A COMPARATIVE STUDY OF ACUTE RESPONSES TO RUNNING IN ELITE BLACK AND WHITE MARATHON ATHLETES

BY

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ABSTRACT

Experienced male marathon runners, 9 black and 10 white, with marathon times of 2 hours 45 minutes or faster, acted as subjects for the study, the purpose of which was to determine whether black runners are better suited to marathon running than whites. Body composition was determined by anthropometry. Maximal oxygen uptake ($\dot{V}O_2$ max) and other physiological variables were measured during a continuous, speed-incremented treadmill protocol using a computer-aided data acquisition system. Subjects also ran a simulated marathon at 92.5% of the running speed at which the ventilatory threshold (VT) occurred. Physiological, gait and RPE variables were measured at 10 minute intervals during the marathon. Major findings are detailed below:-

The $\dot{V}O_2$ max averaged 60.4 $\pm$ 6.5 and 63.2 $\pm$ 2.9 $ml.kg^{-1}.min^{-1}$ in the black and white runners respectively and was highly correlated with best marathon race time ($r = 0.86$ and 0.85 respectively) and VT ($r = 0.84$ and 0.60 respectively) ($p < 0.05$). No significant differences existed between the groups in submaximal oxygen uptake ($\dot{V}O_2$) or % $\dot{V}O_2$ max utilised at 16 $km.hr^{-1}$, but the estimated % $\dot{V}O_2$ max utilised during a marathon race was higher in the black (89.0 $\pm$ 5.5%) than the white runners (81.5 $\pm$ 3.1%) ($p < 0.05$). The % $\dot{V}O_2$ max utilised at 16 $km.hr^{-1}$ (84.8 $\pm$ 9.1 and 78.6 $\pm$ 5.8% in the black and white runners respectively) was significantly correlated with the % $\dot{V}O_2$ max utilised while racing in the white (81.5 $\pm$ 3.1%) ($r = 0.70$) ($p < 0.05$), but not the black runners (89.0 $\pm$ 5.5%). The VT occurred at 82.7 $\pm$ 7.7 and 75.6 $\pm$ 6.2% $\dot{V}O_2$ max in the black and white groups respectively ($p < 0.05$). Post-marathon blood lactic acid levels were lower in the black (1.30 $\pm$ 0.26 $mmol.l^{-1}$) than the white runners (1.59 $\pm$ 0.20 $mmol.l^{-1}$). The respiratory exchange ratio (R) was higher in the blacks than whites when running at 16 $km.hr^{-1}$ (1.03 $\pm$ 0.07 and 0.98 $\pm$ 0.03 respectively) and during the marathon ($p < 0.05$). There was no significant difference in pulmonary minute ventilation ($\dot{V}_I$) between the groups, but breathing frequency (f) was higher

in the black (59 $\pm$ 12 breaths.min^{-1}) than the white runners (45 $\pm$ 8 breaths.min^{-1}) and tidal volume ($\bar{V}_T$) lower in the black (1.33 $\pm$ 0.16 l.breath^{-1}) than the white runners (1.75 $\pm$ 0.36 l.breath^{-1}) during submaximal running at 16 km. hr^{-1} ($p < 0.05$). The same trend was observed during the marathon run. During the time-course of the marathon f increased and $\bar{V}_T$ decreased in both groups ($p < 0.05$). Stroke volume decreased and heart rate increased in both groups during the time-course of the marathon ($p < 0.05$). Cardiac output was therefore maintained. Thermal responses were similar in the two groups. A significant increase in rectal temperature coincided with a decrease in skin temperature and may have been related to an increase in f ($r = 0.86$ and 0.67 in the blacks and whites respectively), H/R ($r = 0.70$ and 0.67 respectively) and "local" (leg) RPE ($r = 0.84$ and 0.82 respectively).

It was concluded that black runners were able to run marathon races at a higher % $\dot{V}O_2$ more than whites due to the blacks having lower blood lactic acid levels when running at a similar % $\dot{V}O_2$ max. Given similar maximal oxygen uptakes,this would enable blacks to run faster. Cardiopulmonary adjustments occur during the time-course of a marathon which maintains Q and $\dot{V}_I$.

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TABLE OF CONTENTS

LIST OF FIGURES

Page

LIST OF TABLES

CHAPTER 1

INTRODUCTION

It is a well known fact among marathon runners in South Africa that black athletes tend to perform better than their white counterparts in marathon races. This is borne out by the results of the South African marathon championships (Le Roux 1985). The race entry has, in the last five years, consisted of approximately 50% black athletes and 50% white. This alone may indicate superior running on the part of the black marathoners as in order to run in the championship event it is necessary to qualify by running faster than a set standard in a marathon prior to the championship. It should be noted, however, that only registered runners can compete and that the registered running population in South Africa consists of only 20% black athletes (Le Roux 1984). The 50% black entry in the championships thus indicates that proportionately more black runners are achieving the qualifying standard than white runners. When the championships results are scrutinised, it is found that of the first twenty finishers approximately 80% are black runners. Further, in the last seven years a black runner has won the event on six occasions and it is a non-white runner who is the holder of the South African marathon record of 2 hours 8 minutes 58 seconds. These statistics suggest that the performance of black marathon runners is superior to that of white marathon runners.

The possibility thus exists of South African blacks being better suited than whites for the marathon with respect to one or more of the factors that determine success in this event.

Bosch and Goslin (1984) recently presented data which revealed that differences may exist in some of the cardiopulmonary responses between faster and slower marathon runners during the running of a simulated marathon on the treadmill.

By chance the faster runners taking part in the study, with a marathon time of under 2 hours 45 minutes, were black athletes while the slower runners, with a time of over 3 hours for the marathon, were white. This raised the question of whether the differences observed between faster and slower runners were in fact entirely fast/slow differences. It is possible that cardiopulmonary differences observed were, to an unknown extent, due to differences that may exist between black and white runners in these responses when running a marathon.

For the reasons outlined above it would be of interest to determine if some of the factors that can affect running performance differ between black and white marathon runners.

Any differences that may exist in these factors could be due to the genetically determined physiological endowment of the runners and thus a difference in physiological responses to severe, prolonged activity. These physiological determinants of success in competitive marathon running include a high maximal oxygen uptake ($\dot{V}O_2$ max) which appears to be a prerequisite for success in this event (Costill 1967, Conley and Krahenbuhl 1980). Amongst runners possessing this attribute, other factors become important in determining a winning performance from within the group. These include the fractional utilisation of $\dot{V}O_2$ max (% $\dot{V}O_2$ max) (Costill et al 1973b), running economy (Daniels 1974, Conley and Krahenbuhl 1980), muscle and blood lactic acid accumulation (Farrell et al 1979) which is probably related to training methods (MacDougall 1977) and muscle fibre composition (Costill et al 1976a). Two major factors that may limit prolonged, strenuous exercise are muscle glycogen depletion and development of hypoglycaemia (Ivy et al 1983, Miller et al 1983). Pugh et al (1967) have suggested that the capacity to withstand a high body temperature is also a requisite of successful marathon running. Other factors that may be associated with success in distance

running include the biomechanical characteristics of the runners' gait (Cavanagh et al 1977), body composition (Pollock et al 1977), and socio-economic and nutritional status (Phillips 1976, Shephard 1980).

Investigations which have examined these and other variables in athletes of differing ability suggest that the elite marathoner differs from the non-elite in some of the factors that are known to be determinants of successful marathon running. It is possible that any differences in these same factors betweeen black and white marathon runners may partly explain the apparent superior performance of black compared to white marathon runners in South Africa. This possibility is supported by some documented differences between black and white athletes. Anatomical differences have been found to exist between elite black and white runners (De Garay et al 1974). Pulmonary minute ventilation ($\dot{V}_I$) and the ventilatory equivalent for oxygen ($\dot{V}_I/\dot{V}O_2$) ($VE\text{-}0_2$) have been found to be higher in American blacks than whites during cycle ergometry (Cerny et al 1982). In the same study it was also found that the black subjects had a higher ventilatory frequency (f) and lower tidal volume ($\bar{V}_T$) for any given $\dot{V}_I$. A study conducted by Leary and Wyndham (1965) revealed that no differences existed in $\dot{V}0_2$ max between South African black and white middle distance athletes, but that at any given oxygen uptake the black athletes could exercise at a higher power output than the whites. It may thus be possible to explain the marathon performance heterogeneity of black and white marathon runners in terms of one or more of the factors that are known to be important for successful marathon running.

Statement of the Problem

It is unknown to what extent black runners may be different physiologically, biomechanically and/or psychologically compared to white marathon runners.

This study aims at the determination of any differences that may exist between elite black and white marathoners within the above mentioned domains. An elite marathon runner for the purposes of this study refers to a runner who has completed a marathon in 2 hours 45 minutes or faster. Any differences between black and white runners within these domains will be determined, as will any change that may occur during the time-course of a simulated marathon on the treadmill.

Research Hypotheses

1. There are no differences between elite black and white marathon runners within the physiological, biomechanical and psychological variables being examined during running.

2. There are no changes in the variables being investigated during the time-course of a simulated marathon.

Statistical (Ho) and Alternative (HA) Hypotheses

1. Ho: $\mu_B = \mu_W$

   HA: $\mu_B \neq \mu_W$

   Where: μ_B and μ_W are the means of a number of physiological, gait and Rating of Perceived Exertion (RPE) variables as measured in black and white marathon runners respectively. These variables were measured during a progressive $\dot{V}O_2$ max test and a simulated marathon on the treadmill. The data from the $\dot{V}O_2$ max tests were analysed at 16 $km.hr^{-1}$ and $\dot{V}O_2$ max, while the data from the simulated marathons were analysed at 10% intervals from 10 to 100% of total running time. The submaximal speed of 16 $km.hr^{-1}$ was chosen for comparisons between the two groups as various physiological variables have most often been reported at this speed and it was close to the average race speed of the subjects.

2. Ho: $\mu_{10} = \mu_{20} = \ldots = \mu_{100}$

   HA: $\mu_{10} \neq \mu_{20} \neq \ldots \neq \mu_{100}$

   Where: μ_{10} to μ_{100} are the means of a number of physiological, gait and RPE variables from 10 to 100% of total running time during a simulated marathon.

3. Ho: $\mu_{B\Delta bl} = \mu_{W\Delta bl}$

   HA: $\mu_{B\Delta bl} \neq \mu_{W\Delta bl}$

   Where: $\mu_{B\Delta bl}$ and $\mu_{W\Delta bl}$ are the differences between the pre-to post-marathon blood glucose and lactic acid concentrations of black and white runners respectively.

4. Ho: $\mu_{B\ comp} = \mu_{W\ comp}$

   HA: $\mu_{B\ comp} \neq \mu_{W\ comp}$

   Where: $\mu_{B\ comp}$ and $\mu_{W\ comp}$ are body compositional factors of black and white runners respectively.

Delimitations

The problem posed was investigated by examination of a number of cardiopulmonary and gait variables, as well as blood glucose, blood lactic acid, thermal, and RPE responses of elite marathon runners.

These variables were examined in both black and white sub 2 hour 45 minute marathon runners during a speed-incremented $\dot{V}0_2$ max test as well as during a simulated marathon on the treadmill. The study investigated any difference that may exist between black and white marathon runners in any of these variables and any changes during the time-course of the marathon.

The runners were selected on availability, provided that they had a best marathon time of 2 hours 45 minutes or faster. This was chosen as it represents a well above average performance, being close to the qualifying time for the South African marathon championships (2 hours 35 minutes). The $\dot{V}0_2$ max of each subject was determined during a $\dot{V}0_2$ max test on the treadmill. After a recovery period of approximately 4 hours each subject then ran on the treadmill for the equivalent of the marathon distance (42.2 km) at a speed close to the ventilatory threshold (92.5% of the speed at which the ventilatory threshold occurred).

Limitations

In order to obtain constant, standardised conditions and because of the monitoring equipment needed, the marathon was run on the treadmill. This, however, only approximates the real-life situation.

The problem posed originated from differences in marathon performance of the 20 fastest black and white marathon runners in the country, but it was impossible to test these runners because of travelling expenses and availability. For this reason, runners not quite of this calibre but who could still be classified as elite served as subjects in the study. These runners were resident in the Eastern Cape and Border regions.

It should be noted that the runners were not peaked at the time of testing as they would be for a marathon. Also, associated with this, they did not taper their training as much before treadmill testing as before a marathon. This, however, applied to both groups tested. Further, some subjects glycogen loaded for a few days before testing while others did not and the content of replacement drinks during the marathon varied, as did the diet and pre-

marathon meal of the subjects. These were the normal procedures of each subject before and during a marathon race.

Problems in contacting and communicating with potential black subjects led to some runners being tested only a week after running a race. This could have influenced results obtained, although a study by Noble et al (1979) in which cardiopulmonary and perceptual recovery from a marathon were investigated, revealed that at one week post-marathon only RPE was significantly different from the pre-marathon testing. These tests were conducted over a period of 30 minutes and not the entire marathon distance.

A Borg RPE Scale translated into Xhosa was used to determine RPE of the black subjects. This scale has, however, not been validated against the original Borg RPE Scale. It is possible that the wording is not an accurate reflection of the English equivalent due to possible differences in perception by different cultural groups, although translation was by staff members of the African Languages Department of Rhodes University who attempted to account for this problem. As the subjects had to travel from neighbouring towns to the laboratory, it was not possible to habituate them to the treadmill on the days prior to testing and it was noticed that the black subjects had difficulty in adapting to treadmill running. The extent to which fear of the technology may have influenced results is unknown, especially in the case of the black subjects who were less acquainted with modern technology than the whites.

The ventilatory threshold (VT) was used as the baseline from which the treadmill speed for the simulated marathon was set. In the case of some black subjects (who were tested after testing of the white group was already completed) it was difficult to accurately determine the VT. With these subjects the speed was adjusted so that the marathon was run

at approximately 35-40 seconds.km^{-1} slower than the speed during the most recent marathon run by the subject. This was used as it was found that the white marathoners (who ran at a speed based on the VT) were running at a speed which, on average, fell within these limits.

CHAPTER 2

SURVEY OF RELATED LITERATURE

A thorough search of the literature dealing with distance running revealed that very few studies have addressed the problem of acute and chronic responses of black athletes to long distance running. This is particularly so as regards physiological and psychological responses.

As was pointed out in the previous Chapter, there appears to be a discrepancy in the performance of black and white marathon runners,suggesting that blacks may be better suited to marathon running than whites. The underlying cause of any difference in performance of the two groups may have a physiological, biomechanical or psychological basis, which may reflect the differences known to exist in these domains between international class, elite and non-elite marathon runners. This survey of the various studies conducted on distance runners will therefore centre on what constitutes the difference in performance ability of these groups of athletes. For the purposes of this study an international class runner is any athlete who has a marathon time in the region of 2 hours 15 minutes and an elite runner any athlete with a time of approximately 2 hours 45 minutes or better.

Studies have disclosed that genetic differences account almost entirely for inter-individual variation in $\dot{V}O_2$ max (Klissouras 1973a), an important determinant of success in marathon running (Conley and Krahenbuhl 1980). Evidence has indicated that as much as 93% of the variability in $\dot{V}O_2$ max can be attributed to genetic influences (La Fontaine 1981). Extragenetic influences can, however, alter the $\dot{V}O_2$ max, but only up to a limit predetermined by the genotype (Klissouras 1971). In addition, it has been found that the genetic factor is also the principal determinant of the variability in physical fitness and that chronic exercise can affect the

expression of this genetic potential (Klissouras 1973b). As with the $\dot{V}O_2$ max, however, this can only occur within the fixed limits of the genotype. Even within a population, $\dot{V}O_2$ max exhibits considerable inter-individual variation, heredity probably exerting the most determinant influence (Lortie et al 1981). These authors have reported a significantly larger inter- than intra-family variation in $\dot{V}O_2$ max. Their results suggest that sexual chromosomes contribute little to $\dot{V}O_2$ max inheritance and that autosomes are probably responsible for most genetic variation. In a study conducted with monozygotic twins as subjects (Prudhomme et al 1983), it was found that the group as a whole had a significantly higher $\dot{V}O_2$ max after a training programme. Although there were considerable inter-individual differences in training gains, members of the same twin pair yielded approximately the same response to training. These results indicate that the genotype plays a significant role in the adaptive capacity of the $\dot{V}O_2$ max to training. Although $\dot{V}O_2$ max may be genotype dependent, so may other variables involved in running.

It should be pointed out that results concerning variance in $\dot{V}O_2$ max inheritance are conflicting. Klissouras (1971) reported that 93% of the variance in $\dot{V}O_2$ max was inherited, a statement based on data from studies on mono- and dizygotic twin pairs. Shephard (1980) reports, however, that a 47% difference in $\dot{V}O_2$ max has been found between two monozygotic twins. An estimate of the degree to which $\dot{V}O_2$ max is inheritable is thus unreliable to the point of having little scientific value. The large difference reported may be attributable to environmental and/or methodological variances.

Genetically determined differences in $\dot{V}O_2$ max have also been investigated by comparing distinctive ethnic groups living in the same environment. African tribes sometimes differ in average body mass and $\dot{V}O_2$ max while living in their homelands (Wyndham 1973). Wyndham et al (1966) and Wyndham (1973) examined this, finding that these different African tribes, when living under

the uniform conditions of mining compounds, showed no inter-tribal differences in $\dot{V}O_2$ max. This suggests that the observed differences may not be due to the genome but rather influences such as diet and/or activity level. A similar study conducted by Glick and Shvartz (1974), however, resulted in a different conclusion being drawn. They compared the $\dot{V}O_2$ max of Jewish settlers of European, Iraqi, North African and Yemenite origin and found that the Yemenites were significantly superior to the other three groups in terms of $\dot{V}O_2$ max. Differences in activity and diet being responsible were ruled out and it was concluded that the observed differences in $\dot{V}O_2$ max were largely a result of genetic variance. It is therefore uncertain to what extent genetic influences and environmental factors are implicated in the variation in $\dot{V}O_2$ max observed in different populations. Considering athletic groups, a study conducted by Leary and Wyndham (1965) revealed that no difference existed in $\dot{V}O_2$ max between South African black and white middle distance athletes. An analysis by Shephard (1980) attributed 27% of the high $\dot{V}O_2$ max observed in cross-country skiers to prolonged athletic training, 26% to normal population differences in habitual activity and 47% to genetic factors.

