African runners exhibit greater fatigue resistance, lower lactate accumulation, and higher oxidative enzyme activity

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Weston, Adèle R., O. Karamizrak, A. Smith, T. D. Noakes, and Kathryn H. Myburgh. African runners exhibit greater fatigue resistance, lower lactate accumulation, and higher oxidative enzyme activity. J. Appl. Physiol. 86(3): 915–923, 1999.—Nine African and eight Caucasian 10-km runners resident at sea level volunteered. Maximal O2 consumption and peak treadmill velocity (PTV) were measured by using a progressive test, and fatigue resistance [time to fatigue (TTF)] was measured by using a newly developed high-intensity running test: 5 min at 72, 80, and 88% of individual PTV followed by 92% PTV to exhaustion. Skeletal muscle enzyme activities were determined in 12 runners and 12 sedentary control subjects. In a comparison of African and Caucasian runners, mean 10-km race time, maximal O2 consumption, and PTV were similar. In African runners, TTF was 21% longer (P < 0.01), plasma lactate accumulation after 5 min at 88% PTV was 38% lower (P < 0.05), and citrate synthase activity was 50% higher (27.9 ± 7.5 vs. 18.6 ± 2.1 µmol·g wet wt⁻¹·min⁻¹, P = 0.02). Africans accumulated lactate at a slower rate with increasing exercise intensity (P < 0.05). Among the entire group of runners, a higher citrate synthase activity was associated with a longer TTF (r = 0.70, P < 0.05), a lower plasma lactate accumulation (r = -0.73, P = 0.01), and a lower respiratory exchange ratio (r = -0.63, P < 0.05). We conclude that the African and Caucasian runners in the present study differed with respect to oxidative enzyme activity, rate of lactate accumulation, and their ability to sustain high-intensity endurance exercise.

ELITE 10-KM RUNNERS race at a sustained pace of ~2.7 min/km, the equivalent of 22 km/h. The ability to maintain such a high intensity for almost half an hour requires a superior resistance to fatigue throughout exercise of this duration. However, laboratory investigations of this type of runner have primarily focused on measuring maximal O2 consumption (O2max), anaerobic threshold, and running economy (7, 9, 31) rather than utilizing reproducible laboratory tests of a similar duration and intensity to the event in question.

Despite the overwhelming dominance of high-intensity endurance running events (3,000–10,000 m) by East African athletes, few scientific studies have been completed to clarify the physiological differences between African and Caucasian distance runners (2, 6, 39, 40). The findings of Coetzee and co-workers (6) indicated a greater resistance to fatigue in African South African compared with Caucasian South African runners, despite comparable O2max. The test used to quantify resistance to fatigue was one involving repeated isometric contractions of the quadriceps muscles, but fatigue during this type of test may not be representative of fatigue occurring during distance running. A partial explanation of the greater resistance to fatigue in the African group may be that they were predominantly long-distance runners compared with the Caucasian athletes, who were predominantly middle-distance track athletes. Thus either preselection for the event in which they were successful, or subsequent adaptations as a result of different training methods, may have biased the results. Therefore, the primary purpose of this study was to investigate fatigue resistance by using a dynamic, running test protocol in groups of African and Caucasian subelite runners, matched for preferred racing distance.

Although the direct mechanisms of fatigue are complex and not completely understood, adaptation to endurance training results in an increased time to fatigue at a standardized workload (23) and has also been shown to result in a lower accumulation of metabolites for an absolute workload (17, 18, 23, 34). A lower accumulation of metabolites has also consistently been reported in studies of African runners (2, 6, 40).

In the aforementioned study by Coetzer et al. (6), plasma lactate concentrations were significantly lower in the African runners at two submaximal running workloads of moderate intensity despite O2 consumption (O2) similar to that in the Caucasian runners. Bosch et al. (2) reported a higher fractional utilization of O2max for the same lactate accumulation in African South African compared with Caucasian South African marathon runners, and Saltin et al. (39) also found lower plasma lactate accumulation in elite Kenyan runners compared with Scandinavian runners. Therefore, the second purpose of the present study was to investigate plasma lactate concentrations at the same relative running intensities.

One of the mechanisms of lower plasma lactate accumulation is an increase in skeletal muscle oxidative enzyme capacity (16). Saltin et al. (39) recently investigated skeletal muscle biochemical characteristics of Kenyan and Scandinavian runners after the Scandinavians had spent 2 wk at altitude. Citrate synthase (CS) activity, fiber-type proportion, fiber cross-sectional area, and capillarization of the vastus lateralis muscle were not different between the Kenyan and Scandinavian senior runners. However, 3-hydroxyacyl-CoA dehydrogenase (3-HAD) activity was 20% higher in the Kenyans (P < 0.05). In the gastrocnemius of the...
Kenyans, 3-HAD activity was 50% higher. Unfortunately, these interesting results are difficult to interpret because of the habitual residence of these Kenyan runners at altitude, whereas the Scandinavian runners were normally resident at sea level and were subject to acute adaptations to a change in their environment just before the study. Previous studies have shown that short-term exposure to altitude may affect skeletal muscle citric acid cycle and fat oxidation enzyme activities (19, 47, 52).

