body temperatures below 39°C.<sup>14</sup> In 2005, Todd and colleagues<sup>15</sup> showed a reduction in force output due to a failure of voluntary drive during passive heating, despite the availability of additional motor cortical output, which would, in theory, allow an increased force output. African runners maintained higher running speeds than white Caucasian runners during self-paced 8 km running trials in the heat, despite core temperatures that were not significantly elevated or even different from those of the white runners.<sup>4</sup> Finally, it was recently found that cycling power output and integrated electromyographic (iEMG) activity (an indirect measure of the activation of skeletal muscle motor units) are reduced in hot (35°C) conditions compared with cool (15°C) conditions during a 20 km cycling time trial, despite rectal temperatures that are similar between conditions and well below the proposed critical level of hyperthermia.<sup>5</sup>

Collectively, these studies provide evidence for a pacing strategy that is regulated in advance of, in this case, the attainment of a critically high body temperature. It has been suggested that the pacing strategy is a key component of a proposed tele-anticipatory system,<sup>16, 17</sup> in which the brain anticipates the endpoint of exercise (telos = “end”) and then regulates exercise intensity and alters the adopted pacing strategy specifically to ensure that potentially catastrophic derangements to homeostasis do not occur.<sup>18–21</sup>

The aim of this review is to examine the regulation of pacing strategy during both short duration and long duration self-paced exercise bouts. We examine studies in which various interventions such as high temperatures, hyperoxia, hypoxia and altered substrate availability have been found to influence the adopted pacing strategy. Based on these findings and recent novel work suggesting that the subjective rating of perceived exertion (RPE) is a key mediator in the regulation of work rate, a model is proposed that aims to explain how exercise intensity is altered both to optimise performance and prevent potentially harmful disturbances to any physiological systems.

## STUDIES OF PACING STRATEGY

Pacing strategy can be examined in one of two ways. The first method is the observation of selected exercise intensity during either laboratory time trials or competitive events. Because pacing strategy is dependent on external factors such as environment, race situation and the influence of other competitors, these studies may not necessarily reflect optimal pacing strategy for a given set of

circumstances. However, they do allow factors such as ambient temperature,<sup>4,6</sup> oxygen content of the inspired air<sup>22-24</sup> and expected exercise duration<sup>25</sup> to be manipulated, and the potential physiological mechanisms that could explain any alterations in pacing strategy and performance induced by these changes can be postulated.

In the second method, the pacing strategy can be experimentally altered by forcing the athlete consciously to begin a trial at a pace that is either faster or slower than the self-selected pace.<sup>1 26-29</sup> This allows the effects of experimental manipulations on overall performance to be studied, and inferences can be drawn regarding the optimal pacing strategy. As a result of the difficulty in accurately controlling the exercise intensity for long periods, such experimental interventions are typically used for shorter duration exercise (less than 4 minutes) only. A variation on experimental manipulations of the pacing strategy is the use of computer models that predict pacing strategies during competitions.<sup>2 30-32</sup> These computer models are used to simulate competitive events and they generate predicted split times that can then be compared to actual performances.

### INFLUENCE OF EXERCISE DURATION ON OPTIMAL PACING STRATEGY

Both the observed and experimentally determined optimal pacing strategy are strongly influenced by exercise duration. In shorter duration events (<4 minutes), a typically selected strategy usually involves a fast start, with power output declining progressively until completion of the trial.<sup>1 25 33</sup> During a 1500 m cycling time trial (approximately 2 minutes), for example, power output and velocity peak in the first 300 m, and then decrease progressively so that the lowest velocity is recorded at the completion of the trial.<sup>1</sup> Exercise trials such as the Wingate Anaerobic test, classically used to assess anaerobic (oxygen independent) exercise performance, show a similar pattern, with power output decreasing by almost 50% from the start to the finish of a 30-s trial.<sup>25</sup>

Analysis of the sprinting events (100 m, 200 m and 400 m) at the 7th International Association of Athletics Federations World Championships of Athletics showed that every single athlete adopted a positive pacing strategy, in which the highest speeds were achieved early on during the event, with a progressive slowing down until the finish line.<sup>33</sup> These results are in agreement with computer simulations and studies in which the pacing strategy has been manipulated, because they support the notion that for shorter duration events, optimal pacing requires a fast start, even if this results in a reduction in velocity at the end of exercise. Recently, Tucker *et al*<sup>34</sup> evaluated the pacing strategies employed during track world records for events from 800 m to 10 000 m. It was found that for the shorter 800 m event, the typical pacing strategy involved a fast first lap, with a significant decrease in speed on the second lap. That this occurred in 26 out of 28 world records suggests that the optimal strategy involves a significant slowing down in the second half of the event.<sup>34</sup>

As exercise duration increases (>4 minutes), the self-selected pacing strategy becomes more even, and during longer duration exercise, self-selected pacing is characterised by the ability to elevate power output or running speed significantly at the end of the event.<sup>4-7 16 34</sup> More specifically, these events typically begin with a relatively high power output, followed by a reduction in power output during the middle part of the trial or race, before power output increases significantly towards the end of the exercise bout, a phenomenon termed an

endspurt.<sup>5 35 36</sup> The physiological significance of this characteristic pacing strategy is discussed subsequently.

To our knowledge, only one study has examined pacing strategy during a single bout of ultra-endurance exercise (>6 h).<sup>37</sup> That study found that running speeds were relatively constant for the first 50 km of a 100 km running race, but then declined progressively over the second half of the race, resulting in an overall positive pacing strategy. The best athletes showed the smallest reduction in running speed during the second half of the race. It must also be noted that it is possible that an endspurt was present at the end of the 100 km race, but this was not detected because split times were available for 10 km intervals only.

### PHYSIOLOGICAL BASIS FOR THE OBSERVED OPTIMAL PACING STRATEGY DURING LONG DURATION EXERCISE BOUTS

Exercise work rate typically increases significantly at the end of longer duration exercise bouts.<sup>7</sup> This observation is of physiological significance, as it indicates that the exercise intensity during the middle part of the exercise bout is submaximal relative to the athlete's capability near the endpoint of the exercise trial. The notion that the athlete maintains a submaximal work rate until near the endpoint suggests that exercise is regulated in a complex, feedforward manner, rather than being the result of a catastrophic failure in one or more homeostatic systems.<sup>9 18</sup>

In such a complex system, the observed pacing strategy may fulfil a teleological role, being the consequence of the underlying physiological regulatory processes occurring during exercise,<sup>20 21</sup> while at the same being the means by which homeostasis is regulated. Changes in the external environment that influence homeostatically regulated variables such as body temperature or oxygen availability would thus be expected to induce a change in the pacing strategy.