As stated previously, $\dot{V}O_2$ max is an important determinant of success in the marathon and elite and international class runners are characterised by a high $\dot{V}O_2$ max (Costill 1967, Conley and Krahenbuhl 1980). Within such a group, however, a wide range of values have been reported. Pollock (1977a) observed $\dot{V}O_2$ max values ranging from 71.3 - 84.4 $ml.kg^{-1}.min^{-1}$ in a group of international class athletes with similar performances. The lowest value (71.3 $ml.kg^{-1}.min^{-1}$) was that of the 1972 Olympic marathon champion (Pollock 1977a), while the $\dot{V}O_2$ max of the holder of the world best marathon time until 1981 and still ranked in the top ten on the world all-time list, has been reported at 69.7 $ml.kg^{-1}.min^{-1}$ (Costill et al 1971a). Although a $\dot{V}O_2$ max in the region of 70 $ml.kg^{-1}.min^{-1}$ will place an athlete in the ranks of the internationalists, other factors play a role to differentiate between athletes

possessing this essential capacity (Costill et al 1973b). Some of these factors include the fractional utilisation of the $\dot{V}O_2$ max (% $\dot{V}O_2$ max), running economy (submaximal oxygen uptake), peak muscle and blood lactic acid accumulation, and muscle fibre composition (Conley and Krahenbuhl 1980). While it has been found that a high correlation (r = 0.91) existed between $\dot{V}O_2$ max and success in running a 15 km race in a cross-section of elite and non-elite runners (Costill et al 1973b), this is in contrast to other observations on marathon runners where a low correlation (r = 0.08) was found to exist within a group of international class runners between $\dot{V}O_2$ max and performance (Costill 1967). Therefore, within international and elite groups, $\dot{V}O_2$ max alone does not discriminate between performers. The high $\dot{V}O_2$ max of elite and international class endurance athletes has been thought to be related to the oxygen transport and delivery systems of these athletes. Stuart and Collings (1959) and Bock (1963) report a larger vital capacity (VC) in athletes than in non-athletes of similar stature, but this was not found to be the case by Kaufmann et al (1974) or Costill (1967). Cerny et al (1982) reported a lower VC in American blacks than whites during cycle ergometry and Kaufmann et al (1974) observed that athletes had larger than normal maximum voluntary ventilation (MVV) and forced expiratory volume (FEV). Costill (1967) found that MVV per unit body surface area was positively related with distance running performance and suggested that faster runners may possess superior respiratory musculature and/or reduced pulmonary resistance, which might enable them to carry on external respiration during exercise at a reduced energy cost. Cerny et al (1982) determined that American blacks have a higher ventilatory equivalent for oxygen during cycle ergometry than whites and suggested increased lung elastic recoil, or breathing higher on the pressure/volume curve as possible reasons. Raven (1977) found no significant differences between international class runners, elite runners and sedentary controls when examining forced vital capacity (FVC), total lung capacity (TLC), residual volume (RV),

RV/TLC ratio, and FEV/FVC ratio. He suggested that at the level of the international class athlete, other factors, such as pulmonary diffusing capacity, pulmonary capillary blood volume and membrane permeability become more important than pulmonary capacity alone. In support of this are findings by Kaufmann et al (1974) and Bannister et al (1960) who report a greater than normal resting diffusing capacity in runners. Holmgren and Åstrand (1966) are also of the opinion that the respiratory system does not limit the $\dot{V}O_2$ max, but that the delivery of oxygen by the cardiovascular system may be the limiting factor. While the pulmonary minute ventilation ($\dot{V}_E$) is not a perfect correlate of oxygen uptake, runners appear to adjust running speed to permit a tolerable level of respiratory distress (Costill 1979). During a simulated cross-country run, Kollias et al (1967) observed a $\dot{V}_E$ of 150 $l.min^{-1}$ and Costill (1979) reports that during a treadmill-simulated 10 000 m run, the $\dot{V}_E$ was between 120-145 $l.min^{-1}$. It was estimated that 9% of the total energy expenditure was utilised by the ventilatory musculature. Although no values were reported, Pollock (1977b) found a lower $\dot{V}_E$ at any given submaximal speed in international class compared to elite athletes. As stated previously, Cerny et al (1982) found the $\dot{V}_E$ to be higher in American blacks than whites during cycle ergometry. Costill (1979), however, does not consider $\dot{V}_E$ a limiting factor in long distance running.

A high $\dot{V}O_2$ max appears to be closely related to the mean number of capillaries per muscle fibre. These are increased with training, the increase being closely related to the activity level of the muscle. A smaller diffusion distance in the tissue may be of importance, as the level of oxygenation of the muscle cell will then be kept high.(Saltin et al 1977). The concentrations of myoglobin and oxidative enzymes would also be higher (Saltin and Rowell 1980). These authors are of the opinion that while $\dot{V}O_2$ max is limited by the transport of oxygen to the working muscle, muscle blood flow, and muscle capillary density, the capacity for submaximal work depends on the

oxidative capacity of the muscle.

The ability to utilise a large fraction of the $\dot{V}O_2$ max (% $\dot{V}O_2$ max) during competition appears to be an important factor in determining a winning performance from within a group of runners who all have a high $\dot{V}O_2$ max (Costill 1972). The average % $\dot{V}O_2$ max employed during a marathon race has been reported to be 75% (Costill and Fox 1969), but amongst trained runners values may vary from 64-90% (Costill 1972). It is thus possible for two runners to have the same $\dot{V}O_2$ max but to differ in the % $\dot{V}O_2$ max that can be utilised during a race. This may be one reason why the marathoner cited previously as having a $\dot{V}O_2$ max of "only" 69.7 $ml.kg^{-1}.min^{-1}$ could run a world record time, for although he had a low $\dot{V}O_2$ max for an international class runner, he was found to utilise a high % $\dot{V}O_2$ max (86%) when running at the speed required to set his world best time, with blood lactic acid accumulation at this speed being only 2.4 $mmol.l^{-1}$ (21.4 mg%) (Costill et al 1971a). Other runners, however, with a greater $\dot{V}O_2$ max can sometimes tolerate running speeds which employ only 64% $\dot{V}O_2$ max for the duration of a marathon (Costill 1970).

The submaximal oxygen uptake ($\dot{V}O_2$) at a given running speed (running economy) appears to vary amongst runners (Daniels 1974). It is thus possible for two runners with an identical $\dot{V}O_2$ max to be running at different percentages of that $\dot{V}O_2$ max when running at the same speed, with the more economical runner at a lower percentage. If both were to run at the same % $\dot{V}O_2$ max, the more economical athlete would be running faster than the less economical (Costill 1972). Results of a study by Leary and Wyndham (1965) showed that at any given $\dot{V}O_2$, black athletes could exercise at a higher power output than a group of white athletes. The importance of a low submaximal $\dot{V}O_2$ is demonstrated by the two previously mentioned world class marathon runners, these being an Olympic marathon winner and a previous

world record holder in the marathon. Both these athletes had a relatively low $\dot{V}O_2$ max compared to many elite and international class athletes at 71 $ml.kg^{-1}.min^{-1}$ (Pollock 1977a) and 69.7 $ml.kg^{-1}.min^{-1}$ (Costill et al 1971a) respectively. Both these athletes, however, were found to be extremely economical at submaximal running speeds. The Olympic marathon champion had an average $\dot{V}O_2$ of 57 $ml.kg^{-1}.min^{-1}$ at 20 $km.hr^{-1}$ and at the same speed the $\dot{V}O_2$ of the previous record holder was 59 $ml.kg^{-1}.min^{-1}$, with a heart rate of 167 $b.min^{-1}$ (Costill et al 1971a). At 16 $km.hr^{-1}$ international class runners have been shown to have a lower $\dot{V}O_2$ than elite runners (Pollock 1977b). At 20 $km.hr^{-1}$, the $\dot{V}O_2$ was near the $\dot{V}O_2$ max for several of the elite runners studies by Pollock (1977b). The mean $\dot{V}O_2$ max of the group of international runners studied was 74.1 ± 2.6 $ml.kg^{-1}.min^{-1}$ and of the elite runners 69.2 ± 3.7 $ml.kg^{-1}.min^{-1}$. At 16 $km.hr^{-1}$ the international runners had a $\dot{V}O_2$ which was lower than that of the elite runners, but at 20 $km.hr^{-1}$ the $\dot{V}O_2$ was 65 $ml.kg^{-1}.min^{-1}$ in both groups. When expressed as % $\dot{V}O_2$ max, the international class runners were utilising 71.5% $\dot{V}O_2$ max at 16 $km.hr^{-1}$, significantly lower than the elite runners at 80.9%. At 20 $km.hr^{-1}$ the international class runners were at 87.7% $\dot{V}O_2$ max, while the elite runners were at 93.9% $\dot{V}O_2$ max. (Pollock 1977b). Whereas $\dot{V}O_2$ at 20 $km.hr^{-1}$ did not discriminate between the groups, the % $\dot{V}O_2$ max utilised did. The % $\dot{V}O_2$ max that can be utilised by an athlete during a marathon may be determined by the point at which muscle and blood lactic acid begins to accumulate exponentially. This is indicated from research which has shown that a marathon can be run at a speed corresponding to the point of blood lactic acid accumulation, often termed the "anaerobic threshold". (Farrell et al 1979). The % $\dot{V}O_2$ max at which this occurs can be increased with training (MacDougall 1977).

Numerous mechanisms have been postulated for the "anaerobic threshold" (AT), a term proposed by Wasserman and McIlroy because they thought the sudden

accumulation of blood lactic acid was due to an oxygen deficiency within the muscle (Wasserman and McIlroy 1964). Support is provided for this from experimental work by Vogel and Gleser (1972) who demonstrated an increase in blood lactic acid concentration at any particular power output and a lower "AT" when the oxygen content of the blood was experimentally reduced. This mechanism, however, is considered incorrect (Brooks 1985a).

A second possible mechanism is that although oxygen delivery to the muscle may be adequate, a point is reached where the oxidative capacity of the muscle is exceeded. This point results in the observed "AT". The term "onset of blood lactic acid/blood lactate accumulation" (OBLA). may thus be better than AT, as these do not imply that skeletal muscle is anaerobic at submaximal exercise (Brooks 1985a). Training causes an increase in the oxidative enzymes as well as the number and size of the mitochondria, which may correspond with the increase in OBLA after training (Davis 1985).

Sjödin et al (1979) suggest that endurance training may shift the lactic acid dehydrogenase (LDH) isozyme patterns of the slow-twitch (ST) fibres towards that of LDH-1, the highly aerobic isozyme. This might decrease the rate of lactic acid production and accumulation at submaximal power outputs. According to the results of Rusko and Rahkila (1979) the OBLA correlated only slightly with the oxidative enzyme activities, and no significant correlations were observed between OBLA, the percentage of ST fibres and LDH activity.

A third postulated mechanism involves the recruitment pattern of muscle fibres. At higher power outputs the fast-twitch (FT) fibres are increasingly recruited and as these fibres are highly glycolytic, a progressive increase in lactic acid production occurs (Davis 1985). This is supported by the suggestion that the point at which lactic acid accumulation occurs is related to the percentage of FT muscle fibres in the contracting muscles (Tesch et al 1978)

as lactic acid formation in these motor units will affect both mixed muscle tissue lactic acid and blood lactic acid concentrations (Tesch 1980). Thus a higher percentage of ST muscle fibres may result in a lower lactic acid accumulation at a given speed (Essén and Häggmark 1978). It was shown by Sjödin et al (1979) that the % $\dot{V}O_2$ max corresponding to a blood lactic acid concentration of 4 mmoles.l^{-1} was positively related to the percentage ST fibres. This is contrary to the results of Rusko and Rahkila (1979).

The most recent mechanism postulated is that of Brooks and Fahey (1984) who have suggested a reduced rate of lactic acid clearance relative to production being the cause of the increase in blood lactic acid, possibly due to an inability of the muscle to extract and oxidise lactic acid at a sufficiently high rate. Even when at rest lactic acid is produced. The blood lactic acid concentration remains low and invariate, however, because production is balanced by removal (Brooks 1985a). A direct, linear relationship exists between oxygen uptake and lactic acid turnover both in dogs (Issekutz et al 1976) and in humans (Hubbard 1973). Further experimental support comes from the work of Connett et al (1984) who demonstrated that in the dog gracilis muscle lactic acid is produced even under fully aerobic conditions. Their results also suggested that lactic acid production is linearly related to the power output of the muscle. Thus the term "anaerobic threshold" is not metabolically correct. The rate of lactic acid oxidation is directly related to the $\dot{V}O_2$ up to and including hard exercise. Although both production and clearance of lactic acid increase linearly with increasing exercise intensity, the increase in clearance lags behind the increase in production, resulting in lactic acid accumulation in the blood. (Brooks 1985a).

There appear to be three clearly defined phases in blood lactic acid levels during the transition from low to maximum intensity exercise. These are identified by breakpoints in various physiological variables that differentiate

the first from the second phase, and second to third phases. The first transition is the "aerobic threshold", characterised by a gradual increase in the fraction of expired oxygen (F_EO_2) and non-linear increases in pulmonary minute ventilaton ($\dot{V}_E$) and CO_2 production ($\dot{V}CO_2$) (Skinner and McLellan 1980), as well as an increase in the ventilatory equivalent for oxygen ($\dot{V}_E/\dot{V}O_2$) ($VE-O_2$)with little or no change in the ventilatory equivalent for CO_2 ($\dot{V}_E/\dot{V}CO_2$) ($VE-CO_2$) (Dawson and Pyke 1984). The second transition from phase two to phase three is the "AT" (OBLA), characterised by a further sharp increase in blood lactic acid concentration, a sharp decrease in the fraction of expired carbon dioxide (F_ECO_2), a second break in $\dot{V}_E$ (Skinner and McLellan 1980) and further sharp increases in F_EO_2, $VE-O_2$ and $VE-CO_2$ (Dawson and Pyke 1984). The $VE-O_2$ and $VE-CO_2$ are the best ventilatory indicators of both the "aerobic threshold" and OBLA especially where the increment in power output is rapid (approximately one minute). This may be better termed the ventilatory threshold when measured by ventilatory variables. When power output increases are rapid the $VE-CO_2$ does not increase at the "aerobic threshold" but remains stable with further increases in power output due to isocapnic buffering of lactic acid. Thus, the criterion of a systematic increase in $VE-O_2$ without a concomitant increase in $VE-CO_2$ is the most specific gas exchange method for detecting the "aerobic threshold". After further increases in power output, the $VE-C0_2$ then also increases, together with a further increase in $VE-O_2$ (Davis 1985). This is the OBLA. Whereas the "aerobic threshold" occurs at approximately 40-60% $\dot{V}O_2$ max and a blood lactic acid concentration of approximately 2 $mmol.l^{-1}$ (18 mg%), the OBLA occurs at approximately 65-90% $\dot{V}O_2$ max and a blood lactic acid concentration of approximately 4 $mmol.l^{-1}$ (36 mg%) (Skinner and McLellan 1980).

Green et al (1983) observed that muscle lactic acid concentration increases before the lactic acid (lactate) threshold identified by either blood analysis or

ventilatory variables. It is usually the latter indicators that are used to determine the OBLA. While some authors have suggested that the correlation between the ventilatory threshold (VT) and OBLA is high (Caiozzo et al 1982, Davis et al 1976) the results of other investigations indicate that the correlation between the VT and the OBLA may be coincidental (Poole and Gaesser 1984). These authors observed a smaller increase in the VT than the OBLA after training and were of the opinion that this showed a separation between the two thresholds. Hughes et al (1982) and Segal and Brooks (1979) also showed an uncoupling of the VT and the OBLA. They found that when subjects were glycogen depleted, the OBLA occurred at a higher power output and % $\dot{V}O_2$ max, but the VT at a lower % $\dot{V}O_2$ max than when subjects were not glycogen depleted. The intervention to lower glycogen stores in the study was prolonged exercise to exhaustion (Segal and Brooks 1979), which can lead to a chronic reduction in PCO_2 (Heigenhauser et al 1983). Such a reduction in PCO_2 could result in an increase in ventilation if $\dot{V}CO_2$ is held constant because of the ventilation equation ($\dot{V}_A = \dot{V}CO_2.K/PCO_2$) Davis 1985). Hagberg et al (1982) demonstrated an uncoupling of the VT and the OBLA in patients suffering from McArdles syndrome. The investigators hypothesised that because these patients do not produce lactic acid during exercise due to a deficiency of the muscle enzyme phosphorylase, they should not demonstrate the ventilatory changes associated with the OBLA. These patients, however, showed the normal VT at a power output which elicited approximately 70% $\dot{V}O_2$ max even though blood lactic acid did not increase. This led Hagberg et al (1982) to suggest that changes in ventilatory variables are independent of increases in blood lactic acid. It should be pointed out, however, that in the patients there was no region of isocapnic buffering, which should be present if the ventilatory variables of $VE-O_2$ and $VE-CO_2$ for the detection of the VT/OBLA are being correctly applied. Thus, McArdles syndrome patients do not in fact show a VT according to the criteria previously stated. It therefore appears that the

arguments against the VT and the OBLA coinciding due to an increase in blood lactic acid levels may be questionable.

Although controversy exists as to the reason for the non-linear increase in blood lactic acid and whether or not the VT and the OBLA are measuring the identical metabolic occurrences, the fact remains that the correlation between VT and marathon running performance is high (Farrell et al 1979). These authors showed that the treadmill velocity corresponding to the ventilatory threshold yielded a correlation of 0.98 with marathon race speed, with marathon speed being an average of 8 $m.min^{-1}$ higher than the treadmill velocity at which the VT occurred. Similar results have been obtained by Tanaka and Matsuura (1984).

A high correlation has also been shown to exist between oxygen uptake ($\dot{V}O_2$) at a given submaximal running speed and the treadmill velocity corresponding to the OBLA. Subjects with a higher $\dot{V}O_2$ at a given submaximal speed (i.e: poor running economy) had a lower running speed corresponding to the OBLA (Sjödin et al 1979). It was demonstrated by these authors that when the $\dot{V}O_2$ at a given submaximal speed was expressed as % $\dot{V}O_2$ max, a higher correlation existed with the treadmill velocity at OBLA than obtained with $\dot{V}O_2$ alone. Runners with a higher velocity at the OBLA utilised a lower % $\dot{V}O_2$ max at any given submaximal running speed. It was therefore concluded that a lower velocity at the OBLA occurs in conjunction with a poor running economy (Sjödin et al 1979). A possible explanation for this is that muscle contractibility and performance can become impaired when even a small accumulation of lactic acid occurs (Tesch et al 1978, Tesch 1980).