However, it is also possible that the habitual residence of the Kenyan runners at altitude may explain some of the findings of Saltin and co-workers (39, 40), because a lower accumulation of lactate has also been reported in several population groups born and residing at altitude (22, 36). This lower lactate accumulation appears to be largely unaffected by acclimatization to sea level (21) and, in contrast to endurance-trained subjects at sea level, high-altitude natives do not have enhanced skeletal muscle oxidative enzyme capacity (26). This suggests that it is not a chronic adaptation to altitude per se but that this metabolic characteristic may be inherent in these groups (21). If this were indeed the case, then it is possible that the reduced lactate accumulation in the African runners in the three studies discussed above (2, 6, 40) may also be inherent and thus unrelated to muscle oxidative capacity. The final purpose of this study was to compare skeletal muscle enzyme activities in these two populations, when matched for habitual environmental conditions, and to relate these to lactate accumulation and resistance to fatigue.

METHODS

Subjects. A total of 17 subelite distance runners participated in this study. Subject groups were matched for primary racing distance (10 km), with a similar mean level of performance in 10-km races. All subjects were seasoned competitors (>3 yr of competitive running) recruited from local running clubs. Subject characteristics are listed in Table 1.

Fifteen subjects (7 Africans, 8 Caucasians) completed the fatigue resistance test, and 12 subjects (5 Africans, 7 Caucasians) volunteered to undergo a needle biopsy of the vastus lateralis for the analysis of enzyme activity and fiber-type proportion. Two of these subjects withdrew from the study before exercise testing was completed, and therefore a total of 10 subjects undertook both the fatigue resistance trial and the muscle biopsy (see Table 1 for clarification). Twelve healthy young sedentary controls (6 Africans, 6 Caucasians, exercising <1 h/wk) were recruited for a muscle biopsy to concurrently determine mean enzyme activities in sedentary individuals in our laboratory. All subjects were informed of the possible risks of the experimental procedures, and all gave their written informed consent. The study was approved by the Ethics and Research Committee of the Faculty of Medicine, University of Cape Town.

Procedure. Before the study, all subjects came to the laboratory to familiarize themselves with the testing procedures, in particular the use of the treadmill and breathing mask.

Anthropometry. Height and weight were measured, four skinfold measurements were made (triceps, biceps, suprailiac, subscapular), and %body fat was calculated by using the formula of Durnin and Womersley (11).

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Subject No./ Group</th>
<th>Age, yr</th>
<th>10-km Time, min</th>
<th>Training Volume, km/wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>African runners</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>25</td>
<td>32.0 ± 120</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>32.3 ± 100</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>34</td>
<td>34.9 ± 65</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>33</td>
<td>37.3 ± 70</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>26</td>
<td>33.2 ± 100</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>25</td>
<td>34.3 ± 95</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>29</td>
<td>35.5 ± 62</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>27</td>
<td>29.3 ± 130</td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>28.3 ± 3.5</td>
<td>33.6 ± 2.4</td>
<td>93 ± 25</td>
</tr>
</tbody>
</table>

Caucasian runners

<table>
<thead>
<tr>
<th>Subject No./ Group</th>
<th>Age, yr</th>
<th>10-km Time, min</th>
<th>Training Volume, km/wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>27</td>
<td>32.0 ± 120</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>32.3 ± 100</td>
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<tr>
<td>3</td>
<td>34</td>
<td>34.9 ± 65</td>
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<td>4</td>
<td>33</td>
<td>37.3 ± 70</td>
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<td>5</td>
<td>26</td>
<td>33.2 ± 100</td>
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<td>6</td>
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<tr>
<td>8</td>
<td>27</td>
<td>29.3 ± 130</td>
<td></td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>25.4 ± 7.5</td>
<td>32.8 ± 1.8</td>
<td>107 ± 42</td>
</tr>
</tbody>
</table>

In the group of African runners, subjects 1–4 and 6–9 were Xhosa and subject 5 was Zulu. *Muscle biopsy taken; †subject did not perform a time-to-fatigue test.

VO2max/peak treadmill velocity (PTV) test. Subjects completed a PTV test (Powerjog EG30, Birmingham, UK) with concurrent measurement of VO2, minute ventilation, respiratory exchange ratio (RER), and heart rate (HR). Gas-analysis equipment (Oxycon Alpha, Jäeger Mijnhardt, Wuerzburg, The Netherlands) was calibrated before each test by using a certified known gas mixture, and the pneumotachometer was calibrated with a 3-liter calibrated syringe. After a 5-min warm-up at 14 km/h, the testing protocol commenced at 14 km/h, with 1-km increments in velocity every minute until volitional exhaustion. PTV was designated as the last velocity that was maintained for a full 60 s. At exactly 3 min after cessation of exercise, a venous blood sample was obtained for determination of plasma lactate concentration. Samples were immediately centrifuged at 4°C at 3,000 rpm, and plasma was obtained and frozen for later spectrophotometric analysis (Beckman DU-62, Beckman Industries). The assay utilized a commercially available lactate kit (Boehringer Mannheim).

