Physiological function and neuromuscular recruitment in elite South African distance runners

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Abstract
Physiological studies of elite and sub-elite black South African runners show that these athletes are typically about 10–12 kg lighter than white athletes and that they are able to sustain higher exercise intensities for longer than white runners. Such superior performance is not a result of higher VO2max values and hence cannot be due to superior oxygen delivery to the active muscles during maximal exercise, as is predicted by the traditional cardiovascular/anaerobic/catastrophic models of exercise physiology. A marginally superior running economy is also unlikely to be a crucial determinant in explaining this apparent superiority. However, black athletes are able to sustain lower rectal and thigh, but higher mean skin, temperatures during exercise. Furthermore, when exercising in the heat, lighter black athletes are able to maintain higher running speeds than are larger white runners matched for running performance in cool environmental conditions. According to the contrasting theory that the body acts as a complex system during exercise, the superiority of black African athletes should be sought in an enhanced capacity to maintain homeostasis in all their inter-dependent biological systems despite running at higher relative exercise intensities and metabolic rates. In this case, any explanation for the success of East African runners will be found in the way in which their innate physiology, training, environment, expectations and genes influence the function of those parts of their subconscious (and conscious) brains that appear to regulate the protection of homeostasis during exercise as part of an integrative, complex biological system.

Keywords: maximum oxygen consumption; economy; size; lactate; fatigue resistance; central governor; muscle recruitment

Introduction
Humans, scientists included, interpret the phenomena they observe according to preconceptions, or models, of how they believe the world works1–4. In this way, our preconception of what we already believe to be true determines how we will interpret any information that we observe and hence the nature of the truth that we will accept. Thus, at a subconscious level, the human brain acts as a self-censoring device, ultimately determining how we interpret the daily phenomena that we observe. Popper5 was one of the first to popularize this concept. He coined the term ‘paradigm’ to identify any model that is widely accepted as the prevailing ‘truth’ by any dominant intellectual majority, as might be the case, for example, in a specific scientific discipline.

The current model or paradigm of the factors believed to determine long-distance running performance has been termed the AV Hill cardiovascular/anaerobic/catastrophic model4,6,7. This model posits that exercise performance is ultimately determined by the capacity of the athlete’s heart to pump as large a volume of blood as possible to the exercising muscles. The maximal functional capacity of the
heart then establishes ('limits') the maximal amount of oxygen that the exercising muscles are able to consume. Since, according to this model, it is the rate of oxygen consumption (VO$_2$) by the exercising muscles that determines their capacity to produce force, so it must be that the very best athletes will be those whose muscles receive and then consume the greatest volumes of oxygen during exercise. This value is known as the maximum oxygen consumption (VO$_{2\max}$).

It is also recognized that some athletes can run faster or further whilst consuming less oxygen and hence less fuel. As a result they are considered to be 'more economical'. Thus a second construct is the belief that it is also helpful if the athlete not only has a very high VO$_{2\max}$, but also runs at a minimum energy cost; that is, with greater economy.

However, the two aforementioned variables are not independent; athletes with a very high capacity to consume oxygen, i.e. a high VO$_{2\max}$, are frequently uneconomical$^{6,8}$ whereas those with a lower VO$_{2\max}$ may be both more economical and, on occasion, even better athletes. This apparent paradox has been explained$^{6,8}$ and, by itself, does not exclude the possibility that the magnitude of oxygen consumption by the muscles is still an important determinant of their function$^9$.

The key to this paradox is that athletes also differ in the peak work rates that they achieve during maximum exercise$^{6,8,10}$. Thus, as shown in Fig. 1, athletes with a high VO$_{2\max}$ can achieve that value either because they are economical and also achieve a high peak work rate (line B in Fig. 1), or they may be uneconomical and achieve the same high VO$_{2\max}$ but at a much lower peak work rate (line A in Fig. 1). Clearly, however, the best athlete will be someone who achieves both a very high peak work rate and a high VO$_{2\max}$ despite being very economical (line C in Fig. 1). As we shall see, this example of athlete C is the most common finding in elite African distance runners.

A defining feature of the cardiovascular/anaerobic/catastrophic model is that it excludes any role for the brain (central nervous system) in determining the exercise performance$^7$. Rather it is argued that the exercise performance is regulated by fatigue in the exercising muscles whose performance is, in turn, regulated by the adequacy of the oxygen supply they receive. As long as the oxygen supply is adequate, the exercising muscles are able to contract 'aerobically' without the need to produce those fatigue by-products of 'anaerobic' metabolism, lactate and hydrogen ions, which, according to this model, are believed to restrict the exercise performance by 'poisoning' the exercising muscles$^7$.