A possible mechanism to explain how an accumulation of lactic acid could result in impaired muscle function and force a rise in $\dot{V}O_2$ involves the effects of acidosis on muscle. A reduction in pH has been shown to

increase the Ca^{2+} binding capacity of the sarcoplasmic reticulum and to interfere with Ca^{2+} binding to troponin. This would decrease the number of calcium ions bound to troponin during the excitation - contraction coupling and thus cause a decrease in contractile force (Fitts 1977). Additional motor units may therefore be recruited, forcing a subsequent rise in $\dot{V}O_2$. This upward drift in $\dot{V}O_2$ with lactic acidosis has also been noted by Davis (1985).

A high running velocity corresponding to the OBLA may also affect the utilisation of free fatty acid (FFA) as a fuel substrate during prolonged, high intensity exercise (Gollnick 1977). Lipolysis is inhibited by metabolic acidosis (Issekutz et al 1975) probably due to an inhibition of lipase activity in adipose tissue when the pH is low (Brooks and Fahey 1984). Therefore a lower muscle pH, which occurs at the OBLA, would lead to a greater dependence on glycogen as a fuel substrate. This obligatory glycogen utilisation could result in earlier glycogen depletion, which in turn could lead to exhaustion during prolonged exercise because long-term, high-intensity exercise may ultimately be limited by muscle glycogen depletion (Costill et al 1971b). Exercise just below the point of the OBLA could thus result in a much slower reduction of muscle glycogen stores than would exercise above the OBLA. The rate of glycogen utilisation would also be reduced if exercise is performed below rather than above the OBLA because of greater "anaerobic" glycolysis, a biochemical pathway which uses glycogen at a rate that is 18-19 times faster than oxidative phosphorylation for the same energy (ATP) yield (Davis 1985) in exercise above the OBLA.

On the average, international class distance runners possess significantly more slow twitch (ST) muscle fibres than do elite runners, middle distance runners and untrained men (Fink et al 1977). The percentage of ST fibres may be a factor in successful distance running and may separate the international class

from the elite and slower runners (Costill 1979). However, as with $\dot{V}O_2$ max, this factor alone does not discriminate between the abilities within a group of international class or elite runners (Fink et al 1977), who may have more than 90% ST muscle fibres in their gastrocnemius (Costill et al 1973a). The ST fibre has a large amount of myoglobin which functions as a facilitator for the diffusion of oxygen within the cell and mitochondrial enzymes (Essén et al 1975) making them highly oxidative. They have low amounts of glycolytic enzymes (Essén et al 1975) but a high lipid content (Buchtal and Schmalbruch 1970). The fast twitch (FT) fibres can be divided into a further two varieties, FTa and FTb. The FTa fibres are oxidative, less so than the ST but more than FTb (Essén et al 1975). Both are glycolytic, but FTb is more so than FTa (Essén et al 1975) and both have an intermediate amount of lipid (Buchtal and Schmalbruch 1970). ST fibres contain 2-3 times as much triglyceride as FT fibres (Essén 1977) but all three subdivisions contain equal amounts of glycogen (Saltin et al 1977). With endurance training the oxidative capacity of FTa and FTb fibres is increased to above that of ST fibres of untrained subjects (Essén et al 1975) and FTb fibres begin to take on the characteristics of FTa fibres, thus gaining greater oxidative ability (Costill 1979). On average, the ST muscle fibres of international class runners have been found to be 29% larger than the FT fibres, but no differences were found between the two fibre types in a comparison of good middle distance runners and untrained men (Fink et al 1977). This is explained by Saltin (1973) as a selective hypertrophy of the ST fibres with endurance training. As a result of the hypertrophied ST fibres, 82.9% of the cross-sectional area of an international class runners' muscle has been found to be composed of these fibres. If ST fibres are responsible for most of the tension developed during distance running, then international class runners are at an advantage. (Fink et al 1977).

Recent advances in histochemical and biochemical techniques have resulted in several advances being made as to the composition and metabolic characteristics of the muscle of elite and international class marathon runners. Various muscles have been sampled, but those of the lower leg, the gastrocnemius and soleus, are more pertinent for examination when considering distance runners, as these muscles have been shown to be metabolically more active than the vastus lateralis during running (Costill et al 1974). In a paper by Costill et al (1976b) it was reported that a strong relationship existed between the LDH activity and the percentage of the muscle area consisting of ST fibres. A more recent paper, however, in which biopsy samples were obtained from the gastrocnemius of international class marathon runners does not agree with this finding as little relationship existed between the two variables (Fink et al 1977). Succinic dehydrogenase (SDH), a Tricarboxylic Acid Cycle enzyme, is representative of the oxidative capacity of a muscle. The activity of this enzyme is 120% greater in international class than elite runners (Fink et al 1977). These authors point out that the greater SDH activity found in international class runners may be related to differences in training duration and/or intensity and is unlikely to be associated with the percentage ST fibres. This was because little relationship was found to exist between percent ST fibres and SDH activity ($r = 0.22$), the highest activity being found in the runner with the lowest percentage ST fibres (50%). In trained runners the activity of SDH is 350% greater than that of non-runners (Costill et al 1976b). Oxidative metabolism occurs in the mitochondria and evidence exists that both quantitative and qualitative changes occur with training (Gollnick and King 1969). One of these would be the 350% increase in the activity of SDH cited previously. When sedentary subjects participate in a conditioning programme the activity of SDH and respiratory chain enzymes are enhanced (Henriksson and Reitman 1977) but this is localised to the fibres most actively involved in training (Henriksson and Reitman 1976). Low to medium intensity training

was found to cause an increase in the SDH activity of the ST fibres, while high intensity training caused an increase in SDH activity mainly in the FT fibres. The glycolytic enzymes, however, did not show an equivalent increase (Henriksson and Reitman 1976). Costill et al (1976b) have demonstrated that international class and elite runners in fact usually have a low level of glycolytic enzymes. Malic dehydrogenase (MDH) activity of the muscle was found to be greater in international class runners compared to non-runners, but there was no difference in a comparison between international and elite runners (Fink et al 1977). These observations are in agreement with Gollnick et al (1973) who found that skeletal muscle undergoes an increase in oxidative potential after training.

Besides enzymatic differences, haematological differences may exist between international class and elite athletes, or athletes and non-athletes. When considering these three groups, no significant differences were found to exists in haemoglobin concentrations in a study conducted by Martin et al (1977). In the same study, the hemotocrit was found to be lower in the runners, but there was no significant difference in the red and white blood cell concentrations between the international and elite runners. The lower hematocrit was probably attributable to an increased blood volume which occurs with training (Martin et al 1977).

Depletion of muscle glycogen stores has been cited as the major explanation of fatigue during prolonged exercise (Bergström et al 1967). During such exercise the liver may not be able to release glucose into the blood sufficiently quickly so as to balance a progressive increase which occurs in the rate of glucose uptake by the active muscles (Ahlborg et al 1974). The progressive increases in glucose uptake from the blood has also been demonstrated by Wahren (1977) whose results indicated that leg glucose uptake increased 7-fold above the resting value after 40 minutes of light

exercise (65W) and 10-to 20-fold at higher power outputs (200W) during cycle ergometry. Blood glucose oxidation accounted for a growing fraction of the total carbohydrate oxidation during exercise. Glycogen was the dominant carbohydrate substrate during the initial phase of exercise, but with time the oxidation of blood glucose increased steadily, to as much as 75 - 90% of the carbohydrate metabolism (Wahren 1977). When this occurred, hepatic glucose output decreased to 1.8 $mmol.min^{-1}$ and was associated with a progressive decline in blood glucose concentration. Foster et al (1979) suggest that a major factor limiting prolonged exercise may be the development of hypoglycaemia. In a study by Ivy et al (1983) it was concluded, however, that fatigue was not associated with hypoglycaemia. Central nervous system dysfunction as a result of hypoglycaemia was also discounted as a possible source of fatigue because psychomotor tests administered were unaffected after exercise. The authors suggested a local cause of fatigue, possibly muscle glycogen depletion. The results of a study by McArthur et al (1983) also discounted hypoglycaemia as a possible cause of fatigue. Noakes and McArthur (1985) presented data which showed that when running at 85% $\dot{V}O_2$ max, liver glycogen depletion occurs after approximately 2 hours 30 minutes of running, while muscle glycogen depletion occurs after approximately 2 hours of running. Thus hypoglycaemia is unlikely to affect performance in fast marathoners. During exercise at 70 - 75% $\dot{V}O_2$ max lasting longer than 3 hours, liver glycogen depletion precedes muscle glycogen depletion, which would result in hypoglycaemia (Noakes and McArthur 1985). Therefore, hypoglycaemia may limit performance in certain situations.

In the study by McArthur et al (1983), serum glucose levels were elevated (7.2 ± 1.1 $mmol.l^{-1}$) in a group of runners at the end of a marathon with an average finishing time of 2 hours 24 minutes. There was a tendency to higher glucose levels in the faster runners in the study, but this was not a significant difference. Maron et al (1975) have also shown higher blood

glucose levels in faster runners. Recently, Noakes and McArthur (1985) demonstrated that blood samples for glucose analysis must be drawn immediately the athlete stops if this is to bear any relationship to glucose levels during exercise. Any delay results in a rapid increase in blood glucose levels. McArthur et al (1983) showed that the runners who achieved the fastest times in a race had significantly lower blood FFA levels (1.2 ± 0.9 mmol.l^{-1}) than the runners with slower finishing times (2.0 ± 0.6 mmol.l^{-1}). The respiratory exchange ratios of faster runners ($\bar{X} = 0.85$) during a 56km race tended to be higher than that of the slower runners ($\bar{X} = 0.71$), and faster runners had higher blood glucose and serum insulin levels.

A study in which pre-race glycogen content of the gastrocnemius was approximately 200 mmols.kg^{-1} wet weight and post-race approximately 30 mmols.kg^{-1} wet weight (Noakes 1985), the extent of glycogen depletion was similar in fast and slower runners and appeared to be most influenced by the pre-race glycogen level (Noakes and McArthur 1985). Ahlborg et al (1967) and Bergström et al (1967) both showed that the resting glycogen content of the muscle may be a limiting factor in the performance of prolonged, strenuous exercise. McArthur et al (1983) concluded their study with the suggestion that faster marathon runners are less carbohydrate depleted, as shown by muscle glycogen content, than slower runners at the end of a marathon race. This was despite the faster runners having higher R values during the marathon. It was pointed out that this is contrary to the general belief that international and elite calibre marathon runners are better able to utilise fat as a fuel substrate than are slower runners. It would be expected that as the faster runners were less carbohydrate depleted, they must have "supplemented" their fuel substrate requirements during the race with FFA to a greater extent than slower runners. This, however, is not indicated by the R values, which were higher in the faster runners.

Although it was suggested by McArthur et al (1983) and Noakes and McArthur (1985) that it is questionable whether muscle glycogen depletion is the cause of exhaustion during marathon running, evidence from other studies indicate that muscle glycogen depletion may limit endurance performance (Miller et al 1983). This latter contention is supported by Karlsson and Saltin (1971) who reported that muscle glycogen depletion to 17-28 mmols.kg^{-1} wet weight muscle coincided with fatigue. When exercising at 70-80% $\dot{V}O_2$ max, nearly all glycogen is depleted in the vastus lateralis after one to two hours of cycling (Hermansen et al 1967), the rate of depletion being dependent on the % $\dot{V}O_2$ max being employed (Saltin 1973). In a study by Costill et al (1973a), however, it was shown that when running was the mode of exercise, exhaustion occurred although glycogen of the vastus lateralis was not depleted. In a separate study where running was the mode of exercise, biopsy samples were taken from the vastus lateralis, soleus and gastrocnemius and it was found that substantially less glycogen was used from the vastus lateralis than the other two groups (Costill et al 1974). This suggests that the vastus lateralis is not a representative muscle group to use when studying exhaustion in running and it may be incorrect to extrapolate results obtained from this muscle group during cycling, to muscle metabolism during running. Thus, in the study cited above (Costill et al 1973a) (where exhaustion occurred during running although glycogen of the vastus lateralis was not depleted), had the biopsy sample been taken from the soleus or gastrocnemius, it may have been found that these two muscle groups were in fact glycogen depleted. This would have meant that fatigue may have coincided with glycogen depletion. Another finding that implicates muscle glycogen depletion as a possible cause of exhaustion during distance running is that during running selective depletion of the ST fibres occurs, and these are metabolically more active during running than the FT fibres, which do not become depleted (Costill et al 1973a). It was suggested that when the ST fibres are depleted the FT fibres cannot generate sufficient tension to compensate for this, the

result of which is that running becomes more difficult and the runner feels exhausted. It has also been shown that glycogen depletion results in an increase in oxygen uptake, heart rate and pulmonary minute ventilation at any given submaximal power output (Heigenhauser et al 1983).

Analysis of the respiratory exchange ratio during prolonged exercise has suggested that both lipids and carbohydrates contribute to energy metabolism. The proportion of energy derived from the intramuscular stores of glycogen and triglycerides appear to be influenced by several factors. At exercise intensities ranging from 55 - 85% $\dot{V}O_2$ max (the range at which a marathon is run) glycogen concentration decreases in a curvilinear fashion with time, the most rapid decline being observed at the onset of exercise (Essén et al 1977). In a study by Karlsson and Saltin (1971) it was indicated that the availability of glycogen could be a limiting factor to the continuation of prolonged exercise. It appears that the rate of glycogen depletion depends upon the % $\dot{V}O_2$ max being utilised, as Saltin (1973) determined that the greater the % $\dot{V}O_2$ max employed, the greater the rate of glycogen utilisation. After training, glycogen utilisation during exercise is decreased in subjects performing at the same absolute power output as before training. Lower values for the respiratory exchange ratio occur, indicating a greater contribution of lipids to the total energy metabolism. If, however, the power output is increased after training so that the % $\dot{V}O_2$ max utilised before and after training is the same, then the amount of glycogen utilised remains unchanged (Karlsson et al 1974)

The glycogen stores in a muscle can be influenced by the composition of the diet consumed, a carbohydrate –rich diet resulting in larger glycogen stores than a low carbohydrate diet (Bergström et al 1967). These authors found a significant decrease in the length of time exercise could be continued when on a low carbohydrate diet. Conversely, a high carbohydrate diet resulted in

an increase in exercise time to exhaustion. In some people the glycogen level can be raised by dietary manipulation to approximately 220 mmol.kg^{-1} wet weight muscle (4g/100g muscle) (Newsholme 1977) and this plays an important role in sustaining muscular activity during a marathon. Assuming 15 kg of leg muscle and a possible muscle glycogen concentration of 4 g/100 g muscle, the total glycogen stored in the muscle would be 600g. As the oxidation of 1g of glucose substrate (or glycogen) produces approximately 15 KJ of energy, the complete oxidation of this glycogen will produce 9000 KJ of energy. The release of glucose by the liver from stored liver glycogen should provide approximately 1480 KJ, resulting in a total of approximately 10480 KJ of energy from the oxidation of carbohydate. A marathon runner expending energy at the rate of appxoximately 72 KJ.min^{-1} should thus have enough energy from the oxidation of carbohydrate for approximately 146 minutes of running (Newsholme 1977). This represents the entire marathon for a fast marathon runner. These calculations, however, assume a 100% efficiency in the conversion of chemical energy to the ADP-ATP energy couple. Free fatty acids (FFA) supply any deficit.

Intramuscular triglyceride stores play an essential role in prolonged submaximal running. Costill et al (1973a) found that the triglyceride content in the vastus lateralis decreased by 30% after a 30 km race. The FFA from these local lipid stores accounts for about 50% of the total lipid oxidation, while the remaining 50% of lipid oxidised is from blood-borne FFA (Havel et al 1967). During exercise only a small fraction of the available FFA is oxidised by the working muscles (Gollnick 1977), the major limiting factor being the diffusion of the FFA molecules from the capillaries into the muscle cell where oxidation occurs (Saltin 1973). An enhancement of the oxidation of FFA occurs with training as a result of a reduction in diffusion distance and an increase in mitochondrial oxidative enzymes (Saltin 1973).

It has been noted by Pugh et al (1967) in a study of 56 marathon runners, that three of the first four runners to finish a particular marathon race had higher rectal temperatures (Tr) (41.1° C, 40.5°C and 40.2°C respectively) at the finish than other runners in the race who had an average post-race Tr of 38.9°C. It was suggested that the capacity to withstand a high body temperature was a requisite for successful marathon running. A significant correlation between race time and Tr was also found to exist in a study conducted by Maron et al (1975). In the Maron study all the runners completed the race within 11 minutes of each other.

Maron et al (1977) reported on two runners where Tr and skin temperature (Ts) from 5 sites were monitored. These two runners finished a race within two minutes of each other. After 45 minutes of running, the Tr of runner one reached 38.9 - 39.2°C and then remained constant for the next two hours at which point he finished the race. The Tr of runner two, however, plateaued at 40.0 - 40.1°C after 45 minutes of running, but a sharp secondary increase of 1°C in Tr occurred between minutes 113 and 119, remaining at that level for the last 44 minutes of the race. A partitional calorimetric analysis suggested that the observed secondary increase of 1°C was due to a transitory decrease in sweating. The arm Ts of runner two was 4.0-6.5°C higher than that of runner one throughout the race, but thigh temperature was 2.5 - 9.5°C lower than that of runner one, demonstrating the variability that can exist between runners of comparable capability. It was suggested that the observed differences in $\overline{Ts}$ between the two runners may have been due to differences in regional sweating and/or cutaneous vasomotor activity.

An investigation by Wyndham and Strydom (1969) revealed that post-race Tr was linearly related to the percentage of mass lost through sweating, once this loss exceeded 3% of initial body mass. All runners with greater than 4% mass loss had a Tr higher than 39.4°C. The authors attribued a lower

Tr to smaller water deficits, but did not suggest a mechanism for this. Senay (1968), however, has suggested that the mechanism may be associated with a change in plasma osmolarity. The relationship between percent mass loss and Tr appears to vary between subjects. This seems to depend upon differences in $\dot{V}O_2$ max and % $\dot{V}O_2$ max (Saltin and Hermanson 1966), body mass/surface area ratio (Shvartz et al 1973), state of hydration (Buskirk et al 1958) and degree of heat acclimatisation (Schvartz et al 1978).

