

Review Article

Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance

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A popular concept in the exercise sciences holds that fatigue develops during exercise of moderate to high intensity, when the capacity of the cardiorespiratory system to provide oxygen to the exercising muscles falls behind their demand inducing “anaerobic” metabolism. But this cardiovascular/anaerobic model is unsatisfactory because (i) a more rigorous analysis indicates that the first organ to be affected by anaerobiosis during maximal exercise would likely be the heart, not the skeletal muscles. This probability was fully appreciated by the pioneering exercise physiologists, A. V. Hill, A. Bock and D. B. Dill, but has been systematically ignored by modern exercise physiologists; (ii) no study has yet definitely established the presence of either anaerobiosis, hypoxia or ischaemia in skeletal muscle during maximal exercise; (iii) the model is unable to explain why exercise terminates in a variety of conditions including prolonged exercise, exercise in the heat and at altitude, and in those with chronic diseases of the heart and lungs, without any evidence for skeletal muscle anaerobiosis, hypoxia or ischaemia, and before

there is full activation of the total skeletal muscle mass, and (iv) cardiovascular and other measures believed to relate to skeletal muscle anaerobiosis, including the maximum oxygen consumption (VO_2 max) and the “anaerobic threshold”, are indifferent predictors of exercise capacity in athletes with similar abilities. This review considers four additional models that need to be considered when factors limiting either short duration, maximal or prolonged sub-maximal exercise are evaluated. These additional models are: (i) the energy supply/energy depletion model; (ii) the muscle power/muscle recruitment model; (iii) the biomechanical model and (iv) the psychological model. By reviewing features of these models, this review provides a broad overview of the physiological, metabolic and biomechanical factors that may limit exercise performance under different exercise conditions. A more complete understanding of fatigue during exercise, and the relevance of the adaptations that develop with training, requires that the potential relevance of each model to fatigue under different conditions of exercise must be considered.

The nature of the physiological and biochemical adaptations that occur in response to physical training has been extensively studied in humans and other mammals. This information is readily available and is likely to be well known to most exercise scientists (Saltin & Gollnick 1983, Holloszy & Coyle 1984). Similarly there is an extensive literature on the cellular mechanisms believed to cause the fatigue that develops during exercise (Fitts 1994).

In contrast, fewer studies have evaluated the extent to which these adaptations explain the improvements

in performance that occur with different types of physical training (Acevedo & Goldfarb 1989, Daniels et al. 1978, Hawley et al. 1997, Houston et al. 1979, Moore et al. 1997, Ramsbottom et al. 1989, Westgarth-Taylor et al. 1997, Weston et al. 1997) and which presumably result from changes that delay the onset or development of fatigue. There are at least three probable reasons for this.

First, many exercise physiologists may consider this to be the work of the coach, not of the scientist. Or, accustomed to the tightly controlled conditions of laboratory research, some scientists may be reluctant to undertake field-based studies of performance in which all the different variables influencing human performance are not easily controlled. Human performance is influenced by many variables, not least those involving the psyche. Many scientists may feel,

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perhaps justifiably, that these variables cannot be sufficiently well controlled in field studies for there to be meaningful findings.

Second, there is a dearth of tools to measure accurately human performance in the laboratory. If sports performance cannot be measured frequently with a high degree of precision in the laboratory, then training-induced changes in exercise performance are not quantifiable. As a result, most studies use physiological surrogates to predict changes in exercise performance. The most widely used performance surrogate is the maximum oxygen consumption (VO_2 max). But, the very use of this specific measure has helped to entrench a particular and, perhaps, unquestioning dogma of the factors that likely determine human exercise performance (Noakes 1988, 1997, 1998).

As a result, most of the training studies reported in the literature have measured the physiological and biochemical responses of the human to training and have paid less attention (i) to the extent to which human exercise performance is altered by different training programmes and (ii) to the specific physiological adaptations which explain training-induced changes in athletic performance.

Indeed, an important weakness in our current thinking in exercise physiology is that we lack certain knowledge of the precise factors that determine fatigue and hence limit performance in different types of exercise under a range of environmental conditions. In part, this is because some scientists remain unaware that their research is based on the (subconscious) acceptance usually of one specific model of human exercise physiology (Noakes 1997, 1998). But it would be very surprising if one single physiological model adequately explains human exercise performance under all conditions.

Accordingly, the aim of this review is not to describe how the body adapts to physical training. This information is freely available, largely descriptive and not particularly contentious, so that its review is unlikely to challenge how we think about our science. Rather I will use this opportunity to pose two questions: What physiological models have exercise scientists developed (and subconsciously accepted) for the study of the physiological and biochemical determinants of fatigue during exercise? And which specific physiological, metabolic or biomechanical attributes

might explain superior athletic performance and enhanced resistance to the development of fatigue?

Current physiological models to understand the physiology of training for enhanced endurance performance

Table 1 lists five different models that are commonly used to study and explain the likely physiological and other training-induced changes that may improve, especially, endurance performance, probably by delaying or preventing the onset of fatigue. Each model has its own proponents, usually those with a special expertise in the specific areas embraced by the model. Thus, the cardiovascular/anaerobic model is promoted usually by cardiovascular and respiratory physiologists; the energy supply/energy depletion model is favoured by the exercise biochemists; the muscle power/muscle recruitment model is advocated by muscle physiologists, and some biomechanists and neuro-physiologists; the biomechanical model by biomechanists, and the psychological/motivational model by sports psychologists.

Yet it is highly improbable that the factors explaining human exercise performance under all conditions are restricted to one physiological system or to one scientific discipline. Thus, human performance is unlikely to be adequately defined by any of these unitary models that are often presented as if they are mutually exclusive. The complexity of the physiological and other factors determining human performance is emphasized when the limitations of each of these models are exposed.

The cardiovascular/anaerobic model

Maximal exercise

This model holds that endurance performance is determined by the capacity of the athlete's large heart to pump unusually large volumes of blood and oxygen to the muscles. This allows the muscles to achieve higher work rates before they outstrip the available oxygen supply, developing skeletal muscle anaerobiosis (Fig. 1) (Noakes 1988, 1997, 1998, Bassett & Howley 1997). This model remains the most popular for explaining why fatigue develops during exercise; how the body adapts to training; how these adaptations enhance performance and, as a consequence, how effective exercise training programmes should be structured.

This model predicts that training increases "cardiovascular fitness" especially by increasing the body's maximum capacity to consume oxygen, measured as the maximum oxygen consumption (VO_2 max). This effect results from an increased maximum capacity of the heart to pump blood (the cardiac output) and an enhanced capacity of the muscles to consume that

Table 1. Current physiological models to understand the physiology of training for enhanced endurance performance

- (a) The cardiovascular/anaerobic model
- (b) The energy supply/energy depletion model
- (c) The muscle recruitment (central fatigue)/muscle power model
- (d) The biomechanical model
- (e) The psychological/motivational model

The classic theory of fatigue according to the Cardiovascular/Anaerobic Model of Exercise Physiology

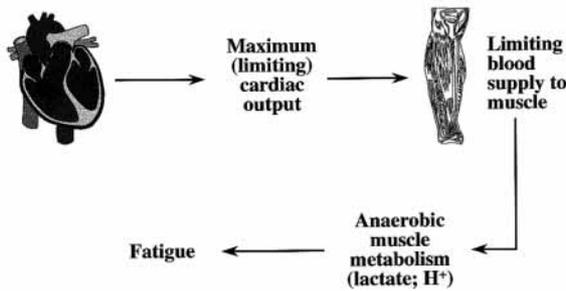


Fig. 1. The Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance holds that the heart has a limiting maximum cardiac output that is reached at the onset of a “plateau phenomenon”, ascribed incorrectly to the work of Hill and colleagues (Hill et al. 1924, Hill 1927). As a result, blood and hence oxygen flow to the exercising muscles falls behind demand, inducing anaerobic metabolism. Metabolites of anaerobic metabolism, in particular hydrogen ions, then inhibit muscle contraction, inducing fatigue.

oxygen, the latter by increasing skeletal muscle capillarization and mitochondrial mass. It is argued that these adaptations delay the onset of skeletal muscle anaerobiosis during vigorous exercise, thereby reducing blood lactate concentrations in muscle and blood at all exercise intensities above the so-called “anaerobic threshold”. The delayed onset of this blood lactate accumulation then allows the exercising muscles to continue contracting for longer at higher intensities before the onset of fatigue.

In addition, these changes increase the capacity of the muscles to use fat as a fuel during exercise, thereby enhancing endurance performance (according to the Energy Depletion model, described subsequently) (Saltin & Gollnick 1983, Holloszy & Coyle 1984). An important but unrecognized prediction of this model is that increases in coronary blood flow must be an essential adaptation to training (Noakes 1998). The higher coronary blood flow allows a greater pumping capacity of the heart producing a greater cardiac output to perfuse the exercising muscles, which can then achieve a higher exercise capacity.

This model finds strong support from the confirmation that these changes, with the exception of a greater coronary flow which is inferred, not proven, do indeed result from training, as fully documented in the literature. The key question is whether these changes are causally linked; that is, do these changes cause the change in exercise performance or do they occur pari-passu with other adaptation(s) that are the real cause of changes in exercise performance. For there are important deficiencies in this model which are fully argued (Noakes 1988, 1997) and counter-argued (Noakes 1998, Bassett & Howley 1997) elsewhere and will not be repeated here.

Perhaps the major but overlooked limitation of this model is that, if the pumping capacity of the heart does indeed limit oxygen utilization by the exercising skeletal muscle, then the heart itself will be the first organ affected by any postulated oxygen deficiency (Noakes 1998). This was first recognized by Hill and his colleagues as early as 1925 (Hill et al. 1924). Paradoxically it was the incorrect interpretation, by others, of the work of Hill and his colleagues, in particular their supposed description of a plateau phenomenon (Noakes 1998), that forms the (mythical) foundation for the cardiovascular/anaerobic model of exercise physiology. Yet those who popularized this mythical interpretation of the work of Hill and his colleagues, failed also to record what Hill considered to be the physiological cause (and equally the consequence) of the fatigue that develops during maximal exercise.

For the interpretation of Hill and his colleagues was unequivocal: “Certain it is that the capacity of the body for muscular exercise depends largely, if not mainly, on the capacity and output of the heart. It would obviously be very dangerous for the organ to be able, as the skeletal muscle is able, to exhaust itself very completely and rapidly, to take exercise far in excess of its capacity for recovery ... When the oxygen supply becomes inadequate, it is probable that the

Which organ is at greatest risk during maximum exercise?

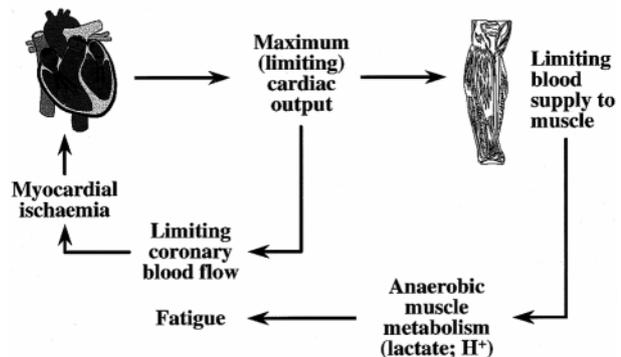


Fig. 2. The weakness of the Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance is that the attainment of a maximum cardiac output has more serious consequences for the heart than it does for the skeletal muscles as the first organ to be affected by a maximum cardiac output would be the heart itself. Continuing to exercise with a fixed (maximum) cardiac output would cause myocardial ischaemia. The heart, unable to increase coronary flow (dependant on an increase in cardiac output), would be unable to balance the increased myocardial oxygen demand caused by the very increase in workrate, theoretically necessary to define the mythical “plateau phenomenon” of Hill and his colleagues. Many early researchers believed that the heart did indeed develop ischaemia during maximal exercise, leading to a fall in cardiac output (Hill et al. 1924, Bainbridge 1931, Hill 1927, Dill 1938).

heart rapidly begins to diminish its output, so avoiding exhaustion ...”.

The point identified by Hill and his colleagues, and since ignored by all subsequent generations of exercise physiologists, is that the heart is also a muscle, dependent for its function on an adequate blood and oxygen supply. But, unlike skeletal muscle, the heart is dependent for its blood supply on its own pumping capacity. Hence any intervention that reduces the pumping capacity of the heart, or demands the heart somehow to sustain an increased work output by the exercising muscles without any increase in cardiac output and coronary flow (as theoretically occurs when the “plateau phenomenon” develops), imperils the heart’s own blood supply. Any reduction in coronary blood flow will consequently reduce the heart’s pumping capacity, thereby inducing a vicious cycle of progressive and irreversible myocardial ischaemia (Fig. 2). It would seem logical that human design should include controls to protect the heart from ever entering this vicious circle.

Hence if (skeletal) muscle function fails when its oxygen demand exceeds supply then, for logical consistency, the inability of the pumping capacity of the heart to “raise the cardiac output” at the VO_2 max (Rowell 1993), must also result from an inadequate (myocardial) oxygen supply caused by a plateau in

coronary flow. This limiting coronary blood flow induces myocardial “fatigue”, causing the plateau in cardiac output and hence in the VO_2 max leading, finally, to skeletal muscle anaerobiosis. Thus, by this logic, the coronary blood flow must be the first physiological function to show a “plateau phenomenon” during progressive exercise to exhaustion (Fig. 3). All subsequent physiological “plateaus” must result from this limiting coronary flow (Noakes 1998).