Thus this model predicts that once the muscles must contract 'anaerobically', the lactic acid they produce begins to accumulate, ultimately 'poisoning' the muscles, leading to localized muscle fatigue and hence the termination of exercise. Since this form of fatigue occurs progressively and exclusively in the muscles, it cannot be influenced by any action of the brain, other than by the voluntary 'choice' of the conscious brain to terminate the exercise. No amount of urging by a 'fresh' brain can undo the 'poisoning' of the exercising muscles$^{9,11}$.

An important and obvious weakness of this 'brainless' model of exercise physiology is that it can offer no reasonable biological explanation for the performance-altering effects of either behavioural or psychological interventions, or for the brain-modifying action of drugs such as amphetamines, which provide perhaps the most profound, acute, performance-enhancing effect known to the sports sciences$^{6,7,9,12}$. Since these interventions are unlikely to slow the rate of lactic acid accumulation in the exercising muscles and thus postpone their 'poisoning' effect, the cardiovascular/anaerobic/catastrophic model$^7,9$ is totally unable to explain the mechanism of action of these other interventions.

Yet clearly the brain must have some functional role in exercise. One obvious analogy might be the role of the driver in determining the speed at which a Ferrari Formula One racing car can travel. Consider, for example, the correct answer to the question 'What speed can a Ferrari Formula One racing car achieve?' This is perhaps not quite as obvious as it might originally appear.

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Fig. 1 The VO$_{2\max}$ value is a function of the athlete's running economy and the peak achieved work rate. Thus in this diagram athlete A is the least economical athlete, having the highest oxygen cost of running at any speed. In addition, A also achieves a lower peak running speed than athlete C during maximal exercise testing. Athlete B also has a lower VO$_{2\max}$ than athlete A but is potentially of equivalent ability by achieving the same peak treadmill running speed as does athlete A. But athlete C is probably the best by virtue of having both the greatest economy and achieving the highest peak running speed. Despite this, C's VO$_{2\max}$ value is no higher than runner B's and is lower than the value of runner A.
This is because the correct answer is ‘It depends’. It depends absolutely on having a live human driver with the ability to exert pressure on the accelerator pedal. Without a live human driver with an intact nervous system, the top speed of the Ferrari is absolute zero. Without a live driver, even the most exotic Ferrari will remain motionless for all time, even with a tank full of petrol and all the other necessary ingredients to allow it to travel at up to 300 km h\(^{-1}\).

But as soon as the live driver with his intact central and peripheral nervous systems is in the driving seat, the engine is started and the clutch released, the speed of the moving Ferrari will initially be determined by one factor—the amount of pressure exerted by the driver’s forefoot on the accelerator pedal; and the magnitude of this pressure will be determined by a complex series of calculations made in the driver’s brain. The driver may not be completely aware of the nature of those calculations, perhaps almost all are made subconsciously. Yet the driver’s overriding priority will be to drive as fast as possible with the lowest possible risk of exterminating the very organ, his brain, that is responsible for both those calculations and for exerting the pressure on the accelerator.

This analogy fits perfectly with the problem faced by the elite long-distance runner, as typified by the great African runners. In competition, they must run just fast enough to win but not so fast that they expire in the process. Indeed, the athletes who finish in their wake are perhaps even better examples of this truth. Their subconscious brain chooses to run more slowly than the winners, thus choosing the ignominy of defeat rather than the finality of death.

This model of exercise physiology in which the brain regulates the exercise performance by altering the mass of muscle, i.e. the number of motor units in the exercising muscles that it chooses to recruit, in order to ensure an optimum performance without risking its health, has been called the central governor model\(^6,7,9,13\). So just as the speed of the Ferrari is controlled by the number of motor units recruited in the driver’s right calf muscles, so the athlete’s running speed is determined by the number of motor units that the brain will allow to be recruited in the exercising muscles. As more motor units are recruited, so speed will increase; as fewer are recruited, so speed will decrease.

It is our belief that only this model can explain a range of observed phenomena in running, in particular the classical ‘end spurt’\(^14,15\) in which subjects, especially some runners in events from 1 to 42 km, speed up near the end of the race\(^6,7\), an observation that absolutely cannot be explained by the traditional AV Hill cardiovascular/anaerobic/catastrophic model\(^7\). The data from South African runners presented here will therefore be interpreted according to this new model despite the fact that these original research studies, on which this more modern interpretation is based, were conceived and completed before the implications of the novel model were fully appreciated. Perhaps these findings and conclusions in South African runners can be extrapolated as a possible explanation of why East African athletes run so swiftly.