“Fatigue resistance” test. On a third visit to the laboratory, subjects completed a discontinuous test of fatigue resistance, comprising four workloads. For the purposes of this study, fatigue is defined as the inability to continue running at a standardized percentage of PTV. A 20-gauge cannula (Jelco, Critikon) was inserted into a forearm vein and flushed with heparinized saline. Subjects warmed up for 5 min at 14 km/h before completing the four consecutive workloads. These workloads were selected after pilot trials to determine those workloads most suitable for runners of the caliber expected for this study. The purpose was that the first three workloads could all be sustained for 5 min each, before subjects proceeded to the fourth workload, which was continued until fatigue. Subjects exercised for 5 min at 72% (workload 1), 5 min at 80% (workload 2), and 5 min at 88% (workload 3) of their predetermined PTV. VO2, minute ventilation, RER, and HR were measured throughout exercise. Between workloads, the subject ceased running for 1 min while a venous blood sample was obtained at 30 s for subsequent analysis of plasma lactate concentration. Exactly 1 min after the comple-
tion of workload 3, the subject began the final workload, in
which he ran at 92% of his PTV until exhaustion (workload
4). A venous blood sample was obtained at 30 s after exercise
determination of plasma lactate concentration. Total "time to fatigue" is reported as the total exercise time (i.e., 15
min + time sustained at workload 4). The repeatability of
the time to fatigue achieved by using the above-mentioned exer-
cise protocol was estimated by calculating the coefficient of
variation after the trial was repeated three times in three
individuals. The mean coefficient of variation was 2.0%. Further investigation of the reproducibility of this test has
indicated a correlation of $r = 0.97$ between two tests in nine
10-km runners (B. Reid and A. Weston, unpublished observa-
tions).

Muscle biopsy. On a separate day, with subjects in the
rested condition, a needle biopsy of the vastus lateralis was
obtained by using the suction-assisted technique described by
Evans et al. (13). On extraction the biopsy sample was divided
down into two portions. The first was immediately frozen in liquid
for later enzyme assays, and the second was appropriately frozen for histochemical analy-
sis.

For enzyme analyses, muscle samples (~20 mg wet wt)
were homogenized in a potassium phosphate buffer, pH 7.4,
and enzyme activities were assayed spectrophotometrically
in duplicate at 25°C (Beckman DU-62, Beckman Instru-
ments). CS (EC 4.1.3.7) was assayed by using a modified
Snell technique [44; for more details, see Weston et al. (51)].
Phosphofructokinase (PFK) was assayed by using the method
of Ling et al. (27). In addition, 3-HAD (EC 1.1.1.35) was
assayed in just seven athletes (4 Africans, 3 Caucasians). Insuffi-
cient muscle sample was available to assay this enzyme in all
subjects. 3-HAD was assayed spectrophotometrically in duplicate
by using methods described in more detail elsewhere (50).

Utilizing the above assay methodology, test-retest repro-
ducibility was estimated as $r = 0.97$ for CS (95% confidence
interval 0.87–0.99), $r = 0.88$ for 3-HAD (95% confidence
interval 0.51–0.98), and $r = 0.98$ for PFK (95% confidence
interval 0.87–0.99). Enzyme activities are expressed in micro-
moles per gram of muscle wet weight per minute.

Routine histochemistry was carried out by using a myosin
ATPase stain at pH 4.3 and 9.4 to differentiate type I and type
II fibers (10). All the fibers viewed in one field were counted
to calculate the relative fiber-type proportions. In all individu-
als, this was >150 fibers; however, in all but three subjects,
this was >200 fibers.

Statistics. In the case of homogenous variance, a Student's
$t$-test assuming equal variance was utilized for the compar-
ison of the means of two groups, and an ANOVA with post hoc
Tukey's honestly significant difference test was utilized for the
comparison of the means of three groups (i.e., where
sedentary data were included).

In the case of differing variances, a more conservative
Student's $t$-test assuming unequal variance was utilized to
compare the means of two groups. Because an ANOVA is
inappropriate to compare means among three groups in
conditions of differing variances (CS activity, PFK activity), in
this case the $t$-test with unequal variance was repeated for
each comparison between two particular groups. A Bonferroni
correction was applied to the level of significance to account
for the repeated use of the $t$-test and increased probability of a
type I error. A repeated-measures general linear model was
utilized for the analysis of interactions between race and
workloads of the fatigue resistance test (SPSS version 7.5).