Whereas the most influential modern exercise physiologists have enthusiastically embraced this mythical basis for a “plateau phenomenon” for the past 75 years, none seems to have grasped this logical prediction of the “plateau phenomenon”, which is that the “plateau phenomenon” requires the heart to fatigue first before skeletal muscle fatigue can develop. But this was clearly a concept with which the pioneering exercise physiologists were entirely comfortable. Thus, in addition to the conclusion of Hill and his colleagues, already quoted, both Bock and Dill (Bainbridge 1931) also believed that myocardial hypoxia causes a fall in the cardiac output at the point of fatigue during high intensity exercise:

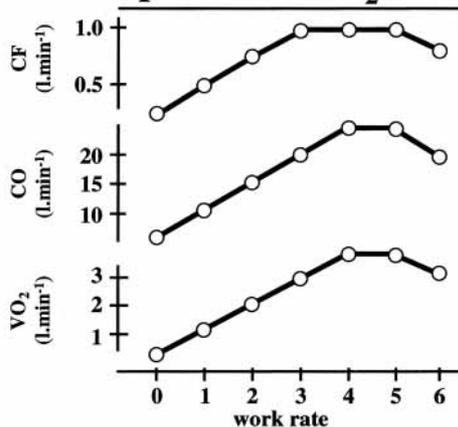
“The blood supply to the heart, in many men, may be the weak link in the chain of circulatory adjustments during muscular exercise, and as the intensity of muscular exertion increases, a point is probably reached in most individuals at which the supply of oxygen to the heart falls short of its demands, and the continued performance of work becomes difficult or impossible” (p. 15). Hence they proposed that: “Another factor, which may contribute to the production of this type of fatigue, is fatigue of the heart itself” (p. 229).

“Although the occurrence of fatigue of the heart in health is not very clearly established, a temporary lowering of the functional capacity of the heart, induced by fatigue of its muscular fibres, might gradually bring about during exercise an insufficient blood supply to the skeletal muscles and brain. The lassitude and disinclination for exertion, often experienced on the day after a strenuous bout of exercise, has been ascribed to fatigue of the heart as its primary cause” (p. 229). Hence they concluded: “The heart, as a rule, reaches the limit of its powers earlier than the skeletal muscles, and determines a man’s capability for exertion”.

In summary, the early physiologists who believed that skeletal muscle anaerobiosis limits maximal exercise clearly understood that any plateau in cardiac output, necessary for there to be a limiting skeletal muscle blood flow, must result from a plateau in coronary blood flow which would expose the heart to a progressive myocardial ischaemia that would worsen as exercise was prolonged.

Perhaps the reluctance of modern physiologists to acknowledge these concepts stems from the current

What causes the “plateau” in cardiac output at the VO_2 max?



From: Noakes TD. *Med Sci Sports Exerc* 1998; 30: 1381-8.

Fig. 3. According to the logic of the Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance, coronary flow must be the first physiological variable to “plateau” during progressive exercise to exhaustion. The peak in coronary flow would then induce a plateau in cardiac output as a result of a progressive myocardial ischaemia. Continuing to exercise with a fixed cardiac output and coronary flow would rapidly cause an ischaemia-induced fall in cardiac output and in coronary flow, and hence in whole body oxygen consumption. This logic was accepted by the early proponents of this model (Hill et al. 1924, Bainbridge 1931, Hill 1927, Dill 1938) but has since been overlooked by exercise physiologists for the past 75 years.

appreciation that progressive myocardial ischaemia does not occur during maximal exercise in healthy athletes (Raskoff et al. 1976), even though there is good evidence that it is a limiting cardiac output that probably determines the VO_2 max (Rowell 1993). Thus, one postulate might be that even if cardiac output limits maximal exercise as seems likely (Noakes 1997), termination of exercise must occur before the heart actually reaches that maximum and hence well before skeletal muscle anaerobiosis can develop (Noakes 1998). Hence for 75 years, exercise physiologists may have focused on the incorrect organ as the site of any potential anaerobiosis that may develop during maximal exercise (Hill et al. 1924, Bainbridge 1931, Hill 1927, Hill et al. 1924).

How might a maximal cardiac output be reached without the development of myocardial ischaemia? The argument that the rate of cardiac filling, due either to a limiting venous return or the effects of a restrictive pericardium (Stray-Gundersen et al. 1986) may limit the maximal cardiac output, whilst superficially attractive, is still unable satisfactorily to explain which physiological events terminates exercise. Such an argument fails for the reason that the continuation of exercise beyond that (however limited) maximal cardiac output must still cause a progressive myocardial ischaemia to develop (Fig. 2). Hence, even if the cardiac output is limited by factors unrelated to the development of myocardial ischaemia (for example, a limiting venous return), the continuation of exercise beyond that point of limitation must induce myocardial ischaemia and the development of chest pain (angina pectoris) that would terminate exercise.

Perhaps it is more logical to speculate that maximal exercise terminates as part of a regulated process before the absolute maximum cardiac output and coronary blood flow are achieved. Interestingly Hill and his colleagues seem to have been the first to suggest a solution to this dilemma as early as 1924: "From the point of view of a well co-ordinated mechanism, ... it would clearly be useless for the heart to make an excessive effort if by doing so it merely produced a far lower degree of saturation of the arterial blood; and we suggest that, in the body (either in the heart muscle itself or in the nervous system), there is some mechanism which causes a slowing of the circulation as soon as a serious degree of unsaturation occurs, and vice versa. This mechanism would tend to act as a governor maintaining a high degree of saturation of the blood" (Hill et al. 1924, p. 161–162).

Clearly no such governor has yet been discovered, perhaps because no physiologists have yet searched for it. But there is clear physiological evidence for the existence of such a governor. The evidence comes from studies of skeletal and cardiac muscle function at altitude. For if oxygen deficiency really does develop in either heart or skeletal muscle during maxi-

mum exercise, its appearance will likely be more easily identifiable during exercise at altitude under conditions of hypobaric hypoxia. Furthermore, such experiments should identify in which organ – heart or skeletal muscle – anaerobiosis first becomes apparent; the heart, according to the ideas of the pioneering British and North American exercise physiologists, or the skeletal muscles, according to the influential group of modern exercise physiologists (Noakes 1998).

The original studies of exercise at altitude were undertaken by a research group co-ordinated by Dill and his colleagues from the Harvard fatigue laboratory. This research established two crucial findings. First, that peak blood lactate concentrations during maximum exercise fell with increasing altitude (Edwards 1936), a phenomenon since labelled the "lactate paradox" (Hochachka 1989). Second, that maximum heart rate and cardiac output likewise fell during exercise at increasing altitude (Christensen 1938, Dill 1938).

Edwards (1936) interpreted the "lactate paradox" at altitude accordingly: "The inability to accumulate large amounts of lactate at high altitudes suggests a protective mechanism preventing an already low arterial saturation from becoming markedly lower ... It may be that the protective mechanism lies in an inadequate oxygen supply to essential muscles, e.g. the diaphragm or the muscles".

The existence of the "lactate paradox" was confirmed during the epic laboratory experiment of exercise and acclimatization at simulated high altitude, Operation Everest II (Green et al. 1989). That study found that muscle lactate concentrations achieved during maximal exercise at the highest equivalent altitude achieved during that experiment (8848 m – equivalent to the summit of Mount Everest) were no higher than when at rest at sea level.

Hence, in as much as high muscle lactate concentrations would have to be present if the exercising muscles were contracting "anaerobically", this study proves that exercise at extreme altitude terminates when the exercising muscles are contracting in fully aerobic conditions.

Similarly Operation Everest II (Sutton et al. 1988) confirmed these original and subsequent studies (Pugh 1964, Vogel et al. 1974) showing that heart rate and cardiac output are substantially reduced during exercise at extreme altitude. The key observation is that the peak cardiac output falls with increasing altitude. This response is equally paradoxical for those who believe that the delivery of an adequate oxygen supply to the exercising muscles is the cardinal priority during exercise (Noakes 1998). For logic demands that *if* the principal responsibility of the cardiovascular system during exercise is the achievement of an (ultimately inadequate) oxygen supply to skeletal

muscle, then the maximum cardiac output during exercise at increasing altitude must either stay the same or even increase at increasing altitude in order to limit the effects of the progressive reduction in the arterial oxygen content.

Yet the evidence is absolutely clear. The heart makes the exactly opposite adjustment – maximum cardiac output falls with increasing altitude (Sutton et al. 1988). The reduction is due to the reduction in heart rate; stroke volume and myocardial contractility are, if anything, enhanced during peak exercise at altitude (Reeves et al. 1987, Suarez et al. 1987). Hence the conclusion must be that some currently unrecognized mechanism must exist to insure that the heart does not become “anaerobic” during maximal exercise at any altitude – from sea level to the summit of Mount Everest – in healthy humans.

Interestingly Christensen, but not Dill, interpreted this phenomenon correctly: “Christensen and I differed in our interpretation of his measurements of respiratory and circulatory function in exercise (at altitude). In his opinion, the chief limiting factor is the ventilation of the lungs. In the hardest grade of work at any station, the pulmonary ventilation reached about as high a value as at sea level, while the maximal cardiac output became less as the altitude increased. He thinks this means that the heart has an untapped reserve; it is circulating blood fast enough to carry to the tissues all the oxygen supplied by the lungs” (Dill 1938, p. 170–171).

These studies invite two precise conclusions. First, that the oxygen demands of the skeletal muscles are not the cardinal priority and hence are not “protected” during maximum exercise, at least at extreme altitude. Second, neither the skeletal muscles nor the heart becomes “anaerobic” during maximal exercise

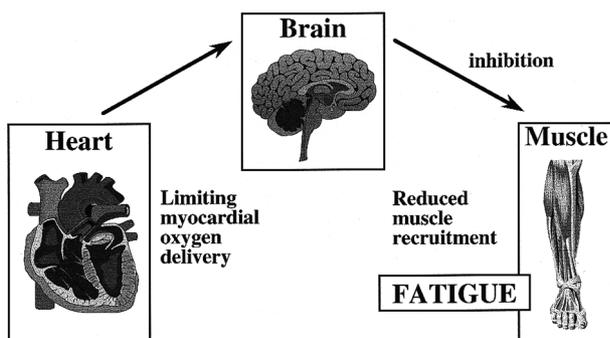


Fig. 4. The “governor” postulated by Hill could be activated by a limiting myocardial oxygen delivery. As a result, there would be a reduced activation of the exercising skeletal muscle by the cerebral motor cortex. There is compelling evidence for the presence of this reflex during exercise at altitude (Kayser et al. 1994) with the result that neither the heart (Sutton et al. 1988, Reeves et al. 1987, Suarez et al. 1987) nor the skeletal muscles (Green et al. 1989) develop “anaerobiosis” during maximal exercise at extreme altitude.

under conditions of hypobaric hypoxia. The sole conclusion must be that some type of “governor”, as originally proposed by A.V. Hill, must limit maximum exercise at altitude. Furthermore, it would be difficult to explain why the same control mechanism should not act similarly during maximum exercise at sea level.

In summary, a number of famous studies have shown that under the precise conditions likely to induce anaerobiosis in either the heart or skeletal muscles – maximal exercise at altitude – neither the heart nor the skeletal muscle show any evidence whatsoever for “anaerobic” metabolism. This unexpected finding can be explained only if there is a “governor”, probably in the central nervous system, whose function is likely to prevent the development of myocardial ischaemia. The same governor could also serve the identical function also at sea level, thereby preventing the development of myocardial ischaemia during maximum exercise at sea level, according to Fig. 2. As Dill (1938) concluded, probably correctly: “The capacity of the heart, as has already been suggested, is restricted at high altitude because of the deficiency in supply of oxygen to it” (p. 15). But the important point is that the heart never actually develops an oxygen deficiency at altitude or at sea level; the governor acts to terminate exercise before that deficiency becomes apparent.

The final confirmation for the presence of this theoretical governor comes from the study of Kayser et al. (1994). They showed that skeletal muscle recruitment, measured as skeletal muscle EMG activity at peak exercise, falls with increasing altitude, but increases acutely with oxygen administration. They conclude: “during chronic hypobaric hypoxia, the central nervous system may play a primary role in limiting exhaustive exercise and maximum accumulation of lactate in blood”. This study therefore proves the existence of the neural effector limb of Hill’s postulated governor (Fig. 4) and its activity during exercise at altitude.

Interestingly, had the human body been designed to function according to the modern physiologists’ cardiovascular/anaerobic model, which requires that anaerobiosis first develops in skeletal muscle before maximal exercise is terminated, no climber would ever have reached the summit of Mount Everest or other high mountains, even with the use of supplemental oxygen. Rather, all would have succumbed to a combination of myocardial ischaemia and cerebral hypoxia whilst their skeletal muscles were exercising vigorously and unrestrainedly, in pursuit of anaerobiosis and fatigue, according to the model depicted in Fig. 1.

Figure 4 therefore summarizes the hypothetical existence and action of the “governor”, first proposed by A.V. Hill. It is postulated that receptor(s) exist in

the heart, to assess the adequacy of any of all of the following: coronary blood flow, coronary oxygen delivery or myocardial or coronary venous oxygen tension. Before any of these reach some predetermined limit, the motor cortex in the brain reduces skeletal muscle activation. As a consequence, skeletal muscle recruitment either fails to rise further or it falls, limiting the work output of the body, indicating the onset of “fatigue”. The fall in work output by the body reduces myocardial oxygen demand and, as a consequence, the threat of myocardial ischaemia is averted.

Alternatively it may be that myocardial adenosine triphosphate (ATP) concentrations are sensed and “defended” in much the same way as appears to be the case for skeletal muscle, as discussed subsequently (Fitts 1994, Spriet et al. 1987). Reduction of myocardial ATP concentrations could lead directly to a reduction in myocardial contractile force as occurs in “myocardial stunning” (Braunwald & Kloner 1982). This could explain the onset of cardiac failure during maximal exercise in persons with coronary artery disease but could not explain why, at altitude, left ventricular function is enhanced during maximal exercise and shows no evidence for “fatigue”.