The study of Bosch et al.\(^16\)

Bosch and colleagues compared physiological changes during a 42 km simulated marathon run on a laboratory treadmill in sub-elite black and white South African marathon runners\(^16\), selected on the basis of their matched speeds during real 42 km marathon races (16.6 ± 0.9 vs. 16.8 ± 0.9 km h\(^{-1}\); white vs. black runners; this difference was not statistically different). White runners were significantly heavier by approximately 12 kg (71.1 ± 8.6 vs. 59.5 ± 4.3 kg); they were also taller although not significantly so (181 ± 6 vs. 171 ± 6 cm) and had marginally higher \(\text{VO}_{2\text{max}}\) values (63.2 ± 2.9 vs. 60.4 ± 6.5 ml O\(_2\) kg\(^{-1}\) min\(^{-1}\)).

When subjects ran the simulated marathon at the same percentage (~75%) of their average speed during their best 42 km marathon performance, it was found that black athletes ran at a significantly higher percentage of \(\text{VO}_{2\text{max}}\), and a higher respiratory exchange ratio, indicating a greater carbohydrate oxidation. Despite running at a higher percentage \(\text{VO}_{2\text{max}}\) and hence a higher relative exercise intensity, black athletes maintained lower rectal temperatures, but had higher pectoral and mean skin temperatures (Fig. 2).

Thus apparently these sub-elite black athletes had the capacity to maintain higher skin temperatures and lower rectal temperatures, despite running at a higher relative exercise intensity. Maintaining a higher skin temperature would increase the potential for convective (skin to circulating air) heat losses, reducing the necessity to sweat as much when running in the heat. This would clearly be advantageous during very prolonged exercise in the heat if no fluid is available to replace those sweat losses. It is notable, for example, that the Khoi-San peoples of Southern Africa still hunt large antelope to their exhaustion by chasing the antelope for 4 h or more in the midday heat whilst ingesting little if any fluid. Some believe that the ability to sweat and therefore to regulate the body temperature homeostatically below some ‘critical’ value\(^17–21\) provided humans with a critical biological advantage, since they became able to chase non-sweating mammals in the heat until those mammals became incapacitated as a result of overheating\(^22\). Indeed, the ability to perform prolonged exercise in
a waterless environment whilst sweating just enough to maintain a sub-critical body temperature would clearly be of substantial survival value since it would allow more exercise to be performed without developing a critical water deficit.\(^2\)\(^3\),\(^2\)\(^4\).

Unfortunately, Bosch et al.\(^1\)\(^6\) did not compare sweat losses in their black and white marathon runners. Nor are we aware of any other data that address the question of whether the brain of some human athletes might ‘choose’ to maintain either higher skin temperatures or higherrectal temperatures and lower sweat rates, or conversely higher sweat rates and lower skin and rectal temperatures, and the effects of these different ‘choices’ on their respective exercise performances.

However, the lower rectal temperatures in the black runners, although clearly unexpected, could provide a performance advantage since it is known that exercise terminates when a particularrectal temperature (\(\geq 40\text{–}41^\circ \text{C}\)) is achieved.\(^1\)\(^7\)–\(^2\)\(^0\). This ability to maintain a lower rectal temperature during exercise should allow a faster running speed to be maintained in self-paced activity in the heat.\(^2\)\(^1\).

Note that in the study of Bosch et al.\(^1\)\(^6\), as is usually the case, the experimenters determined the athletes’ pacing strategies, thereby excluding any possible contribution of the subconscious brain to the measured physiological responses to that exercise. Clearly in competition, each athlete determines their own pace and thus the physiological response to that exercise bout.

The study of Coetzer et al.\(^2\)\(^5\)

Coetzer et al.\(^2\)\(^5\) completed the first study of elite South African middle- and long-distance runners. They compared the physiological characteristics of elite black South African distance runners, whose best performances were achieved in races of between 5 and 42 km, with those of white South African middle-distance runners who performed best over 800–1500 m. This white comparison group was necessary because these middle-distance runners could equal the performance of the black distance runners over at least one running distance, i.e. in races of less than 2–3 km. No other comparison group of South African runners could match the performances of the elite black runners at any other running distance.

An important finding was that the black distance runners were again substantially lighter by approximately 13 kg (56.0 ± 5.4 vs. 69.9 ± 5.6 kg, black vs. white); they were also shorter (168.9 ± 5.1 cm vs. 181.3 ± 3.0 cm) with a higher proportion of type II fast-twitch muscle fibres (63 ± 13 vs. 53 ± 5% type II fibres). \(\text{VO}_{\text{2max}}\) values were, however, not different between the two groups (71.5 ± 4.6 vs. 71.0 ± 5.3 ml O\(_2\) kg\(^{-1}\) min\(^{-1}\); black vs. white).