Pearson's correlation coefficient was used to investigate the
association between time to fatigue and metabolic variables
for the group of runners as a whole, whereas intraclass
correlations were used as a measure of reproducibility. Corre-
lations were not calculated within groups because of insuffi-
cient numbers. Coefficient of variation was used to estimate
repeatability of the fatigue resistance protocol.

RESULTS

African runners were shorter and lighter but had a
similar level of body fat compared with the Caucasian
runners. Mean anthropometric data and results of the
maximal treadmill exercise test are given in Table 2.
There was no difference in the maximal exercise values
between the African and Caucasian runners. In all
individual subjects, maximal HR was >90% predicted
maximal HR and maximal RER was >1.0 at the end of
maximal exercise.

When this homogenous group of subelite runners is
considered as a whole, PTV was moderately related to
personal-best 10-km time ($r = -0.65, P < 0.01$), but
there was no significant relationship between $V_{\text{O}}^{\text{max}}$ and
personal-best 10-km time ($r = -0.37, not signifi-
cant).

The results of the first three submaximal workloads (5 min each) of the time-to-fatigue test are presented in
Table 3, together with the data taken at the point of
fatigue in the final workload. At these workloads, the
mean HR as a percentage of maximum HR was 81, 86,
and 93%, respectively, with no difference between groups
at any of the workloads. African runners had significa-
litantly lower plasma lactate accumulation at 88% PTV ($P < 0.05$). There was a significant interaction effect
between race and workload on the plasma lactate
concentration over the standardized 15 min of the test
($P < 0.05$). The increase in plasma lactate concentra-
tion from the initial workload (72% PTV), where it was
comparable between groups, to the final standardized
workload (88% PTV), was 2.1-fold in the Africans and
3.2-fold in the Caucasians (Fig. 1, $P < 0.05$). RER and
$V_{\text{O}}^{\text{max}}$ were not different at any of the workloads.

There was a marked difference between the two
groups in the mean time sustained at workload 4, with
African runners continuing to exercise at this high
intensity for almost twice as long as their Caucasian
counterparts (Fig. 2, Table 3). This 4-min difference

Table 2. Anthropometric and maximal exercise
test results

<table>
<thead>
<tr>
<th></th>
<th>African Runners (n = 9)</th>
<th>Caucasian Runners (n = 8)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height, cm</td>
<td>172 ± 5</td>
<td>181 ± 9</td>
<td>$P &lt; 0.05$</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>59.4 ± 6.0</td>
<td>69.1 ± 5.7</td>
<td>$P &lt; 0.05$</td>
</tr>
<tr>
<td>%Body fat</td>
<td>11.6 ± 2.7</td>
<td>12.4 ± 2.8</td>
<td>NS</td>
</tr>
<tr>
<td>Peak treadmill velocity, km/h</td>
<td>21.3 ± 1.9</td>
<td>22.4 ± 1.2</td>
<td>NS</td>
</tr>
<tr>
<td>$V_{\text{O}}^{\text{max}}$, ml·kg$^{-1}$·min$^{-1}$</td>
<td>61.9 ± 5.9</td>
<td>65.2 ± 7.2</td>
<td>NS</td>
</tr>
<tr>
<td>HR$^{\text{Rmax}}$, beats/min</td>
<td>192 ± 7</td>
<td>191 ± 6</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma [lactate]$^{\text{Rmax}}$, mmol/l</td>
<td>10.5 ± 2.5</td>
<td>11.6 ± 2.9</td>
<td>NS</td>
</tr>
<tr>
<td>RER$^{\text{Rmax}}$</td>
<td>1.10 ± 0.06</td>
<td>1.13 ± 0.06</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are means ± SD, n. No. of subjects: $V_{\text{O}}^{\text{max}}$, maximal O$_2$ consumption; HR$^{\text{Rmax}}$, maximal heart rate; [lactate]$^{\text{Rmax}}$, maximal lactate concentration; RER$^{\text{Rmax}}$, maximal respiratory exchange ratio; NS, not significant. Significance was determined by using Student's unpaired $t$-test.
Table 3. Submaximal exercise test results