Numerous studies have examined the effects of changing the ambient temperature,<sup>4 6 7</sup> the oxygen content of the inspired air,<sup>22 23 38</sup> muscle glycogen content<sup>39</sup> and feedback regarding distance or duration<sup>25 36 40</sup> on exercise performance. The following section describes those studies, evaluating the physiological basis for the characteristically observed pacing strategies during endurance exercise.

### Exercise in the heat

#### The critical internal temperature hypothesis

Environmental temperature has long been recognised as a critical factor affecting endurance exercise performance. It is known that hot (30–40°C) conditions markedly impair exercise performance compared with cool (3–20°C) conditions.<sup>10 12 13 41-43</sup> Originally, it was believed that the impairment of exercise performance in the heat was the result of a reduction in skeletal muscle blood flow<sup>41 44</sup> as a result of reduced stroke volume and cardiac output,<sup>44</sup> due to the challenges imposed on the circulatory system by the hot environment, in particular the need to perfuse both working muscle to maintain the power output and the skin in order to thermoregulate.

However, it is now known that the termination of exercise in the heat is not caused by reductions in cardiac output or exercising muscle blood flow, by impaired substrate availability or utilisation, or by the accumulation of lactate or potassium ions.<sup>10 42 43 45 46</sup> Such fatigue in well-trained individuals has been observed to occur at a core temperature of approximately 40°C,<sup>10 12 13 45</sup> irrespective of the rate of heat storage, the pre-exercise core temperature,<sup>46</sup> or the extent of previous heat

acclimatisation.<sup>45-47</sup> In moderately fit individuals, this “limit” has been established at a rectal temperature of approximately 38.7°C, regardless of hydration or acclimation status.<sup>48</sup>

Symptoms commonly associated with volitional exhaustion during exercise include confusion, loss of coordination and syncope,<sup>49</sup> suggesting the possible involvement of the central nervous system in fatigue. Accordingly, it has been proposed that fatigue during exercise in the heat is associated with a “critical internal temperature limiting exercise performance”,<sup>46-50-51</sup> in which a high body temperature directly affects central nervous functions,<sup>10-49</sup> including a failure to maintain central drive to continue exercise.<sup>12</sup>

In 2001, Nybo and Nielsen<sup>12</sup> showed that force production and voluntary activation percentage in the exercised muscle groups (knee extensors) were lower during a sustained isometric maximal voluntary contraction (MVC) following cycle exercise in hot (40°C, sufficient to raise body temperature to 40°C) than in temperate (18°C, final core temperature 38°C) conditions. Significantly, the overall force produced when electrical stimulation was superimposed upon voluntary contraction was unchanged from values measured during the temperate trial. This indicates that the force-generating capacity of the exercised muscle was unaffected by the elevated core and muscle temperatures after exercise in the heat. It was concluded that exercise-induced hyperthermia caused a form of “central fatigue”, in which elevated body temperature (>40°C) caused reduced central activation of the exercised muscles by the motor cortex leading to a lower force production.

Recent novel research<sup>52-54</sup> has demonstrated a possible effect of hyperthermia on arousal levels (a proxy for motivation or “drive”) by examining changes in the electroencephalographic signal during exercise in hot (40°C) and cool (~19°C) conditions. Subjects cycled to volitional fatigue at a fixed work rate, and the  $\alpha$ -to- $\beta$  wave ratio was measured as an index of arousal levels, with an increase in the ratio suggesting that arousal levels are reduced.<sup>52-54</sup> It was found that the  $\alpha$ -to- $\beta$  ratio increased during exercise in the heat, and this increase was strongly correlated with the increase in core temperature,<sup>52-54</sup> and with the increase in the RPE.<sup>53-54</sup>

The strong linear correlation ( $r^2 = 0.98$ ,  $p < 0.001$ ) between the reduction in arousal (as measured by the increase in the  $\alpha$ -to- $\beta$  ratio) and the increased body temperature<sup>52</sup> is of interest, because it suggests that arousal levels decrease progressively as body temperature increases, rather than simply falling after the core temperature reaches 40°C. Therefore, at a slightly elevated body temperature of 38.5°C, arousal levels were already reduced compared with at a body temperature of 37.5°C. However, because of the study design and exercise modality used in that study,<sup>52</sup> no graded effect of hyperthermia on performance could ever be established. That is, subjects cycled at a fixed work rate until volitional exhaustion occurred. Under such conditions, progressive reductions in motivation or arousal as body temperature increases would have no impact on exercise performance, until such time that arousal/motivation declines to levels at which the athlete is no longer sufficiently motivated to continue exercise and volitionally terminates exercise.

In contrast, when exercise is self-paced, then any reduction in arousal would presumably cause the exercising athlete voluntarily to reduce their exercise intensity even though body temperatures are below the limiting level, rather than waiting until after the body temperature reaches 40°C. The authors did not, however, acknowledge this possibility, suggesting instead that the data supported the notion that impaired performance in the heat is associated with the attainment of a body

temperature of approximately 40°C due to a reduction in arousal levels, leading to reduced voluntary activation levels. This explanation<sup>52</sup> fits a catastrophic model of how exercise performance is regulated,<sup>9</sup> because<sup>18</sup> the conclusion drawn is that fatigue in exercise in the heat is the result of a fall in arousal only after body temperatures had risen beyond critical levels.

Observations that the RPE is strongly correlated with both the body temperature ( $r = 0.98$ ,  $p < 0.001$ ) and to the  $\alpha$ -to- $\beta$  ratio ( $r = 0.98$ ,  $p < 0.001$ )<sup>53</sup> are also noteworthy, for they suggest that the RPE may be an important variable that is progressively influenced by the increase in body temperature. Changes in core temperature and the  $\alpha$ -to- $\beta$  index were the best predictors of an increase in RPE, and this was “associated with increased difficulty to maintain the required exercise intensity”.<sup>54</sup> If the increase in RPE suggested the work rate was becoming more difficult to maintain, then it seems probable that the subjects would reduce the work rate, if they had the opportunity to do so, as during self-paced exercise. This possibility is discussed below.

Finally, there was no change in electromyographic activity during the trials<sup>52-53</sup> and the authors concluded that hyperthermia does not affect the electrical activation pattern of the active skeletal muscles. However, because the trials used a fixed work rate protocol, in which the subject cycled at a predetermined, fixed power output, it may be argued that this finding is expected, and shows simply that the degree of muscle activation required to maintain the power output was not different between hot and cool conditions. Instead, the authors might have concluded that not allowing the subjects to alter their work rate during exercise in the heat resulted in a faster rise in body temperature and a reduced exercise time to fatigue in the presence of a constant, imposed degree of muscle activation.