However, despite similar \(\text{VO}_{\text{2max}}\) values, the black runners were able to sustain a substantially higher proportion of their \(\text{VO}_{\text{2max}}\) value when racing at 21 km (89 ± 6 vs. 82 ± 4% \(\text{VO}_{\text{2max}}\); black vs. white). This is shown graphically in Fig. 3, which compares the percentage \(\text{VO}_{\text{2max}}\) sustained by black and white runners...
when competing at different racing distances. At distances beyond 5 km, black runners sustained a significantly higher percentage \(\text{VO}_2\text{max}\) and a significantly higher running speed than did white runners. This difference increased with increasing racing distance. Thus the crucial finding was that the black distance runners were better able to resist fatigue; hence they showed superior fatigue resistance in running events longer than 5 km.

Accordingly, the findings of this study support the conclusion that the superior performance of elite black South African distance runners is not likely to be due to a superior maximal capacity to transport and utilize oxygen, as would occur if they had higher \(\text{VO}_2\text{max}\) values. Rather their superiority appears to be due to a greater capacity to utilize a higher proportion of the same \(\text{VO}_2\text{max}\) for longer periods of time during competition, as suggested by Bosch et al. \(^{16}\).

The finding that the superiority of black South African athletes is not due primarily to a greater capacity to consume oxygen, i.e. as a result of higher \(\text{VO}_2\text{max}\) values, is perhaps predictable on other grounds. Figure 3 shows that athletes can sustain exercise intensities eliciting 100% of their \(\text{VO}_2\text{max}\) values only over racing distances of 1–2 km. Yet it is not at those distances that the superiority of African runners in international competition is most evident.

Rather, if the Africans’ success were to be due solely to unusually high \(\text{VO}_2\text{max}\) values (which has never been shown), one would expect these runners to be substantially better than all other runners, also at 1500 m (or the mile). This is not the case; historically, the mile was dominated by Caucasians, especially runners from Great Britain and the Commonwealth. More recently, North African runners from Algeria and Morocco have shown superior ability at this distance. The clear evidence, therefore, is that the success of the East Africans, like that of the black South Africans studied by Coetzer et al. \(^{25}\), is not due to higher \(\text{VO}_2\text{max}\) values and another explanation must be sought. This explanation must presumably include the reason for the superior fatigue resistance of black athletes.

Take, for example, the historical performances of Daniel Komen. Komen has run the mile in 3 min 46 s, marginally faster than the best time achieved by the great British miler and former world record-holder, Sebastian Coe. Komen’s \(\text{VO}_2\text{max}\), however, is unlikely to be greater than that of Coe (\(\sim 77 \text{ ml O}_2 \text{ kg}^{-1} \text{ min}^{-1}\))\(^{6}\) and other great mile runners. The key observation is that Komen runs the 5000 m more than 80 s faster than Coe’s best time, an astonishing 10% difference in performance. Thus Komen’s success is due to his ability to sustain a higher running speed and hence, presumably, a higher percentage \(\text{VO}_2\text{max}\) during races longer than 2 km (Fig. 4). The key to Komen’s success in races of 2–10 km therefore is his superior fatigue resistance, as also found in our black South African runners.

What might explain the superior fatigue resistance of these athletes? Traditionally it is argued, according to the Hill model, that superior exercise performance is determined by the superior capacity of the heart to pump more blood and oxygen to the exercising muscles, and the superior capacity of those muscles to take up that oxygen\(^7\). According to this model, therefore, the superior fatigue resistance of the East African runners must be due to their superior maximal cardiovascular function and skeletal muscle metabolism, in particular with regard to oxygen metabolism.

But intuitively this makes no sense because the superiority of the East Africans increases as the duration of the exercise increases, i.e. as the exercise becomes increasingly submaximal in intensity (Figs 3 and 4). How do cardiovascular function and oxygen delivery set the limit for submaximal exercise.
performance when it is clear that an oxygen deficiency does not occur in the exercising skeletal muscles even in the untrained state, or even during maximal, all-out running exercise of short duration.

Our alternative explanation is that the component missing in Hill’s model is the action of a central governor in the brain, the function of which is to modify the extent of skeletal muscle recruitment (and hence muscle oxygen use) specifically to ensure that exercise is completed within safe physiological limits and without the catastrophic loss of physiological homeostasis.

This is the only model that can explain all the current physiological findings, most especially the ‘lactate and cardiac output paroxes’ of exercise in hypoxia, as well as the more obviously observable phenomena in sport such as the ‘end spurt’ during exercise in which athletes markedly increase their pace during the final 5% of running races and other athletic events; the anticipatory nature of the pacing strategy during exercise, and the fact that many different paces can be chosen during exercise of different durations. The prediction of the Hill model must be that only one pace can ever be chosen and that is the pace immediately below that which produces a catastrophic rise in blood and muscle lactate concentrations.

