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The human endurance athlete: heterogeneity and adaptability of selected exercise and skeletal muscle characteristics

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In human subjects, large variations between individuals (up to 3-fold) exist in the capacity for endurance exercise performance. In a heterogeneous population, endurance performance is strongly related to whole body maximal oxygen uptake (VO_2max). This is in part genotype dependent (~25%) but is adaptable with training. However, this relationship does not hold within a homogeneous group of well-trained runners. Other physiological characteristics must contribute to endurance performance and these may include specific advantageous skeletal muscle phenotypes. Muscle fibre type distribution is also heterogeneous, although less adaptable. In contrast, muscle oxidative enzyme capacity is highly adaptable with training. The genetic influences on these muscle characteristics have been indirectly investigated by comparing African endurance athletes, who dominate world-class events, to Caucasian endurance athletes. We have established that African runners have greater resistance to fatigue than Caucasians ($p < 0.01$) and 50% greater oxidative enzyme activity in vastus lateralis samples ($p < 0.05$), despite somewhat lower Type I fibre proportion. These differences were not inherently present in a group of sedentary Africans, suggesting that the genotypic influence on athletic performance may be a superior adaptation to training, rather than a baseline genetic effect. Combined physiological and genetic studies are likely to elucidate a polygenetic basis for superior endurance performance.

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Introduction

Within the human species endurance performance capacity is characterised by a large degree of variation between individuals. Previous research has shown that this heterogeneity is at least partially dependent on genotype (Bouchard & Lortie 1984; Bouchard, Dionne, Simoneau & Boulay 1993). Although the magnitude of the genotypic influence is not clear at this time, it is accepted that it is most likely a result of several different DNA variants (Dionne, Turcotte, Thibault, Boulay, Skinner & Bouchard 1991). Acceptance of a genetic component to the endurance performance phenotype raises the possibility that there may be racial differences in endurance capacity (Boulay, Ama & Bouchard 1988).

Considerable anecdotal evidence suggests an advantageous endowment for endurance performance in individuals of East African origin. These runners dominate international men's distance running. At the 1996 World Cross-Country Championships, African runners claimed 14 of the first 15 places in the senior men's event. African runners currently hold the world records at all distances between 800 m and 42.2 km (as at September 1997). There is an equivalent dominance of distance events by Africans within South Africa (Coetzer, Noakes, Sanders, Lambert, Bosch, Wiggins & Dennis 1993) and recent successes of African South African distance runners in the international arena are also apparent. Although no studies have as yet identified any specific genetic polymorphism associated with enhanced endurance capacity in these groups, some scientific studies have attempted to elucidate the physiological and biochemical characteristics that may contribute to the apparently superior endurance phenotype of runners of East or South African origin (Bosch, Goslin, Noakes & Dennis 1990; Coetzer *et al.* 1993; Saltin, Kim, Terrados, Svedenhag & Rolf 1995a; Saltin, Larsen, Terrados, Bangsbo, Bak, Kim, Svedenhag & Rolf 1995b; Weston, Karamizrak, Smith, Noakes & Myburgh 1998).

A significant correlation between a physiological or biochemical variable and performance suggests, but does not prove, causality. In contrast, adaptations of physiological or biochemical variables to training regimens imply that these adaptations serve to improve the homeostasis of the organism in response to the external influence of additional exercise. The extent of adaptability may be a factor determining subsequent performance capacity. Twin studies have shown that the marked heterogeneity between individuals in their response to identical training regimens is at least, in part, genetically determined (Hamel, Simoneau, Lortie, Boulay & Bouchard 1986; Lortie, Simoneau, Hamel, Boulay, Landry & Bouchard 1984; Prud'homme, Bouchard, LeBlanc, Landry & Fontaine 1984). This indicates that there is an interaction between the influence of genetics and training on performance.

The aim of this review is to discuss the heterogeneity and adaptability of selected physiological and biochemical characteristics that may influence endurance performance, namely VO_2max, muscle fibre type, muscle oxidative enzyme capacity and resistance to fatigue during submaximal exercise. Particular reference will be made to evidence for and against a genotypic effect on these variables and any distinctly different phenotypic expression of these variables between African and Caucasian distance runners.

1. Whole body maximal oxygen uptake (VO_2max)

1.1 Heterogeneity

Early studies by Hill & Lupton (1923) suggested that VO_2max was higher in endurance-trained athletes when compared to less trained controls. These investigators suggested that the individual with the highest VO_2max was able to maintain higher levels of exercise without requiring a significant contribution from fatigue-inducing anaerobic metabolism, and therefore had the best endurance performance capacity.

In general, VO_2max is a good predictor of performance in a heterogeneous group. The relationship between VO_2max and endurance performance in individuals ranging from less-trained to well-trained, shows a high correlation (e.g. r = –0.88, in runners with a marathon time ranging from 2 h 19 min to 4 h 53 min) (Maughan & Leiper 1983). Even in a group of trained athletes with somewhat less range of race performances, the correlation remains high. Costill (1967) found a strong relationship between VO_2max and endurance performance in collegiate cross-country athletes (r = –0.82). Farrell, Wilmore, Coyle, Billing & Costill (1979); Foster, Costill, Daniels & Fink (1978); and Sjödin & Svedenhag (1985) all report similar correlations (r = –0.91, –0.86 and –0.78 respectively) for their studies of this relationship in trained individuals.

1.2 Elite athletes

However, this is not the case in populations with more homogeneous race performances. In a group of very well-trained runners with a relatively narrow range of 10 km race time, (30–33 min), there was no relationship between VO_2max and race performance (r = 0.12) (Conley & Krahenbuhl 1980). Similarly, the documented VO_2max of elite runners shows that the best runner does not necessarily have the highest VO_2max (Costill, Fink & Pollock 1976; Sjödin & Svedenhag 1985). Some very good runners do not have excessively high VO_2max test results (for example, Derek Clayton marathon time 2:08:33, VO_2max = 69.7 ml/kg/min) (Costill, Branam, Eddy & Sparks 1971).