#### Pacing strategies during self-paced exercise performance in the heat

A different picture emerges when the athlete is able to increase or decrease the exercise work rate volitionally. Cycling power output<sup>6-55</sup> and running speed<sup>4-7</sup> are reduced soon after the onset of exercise in hot compared with cool conditions. Most importantly, these changes occur before body temperatures reach values that are commonly associated with a performance limitation or bodily harm.<sup>12-47</sup>

For example, Tucker *et al*<sup>5</sup> found that power output and skeletal muscle activation were reduced during the first 6 km of a 20 km cycling time trial in hot (35°C) compared with cool (15°C) conditions. The rectal temperatures, heart rates and RPE values recorded at this stage, when pacing strategy began to differ, were similar between the two conditions. As a result of the progressive decrease in power output, the body temperatures in the hot condition remained similar to those measured in the cool condition until the very final kilometre of the trial. In the final kilometre, power output and iEMG activity increased in both conditions. Significantly, the rectal temperature had increased to 39.2°C, but subjects were still able to increase both skeletal muscle activation and power output in this characteristic “endspurt”. In the heat, the activation of skeletal muscle motor units, and thus power output, was reduced as part of an anticipatory regulation of performance, the function of which was to prevent excessive heat storage and increases in body temperature.<sup>5</sup>

A similar anticipatory system was suggested by Marino *et al*,<sup>4</sup> who found that African and white Caucasian runners adopt different pacing strategies in the heat. The smaller African runners paced themselves similarly in hot and cool conditions, whereas larger white runners showed a significant decrease in

running speed in hot conditions, but not cold conditions. As body size is known to be an important determinant of the rate of heat storage,<sup>56</sup> it was proposed that the difference in pacing strategy occurred because the brain was sensitive to the rate of heat storage. As a result, it reduced the running speed of the larger white runners during time trials in hot conditions to ensure that excessive heat storage did not occur.

Morrison *et al*<sup>14</sup> measured voluntary muscle activation during self-paced exercise in the heat. They examined the effect of core and skin temperature on MVC during an isometric knee extension, performed while subjects were passively heated from a core temperature of 37.5°C to 39.5°C and then gradually cooled again to starting temperatures. A 10-s MVC was performed at every 0.5°C increase in core temperature, with two muscle stimulations performed during each MVC to determine the magnitude of voluntary activation of the muscle. Their results showed a gradual and progressive decrease in force production during the MVC and electrical stimulation as body temperature increased, with a subsequent return to baseline with cooling. Therefore, the brain selectively activated a smaller muscle mass throughout the range of increasing body temperatures, rather than only after a “critical temperature” had been reached.

These findings<sup>4 5 14</sup> are inconsistent with a hypothesis that a reduction in skeletal muscle recruitment occurs only after a critically high core temperature is reached. Rather, these data contribute to the growing body of evidence for anticipatory regulation of exercise in the heat,<sup>57</sup> which suggests that during exercise in which force output is selected by the individual and is free to vary, motor command and voluntary activation are reduced incrementally as core temperature rises.

Todd *et al*<sup>15</sup> attributed hyperthermia-induced fatigue to a combination of factors in both the muscle and the motor cortex. They measured a reduction in force output that occurred due to a failure of voluntary drive during passive heating despite the availability of additional motor cortical output, which would, in theory, allow increased force output.<sup>15</sup> Therefore, fatigue occurred in the presence of a “motor cortex” reserve and a motor unit reserve,<sup>12 14</sup> suggesting that the brain may play a regulating role.

Recently, Tucker *et al*<sup>19</sup> found that when the RPE was “clamped” at a fixed, predetermined level during exercise trials in hot and cool conditions, the selected work rate in order to prevent the RPE from rising above the clamped value was regulated differently in the heat. Power output thus declined more rapidly in the hot trial, resulting in a similar rate of increase in body temperature by virtue of rates of heat storage that were similar between the hot and cool trials. It was suggested that the regulation of work rate integrated afferent feedback including the rate of heat storage (including both body and skin temperature), and then utilised the RPE to mediate a regulation of exercise work rate specifically so that the rate of heat storage did not reach excessive levels.

In conclusion, self-paced exercise performance in the heat is impaired before body temperature reaches critically high levels. There may be a centrally mediated mechanism to decrease muscle activation and thus exercise work rate when the rate of heat storage is high early on during exercise, or when there is a risk that the core temperature will rise to limiting levels before the anticipated end of the exercise bout.<sup>4 6 7 57</sup>

### Exercise with different inspired oxygen content

Changes in the oxygen content of the inspired air also alter the pacing strategy<sup>23 38</sup> and skeletal muscle activation patterns<sup>22</sup>

during self-paced exercise. In 1997, Peltonen *et al*<sup>22</sup> examined iEMG activity in seven active muscles during a 2500 m rowing ergometry trial, and found that hypoxia (fractional inspired oxygen (FiO<sub>2</sub>) 15.8%) impaired overall performance as a result of differences in pacing strategy during the trials. That is, force output during maximal rowing strokes decreased progressively during the trials, but the reduction was greater in hypoxia than in normoxia.<sup>22</sup> Furthermore, the decline in force production in hypoxia was accompanied by a reduced iEMG activity, suggesting that the level of muscle activation was influenced by the oxygen content of the inspired air.

Kayser *et al*<sup>58</sup> have provided evidence for this effect, showing that when cyclists were given hyperoxic air at the point of volitional exhaustion in hypoxic conditions, while at the same moment the exercise load was increased, the subjects were able to continue cycling at the higher power output. The continuation of exercise was associated with an immediate increase in the iEMG activity of one of the active muscles (vastus lateralis). The authors proposed that the extent of skeletal muscle activation during exercise is influenced by the FiO<sub>2</sub>.

Interestingly, Peltonen *et al*<sup>22</sup> found that altered pacing strategy and improved performance in hyperoxia were not associated with any differences in iEMG activity compared with normoxia. This finding was attributed to “other factors related to the availability of oxygen”, or due to a neural limitation of muscle recruitment, because “full motor unit recruitment is achieved during normoxia”.<sup>22</sup> However, inspection of the results indicates that the iEMG activity was never greater than 75% of the iEMG activity measured during a maximal effort stroke before the trial, and so skeletal muscle recruitment was clearly submaximal during the trial. It may, however, be that the method of measurement of iEMG in that study,<sup>22</sup> which summed seven active muscles during rowing, was not sensitive enough to detect differences in activation in hyperoxic condition, because only small differences in power output were observed and these differences may have been the result of increased activation of only one of the seven muscle groups that were studied.