Accordingly, it is proposed that maximal exercise is limited by a regulated process that terminates exercise before the development of a progressive myocardial ischaemia, that would precede the development of skeletal muscle anaerobiosis. This model further predicts that peak coronary blood flow is an important determinant of maximum exercise performance, and that interventions, including exercise training, that increase the maximum cardiac output probably also increase the maximum coronary blood flow, as their more important effect.

But this model does not exclude the possibility that interventions could also improve exercise performance by altering either skeletal muscle or myocardial contractile function or the efficiency of oxygen utilization, or both (Fig. 5).

Interestingly, the presence of a “governor” preventing the development of anaerobiosis in either heart or skeletal muscle during exercise at altitude has interesting implications for theories of the value of exercise training at altitude. For its presence means that any beneficial effect of altitude training cannot result from repeated exposure of either the heart or exercising skeletal muscle to greater levels of “anaerobiosis” than can be achieved during maximal exercise at sea level. This might explain why there remains considerable controversy about the proven value of high intensity training at altitude (Bonning 1997).

The additional paradoxes that (i) adaptation to extreme altitude is associated with reduced skeletal muscle mitochondrial volume and enzyme content (Green et al. 1989, Oelz et al. 1986, Hoppeler et al. 1990); (ii) the skeletal muscle morphology of altitude-

What limits maximal exercise?

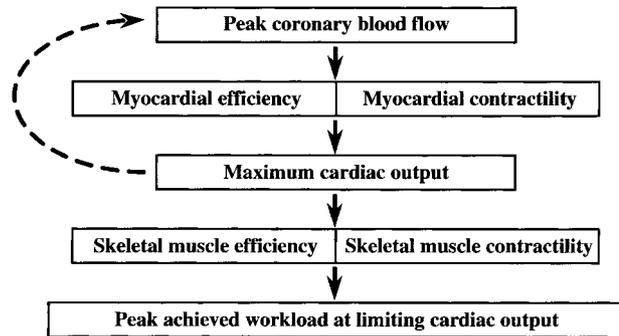


Fig. 5. According to the Hill/Noakes Cardiovascular/Neural Model of Exercise Physiology and Athletic Performance, performance during maximal exercise is ultimately limited by the peak coronary blood flow. However, the actual workrate achieved at that peak coronary blood flow would be determined by the efficiency and contractility of both the heart and the active skeletal muscles.

adapted Nepalese Sherpas is not different from that of acclimatized Caucasian climbers (Kayser et al. 1991) except that (iii) the volume density of skeletal muscle mitochondria is significantly smaller in Sherpas than in untrained sedentary subjects (Kayser et al. 1991), can best be explained if exercise performance at altitude is more likely determined by factors producing superior oxygenation of the heart, than of the skeletal muscles. This model therefore predicts that coronary blood flow and perhaps cardiac mitochondrial mass would be higher in high altitude natives and superior performers at high altitude. This would explain why high altitude natives achieve almost similar cardiac outputs at sea level and at altitude (Vogel et al. 1974), indicating a lesser activation of the “governor” during exercise at altitude.

Indeed the ability of high altitude natives to achieve high heart rates and cardiac outputs during exercise at altitude is associated with “relatively high oxygen tension and saturation” (Vogel et al. 1974) compatible with this postulate that superior myocardial oxygenation might be an important factor determining exercise capacity at altitude.

Prolonged submaximal (endurance) exercise

Many physiologists, notably in the past (Bainbridge 1931, Hill 1927, Dill 1938) but even today (Bassett & Howley 1997), have used this cardiovascular/anaerobic model also to explain the fatigue that develops during prolonged submaximal exercise and consequently have evoked changes in cardiovascular function to explain the mechanisms by which exercise training improves (endurance) performance during prolonged submaximal exercise.

Yet it is not entirely apparent why changes in the

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Table 2. World rankings of male Kenyan runners in 1996

Distance	Rankings in world top 10
800 m	1*, 4, 6, 7, 10
1500 m	4, 5, 7, 8
5000 m	1, 7, 8, 9, 10
10 000 m	3, 4, 5, 6
3000 m steeplechase	1, 2, 3, 4, 5, 6, 8, 9, 10
3000 m	1, 3, 4, 5
Marathon	4, 6

* The naturalized Dane, Wilson Kipketer, is considered a Kenyan for the purposes of this analysis.

Table 3. Performances of Kenyans in the I.A.A.F. world cross-country championships (1986–1997)

Kenyan Senior Men, 1st for the last 12 years (1986–1997)
Kenyan Junior Men, 1st for the last 10 years (1988–1997)
Kenyan Senior Women, 1st 5 times in the last 7 years
Kenyan Junior Women, 1st 8 times in the last 9 years
Total: 35 Championships in 49 competitions including 24 individual championships.

maximum capacity to transport and utilize oxygen must also explain alterations in performance during *submaximal* exercise when oxygen transport cannot be limiting. An early proponent of this (il)logic was Sir Roger Bannister who wrote in 1956 that: “The muscular effort in long-distance running appears to be limited by cardio-respiratory failure as a whole and not by premature failure of any part of the integration” (Bannister 1956).

The obvious point is that, whereas the cardiovascular system could indeed set the limit for maximal exercise performance because of a limiting capacity to increase blood flow first to the heart, and then to the active muscles, it is not clear why cardiovascular function should limit prolonged submaximal exercise when blood flow and oxygen supply to muscle must be adequate. An Olympic analogy from my (African) continent highlights the issues that require debate.

In the years since Wilson Kiprugut won Kenya's first Olympic medal by finishing third in the 800 m at the 1964 Olympic Games, the dominance by Africans, especially Kenyans, in distance running has become a phenomenon unequalled in any other sport in the world (Bale & Sang 1996, Tanser 1997). Two measures of that dominance are provided by the world rankings of male Kenyan track runners in 1996 (Tanser 1997) (Table 2) and of the performances of the men and women's team, both senior and junior, in the World Cross-Country championships over the past 12 years (Tanser 1997) (Table 3). Of particular interest is the almost total dominance of the 3000 m steeplechase by Kenyans (Table 2). Indeed in excess of 90% of the 100 fastest-ever 3000 m steeplechase times in the world have been set by Kenyans. Any

physiological explanation for the Kenyan's success must be able also to explain why these physiological attributes, uniquely common in Kenyan runners, offer an even greater advantage in cross-country events and in the steeplechase, rather than at other distances in which repeated jumping and changes in speed do not occur.

Two studies of Kenyan runners failed to provide a definitive physiological answer for their manifest superiority as distance runners although two of the best Kenyan runners were the most efficient runners yet studied (Saltin 1996, Saltin et al. 1995a,b) (Fig. 6). The overriding conclusion was that the Kenyans' VO_2 max values were not inordinately high; hence, a superior capacity for oxygen consumption during *maximum* exercise did not explain the Kenyans' manifest superiority during more prolonged *submaximal* exercise. In the words of Bengt Saltin, the senior author: “A comparison of some data on some of the very best runners in Kenya during the last decades and world class runners in Scandinavia does not reveal much that was not already known or could be anticipated” (Saltin 1996).

The only other study of elite (South) African distance runners is that of Coetzer et al. (1993). That study, which reported physiological data in the best group of distance runners yet evaluated anywhere in the world, has been largely ignored, for reasons that are not immediately clear. The sole weakness of the study was that the physiological characteristics of el-

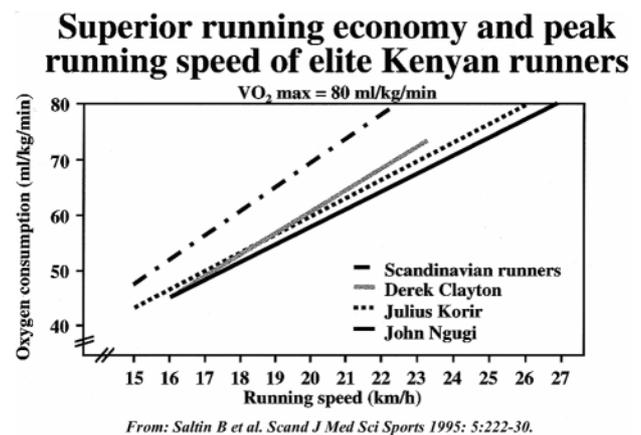


Fig. 6. In comparison to a group of elite Scandinavian runners (the highest running curve), the running economy (VO_2 at any running speed) of former world marathon record holder, Derek Clayton, and two of the greatest Kenyan runners, Julius Korir and John Ngugi, is substantially better. The Biomechanical Model of Exercise Physiology and Athletic Performance predicts that superior running economy would aid elite athletic performance by reducing both the rate at which heat is produced during exercise as well as the extent to which Stretch Shortening Cycle Fatigue develops during both training and racing. This theory predicts that the superior running ability of the Kenyans may relate, at least in part, to characteristics of their skeletal muscle and tendon elasticity (see also Fig. 12).

Table 4. Characteristics of elite South African distance runners

	Middle distance	Long distance
Height (cm)	181	169*
Weight (kg)	70	56*
Aerobic capacity (ml/kg/min)	72	71
Fatigue resistance (% VO ₂ max for 21 km)	82	90*
Muscle fibre composition (% Type I)	63	53

* $P < 0.05$.

Data from Coetzer et al. 1993.

ite black South African distance runners were, perforce, compared to those of white South African middle distance runners. This comparison group was not ideal but was necessary because no other readily available group could match the performances of the black distance runners at any distance over 5 km. But the performances of the black and white runners in that study were at least equal in races of 1–3 km. Table 4 lists the important findings of that study.

The black distance runners were lighter and smaller, as also reported by Saltin (1996), with a slightly lower proportion of type I muscle fibres. But the key finding was that the black runners were able to run substantially faster at all distances beyond 5 km despite VO₂ max values that were the same as those of the middle distance runners. Hence the cardiovascular/anaerobic model failed to explain the superior endurance capacity of the black distance runners in that study and perhaps also in the studies of Saltin and colleagues (Saltin 1996, Saltin et al. 1995a,b).

Rather, the important difference was that the black runners were able to sustain a substantially higher proportion of their VO₂ max when racing. This is shown graphically in Fig. 7, which compares the % VO₂ max sustained by the black and white runners at different racing distances. At distances beyond 5 km, black runners sustained a significantly higher % VO₂ max than did the white runners and the difference increased with increasing racing distance. Thus, the crucial finding was that the black distance runners have superior fatigue resistance, not a higher aerobic capacity (VO₂ max). Hence factors distal to the heart, perhaps in the brain or in the muscles, appear to distinguish the very best runners in the world from those who are almost as good.

Interestingly, physiologists have known for at least two decades that the % VO₂ max that athletes can sustain during exercise is an important predictor of performance (Costill et al. 1973, Davies & Thompson 1979). Yet we have perhaps failed to emphasize, probably because of a devotion to the cardiovascular/anaerobic model, that this is likely to be a more important determinant of performance in prolonged exer-

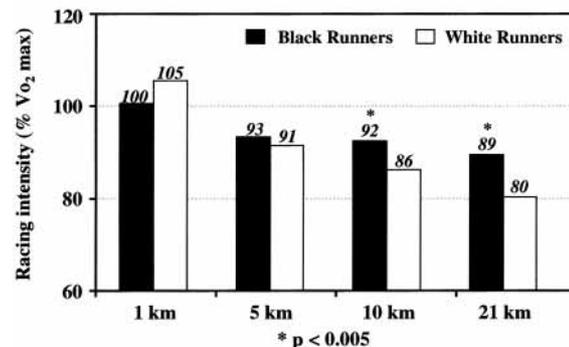
cise than is the VO₂ max alone. Furthermore, it has not been appreciated that the % VO₂ max sustained during exercise is a measure of the athlete's resistance to fatigue.

Hence, the important finding of that study was to show that the cardiovascular/anaerobic model may be unable to discriminate between very good and superior performance in events lasting more than a few minutes and which constitute the bulk of sporting events. It is consistent with the finding that the VO₂ max is a relatively poor predictor of endurance performance in athletes whose abilities are relatively homogenous (Noakes 1988, 1997, 1998, Davies & Thompson 1979, Noakes et al. 1990). The failure stems from the inability of this model to measure or predict fatigue resistance during prolonged *submaximal* exercise on the basis of physiological variables and performance measured during a single bout of progressive, maximal exercise to exhaustion. This confirms that the VO₂ max test does not measure *all* the physiological variables determining success during more prolonged exercise.

Further support for this explanation can be surmised from other information in Fig. 7, which shows that these athletes run at 100% or greater of their VO₂ max in race distances of 1–2 km. Yet it is not at those distances that the Kenyans' dominance is most apparent. If the Kenyans' success was due to their unusually high VO₂ max values, one would expect Kenyans also to be dominant at race distances of 800 m to the mile, which is not the case (Table 2).

Indeed, comparison of the performances of the

Superior Fatigue Resistance of Black SA Distance Runners



From: Coetzer et al. *J Appl Physiol* 1993; 75: 1822-7.

Fig. 7. The % VO₂ max sustained by elite South African black and white distance runners falls with increasing racing distance. However, black runners sustain a significantly higher % VO₂ max at race distances of 10 km and 21 km, indicating superior fatigue resistance. Such superiority cannot be explained by the Cardiovascular/Anaerobic Model of Exercise Physiology and Athletic Performance as VO₂ max values of black and white athletes in this study (Coetzer et al. 1993) were the same.

great British runner, Sebastian Coe, with those of a current Kenyan champion, Daniel Komen, provides further evidence for this interpretation. Remarkably, Komen's best time for the mile is 1 s faster than Coe's best. Yet the real performance difference occurs at 5 km: Komen's best time is 83 s faster than Coe's best, a performance difference of 10%.