Nevertheless, it is apparent that a VO_2max of a reasonable level [approximately above 63 mmol/kg/min (Sjödin & Svedenhag 1985)] is a prerequisite for elite performance. The following question therefore arises: is this variable a trait influenced by genotype or is it highly adaptable to training, or both?

1.3 Influence of genotype

Early studies investigated familial resemblance of VO_2max (ml/kg/min). Montoye & Gayle (1978) showed a strong relationship (r = 0.66) between young fathers and their sons with respect to VO_2max, but the relationship did not hold for fathers in an older age group. It is well known that ageing is associated with a decrement in VO_2max in human subjects. This suggests that factors other than genetics can eclipse the genotypic effect on VO_2max that may have been measurable prior to outside influences such as age, environment and lifestyle. This conclusion is supported by Lortie, Bouchard, LeBlanc, Tremblay, Simoneau, Thériault & Savoie (1982) who reported a low correlation for VO_2max between parents and their children. These authors also noted that the correlation was not different from that between spouses. Clearly, spouses have no genetic link, but considerable similarities in age, environment and lifestyle.

Since these early familial resemblance studies, investigators have utilised a different, more robust, approach by studying twin pairs. Engstrom & Fishbein (1977) investigated the physical capacity of twins by determining the highest workload they could sustain for 6 min on a cycle ergometer. After correcting for body mass, they found a significant relationship within twin pairs (r = 0.70). Twin studies also allow a comparison to be made between the intra-twin pair correlation of monozygotic twins and the intra-twin pair correlation of dizygotic twins, as well as the correlation between pairs of siblings. Similarity between monozygotes that is over and above that of dizygotes and siblings is likely to predominantly reflect genetic dependence and can be determined by calculating the intra-pair differences. Howald (1976) found that monozygotic pairs were not significantly more similar than dizygotic pairs. However, as discussed by the authors, these data may be biased by the diverse physical activity history of two of the sets of monozygotic twins, that resulted in less intra-pair resemblance in the monozygotic group than would have been achieved without this environmental difference. The analysis excluding these pairs and one dizygotic pair with very different body mass, resulted in a high heritability index. This indicates that when it is possible to exclude clear environmental and lifestyle influences, a large amount of the variance was caused by genetic factors.

More recently Bouchard, Lesage, Lortie, Simoneau, Hamel, Boulay, Pérusse, Thériault & LeBlanc (1986a) conducted a study with a large number of siblings (n = 42), dizygotic twins (n = 66) and monozygotic twins (n = 106). The study aimed to control bias by using siblings of the same gender or twins who were living together in an attempt to isolate and quantify the genetic influence on VO_2max. There was a trend for the VO_2max (ml/kg/min) within-pair correlation to decline from monozygotic (r = 0.85) to dizygotic (r = 0.74) to brothers (r = 0.55) although statistically significant to the 1% level in all cases. The fact that the dizygotic intra-class correlation was slightly higher than that for brothers, implies that a portion of the correlation is environmental and therefore the genetic effect may be slightly inflated.

In summary, it would appear that the heritability fraction for VO_2max is significant but smaller than first thought (somewhere between 10–30%) and may be over-estimated by twin studies because of more similar environmental conditions for twins than for siblings.

1.4 Adaptability

The 'textbook' concept that the adaptation of VO_2max to training is only approximately 15%, is based on early data. For example Gollnick, Armstrong, Saltin, Saubert, Sembrowich & Shepherd (1973) showed a five-month training effect averaging 13% in subjects previously fairly active but not endurance-trained. Examination of individual data indicates variability between individuals ranging from 4% to 25%. More recently, the subjects investigated by Lortie *et al.* (1984) responded to a five-month training regimen with an even greater range in improvement in VO_2max of between 5

and 88%. However, it should be noted that the former study expressed increases in VO_2max in absolute terms i.e. not inflated for any loss in body mass owing to exercise. In the latter study, if the increases are not expressed relative to body mass, women still showed a wide variation in response (10–87%), but the variation in response for men was somewhat less (7–43%). Only 25 to 35% of the variation in response can be attributed to the baseline level of VO_2max. It has been postulated that at least a part of the remaining variance may be genetically determined i.e. some genotypes may respond to a greater extent to training than others may.

To investigate the above hypothesis, ten pairs of monozygotic twins involved in a 20 week aerobic training programme were evaluated (Prud'homme *et al.* 1984). The results indicated that twin pairs responded to training with similar magnitudes of change in VO_2max (ml/kg/min) that resulted in a correlation for change in VO_2max between the various twin pairs of 0.74 ($p < 0.01$). These results were supported by Hamel *et al.* (1986), suggesting that there is indeed a genotypic effect on sensitivity of VO_2max to training.

Variations in mitochondrial DNA sequence may be responsible for differences in gene expression. Dionne *et al.* (1991) investigated mitochondrial DNA sequence variants in sedentary males in an attempt to relate variants to either VO_2max or to the trainability of VO_2max. A high baseline VO_2max was prevalent in carriers of three particular mitochondrial DNA morphs whilst carriers of another morph exhibited a lower baseline VO_2max. Three carriers of a further variant had a significantly lower VO_2max response to training. This study further supports the notion that genetic factors may contribute to the magnitude of VO_2max as well as its response to training.

It is now believed that most human genetic material is common to all humans and that perhaps 10% is specific to population or racial groups. A degree of that percentage encodes for skin or facial characteristics. However, if this 10% was to include any of the exercise performance-determining genes it may explain the dominance of East and South African athletes in endurance races as described in the introduction.

1.5 Different populations

Davies, Mbelwa, Crockford & Weiner (1973) and Di Prampero & Ceretelli (1969) found lower VO_2max in untrained Africans (Tanzanians and Kenyans respectively) compared to Europeans. However when this data was corrected for their smaller muscle mass, the difference was no longer apparent, emphasising the importance of matching subjects for mass where possible. Similarly, VO_2max values of South African Africans of various tribal groups were similar to control Caucasian subjects only when corrected for body mass (Wyndham, Strydom, Morrison, Peter, Williams, Bredell & Joffe 1963). Boulay *et al.* (1988) have summarised these studies and concluded that there are differences in endurance capacity but that these are small and that 'differences observed at times between some racial groups can be attributed to such factors as age, gender, body size, habitual physical activity, altitude, ambient temperature, and test mode.' A recent study of sedentary African and Caucasian university students supports the earlier findings with a lower VO_2max in the African subjects (45 vs 54 ml/kg/min). Although sedentary for several years, two of the Caucasian subjects had been good endurance athletes at school and this may have biased the results somewhat (unpublished data).