Most recently, 20 km cycling performance was improved in hyperoxic (FiO<sub>2</sub> 40%) compared with normoxic conditions.<sup>24</sup> The improved performance was associated with a different pacing strategy, in which the power output was maintained throughout the trial in hyperoxia, but decreased over the course of the trial in the normoxic condition. In both conditions, a significant increase in power output and skeletal muscle activation levels (measured as iEMG activity) occurred in the final kilometre of the time trials. Furthermore, the skeletal muscle activation levels were maintained at higher levels in hyperoxia than in normoxia, which was interpreted as an indication that the increased availability of oxygen enabled a higher degree of muscle activation and thus power output in hyperoxia than in normoxia.<sup>24</sup>

The theory that muscle activation levels and exercise intensity are regulated differently in hypoxia and hyperoxia does not discount the observation that peripheral factors such as changes in metabolite levels may result in “myographical signs of muscle fatigue”, in which the measured iEMG activity increased despite no change in force production.<sup>58–60</sup> For example, Taylor *et al*<sup>59</sup> found that iEMG activity was greater during submaximal cycling at a fixed power output in hypoxic (FiO<sub>2</sub> 11.6%) compared with normoxic conditions, suggesting that the force-generating capacity of the muscle was impaired in hypoxia. Furthermore, the ratio of force/electromyographic activity decreased progressively during exercise in hypoxia. This

suggests that an increase in motor unit recruitment was required to maintain the power output due to a progressive reduction in muscle force generating capacity.

However, the critical point is that power output and iEMG activity can be increased volitionally at the end of exercise time trials in hyperoxia or hypoxia,<sup>7 22</sup> indicating that the reduction in power output during the middle part of the trial is not solely due to a failure of muscle contractility, but must be part of a regulated process because a greater power output could have been achieved with a greater level of muscle recruitment. These studies thus provide evidence for regulation of the degree of motor unit recruitment, which is sensitive to the oxygen content of the inspired air.<sup>22 58</sup>

### Energy substrate availability

Substrate availability is often implicated as a limiting factor during exercise performance. Volitional fatigue during exercise at a constant workload is often thought to coincide with muscle or liver glycogen depletion.<sup>39 61 62</sup> Therefore, if the pacing strategy is regulated to prevent absolute or “catastrophic” fatigue, as proposed in previous examples, then the overall pacing strategy during self-paced exercise should be altered by dietary interventions that result in either different amounts of stored energy (particularly muscle and liver glycogen) before exercise or altered substrate utilisation during exercise.

Indeed, a high-fat diet for 6 days, followed by a single day of high carbohydrate feeding to normalise muscle glycogen stores, impairs performance during repeated 1 km sprints during a self-paced 100 km time trial when compared with performance after a 7-day high carbohydrate diet.<sup>62</sup> However, the slower overall performance (3 minutes and 44 s) in the 100 km time trials following the high fat diet was not significantly different from the high carbohydrate diet, although five out of eight subjects improved while eating a high carbohydrate diet.

It was suggested that the higher intensity sprints were impaired because of an increase in sympathetic activation and consequent increase in effort perception<sup>62 63</sup> following the high fat intake. However, and most importantly, the measured RPE during trials was not different.<sup>62</sup> Therefore, if the high fat diet influenced sympathetic activity and the RPE, then it might have done so by altering the self-selected work rate that could be sustained to generate the given RPE.

Previous studies using a similar dietary regime failed to find an effect of dietary regimes on time trial performance.<sup>64 65</sup> These studies are often affected by large individual responses to the different diets, thereby reducing the statistical power. For example, in the study by Havemann *et al*,<sup>62</sup> five out of eight subjects improved their time trial performance when on a high carbohydrate diet. In contrast, in studies by Carey *et al*<sup>64</sup> and Burke *et al*,<sup>64</sup> most subjects improved performance on a high fat diet, with this improvement being attributed to a muscle glycogen-sparing effect as a result of increased rates of fat oxidation on the high fat diet. However, the studies differ in that they have used a constant effort time trial following prolonged submaximal exercise,<sup>64 65</sup> whereas the study by Havemann *et al*<sup>62</sup> included high intensity sprints during endurance exercise in an attempt to simulate pacing strategies during competition. In all these studies, it appears that altering the pre-exercise glycogen levels and utilisation of substrates through dietary manipulations may cause changes in pacing strategy and self-selected power output, but individually different responses make definitive conclusions difficult.

A putative role for glycogen concentration as a signaller and regulator of pacing strategy has been proposed by Rauch *et al*,<sup>39</sup>

who found that one hour cycling time trial performance was improved by a high carbohydrate diet that elevated the muscle glycogen content. The difference in performance was evident from the onset of the time trial, as power output in the normal diet group decreased and became lower than in the carbohydrate loaded group after the first minute. Therefore, subjects did not become glycogen “depleted” before the differences in performance were observed, but slowed down, apparently in advance of such an effect developing. Interestingly, each subject ended the trials with similar muscle glycogen concentrations, irrespective of whether they had been carbohydrate loaded or depleted. Therefore, subjects made use of the extra carbohydrate in the loaded trials, leading to the hypothesis that subjects paced themselves to reach a critical level of muscle glycogen at the termination of the exercise trial. Pacing was suggested to be the result of afferent feedback, signalling alterations in total substrate availability, thereby allowing a higher power output to be maintained in the carbohydrate loaded state.<sup>39</sup>

In conclusion, large individual differences in response to energy substrate availability have made definitive conclusions regarding its effects on pacing strategy difficult. Evidence does exist that self-paced exercise performance and pacing strategies are sensitive to alterations in muscle substrate utilisation. A role of glycogen as a signaller has been proposed in this regard. Pacing strategy may be regulated during endurance exercise to ensure that a limiting level of glycogen depletion does not occur.

### Provision of incorrect distance or duration feedback

The previous sections have described studies in which interventions such as temperature, diet or oxygen content have produced differences in pacing strategies, which have subsequently been used to develop a physiological model for self-paced exercise. In this model, the pacing strategy is altered by a central controller to prevent limiting physiological changes from occurring before the known endpoint of exercise is reached. Implicit in this model is that the endpoint of exercise must be known before the commencement of the exercise bout, because any anticipatory calculation cannot be made unless the duration of exercise is known with some accuracy. That is, if the adjustments in pacing strategy serve to prevent harmful or limiting disturbances to homeostasis before the end of exercise, as is described in this review, then the expected duration of exercise would serve as the “anchor point” against which this regulation would occur.

To confirm this, the following two hypotheses must be valid: First, if the athlete is correctly informed of the upcoming exercise duration before the commencement of exercise, then the provision of incorrect information regarding time and distance intervals during exercise would not be expected to result in changes in performance, provided the mismatch created by the misinformation is sufficiently small so as not to be consciously detected by the subjects. Second, if the athlete is incorrectly informed about the duration of exercise about to be undertaken, then performance will be negatively affected if the mismatch is eventually revealed or detected (either consciously or subconsciously), because the allocation of physiological resources will have been based on an incorrect expectation of exercise time before exercise began. Alternatively, if the athlete is incorrectly informed of duration, but the discrepancy between expected and actual duration is small and is not detected, then performance would be expected to be the same as when the athlete is reliably informed of the exercise duration.