In summary, there are serious theoretical flaws in the proposed cardiovascular/anaerobic model of exercise physiology and athletic performance (Noakes 1998), not least because the model predicts that a "plateau" in cardiac output must develop before skeletal muscle anaerobiosis can begin to occur. But any "plateau" in cardiac output requires that myocardial ischaemia be present either to cause that plateau (according to the theory that anaerobiosis limits muscle function) or as a result of it, as the cardiac output determines both coronary and skeletal muscle blood flow. As myocardial ischaemia has never been shown to develop during maximal exercise in healthy humans, so it would seem unlikely that skeletal muscle anaerobiosis can develop during progressive exercise to exhaustion (Noakes 1998). Rather, it would seem that "fatigue" during maximal exercise of short duration is part of a regulated neural process that prevents the development of myocardial ischaemia during maximal exercise.

Whilst this mechanism is designed to protect the heart from myocardial ischaemia, only indirectly does it determine the actual peak work rate achieved during maximal exercise (Fig. 5). The actual peak work rate achieved will depend on the "quality" of the skeletal and cardiac muscle. Superior myocardial contractility and efficiency of oxygen use would increase the maximum cardiac output achieved at any maximum (limiting) coronary flow. Similarly at any maximum skeletal muscle blood flow, superior contractility and efficiency of skeletal muscle contraction would increase the peak work rate achieved at that maximum cardiac output. This hypothesis forms what might be called the Cardiovascular/Neural Recruitment Model of Exercise Physiology and Athletic Performance.

Thus, this analysis of the traditional cardiovascular/anaerobic model of exercise performance leads to the alternate hypothesis that superior fatigue resistance, determined perhaps by the central nervous system or skeletal muscle contractile function, might explain superior performance in events lasting more than a few minutes. This superior fatigue resistance cannot be predicted by the cardiovascular/anaerobic model which uses exercise tests of short duration and in which the fatigue resistance component of endurance performance is not measured. By extension, it would seem that fatigue resistance is not causally determined by the magnitude of the athlete's cardiovascular capacity. There is also no logical reason to believe that fatigue resistance during *submaximal* ex-

ercise is determined by either the presence or absence of skeletal muscle anaerobiosis.

Accordingly, changes in exercise performance that result from endurance training are unlikely to be determined solely by changes in cardiovascular function, with the exception that increases in maximum coronary blood flow would likely be crucial for any increases in maximal cardiac output and hence in VO_2 max. It is of interest that the vasodilator capacity of the major epicardial coronary vessel is greatly increased in veteran long distance runners (Haskell et al. 1993). Perhaps this indicates that an important effect of endurance training, possibly at some critical growth periods, may be to increase maximum coronary blood flow as shown in animal models (Scheuer & Tipton 1997).

The energy supply/energy depletion model

The energy supply model

The central premise in the cardiovascular/anaerobic model is that it is the provision of a substrate (oxygen) to muscle that limits exercise performance so that fatigue is a direct consequence of a failure of oxygen delivery to the exercising muscles. A subtle extension of this idea produces a second model which proposes that fatigue during high intensity exercise may, alternatively, result from the inability to supply another substrate (ATP) at rates sufficiently fast to sustain exercise. Nobel Laureate A. V. Hill, whose research in the 1920s was directly responsible for the development of the cardiovascular/anaerobic model of exercise performance, also wrote: "The fact remains, however, that the chief factor in many forms of athletic achievement is the supply of energy and its proper and economic utilization" (Hill 1927, p. 237). Dr Peter Snell, Olympic Gold medallist in the 800 and 1500 m and former world record holder has stated similarly: "Performance in middle and (long) distance running ultimately depends upon the runner's capacity to produce energy for the duration of the event, and on the efficiency with which that energy is translated to running velocity. Thus the purpose of training is to improve the energy delivery systems, according to the demands of the event and to improve running economy" (Snell 1997).

Thus, this model predicts that performance in events of different durations is determined by the capacity to produce energy (ATP) by the different metabolic pathways including the phosphagens, oxygen-independent glycolysis, aerobic glycolysis and aerobic lipolysis. Superior performance is then explained by a greater capacity to generate ATP in the specific metabolic pathway(s) that predominates during that activity. Thus, the sprinter is assumed to have a greater capacity to generate ATP from the intramuscular phosphagen stores and from oxygen-indepen-

dent glycolysis, whereas the ultramarathon runner has a superior capacity to oxidize fat (aerobic lipolysis) (Hawley & Hopkins 1995).

Whether this hypothesis is true is uncertain as it has yet to be systematically evaluated. To prove this model would require (i) that the metabolic capacities of these different pathways be shown to be causally related to performance in events lasting the different durations; (ii) that the specific metabolic pathways be shown to adapt predictably with specific training, and (iii) that these adaptations alone explain the changes in performance that result from training with exercises lasting the different durations. Until these studies are completed, this model remains hypothetical, but interesting. It must be remembered that the truth of this model would need to disprove the opposing model, described in the previous section, which holds that maximal exercise performance is a regulated process limited by a failure of central neural recruitment. Fatigue at exhaustion, caused by a failure of central recruitment, will always *appear* to be due to a failure of ATP production unless the alternate possibility is studied (and excluded) simultaneously.

The argument against this model has been introduced elsewhere (Noakes 1997). In short, the prediction of this model is that exercise must terminate when muscle ATP depletion occurs (Fitts 1994), that is when the muscle develops rigor. Yet here again, the evidence appears clear. ATP concentrations, even in muscles forced to contract under ischaemic conditions, do not drop below about 60% of resting values (Fitts 1994, Spriet et al. 1987, Hochachka 1994) indicating that muscle ATP concentrations are “defended” in order to prevent the development of skeletal muscle rigor. As Fitts (1994) has concluded: “The overriding evidence suggests that (the high energy phosphates) do not participate in the fatigue process; that fatigue produced by other factors reduces the ATP utilization rate before ATP becomes limiting. The most compelling evidence for this conclusion is that cell ATP rarely falls below 70% of the pre-exercise level, even in cases of exercise fatigue” (p. 82).

Hence, it appears that the rate of ATP demand by the contracting muscles can never exceed the maximum rate of ATP supply because of the close matching of ATP demand to the available ATP supply (Spriet et al. 1987, Hochachka 1994).

There is an obvious analogy to the centrally situated neural “governor” that prevents the development of myocardial ischaemia during maximal exercise at either sea level or altitude. The difference is that the “governor” identified by Spriet et al. (1987) is clearly located in the periphery and acts even in muscles stimulated to contract with an externally applied current.

It is of interest that the presence of this peripheral “governor” is an essential component of the cardiovascular/anaerobic model as originally conceived from the work of Hill and his colleagues, and still widely promoted (Fig. 1). This hypothesis holds that when the rate of ATP production by oxidative sources becomes inadequate, high rates of “anaerobic” glycolytic ATP production produce metabolites, particularly H^+ , which interfere with energy production and cross-bridge cycling causing fatigue and a failure of muscle contraction (Fitts 1994). In this way, muscle contraction fails not because of a failure of central recruitment (as predicted by the Cardiovascular/Neural Recruitment Model – previous section), but because of a peripherally located inhibition of muscular contraction. Proponents of this model can cite a large body of evidence showing that a number of metabolites can interfere with muscle cross-bridge cycling measured *in vitro* in isolated muscle fibres (Fitts 1994). The necessary assumption is that skeletal muscle contracting *in vitro* in the absence of an intact neural system behaves exactly as it would *in vivo* when the influences from the central nervous system are intact. But there is a body of evidence that is not compatible with these assumptions and conclusions.

For example, one of the few studies to evaluate critically this hypothesis that metabolites, particularly H^+ , can induce skeletal muscle fatigue, is that of Mannion et al. (1995). They found that there is a wide range of muscle pH concentrations reached at exhaustion during intense exercise showing that if an accumulation of H^+ limits high intensity exercise *in vivo*, then “considerable interindividual differences must exist in the pH sensitivities of the various processes involved” (Mannion et al. 1995, p. 98).

Next, they found that in contrast to the prediction from *in vitro* studies, subjects with the highest proportion of type II muscle fibres were able to exercise to the lowest muscle pH concentrations. In contrast, *in vitro* studies have suggested that type I muscle fibres are more resistant to acidosis than are type II fibres. Finally, the authors found that subjects with a greater skeletal muscle buffering capacity did not accumulate more lactate during maximal exercise; nor were they able to exercise for longer than did those with lesser muscle buffering capacity. They concluded that “if acidosis makes any contribution to the fatigue during performance of this (high intensity) type of exercise, it is an indirect one ...”.

One possibility is that such exercise is terminated by a central governor responding to factors other than skeletal muscle pH. Under these circumstances there would be no relationship between the onset of fatigue and muscle acidosis. This would not negate the established finding that *in vivo*, acidosis inhibits crossbridge cycling (Fitts 1994). It would mean only that this mechanism is not relevant in exercising

humans, perhaps because exercise terminates for other reasons, in particular to prevent the development of myocardial ischaemia, before the limiting skeletal muscle pH is reached.

Other relevant findings include the study of Bogdanis et al. (1995), who showed that recovery of muscle function following maximal sprint cycling exercise was related to recovery of muscle phosphocreatine concentrations and unrelated to muscle pH concentrations during recovery. In addition, Vollestad et al. (1988) showed that the gradual decline in maximum force generation in subjects performing repeated submaximal contractions for 40–70 min was “not due to lactacidosis or lack of substrates for ATP resynthesis and must have resulted from excitation/contraction coupling failure ...”. Yet terminal exhaustion was associated with depletion of intramuscular phosphagen stores, but without evidence for acidosis.

In summary, a metabolic basis limiting high intensity exercise of short duration is widely assumed but incompletely documented. There is a need to establish whether those metabolic factors that appear to limit muscle function *in vitro* also play a role *in vivo* when the muscle is also under the influence of the central nervous system. Thus, the possible contribution of neural factors to this form of fatigue needs to be excluded before results from *in vitro* studies are extrapolated, without qualification, to the *in vivo* condition.

The energy depletion model

The related energy depletion model of exercise performance is specific for exercise lasting more than 2–3 h. It holds, in essence, that: “Depletion of endogenous carbohydrate stores has been shown to be a limiting factor in the ability to perform long term exercise” (Costill et al. 1973). The findings that support this conclusion are (i) that fatigue during prolonged exercise is *associated* with depletion of liver (causing hypoglycaemia) or muscle glycogen stores (Fitts 1994, Bosch et al. 1993, Coggan & Coyle 1987, Coyle et al. 1986, Tsintzas et al. 1996), or both; (ii) that reversal of hypoglycaemia allows exercise to continue (Coggan & Coyle 1987, Coyle et al. 1986, Tsintzas et al. 1996, Christensen & Hansen 1939) and (iii) that pre-exercise muscle glycogen supercompensation (carbohydrate-loading) (Hawley et al. 1997) or carbohydrate ingestion during exercise (Coyle et al. 1986), or both (Bosch et al. 1996), delays the onset of fatigue and improves exercise performance.

However, it must be remembered that relatively few carbohydrate-loading studies have been conducted with an adequate placebo control group. It seems highly improbable that neither athletes nor researchers are completely unaware of the widely reported benefits of carbohydrate-loading and that such knowledge is without effect on the findings of

these trials. Indeed, two of the first such trials which included adequate placebo-controlled groups have both failed to find any ergogenic effect of pre-exercise carbohydrate loading (Burke et al. 1999, Hawley et al. 1997) under experimental conditions when such an effect might have been expected.

The finding that the reversal of hypoglycaemia alone allows exercise performance to continue (Coggan & Coyle 1987, Christensen & Hansen 1939) proves conclusively that liver glycogen depletion is one form of energy (carbohydrate) depletion that can definitely limit exercise performance. Interestingly, the rapidity with which the reversal of hypoglycaemia restores exercise performance indicates that a central neural “governor” must be active, similar to that activated during high intensity exercise at altitude. However, this control would be activated by changes in blood glucose concentrations and would act to prevent continuing high rates of muscle contraction and blood glucose oxidation that would further reduce the blood glucose concentration, risking hypoglycaemic cerebral damage.

But, in as much as no technique has yet been devised that will instantly reverse muscle glycogen depletion, in the same way that intravenous glucose infusion or oral glucose ingestion rapidly reverses hypoglycaemia, so it is impossible to prove conclusively that muscle glycogen depletion *alone* limits prolonged exercise performance. It needs to be remembered that there are many physiological changes besides muscle glycogen depletion that develop during exercise, and that any or all of these could contribute to, or cause fatigue during prolonged exercise. In addition, relatively little attention has been paid to the possible role of central (neural) fatigue (Davis & Bailey 1997) as *the* factor limiting prolonged exercise when muscle glycogen concentrations are also very low. Future studies of the energy depletion model need to show that central neural factors do not cause the fatigue currently ascribed to the development of muscle glycogen depletion during prolonged exercise.

Thus, the belief that muscle glycogen depletion causes fatigue is an interesting hypothesis that is supported logically by the findings that subjects who are exhausted during prolonged exercise develop very low muscle glycogen content (Fitts 1994, Bosch et al. 1993, Tsintzas et al. 1996, Burke et al. 1999), and that muscle glycogen is the metabolic fuel required for sustained high intensity exercise (Bosch et al. 1993). It would seem logical to assume that the two are causally linked at exhaustion during prolonged exercise; namely, that the near absence of muscle glycogen in exhausted subjects explains why they are unable to maintain, let alone increase, their exercise intensity at exhaustion. The finding that the vast majority of the modern and historical carbohydrate-loading studies show that this technique improves endurance per-

formance, presumably by increasing muscle glycogen utilization and delaying the onset of terminal muscle glycogen depletion, strongly supports the theory (Hawley et al. 1997). However, the possibility that part or all of these findings could also result from a placebo effect, acting through the central nervous system, needs to be considered (Burke et al. 1999). In addition, it is unclear how the inability to produce ATP at sufficiently high rates from one fuel source can explain this form of fatigue, given that skeletal muscle ATP concentrations remain high at exhaustion (as they do in all other forms of exhaustion (Fitts 1994)).