Indeed, the finding that changes in the pacing strategy, measured as force output or running speed, during exercise are associated with changes in the extent of the neuromuscular recruitment of the exercising skeletal muscles can only be interpreted according to the central governor model which predicts, as now confirmed, that the increased pace during the ‘end spurt’ is due to the recruitment of a larger number of skeletal muscle motor units.

Thus, according to the central governor model, the reason why East African runners can maintain a higher percentage VO_{2max} for longer during races in excess of 5–10 km must be because they can maintain higher levels of skeletal muscle recruitment for much longer than other runners. What enables them to maintain this higher level of recruitment needs to be determined.

Since there is evidence that the pacing strategy is probably determined in anticipation of the start of exercise, the role of a host of factors, not purely physiological, that will influence the superiority of the East African runners needs to be considered.

The study of Weston et al.

This study compared laboratory-derived measures of performance in sub-elite black and white South African 10 km runners, matched for best 10 km running times (32.8 ± 1.8 vs. 33.6 ± 2.4 min, black vs. white) and average weekly training volumes (107 ± 42 vs. 93 ± 25 km).

Black runners were again shorter (172 ± 5 vs. 181 ± 9 cm) and lighter by approximately 10 kg (59.4 ± 6.0 vs. 69.1 ± 5.7 kg) than white runners, but VO_{2max} values were similar in both groups of runners (61.9 ± 5.9 vs. 65.2 ± 7.2 ml O_2 kg^{-1} min^{-1}). However, the key finding was that black athletes were able to run at 92% of the peak treadmill velocity they achieved during the VO_{2max} test for significantly longer (7.56 ± 3.45 vs. 3.57 ± 2.05 min) than were white runners. In addition, the increase in venous blood lactate concentrations when running at 88% VO_{2max} was significantly less in black than in white runners (2.4 ± 2.6 vs. 5.3 ± 2.4 mmol l^{-1}). Even at the end of the test when black runners had run for twice as long at 92% VO_{2max} than had white runners, their final blood lactate concentrations tended to be lower (7.2 ± 4.8 vs. 11.1 ± 3.5 mmol l^{-1}), although not significantly.

The surprising finding was that black runners clearly outperformed white runners in an unfamiliar laboratory trial; yet in competition their performances were more similar. However, the data are compatible with the interpretation that black athletes have superior fatigue resistance during high-intensity exercise. There might be a number of reasons why these sub-elite black athletes have yet to express this apparent physiological superiority also in real competition.

The study of Marino et al.

Intrigued by the possibility that their lightness might explain the superiority of African runners during competition in the heat, Dennis and Noakes calculated that an athlete of 50 kg would be able to maintain thermal homeostasis when running at 20 km h^{-1} in hot, humid conditions whereas an athlete of 65 kg would be able to maintain the same thermal balance when running only at about 16 km h^{-1}.

To evaluate this theory, Marino et al. compared racing performance in the heat of lighter black, and heavier and taller white, runners matched for VO_{2max} (62.6 ± 3.5 vs. 64.3 ± 3.0 ml O_2 kg^{-1} min^{-1}, black vs. white) and peak treadmill velocity (21.2 ± 0.8 vs. 20.8 ± 1.2 km h^{-1}) but not for height (167.4 ± 4.4 vs. 183.4 ± 6.5 cm; P < 0.01) or weight (59.3 ± 4.4 vs. 76.6 ± 9.3 kg; P < 0.01). Subjects performed two 8 km time trials in an environmental heat chamber under either cool (15°C; 60% relative humidity) or hot (35°C; 60% relative humidity) conditions. There were two important findings.

First, although running performance was not different between groups when running the 8 km time trial in the cool (27.4 ± 1.0 vs. 27.4 ± 0.4 min, black vs. white), when running in the heat, black runners ran only marginally more slowly (29.7 ± 2.3 min)
than they did in the cool, whereas white runners ran significantly more slowly (33.0 ± 1.6 min; \( P < 0.05 \)) than they had in the cool conditions. Second, the slower running speeds of white runners in the heat occurred from the outset of the exercise bout. Thus they did not wait to overheat before they slowed down. Rather they began the time trial at a much slower running speed. This indicates that they ran more slowly from the outset of the exercise bout, i.e. ‘in anticipation’, presumably to ensure that they did not overheat.