Bosch *et al.* (1990) compared African and Caucasian marathon runners resident in South Africa. The Caucasian runners were considerably taller and heavier than the African runners. When corrected for body mass, VO_2max was not different between groups. Similarly, sub-elite African and Caucasian runners specialising in shorter distance racing (10 km) did not differ significantly with respect to VO_2max (61.9 ± 5.9 and 65.2 ± 7.2 ml/kg/min respectively) (Weston *et al.* 1998). Also, elite South African marathon athletes, Zithulele Sinqe and Willie Mtolo (marathon times 2:08:04 and 2:08:15 respectively) did not exhibit extraordinary values for VO_2max (72.0 and 70.3 ml/kg/min respectively) (Noakes, Myburgh & Schall 1990).

Recently, Saltin *et al.* (1995b) measured aerobic capacity in 42 Kenyans (active boys and junior and senior runners) and 12 elite Scandinavian runners. When comparing the seniors of Kenya and Scandinavia, VO_2max was not different.

In summary, there appears to be very little racial difference in maximal aerobic power when values are corrected for body size. Clearly, the successful endurance performance of East Africans can not be explained by VO_2max alone. Therefore, despite a genotypic effect on VO_2max and on the sensitivity of VO_2max to training, this is apparently not different in different populations. Other physiological parameters are important in determining endurance performance. In particular, a high percentage of Type I fibres and high oxidative enzyme capacities have been associated with superior endurance performance (Gollnick, Armstrong, Saubert, Piehl & Saltin 1972; Holloszy & Coyle 1984; Lortie, Simoneau, Hamel, Boulay & Bouchard 1985).

2. Skeletal muscle fibre type

2.1 Heterogeneity

It is well known that in small mammals, such as the rat, a particular muscle will consist predominantly (or solely) of the fibre type that is suitable for the function of the muscle. A muscle continuously involved in maintenance of balance will consist of slow twitch fibres whilst a muscle used only for locomotion may contain a substantial proportion of fast twitch fibres. In humans, most muscle is of mixed fibre type and it is conceivable that the proportions of slow to fast twitch fibres could be influenced by the anatomically based function of the specific muscle. It is also likely that, within the restraints of the anatomically based fibre type proportion, any variability in the latter could in itself further influence functional capacity of the muscle.

There has been considerable interest in the fibre type composition of both sedentary individuals and various athletic populations as researchers attempt to elucidate the mechanisms important in successful endurance performance. A summary of some of the published data for fibre type composition of the vastus lateralis muscle of sedentary human subjects is presented in Table 1. These early studies indicated that in most sedentary individuals there is a fairly equal proportion of Type I and Type II fibres.

A more recent study summarising the skeletal muscle fibre

Table 1 Fibre type composition in the vastus lateralis muscle of sedentary populations

n =	% Type I	% Type II	Reference
26	36	64	Gollnick *et al.* 1972
11	60	40	Larsson *et al.* 1979
10	63	37	Larsson *et al.* 1979
70	54	32/13*	Saltin *et al.* 1977
10	47	38/14*	Simoneau *et al.* 1985

* = % Type IIA / % Type IIB

type of more than 400 Caucasian men and women from North America, has shown that in fact there is considerably more heterogeneity than previously believed (Simoneau & Bouchard 1989). Twenty-five per cent of the male subjects and 19% of the female subjects in this study had less than 35% or more than 65% Type I fibres (i.e. only approximately 75% of all the subjects fell within the middle 'normal' range). Overall the range in the percentage of Type I fibres was 15% to 85%.

2.2 Influence of genotype

Twin studies have shown that fibre type phenotype is genotype dependent (Komi, Viitasalo, Havu, Thorstensson, Sjödin & Karlsson 1977). These authors concluded from twin studies (n = 15 monozygotic pairs, n = 16 dizygotic pairs) that fibre type composition was highly genetically determined (heritability coefficient = 0.93). More recent studies (Bouchard, Simoneau, Lortie, Boulay, Marcotte & Thibault 1986b; Lortie, Simoneau, Boulay & Bouchard 1986) have refuted this finding showing considerably lower heritability coefficients. It may be significant that the subjects in the early study by Komi *et al.* (1977) were all young (under age 25) and therefore had less environmental influence on their fibre type than in the latter studies. Nevertheless, in their recent review of the human studies that investigated the heritability of fibre type, Simoneau & Bouchard (1995) concluded that the genetic component accounts for between 40 and 50% of the variability in the proportion of Type I fibres in human muscles. Therefore, an individual's fibre type proportion is partially genotypically pre-determined and probably to a greater extent than is VO_2max. However, VO_2max is responsive to substantial adaptations to training, at least in some individuals. The following question therefore arises: to what extent is human muscle fibre type responsive to exercise?

2.3 Adaptability

Cross innervation and electrical stimulation of skeletal muscle in animals and transcutaneous electrical stimulation in humans have both been shown to change Type II fibres to Type I fibres (Bárány & Close 1971; Munsat, McNeal & Waters 1976; Pette & Vrbová 1992). However, early studies provided little evidence for gross changes from Type II to Type I fibres as a result of normal endurance training in humans. Gollnick *et al.* (1973) investigated the effect of a five month training programme on fibre type composition and cross-sectional area. Although mean VO_2max increased by 13%, no change occurred in the mean percentage of the Type I or Type II fibres. Only the cross-sectional area of the slow twitch fibres was larger after training. No differentiation was made between Type IIA and IIB and therefore it was not possible to investigate changes between the sub-groups of fibre types. Saltin, Nazar, Costill, Stein & Jansson (1976) reported that fibre type proportions did not change after either endurance or sprint training in human subjects using the one-leg training model. Similarly, Henriksson, Jansson & Schantz (1980) reported no change in triceps brachii Type I fibre percentage after a 50-day endurance regimen of cross-country skiing.