A marathon runner is able to maintain a high level of exercise for a number of hours. During this time body core temperature is maintained at a level between 39 - 41°C (Adams et al 1975), even though international class marathon runners sustain a metabolic heat production exceeding 650 $W.m^{-2}$ for approximately 2 hours 15 minutes (Costill and Fox 1969). Since a large amount of heat is produced in the contracting muscles while running, a highly efficient temperature regulating system is necessary. In the presence of a thermal load, receptors in the hypothalamus sense any increase in temperature and in response directs an integrated reflex causing cutaneous vasodilation, resulting in increased circulation to the skin surface where heat can be dissipated primarily by the evaporation of sweat (Nadel et al 1977). It is also well established that heat-acclimatised individuals maintain a lower core temperature when exposed to heat stress (Wyndham 1967) and that heat acclimation results in an increased sweat rate for a given central drive by lowering the core temperature threshold for the onset of sweating (Nadel et al 1974). Testing of various African groups has indicated better heat tolerance in black subjects relative to unacclimatised whites (Wyndham et al 1966). Blacks have been found to have more active sweat glands than whites (Wyndham et al 1966), and a reduced salt content of the sweat (Shephard 1980).

Improved sweating responsiveness may provide a small cardiovascular advantage

as skin blood flow demand would be less, while core temperature would be maintained during steady state activity (Nadel et al 1977). Even mild dehydration, however, results in a core temperature that is higher during steady state exercise (Greenleaf and Castle 1971). The primary advantage of a reduction in skin blood flow due to a reduced sweating response threshold is an improved ability to maintain an adequate circulating blood volume (Nadel et al 1977). As a 15% decrease in plasma volume occurs after approximately 50 minutes of exercise, probably due to hydrostatic fluid shifts from the intravascular to extravascular compartments (Lundvall 1972), maintenance of cardiac filling pressure could become difficult (Harrison et al 1975). Nadel et al (1977) and Saltin and Stenberg (1964) found that reduced circulating plasma volume resulted in a lower stroke volume, but that increased heart rate resulted in the maintenance of cardiac output. This includes exercise when ambient temperature is high (Williams et al 1962). Hartley (1977) and Johnson (1977) suggest that the increase in heart rate is caused by the thermoregulatory shunting of blood to the cutaneous vessels. As previously described this is probably because as core temperature increases, cutaneous vasodilation occurs in response to a reflex from the hypothalamus (Nadel et al 1977) resulting in a decrease in venous resistance and venous pressure (Hartley 1977 , Johnson 1977). These adjustments occur gradually during the time-course of prolonged exercise, but may eventually result in competition for circulation between cutaneous vessels and the metabolically active skeletal muscles (Hartley 1977, Johnson 1977). It has also been noted that Ts (Adams 1977), sweat rate (Costill et al 1970b) and Tr (Maron et al 1977) are higher at faster running speeds. During a fast treadmill run ($293 m.min^{-1}$) at approximately 80% $\dot{V}O_2$ max (with cooling via a fan) Tr increased gradually for approximately 35 minutes, but from 35 - 40 minutes (when testing was terminated) began to plateau at 39.5°C (Adams 1977). In one of the subjects tested by Adams the Tr increased at a rate which would have resulted in a temperature exceeding

41°C after 45 minutes of running. This subject would either have had to stop, or reduce running speed. It therefore appears that some runners may not be able to maintain thermoregulation over the entire marathon distance at a particular running speed (Adams 1977). It has been estimated that a 60 kg runner sweats at a rate of 1250 $ml.hr^{-1}$ (Wyndham 1977) or 1.09 $l.m^{-2}.hr^{-1}$ (Costill et al 1970b). Contrary to the Tr data of Adams (1977), Wyndham (1977) suggests that if the humidity is sufficiently low to allow for evaporation of sweat, this sweat rate would enable marathon runners to remain in thermal balance, provided sufficient liquid is replaced during the run. Runners who drank sufficient liquid to keep their level of dehydration below 3% of body mass were found to have a Tr of approximately 38.5°C at the end of 30 km race, but runners who developed more than 5% dehydration finished with a Tr of about 41°C (Wyndham 1977). The latter would result in impaired performance and physiological function during the run (Saltin 1964). If dehydration greater than 2% (Costill 1979) to 3% (Wyndham and Strydom 1969) of body mass occurs, peformance will be impaired, probably due to increased demand on the cardiovascular and thermoregulatory systems (Saltin 1964). Such a decrease in performance may be compounded by an increase in muscle glycogen metabolism when ambient temperature is high, as Fink et al (1975) demonstrated that muscle glycogen utilisation was greater at an ambient temperature of 40°C than at 9°C. Therefore, when racing in the heat, muscle glycogen depletion may be a greater problem than when racing in cold environmental conditions. A higher environmental temperature may also cause a decrease in the running speed at which the OBLA and ventilary thresholds occur (Gilman and Lemon 1982).

Cardiac output remains relatively constant during prolonged exercise (Saltin and Stenberg 1964). Therefore, if additional blood is shunted to the skin to aid cooling during prolonged running, a concomitant decrease in blood

flow to the active muscle will occur. A major demand on the circulation, however, is the maintenance of blood flow to these muscles. If this is maintained during prolonged exercise, however, skin blood flow and therefore the regulation of core temperature may suffer. Due to this competition, peripheral blood flow is controlled during exercise. Vasoconstriction occurs in response to exercise in order to maintain blood flow to the active muscle, but a competing drive for vasodilation exists due to a rising core temperature. (Johnson 1977).

During prolonged exercise at 66% $\dot{V}O_2$ max it has been observed that there is a gradual increase in both pulmonary minute ventilation and ventilatory frequency during the time-course of the exercise. The stimulus for this could be a thermal drive caused by an increasing core temperature, but the exact mechanism is unknown. (Dempsey et al 1977). Cerny et al (1982) found that during cycle ergometry, American blacks had a higher ventilatory frequency and lower tidal volume for any given pulmonary minute ventilation than did whites. Increased lung elastic recoil or breathing higher on the pressure-volume curve in blacks were suggested by the authors as possible reasons for this.

An examination of international class white runners has revealed an average pulse rate of 47 $b.min^{-1}$ and mean blood pressures of 117/74 mmHg (Gibbons et al 1977). In a study of black and white untrained males exercising on a cycle ergometer, it was found that blacks had higher heart rates than the whites (140.0 $\pm$ 13.2 and 137.9 $\pm$ 91 $b.min^{-1}$ respectively), systolic blood pressure (190.5 $\pm$ 21.3 and 179.8 + 17.4 mmHg respectively), diastolic blood pressure (85.9 $\pm$ 14.6 and 80.9 $\pm$ 12.5 mmHg respectively), and pressure-rate product (261.7 $\pm$ 65.3 and 248.4 $\pm$ 7 respectively) at maximal exercise. (Cieslik et al 1984). Cardiac hypertrophy has been noted in runners by Arstila and Koivikko (1966). As heart size varies

in people of the same height, mass and age, it is possible that some people are born with a heart distinctly larger than average, which could give a potential advantage in endurance activities (Costill 1979). The heart volume of endurance athletes is greater than normal (Underwood and Schwade 1977), and Pollock (1977b) found that when expressed relative to body weight, international class runners possess a greater heart volume than elite runners. Low correlations, however, were found to exist between heart volume and both $\dot{V}O_2$ max and treadmill running time to exhaustion (Pollock 1977b). Both Costill (1979) and Pollock (1977b) suggest that a possible explanation of the enlarged heart volume is that a genetic factor is responsible. This appears plausible since heart volume has not been shown to increase when sedentary people indulge in endurance training (Åstrand and Rodahl 1977). In a study by Underwood and Schwade (1977) runners were found to have greater left atrial diameter, greater left ventricular mass, and greater stroke volume at rest than control subjects. A physiological examination of international class and elite marathon runners revealed a greater stroke volume at rest in the case of international runners (Pollock 1977b) which is in agreement with Saltin (1973) who suggested that stroke volume may separate the champion from the good athlete. Underwood and Schwade (1977) have reported resting stroke volumes of 95 $ml.beat^{-1}$ for international class runners, 85 $ml.beat^{-1}$ for elite runners and 72 $ml.beat^{-1}$ for non-runners. Costill (1979) reported a stroke volume of 149 $ml.beat^{-1}$ in marathoners performing a 2 hour 26 minute marathon. It has also been found that there was a greater difference between resting H/R and H/R max in international class runners than elite runners (151 $b.min^{-1}$ and 143 $b.min^{-1}$ respectively) (pollock 1977b). The international class runners had a slightly lower resting H/R (47 $b.min^{-1}$) compared to elite runners (52 $b.min^{-1}$) and a slightly higher H/R max was recorded in the international class runners (198 $b.min^{-1}$) compared to the elite runners (195$b.min^{-1}$). (Pollock 1977b).

These H/R maxima are somewhat higher than the 161-197 $b.min^{-1}$ ($\bar{X}$ = 179 $b.min^{-1}$) reported by Costill et al (1973b) for a group of highly trained distance runners of varying ability.

The differences between resting and maximum H/R reported above respresents a 4.2-fold increase in the case of international runners and a 3.7-fold increase for the elite runners, reflecting a greater cardiac reserve in the international class runners. People with identical cardiovascular fitness may have a different submaximal H/R for the same exercise load. As any one particular person gets fitter, however, the submaximal H/R at a given exercise load will be less (Clausen 1977), stroke volume will be greater (Ekblom et al 1968) and the recovery rate towards normal will be faster (Åstrand and Rodahl 1977). The H/R of international class runners was found to be lower than that of elite runners at a given submaximal speed (Pollock 1977b).

Whereas some of the physiological aspects such as muscle fibre composition and $\dot{V}O_2$ max are largely a reflection of the genotype of the individual, the biomechanical measurements are essentially quantitative expressions of running style (Cavanagh et al 1977). In a longitudinal study conducted by Nelson and Gregor (1976) it was found that nine out of ten distance runners tested tended to decrease their stride length and increase stride rate at a given velocity over a period of four years. During this time the performances of the runners improved. The decrease in stride length may be related to a minimisation of the oxygen uptake at any given speed. (Cavanagh and Williams 1982). The changes observed could not be attributed to direct intervention by the coach (Nelson and Gregor 1976). It was determined by Cavanagh et al (1977) that a significant difference ($p < 0.05$) existed between international and elite runners in the step length/leg length relationship. The correlation between these two variables was 0.67

for the international class runners, but -0.10 for the elite runners. These correlations indicated that the elite runners took long steps (172% of leg length) irrespective of their leg length. The international class runners, however, took shorter steps (165% of leg length), with the step length being related to leg length in these runners. Cavanagh and Williams (1982) found no relationship between leg length and step rate of recreational runners. As blacks have a greater leg length (Jordan 1969), these relationships may differ in black runners. In the Cavanagh and Williams (1982) study it was noted that of all possible combinations of step length and step rate all subjects exhibited a step length at which oxygen uptake ($\dot{V}0_2$) was minimised. When step length was shortened, $\dot{V}O_2$ increased an average of 2.6 $ml.kg^{-1}.min^{-1}$ and when lengthened, $\dot{V}O_2$ increased an average of 3.4 $ml.kg^{-1}.min^{-1}$. The authors are of the opinion that the naturally chosen step length is the most economical due to an adaptation through training or a process of energy optimisation.

The patterns of hip and knee joint motions have been shown to be similar between international class and elite runners with only a slight difference existing between the two groups during the swing phase. Considerable hip extension and knee flexion was shown to occur before foot-strike, and the authors (Cavanagh et al 1977) point out that the knee was never fully extended at any stage of the running cycle. Toe-off occurred at the point of maximum knee extension. No significant differences existed between groups in maximum knee flexion during support and swing phases, maximum hip flexion during the swing phase, or the position of the limb at foot-strike. A significant difference did exist between groups, however, in the final phases of plantar thrust, where elite runners were shown to plantar flex approximately 10° more than international class runners (Cavanagh et al 1977). It is therefore interesting to note that the only significant difference which existed between the groups was at the ankle joint. This may account

for the differences in step length, as the difference in ankle angle may result in a greater horizontal component to the force being exerted. It has also been determined that elite runners tend to have greater amplitude of vertical oscillation than international class marathon runners, although the difference was not statistically significant (Cavanagh et al 1977). These authors concluded that the lack of major significant differences in the biomechanical variables between elite and international class runners suggests that both groups in the study had a similar range of running styles.

An investigation into the mechanical efficiency of positive work in running (Ito et al 1983) revealed that efficiency stayed approximately the same across a range of running speeds from 7 - 22 $km.hr^{-1}$. It was found that the elastic recoil during the stretch-shortening cycle of the contact period provided an appreciable amount of extra work and could occur at the hip, knee and ankle joints. The authors suggested that this could explain the constant efficiency observed and pointed out that their results differed from those of previous studies where the transfer of energy between segments was not considered. Williams and Cavanagh (1981) considered between-segment energy transfer an important factor when explaining differences in mechanical work among runners. The percent ST fibre and elastic storage, however, did not appear to exert any influence on differences in efficiency between runners.

Although biomechanical measurements of international and elite class distance runners are generally similar, some differences do exist between these groups in factors of body composition. As regards percentage body fat, a study on a marathon winner showed lower body fat values when body density was measured by the hydrostatic weighing technique rather than by prediction equations using anthropometric measurements. (Pollock et al 1977). The same authors point out that research findings have suggested the use of population-specific

equations, as the accuracy of prediction when using equations from either normal or athletic populations with a different body type to that of the marathoner is questionable. An investigation by Schutte et al (1984) revealed that in whites the observed body density does not vary significantly from density predicted by anthropometry. In blacks, however, the observed density was significantly greater than the predicted density and the authors suggested that a separate formula should therefore be used when converting density to percent fat in black subjects (Schutte et al 1984). Regression analysis revealed that the equation for converting density to percent fat in blacks was:

$$\% \text{ fat} = 100 \times ((4.374/\text{density}) - 3.928)$$

In a study conducted by Pollock et al (1977) it was found that international class runners had an estimated body fat of 5.1% when this was calculated using the Brožek and Keys equation, but this estimate was 4.7% when the Siri formula was used. The percentage fat of elite runners was 6.1% using the Siri formula. These values were lower than the 6 - 8% reported previously for well trained distance runners (Costill et al 1970a). The difference may be due to previous studies having included both elite and international runners as a single group, using anthropometric techniques to estimate body density and/or using runners whose training was less intense than that of the runners in the Pollock study (Pollock et al 1977). International class marathon runners have been found to be shorter in stature than elite runners (176.8 and 181.1 cm respectively), lighter in body mass (62.1 and 67.5 kg respectively), and higher in body density (1.08954 and 1.08527 $g.ml^{-1}$ respectively) (Pollock et al 1977). The previous world marathon record holder (2 hours 8 minutes 33 seconds - still one of the fastest times ever recorded) was, however, 188 cm tall (Costill et al 1971a). Although stature may not affect performance it appears that body mass does, possibly due to extra energy being needed to run if body

mass is high (Costill 1979).

The reason that only a few marathoners achieve noteable success although many meet the physiological, biomechanical and body compositional pre-requisites for successful marathon running as described, may well be found in the psychological attributes of the international and elite marathon runner. Morgan and Costill (1972) indicated that marathon runners as a group were characterised by introversion, stability and low anxiety levels, but none of these variables appeared to be correlated with performance in the marathon. Other studies have also shown marathon runners to be more introverted than the general population (Clitsome and Kostrubala 1977). These researchers used the Myers-Briggs Type Indicator, a psychological test that delineates personality type, on a sample of 100 marathoners. It was found that the ratio of extroverts/introverts was approximately 1/1 (54 extroverts, 46 introverts) whereas the usual distribution in the general population (USA) is 3/1. Marathon runners therefore differ from the general population in a tendency towards introversion by a ratio of approximately 2/1. Other studies, however, have not found marathon runners to be more introverted than normal (Morgan and Pollock 1977). An analysis of psychological data collected from international class runners has shown that these runners do not differ significantly from elite runners in any of 16 variables examined (Morgan and Pollock 1977). These variables included measures of state and trait anxiety, perception of somatic activity during stressful situations, depression, tension, anger, vigour, fatigue, confusion, extroversion, neuroticism, conformity, attraction toward physical activity, estimation of physical ability and field dependence. The authors reported that the runners scored below the population mean for tension, depression, anxiety, fatigue and confusion, but above the mean for vigour.

In the study by Morgan and Pollock (1977) data revealed that international

and elite class marathon runners are different from the general population and non-elite runners in their cognitive activity during competition. Whereas non-elite runners employed a cognitive strategy of dissociating painful input by thinking of things other than running while racing, international and elite runners used a technique of association, by concentrating and acting on any input such as pain and fatigue, attempting to process this information and modulating speed as well as strategy accordingly. The input used by the runners in this strategy of association included respiration as well as sensory information from the feet, calves, and thighs. The speed of running was largely determined by "reading" these sensations. During a race international class runners did not encounter "pain zones" or "the wall" and constantly reminded themselves to "relax". (Morgan and Pollock 1977). If the ability to relax is important, the black runner may be at an advantage as Phillips (1976) states that blacks may have a culturally conditioned tendency to relax rather than become tense under pressure. This could be an advantage in a competitive situation where "tensing up" could hamper the efficiency of a runner.

It has been suggested that whether a marathoner runs at 65% $\dot{V}O_2$ max or 85% $\dot{V}O_2$ max depends on his "willingness" to tolerate the discomfort associated with running at the latter. This may have a physiological basis. It was described earlier when considering the ventilatory threshold that some runners may accumulate large amounts of blood lactic acid at relatively low percentages of $\dot{V}O_2$ max, whereas in other runners this may occur at a much higher % $\dot{V}O_2$ max. Some runners can run at 85% $\dot{V}O_2$ max before lactic acid accumulation occurs. Both groups of runners may therefore be running equally "hard" although at vastly different percentages of $\dot{V}O_2$ max. As previously pointed out, the speed at which lactic acid accumulation occurs can be attributed to various physiological attributes possessed by the runner. This, however, does not rule out psychological factors playing

a role in distinguishing the international class athlete from the merely "elite".

When running at 20 $km.hr^{-1}$ international class marathon runners scored lower on the Borg RPE Scale than did elite runners. At lower speeds there was no difference in RPE ratings between the two groups of runners, even though the elite athletes were running at a greater oxygen uptake. For instance, although RPE was the same in both groups when running at 16 $km.hr^{-1}$, the mean H/R of the elite athletes was 16 $b.min^{-1}$ higher than that of the international class runners after two minutes of running, and 19 $b.min^{-1}$ higher after six minutes of running. The pulmonary minute ventilation ($\dot{V}_E$) of the elite runners was 23 $l.min^{-1}$ higher after six minutes of running (Morgan and Pollock 1977). The elite runners were therefore running at a higher $\dot{V}_E$ and % $\dot{V}O_2$ max than the international runners, yet the RPE was the same in both groups. The RPE of the elite runners increased concomitantly with an increase in blood lactic acid. During constant intensity exercise above the ventilatory threshold, however, increases in RPE appear to be independent of changes in H/R, oxygen uptake, $\dot{V}_E$ and blood lactic acid (Simon et al 1984). The independance of $\dot{V}_E$ to RPE is supported by results from a study by Montgomery and Constantinides (1984) in which $\dot{V}_E$ was found to have no influence on RPE during 2 hours of treadmill running at the OBLA. Accumulation of blood lactic acid has been considered a "local" cue to increased RPE, whereas H/R (Borg 1973) and $\dot{V}_E$ (Morgan and Pollock 1977) have been considered "central" cues.