This is in keeping with the prediction of the central governor model and contrasts absolutely with the cardiovascular/anaerobic/catastrophic model, which predicts that homeostasis must first be lost before there is any change in the pacing strategy. Elsewhere we have shown that this anticipatory response is due to modifications in the extent of skeletal muscle recruitment as predicted by our model.

Another possibility is that the apparently superior cooling strategy of black runners identified by Bosch et al.\(^{16}\) will allow them to run faster ‘in anticipation’ since they may be better able to maintain a lower body temperature under any environmental conditions, even when exercising at the same rate of energy expenditure as white runners.

**Discussion**

A familiar mindset that has had shocking social consequences, most especially in South Africa in the past, is that black and white people come from genetically distinct human populations so that any apparent phenotypical differences are purely the result of genetic differences. Whilst our research reported here appears to show that black South Africans runners are phenotypically different from white South African runners, we have no findings that might explain the origins of those differences. It would seem logical to assume that different environmental exposures over generations and centuries might select for a unique genotype that provides a specific advantage, confined to one unique biological function, such as the ability to run long distances at a high percentage VO\(_{2}\max\). Thus our research shows that black South African runners differ from white South African runners in the following important ways.

**Anthropometry**

Black athletes are significantly smaller and substantially lighter, often by as much as 15 kg\(^{16,25,35,37}\). There may also be other anthropomorphic differences, for example, in the relative mass and length of the lower limb, but we did not specifically address that question. Clearly these differences are likely to have a strong genetic component. As a result, the smaller mass of black athletes provides significant advantages, some of which we have already established\(^{31}\).

Thus their smaller size may reduce the risk of injury since the absolute loading on the musculoskeletal structures will be less. To our knowledge, this has yet to be addressed. However, the ability to train harder and longer because of a reduced stress on these structures would probably provide a competitive advantage.

During exercise in the heat, the smallness of black runners provides a clearly measurable advantage. Since it appears that the rate of heat loss into the environment is an important regulator of exercise performance in warm to hot conditions\(^{36}\), those athletes who produce less heat at any running speed will be at a competitive advantage since they will be able to run faster in limiting environmental conditions, as shown in the study of Marino et al.\(^{21}\). In addition, the higher skin temperatures of the black runners\(^{16}\) will favour heat loss by convection.

Indeed that study tends to confirm the prediction\(^{38}\) that key adaptations that would enhance the likelihood of success in distance running would be a smaller body mass and greater running economy, rather than making the athlete ever more powerful with a larger muscle mass and a greater VO\(_{2}\max\). In African terms, an analogy is the difference between the running abilities of the lion, or more correctly the lioness who does most of the hunting, and the cheetah\(^2\).

The cheetah, whose chase is terminated by an elevated rectal temperature\(^{39}\) after running at up to 100 km h\(^{-1}\) for up to 40 s, succeeds because of the animal’s small size and high degree of running economy (due to elasticity provided by the flexible spine and perhaps other adaptations), which slows its rate of heat accumulation. The small size of the cheetah forces it to hunt even smaller mammals, whose attempts at escape are also limited by their rapid rates of heat production when running very fast\(^{40}\). In contrast, the heavier, more muscular lioness is able to run at high speed for only short distances and thus has evolved a different, co-operative hunting strategy. This analogy suffers of course from the obvious limitation that the cheetah remains a sprinter whereas the focus of this symposium is on endurance athletes. However, the single most important biological adaptation that allowed humans to become distance runners was specifically their capacity to sweat and thus to maintain lower body temperatures when running in the heat\(^{22-24}\).

Perhaps the point is that smallness and greater running economy would seem to be the means used to increase endurance capacity in one animal, the cheetah. Logic suggests that these attributes may also explain the superior endurance capacity of elite human distance runners. Indeed, some of the most successful Kenyan runners are also amongst the most economical yet studied\(^{31,42}\).
**Oxygen consumption**

Black athletes do not have higher \(\text{VO}_{2\text{max}}\) values than elite white runners. Thus the ability to transport and consume larger maximal volumes of oxygen cannot explain the superiority of East African runners.

This is despite the evidence, presented at this symposium, that many of the elite East African athletes originate from high altitude. Hence the benefit of living at altitude is not expressed directly as a superior capacity for maximum oxygen consumption when tested at sea level. Indeed, in studies of sub-elite athletes, black South African athletes tend to have lower \(\text{VO}_{2\text{max}}\) values than do equally performing white runners\(^{16,35}\), perhaps as a result of their generally better running economy, according to the logic presented in Fig. 1.

**Fatigue resistance**

The key advantage enjoyed by black athletes appears to be their superior ability to run for longer at a higher percentage \(\text{VO}_{2\text{max}}\), an attribute we have termed superior fatigue resistance.