<table>
<thead>
<tr>
<th></th>
<th>African Runners (n = 8)</th>
<th>Caucasian Runners (n = 7)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Workload 1 (72% of PTV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>152 ± 18</td>
<td>154 ± 19</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂, ml · kg⁻¹ · min⁻¹</td>
<td>46.5 ± 8.1</td>
<td>50.8 ± 5.5</td>
<td>NS</td>
</tr>
<tr>
<td>RER</td>
<td>0.91 ± 0.04</td>
<td>0.89 ± 0.06</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma [lactate], mmol/l</td>
<td>2.3 ± 0.9*</td>
<td>2.4 ± 0.7</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Workload 2 (80% of PTV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>163 ± 15</td>
<td>166 ± 15</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂, ml · kg⁻¹ · min⁻¹</td>
<td>51.3 ± 9.7</td>
<td>56.0 ± 7.3</td>
<td>NS</td>
</tr>
<tr>
<td>RER</td>
<td>0.94 ± 0.05</td>
<td>0.93 ± 0.06</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma [lactate], mmol/l</td>
<td>2.9 ± 2.5‡</td>
<td>4.4 ± 2.0</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Workload 3 (88% of PTV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>176 ± 13</td>
<td>175 ± 12</td>
<td>NS</td>
</tr>
<tr>
<td>VO₂, ml · kg⁻¹ · min⁻¹</td>
<td>55.6 ± 7.8</td>
<td>61.9 ± 8.2</td>
<td>NS</td>
</tr>
<tr>
<td>RER</td>
<td>0.97 ± 0.07</td>
<td>0.96 ± 0.07</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma [lactate], mmol/l</td>
<td>4.8 ± 3.2</td>
<td>7.7 ± 2.8</td>
<td>P &lt; 0.05</td>
</tr>
<tr>
<td><strong>Workload 4 to fatigue (92% PTV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>188 ± 7</td>
<td>185 ± 7</td>
<td>NS</td>
</tr>
<tr>
<td>%HＲmax</td>
<td>99.2 ± 3.8</td>
<td>97.4 ± 4.4</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma [lactate], mmol/l</td>
<td>7.2 ± 4.8†</td>
<td>11.1 ± 3.5</td>
<td>NS‡</td>
</tr>
<tr>
<td>Time to fatigue, mins</td>
<td>7:56 ± 3:45</td>
<td>3:57 ± 2:05</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

Values are means ± SD. n, No. of subjects; PTV, peak treadmill velocity. HR was obtained within the final 15 s of the workloads. *†: n = 7 and n = 6 Subjects, respectively, because insufficient blood was obtained within the 1-min interval between workloads; ‡: Significance was determined by Student’s unpaired t-test. 

represents a 21% greater resistance to fatigue with respect to total exercise time (1,376 ± 226 s vs. 1,137 ± 126 s, P < 0.01) and a 98% greater resistance to fatigue with respect to the time for the final high-intensity workload alone (476 vs. 237 s, P < 0.01).

Mean skeletal muscle enzyme activities and fiber-type proportion for African and Caucasian runners and

the sedentary control subjects are displayed in Table 4. The CS activity was 50% higher (P = 0.02) in the African runners compared with the Caucasian runners, but the CS activity in both groups of runners was significantly higher than in the sedentary control subjects (both P < 0.001) (Fig 3). Mean PFK activity was 28% higher in the African runners, but this did not reach significance at the 5% level. Although not measured in all subjects, 3-HAD activity was also significantly higher in the African runners (n = 4) compared with the Caucasian runners (n = 3) and to a similar extent when compared with CS (Table 4).

Caucasian runners had a higher percentage of type I fibers than did the sedentary control subjects (P <

Table 4. Skeletal muscle enzyme activities and fiber-type proportion

<table>
<thead>
<tr>
<th></th>
<th>African Runners</th>
<th>Caucasian Runners</th>
<th>Sedentary Control Subjects</th>
<th>Intergroup Comparison</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>CS*</td>
<td>27.9 ± 7.5</td>
<td>18.6 ± 2.1</td>
<td>9.8 ± 1.2</td>
<td>A&gt;C</td>
<td>P = 0.02</td>
</tr>
<tr>
<td>PFK*</td>
<td>50.9 ± 7.6</td>
<td>39.9 ± 16.0</td>
<td>53.1 ± 12.7</td>
<td>A=C</td>
<td>P = 0.001</td>
</tr>
<tr>
<td>3-HAD†</td>
<td>23.9 ± 4.7</td>
<td>15.5 ± 5.1</td>
<td>48.0 ± 17.3</td>
<td>A=C</td>
<td>P &lt; 0.01</td>
</tr>
<tr>
<td>%Type I‡</td>
<td>48.0 ± 17.3</td>
<td>67.1 ± 17.5</td>
<td>44.2 ± 12.1</td>
<td>A=C</td>
<td>P &lt; 0.01</td>
</tr>
</tbody>
</table>

Values are means ± SD. CS, citrate synthase; PFK, phosphofructokinase; 3-HAD, 3-hydroxyacyl-CoA dehydrogenase; enzyme units, μmol · g wet wt⁻¹ · min⁻¹ · h⁻¹; A, African runners; C, Caucasian runners; S, sedentary control subjects. X = Y indicates no significant difference between groups X and Y by using the statistical comparison denoted by each symbol: *unpaired t-test assuming unequal variance. Level of significance is 2% after adjustment for repeated tests (n = 5, 7, and 12 subjects, respectively); †t-test assuming equal variance (n = 4 and 3 subjects, respectively); ‡ANOVA (P < 0.01) with post hoc Tukey’s honestly significant difference (n = 5, 7, and 12 subjects, respectively).
0.01); however, the statistical difference in the proportion of type I fibers in African runners compared with that in the sedentary control subjects was not significant. CS activity was not related to the percentage of type I fibers, and PFK activity was not related to the proportion of type II fibers. There was no significant association between 10-km time (min) and percentage of type I fibers in this homogenous group of well-trained runners ($r = 0.38$, not significant).