To test the first hypothesis, Albertus *et al*<sup>36</sup> had well-trained male cyclists perform five 20-km cycling time trials, during which they received either correct or incorrect distance feedback every kilometre. In the incorrect feedback trials, subjects were told they had completed a kilometre when in fact the actual distance was either shorter or longer, by up to 250 m per kilometre. It was found that overall performance, pacing strategies and the subjective ratings of perceived exertion were not different at any stage in the different trials. This indicates that the control of pacing strategy is rigorous and is unaffected by the provision of incorrect feedback, and might be set before exercise based on the anticipated exercise duration, at least in exercise of this duration. Presumably, if the mismatches between actual and informed distances were larger, or if the subjects had been aware of their split times at each incorrectly informed kilometre, then the pacing strategy would have been altered in response to these conscious cues. However, because only distance feedback was provided, pacing strategy was based on the conscious expectation of the overall exercise duration of 20 km and the subconscious regulation was based on afferent feedback, as discussed previously.

In contrast, the second requirement of the present model is that exercise performance should be altered when the overall exercise duration differs from what was anticipated before the start of the exercise bout and large mismatches are detected by the exercising athlete. In support of this, Ansley *et al*<sup>25</sup> found evidence of a preprogrammed, centrally regulated pacing strategy during supramaximal exercise lasting only 36 s. That study found that when subjects performed a supramaximal cycling trial lasting 36 s after being informed that they would be cycling for only 30 s, their power output in the final 6 s was significantly lower than when they were correctly informed of the correct duration of the activity—that is, 36 s. Therefore, when the actual duration of the exercise bout exceeded the anticipated duration by 20%, a significant impairment in performance occurred, suggesting that the physiological resources had been “incorrectly” allocated. It was concluded that a pacing strategy was set based on the anticipated duration of exercise as a result of previous experience.

A third example that work rate is set in anticipation of exercise duration was provided by Nikolopoulos *et al*,<sup>66</sup> who informed well-trained cyclists that they would be completing 40 km time trials when the actual distances were 34 km, 40 km and 46 km (a difference of 15% compared with the expected duration). It was found that the pacing strategies and performances during the 34 km and 46 km rides were not different from those measured during the 40 km time trial, suggesting that the subjects were able to maintain a power output based on the expected distance (40 km) from the onset of exercise. Performances during subsequent 34 km and 46 km time trials in which subjects were correctly informed of the duration were not different from those trials with incorrect distance information.<sup>66</sup> This may suggest that the difference in distance between the actual 34 km or 46 km and the expected 40 km was not sufficiently large to allow the subjects to detect it and force them to alter their pacing strategy.

Also, subjects were provided with distance feedback in the form of the percentage of the distance remaining—this would allow the pacing strategy to be modified constantly with respect to the endpoint, such that the discrepancy of 6 km in the beginning is progressively reduced as the trial progresses. That study<sup>66</sup> does, however, provide evidence that well-trained

cyclists self-select power outputs and pacing strategies based on their perception or expectation of distance, and not solely on the physiological feedback during exercise.

A final interesting illustration of the importance of the expectation of exercise duration, even during exercise at a fixed work rate, was provided by Baden *et al*.<sup>40</sup> Their study suggested that the anticipated exercise duration influences running economy,<sup>40</sup> as the oxygen consumption ( $\text{VO}_2$ ) at a submaximal running speed (corresponding to 75% of previously measured peak treadmill running speed) was lower when subjects were told they would be running for an indeterminate length of time compared with trials in which they were told they would run for only 10 minutes. It was suggested that when an unknown, perhaps longer, exercise bout was undertaken, subjects attempted to conserve their resources by improving their energy efficiency.<sup>40</sup> It has also been shown that electromyographic activity in the biceps muscle is lower during a task of long duration than a task of short duration, despite similar work being performed,<sup>67</sup> supporting the idea of greater muscular efficiency in longer tasks.

### PHYSIOLOGICAL BASIS FOR POSITIVE PACING STRATEGY DURING SHORT DURATION EXERCISE

In contrast to the endspurt observed during longer duration exercise, both observational studies and studies in which the initial pacing strategy is manipulated have shown that during short duration exercise bouts lasting less than 2 minutes, there is an inability to increase work rate at the end of exercise.<sup>26–28 33 34</sup> Instead, there is typically a progressive reduction in power output or velocity in these events. This suggests that exercise intensity is not regulated during short duration exercise, but decreases as a result of a progressive failure of the muscle to produce force.<sup>59 68</sup> This argument is evaluated subsequently.

### EVIDENCE FOR DECLINING MUSCLE FORCE PRODUCTION DURING SHORT DURATION SELF-PACED EXERCISE

Kayser *et al*<sup>58</sup> found that electromyographic activity increases progressively during cycling exercise at a constant power output. They interpreted this as evidence for a “myographical sign of muscle fatigue”.<sup>60</sup> It was suggested that muscle can develop signs of metabolic fatigue but only if the volume of muscle at work is small, or if the workload achieved with larger muscle groups is very high,<sup>60</sup> as would occur during short duration, high intensity exercise. Taylor *et al*<sup>59</sup> found that during high intensity cycling exercise in hypoxia, the ratio of force/electromyographic activity decreased progressively, suggesting that an increase in motor unit activation was required to maintain the power output caused by a progressive reduction in muscle contractility.<sup>59</sup>

Similarly, Nummela *et al*<sup>68</sup> found that drop jump performance was impaired by 39% following a maximal 400 m sprint, and that the reduction was negatively correlated with increases in blood lactate concentrations. The electromyographic activity in the active sprinting muscles increased significantly over the course of the sprint. It was concluded that additional motor units were being activated to compensate for the progressive reduction in muscle force production as a result of metabolic acidosis in the muscle.<sup>68</sup> This supports the notion that the progressive decline in power output or running speed during shorter duration exercise bouts is the result of an attenuation of muscle contractility.

## EVIDENCE FOR PACING STRATEGIES DURING SHORT DURATION, HIGH INTENSITY EXERCISE

Whereas these studies<sup>58–68</sup> provide evidence that exercised muscle is less capable of producing force, studies have also shown an anticipatory component to short duration, high intensity exercise. Most significant is that when short duration exercise is undertaken, the initial power output is lower than is possible if the athlete is instructed to perform an all-out effort with no regard for overall performance.<sup>69</sup> Therefore, some form of pacing must be present to regulate the initial exercise intensity, even though a progressive reduction in intensity still occurs as exercise continues.

Foster *et al.*<sup>1</sup> evaluated the pattern of energy system contribution to power output during high intensity cycling lasting less than 2 minutes, and found that energetic resources were distributed over the duration of the event, apparently to preserve the contribution of non-oxidative energy production to power output throughout the time trial. It was suggested that the intracellular changes occurring during exercise, such as metabolite accumulation<sup>70–73</sup> or phosphagen depletion,<sup>74</sup> were being monitored continually and that power output was reduced in advance of these changes becoming critical or harmful,<sup>1</sup> in agreement with the model proposed for longer duration exercise.