Nevertheless, there is a body of evidence that conflicts with the predictions of this hypothesis. For example, the classic study of Coyle et al. (1986) showed that athletes ingesting carbohydrate terminated exercise after 4 h when their muscle glycogen concentrations and rates of carbohydrate oxidation were the same as values measured 1 h earlier when the athletes were not exhausted. Another study found that athletes who adapted to a high fat diet were able to exercise to significantly lower muscle glycogen concentrations at exhaustion than when they were carbohydrate adapted (Lambert et al. 1994).

Conversely, Helge et al. (1996) showed that previously untrained subjects who trained on a high fat diet for 7 weeks before switching to a high carbohydrate diet for one week increased their pre-exercise muscle glycogen concentrations by 44% with only a small further increase in performance between the seventh and eighth weeks of the trial. Furthermore, performance was still substantially worse in fat-adapted subjects than it was in subjects who trained for 8 weeks on a high carbohydrate diet and whose pre-exercise muscle glycogen concentrations were 32% lower than fat-adapted athletes exposed to a high carbohydrate diet for one week.

In addition, exercise performance evaluated on 3 occasions in both dietary groups terminated before there was marked muscle glycogen depletion. The authors concluded: "Factors other than carbohydrate availability are responsible for the differences in endurance time between the groups" (p. 303); and "These observations also indicate that fatigue during prolonged moderately intense exercise does not always seem to be closely related to glycogen depletion, as is usually stated" (Christensen & Hansen 1939, Bergstrom et al. 1967). In his extensive review, Fitts (1994) similarly concludes: "It seems unlikely that muscle glycogen depletion, low blood glucose, and the resultant decline in carbohydrate oxidation is an exclusive fatigue factor during prolonged exercise". He does, however, acknowledge that "a possibility exists that muscle glycogen depletion is causative in fatigue via a mechanism independent of its role in energy production" (p. 83).

In addition, to my knowledge, no study has yet established that training improves endurance performance exclusively by increasing body carbohydrate stores and by delaying the onset of carbohydrate depletion during prolonged exercise in humans, although this finding has been reported in rats (Fitts et al. 1975) whose metabolism is substantially different from that of humans.

Similarly, it is currently difficult to explain performance in ultra-endurance events, especially the final 42 km running leg of the 226 km Ironman triathlon events according to this model, which holds that exercise of moderately high intensity is not possible once there is marked muscle glycogen depletion. After cycling at $40 \text{ km} \cdot \text{h}^{-1}$ for 4.5 h, the lead cyclists would be expected to have near total muscle glycogen depletion according to data from laboratory studies (Bosch et al. 1993). Yet the best performers in that event are able to run at close to $16 \text{ km} \cdot \text{h}^{-1}$ for a further 160 min. This probably represents an exercise intensity of $>66\% \text{ VO}_2 \text{ max}$. The studies of Rauch et al. (1998) and O'Brien et al. (1993) suggest that total carbohydrate oxidation during very prolonged exercise of up to 6 h duration exceeds the estimated carbohydrate stores in liver and active muscle by up to 100%. Either these calculations are incorrect, or other sources of carbohydrate, in addition to those in the active muscles and liver, must contribute to fuel oxidation in events lasting more than 4–6 h. One possibility is that lactate oxidation of glycogen stored in the inactive skeletal muscles contributes a substantial additional amount to fuel use during very prolonged exercise (Rauch et al. 1998). How increased lactate oxidation contributes to performance is not known.

Similarly, provision of carbohydrate at high rates intravenously (Coggan & Coyle 1987) cannot extend exercise performance indefinitely. Whilst this could support the argument that muscle glycogen is *the* important carbohydrate source limiting exercise performance, the alternate possibility is that another factor, unrelated to depletion of body carbohydrate stores, perhaps a rising body temperature discussed subsequently, or central neural fatigue induced by other factors, may also limit endurance performance.

Perhaps there is a necessary *rate* of carbohydrate oxidation that is required to sustain a specific exercise intensity and that progressive whole body carbohydrate depletion lowers that rate, inducing fatigue. If this is correct, then fatigue resistance during prolonged exercise could be due to the capacity to sustain a higher rate of carbohydrate oxidation, and hence a *higher* respiratory quotient (RQ) during prolonged exercise. Again, this model suffers from the persisting logical impasse that a failure to generate ATP sufficiently rapidly must cause exercise to terminate because of muscle ATP depletion and rigor, a phenomenon which does not occur (Fitts 1994).

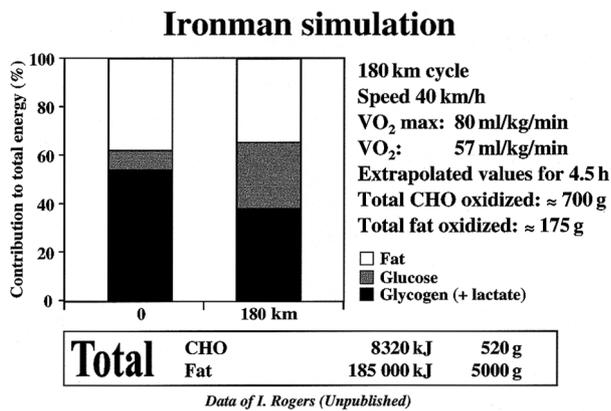


Fig. 8. Laboratory simulation suggests that an elite cyclist cycling 180 km in 4 h 30 min would cycle at an oxygen consumption of 57 ml/kg/min. Based on the measured contribution of fat and carbohydrate oxidation to this energy requirement (columns on the left of the figure), this would require the oxidation of about 700 g of carbohydrate and 175 g of fat. This compares to the predicted maximum body stores of 520 g of carbohydrate and 5000 g of fat in an elite triathlete. Hence this model predicts that the elite triathlete must commence the running leg of the triathlon with very low or absent whole body carbohydrate stores.

An equally plausible alternate theory postulates that superior endurance capacity may be determined by the exact opposite; by a superior capacity to oxidize fat and hence maintain a *lower* RQ during prolonged exercise. The latter possibility is supported by at least some evidence. In the studies of Bosch et al. (1993), those athletes unable to complete 3 h of exercise at 70% VO₂ max after carbohydrate-loading had significantly higher RQ during exercise and were therefore characterized by an inability to sustain high rates of fat oxidation during prolonged exercise. Indeed, simulated metabolic balance studies for the 226 km Hawaiian Ironman triathlon suggest it to be very likely that the capacity to oxidize fat at high rates will influence running speed late in the race when calculations suggest that muscle glycogen stores are likely to be depleted.

Figure 8 shows the expected energy metabolism during 4.5 h cycling at an oxygen consumption of 57 ml/kg/min. This is equivalent to cycling at 40 km/h and completing the 180 km cycle leg of the Ironman triathlon in the time necessary to be amongst the race leaders. The data for this simulation come from laboratory data measured on elite South African cyclists (I. Rodgers. Unpublished data).

The simulation predicts that after 4.5 h of cycling an elite male Ironman triathlete would be expected to have oxidized about 700 g of carbohydrate and 175 g of fat. This compares to predicted whole body carbohydrate and fat stores of 520 g and 5000 g, respectively. Hence this model predicts that, at the end of the cycle leg, an elite athlete would have depleted his

body carbohydrate stores, yet must still run 42.2 km at close to 16 km/h if he wishes to be successful.

Our other laboratory data suggest that after 4.5 h of such exercise, the carbohydrate contribution to whole body energy metabolism would comprise a blood glucose oxidation rate of 1.2 g/min (21 kJ/min) and a lactate oxidation rate of 0.6 g/min (10.5 kJ/min). Together with the average maximum rate of fat oxidation that we have measured after 6 h of laboratory cycling (0.76 g/min; 28 kJ/min), this provides a total rate of energy production of 59.5 kJ/min. This would provide energy at a rate sufficient to sustain a running speed of approximately 12 km/h, sufficient to complete the 42 km marathon leg of the Ironman triathlon in 3 h 30 min (Fig. 9). To equal the best marathon time yet run in that race, the athlete would be required to oxidize fat at a rate of 1.15 g/min (Fig. 10). This rate is approximately 50% faster than we have measured in cyclists in our laboratory.

Accordingly, if this metabolic model of fatigue in the Ironman triathlon is correct, then the difference between running the final marathon in 2 h 40 min versus 3 h 30 min may simply be a 51% (0.4 g/min)

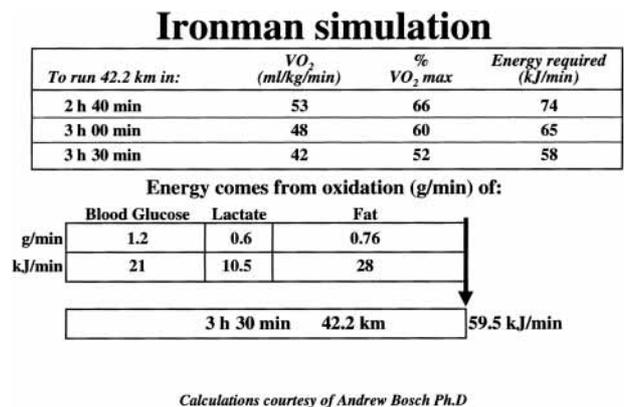


Fig. 9. The top panel of this figure shows the oxygen requirement (VO₂), the exercise intensity (% VO₂ max) and the rates of energy expenditure that would be sustained by a world-class athlete completing the final 42.2 km marathon running leg of the Ironman triathlon in times of either 2 h 40 min; 3 h 00 min; or 3 h 30 min. Laboratory studies suggest that after a 3.8 km swim and a 180 km cycle, the athlete's body carbohydrate stores would be depleted so that energy for the running leg would come from oxidation of (mainly ingested) blood glucose, blood lactate and from circulating free fatty acids derived from muscle and adipose tissue triglyceride. To complete the marathon running leg of the Ironman triathlon in 3 h 30 min, the athlete would have to sustain a VO₂ of 42 ml/kg/min (52% VO₂ max), equivalent to an energy expenditure of 59.5 kJ/min. Laboratory simulations (Fig. 8) suggest that under these conditions of near total carbohydrate depletion, peak glucose oxidation rates are 1.2 g/min and peak lactate (from glycogen) oxidation rates are 0.6 g/min. If these data for the maximum capacity to oxidize glucose and lactate in the carbohydrate-depleted state are correct, then to sustain the rate of energy expenditure necessary to run the marathon in 03:30:00, the carbohydrate-depleted triathlete must oxidize fat at a rate of 0.76 g/min.

Ironman simulation

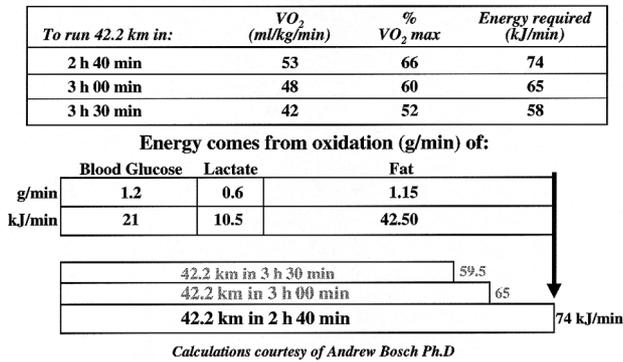


Fig. 10. To complete the marathon running leg of the Ironman triathlon in 2 h 40 min, currently the fastest running time yet recorded in the Hawaiian Ironman triathlon, the athlete would have to sustain a VO₂ of 53 ml/kg/min (66% VO₂ max), equivalent to an energy expenditure of 74 kJ/min. If the maximum capacity to oxidize glucose and lactate in the carbohydrate-depleted state is unchanged from values given in Fig. 9, then to sustain such a high rate of energy expenditure, the athlete must oxidize fat at a rate of 1.15 g/min. This model predicts that the superior ability of the elite Ironman triathlete may result from a much greater (approximately 50%) capacity to oxidize fat than has been measured in our laboratory experiments of very prolonged laboratory exercise involving sub-elite athletes (Rauch et al. 1998).

greater capacity to oxidize fat when body carbohydrate and, especially, muscle glycogen stores are depleted. Of course, this model does not negate the requirement that such high rates of fat oxidation can only be achieved if the central nervous system continues to recruit an appropriately large number of muscle fibres able to produce an appropriate force.

In summary, the human body has a limited capacity to store carbohydrates. In addition, high rates of carbohydrate oxidation are necessary to sustain high rates of energy expenditure (in the fed state). Furthermore, studies of very prolonged exercise (6 h) show that rates of carbohydrate oxidation remain high in athletes who ingest appropriate amounts of carbohydrate during exercise (Rauch et al. 1998). As both muscle and liver glycogen depletion occur in the fatigued state, it has popularly been assumed that there is a direct causal relationship between, especially, muscle glycogen depletion and the development of fatigue during prolonged exercise. Yet some findings suggest that this relationship may not be strictly causal under all circumstances. In addition is the logical impasse which requires that any energy depletion model predicts that exercise must terminate when muscle ATP depletion occurs, leading to muscle rigor. In the absence of such evidence, it would seem that factors in addition to depletion of body carbohydrate store may contribute to, or even cause, fatigue during prolonged exercise.

At present, no study has conclusively established that training-induced changes in the capacity to store and metabolize carbohydrate during prolonged exercise are causally related to training-induced changes in performance in humans, although this relationship is frequently assumed. The alternate possibility is that the capacity to oxidize fat at high rates when body carbohydrate stores are depleted, may delay fatigue and determine performance during exercise of moderately high intensity that lasts more than 4 h and is typified by ultradistance running and triathlon events (Fig. 8–10).