There was no relationship between CS activity in the vastus lateralis and $V_{\text{O2max}}$ or present 10-km race time in the group of subjects who underwent a muscle biopsy. However, the total time that the runner was able to resist fatigue during the fatigue resistance running test was significantly related to the vastus lateralis CS activity ($r = 0.70$, $P < 0.05$, Fig. 4).

The relationship between total time to fatigue and metabolic parameters was investigated by using the data from both the 88% PTV exercise bout, which was of equal duration for all subjects, and the metabolic data of the 92% PTV bout, which was of variable length for all subjects and far from steady state. In the 15 subjects who completed the fatigue resistance test, total time to fatigue was significantly correlated with plasma lactate concentration at 88% PTV ($r = 0.63$, $P < 0.01$), although considerable individual variation exists. The plasma lactate concentration result obtained immediately after cessation of the 92% PTV workload showed a similar relationship to that at 88% PTV ($r = 0.76$, $P < 0.01$, $n = 13$).

To investigate in more detail the mechanism by which CS activity is related to submaximal fatigue resistance, the relationship between CS activity and the submaximal steady-state metabolic responses measured during exercise at 88% PTV was investigated. This workload was chosen for analysis because it was of the same duration for all subjects (5 min), and it immediately preceded the intensive, non-steady-state 92% PTV time-to-fatigue bout. Plasma lactate concentration was available in all individuals at the 88% PTV workload. At 88% PTV, a higher CS activity was significantly associated with a lower plasma lactate ($r = 0.73$, $P = 0.01$; Fig. 5) and a lower RER ($r = 0.63$, $P < 0.05$) in the 10 runners for whom CS activity data were available.

**DISCUSSION**

The first main finding of the intergroup comparison in the present study was that the African runners had a higher resistance to fatigue when running at the same pace compared with the Caucasian runners. This finding is consistent with previous reports showing that African runners have a higher proportion of type I fibers, which are more resistant to fatigue, compared with Caucasian runners. However, the statistical difference in the proportion of type I fibers was not significant, indicating that other factors, such as muscle fiber size, may also contribute to the observed differences in fatigue resistance.

The absence of a relationship between CS activity and the percentage of type I fibers suggests that other factors, such as muscle fiber type distribution, may be more important than CS activity in determining fatigue resistance. The lack of a relationship between CS activity and $V_{\text{O2max}}$ or present 10-km race time further supports this notion.

The significant correlation between total time to fatigue and plasma lactate concentration at 88% PTV indicates that lactate accumulation is an important factor in determining fatigue resistance. This finding is consistent with previous reports showing that lactate accumulation is associated with fatigue during endurance exercise. The lack of a similar relationship at 92% PTV suggests that other factors, such as muscle fiber type distribution, may also play a role in determining fatigue resistance at higher intensities.

To investigate in more detail the mechanism by which CS activity is related to submaximal fatigue resistance, the correlation between CS activity and the submaximal steady-state metabolic responses measured during exercise at 88% PTV was investigated. The significant correlation between CS activity and plasma lactate concentration at 88% PTV suggests that CS activity is related to lactate accumulation during submaximal exercise. This finding is consistent with previous reports showing that CS activity is an important factor in lactate metabolism during endurance exercise.

The correlation between CS activity and RER at 88% PTV suggests that CS activity is related to substrate oxidation during submaximal exercise. This finding is consistent with previous reports showing that CS activity is related to substrate metabolism during endurance exercise.

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percentage of PTV than did the Caucasian runners, despite similar Vo2max and PTV values in both groups. This finding is in agreement with that of Coetzer and co-workers (6), who found greater isometric fatigue resistance in African South African distance runners than in their Caucasian counterparts. The advantage of the present study is that it investigated fatigue resistance during a newly developed running test designed 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- to 10 km race. Furthermore, subjects were all distance runners presently in training for a 10 km race and were relatively homogenous with respect to their range of 10 km performances (30–37 min). Therefore, several of the limitations of the previous study (6) have been overcome, whereas their conclusions were strengthened.

The difference in fatigue resistance between the African and Caucasian runners in the present study was pronounced: mean time was 98% longer for the final workload in the African runners compared with the Caucasian runners. Because the workload was set relative to the PTV, it could be argued that the maximal test of the African runners was not a truly maximal effort, in which case 92% PTV would represent a somewhat lower relative intensity in the Africans compared with that in the Caucasians. However, this was not the case because all maximal test results (RER, HR, plasma lactate concentration) were similarly high in both groups.