Whilst the traditional model posits that this superiority must be due to reduced ‘anaerobic’ conditions in skeletal muscle during exercise and hence a lesser rate of accumulation of ‘poisonous’ lactic acid, this model can be discounted for all the reasons argued in extensive detail elsewhere\(^7,9\). First, there is no evidence that skeletal muscles ever develop ‘anaerobiosis’ during maximal exercise\(^7\). That this most certainly does not occur during submaximal exercise is also now firmly established\(^7\).

Second, there is no evidence that lactic acid is ‘poisonous’ or that it can ever be the exclusive pacing molecule. For if lactic acid were indeed the unique pacing molecule, then it would allow only one peak running speed, specifically the speed exactly below the point at which the exercising muscles supposedly become ‘poisoned’ by the elevated muscle lactic acid concentrations.

If lactic acid is to act as the ‘poison’ that determines the pacing speed, then this model also requires that all available muscle fibres must be active at the point of exhaustion. Yet it is not immediately apparent how lactic acid can completely ‘poison’ muscles that are quiescent because they have yet to be recruited by the central nervous system, whilst the same poison acts only partially on those fibres that are actively contracting. However, all the available motor units in the exercising skeletal muscles are never fully activated during either submaximal or maximal exercise\(^7\), discounting this mechanism.

Third, if this model were true, African runners would run faster at the same blood and muscle lactate concentrations. But they do not. They run faster at *lower* blood lactate concentrations, indicating that the lower blood lactate concentrations are a marker of some other biological difference between white and black runners and are not a primary determinant of the black runners’ superior fatigue resistance. In other words, there is no direct causal relationship between the lower blood lactate concentrations of the black runners and their superior running performance. If black athletes ran faster at the same blood lactate concentrations, this distinction would not be as obvious.

Rather, we believe that this difference originates in the central nervous system, which allows the black runners to recruit a larger muscle mass for longer during prolonged exercise than can white runners. Since it is our theory that the need to protect homeostasis is the overriding regulator of skeletal muscle recruitment during exercise\(^7,9\), these data suggest that black athletes are better able to maintain homeostasis at higher levels of skeletal muscle recruitment than are white runners. Since we do not yet know which are the most important homeostats that regulate this response during exercise, we can only speculate on the location of the physiological systems in which this superiority resides.

Clearly thermal regulation is one such homeostat and we have established the superiority of black runners in the ability to maintain thermal homeostasis during exercise in the heat\(^16\), in part because of their smaller size\(^21\) and their ability to maintain higher skin but lower core body temperatures during exercise\(^16\).

Another homeostat appears to be the muscle glycogen concentration\(^33\). A novel theory is that muscle glycogen is the sole fuel for exercising muscles\(^43,44\). If blood and muscle lactate concentrations are lower in black runners when running at the same \(\text{VO}_{2}\) during exercise, this could indicate an altered skeletal muscle metabolism.

Perhaps black runners have a greater capacity for more rapid resynthesis of glycogen in the contracting muscles, from glucose and other substrates. Such a mechanism would allow more rapid glycogen resynthesis even in the contracting muscles. This information relayed to the central governor would indicate that more glycogen is available and hence a longer exercise bout is possible. Other metabolic differences might act in the same way.

**Running economy**

There is some evidence for differences in running economy between black and white runners. The benefits of a superior running economy would be to further reduce their rates of heat production, slow the rates of energy use and hence allow black runners to travel either faster or longer on the same amount of fuel. The threat to thermal homeostasis is also lessened at any exercise intensity in those who are the most economical.

There is growing interest in the role of muscles as elastic energy return systems\(^45\), which act more like
springs than torque producers during exercise. The more the muscle acts as a spring, the less energy it consumes and hence the more efficient it is. The more efficient, more elastic muscle will enhance performance by slowing (1) the rate of accumulation of metabolites that may act as afferent signallers of homeostatic deviation, and (2) the rate of rise of body temperature, as discussed above.

Empirical observation of the running stride and the anatomical structure of the lower limb of East African runners suggests, at least to these authors, that an evaluation of the elastic elements of the legs of elite East African runners would probably be very rewarding. An important theory inviting attention is that a superior muscle elasticity might contribute to an apparently superior running economy in these athletes and, independently, to their extraordinary running ability. Better running ability might also result from more efficient muscle contraction.

Psychological/motivational factors
Psychological/motivational factors may explain the inordinate motivation to train and race as hard as do the elite East African runners. The psychological factors explaining this extraordinary commitment of effort and focus need to be explored.