Further evidence that the pacing strategy during shorter duration exercise bouts has an anticipatory component comes from the study of Ansley *et al.*,<sup>25</sup> described previously, which showed that performance during a supramaximal exercise bout was impaired only after the expected exercise duration had elapsed. This shows that the appropriate allocation of physiological resources is essential in even very short (36 s) exercise bouts, and suggests that the reduction in power output in the first 30 s occurs as part of this subconscious allocation, because a greater reduction in power output occurred after the pacing strategy had “failed”.

Collectively, these studies indicate that pacing strategy is regulated during exercise lasting less than 2 minutes, even though power output decreases progressively during exercise.<sup>1, 27–28</sup> This progressive reduction in work output is attributed to the impaired ability of muscle to produce force due to changes in metabolite levels, so-called “peripheral fatigue”.<sup>8, 21, 59–68</sup> This is based on the theory that impaired oxygen delivery or uptake results in the accumulation of metabolites, leading to a reduction in muscle pH, which impairs glycolysis<sup>75</sup> and muscle contractile processes.<sup>76</sup>

As such, much of the early focus on the optimal pacing strategy was on the effect of a faster or slower starts on oxygen kinetics, including oxygen uptake ( $\text{VO}_2$ ), incurred oxygen debt, post-exercise lactate levels and concentrations of other metabolites, including phosphocreatine and adenosine triphosphate. Robinson *et al.*<sup>29</sup> suggested that even pacing was optimal for middle distance running events, based on the finding that a faster start resulted in elevated blood lactate concentrations and oxygen uptake.

Similarly, Thompson *et al.*<sup>27</sup> found higher lactate levels, respiratory exchange ratio and RPE after 200 m breaststroke trials that began at speeds corresponding to 102% of a previously performed self-paced effort compared with trials performed at 100% of previous swimming times. The authors suggested that this impaired muscle function results from proton accumulation and reduced muscle pH,<sup>27</sup> and caused the large reduction in speed in the second half of the trial. Regardless of the potential factors responsible for this reduction in swimming speed, this conclusion<sup>27</sup> fails to acknowledge that

the best overall performance was achieved with the faster starting pace, and so performance was optimised despite these apparent “limitations” to muscle function as a result of higher lactate concentrations.

Bishop *et al.*<sup>28</sup> showed that performance time was improved even though initial and total oxygen consumption were greater when a kayaking trial was performed with an all-out start compared with an even-paced start. No differences were found in accumulated oxygen deficit, blood lactate concentrations or pH. They proposed that the higher initial  $\text{VO}_2$  was the result of greater rates of phosphocreatine breakdown. Interestingly, as with the study of Thompson *et al.*,<sup>27</sup> the power output declined significantly in the second half of the trial, but overall performance was still improved with the faster start. Therefore, rather than being detrimental to performance, the proposed elevation in phosphocreatine breakdown (and consequent reduction in phosphocreatine levels), as well as changes in the levels of other metabolites such as adenosine triphosphate and lactate as a result of the faster start, were associated with (although not necessarily responsible for) improved overall performance.

We therefore suggest that if an exercise trial is to be performed in the shortest possible time, these metabolic changes are simply the consequences of the high work output required early on during exercise in order for performance to be optimised. Whereas it must be acknowledged that the later reduction in power output is attributable to metabolic changes in the muscle, the observation that pacing strategies are still present in these events suggests that the observed reduction in power output is “tolerated” or controlled as a consequence of the overall pacing strategy, which ensures a balance between protecting against harmful disturbances to homeostasis while still optimising exercise performance.

It is interesting to speculate whether the requirements to defend homeostasis and optimise performance are in conflict with one another. That is, in order to prevent large disturbances in metabolic and physiological systems, a relatively lower work rate would have to be selected from the onset of exercise and would negate any possibility of achieving the best possible performance. During shorter duration bouts, an even pacing strategy may fall into this “suboptimal” category, with the initial exercise intensity being reduced and the resultant performance suboptimal.<sup>26</sup> In contrast, excessively high work rates early on would result in a greater threat to the maintenance of homeostasis, causing a rapid decline in exercise work rate, leading to impaired performance, as demonstrated by Foster *et al.*<sup>26</sup>

Consequently, if the pacing strategy is a marker of the complex regulation of physiological systems and performance during exercise, then an optimal pacing strategy does not necessarily imply minimal physiological derangements during exercise, because these would result in suboptimal performance. Nor does it suggest that an all-out pacing strategy with resultant peripheral fatigue is optimal, if the systems were pushed beyond their limits. In longer duration exercise, in which there may for example be complete failure to continue exercise when a critical core temperature is reached,<sup>12</sup> or when energy substrates are depleted,<sup>62</sup> performance would clearly be suboptimal if these “limitations” occurred before the exercise bout was completed. Therefore, the maintenance of homeostasis is an essential requirement for optimal performance during exercise of all durations.

However, in shorter duration exercise bouts, it is possible that metabolic changes such as metabolite accumulation or acidosis,

sufficient to cause a progressive and gradual decline in exercise intensity even at the same level of muscle activation, are controlled in order to optimise performance, possibly because these changes in metabolite concentrations are short-lived and reversible. The greater kinetic energy in these events<sup>26–69</sup> may also mean that reductions in power output do not affect overall performance times to the same extent as in longer duration exercise, because the decline in work output has a minimal impact on the “decay” in velocity and thus overall performance. This is particularly true for speed skating events.<sup>31</sup>

## CONCLUSION

There is evidence that during long duration exercise, the overall pacing strategy is mediated to prevent premature fatigue caused by a failure of one or more physiological systems. The resulting pacing strategy is thus proposed to be a marker of underlying physiological regulation, and alterations in pacing strategy occur due to changes in muscle activation in an anticipatory manner, based on afferent feedback from the various physiological systems and previous experience.

Evidence from short duration exercise trials is that performance is optimised when initial power output is high, even though the work rate may decline in the second half of the bout and the muscle force-generating capacity may decline. A centrally mediated pacing strategy for self-paced exercise of all durations is proposed, the role of which is to balance the requirement for optimal performance with the requirement to complete the task without causing irreparable harm to the muscle and other organs. This results in a certain degree of physiological disturbance being tolerated, which may cause a progressive reduction in power output as exercise continues, particularly during short duration (<4 minutes) exercise. However, as the pacing strategy is regulated based on previous experience and afferent information from the periphery, the task can be completed without the development of bodily harm.