Although Phillips (1976) rejects the possibility of blacks being superior in certain psychological characteristics that provide a competitive advantage, black runners may have other psychological as well as socio-economic incentives to perform at a higher level. Jokl (1977) has stated that sport represents a form of social elevation and a means of achieving greater status

and freedom for the less privileged. Talented blacks may pursue success in sport because this is one of the areas in which they perceive a fair chance of success (Phillips 1976). Running is one of the less expensive sports, enabling the average black to participate. Further, blacks who perform well in athletics in SouthAfrica often get career offers at one of the South African mines where they are assured of a regular income and ample training opportunity. This is in agreement with a statement by Edwards (ex Phillips 1976) that black athletes concentrate on sports in which they can earn money and gain prestige. The absence of blacks in sports such as tennis and golf does not support this contention (Phillips 1976). These sports require skill and thus coaching in order to excel, however, and in South Africa this is not generally available to the young black due to economic and political factors. Blacks may prefer running to many other sports as running does not require much skill and for the young black it may be the only sport in which he perceives he can earn a living and prestige without the necessity of coaching. This is borne out by Phillips (1976) who suggests that sports in which blacks excel are distinguished by having facilities, coaching and competition available in schools. Sports in which black competitors are rare usually have facilities and coaching available only at private clubs. There may therefore be a concentration of black talent in athletics.

Summary

Few studies have addressed the problem of acute and chronic responses of black athletes to long distance running. However, any physiological and biomechanical differences between black and white runners may reflect the differences known to exist in these domains between international class, elite and non-elite runners, and thus this review of literature has centred on studies conducted in this area.

Genetic influences play a major role in the $\dot{V}O_2$ max of an individual and some differences have been noted in different ethnic groups. The $\dot{V}O_2$ max of international runners is higher than elite and non-elite, but this variable alone cannot be used to discriminate between individuals in a group in which all members have a high $\dot{V}O_2$ max.

During cycle ergometry, the $VE-O_2$, pulmonary minute ventilation and ventilatory frequency, have been found to be higher in American blacks than whites, but tidal volume was lower in the blacks. Generally, ventilatory variables have not been found to differ between international and elite athletes.

The ability to utilise a large % $\dot{V}O_2$ max appears to be an important factor in determining a winning performance. International class runners can run at 85% $\dot{V}O_2$ max with little lactic acid/lactate accumulation. Running economy is another important determinant of success, a lower submaximal oxygen uptake at a given speed being an advantage. International class runners are more economical than elite runners and have lower pulmonary minute ventilation at submaximal running speeds. They have lower heart rates than the elite runners but higher stroke volumes. Black athletes appear to be able to exercise at a higher power output than whites at a given submaximal oxygen uptake.

A high correlation has been found to exist between the running speed at the onset of blood lactic acid accumulation (OBLA) and the running speed during a marathon. Runners with a higher running velocity at the OBLA utilise a lower % $\dot{V}O_2$ max at a given submaximal running speed.

International class runners have been found to possess a larger percentage of slow twitch muscle fibres, and greater succinic dehydrogenase activity than elite runners. Faster runners have been shown to finish a marathon

with significantly higher R values than slower runners, indicating that a larger percentage of the energy requirements were from glycogen (carbohydrate) fuel substrate even at the end of a marathon. This suggests that faster runners are less glycogen depleted at the end of a marathon as they are still utilising carbohydrate fuel substrate. Glycogen depletion has been shown to be associated with fatigue and a decrease in performance.

A significant correlation has been shown between race time and rectal temperature (Tr), with faster runners having a higher Tr at the finish of a marathon. Blacks may have better heat tolerance than unacclimatised whites.

It has been found that distance runners tend to decrease step length over a period of years and that inernational class runners take shorter steps than elite runners. The internationalists have also been shown to plantar flex approximately 10° less than elite runners.

International class runners have between 4.7 - 5.1% body fat and elite runners 6 - 8%. The former are also shorter in stature and lighter in body mass. While running at 16 $km.hr^{-1}$, international class runners scored the same on the Borg RPE Scale as elite runners, even though the latter were running at a higher % $\dot{V}O_2$ max and had higher heart rates and pulmonary minute ventilation ($\dot{V}_E$). Heart rate and $\dot{V}_E$ have been considered "central" cues, while accumulation of blood lactic acid a "local" cue to increased RPE. The explanation of superior performance of blacks in running may lie in the concentration of black talent in sports such as athletics and because blacks may perceive a chance of earning money and gaining prestige by running, giving added incentive for a high level of performance.

CHAPTER 3

METHODS

Subject Selection

Nineteen experienced male marathon runners, 9 black and 10 white, were selected as subjects for the study. Selection was based on their recent marathon times, the standard being set at 2 hours 45 minutes or better, as this represents a fast time for the marathon. As few such runners live in the Eastern Cape and Border regions, any runner who fell into this time category and was prepared to participate in the study after the purpose and format had been explained, was selected as a subject. These athletes were contacted by personal communication, via their coaches, and through other runners.

Pilot Testing

No pilot testing over the entire marathon distance was conducted as this was impractical. The technique and equipment used, however, has been found to be reliable in a separate study conducted by Rorke (1985). Rorke used oxygen uptake ($\dot{V}O_2$) as a measure to ascertain whether the system was consistent using a test-retest procedure. On two consecutive days the mean $\dot{V}O_2$ for each of two subjects was found to be statistically the same. ($p < 0.05$).

Assistants

Assistants aided during both the $\dot{V}O_2$ max determinations and simulated marathon runs. They were familiar with the protocols and equipment being used and were experienced in this type of research.

Design of the Study

Each subject reported individually to the laboratory where the testing was

conducted. On arrival, the subject was informed again of the purpose and format of the study, as well as possible benefits to him as a subject. In addition, each subject was given an information sheet (Appendix A) to read and then signed a "subject consent" form (Appendix B). When this was completed various anthropometric measurements were taken and a $\dot{V}O_2$ max test then performed. Before testing, the subject was given time to habituate to treadmill running. When running naturally and comfortably, the $\dot{V}O_2$ max test was commenced. On completion of the test each subject was allowed a four hour rest interval before the simulated marathon.

Anthropometry

The following anthropometric data were obtained from each subject: stature, biacromial (shoulder) diameter, mass, trochanterion height and skinfolds.

1. Stature

   The subject stood barefoot on the platform of a Holtain Stadiometer, with heels together and stretched upward to the fullest extent after taking a deep breath. The Frankfurt plane was kept horizontal. The arm of the stadiometer was then brought down to touch the head of the subject (Weiner and Lourie 1981) and stature recorded in centimetres to one decimal place.

2. Biacromial Diameter

   The subject stood with shoulders relaxed, but not slumping forward. From behind the subject the outside edge of the acromion process of the scapula was located. The arms of the anthropometer were placed on the lateral border of each acromion process and pressure applied to compress the overlying tissues (Weiner and Lourie 1981) and the biacromial diameter recorded in centimetres to one decimal place.

3. Body Mass

   This was recorded in kilograms to one decimal place on a Seca beam

balance with the subject wearing only running shorts. Mass was recorded again prior to and after the simulated marathon run in order to determine any mass change.

4. Trochanterion Height

With the subject barefoot, the height (cm) of the right trochanterion above the ground was measured, with the anthropometer kept vertical to the ground and the arm horizontal (De Villiers and Tobias 1974).

5. Skinfolds

The triceps, biceps, subscapular, supra-iliac, abdominal, front thigh and medial calf skinfolds were measured by means of a Harpenden skinfold caliper (jaw pressure 10 $g.mm^{-2}$). The skinfold was picked up between thumb and forefinger and the caliper jaws applied so that the jaws were approximately the thickness of the skinfold in towards the body. The measurement in millimetres was read approximately two seconds after the full pressure of the caliper was applied to the skinfold (Weiner and Lourie 1981).

a) Triceps

The skinfold was picked up at the back of the arm, about 1 cm above the maximum circumference of the biceps with the elbow flexed and directly in line with the olecranon process. The subject relaxed his arm and the caliper jaws applied (Weiner and Lourie 1981).

b) Biceps

The skinfold was picked up at the front of the arm, directly above the centre of the cubital fossa, at the same level as the triceps skinfold (Weiner and Lourie 1981).

c) Subscapular

The skinfold was picked up under the inferior angle of the scapula and inclined slightly laterally downward (Weiner and Lourie 1981).

c) Supra-iliac

The skinfold was picked up approximately 1 cm above and 2 cm medial

to the anterior superior iliac spine on a vertical plane (Weiner and Lourie 1981).

e) Abdominal

The skinfold was picked up at the level of the umbilicus, 5 cm to the left and in a vertical plane (Weiner and Lourie 1981).

f) Thigh

The skinfold was picked up at the anterior aspect of the thigh, halfway between the mid-inguinal point and the upper border of the patella, with the knee flexed at 90° by means of the foot being placed on a foot-rest (Weiner and Lourie 1981).

g) Medial Calf

The skinfold was picked up at the level of the maximal circumference of the calf, on the medial border of the leg (Weiner and Lourie 1981).

Computer - Aided Data Acquisition System

A computer-aided on-line data acquisition system which provided a print-out of a range of physiological data was used to acquire cardiopulmonary data during both $\dot{V}O_2$ max testing and simulated marathon runs. This system, designed by B.R. Goslin of the Human Movement Studies and Physical Education Department, Rhodes University, has been shown to be both accurate and reliable (Goslin et al 1984). The validity of the system was established by having 10 trained distance athletes running on the treadmill on two occasions, using the same protocol on each. On each occasion data was collected for both steady and unsteady state running, once using a bag collection technique and once using the computer-aided on-line data acquisition system. The software programmes for use with the system were developed by B.R. Goslin and M. Campbell, and computed a range of physiological data (Appendix D). During this developmental phase, gas fractions from expired air in the mixing chamber were compared to that from simultaneously collected bags of expired air. Submaximal oxygen uptake as well as $\dot{V}O_2$ max were compared using the computer-aided

technique and a bag technique and found to be statistically identical. In addition, during the development of the system, volume calibration was performed in a pulsatile manner against a Singer gasmeter which had been previously calibrated by a negative pressure water removal method (Goslin et al 1984). This system,shown in Figure 1, enabled the subject to inhale ambient air through the inlet port of a Mijnhardt dry gasmeter at which point the volume of air inspired was measured. The air was directed from the exit port of the gasmeter via 3 cm diameter Collins ridged tubing to the subject. A Hans Rudolf 2700 pulmonary valve was incorporated into the system so that the subject could inhale air via only the gasmeter. Total inspiratory resistance in this system was quite low at 0.3 - 0.5 cm water pressure, depending on the air flow. Expired air was directed by means of the valve and the same type of tubing to a 4 l perspex mixing chamber. The expired gas was kept circulating in the chamber by means of an internal electric fan. Air for analysis was sampled from a sample port on the side of the chamber at a rate of 300 $ml.min^{-1}$. The remaining air exited from the chamber into the laboratory via a 1m section of Collins ridged tubing, thus preventing contamination of the expired gas in the mixing chamber with ambient air. The volume of the chamber was set at 4 l so as to avoid wide fluctuations in the expired gas fractions. This resulted in a more sensitive response to changes in expired gas composition due to a closer association between these fractions and the corresponding inspiratory volume.

The temperature of the inspired air was measured as it passed through the gasmeter by a thermister located inside the meter. Gas analysis of the air drawn from the sample port was via Beckman LB-2 and OM-14 analysers for carbon dioxide and oxygen respectively. Later in the study, analysers with a faster response time were installed. These were the Ametek S-3AI oxygen analyser with N-22 oxygen sensor, and Godart Capnograph Mark III

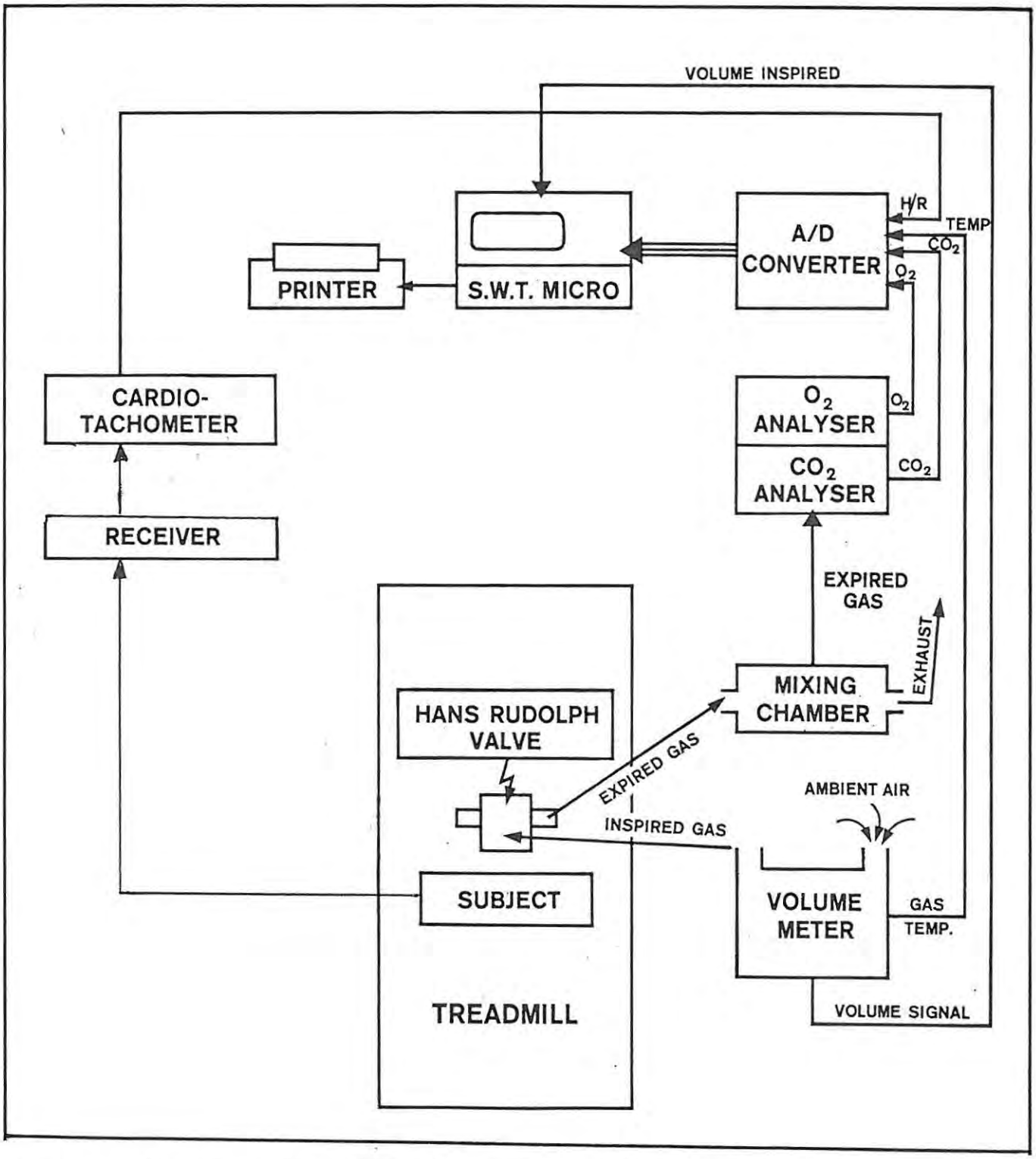

Figure 1: Schematic configuration of the computer-aided data acquisition system (Goslin et al (1984))

carbon dioxide analyser. Gas mixtures of known concentration were used to calibrate the analysers before, during and after testing.

Heart Rate Monitoring

Each subject wore a holter-type heart rate monitoring system during the $\dot{V}O_2$ max test and again during the running of the simulated marathon. This system was used as it was found that stress-test electrodes worked loose after about 1 hour of running. The system consisted of a modified "Exersentry" heart rate monitoring system. The modification involved a lengthening of the connecting wires between the belt with its electrodes and the receiver, so that the subject did not have to carry the receiver while running. In order to smooth the output signal from the receiver it was passed through an optical-isolater before input into a Quinton 611 Cardiotachometer. To improve contact between the skin of the subject and the pick-up electrodes of the belt, the skin surface was thoroughly cleaned with surgical spirits (isopropyl alcohol) and the belt electrodes coated with electrolyte paste. The electrodes were placed so that the left electrode was at the V5 position and the right electrode at a corresponding position on the right of the subjects' chest.

Input to Computer and Printer

Analogue signals from the temperature probe in the gasmeter, the oxygen and carbon dioxide gas analysers, and the Quinton 611 Cardiotachometer were fed into a multiplexor and then into a 12 bit, 8 port analogue-to-digital converter. The digital output was sampled at a rate of 220 times per minute by a South Texas 6800 microcomputer with 32 K core memory. Results of oxygen consumption, respiratory exchange ratio, elapsed time, % $\dot{V}O_2$ max and treadmill speed were displayed on the visual display unit and this together with further physiological data printed out via a Centronics printer (Appendix E). The information on the print-out included relative ($ml.kg.^{-1}min^{-1}$) and absolute ($l.min^{-1}$) oxygen uptake, volume of carbon dioxide produced ($l.min^{-1}$), respiratory exchange ratio, inspired volume

($l.min^{-1}$), heart rate ($b.min^{-1}$), breathing frequency ($breaths.min^{-1}$), inspired gas temperature ($^{\circ}C$), fraction of expired oxygen(F_EO_2), fraction of expired carbon dioxide, (F_ECO_2), ventilatory equivalent for oxygen ($VE\text{-}O_2$) ($\dot{V}_I/\dot{V}O_2$) ($l.100ml^{-1}$), ventilatory equivalent for carbon dioxide ($VE\text{-}CO_2$) ($\dot{V}_I/\dot{V}CO_2$) ($l.100\ ml^{-1}$), estimated cardiac output ($l.min^{-1}$), estimated stroke volume ($ml.beat^{-1}$), oxygen pulse ($ml.beat^{-1}$), tidal volume ($l.breath^{-1}$), % $\dot{V}O_2$ max and treadmill speed ($km.hr^{-1}$).