To the best of our knowledge very few studies have demonstrated increased Type I fibre percentage in human subjects following endurance training (Howald, Hoppeler, Claassen, Mathieu & Straub 1985; Simoneau, Lortie, Boulay, Marcotte, Thibault & Bouchard 1985). Simoneau *et al.* (1985) investigated the response of fibre type proportions to 15 weeks of strenuous training. Training resulted in an increase in the proportion of Type I fibres (from 41 to 47%) and a decrease in the proportion of Type IIB fibres (from 17 to 11%) (both $p < 0.01$), whereas the proportion of Type IIA fibres remained essentially unchanged. Howald *et al.* (1985) also showed a 6% mean increase in Type I fibres from 50 to 56% following only six weeks of high intensity endurance training. These two studies indicate that the direction of change in fibre type proportion in humans can follow similar patterns to those found in animal muscle in response to continuous stimulation. Current studies addressing this issue are utilising gel electrophoresis rather than histological fibre typing to investigate the plasticity of contractile protein isoforms.

Different fibre types contain different isoforms of subunits of myosin e.g. myosin heavy chain (MHC) (Bárány & Close 1971). The subunits are distinguishable by their different mobilities on gel electrophoresis. For example, Type I fibres contain MHCs (slow) and Type II fibres contain MHCf (fast) (Schantz, Billeter, Henriksson & Jansson 1982). Subdivision of Type II fibres has led to the following nomenclature for MHC isoforms: MHC IIa in Type IIA fibres and MHC IIb in Type IIB fibres (Myburgh 1994). Support for the concept of plasticity of muscle fibre type is lent by the fact that 'hybrid' fibres contain more than one MHC isoform. Hybrid fibres have been clearly identified in and associated with experimentally induced transformation of fibre types in animal models (Pette & Vrbová 1992; Termin, Staron & Pette 1989). Physiological levels of exercise training in rats increase the percentage of MHC IIa in plantaris muscle within four weeks (Sugiura, Morimoto & Murakami 1992), whereas changes in the costal diaphragm which is accustomed to a higher workload were only evident after 10 weeks. No changes were apparent in the slow isoforms. However, in a different study, 10 weeks of endurance training altered the ratio between two slow isoforms of MHC in vastus intermedialis in rats (Fitzsimmons, Diffee, Herrick & Baldwin 1990), indicating that different muscle will respond differently to similar workloads. This was further emphasised in the same study which showed an increase in slow isoforms of MHC at the expense of fast isoforms in red medial gastrocnemius and the plantaris, but not in the red vastus lateralis, white medial gastrocnemius or white vastus lateralis. Another important conclusion that can be drawn from this study is that the MHC isoforms can complete the transition from fast to slow isoforms in response to a physiological level of training.

The Type IIC fibres identified in human muscle have been shown to contain both MHCs and MHCf (Schantz *et al.* 1982), suggesting that these fibres may be in transition from fast to slow fibre type. Although the IIC fibres are usually very scarce (< 1%) in human muscle, after 50 days of extremely strenuous endurance activity the percentage IIC fibres has been shown to increase to 5% (Henriksson *et al.* 1980) and even to 15% in one subject following six weeks of high intensity strength training (Staron & Hikida 1992). It has been hypothesised that with continued exposure to the appropriate environmental influence, in this case increased workload, these fibres could fully transform into Type I fibres containing only the slow MHC isoform (Schantz & Dhoot 1987). This hypothesis appears to be indirectly supported by the cross-sectional data of Baumann, Jaggi, Soland, Howald & Schaub (1987) who showed that professional cyclists with many years of training had a mean of 80% slow twitch fibres. We hypothesise that baseline muscle characteristics will profoundly influence the response to training interventions, although at this point definitive predictions of response to training cannot be made. This hypothesis is supported by the individual longitudinal data of Baumann *et al.* (1987). After eight weeks of high intensity endurance training, one of four subjects increased their percentage Type I fibres. This subject had approximately 10% lower Type I fibres at baseline than the other three subjects. However, only two of the remaining three subjects increased percentage IIA, but they did not have baseline characteristics substantially different from the one subject who exhibited no changes in fibre type. The extent of transformation was in the region of 5% on average but was as high as 12% in one case.

Although these studies indicate the capacity for transformation of skeletal muscle contractile protein isoform, the extent of plasticity in response to changes in workloads within a physiologically normal range are unlikely to be as large as that seen in response to electrical stimulation. Nevertheless, the functional effect of smaller changes may also be significant as indicated by Larsson & Moss (1993) in a study investigating the *in vitro* mechanics of single fibres of human muscle. These authors clearly showed differences in the force velocity relationship of different fibres each with various proportions of myosin heavy chain isoforms.

2.4 Elite athletes

As noted above trained endurance athletes tend to have a higher percentage of Type I fibres (see also Table 2). However, well-trained individuals, in particular, are reluctant to have a muscle biopsy. Therefore, the number of subjects in most studies is small and the total data base of skeletal muscle fibre type information on well-trained individuals is relatively small. This may explain some of the apparently inconsistent results reported in the literature. By far the majority of studies have investigated the vastus lateralis muscle and even less information is available on other muscles of the body.

Table 2 Fibre type composition in the vastus lateralis muscle of endurance athletes

n =	% Type I	% Type II	Reference
8 orienteers	68	25/3*	Saltin *et al.* 1977
8 dist. runners	59	41	Gollnick *et al.* 1972
14 dist. runners	79	21	Costill *et al.* 1976
18 middle dist.	62	38	Costill *et al.* 1976
11 cyclists	57	43	Burke *et al.* 1977
13 prof. cyclists	80/1**	17/1*	Baumann *et al.* 1987

* = % Type IIA / % Type IIB; ** = % Type I / % Type IIC; dist. = distance; prof. = professional

There remains considerable debate whether those individuals with a high proportion of Type I fibres have selected endurance sports because of success at this type of activity, or whether the high proportion of Type I fibres is the result of an adaptation to this form of training. Taking the above data of baseline heterogeneity in fibre type and potential for moderate adaptability into consideration, we would suggest that a combination of both is likely.