Critically, this complex central regulation of exercise does not completely prevent homeostatic disturbances from occurring. For example, exercise intensity is not reduced so much that heat storage becomes zero<sup>4</sup>—a certain level of heat storage is “allowed”. Similarly, energy substrate levels<sup>59–62</sup> and oxygen saturation levels<sup>77</sup> do decline during self-paced exercise, and there is evidence that muscle force-generating capacity is reduced during dynamic cycling exercise.<sup>58–59</sup> However, these changes do not reach those critical levels at which exercise would terminate or bodily harm would occur. Rather, a reserve exists in which skeletal muscle activation can be increased to cause increases in whole limb power output even in the presence of such peripheral changes.

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## REFERENCES

1. Foster C, de Koning JJ, Hettinga F, *et al.* Pattern of energy expenditure during simulated competition. *Med Sci Sports Exerc* 2003;**35**:826–31.
2. de Koning JJ, Bobbert MF, Foster C. Determination of optimal pacing strategy in track cycling with an energy flow model. *J Sci Med Sport* 1999;**2**:266–77.
3. Hettinga FJ, de Koning JJ, Meijer E, *et al.* Biodynamics. Effect of pacing strategy on energy expenditure during a 1500-m cycling time trial. *Med Sci Sports Exerc* 2007;**39**:2212–18.
4. Marino FE, Lambert MI, Noakes TD. Superior performance of African runners in warm humid but not in cool environmental conditions. *J Appl Physiol* 2004;**96**:124–30.
5. Tucker R, Rauch L, Harley YXR, *et al.* Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. *Pflügers Arch* 2004;**448**:422–30.
6. Tattersson AJ, Hahn AG, Martin DT, *et al.* Effects of heat stress on physiological responses and exercise performance in elite cyclists. *J Sci Med Sport* 2000;**3**:186–93.
7. Marino FE, Mbambo Z, Kortekaas E, *et al.* Advantages of smaller body mass during distance running in warm, humid environments. *Pflügers Arch* 2000;**441**:359–67.
8. Noakes TD. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand J Med Sci Sports* 2000;**10**:123–45.
9. Noakes TD, St Clair Gibson A. Logical limitations to the “catastrophe” models of fatigue during exercise in humans. *Br J Sports Med* 2004;**38**:648–9.
10. Nielsen B, Savard G, Richter EA, *et al.* Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol* 1990;**69**:1040–6.
11. Nielsen B. Olympics in Atlanta: a fight against physics. *Med Sci Sports Exerc* 1996;**28**:665–8.
12. Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* 2001;**91**:1055–60.
13. Galloway SD, Maughan RJ. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med Sci Sports Exerc* 1997;**29**:1240–9.
14. Morrison S, Sleivert GG, Cheung SS. Passive hyperthermia reduces voluntary activation and isometric force production. *Eur J Appl Physiol* 2004;**91**:729–36.
15. Todd G, Butler JE, Taylor JL, *et al.* Hyperthermia: a failure of the motor cortex and the muscle. *J Physiol* 2005;**563**:621–31.
16. St Clair Gibson A, Schabert EJ, Noakes TD. Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Reg Int Comp Physiol* 2001;**281**:R187–96.
17. Ulmer HV. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia* 1996;**52**:416–20.
18. Noakes TD, St Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br J Sports Med* 2005;**39**:120–4.
19. Tucker R, Marle T, Lambert EV, *et al.* The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *J Physiol* 2006;**574**:905–15.
20. St Clair Gibson A, Noakes TD. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br J Sports Med* 2004;**38**:797–806.
21. Lambert EV, St Clair Gibson A, Noakes TD. Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *Br J Sports Med* 2005;**39**:52–62.
22. Peltonen JE, Rantamäki J, Niittymäki SPT, *et al.* Effects of oxygen fraction in inspired air on force production and electromyogram activity during ergometer rowing. *Eur J Appl Physiol* 1997;**76**:495–503.
23. Brosnan MJ, Martin DT, Hahn AG, *et al.* Impaired interval exercise responses in elite female cyclists at moderate simulated altitude. *J Appl Physiol* 2000;**89**:1819–24.
24. Tucker R, Kayser B, Rae E, *et al.* Hyperoxia improves 20 km cycling time trial performance by increasing muscle activation levels while perceived exertion stays the same. *Eur J Appl Physiol* 2007;
25. Ansley L, Robson PJ, St Clair GA, *et al.* Anticipatory pacing strategies during supramaximal exercise lasting longer than 30 s. *Med Sci Sports Exerc* 2004;**36**:309–14.
26. Foster C, Snyder AC, Thompson NN, *et al.* Effect of pacing strategy on cycle time trial performance. *Med Sci Sports Exerc* 1993;**25**:383–8.
27. Thompson KG, MacLaren DP, Lees A, *et al.* The effects of changing pace on metabolism and stroke characteristics during high-speed breaststroke swimming. *J Sports Sci* 2004;**22**:149–57.
28. Bishop D, Bonetti D, Dawson B. The influence of pacing strategy on  $\dot{V}O_2$  and supramaximal kayak performance. *Med Sci Sports Exerc* 2002;**34**:1041–7.
29. Robinson S, Robinson DL, Mountjoy RJ, *et al.* Influence of fatigue on the efficiency of men during exhausting runs. *J Appl Physiol* 1958;**12**:197–201.
30. Ingen Schenau GJ, de Koning JJ, de Groot G. The distribution of anaerobic energy in 1000 and 4000 metre cycling bouts. *Int J Sports Med* 1992;**13**:447–51.
31. de Koning JJ, Foster C, Lampen J, *et al.* Experimental evaluation of the power balance model of speed skating. *J Appl Physiol* 2005;**98**:227–33.
32. Ingen Schenau GJ, de Koning JJ, de Groot G. Optimisation of sprinting performance in running, cycling and speed skating. *Sports Med* 1994;**17**:259–75.
33. Ferro A, Rivera A, Pagola I, *et al.* Biomechanical analysis of the 7th World Championships in Athletics Seville 1999. *IAAF New Studies in Athletics* 2001;**16**:25–60.
34. Tucker R, Lambert MI, Noakes TD. An analysis of pacing strategies during men's world-record performances in track athletics. *Int J Sports Physiol Performance* 2006;**1**:233–45.
35. Baden DA. Goals and expectations: psychological and physiological effects of anticipating the end. 2002;
36. Albertus Y, Tucker R, St Clair Gibson A, *et al.* Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Med Sci Sports Exerc* 2005;**37**:461–8.
37. Lambert MI, Dugas JP, Kirkman MC, *et al.* Changes in running speeds in a 100 km ultra-marathon race. *J Sports Sci Med* 2004;**3**:167–73.
38. Peltonen JE, Rantamäki J, Niittymäki SPT, *et al.* Effects of oxygen fraction in inspired air on rowing performance. *Med Sci Sports Exerc* 1995;**27**:573–9.
39. Rauch HG, St Clair Gibson A, Lambert EV, *et al.* A signalling role for muscle glycogen in the regulation of pace during prolonged exercise. *Br J Sports Med* 2005;**39**:34–8.
40. Baden DA, Mclean T, Tucker R, *et al.* Effect of anticipation during unknown or unexpected exercise duration on rating of perceived exertion, affect, and physiological function. *Br J Sports Med* 2005;**39**:742–6.