In this regard, the “crossover” concept of Brooks and Mercier (1994) is of particular interest. These authors argue that fuel choice during exercise “crosses over” from predominantly fat to exclusively carbohydrate at exercise intensities above about 80% VO₂ max. They conclude that training produces different effects depending on the intensity of the exercise being evaluated. At exercise intensities below their “crossover” point, training increases fat oxidation whereas at higher exercise intensities, training increases the capacity to burn carbohydrates. According to their concept, exercise at higher intensities (>70% VO₂ max) would be limited as an example of energy supply limitations (an inability to sustain high rates of carbohydrate oxidation), whereas exercise at lower intensities would be limited by muscle glycogen depletion. In fact the models are identical – according to the energy (muscle glycogen) depletion model, fatigue results from an inability to supply ATP sufficiently rapidly from fat oxidation (failure of energy supply from fat oxidation). The opposite pertains during high intensity exercise. In fact, as argued here and elsewhere (Fitts 1994), both the energy supply and the energy depletion models predict that muscle ATP depletion limits exercise. This does not occur; hence, the models must be too simplistic to explain what has been found.

The muscle recruitment (central fatigue)/muscle power model

The two previous models are based on the assumption that it is either the delivery of substrate either in blood (oxygen) or via the glycolytic and oxidative pathways (ATP) that limits exercise performance. The steps of (il)logic that have influenced these assumptions have been described (Noakes 1997, 1998). It remains difficult to prove whether or not either of these models is correct. Yet both continue to dominate, perhaps subconsciously, research and teaching in the exercise sciences, often to the exclusion of competing possibilities.

An alternate view is that it is not the rate of supply of substrate, either oxygen or fuel, to muscle that limits its performance but rather the processes involved

in skeletal muscle recruitment, excitation and contraction.

A failure of central nervous system recruitment of skeletal muscle forms the basis for the “central (nervous system) fatigue” hypothesis (Davis & Bailey 1997). This model holds that the brain concentration of serotonin (and perhaps other neurotransmitters, including dopamine and acetylcholine) alters the density of the neural impulses reaching the exercising muscles, thereby influencing the rate at which fatigue develops, especially during exercise. Alternatively, there may be inhibitory reflexes arising from the exercising muscles and which feedback to the spinal cord, reducing skeletal muscle recruitment at the level of the α -motoneuron. The evidence for both mechanisms has been extensively reviewed (Davis & Bailey 1997).

In brief, a number of studies indicate that manipulation of central nervous system neurotransmitter concentrations, in particular increasing dopamine and reducing serotonin concentrations, can enhance exercise performance whereas the opposite impairs performance. In addition, there is direct evidence for reduced central neural drive to muscle after fatiguing muscle contractions (Behm & St-Pierre 1997, Baker et al. 1993, Newham et al. 1991). This evidence is sufficiently persuasive to believe that central nervous system fatigue contributes to fatigue during prolonged exercise lasting tens of minutes to hours.

The clear evidence that fatigue at high altitude is caused by reduced central nervous system recruitment of the exercising muscles has been described. It is very likely that the fatigue that occurs during exercise in the heat is also likely limited by a failure of central recruitment as this form of fatigue cannot be explained by any other model. Thus, there is now substantive evidence that each athlete can store only some limiting amount of heat before being forced to reduce the exercise intensity or alternatively cease exercising altogether (Nielsen et al. 1990, 1993, 1997). There is no evidence from these studies that exhaustion under these conditions is associated with either skeletal muscle “anaerobiosis” or energy depletion. Rather, that it is either the total heat accumulated or its rate of accumulation that limits exercise is confirmed by intervention studies which show that pre-cooling improves performance (Nielsen et al. 1997, Booth et al. 1997) whereas pre-heating has the opposite effect (Gonzalez-Alonso et al. 1999).

In summary, there is sufficient evidence to suggest that a reduced central nervous system recruitment of the active muscles terminates maximum exercise at high altitudes (and probably also at sea level). The same mechanism likely terminates exercise in the heat when the body temperature reaches some limiting maximum and also when hypoglycaemia develops. In all these examples, reduced central recruitment of

muscle would function to prevent organ damage (Noakes 1997).

However, the contrasting finding that skeletal muscle recruitment, measured as skeletal muscle electromyographic activity, rises during exercise at the same workload (Takaishi et al. 1994), is usually interpreted as evidence for a progressive failure of muscle fibre contractile function requiring additional fibre recruitment if the required force is to be sustained. Thus, this model holds that there is a progressive peripheral fatigue for which the central nervous system makes an appropriate adjustment.

Yet these studies usually impose a constant workload on the subject for the duration of the activity. But competitive sport does not usually involve such constant workrates; workrates tend to vary in a random, stochastic way (Palmer et al. 1994). We found that during prolonged exercise, which includes bouts of self-chosen high intensity exercise designed to simulate stochastic exercise, there is a progressive reduction in power output during the bouts of high intensity exercise (Burke et al. 1999). This strongly suggests central fatigue in which, during bouts of exercise requiring a near maximum effort, there is an inadequate increase in central neural drive to compensate for the expected reduction in the power output of the fatiguing skeletal muscle fibres (peripheral fatigue). That a relatively small percentage of the available muscle mass is ever recruited, even during maximal exercise (Sloniger et al. 1997), remains a perplexing but relatively unrecognized enigma. Proponents of any model of peripheral limitations for exercise performance need to explain why the body does not recruit all its available muscle mass to produce the necessary force under varying exercise conditions as so-called “peripheral fatigue” develops.

In summary, one interpretation of the muscle recruitment (central fatigue) model is that changes in central neurotransmitters induce fatigue simply as a natural consequence of prolonged exercise and changes in the relative balance of the different (ergogenic and ergolytic) neurotransmitters in the brain. No specific physiological value or importance is assigned to this phenomenon.

Alternatively, I have argued that a reduced central activation of the exercising muscles may be necessary to protect the human under specific conditions (Noakes 1997, 1998). It is postulated that these control mechanisms are necessary (i) to prevent myocardial ischaemia during exercise at high intensity; (ii) to prevent the development of muscle ATP depletion and muscle rigor during high intensity exercise; (iii) to prevent myocardial ischaemia or cerebral hypoxia during exercise at altitude; (iv) to prevent a fall in blood pressure during exercise in patients with chronic heart failure; (v) to prevent heatstroke during

prolonged exercise in the heat, and (vi) to prevent glucopaenic brain damage during prolonged exercise when hypoglycaemia results from liver glycogen depletion. The likely mechanism of control is through the regulation either of skeletal muscle recruitment or of excitation/contraction coupling in the muscle.

Muscle power model

This model holds that muscle contractile capacity, that is the ability of individual muscle cross-bridges to generate force, is not the same in the muscles of all humans, so that those with superior athletic ability have muscles with a superior capacity to generate force (superior contractility) by the individual cross-bridges of the different muscle fibres. This model is well accepted by cardiac physiologists, the majority of whom would argue that calcium delivery to the myofibres and the activity of the enzyme involved in ATP hydrolysis, myosin ATPase, rather than substrate supply, determine the contractile state of the myocardium in both health and disease (Opie 1998).

I could find only one recent statement using this model to explain superior athletic performance, specifically in swimming: "First, the strength of the muscles used in swimming is a major determinant of success in events from 50 m to 1500 m. Though this may not seem surprising, it must be remembered that strength *per se* does not dictate fast swimming. The forces generated by the muscle must be effectively applied to the water if they are to propel the body. Thus, strength specificity is the key to swimming success" (Costill et al. 1992).

There are rather few studies of the contractility of skeletal muscle isolated from athletes. These studies generally show that endurance training reduces skeletal muscle contractility (Fitts et al. 1989, Widrick et al. 1996). This establishes that skeletal muscle contractility is not an immutable characteristic of the different muscle fibre types (Fitts & Widrick 1996). By extension, one might speculate that the contractility of the specific muscle fibre types might differ between athletes of different abilities in different sporting disciplines, compatible with this muscle power model.

In summary, these two models of exercise performance predict that changes in exercise performance may result from increased skeletal muscle recruitment resulting from enhanced central neural drive, or from increased muscle contractile function resulting from biochemical adaptations in muscle that increase either force production or the rate of sarcomere shortening, or both.

However, the increase in performance resulting from these adaptations would occur only to the extent that the cardiovascular limits for exercise performance were not exceeded, according to the Cardiovascular/Neural Model.

The biomechanical model

There is growing interest in the role of muscles as elastic energy return systems which function both as springs and torque producers during exercise (Pennisi 1997, Roberts et al. 1997). Central to this model is the prediction that the greater the muscle's capacity to act as a spring, the less torque it must produce and hence the more efficient it is. The more efficient, more elastic muscle will enhance exercise performance, especially in weight-bearing activities, by slowing (i) the rate of accumulation of those metabolites that may cause fatigue during exercise, and (ii) the rate of rise of body temperature, thereby delaying the achievement of the core temperature that prevents the continuation of exercise.

This new information underscores another important logical weakness of the cardiovascular/anaerobic model for explaining enhanced endurance performance. For that model predicts that superior performance during prolonged exercise results from an *increased* oxygen delivery to muscle and an *increased* rate of energy and hence heat production. Thus, according to that model, the price of running faster is that more heat must be produced. But a higher rate of heat production would induce fatigue prematurely due to excessive heat accumulation, according to the findings of Nielsen and colleagues (Nielsen et al. 1993, 1997). A more logical biological adaptation would be to *reduce* the rate of oxygen consumption and hence the rate of heat production by increasing the athlete's efficiency (economy) of movement.

Indeed, if the rate of heat accumulation limits exercise performance under specific conditions, then factors that slow the rate at which heat accumulates when running fast should enhance performance. Two such factors are small size (Dennis & Noakes 1999) and superior running economy. A smaller size reduces the amount of heat produced when running at any speed. When environmental conditions limit the capacity for heat loss, smaller runners will be favoured (Dennis & Noakes 1999).

Further evidence supporting this argument that heat accumulation is a factor limiting endurance performance, is the finding that race times in both the marathon (Noakes 1992) and the longer distance track races including the 3000 m steeplechase and the 10 000 m (McCann & Adams 1997) deteriorate as the environmental heat load increases. Thus, there is an inverse relationship between the environmental heat load, measured as the Wet Bulb Globe Temperature Index, and the reduction in race performance.

Therefore, according to this model, the more economical the athlete, the faster he or she will be able to run before reaching a limiting body temperature. A number of studies indicate that the best endurance athletes are also frequently the most economical

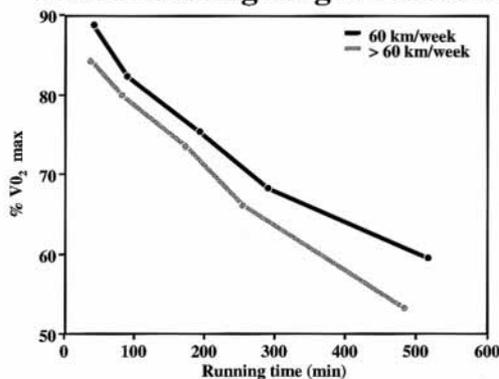
(Noakes 1992; Fig. 6). Indeed, most training studies show that improvements in running economy are perhaps the most likely response to training, especially in those who are already well-trained (Svedenhag & Sjodin 1985). This adaptation allows the athlete to run faster at the same oxygen consumption; thus, he or she completes a given distance more rapidly for the same average rate of heat accumulation but a reduced overall heat expenditure. This would be advantageous under conditions in which the heat load on the athlete increases (during the day).

Figure 11 shows that this adaptation may indeed exist. In a cross-sectional study of recreational (not elite) ultramarathon runners, it was found that those who trained more were more economical and hence could run faster at the same oxygen consumption or % VO_2 max. During competition, the better trained athletes ran at the same or a slightly lower % VO_2 max but completed the races in a shorter time (Scrimgeour et al. 1986). Hence being more economical, not having a higher VO_2 max, appears to be a more logical technique to enhance endurance performance.

In contrast, a high aerobic capacity, often a marker of poor running economy (Noakes 1988, 1992), would likely cause more rapid rates of heat accumulation and hence the more rapid onset of fatigue during prolonged exercise. This finding alone could explain why the best marathon runners usually have VO_2 max values in the range of 63–74 ml/kg/min. Less economical runners with higher VO_2 max values (Noakes 1988) have not necessarily been more successful (Noakes et al. 1990, Noakes 1992).

Thus, this model predicts that success in endurance

Training may improve racing performance without altering fatigue resistance



Scrimgeour AG et al. *Eur J Appl Physiol* 1986; 55:202-9.

Fig. 11. Ultramarathon athletes who trained more than 60 $\text{km} \cdot \text{week}^{-1}$ (the lower running curve) outperformed less well trained athletes with similar VO_2 max values because their superior running economy allowed them to race faster but at a lower % VO_2 max, thereby finishing races of 10–90 km in shorter times. Hence changes in running economy with training allow athletes to run faster and at a lower exercise intensity, the opposite of what is usually assumed.

events is not likely to result from training that makes the athlete ever more powerful with a larger muscle mass and greater VO_2 max. A more likely adaptation would be to reduce the athlete's size and increase his or her running efficiency. That runners believe they run better when lighter, is well known.

Another African analogy for this prediction is provided by the physiological strategy that the cheetah has evolved to survive as a successful predator. The cheetah, whose chase is terminated by an elevated rectal temperature after running at up to 100 $\text{km} \cdot \text{h}^{-1}$ for less than a minute (Taylor & Rowntree 1973), succeeds because of the animal's small size and probably a high degree of running economy (due to elasticity provided by the flexible spine). Thus, laboratory experiments showed that when the cheetah's rectal temperature reached 40.5–41°C, "the cheetahs refused to run ... They would simply turn over with their feet in the air and slide on the tread(mill) surface" (Taylor & Rowntree 1973).