In summary, the early cross-sectional studies comparing non-trained with endurance-trained athletes do indicate a higher proportion of Type I fibres in the latter. This suggests that a high proportion of Type I fibres is a contributor to success in endurance events when considering a heterogeneous group of subjects. Whether the relative proportion of the fibre types is a predictor of endurance performance in a homogeneous group of well-trained athletes is less clear, particularly given the more recent data outlined below.

2.5 Different populations

Only three studies to date have documented skeletal muscle characteristics in endurance athletes of East/South African origin: Coetzer *et al.* (1993) measured fibre type proportions in vastus lateralis in South African distance runners of African and Caucasian descent and found no statistically significant difference (53.3 ± 4.5 and 63.4 ± 13.3% Type I respectively). Saltin and co-workers (1995a) compared Kenyan and Scandinavian runners after the Scandinavians had spent two weeks at altitude. Fibre type proportion of vastus lateralis was not different between Kenyan and Scandinavian runners (72.5 vs 67.7% Type I fibres respectively). Finally, a study by Weston *et al.* (1998) indicated that South African Caucasian runners had a significantly higher percentage of Type I fibres (67.1 ± 17.5%) than the sedentary subjects (44.2 ± 12.1%; $p < 0.01$) whilst the African runners (49.0 ± 17.3 %) were not different from controls. It is not possible from these data collected on relatively small numbers of subjects to decide whether there are differences in per cent fibre type between the endurance runners in these different populations.

Nevertheless, what this data does demonstrate is that subjects with only approximately 50% slow twitch fibres can be quite competent endurance athletes, and the best athletes in the world do not necessarily have upwards of 80% slow twitch fibres. As a function of the running speed of endurance events at the current elite level (where a 10 km race is run as a series of 400 m laps each as fast as 67 s), it is clear that a large proportion of all fibres will be recruited and that they will be required to generate considerable force for a considerable length of time. The combined physiological and biochemical characteristics required to successfully provide both a high force output and endurance would teleologically argue against conversion of fast to slow twitch fibres, and rather argue for the enhancement of oxidative capacity in all availa-

ble fibres regardless of type. Gollnick *et al.* (1973) showed early in the history of sport science that adaptations to endurance exercise are accompanied by large changes in muscle oxidative enzyme activities without any change in fibre type. Therefore, skeletal muscle oxidative enzyme capacity is more likely than fibre type *per se* to be a potential determinant of endurance performance.

3. Skeletal muscle oxidative enzyme capacity

3.1 Heterogeneity

Skeletal muscle oxidative capacity is usually determined by the measurement of oxidative enzyme activities using marker enzymes such as succinate dehydrogenase (SDH), cytochrome oxidase (CYTOX), citrate synthase (CS), oxoglutarate dehydrogenase (OGDH) and 3-hydroxyacyl-CoA dehydrogenase (HAD). A less commonly used marker is malate dehydrogenase (MDH) since it occurs both in the cytosol and the mitochondrial membrane. All seem to adapt to increased or decreased submaximal muscle use in constant ratios and are directly related to the mitochondrial content of the muscle in rats (Gollnick & King 1969) and human subjects (Kiessling, Pilström, Bylund, Saltin & Piehl 1974).

Similar to the strong relationship between racing performance and VO_2max in heterogeneous groups, a relationship has been found between skeletal muscle oxidative capacity and VO_2max in populations relatively heterogeneous for exercise capacity. For example, Hoppeler, Luthi, Claasen, Weibel & Howald (1973) report a significant correlation between VO_2max and skeletal muscle mitochondrial volume density (r = 0.82) in human subjects. Recalculating the data presented by Henriksson & Reitman (1976), indicates that in their relatively untrained subjects, who had a wide range of VO_2max values (36–61 ml/kg/min), there was a significant correlation between VO_2max and SDH activity in the vastus lateralis muscle (r = 0.59; $p < 0.05$). Although the correlation was relatively weak in the previously mentioned study, Booth & Narahara (1975) reported a stronger relationship (r = 0.75) between VO_2max and CYTOX activity in the vastus lateralis of untrained men.

3.2 Influence of genotype

Limited studies have investigated the heritability of skeletal muscle oxidative characteristics. Komi and co-workers (1977) investigated the heritability of skeletal muscle enzymes in 31 pairs of twins and reported no evidence of heritability of any of the enzyme activities measured. However, other studies have provided conflicting evidence as discussed below.

Howald (1976) investigated the ultrastructure and biochemical function of skeletal muscle in 17 pairs of twins. There was no statistical difference between monozygotic and dizygotic twins for either mitochondrial volume density or for the activity of SDH. However, 3-HAD was more closely correlated within monozygotic pairs than within dizygotic pairs ($p < 0.01$, $p < 0.05$ respectively). This finding implies that the activities of the latter enzyme may be considerably more genetically determined or alternatively stated: considerably less influenced by environmental factors. Bouchard *et al.* (1986b) combined the monozygotic and brother data of Lortie *et al.* (1986) with their own dizygotic data to calculate the heritability coefficient for skeletal muscle enzyme activities. Notably, data were adjusted for age and gender where appropriate. The heritability coefficients indicating a significant influence of genetics on oxidative enzyme activities were: OGDH = 0.42; 3-HAD = 0.56 and MDH = 0.63. Subsequent comparison of brothers and dizygotes showed that brother intra-class coefficients were higher than those of dizygotes for some variables and in some cases the dizygote resemblance was near zero. Hence these data must be interpreted with caution.

The relatively large extent of plasticity of oxidative enzymes to various environmental influences, especially exercise, may be a factor confounding the ability to gain a true estimate of the genetic effect on enzyme activities. A true baseline, completely unaffected by the environment, is virtually impossible to achieve. Nevertheless, this same fact points to the ability of the environmental effects to overcome differences in genotypic influence.

In summary, the above studies provide some evidence for a limited measurable genetic effect on enzyme activities. However, the heritability of baseline skeletal muscle oxidative enzyme activities does not appear to be of a really significant magnitude.