Each sample by the computer to obtain the above information involved first determining an initial reading of the gasmeter via a 16 bit cascade incremental counter, accurate to 0.11. Then during the sampling period, the oxygen, carbon dioxide, inspired air temperature, heart rate and treadmill speed readings were accumulated sequentially. If a new breath had started the ventilatory frequency counter was incremented. Finally, when the sample time had elapsed, a final gasmeter reading was taken (Goslin et al 1984).

$\dot{V}O_2$ Max Test

During the $\dot{V}O_2$ max test, subjects wore running shorts and the shoes they would normally wear when running a marathon. Body mass recorded during anthropometric assessment was entered into the computer for determination of oxygen uptake ($ml.kg^{-1}.min^{-1}$). Barometric pressure corrected for brass was also entered, as was relative humidity obtained from wet and dry bulb thermometers.

The skin of the subject was prepared as previously described for heart rate monitoring and the belt and electrodes positioned. The subject was informed of the nature of the $\dot{V}O_2$ max protocol and then allowed time to habituate to treadmill running. When the subject appeared to be running normally and comfortably on the treadmill, the $\dot{V}O_2$ max test was started.

During the test a fan was used to cool the subject. The protocol used involved the subject starting running at 8 $km.hr^{-1}$ with the treadmill horizontal. Every 1.5 minutes the speed of the treadmill was increased by 1 $km.hr^{-1}$ by an automatic treadmill control which had been previously calibrated for speeds from 8 $km.hr^{-1}$ through to 17 $km.hr^{-1}$. Once 17 $km.hr^{-1}$ was reached, the speed was incremented manually via the control panel until a speed 2 $km.hr^{-1}$ faster than the subjects' best race speed was reached. At this point, the speed was kept constant and the grade increased by 1% every 60 seconds until the subject was unable to continue.

$\dot{V}O_2$ max was established using the following criteria:
Subjective exhaustion, confirmed by a plateau in oxygen uptake ($\dot{V}O_2$) where there was less than a 50 ml increase in $\dot{V}O_2$ from one sample to the next (Morehouse 1972), a decrease in $\dot{V}O_2$ after a further increment in power output, and a respiratory exchange ratio greater than one. Various physiological data were determined for a 25 second sample every 30 seconds at each speed during the test and recorded on the computer print-out. In addition, the number of steps per minute was recorded for each speed up to 17 $km.hr^{-1}$ by manually counting each time the foot of the subject made contact with the treadmill belt. The maximum heart rate attained during the test was regarded as the maximum heart rate of the subject. The formulae (Consolazio et al 1963 , Faulkner et al 1977) used to compute the various physiological data are provided in Appendices D and F.

Simulated Marathon

Each subject returned to the laboratory after a minimum of four hours rest in order to run a simulated marathon on the treadmill. The ventilatory threshold (VT) determined from the ventilatory equivalent for oxygen ($VE\text{-}O_2$) and ventilatory equivalent for carbon dioxide ($VE\text{-}CO_2$) during the submaximal portion of the $\dot{V}O_2$ max test was used as the basis for

the calculation of the speed at which the marathon was run. The VT was used as it is well documented that the speed at which a marathon can be run is similar to the speed corresponding to the VT of the runner at that time (Farrell et al 1979). If $\dot{V}O_2$ max was used as a basis for the determination of running speed, a runner with a high $\dot{V}O_2$ max may have had to run at a speed which was too fast for this training status at the time of testing. Based on the VT, however, each subject ran at an equivalent intensity of effort relative to their fitness at the time of testing, as VT is an indicator of the current capability of a runner.

The VT (in $km.hr^{-1}$ and $\dot{V}O_2$) of each subject was determined after the $\dot{V}O_2$ max test had been completed. This was done by visual inspection after plotting curves of the responses of pulmonary minute ventilation ($\dot{V}_I$), the F_EO_2, F_ECO_2, $VE-O_2$ and $VE-CO_2$ versus submaximal running speed. One of the criteria used to determine the point of the VT was a secondary sharp increase in $VE-O_2$ with a corresponding smaller increase in $VE-CO_2$ (Dawson and Pyke 1984). Figure 2 (Page 56) is an example of the determination of VT using the above variables. The $VE-O_2$ and $VE-CO_2$ were the most useful parameters to determine VT. During the early power outputs the $VE-O_2$ and $VE-CO_2$ decreased as the physiological dead space to tidal volume ratio decreased (Davis 1985). As the power output continued to increase, a point was reached where the $VE-O_2$ began to systematically increase. The $VE-CO_2$, however, remained stable for a few increases in power output beyond the point at which $VE-O_2$ started increasing before it also began to increase (Figure 2, page 56). This stable region was the area of isocapnic buffering of lactic acid (Davis 1985). The criteria for the determination of VT were therefore a secondary increase in $VE-O_2$ and a smaller increase in $VE-CO_2$. The point of the VT was confirmed by a secondary sharp increase in $\dot{V}_I$ and F_EO_2, and a decrease in F_ECO_2 (Figure 2). (Dawson and Pyke 1984).

Once the speed at which the VT occurred was determined, 92.5% of that speed was calculated, this being the speed at which the simulated marathon was run. This was chosen as it enabled most subjects to complete the full marathon distance, but left them feeling subjectively exhausted at the finish as in the case of a marathon race.

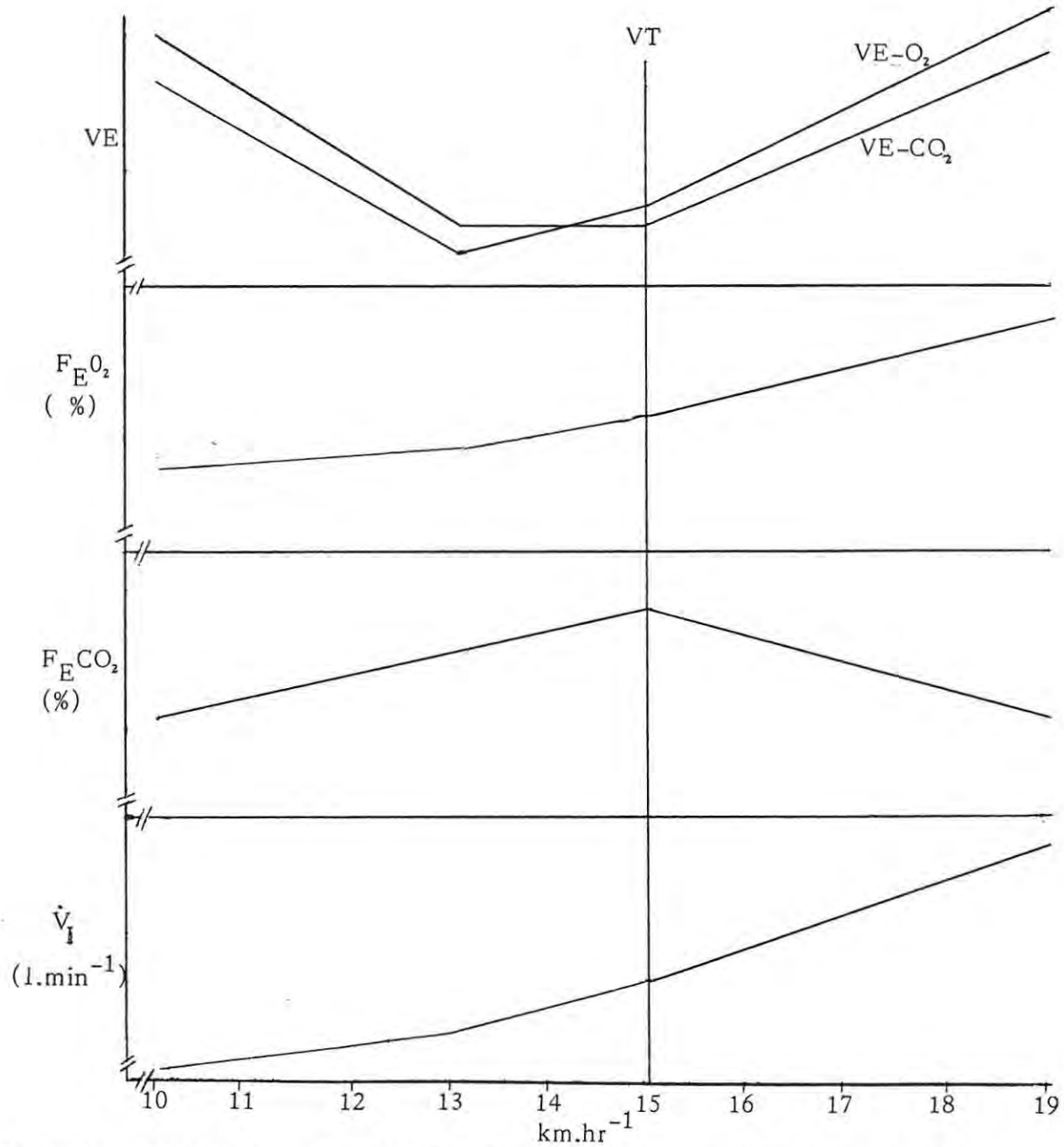

Figure 2 : Schematic representation of the procedure used to determine the VT by ventilatory measures.

Before the marathon run each subject was re-weighed, the heart rate monitoring system attached as for the $\dot{V}O_2$ max test, and YSI-400 series temperature measuring equipment also attached, as described below.

Temperature Measurement

The YSI model 408 surface skin temperature probes were used to measure pectoral and thigh surface temperature. It was found that the most suitable method for attachment of the pectoral temperature probe was by first placing a section of "Elastomesh" around the chest of the subject, attaching the probe to a loose mesh screen,and then placing this between the mesh "vest" and skin of the subject, so that the probe was in contact with the skin surface. The thigh temperature probe was held in place in a similar manner. A section of "Elastomesh" was placed around the thigh of the subject, with the probe placed between the skin and a loose mesh screen attached to the undersurface of the "Elastomesh". The "Elastomesh" was prevented from slipping down the leg of the subject by tying it to the subjects' running shorts. The screen and "Elastomesh" combination allowed free air movement around the probes. Core temperature was monitored by means of a YSI model 401 rectal temperature probe inserted approximately 12 cm.

Blood Analysis

The methods described by Wroblewski and La Due (1956) and Parrott (1968) were used for the determination of blood lactic acid and glucose respectively, details of which are presented in Appendix H.

Physiological Data

During the marathon, the same physiological variables were recorded as during the $\dot{V}O_2$ max test, using the data acquisition system previously described. A 60 second expired gas sample was analysed once every 10 minutes. At the same time the pectoral, thigh and rectal temperatures

were recorded and the step rate determined by counting the number of times the foot made contact with the treadmill belt (steps.minute^{-1}). In addition, the Rating of Perceived Exertion (RPE) was assessed for both "general" RPE as well as "local" (legs only) RPE, by use of the Borg RPE Scale (Borg 1973) (Appendix I and Appendix J). Before and during the run the subject was allowed to drink ad libitum of whatever replacement fluid he normally used during a marathon. This was usually a mixture of "Coca Cola" and water. The amount of liquid consumed was recorded, as was any urine voided.

As with the $\dot{V}O_2$ max test, a fan was used to aid cooling during the run. Subjects stopped either when 42.2 km had been completed or when too tired to continue. If exhaustion was the cause of the subject stopping, this was considered to be 100% running time. If the subject was forced to stop for reasons such as injury or lack of motivation, however, this was not counted as being 100% of running time and in these cases data were lost for the percentages of time not completed.

Statistical Treatment

The data obtained from each 10 minute interval during the marathon was categorised into 10% temporal intervals from 10 - 100% of total running time. This information was then used to determine if any differences existed between the black and white subject groups, if any changes occurred during the time-course of the marathon and if any interaction between subject groups occurred.

Data from the simulated marathon was analysed using a 2 x 10 ANOVA with repeated measures on one factor to test for significant differences between groups and during the time-course of the marathon. There were two levels of factor 1, a black and a white group of runners and 10 levels of factor 2, the temporal intervals of 10%, 20%, 30%, 40%, 50%, 60%, 70%,

80%, 90% and 100% of total running time. The Sheffé test was utilised for a post-hoc analysis on the ANOVAs in which significant differences were found to exist during the time-course of the marathon, except in some instances where this was too stringent for the repeated measures design, in which case the Tukey test was used. This determined where any significant differences between the time intervals occurred and was done by a comparison of means. The groups were also compared at 16 $km.hr^{-1}$ and at $\dot{V}O_2$ max by means of t-tests for independent samples. The submaximal speed of 16 $km.hr^{-1}$ was used for comparison as a wide range of physiological variables have been reported at this speed, and it was close to the average race speed of subjects participating in this study. Correlation analyses were computed for selected data (see "Correlation Analyses at end of this Chapter). A t-test for related samples was used in the case of the pre- to post-run blood analysis and a t-test for independent samples in the case of anthropometric and other between-group data comparisons (Cohen and Holiday 1979), details of which will be presented subsequently.

The level of significance chosen was 0.05 as this limited the probability of committing a Type I error, without greatly increasing the probability of committing a Type II error.

The statistical approach taken with the measured and derived data analysed is documented below:

1. Independent t-test (between groups)
   a) General: $\dot{V}O_2$ max; H/R max; VT; H/R at the VT; % H/R max at VT; VT as % $\dot{V}O_2$ max; race time; simulated marathon run time; estimated best race $\dot{V}O_2$; estimated % $\dot{V}O_2$ max in best race.
   b) At 16 $km.hr^{-1}$ and VO_2 max: $\dot{V}O_2$; $\dot{V}CO_2$; % $\dot{V}O_2$ max; % VT; H/R; % H/R max; R; estimated cardiac output ($\dot{Q}$); $\dot{V}_I$; alveolar ventilation ($\dot{V}_A$); tidal volume ($\bar{V}_T$); breathing frequency (f);

$VE\text{-}O_2$; $VE\text{-}CO_2$; step-length; step rate.

c) Simulated marathon: Energy utilised; carbohydrate utilised; fat utilised; sweat rate; body heat content; % mass loss; marathon performance quotient (MPQ = % $\dot{V}O_2$ max in race ÷ % $\dot{V}O_2$ max at 16 $km.hr^{-1}$); pre- and post-marathon blood lactic acid and glucose concentrations (white vs black).

d) Anthropometric data: body density; body volume; body surface area (BSA); BSA/volume ratio; mass; % fat; stature; trochanterion height; trochanterion height as % stature; step length as % stature; step length as % trochanterion height.

2. Related t-test

Pre- and post-simulated marathon blood lactic acid and glucose levels in both groups of runners.

3. ANOVA with repeated measures on one factor (percent running time)

$\dot{V}O_2$; $\dot{V}CO_2$; % $\dot{V}O_2$ max; H/R max; % VT; $\dot{V}_I$; $\dot{V}_A$; f; R; $VE\text{-}O_2$; $VE\text{-}C0_2$; stroke volume; $\dot{Q}$; one minute gross energy cost; % carbohydrate utilised; "general" RPE; "local" (leg) RPE; step rate; pectoral temperature; thigh temperature; rectal temperature; mean skin temperature; mean body temperature.

4. Correlation analyses

This was conducted on selected data where significant differences were shown to exist by either the t-tests or ANOVA's. These were stature and trochanterion height vs step rate; trochanterion height and step rate vs relative economy; $\dot{V}O_2$ max, % $\dot{V}O_2$ max, VT as % $\dot{V}O_2$ max, MPQ, and % $\dot{V}O_2$ max utilised when racing vs race time; % $\dot{V}O_2$ max at 16 $km.hr^{-1}$ vs VT as % $\dot{V}O_2$ max, % $\dot{V}O_2$ max utilised when racing, post-run blood lactic acid concentration, R and VT; VT as % $\dot{V}O_2$ max vs % $\dot{V}O_2$ max utilised when racing; $\dot{V}O_2$ max vs VT, $\dot{V}O_2$ at 16 $km.hr^{-1}$ and post-run blood lactic acid concentration; VT vs $\dot{V}O_2$ at 16 $km.hr^{-1}$, post-race blood lactic acid concentration and R values.

CHAPTER 4

RESULTS AND DISCUSSION

Cardiopulmonary variables

The subjects in the study were characterised by a high $\dot{V}O_2$ max (Table I , Page 62), there being no statistically significant difference between the black runners (60.4 $\pm$ 6.5 $ml.kg.min^{-1}$) and white runners (63.2 $\pm$ 2.9 $ml.kg^{-1}.min^{-1}$) ($p < 0.05$). As may be expected, these values are slightly lower than those reported from studies conducted on international class runners, where values ranged from 69.7 $ml.kg^{-1}.min^{-1}$ in the case of an international class runner with a best marathon time of 2 hours 8 minutes 33 seconds (Costill et al 1971a), to 71.3 - 84.4 $ml.kg^{-1}.min^{-1}$ in a group of international class marathon runners studied by Pollock (1977b). The black and white runners could not be separated using the $\dot{V}O_2$ max variable. This is not entirely unexpected as there was no statistically significant difference in race performances of the two groups (Table I), Costill et al (1973b) stating that factors other than $\dot{V}O_2$ max are needed to separate athletes of similar performance ability.