The small size of the cheetah and its likely high running economy slows its rate of heat accumulation just sufficiently for it to outrun the smaller gazelles (≈ 25 kg) on which it preys and whose escape is also restrained by a rising body temperature (Taylor & Lyman 1972). Thus the chase between the gazelle and the cheetah is probably decided by which individual animal accumulates heat more slowly during the chase. In contrast, the heavier, more muscular lion has evolved a different co-operative, hunting strategy, targeting larger but slower mammals.

Perhaps the point is that smallness and greater running economy would seem to be a technique used to increase endurance capacity in one animal, the cheetah. Logic suggests that this technique may also be applicable to elite human athletes.

A second component of the biomechanical models stems from the accumulating evidence that repeated high velocity, short duration eccentric muscle contractions, as occur during running, induce a specific form of fatigue that develops during running races and is measurable for at least 7 days after a marathon race (Komi & Nicol 1998; Nicol et al. 1991).

Characteristics of this fatigue are a failure of the contractile capacity of the exercised muscles with a reduced tolerance to muscle stretch and a delayed transfer from muscle stretch to muscle shortening in the stretch/shortening cycle. As a result, the durations of both the braking and push-off phases in the running stride are increased, leading to mechanical changes in the stride with landing occurring on a more extended leg but with greater subsequent knee flexion.

As these abnormalities persist for many days after the race (Fig. 12), they cannot be explained by acute changes in oxygen or substrate delivery to the muscles, or by the elevated body temperature during

exercise, as required by the first 3 models. Rather, Komi and Nicol (1998) conclude that: "Stretch shortening fatigue results usually in a reversible muscle damage process and has considerable influence on muscle mechanics, joint and muscle stiffness as well as on reflex intervention". Thus any evaluation of fatigue resistance, especially in weight-bearing activities like running, needs to consider this specific form of stretch/shortening cycle fatigue.

To return to the African analogy, empirical observation of the running stride and the anatomical structure of the lower limb of Kenyan runners suggests, at least to this author, that an evaluation of the elastic elements of the legs of elite Kenyan runners and their resistance to stretch/shortening cycle fatigue would likely be very rewarding.

For example, it appears that African athletes generally train harder than do Caucasian runners (Tanser 1997, Coetzer et al. 1993). Especially the training volumes and intensities of the Kenyan runners (Tanser 1997) are unmatched by other athletes. But to achieve such training volumes, there must be superior resistance to the stretch/shortening cycle damage proposed by Komi & Nicol (1998), both in training and in marathon racing.

Hence, another possibility is that the more elastic muscles of elite distance runners are better able to resist eccentrically induced damage in training. This may allow more intensive daily training and hence superior adaptations to training. That same superiority would also enhance performance during competitive racing by delaying the onset of the stretch/shortening cycle fatigue that is an inevitable consequence of repeated eccentric muscle contractions.

In summary, the biomechanical model predicts that superior performance, especially in a weight-bearing activity like running, may be influenced by the capacity of the muscles to act as elastic energy return systems. Changes in the efficiency and durability of this process would (i) enhance movement economy and reduce the rate of heat production during exercise, thereby enhancing exercise capacity by slowing the rate at which the body temperature rises when environmental conditions are severe; (ii) enhance the quality of training by allowing more rapid recovery from stretch/shortening cycle fatigue so that more frequent bouts of intensive training can be undertaken and (iii) enhance fatigue resistance during competition by increasing resistance to that form of muscle damage that develops during repeated cycles of stretch/shortening contractions.

The psychological/motivational model

This model holds that the ability to sustain exercise performance results from a conscious effort and is often included as a component of the central fatigue

Fatigue during running: the biomechanical model

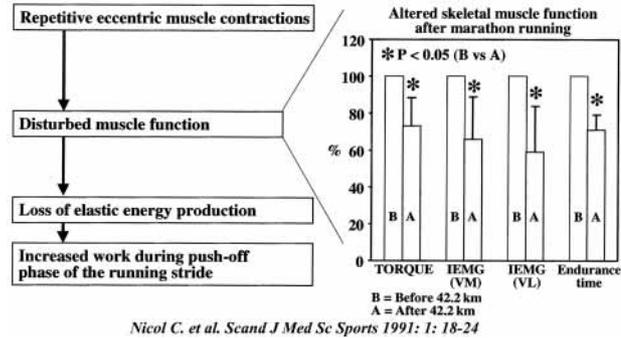


Fig. 12. According to the Biomechanical Model of Exercise Physiology and Athletic Performance, repetitive eccentric muscle contractions, as occurs in the quadriceps and calf muscles during running, produces altered muscle function with a loss of elastic energy production, requiring an increased work during the push-off phase of the running stride. The studies of Komi & Nicol (1998) and Nicol et al. (1991) show that there is a persistent reduction in peak torque and endurance performance time of the quadriceps muscle of subjects after they completed a 42.2 km marathon race. Integrated electromyographic activity (IEMG) in both the vastus medialis (VM) and the vastus lateralis (VL) was also reduced after the marathon. These studies show that there is a long-term effect of marathon running on skeletal muscle performance that persists after the muscle's energy stores are replaced, indicating that this abnormality is not explained by the Energy Depletion Model. It seems probable that the marathon "wall" (Noakes 1992) is probably explained by these alterations in skeletal muscle function.

hypothesis (Davis & Bailey 1997). But it conflicts with one proposal of the muscle recruitment model, which holds that exercise performance is regulated at a sub-conscious level and which exists, in part, to prevent conscious override that might damage the human.

It would seem that exercise performance must include at least some component that can be influenced by conscious effort. The dichotomy of physiology and psychology has generally prevented adequate laboratory evaluation of this model. Any studies showing an ergogenic effect of any placebo intervention on exercise performance would prove that this model contributes, in part, to athletic performance. Any detailed discussion of this model is beyond the scope of this author's expertise.

Summary

This paper has reviewed some of the models currently promoted to describe how exercise performance is limited by fatigue and enhanced by training. The argument advanced here is that until the factors determining both fatigue and athletic performance are established more definitely, it remains difficult to define which training adaptations are the most important for enhancing exercise performance, or how training

should be structured to maximize those adaptations. This remains a serious weakness for the practical application of much research in the exercise sciences.

However, within the constraints provided by these models, it would seem that the following training adaptations would contribute to enhanced exercise performance with training.

(a) *Cardiovascular/neural recruitment model*: Relevant training adaptations would be those that result in an increased VO_2 max and skeletal muscle blood flow during both maximal and prolonged submaximal exercise.

According to the model I have presented here, a plateau in coronary flow would appear to be the factor that limits the cardiac output, and hence the VO_2 max. Thus, an essential component of training would be to increase the maximal coronary flow.

However, even if the maximal coronary blood flow limits the maximal cardiac output, the actual peak workrate or peak VO_2 max that is achieved will depend on the contractile state of the myocardium and the efficiency with which the heart is able to convert that maximum coronary flow into a peak cardiac output. Similarly, the actual maximum work of which the muscles are capable at the VO_2 max, will equally depend on the economy and contractility of the skeletal muscles. Thus, optimum training would not only maximize coronary flow but also the efficiency and contractility of both the heart and skeletal muscles.

Incidentally, according to this model, interventions such as EPO therapy, blood re-infusion, or the administration of oxygen, all of which improve performance, would act in part by increasing oxygenation of the myocardium at maximal exercise, thereby allowing a greater cardiac output and hence a higher VO_2 max.

(b) *Energy supply model*: The important training adaptation would be an increased capacity to store and utilize metabolic substrates during exercise with a greater capacity to produce ATP and prevent a reduction in muscle phosphagen concentrations under all exercise conditions. Adaptations in different metabolic pathways would be necessary for optimizing performance in activities of different durations and intensities.

(c) *Energy depletion model*: A reduced rate of carbohydrate utilization during prolonged exercise would enhance performance by delaying the onset of whole body carbohydrate depletion. This model predicts that an increased capacity to burn fat during prolonged exercise would enhance endurance performance. That this model can also

be considered as an “energy supply” model was described above.

(d) *Muscle recruitment and muscle power models*: These models predict that increased skeletal muscle contractile function, a peripheral effect, or increased neural recruitment, a central nervous system effect, would be advantageous for performance during exercise.

(e) *The biomechanical model*: A key predictor of the biomechanical model is that increased movement economy would improve performance by reducing the rate of heat accumulation during exercise. This model also explains why a reduced body mass would improve performance during prolonged exercise as it slows the rate of heat accumulation.

The importance of elastic return energy, especially in weight-bearing sports, and the identification of stretch/shortening cycle fatigue suggests that training may improve elasticity and delay stretch/shortening cycle fatigue, perhaps by altering the elastic component of skeletal muscle, tendons and ligaments.

Conclusion

The importance of these conceptual models is that they indicate that different physiological systems may determine performance under different exercise conditions. Hence, exercise physiologists need to consider all these models when they design studies to determine which are the most important physiological, biochemical, neural and other factors determining the changes in exercise performance that result from training.

More importantly, this review shows that many findings are incompatible with the predictions of one or more of these models. Rather than simply continuing to accept these inconsistencies uncritically, the modern generation of exercise physiologists should challenge old dogmas and so approach more closely the unattainable truth (Noakes 1997, 1998).

Key words: fatigue; VO_2 max; heart; skeletal muscle; glycogen; fat metabolism; muscle recruitment; contractility.

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References

- Acevedo EO, Goldfarb AH. Increased training intensity effects on plasma lactate, ventilatory threshold, and endurance. *Med Sci Sports Exerc* 1989; 21: 563–8.
- Bainbridge FA. The physiology of muscular exercise. 3rd edn. Bock AV, Dill DB, eds. London: Longmans, Green and Co. 1931: 1–272.
- Baker AJ, Kostov KG, Miller RG, Weiner MW. Slow force recovery after long-duration exercise: metabolic and activation factors in muscle fatigue. *J Appl Physiol* 1993; 74: 2294–300.
- Bale J, Sang J. Kenyan running. London, U.K.: Frank Cass, 1996: 1–209.
- Bannister RG. Muscular effort. *Br Med Bull* 1956; 12: 222–5.
- Bassett DR, Howley ET. Maximal oxygen uptake: “classical” versus “contemporary” viewpoints. *Med Sci Sports Exerc* 1997; 29: 591–603.
- Behm DG, St-Pierre DMM. Effects of fatigue duration and muscle type on voluntary and evoked contractile properties. *J Appl Physiol* 1997; 82: 1654–61.
- Bergstrom JB, Hermansen L, Hultman E, Saltin B. Diet, muscle glycogen and physical performance. *Acta Physiol Scand* 1967; 71: 140–50.
- Bogdanis GC, Nevill ME, Boobis LH, Kalomy HK, Nevill AM. Recovery of power output and muscle metabolites following 30 s of maximal sprint cycling in man. *J Physiol (London)* 1995; 482: 67–480.
- Boning D. Altitude and hypoxia training – a short review. *Int J Sports Med* 1997; 18: 565–70.
- Bosch AN, Dennis SC, Noakes TD. Influence of carbohydrate loading on fuel substrate turnover and oxidation during prolonged exercise. *J Appl Physiol* 1993; 74: 1921–7.
- Bosch AN, Weltan SM, Dennis SC, Noakes TD. Fuel substrate kinetics of carbohydrate loading differs from that of carbohydrate ingestion during prolonged exercise. *Metabolism* 1996; 45: 415–23.
- Booth J, Marino F, Ward JJ. Improved running performance in hot humid conditions following whole body precooling. *Med Sci Sports Exerc* 1997; 29: 943–9.
- Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. *Circulation* 1982; 66: 1146–9.
- Brooks GA, Mercier J. Balance of carbohydrate and lipid utilization during exercise: the “crossover” concept. *J Appl Physiol* 1994; 76: 2253–61.
- Burke LM, Hawley JA, Schabort EJ, St Clair Gibson A, Mujika I, Noakes TD. Carbohydrate loading failed to improve 100-km cycling performance in a placebo-controlled trial. *J Appl Physiol* in press.
- Christensen EH, Forbes WH. *Skand Arch Physiol* 1937; 76: 75. Quoted in: Dill DB. *Life, heat and altitude*. Cambridge, MA: Harvard University Press, 1938: 168.
- Christensen EH, Hansen O. *Arbeitsfähigkeit und Ernährung*. *Skand Arch Physiol* 1939; 81: 160–71.
- Christensen EH, Hansen O. Hypoglykämie, arbeitsfähigkeit und Ernährung. *Skand Arch Physiol* 1939; 81: 172–9.
- Coetzer P, Noakes TD, Sanders B, et al. Superior fatigue resistance of elite black South African distance runners. *J Appl Physiol* 1993; 75: 1822–7.
- Coggan AR, Coyle EF. Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. *J Appl Physiol* 1987; 63: 2388–95.
- Costill DL, Thomason H, Roberts E. Fractional utilization of the aerobic capacity during distance running. *Med Sci Sports Exerc* 1973; 5: 248–52.
- Costill DL, Bennett A, Branam G, Eddy D. Glucose ingestion at rest and during prolonged exercise. *J Appl Physiol* 1973; 34: 764–9.
- Costill DL, Maglischo EW, Richardson AB. *Swimming*. London: Blackwell Science Ltd, 1992: 1–214.
- Coyle EF, Coggan AR, Hemmert MK, Ivy JL. Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *J Appl Physiol* 1986; 61: 165–72.
- Daniels JT, Yarbrough RA, Foster C. Changes in VO_2 max and running performance with training. *Eur J Appl Physiol* 1978; 39: 249–54.
- Davies CTM, Thompson MW. Aerobic performance of female marathon and male ultramarathon athletes. *Eur J Appl Physiol* 1979; 61: 611–7.
- Davis JM, Bailey SP. Possible mechanisms of central nervous system fatigue during exercise. *Med Sci Sports Exerc* 1997; 29: 45–57.
- Dennis SC, Noakes TD. Advantages of smaller body mass for distance running performances in warm, humid conditions. *Eur J Appl Physiol* 1999; 79: 280–4.
- Dill DB. *Life, heat and altitude*. Cambridge, MA: Harvard University Press, 1938: 1–211.
- Edwards HT. Lactic acid in rest and work at high altitude. *Am J Physiol* 1936; 116: 367–75.
- Fitts RH, Booth FW, Winder WW, Holloszy JO. Skeletal muscle respiratory capacity, endurance, and glycogen utilization. *Am J Physiol* 1975; 228: 1029–33.
- Fitts RH, Costill DL, Gardetto PR. Effect of swim exercise training on human muscle fiber function. *J Appl Physiol* 1989; 66: 465–75.
- Fitts RH. Cellular mechanisms of muscle fatigue. *Physiol Rev* 1994; 74: 49–94.
- Fitts RH, Widrick JJ. Muscle mechanics: adaptations with exercise-training. *Exerc Sports Sci Rev* 1996; 24: 427–73.
- Gonzalez-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *Eur J Appl Physiol* 1999; 86: 1032–9.
- Green HJ, Sutton JR, Young P, Cymerman A, Houston CS. Operation Everest II: muscle energetics during maximal exhaustive exercise. *J Appl Physiol* 1989; 66: 142–50.
- Green HJ, Sutton JR, Cymerman A, Young PM, Houston CS. Operation Everest II: adaptations in human skeletal muscle. *J Appl Physiol* 1989; 66: 2454–61.
- Haskell WL, Sims C, Myll J, Bortz WM, Goar FGS, Alderman EL. Coronary artery size and dilating capacity in ultradistance runners. *Circulation* 1993; 87: 1076–82.
- Hawley JA, Hopkins WG. Aerobic glycolytic and aerobic lipolytic power systems. *Sports Med* 1995; 19: 240–50.
- Hawley JA, Myburgh KH, Noakes TD, Dennis SC. Training techniques to improve fatigue resistance and enhance endurance performance. *J Sports Sci* 1997; 15: 325–33.
- Hawley JA, Schabort EJ, Noakes TD, Dennis SC. Carbohydrate-loading and exercise performance. An update. *Sports Med* 1997; 24: 73–81.
- Helge JW, Richter EA, Kiens B. Interaction of training and diet on metabolism and endurance during exercise in man. *J Physiol* 1996; 492: 293–306.
- Hill AV, Long CNH, Lupton H. Muscular exercise, lactic acid, and the supply and utilization of oxygen: parts I–III. *Proc Royal Soc Bri* 1924; 97: 438–75.
- Hill AV. *Living machinery*. London: G Dell and Sons Ltd, 1927: 1–241.
- Hill AV, Long CHN, Lupton H. Muscular exercise, lactic acid and the supply and utilisation of oxygen: parts VII–VIII. *Proc Royal Soc Bri* 1924; 97: 155–76.
- Hochachka PW. The lactate paradox: analysis of underlying mechanisms. *Ann Sports Med* 1989; 4: 184–8.
- Hochachka PW. Muscles as molecular