3.3 Elite athletes

Gollnick *et al.* (1972) compared oxidative enzyme activities in groups of untrained and trained individuals, clearly showing higher mean SDH activity in endurance-trained athletes (1.6-fold higher) than untrained controls. Similarly, Jansson & Kaijser (1987) report values for SDH and 3-HAD activities in trained subjects that were twice the values of untrained individuals. Costill *et al.* (1976) reported an even greater difference between their endurance-trained and untrained subjects. This group reported that SDH activities in elite distance runners and well-trained middle-distance runners were 3.4- and 2.8-fold greater respectively than activities in untrained individuals. The magnitude of the difference between well-trained and untrained subjects in the studies mentioned above might reflect just how untrained the untrained subjects were. There are considerable differences between merely sedentary subjects and subjects specifically deconditioned (Saltin & Gollnick 1983).

Similarly, even within trained subjects, Sjøgaard (1984) showed muscle enzyme activities (CS and 3-HAD) that were 30–60% higher in elite, when compared to merely competitive cyclists. These data again highlight the magnitude of the trainability of the oxidative enzymes. However, it is necessary to consider the results of longitudinal studies, to confirm that enzyme differences are an adaptation to endurance training and not a pre-determined selection requirement for successful endurance performance.

3.4 Adaptability

A five month longitudinal training study in subjects not previously participating in endurance exercise (Gollnick *et al.* 1973) showed that oxidative enzyme activity (SDH) is adaptable and that the increase can indeed be sizeable (2-fold increase, $p < 0.01$). Subsequently, many studies have supported the findings of this study although inter-study comparisons of absolute values are difficult because of variance in

methodology (Bylund, Bjurö, Cederland, Holm, Lundholm, Sjöström, Ängqvist & Scherstén 1977; Chi, Hintz, Coyle, Martin, Ivy, Nemeth, Holloszy & Lowry 1983; Costill *et al.* 1976). Nevertheless, it is clear that the extent of adaptation to the same training protocol can vary extensively and is not only influenced by the baseline levels (see Figure 1) (Henriksson & Reitman 1976).

Henriksson & Reitman (1976) attempted to isolate the adaptations with respect to fibre type after either continuous or high-intensity training, using single fibre techniques. In a continuous training group, SDH activity increased only in Type I fibres (by 32%), whilst in the higher intensity training group, SDH activity increased only in Type II fibres (by 49%). These results imply that the recruitment pattern during training is important in stimulating oxidative adaptations and that Type II fibres can adapt substantially.

The lack of an exclusive association between oxidative enzyme capacity and percentage Type I fibres has been confirmed by other studies. In a study by Costill *et al.* (1976), the individual with the lowest proportion of Type I fibres in fact had the highest total SDH activity and there was no correlation between Type I percentage and SDH activity for the whole group. Furthermore, the study of Essén-Gustavsson & Henriksson (1984) determined enzyme profiles in pools of single fibres from endurance-trained and control subjects. The oxidative capacity of the Type II fibres of the endurance-trained was higher than the oxidative capacity of the Type I fibres of the controls. Clearly, the oxidative capacity of even Type II muscle fibres can be increased considerably and this may be a reason that fibre type alone cannot explain differences in endurance performance capacity.

3.5 Genotype dependence of adaptability

Simoneau, Lortie, Boulay, Marcotte & Thibault (1986) investigated whether there was any genotypic influence in the response to training of the skeletal muscle in 14 pairs of monozygotic twins. The results showed marked inter-individual variation in response to training with a significant intra-twin correlation for change in MDH. Hamel *et al.* (1986) also investigated the trainability of skeletal muscle enzyme activities in twins ($n = 6$). The changes in enzyme activities in the later half of the endurance training programme (7–15 weeks) appeared to be more genotype dependent than the changes in the first seven weeks. Change in MDH and 3-HAD showed sizeable intra-pair correlation coefficients, e.g. over the entire training programme, 3-HAD had an intra-pair correlation coefficient of $r = 0.69$. The intra-pair correlation coefficient was even higher over the second seven-week period ($r = 0.89$). It is difficult to explain why the intra-pair variability is so vastly different between the two seven-week periods, however, absolute change in 3-HAD activity did occur during both periods. The results of this study must be interpreted with caution for two reasons: (a) the small number of twins and (b) the absence of dizygotic twin data for comparison.

In summary, there appears to be genetic determination of the response of some skeletal muscle enzyme activities to training. A relatively high coefficient of variation for enzyme activity analysis from repeated biopsies remains an inherent problem in the interpretation of this data.

3.6 Different populations

Saltin and co-workers (1995a) recently undertook a study to investigate skeletal muscle biochemical characteristics in Kenyan Africans and Scandinavian Caucasians. In the vastus lateralis, CS activity was similar whilst 3-HAD activity was 20% higher in the Kenyans (55.8 vs 45.0 µmol/min/g d.w., $p < 0.05$). In the gastrocnemius, 3-HAD activity was 50% higher in the Kenyans. These data may have been influenced by acute alterations in enzyme activities of the Scandinavian athletes who were not accustomed to altitude, since the biopsies were taken after a two week training camp at altitude. Acute exposure to altitude has previously been shown to influence oxidative capacity (Hoppeler, Howald & Cerretelli 1990). Nevertheless, it is interesting that the differences were seen in 3-HAD and not CS and the discussion above noted that 3-HAD may be the oxidative enzyme most influenced by genotype.