In the case of subject WM, the data at 95% running time was used as representative of 100% time as this subject did not run for the full 42.2 km, but was not exhausted when he stopped. He and subject GN did not complete the full 42.2 km as they were unwilling to continue the run and seemed to be unmotivated and uninterested. In the case of subject GN, regression analyses were performed on the data and the regression equation with the highest coefficient of determination was used to estimate data points at 90% and 100% running time. Subjects EM,EN, MN, MB, HE and RJ were unable to continue due to exhaustion. There was no single variable such as % $\dot{V}O_2$ max or blood lactic acid concentration which accounted for these subjects being unable to continue the marathon, although MN displayed symptoms of hypoglycaemia. Unfortunately, he would not consent to a blood sample being drawn. Subject VK was unable to run for longer than 40 minutes

Table I: Characteristics of subjects

Category	Subject	Stature (cm)	Mass (kg)	$\dot{V}O_2$ max ($ml.kg.min^{-1}$)	Best Marathon Speed ($km.hr^{-1}$)	**Sim. Marathon Speed ($km.hr^{-1}$)	VT as % $\dot{V}O_2$ max	Time to 42.2 km/ exhaustion (mins)	***Sim. Mar. Dist. Completed (km)
BLACK (n = 9)	TG	166.4	57.6	54.2	16.05	14.07	81.7	180	42.2
	GN	162.1	59.1	61.1	17.09	14.40	86.7	140	33.6
	CM	173.0	56.1	59.8	16.05	13.88	70.0	182	42.2
	EM	181.3	67.2	49.2	15.75	12.98	78.3	160	34.6
	EN	168.6	59.9	62.7	17.22	15.23	91.4	160	40.6
	MN	172.3	57.6	70.0	16.68	14.81	74.6	140	34.6
	WM	174.3	53.3	66.6	18.92	16.88	80.6	140	39.4
	MB	164.7	60.2	64.8	17.22	15.75	93.2	105	27.6
	VK	177.2	64.9	56.0	16.23	14.31	87.5	-	-
	$\bar{X}$	171.1	59.5	60.4	16.75	14.70	82.7	151	36.9
	SD	6.2	4.3	6.5	0.91	1.08	7.7	23.6	4.8
WHITE (n = 10)	SG	189.7	83.7	63.4	16.55	13.84	70.7	182	42.2
	RB	184.4	66.4	61.6	15.46	14.07	76.9	180	42.2
	RS	179.9	71.0	64.1	16.68	13.61	71.6	186	42.2
	HE	181.7	64.0	64.7	16.17	12.98	68.2	170	36.8
	CG	164.9	56.7	64.1	17.09	14.81	78.0	171	42.2
	BE	181.1	81.4	60.1	15.81	13.88	91.3	181	42.2
	RJ	186.6	75.2	62.4	16.55	13.88	74.7	140	32.4
	KS	178.9	72.3	65.9	18.11	15.75	77.2	160	42.2
	SB	187.4	77.1	57.8	15.51	13.19	75.6	192	42.2
	AB	177.8	63.1	67.9	17.81	15.75	75.3	160	42.2
	$\bar{X}$	180.7	71.1	63.2	16.57	14.18	75.6	172	40.7
	SD	6.4	8.6	2.9	0.85	0.96	6.2	14.7	3.2

* $p < 0.05$
** Simulated
*** Simulated Marathon Distance

due to a muscle injury. Compared with white subjects fewer black subjects completed the full 42.2 km (Table I , Page 62). This may be due to these subjects being at a higher % $\dot{V}O_2$ max ($p < 0.05$) during the simulated marathon than the white runners (Figure 3, Page 64) . The black runners were, however, at the same % VT as the white subjects (92.3 $\pm$ 5.9% and 91.1 $\pm$ 3.1%respectively), and had lower post-marathon blood lactic acid levels than the whites (Table II, Page 65).

The correlation between $\dot{V}O_2$ max and marathon race time in the group of runners studied was 0.86 in the case of the black runners and 0.85 in the white group. This was lower than the correlation of 0.91 found to exist between $\dot{V}O_2$ max and running success in a cross-section of elite and non-elite runners (Costill et al 1973b),but higher than the correlation ($r = 0.08$) reported within an elite group of runners between $\dot{V}O_2$ max and performance (Costill 1967). This difference may have resulted from the runners in the present study having a wider range in race times (speed) and $\dot{V}O_2$ max values (Table I , Page 62) than the group studied by Costill (1967). A lower correlation between $\dot{V}O_2$ max and race performance may have been obtained in the present study had the criteria for subject selection been more stringent, as this would have resulted in a smaller range in race times, and a more homogenous group. The low correlation found by Costill et al (1973b) does not suggest that $\dot{V}O_2$ max is unimportant in running success, as all subjects exhibited high values. These data are in agreement with Conley and Krahenbuhl (1980) who also found a low correlation between $\dot{V}O_2$ max and running peformance within a group with similar performance ability. They suggested that a high $\dot{V}O_2$ max was responsible for each subject gaining membership to the elite group, but did not discriminate between runners within the group.

The submaximal oxygen uptake ($\dot{V}O_2$) at any given running speed (running

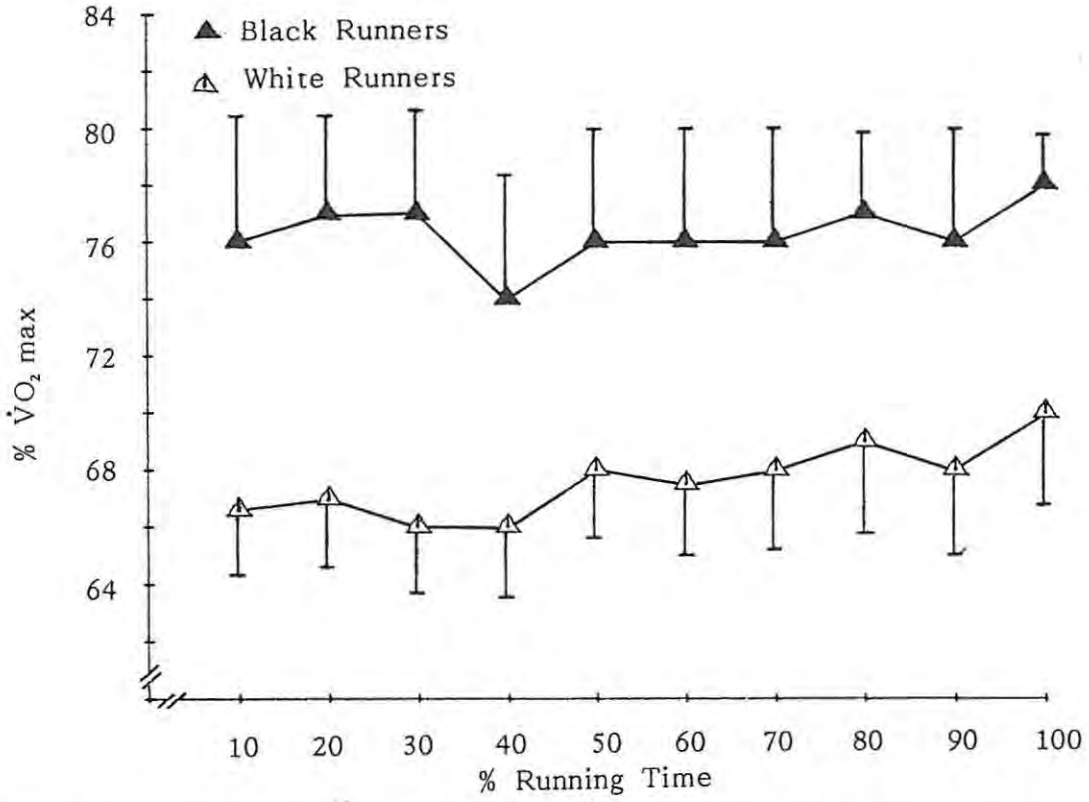

Figure 3 : Black runners were at a significantly ($p < 0.05$) higher % $\dot{V}O_2$ max than white runners during a simulated marathon. The upward drift in % $\dot{V}O_2$ max during the time-course of the marathon was not significant.

Table II: Pre- and post-marathon blood lactic acid and glucose concentration ($mmol.l^{-1}$)

CATEGORY		Pre-run Lactic Acid	Post-run Lactic Acid	Pre-run Glucose	Post-run Glucose
Black (n=9)	$\bar{X}$	1.02	1.30	10.8	9.7
	SD	0.08	0.26	0.3	1.5
White (n=10)	$\bar{X}$	1.01	1.59	10.4	10.9
	SD	0.09	0.20	0.6	0.9

*

* Post-marathon blood lactic acid significantly ($p < 0.05$) lower in the blacks.

Table III: Descriptive statistics of submaximal performance characteristics.

CATEGORY		VT ($ml.kg^{-1}.min^{-1}$)	VT as % $\dot{V}O_2$ max	% $\dot{V}O_2$ max in Best Race	% $\dot{V}O_2$ max in Sim** Marathon	MPQ	Gross Energy Cost ($KJ.kg^{-1}$)
Black (n=9)	$\bar{X}$	50.0	82.7	89.0	76.0	1.06	146
	SD	7.3	7.7	5.5	7.3	0.10	24
White (n=10)	$\bar{X}$	47.2	75.6	81.5	67.5	1.04	152
	SD	2.7	6.2	3.1	5.3	0.06	14

* *

* $p < 0.05$
** Simulated

economy) varies considerably amongst runners (Costill et al 1973b, Daniels 1974, Pollock 1977b). The $\dot{V}O_2$ at 16 km.hr^{-1} of the black runners was 49.9 $\pm$ 1.7 ml.kg^{-1}.min^{-1} and that of the white runners 51.0 $\pm$ 5.3 ml.kg^{-1}.min^{-1}, but this was not a significant difference ($p < 0.05$) (Table IV, Page 67). In a study by Leary and Wyndham (1965) it was observed that black athletes could exercise at a higher power output than whites at any given $\dot{V}O_2$. This implies that at the same power output black athletes should have a lower $\dot{V}O_2$ than whites. Data presented from the current study shows that this was not the case in the athletes tested. Considerable variation was observed in $\dot{V}O_2$ at 16 km.hr^{-1} with values ranging from 47.7 - 53.6 ml.kg^{-1}. min^{-1} in the case of the white runners and from 43.9 - 61.1 ml.kg^{-1}. min^{-1} in the black group. Correlation analysis revealed that no significant relationship existed between $\dot{V}O_2$ at 16 km.hr^{-1} and $\dot{V}O_2$ max which agrees with the results of Farrell et al (1979). Correlation analysis also revealed that no significant relationship existed between $\dot{V}O_2$ at 16 km.hr^{-1} and race performance in either group. This is in agreement with Costill et al (1973b) who found that at 16 km.hr^{-1} the $\dot{V}O_2$ of a group of runners studied ranged from 48.4 - 54.8 ml.kg^{-1}. min^{-1}, with no relationship to running performance being apparent. The $\dot{V}O_2$ at selected submaximal speeds was stated as being of little value in differentiating distance running ability. Similar results were obtained by Farrell et al (1979) who also found no relationship between $\dot{V}O_2$ at race speed and performance. The results of the present study do not agree with data obtained by Conley and Krahenbuhl (1980) who found a high correlation ($r = 0.82$) between $\dot{V}O_2$ at 16 km.hr^{-1} and performance in a 10 km race among runners of comparable ability. It should be noted that the distance was 10 km and not a marathon in the case of the Conley and Krahenbuhl (1980) study, and that these authors were dealing with a group of runners who were more homogenous as regards race time than the subjects in the current study.

Table IV: Descriptive statistics of variables not significantly different between groups at 16 km.hr^{-1}. ($p < 0.05$).

CATEGORY		% $\dot{V}O_2$ max	$\dot{V}O_2$ (ml.kg.$^{-1}$min^{-1})	$\dot{V}CO_2$ (ml.kg.$^{-1}$min^{-1})	H/R (b.min^{-1})	% H/R max	%VT	$\dot{Q}$ (ml.kg.min^{-1})	$\dot{V}_I$ (l.min^{-1})	$\dot{V}_I$ (l.kg.$^{-\frac{2}{3}}$min.$^{-1}$)	$\dot{V}_A$ (l.min^{-1})	$\dot{V}_A$ (l.kg.$^{-\frac{2}{3}}$min.$^{-1}$)	Step Rate (Steps.min^{-1})
Black (n=9)	$\bar{X}$	84.8	49.9	52.6	167	88.9	103	331.0	77.63	5.09	68.79	4.51	180
	SD	9.1	1.7	6.9	8	5.6	11	27.6	13.87	0.83	12.55	0.79	6
White (n=10)	$\bar{X}$	78.6	51.0	48.9	164	89.6	106	325.7	76.33	4.46	69.64	4.07	182
	SD	5.8	5.3	2.2	8	5.2	6	8.6	8.88	0.42	8.82	0.38	8

Table V: Descriptive statistics of variables significantly ($p < 0.05$) different between groups at 16 km.hr.$^{-1}$

CATEGORY		R. value	$\bar{V}_T$ (l.breath^{-1})	$\dot{V}_{T/kg}$ (ml.kg.$^{-1}$breath^{-1})	f (breaths.min^{-1})	Step Length as % Stature(16km.hr^{-1})
Black (n=9)	$\bar{X}$	1.03	1.33	87	59	80.8
	SD	0.07	0.16	9	12	2.8
White (n=10)	$\bar{X}$	0.98	1.75	101	45	86.9
	SD	0.03	0.36	14	8	1.9

Although the $\dot{V}O_2$ at submaximal speeds may possibly not differentiate distance running ability, Costill et al (1973b) states that when considered as % $\dot{V}O_2$ max, $\dot{V}O_2$ at 16 km.hr^{-1} is highly related to running performance and the existence of a high correlation between these two variables ($r = 0.94$) was shown. In the current study there was no difference between the groups in $\dot{V}O_2$ at 16 km.hr^{-1}, but the % $\dot{V}O_2$ max utilised at 16 km.hr^{-1} tended to be higher in the black runners (84.8 $\pm$ 9.1 %) than the white runners (78.6 $\pm$ 5.8 %) (Table IV, Page 67). This difference was not found to be statistically significant. In a study conducted by Pollock (1977b) it would found that at a speed of 16 km.hr^{-1}, international class runners utilised 71.5% $\dot{V}O_2$ max and elite runners 80.9% $\dot{V}O_2$ max, which is in agreement with the % $\dot{V}O_2$ max utilised by the white runners in the current study. In the case of the white runners, a low correlation ($r = 0.68$) was found to exist between % $\dot{V}O_2$ max utilised at 16 km.hr^{-1} and best marathon race time ($p < 0.05$) but in the case of the black runners, no significant correlation existed between these two variables. The results of the present study thus differ from those of Costill et al (1973b), possibly due to the different performance ability of the runners tested.

The % $\dot{V}O_2$ max utilised during the fastest marathon completed by each subject in the current study was estimated from the $\dot{V}O_2$ of each subject while running at the speed of his best marathon on the treadmill. The mean estimated $\dot{V}O_2$ during a race was 53.8 $\pm$ 6.0 ml.kg^{-1}.min^{-1} in the case of the black runners, and 51.5 $\pm$ 2.5 ml.kg.$^{-1}$min^{-1} in the case of the white runners, the difference not being statistically significant. The % $\dot{V}O_2$ max utilised at race speed was, however, significantly ($p < 0.05$) higher in the black runners (89.0 $\pm$ 5.5%)than the white runners (81.5 $\pm$ 3.1%),(Table III , Page 65) although the race times of the two groups were not statistically ($p < 0.05$) different. The mean race time of the white runners was 2 hours 33 minutes and the blacks 2 hours 31 minutes. Correlation analysis showed that in the case of the white subjects a significant relationship existed between

the % $\dot{V}O_2$ max utilised at 16 $km.hr^{-1}$ and the % $\dot{V}O_2$ max utilised during the subjects' fastest marathon race ($r = 0.70$). There was no significant relationship between the same variables in the case of the black subjects.

Although the value of 81.5% $\dot{V}O_2$ max utilised during a race has been previously reported (Costill et al 1971a) the value of 89.0% $\dot{V}O_2$ max in the case of the black subjects is higher than normal (Table III , Page 65). Costill et al (1973b) suggests that the ability to run at a high % $\dot{V}O_2$ max during competition is an advantage, while Farrell et al (1979) are of the opinion that the % $\dot{V}O_2$ max that can be utilised during a marathon may be determined by the point at which muscle and blood lactic acid begin to accumulate exponentially.

In a study by Costill et al (1971a) the following equation was derived:

$$MPQ = \frac{\%\ \dot{V}O_2 \text{ max during a marathon race}}{\%\ \dot{V}O_2 \text{ max at } 16\ km.hr^{-1}}$$

where MPQ = marathon performance quotient. Better marathon runners tend to have a higher MPQ. Whereas the average MPQ for a group of marathon runners in the Costill study was 0.97, that of a runner with a marathon time of 2 hours 8 minutes 33 seconds was 1.27. It was found by these authors that a high correlation ($r = 0.94$) existed between MPQ and marathon race performance. The MPQ of each subject in the present study was calculated. It was determined that no significant difference existed in MPQ between the black and white groups, (Table III), but that the MPQ of the runners (1.06 ± 0.10 and 1.04 ± 0.06 respectively) was above the value of 0.97 reported by Costill et al (1971a). Whereas the correlation between MPQ and marathon race performance in the study by Costill et al (1971a) was 0.94, in the present study the correlation was 0.86 in the case of the black runners and 0.92 in the case of the white runners.

The black and white groups of runners studied had similar marathon race times and $\dot{V}O_2$ max values, (Table I , Page 62), a similar $\dot{V}O_2$ at 16 $km.hr^{-1}$ (Table IV, Page 67) and similar MPQ's (Table III , Page 65). In addition, the black and white runners had similar maximum heart rates (186 $\pm$ 6 and 183 $\pm$ 5 $b.min^{-1}$ respectively) (Table VI, Page 72). Pollock (1977b) in a study on international and elite athletes found that at 16 $km.hr^{-1}$ international class runners had a lower heart rate than elite runners. Cieslik et al (1984) found the maximal heart rate of black subjects to be higher than whites. This trend was not found in the present study, which may be due to the subjects being more highly trained than those in the study by Cieslik et al (1984).

It is apparent that the variables of $\dot{V}O_2$ max, submaximal $\dot{V}O_2$, % $\dot{V}O_2$ max utilised, H/R and MPQ, which have been suggested as capable of discriminating between athletes with similar race times, did not distinguish between the present black and white groups of runners.

A high correlation ($r = 0.98$) between the ventilatory threshold (VT) and running speed in a marathon has been demonstrated by Farrell et al (1979). A significant ($p < 0.05$) relationship was found to exist between $\dot{V}O_2$ max and VT in both the black and white groups , a relationship which was stronger in the black group of runners ($r = 0.84$) than the white group ($r = 0.60$). This suggests that a high $\dot{V}O_2$ max contributes to a higher VT. Runners with a higher $\dot{V}O_2$ max would thus be at an advantage due to a higher running velocity corresponding to the VT. No difference in $\dot{V}O_2$ max or VT existed between the blacks and whites. Although Sjödin et al (1979) suggest that a low running economy is responsible for a lower velocity corresponding to the VT, the data from the current study do not support this contention as it was found that no significant relationship existed between the $\dot{V}O_2$ at 16 $km.hr^{-1}$ and the VT. This finding is supported by data from

Table VI: Descriptive statistics of variables not significantly different between groups at $\dot{V}O_2$ max ($p < 0.05$).

CATEGORY		$\dot{V}O_2$ max ($ml.kg.min^{-1}$)	H/R max ($b.min^{-1}$)	$\dot{Q}$ ($ml.kg.^{-1}min^{-1}$)	R value	f ($breaths.min^{-1}$)
Black (n=9)	$\bar{X}$	60.4	186	381.6	1.13	60
	SD	6.5	6	33.3	0.11	6
White (n=10)	$\bar{X}$	63.2	183	394.9	1.12	57
	SD	2.9	5	15.1	0.04	8

Table VII: Descriptive statistics of variables significantly ($p < 0.05$) different between groups at $\dot{V}O_2$ max.