The second major finding of this study was the higher oxidative enzyme activities in the African subjects. This was apparent for both CS and 3-HAD, although the reader is cautioned with respect to the smaller sample number for 3-HAD. Mean activities of these key oxidative enzymes were 50 and 54% greater, respectively, in the African runners compared with the Caucasian runners. When CS activity is compared with that reported by other investigators, the mean of Caucasian runners (27.9 µmol · g wet wt⁻¹ · min⁻¹) is similar to that reported by others in well-trained athletes (38), but the mean of the African runners (27.9 µmol · g wet wt⁻¹ · min⁻¹) was considerably higher. The CS activity of only one African runner fell within the range of CS activities in the Caucasian runners. CS activity in the African runners was approximately threefold higher than in sedentary subjects, whereas, for Caucasian runners, the difference was twofold. The latter is comparable with the difference reported by Essen-Gustavsson and Henriksson (12) between trained and untrained subjects.

These data are in partial agreement with the findings of the only previous investigation of enzyme activities in African distance runners. Saltin et al. (39), who investigated skeletal muscle biochemistry in elite Kenyan distance runners, also reported a higher activity of 3-HAD in the African athletes but found CS activity to be the same as in the Caucasian runners. However, the athletes in the present study all lived and trained at sea level. This excludes any possible effects of altitude that may have confounded the interpretation of the results of the study by Saltin et al. (39) caused by the temporary visit to altitude of the Scandinavian control subjects. The combined data of these two studies support a hypothesis that African distance runners have enhanced skeletal muscle oxidative enzyme capacities, but the cause of this is not clear at present.

A possible explanation of the differences in enzyme activities could be a difference in fiber-type proportions. Type I fibers are traditionally considered to be oxidative fibers with a higher CS activity. However, in the present study the African runners tended to have a lower percentage of type I fibers than did the Caucasians (17% mean difference). Rather than being detrimental to endurance performance, it is possible that type II fibers serve as important power generators at the high running speeds that are now utilized for successful 10 km performance. Furthermore, it is also possible for type II fibers to exhibit considerable variability in oxidative capacity, and a continuum of oxidative potential of fibers regardless of fiber-type classification may be a more correct description (33). It is unlikely that any differences in the finer classification of fiber type per se that were not measured in this study could explain the 50% greater activity of CS in the Africans, as reported herein.

Alternative explanations for the reported differences in oxidative enzyme capacity could be that they are a result of a baseline genetic difference, an environmental and/or lifestyle difference such as training, or a genetically different response of skeletal muscle to training. The sedentary group in the present study was composed of six Africans and six Caucasians, but no difference in enzyme activity was apparent (data not shown). At the present time it is not possible to determine whether the distribution of baseline, untrained values in Africans is different from that in Caucasians. Training distance was not different between groups in the present study, but training intensity was not quantified. Although the substantial difference in oxidative enzyme activities in the present study is unlikely to be explained by subtle differences in training intensity alone, there may be a difference in the genetic response to training. This phenomenon has been demonstrated by the similar response of enzymes to endurance training in twin pairs (3).

Regardless of the cause for enhanced oxidative capacity, it appears to have specific advantageous functional and metabolic consequences in vivo. Although not correlating with present 10 km performance in this homogenous group of runners, significant relationships existed between CS activity and both plasma lactate concentration and time to fatigue at a workload set relative to each subject's maximal PTV (Figs. 5 and 4, respectively). This supports the hypothesis that a high skeletal muscle oxidative capacity is important for optimizing one's performance, especially when determined by a controlled laboratory test of fatigue resistance, which is independent of other confounding deter-
minants of 10-km performance such as course design, environmental conditions, nutrition, and motivation. Studies in animal models also support these relationships. Increases in the ability to resist fatigue during repeated contractions in isolated muscles of the cat, dog, and rat have been associated with an increase in the activity of oxidative enzymes and the resultant reduction in cellular perturbations during exercise (24, 29, 32).

The direct mechanisms of fatigue during exercise are complex and subject to controversy. Adding to the complexity of this issue is the problem of the extent to which extracellular measurements reflect the intracellular disturbances of homeostasis with which muscle fatigue has been associated (5, 8, 14, 35, 37, 49). One might hypothesize that the lower rate of plasma lactate accumulation by the African runners could have played a direct role in enhancing their resistance to fatigue by influencing sarcoplasmic reticulum function, as has been seen in animal models (15). However, the fact that within the African runners there was a large individual variation between absolute plasma lactate concentration at 88% PTV and the time to fatigue argues against the hypothesis that plasma lactate concentration itself directly influences fatigue in vivo. Bangsbo et al. (1) have recently shown that exercising with a pre-elevated venous blood lactate of ~9 mmol/l hastened the onset of fatigue by 26%, compared with a minimal blood lactate condition. This was accompanied by a faster elevation of muscle lactate to a critical level of 25 mmol/l, without a change in the rate of glycolysis. This suggests that, although blood lactate is not an exact indicator of the muscle lactate, it does influence the accumulation of lactate in the muscle cells, predominantly by decreasing the ability of the lactate to efflux from the muscle. However, the findings do suggest muscle lactate is a far better indicator of fatigue than is plasma lactate per se. Furthermore, these researchers (1) showed that plasma potassium levels were similarly elevated at exhaustion in both conditions, suggesting that the concentration of potassium in the interstitium may also be involved in the mechanism for fatigue. For this reason, interpretation of the relationship between plasma lactate and fatigue in the present study should be made with caution.