We have investigated the enzyme activities of sub-elite African and Caucasian runners habitually resident and training at sea level in South Africa (Weston *et al.* 1998)(Figure 2). Mean CS activity was 1.5-fold higher ($p < 0.05$) in the five African runners when compared to the seven Caucasian run-

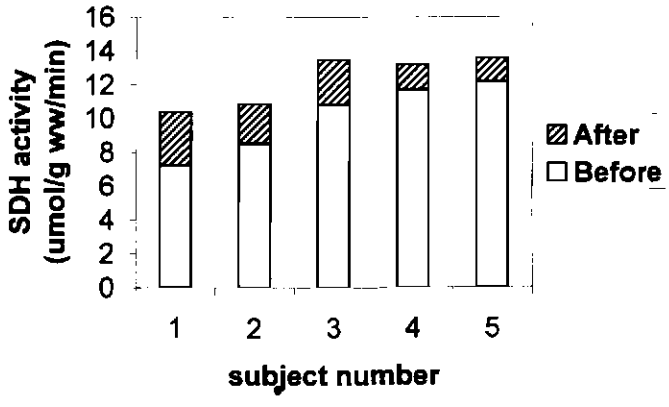

Figure 1 Heterogeneity in both baseline skeletal muscle oxidative enzyme activity and its adaptation to endurance training. Data from Henriksson & Reitman 1976. Clear bars = baseline and hatched bars = improvement in SDH activity in response to 8 weeks of training, three times a week for approximately 30 min at 80% VO_2max. SDH = succinate dehydrogenase activity.

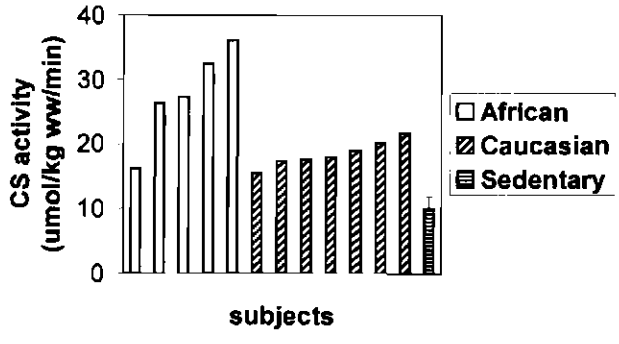

Figure 2 Individual values of skeletal muscle oxidative enzyme activity in African (clear bars) and Caucasian (hatched bars) South African distance runners. For comparison mean values are given for sedentary subjects recruited from both populations (horizontal line bar). CS = citrate synthase.

ners (A: 27.9 ± 7.5 and C: 18.6 ± 2.1 μmol/g w.w./min). Groups were well matched for best 10 km race time (A: 32.6 ± 1.7 vs C: 33.8 ± 2.6 min). There was also no difference in average weekly training distance, although training intensity was not reported. Only one African fell within the range of values for CS activity of the seven Caucasians and it is unlikely that training intensity alone could account for such a large difference. It is therefore tempting to speculate that the substantially higher values in the African runners is genotype- dependent.

A similar magnitude of difference was seen for 3-HAD activities (A: 23.9 ± 4.7 vs C: 15.5 ± 5.1 μmol/g w.w./min, $p < 0.01$). It is also interesting to note that this greater oxidative enzyme activity occurred despite somewhat lower percentage Type I fibres in Africans as discussed earlier. In this study, enzyme activities did not correlate with VO_2max or 10 km race time, but did correlate with a submaximal exercise test, as will be discussed below in Section 4.3. Therefore, once again, the correlation between oxidative enzyme activity and VO_2max that is observed in heterogeneous groups of subjects was not present in this group which was relatively homogeneous for performance and weekly training.

3.7 VO_2max and oxidative enzyme activities

It is clear from the many training studies that the changes in enzyme activities are disproportionately higher than the changes in VO_2max (Henriksson & Reitman 1976; Gollnick *et al.* 1973). In addition, Sjøgaard (1984) followed a group of competitive cyclists throughout a season and observed considerable further improvement of CS and 3-HAD (40–70%) whilst VO_2max remained the same. These data are evidence that a significant correlation between these two variables is likely to be non-causal. This becomes particularly evident when one studies a homogeneous population of trained subjects (Weston *et al.* 1998; Costill *et al.* 1976).

In further support of the dissociation between mitochondrial enzyme activity and VO_2max, is the work of Henriksson & Reitman (1977) who investigated the time course of changes in VO_2max and skeletal muscle enzyme activities, showing that they adapted asynchronously. They suggest that changes in skeletal muscle enzyme activities may be more relevant to submaximal endurance performance.

4. Resistance to fatigue

4.1 Heterogeneity

Prior to discussing the heterogeneity and adaptability of resistance to fatigue, it is necessary to discuss the exercise testing methods *per se*. Because athletes rarely race at VO_2max, simulated time trials and tests of time to fatigue during high-intensity submaximal exercise may be more useful tests than the traditional incremental VO_2max test. These types of tests have been utilised to determine the effect on exercise capacity of e.g. nutritional supplements (Bredle, Stager, Brechue & Farber 1988), training interventions (Weston, Myburgh, Lindsay, Dennis, Noakes & Hawley 1997) and genotype (Bouchard *et al.* 1986).

Because of the diverse objectives behind selecting exercise tests to fatigue, there are numerous different protocols. However, two common factors are, firstly, that the specific test should have a low coefficient of variation (CV) for repeated tests on the same subject, and secondly, that in the case of athletes the duration of the test should be related to the duration of the athlete's competitive event. For example, cyclists are now commonly required to perform time trials (set distance in the shortest possible time) or distance trials (furthest possible distance in a set time) in the laboratory utilising their own bicycle attached to a cycle ergometer (Weston *et al.* 1997). Typically the CV for these tests is between 1% and 2%.

Tests based on similar principles can be performed by non-athletes on laboratory cycles. Boulay, Hamel, Simoneau, Lortie, Prud'homme & Bouchard (1984) developed a 90-minute test of 'aerobic capacity' that quantifies the total amount of work performed on a cycle ergometer within the specified time. These investigators subsequently utilised this test for several studies indicating that the test is reliable and repeatable with an approximately 2% variation between tests.

In sedentary subjects with no history of physical exercise the range of performances differed approximately 2-fold (Lortie *et al.* 1984), once again indicating the wide variation in endurance capacity phenotype amongst individuals.

4.2 Adaptability

The effect of training status on the 90-min work output test can be seen by comparing the sedentary subjects in the study by Lortie *et al.* (1984) with active physical education students studied by Boulay *et al.* (1984). Mean work capacity was 9.6 kJ/kg in the former and 14.7 kJ/kg in the latter.