41. **Fink WJ**, Costill DL. Leg muscle metabolism during exercise in the heat and cold. *Eur J Appl Physiol* 1975;**34**:247–8.
42. **Savard GK**, Nielsen B, Laszczynska J, *et al.* Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J Appl Physiol* 1988;**64**:649–57.
43. **Drust B**, Rasmussen P, Mohr M, *et al.* Elevations in core and muscle temperature impairs repeated sprint performance. *Acta Physiol Scand* 2005;**183**:181–90.
44. **Rowell LB**, Marx HJ, Bruce RA, *et al.* Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest* 1966;**45**:1801–16.
45. **Nielsen B**, Hales JR, Strange S, *et al.* Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol* 1993;**460**:467–85.
46. **Gonzalez-Alonso J**, Teller C, Andersen SL, *et al.* Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 1999;**86**:1032–9.
47. **Nielsen B**, Strange S, Christensen NJ, *et al.* Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflügers Arch* 1997;**434**:49–56.
48. **Cheung SS**, McLellan TM. Influence of short-term aerobic training and hydration status on tolerance during uncompensable heat stress. *Eur J Appl Physiol* 1998;**78**:50–8.
49. **Cheung SS**, Sleivert GG. Multiple triggers for hyperthermic fatigue and exhaustion. *Exerc Sport Sci Rev* 2004;**32**:100–6.
50. **Bruck K**, Olschewski H. Body temperature related factors diminishing the drive to exercise. *Can J Physiol Pharmacol* 1987;**65**:1274–80.
51. **Fuller A**, Carter RN, Mitchell D. Brain and abdominal temperature at fatigue in rats exercising in the heat. *J Appl Physiol* 1998;**84**:877–83.
52. **Nielsen B**, Hyldig T, Bidstrup F, *et al.* Brain activity and fatigue during prolonged exercise in the heat. *Pflügers Arch* 2001;**442**:41–8.
53. **Nybo L**, Nielsen B. Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *J Appl Physiol* 2001;**91**:2017–23.
54. **Rasmussen P**, Stie H, Nybo L, *et al.* Heat induced fatigue and changes of the EEG is not related to reduced perfusion of the brain during prolonged exercise in humans. *J Therm Biol* 2004;**29**:731–7.
55. **Kay D**, Marino FE, Cannon J, *et al.* Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Eur J Appl Physiol* 2001;**84**:115–21.
56. **Dennis SC**, Noakes TD. Advantages of a smaller body mass in humans when distance-running in warm, humid conditions. *Eur J Appl Physiol* 1999;**79**:280–4.
57. **Marino FE**. Anticipatory regulation and avoidance of catastrophe during exercise-induced hyperthermia. *Comp Biochem Physiol B Biochem Mol Biol* 2004;**139**:561–9.
58. **Kayser B**, Narici M, Binzoni T, *et al.* Fatigue and exhaustion in chronic hypobaric hypoxia: influence of exercising muscle mass. *J Appl Physiol* 1994;**76**:634–40.
59. **Taylor AD**, Bronks R, Smith P, *et al.* Myoelectric evidence of peripheral muscle fatigue during exercise in severe hypoxia: some references to m. vastus lateralis myosin heavy chain composition. *Eur J Appl Physiol* 1997;**75**:151–9.
60. **Kayser B**. Exercise starts and ends in the brain. *Eur J Appl Physiol* 2003;**90**:411–19.
61. **Bergstrom J**, Hermansen L, Hultman E, *et al.* Diet, muscle glycogen and physical performance. *Acta Physiol Scand* 1967;**71**:140–50.
62. **Havemann L**, West SJ, Goedecke J, *et al.* Fat adaptation followed by carbohydrate-loading compromises high-intensity sprint performance. *J Appl Physiol* 2005;
63. **Stepto NK**, Carey AL, Staudacher HM, *et al.* Effect of short-term fat adaptation on high-intensity training. *Med Sci Sports Exerc* 2002;**34**:449–55.
64. **Burke LM**, Hawley JA, Angus DJ, *et al.* Adaptations to short-term high-fat diet persist during exercise despite high carbohydrate availability. *Med Sci Sports Exerc* 2002;**34**:83–91.
65. **Carey AL**, Staudacher HM, Cummings NK, *et al.* Effects of fat adaptation and carbohydrate restoration on prolonged endurance exercise. *J Appl Physiol* 2001;**91**:115–22.
66. **Nikolopoulos V**, Arkinstall MJ, Hawley JA. Pacing strategy in simulated cycle time-trials is based on perceived rather than actual distance. *J Sci Med Sport* 2001;**4**:212–19.
67. **Vidacek S**, Wishner J. Influence of expectation of task duration on efficiency of muscular activity. *J Appl Psychol* 1971;**55**:564–9.
68. **Nummela A**, Vuorimaa T, Rusko HK. Changes in force production, blood lactate and EMG activity in the 400-m sprint. *J Sports Sci* 1992;**10**:217–28.
69. **Foster C**, Schrage M, Snyder AC, *et al.* Pacing strategy and athletic performance. *Sports Med* 1994;**17**:77–85.
70. **Jacobs I**, Tesch PA, Bar-Or O, *et al.* Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. *J Appl Physiol* 1983;**55**:365–7.
71. **Jacobs I**, Kaiser P. Lactate in blood, mixed skeletal muscle, and FT or ST fibres during cycle exercise in man. *Acta Physiol Scand* 1982;**114**:461–6.
72. **Karlsson J**, Saltin B. Lactate, ATP, and CP in working muscles during exhaustive exercise in man. *J Appl Physiol* 1970;**29**:596–602.
73. **Diamant B**, Karlsson J, Saltin B. Muscle tissue lactate after maximal exercise in man. *Acta Physiol Scand* 1968;**72**:383–4.
74. **McLester JR Jr.** Muscle contraction and fatigue. The role of adenosine 5'-diphosphate and inorganic phosphate. *Sports Med* 1997;**23**:287–305.
75. **Hermansen L**. Muscle fatigue during maximal exercise of short duration. 1981:45–52.
76. **Fabiato A**, Fabiato F. Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from the cardiac and skeletal muscles. *J Physiol* 1978;**276**:233–55.
77. **Peltonen JE**, Leppavuori AP, Kyrö K-P, *et al.* Arterial haemoglobin oxygen saturation is affected by F<sub>2</sub>O<sub>2</sub> at submaximal running velocities in elite athletes. *Scand J Med Sci Sports* 1999;**9**:265–71.