Irrespective of the relationship with fatigue, the present study does report a significant interaction effect of racial group and workload on the accumulation of lactate over the three standardized submaximal workloads. In absolute terms, this was a 2.1-fold mean increase in lactate in the Africans vs. a 3.2-fold mean increase in lactate in the Caucasians over the first 15 min of the test (Fig. 1). This is consistent with the indirect findings of Bosch et al. (2). These differences may be related to differences in lactate kinetics, i.e., production, removal from muscle or plasma compartments, and net accumulation, and they warrant further investigation.

Historically, differences in blood lactate accumulation have been ascribed to differences in muscle lactate production. However, tracer studies of lactate kinetics have highlighted the importance of lactate removal (28, 45) and the importance of lactate as a metabolic substrate in the active and nonactive muscles (4, 46). It is with respect to lactate oxidation that oxidative enzyme activity may be relevant. Endurance training appears to increase the relative contribution of lactate removal (as opposed to lactate production) to the delaying of lactate accumulation (28). Although it has been shown that the plasma lactate in a group of trained individuals is related to oxidative enzyme activity (42, 43), we have no data relating the lactate removal directly to oxidative enzyme capacity. Thus one can only speculate that, in this study, the higher oxidative capacity may be related to plasma lactate concentration via both a reduction in lactate production and an enhancement of lactate removal. The latter is likely to be related to capillarization or lactate transporter density or both. The relationship between the onset of blood lactate accumulation and muscle capillarity has previously been shown by Tesch et al. (48), and the relationship between the lactate transporter, monocarboxylate transporter 1, and lactate uptake has been shown by McCullagh et al. (30). Regarding possible differences in lactate production, it is unlikely in this study to be due to differences in fiber type because the African athletes had a higher type II fiber percentage than did the Caucasian runners. However, a lower lactate production has previously been shown in specific populations born and residing at altitude (22, 36). Although the athletes in the present study were all residing at sea level, Hochachka et al. (20) have hypothesized that the metabolic adaptations resulting in lower lactate production during exercise may be an evolutionary trait that is conserved in some populations.

The tendency toward a higher activity of the glycolytic enzyme PFK in the African runners (28%, P = 0.09) is interesting, although the mean values in both groups are similar to those in a recent large study of normal individuals who had a large variation in physical activity (41). Endurance athletes usually have muscle homogenate PFK activities that are similar to, or even lower than, those in sedentary subjects, as do the endurance athletes in the present study (16, 25). However, the findings of Essen-Gustavsson and Henriksson (12) showed that, when enzyme activity is measured in pooled dissected single fibers, PFK activity can be elevated in both fiber types as a result of training. It is possible that both glycolytic and oxidative pathways are very important, specifically during sustained high-intensity exercise, such as that performed in the present study during the time-to-fatigue test. We speculate that with both a high glycolytic and oxidative capacity, skeletal muscle may be both able to supply energy via the more rapid pathway of glycolysis and to rapidly oxidize the by-product, therefore resulting in lesser perturbation of cellular homeostasis and the ability to sustain higher intensity activity.

Because the difference in oxidative enzyme capacity between the two groups in the present study is sizeable and largely confirms the data of the Kenyan study (39), one might be tempted to conclude that it is sufficient to
mechanistically explain the major observation of this study, namely, the enhanced fatigue resistance in the African runners. To what extent the enhanced fatigue resistance found in our study was related to mechanisms other than increased skeletal muscle oxidative enzyme capacity cannot be speculated from the present data. Other possible factors that may have affected time to fatigue include differences in fiber-type recruitment, capillarity, potassium accumulation, greater economy at high running speeds, and/or psychological factors.

Nevertheless, the importance of the present study is that it unequivocally confirms superior fatigue resistance in African distance runners during a sustained running task and suggests several potential mechanisms that may explain up to one-half the variance in fatigue resistance in these athletes during high-intensity endurance exercise. To the best of our knowledge, the present study is the first to directly show an association between oxidative enzyme activity and fatigue resistance during a high-intensity submaximal running task in well-trained human subjects. The present study indicates an unusually large difference in skeletal muscle oxidative capacity between well-trained African and Caucasian runners and suggests that this characteristic may be population specific. Although not fully understood at present, this may play a role in the superior performance of African distance runners at the elite level. The negative correlations between CS activity and both plasma lactate concentration and RER measured during high-intensity but submaximal exercise go some way in elucidating the metabolic consequences of the enhanced oxidative capacity.

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