A significant training effect on the 90-min work output test has been demonstrated: 20 weeks of endurance training resulted in a mean increase of 50% ($p < 0.01$) (Lortie *et al.* 1984). The variation in training effect ranged from 16 to 97% and once again indicates the wide range of phenotypic response to endurance training.

Animal studies have shown a significant relationship between submaximal fatigue resistance and skeletal muscle enzyme activities in response to training. In isolated muscles of, for example, the cat and rat, increases in the ability to resist fatigue during repeated contractions have been associated with an increase in the activity of oxidative enzymes (Peckham, Mortimer & van der Meulen 1973; Hudlická, Brown, Cotter, Smith & Vrbová 1977). These studies suggest that the ability of the muscle to sustain submaximal contractile activity is dependent upon its mitochondrial oxidative capacity. Certainly the magnitude of changes in submaximal endurance capacity after training are similar to the magnitude of the enzyme changes (50% or greater), although this does not necessarily mean that the relationship is unequivocally causal (Davies, Packer & Brooks 1981; Gollnick *et al.* 1973; Karlsson, Nordesjö & Saltin 1974; Simoneau, Kaufmann & Pette 1993).

We have recently shown that a similar relationship exists between submaximal fatigue resistance and oxidative enzyme capacity in humans ($r = 0.7$, $p < 0.05$) (Weston *et al.* 1998). The test designed for this study included several submaximal workloads with the final workload (92% of peak treadmill velocity) continued to exhaustion.

These studies all seem to indicate that the benefit of high oxidative enzyme capacity in the skeletal muscle is not primarily to increase the VO_2max as was previously believed,

but rather to improve the ability to sustain submaximal exercise.

4.3 Influence of genotype

A genotypic influence has been shown for the 90-min work output test. A strong within-pair correlation for monozygotic twins was found ($r = 0.66$, $p < 0.001$) (Bouchard *et al.* 1986a) and it was better than the correlation for dizygotic twins. The heritability for the submaximal test was shown to be greater than for a VO_2max test in the same subjects.

The next question that arises, is whether or not the adaptability of resistance to fatigue is influenced by genotype. The results of another twin study (Hamel *et al.* 1986) indicated that the response to training measured by this test is indeed highly significantly influenced by genotype. The genotypic influence was quantified as accounting for approximately 70% of the variance. No other studies using alternative protocols to test aerobic resistance to fatigue have been reported.

4.4 Different populations

Bosch *et al.* (1990) investigated African and Caucasian marathon runners who were all resident in South Africa. As in the earlier South African studies, the Caucasian runners were considerably taller and heavier than the African runners. When corrected for body mass, VO_2max was not different between groups. Runners ran a simulated marathon on a treadmill at the same percentage of their best marathon time (mean 87%). One of the main findings was that the African runners ran at a higher percentage of VO_2max (76% vs 68%), suggesting that at the same percentage of VO_2max, the African runners would have been able to resist fatigue for longer. However, it should be noted that their absolute running speed would also then have been slower. This raises the complex issue of the best method for setting the workload of submaximal tests. Workloads can be set relative to either VO_2max, HRmax, peak workload or race pace. This remains an unresolved area for future research.

Some tests to fatigue focus on a localised area such as the quadriceps and determine the time taken to reduce force production by e.g. 30% or 50% of the peak force produced by a maximal contraction in a rested condition. Coetzer *et al.* (1993) compared quadriceps fatigue in elite South African African and Caucasian athletes. The African athletes were able to resist fatigue longer during a repeated isometric test of knee extension performed in the seated position with the ankles attached to a stationary strain gauge (6 s maximal voluntary contractions interspersed with 4 s rest). These results may have been influenced by the fact that Caucasian runners were middle distance runners whilst African runners were longer distance runners.

We therefore followed up this study with a study on sub-elite African and Caucasian runners matched for similar peak treadmill velocity and 10 km race times. The mean 10 km race time for African subjects was 33.2 ± 1.6 min and for Caucasian subjects 34.2 ± 1.9 min. The runners were tested on a treadmill and the fatigue resistance task was designed to be repeatable (CV of 2%) and to closely represent the fatigue occurring during a high intensity distance running event. The cumulative time of the running test was between 17 and 30 min, which is approximately equivalent to the duration of a 6–10 km race. The difference in the mean fatigue resistance of the African and Caucasian runners was pronounced: mean time was 21% longer in the African runners. Factors such as age, body size, habitual physical activity, preferred race distance, altitude and test mode were equivalent for both groups. Therefore, several of the limitations of the study by Coetzer *et al.* (1993) were overcome whilst strengthening their conclusion that African athletes have a superior ability to resist fatigue.

In addition to the studies detailed above comparing African and Caucasian individuals, several studies have compared populations native to high altitude (e.g. Andeans) with Caucasians. The Andeans displayed differences in work capacity and metabolism when compared to Caucasians. Specifically, the Andeans accumulate less lactate for a given work rate at altitude than lowlanders exercising at the same work rate at sea level (Hochachka, Stanley, Matheson, McKenzie, Allen & Parkhouse 1991). Furthermore, their work capacity during plantar flexion was significantly higher than Caucasian sedentary individuals, power athletes and endurance athletes (Matheson, Allen, Ellinger, Hanstock, Gheorghiu, McKenzie, Stanley, Parkhouse & Hochachka 1991). This could not have been due to a higher training status of the Andeans because, although habitually active, they were less trained than the athletic lowlanders. This paradox did not disappear after the Andeans had been removed from altitude for six weeks and re-tested at sea level. Therefore the authors concluded that this paradox 'is a developmentally or genetically fixed characteristic of these Andean natives'. Hochachka, Gunga & Kirsch (1997) further elaborate on this theme and lend credence to the hypothesis that there are indeed genotypic influences on endurance exercise phenotype. Specifically, these authors suggest that they may be, at least in part, situated in the portion of the genome that is distinctive for different racial groups.

Conclusion

It is clear that many factors influence endurance performance. The extent to which these factors are genetically determined is still unclear despite numerous studies. There is sufficient evidence to say that at least a component of endurance performance is genetically determined. Finally, it appears that certain differences may occur between individuals of different racial origins although definitive, direct identification of the responsible genes remains to be accomplished.

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