For example, the traditional explanation is that the ratings of effort (perceived exertion – RPE) during exercise are a function of the exercise intensity alone. But this is clearly incorrect since RPE rises during prolonged exercise even when the exercise intensity may vary, usually falling progressively during self-paced prolonged exercise before rising steeply near the finish – the "end spurt". Misinformation about the exercise intensity or duration can also alter the RPE, which should not occur if the RPE is purely a simple measure of the exercise intensity.

More recently we have observed that the RPE rises as a linear function of the duration of the exercise and that individuals usually terminate exercise at the same terminal RPE. This suggests that, either before the exercise begins or shortly after the onset of exercise, the brain must establish, in those activities in which the exercise intensity is fixed externally by the experimenter, the duration of the allowed exercise bout. In contrast, when pacing is self-imposed by the athlete, the brain must determine the overall pacing strategy shortly after the beginning of exercise.

Figure 5 compares a hypothetical increase in RPE in a group of white marathon runners who complete a 42 km marathon race in a slightly longer time (line D) than a group of elite black runners. Black runners complete the same distance in a shorter time but with three different theoretical patterns of RPE rise during exercise (lines A, B and C). This model is more likely to be correct in athletes who maintain the same pace during the entire race; the effect of changes in pace during a marathon race on RPE or vice versa has, to our knowledge, not been studied.

Theoretical line A would indicate that black runners have the same RPE as white runners throughout exercise; as a result they terminate exercise at the same maximal RPE. This would indicate that specific biological advantages, due either to environmental or genetic influences, allow black athletes to maintain homeostasis more easily during exercise. As a result, these biological advantages would make running easier for black than for white runners.

Theoretical line B would indicate that black athletes run at a higher RPE during exercise but terminate exercise at the same maximal RPE as white runners. This would indicate that the extra speed of the black runners is achieved at the expected cost – a greater stress on homeostatic function without any greater capacity to stress the system closer to its physiological limits, an adaptation which would be shown as a higher peak RPE at exhaustion.

Theoretical line C would indicate that black athletes run faster but at an unexpectedly large increase in homeostatic challenge, so that they must also suffer a higher limiting RPE in order to run faster.
Our bias is to believe that either line A or B could be correct. Line A would predict that a superior capacity to maintain homeostasis allows black athletes to run faster but at the same psychic cost as slower-running white athletes. Line B predicts that black athletes are better able to sustain a higher homeostatic challenge and hence a higher psychic cost than are white runners. Since they run faster but for a shorter time, they terminate exercise at the same terminal RPE as white runners.

Conclusions

Certain physiological differences appear to exist between black and white endurance athletes. The most obvious is the ability of black athletes to sustain a higher percentage VO₂max for longer when racing. This is possibly due to a greater capacity to recruit a larger muscle mass for longer during prolonged exercise.

Since the exercising body acts as a complex system⁹ in which the brain integratively moderates the activities of all peripheral systems to ensure that catastrophic physiological failure does not occur, this difference could be due to any or all of the following factors.

1. A true metabolic difference that makes the regulation of homeostasis easier in black runners when running at higher exercise intensities. Two possibilities suggest themselves. First, black runners appear better able to maintain lower body temperatures during exercise, perhaps as a result of their smaller size and the ability to maintain higher skin and lower core body temperatures during exercise in the heat. Second, an altered muscle glycogen metabolism might allow the same muscle glycogen content to fuel a longer running distance, perhaps as a result of a superior capacity to resynthesize muscle glycogen in the contracting muscle filaments from other carbohydrate sources including glucose and lactate, even whilst the exercise continues. Of course, a host of other homeostats, including their integrative control, may also function more effectively in black athletes.

2. A neurological difference which allows black athletes the ability to recruit a larger skeletal muscle mass even though the threat to homeostasis is greater than in white athletes, who are unable to sustain equally high rates of energy expenditure for as long during prolonged exercise. This explanation would require that black athletes will need to conquer the symptoms of distress¹¹,³⁰ more effectively during exercise than can white runners. That is, their higher running speeds are won at the cost of greater feelings of discomfort. Alternatively, it is also possible that black athletes might feel less discomfort and therefore have a lower RPE at the same level of homeostatic distress than do white athletes. These different possibilities are shown graphically in Fig. 5 and all invite scientific evaluation.

Perhaps the point of our argument is that the human acts as a complex system during exercise⁹ so that any attempt to explain superior performance on the basis of a single organ system such as the heart, lungs or muscles will not be correct. Rather we argue that superior function is related to the central (brain) control of whole-body homeostasis during exercise.

In which case, any explanation for the success of the East African runners will be found in the way in which their innate physiology, training, environment, expectations and genes influence the function of those parts of their subconscious (and conscious) brains that appear to regulate the protection of homeostasis during exercise.

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References

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