Noakes

- and metabolic machines. Boca Raton, Florida, USA: CRC Press, 1994.
- Holloszy JO, Coyle EF. Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *J Appl Physiol* 1984; 56: 831–8.
- Hoppeler H, Kleinert E, Schlegel C, et al. Morphological adaptations of human skeletal muscle to chronic hypoxia. *Int J Sports Med* 1990; 1: S3–9.
- Houston ME, Bentzen H, Larsen H. Interrelationships between skeletal muscle adaptations and performance as studied by detraining and retraining. *Acta Physiol Scand* 1979; 105: 163–70.
- Kayser B, Hoppeler H, Claassen H, Cerretelli P. Muscle structure and performance capacity of Himalayan Sherpas. *J Appl Physiol* 1991; 70: 1938–42.
- Kayser B, Narici M, Binzoni T, Grassi B, Cerretelli P. Fatigue and exhaustion in chronic hypobaric hypoxia: influence of exercising and muscle mass. *J Appl Physiol* 1994; 76: 634–40.
- Komi PV, Nicol C. Stretch-shortening cycle fatigue. *Biomechanics and Biology of Movement* 1998.
- Lambert EV, Speechley DP, Dennis SC, Noakes TD. Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet. *Eur J Appl Physiol* 1994; 69: 287–93.
- Mannion AF, Jakeman PM, Willan PLT. Skeletal muscle buffer value, fibre type distribution and high intensity exercise performance in man. *Exp Physiol* 1995; 80: 89–101.
- McCann DJ, Adams WC. Wet bulb globe temperature index and performance in competitive distance runners. *Med Sci Sports Exerc* 1997; 29: 955–61.
- Moore RL, Thacker EM, Kelley GA, et al. Effect of training/detraining on submaximal exercise responses in humans. *J Appl Physiol* 1987; 63: 1719–24.
- Newham DJ, McCarthy T, Turner J. Voluntary activation of human quadriceps during and after isokinetic exercise. *J Appl Physiol* 1991; 71: 2122–6.
- Nicol C, Komi PV, Marconnet P. Fatigue effects of marathon running on neuromuscular performance. I. Changes in muscle force and stiffness characteristics. *Scand J Med Sci Sports* 1991; 1: 10–7.
- Nielsen B, Savard G, Richter EA, Hargreaves M, Saltin B. Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol* 1990; 69: 1040–6.
- Nielsen B, Hales JRS, Strange S, Christensen NJ, Warberg J, Saltin BN. Human circulatory and thermoregulatory adaptations with heat acclimatization and exercise in a hot, dry environment. *J Physiol* 1993; 460: 467–85.
- Nielsen B, Strange S, Christensen NJ, Warberg J, Saltin B. Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflügers Arch* 1997; 434: 49–56.
- Noakes TD. Implications of exercise testing for prediction of athletic performance: a contemporary perspective. *Med Sci Sports Exerc* 1988; 20: 319–30.
- Noakes TD, Myburgh KH, Schall R. Peak treadmill running velocity during the VO_2 max test predicts running performance. *J Sports Sci* 1990; 8: 35–45.
- Noakes TD. *Lore of Running*. 2nd edn. Cape Town: Oxford University Press, 1992: 1–535.
- Noakes TD. Challenging beliefs: ex Africa semper aliquid novi. *Med Sci Sports Exerc* 1997; 29: 571–90.
- Noakes TD. Maximal oxygen uptake: “classical” versus “contemporary” viewpoints. A rebuttal. *Med Sci Sports Exerc* 1998; 30: 1381–98.
- O’Brien MJ, Viguie CA, Mazzeo RS, Brooks GA. Carbohydrate dependence during marathon running. *Med Sci Sports Exerc* 1993; 25: 1009–17.
- Oelz O, Howald H, Di Prampero PE, et al. Physiological profile of world-class high altitude climbers. *J Appl Physiol* 1986; 60: 1734–42.
- Opie LH. *The Heart: Physiology from cell to circulation*. 3rd edn. New York: Lipincott-Raven, 1998: 1–637.
- Palmer GS, Hawley JA, Dennis SC, Noakes TD. Heart rate response during a 4-d cycle stage race. *Med Sci Sports Exerc* 1994; 26: 1278–83.
- Pennisi E. A new view of how leg muscles operate on the run. *Science* 1997; 275: 1067.
- Pugh LGCE. Cardiac output in muscular exercise at 5,800 m (19,000 ft). *J Appl Physiol* 1964; 19: 441–7.
- Ramsbottom R, Williams C, Fleming N, Nute MLG. Training induced physiological and metabolic changes associated with improvements in running performance. *Br J Sports Med* 1989; 23: 171–6.
- Raskoff WJ, Goldman S, Cohn K. The “Athletic Heart”. Prevalence and physiological significance of left ventricular enlargement in distance runners. *JAMA* 1976; 236: 158–62.
- Rauch LHG, Hawley JA, Noakes TD, Dennis SC. Fuel metabolism during ultra-endurance exercise. *Pflügers Arch* 1998; 436: 211–9.
- Reeves JT, Groves BM, Sutton JR, et al. Operation Everest II: preservation of cardiac function at extreme altitude. *J Appl Physiol* 1987; 63: 531–9.
- Roberts JR, Marsh RL, Weyand PG, Taylor CR. Muscular force in running turkeys: The economy of minimizing work. *Science* 1997; 275: 1113–5.
- Rowell LB. *Human cardiovascular control*. New York: Oxford University Press, 1993: 1–500.
- Saltin B, Gollnick PD. Skeletal muscle adaptability: Significance for metabolism and performance. In: Peachey LD, Adrian RH, Geiger SR (Eds). *Handbook of Physiology*, Section 10, Skeletal Muscle. Bethesda, Maryland: American Physiological Society, 1983.
- Saltin B, Kim CK, Terrados N, Larsen H, Svedenhag J, Rolf C. Morphology, enzyme activities and buffer capacity in leg muscles of Kenyan and Scandinavian runners. *Scand J Med Sci Sports* 1995a; 5: 222–30.
- Saltin B, Larsen H, Terrados N, et al. Aerobic exercise capacity at sea level and at altitude in Kenyan boys, junior and senior runners compared with Scandinavian runners. *Scand J Med Sci Sports* 1995b; 5: 209–21.
- Saltin B. Adaptive responses to training at medium altitude; with a note on Kenyan runners and a proposal for a multi-centre study. Revised and extended version of Saltin B. *Exercise and the Environment: focus on altitude*. *Res Quart Exerc Sport* 1996; 67: 1–10.
- Scheuer J, Tipton CM. Cardiovascular adaptations to physical training. *Annu Rev Physiol* 1997; 39: 221–51.
- Scrimgeour AG, Noakes TD, Adams B, Myburgh K. The influence of weekly training distance on fractional utilization of maximum aerobic capacity in marathon and ultramarathon runners. *Eur J Appl Physiol* 1986; 55: 202–9.
- Sloniger MA, Cureton KJ, Prior BM, Evans EM. Lower extremity muscle activation during horizontal and uphill running. *J Appl Physiol* 1997; 83: 2073–9.
- Snell P. Training of the distance runners. Second Annual Congress of the European College of Sport Science. Copenhagen, August 20–23, 1997. Abstract book, p. 57.
- Spriet LL, Soderlund K, Bergstrom M, Hultman E. Anaerobic energy release in skeletal muscle during electrical stimulation in men. *J Appl Physiol* 1987; 62: 611–5.
- Stray-Gundersen J, Musch TI, Haidet GC, Swain DP, Ordway GA, Mitchell JH. The effect of pericardiectomy on maximal oxygen consumption and maximal cardiac output in untrained dogs. *Circulation Res* 1986; 58: 523–30.
- Suarez J, Alexander JK, Houston CS. Enhanced left ventricular systolic performance at high altitude during Operation Everest II. *Am J Cardiol* 1987; 60: 137–42.
- Sutton JR, Reeves JT, Wagner PD, et al.

- Operation Everest II: oxygen transport during exercise at extreme simulated altitude. *J Appl Physiol* 1988; B64: 1309–21.
- Svedenhag J, Sjodin B. Physiological characteristics of elite male runners in and out of season. *Can J Appl Sport Sci* 1985; 10: 127–33.
- Takaishi T, Yasuda Y, Moritani T. Neuromuscular fatigue during prolonged pedalling exercise at different pedalling rates. *Eur J Appl Physiol* 1994; 69: 154–8.
- Tanser T. Train hard, win easy. Mountain Views, California: Tafnews Press Inc., 1997: 1–198.
- Taylor CR, Lyman CP. Heat storage in running antelopes: independence of brain and body temperatures. *Am J Physiol* 1972; 22: 114–7.
- Taylor CR, Rowntree VJ. Temperature regulation and heat balance in running cheetahs: a strategy for sprinters? *Am J Physiol* 1973; 224: 848–51.
- Tsintzas O, Williams C, Boobish L, Greenhaff P. Carbohydrate ingestion and single muscle fiber glycogen metabolism during prolonged running in men. *J Appl Physiol* 1996; 81: 801–9.
- Vogel JA, Hartley LH, Cruz JC, Hogan RP. Cardiac output during exercise in sea-level residents at sea level and high altitude. *J Appl Physiol* 1974; 36: 169–72.
- Vogel JA, Hartley LH, Cruz JC. Cardiac output during exercise in altitude natives at sea level and high altitude. *J Appl Physiol* 1974; 36: 173–6.
- Vollestad NK, Sejersted OM, Bahr R, Woods JJ, Bigland-Ritchie B. Motor drive and metabolic responses during repeated submaximal contractions in humans. *J Appl Physiol* 1988; 64: 1421–7.
- Westgarth-Taylor C, Hawley JA, Rickard S, Myburgh KH, Noakes TD, Dennis SC. Metabolic and performance adaptations to interval training in endurance-trained cyclists. *Eur J Appl Physiol* 1997; 75: 298–304.
- Weston AR, Myburgh KH, Lindsay FH, Dennis SC, Noakes TD, Hawley JA. Skeletal muscle buffering capacity and endurance performance after high-intensity interval training by well-trained cyclists. *Eur J Appl Physiol* 1997; 75: 7–13.
- Widrick JJ, Trappe SW, Blaser CA, Costill DL, Fitts RH. Isometric force and maximal shortening velocity of single muscle fibers from elite master runners. *Am J Physiol* 1996; 271(2 Pt 1): C666–75.
- Widrick JJ, Trappe SW, Costill DL, Fitts RH. Force-velocity and force-power properties of single muscle fibers from elite master runners and sedentary men. *Am J Physiol* 1996; 271(2 Pt 1): C676–83.