CATEGORY		$\dot{V}_I$ ($l.min^{-1}$)	$\dot{V}_I$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	$\dot{V}_A$ ($l.min^{-1}$)	$\dot{V}_A$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	$\dot{Q}$ ($l.min^{-1}$)	$\bar{V}_T$ ($l.breath^{-1}$)	$\bar{V}_T$ ($ml.kg.^{-\frac{2}{3}}breath^{-1}$)
Black (n=9)	$\bar{X}$	94.63	6.22	85.58	5.62	22.6	1.54	101
	SD	7.31	0.54	6.68	0.48	1.5	0.13	8
White (n=10)	$\bar{X}$	116.44	6.82	107.78	6.31	28.0	2.04	119
	SD	11.47	0.66	11.0	0.59	3.0	0.27	11

the study by Farrell et al (1979) in which it was also shown that no significant relationship existed between these two variables. It is interesting to note that there was also no significant relationship between $\dot{V}O_2$ at 16 $km.hr^{-1}$ and $\dot{V}O_2$ max in both the present study and the study by Farrell et al (1979).

Since lactic acid production is a function of the % $\dot{V}O_2$ max being utilised (Costill et al 1973b), it is possible that lactic acid production and the ability to utilise a large % $\dot{V}O_2$ max are related. This relationship suggests that runners who have a low blood lactate concentration when running at approximately 85 % $\dot{V}O_2$ max are able to utilise such a large % $\dot{V}O_2$ max during prolonged endurance activity because of the low blood lactic acid levels (Costill et al 1973b). Therefore, the black group of subjects who were exercising at a higher % $\dot{V}O_2$ max during their best race, (Table III , Page 65) may have been able to utilise such a large % $\dot{V}O_2$ max because of low blood lactic acid levels despite the high % $\dot{V}O_2$ max employed. This contention is supported by the lower blood lactic acid levels of the black runners (Table II, Page 65) at the finish of the simulated marathon, despite having run at a higher % $\dot{V}O_2$ max (Figure 3, Page 64). This will be addressed subsequently.

Examination of the VT showed that this occurred at a higher % $\dot{V}O_2$ max (Table III, Page 65) in the case of the black subjects (82.7 $\pm$ 7.7%) than the white subjects (75.6 $\pm$ 6.2%) ($p < 0.05$) which may enable the black runners to utilise a greater % $\dot{V}O_2$max during a race before blood lactic acid accumulation. During the simulated marathon run on the treadmill, the black and white groups ran at a statistically similar speed (table I , Page 62), $\dot{V}O_2$ (Figure 4, Page 74), VT, VT as a % of $\dot{V}O_2$ max (Table III) and a % H/R max (Figure 5, Page 75), but the black runners were at a higher % $\dot{V}O_2$ max(76.0 $\pm$ 7.3) than the white runners (67.5 $\pm$ 5.3) as shown

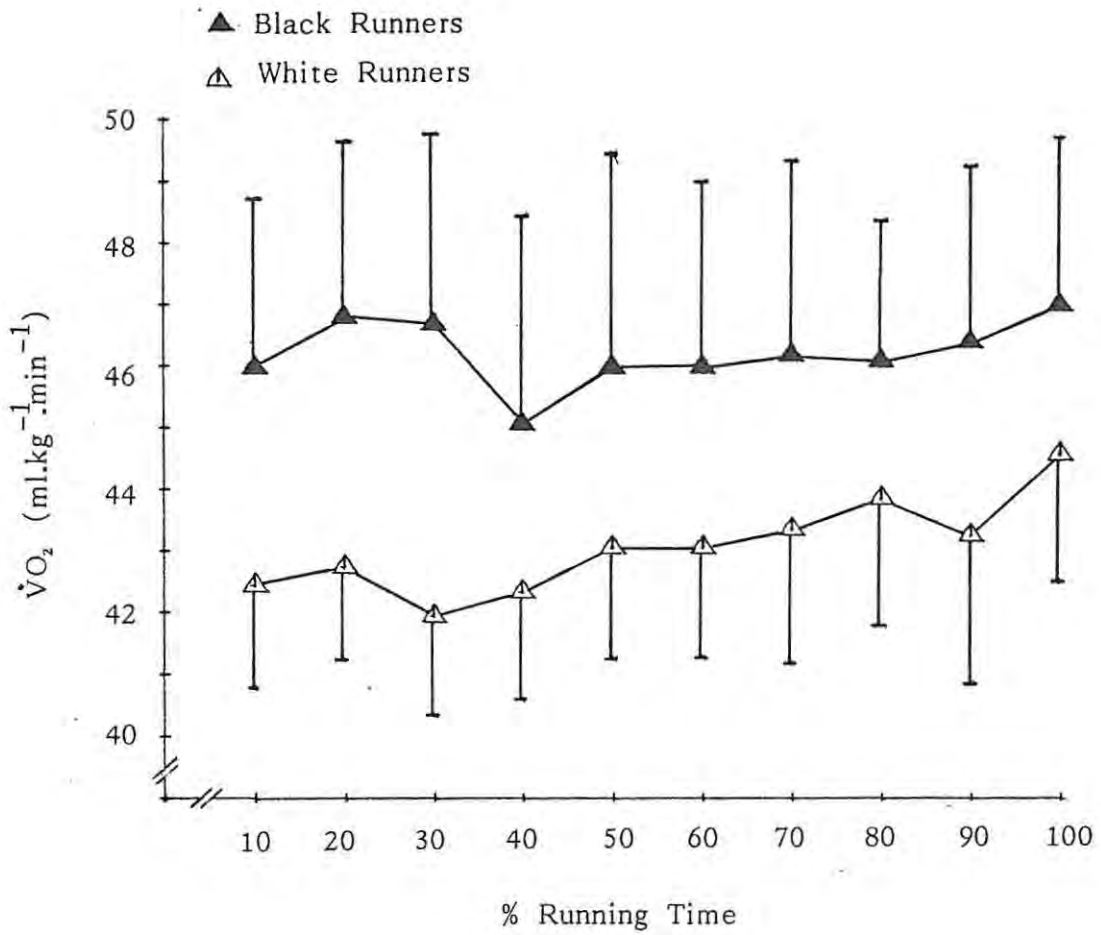

Figure 4 : No significant differences existed in $\dot{V}O_2$ between the black and white runners during a simulated marathon. The upward drift in $\dot{V}O_2$ during the time-course of the marathon was significant only between the mean at 40% running time and 100% running time. ($p < 0.05$).

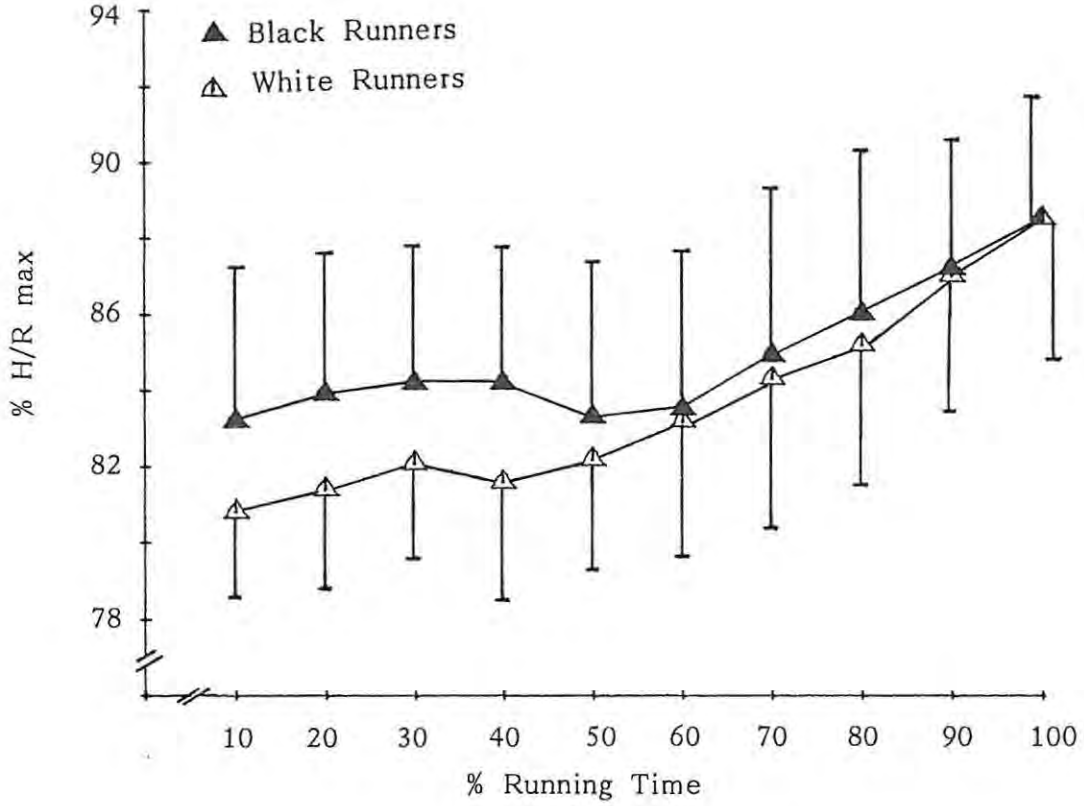

Figure 5: No significant differences existed in % H/R max between the black and white runners during a simulated marathon. The increase in % H/R max during the time-course of the marathon was significant from 70% running time.
Other significant differences were: 10% < 70-100%;
10-50% < 80-100%; 10-60% < 90-100%; 10-80% < 100%. ($p < 0.05$)

in Figure 3 (Page 64) ($p < 0.05$). The difference in % $\dot{V}O_2$ max utilised between the best race and the simulated marathons, however, was 13% in the case of the black runners and 14% in the whites (Table III , Page 65). Despite the similarity in all these variables (except for the % $\dot{V}O_2$ max), as was pointed out previously at the conclusion of the simulated marathon run the black subjects had significantly ($p < 0.05$) lower blood lactic acid levels at 1.30 ± 0.26 mmol.l^{-1} (11.7 mg%) than the white runners at 1.59 ± 0.20 mmol.l^{-1} (14.3 mg%) (Table II , Page 65). These values are similar to those reported by Costill and Fox (1969) and Costill (1970) for marathoners running at a similar % $\dot{V}O_2$ max as those in the present study. The lower values in the black subjects who were at a higher % $\dot{V}O_2$ max is contrary to what is expected, as it is well documented (Costill et al 1973b) that the higher the % $\dot{V}O_2$ max utilised, the greater the resulting blood lactic acid concentration, a response which was also found in the present study within the white group of runners ($r = 0.70$). It might be argued that the lower blood lactic acid concentration of the black runners was related to these subjects running for a 150.9 minutes compared to the 172.2 minutes of the whites (Table I , Page 62), a difference of just over 21 minutes. It is unlikely that the shorter running time of the black runners would have resulted in the lower blood lactic acid levels observed as Costill (1970) reports data which shows a plateau in blood lactic acid concentration after approximately 90 minutes of running in subjects running at a similar % $\dot{V}O_2$ max as those in the current study. Åstrand and Rodahl (1977) also present data which shows a plateau in blood lactic acid concentration during prolonged, submaximal exercise performed at a similar % $\dot{V}O_2$ max. Within the group of white runners a correlation of 0.70 existed between the % $\dot{V}O_2$ max utilised during the simulated marathon and post-marathon blood lactic acid concentration. In the black group, however, no significant relationship existed between these two variables ($p < 0.05$), suggesting that the % $\dot{V}O_2$ max utilised by the black runners did not have much effect

on the amount of lactic acid present in the blood. These findings are particularly interesting due to the high % $\dot{V}O_2$ max (89.0 $\pm$ 5.5%) utilised by the black group of runners during their best marathon races (Table III , Page 65), and the lack of a significant correlation in the black group between the % $\dot{V}O_2$ max utilised at 16 $km.hr^{-1}$ and marathon race times. An explanation of these observations may be related to either the buffering reaction which occurs between sodium bicarbonate in the blood and any accumulating blood lactic acid (MacDougall 1977) or, more likely, the "lactate shuttle" described by Brooks (1985b). If black runners possess a superior capacity for either of these systems than white runners, the lactate/lactic acid levels of the black subjects would be lower at any given speed, ultimately allowing a greater % $\dot{V}O_2$ max to be utilised. The buffering and "lactate shuttle" systems will be addressed in greater detail following discussion of the results obtained for the respiratory exchange ratios (R), as these add support to subsequent discussion of the possible role of these systems in maintaining lower lactic acid levels in the black runners.

Examination of the R values of the two groups revealed that a decrease occurred during the time-course of the marathon, confirming the progressive importance of fats as a fuel substrate during marathon running. Although the mean blood lactic acid concentration at the end of the simulated marathon was lower in the black runners (Table II , Page 65), the mean R R value was significantly ($p < 0.05$) higher in the black group (Figure 6, Page 78). It may be argued that this was due to the black runners being at a higher % $\dot{V}O_2$ max during the simulated marathon (Figure 3, Page 64), but when the mean R value of the two groups were examined at 16 $km.hr^{-1}$, it was observed that the mean value of the black runners (1.03 ± 0.07) was significantly ($p < 0.05$) higher than that of the white runners (0.98 ± 0.03) (Table V , Page 68). At this speed the % $\dot{V}O_2$ max

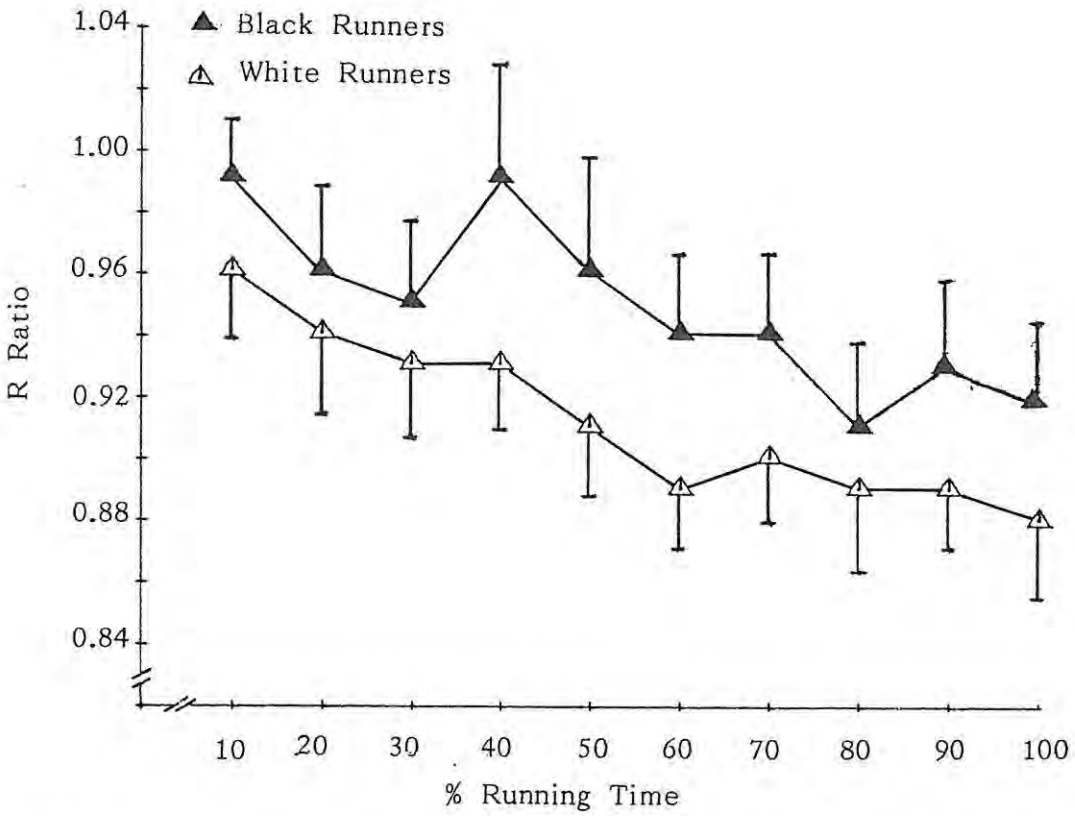

Figure 6: Black runners had significantly higher R values than white runners during a simulated marathon. The respiratory exchange ratio during the time-course of the marathon decreased so that the values from 60-100% running time were significantly less than the 10% value. The 40% values were significantly greater than 80-100%. ($p < 0.05$).

utilised by the black runners was not statistically different from that of the white runners (Table IV, Page 67), ruling out this factor as being responsible for the higher R value in the black runners at 16 $km.hr^{-1}$. This suggests that the higher % $\dot{V}O_2$ max utilised by the blacks during the simulated marathon was not related to the higher R values obtained. The $\dot{V}O_2$ at 16$km.hr^{-1}$ was also similar in the two groups (Table IV , Page 67), as was the percent of the VT at which the groups were running. Besides these variables, it should be reiterated that the VT and $\dot{V}O_2$ max were also similar. Thus, the higher R value observed in the black runners at 16 $km.hr^{-1}$was unexpected. Possible hyperventilation is an unlikely cause of the higher R in the black runners, since both the absolute and relative ($l.kg^{-\frac{2}{3}} min^{-1}$) alveolar minute ventilation ($\dot{V}_A$) and $\dot{V}_I$ at 16 $km.hr^{-1}$ (Table IV , Page 67) were not significantly different between the black and white groups. $\dot{V}_A$ was calculated as $\dot{V}_I$ does not always reflect the actual $\dot{V}_A$. If the $\bar{V}_T$ is small and f is high, as opposed to a high $\bar{V}_T$ and low f, $\dot{V}_A$ is different due to more air from the dead-space being exchanged in the former situation. It is the $\dot{V}_A$ that determines the gaseous concentrations at the alveolar capillary membrane. (McArdle et al 1981). Once the $\dot{V}_A$ was calculated, this was divided by $(body\ mass)^{\frac{2}{3}}$ to account for differences in body size of the two groups, as Åstrand and Rodahl (1977) state that $\dot{V}_I$ and $\dot{V}_A$ are related to body size by both $(stature)^{2}$ and $(mass)^{\frac{2}{3}}$. The $\dot{V}_A$ was calculated by substracting the anatomic dead-space from the $\dot{V}_I$:

$$\dot{V}_A = \dot{V}_I - (0.15\ l \times f)$$ (McArdle et al 1981)

where: 0.15 l is the average anatomic dead-space (McArdle et al 1981) and f is the breathing frequency.

As with hyperventilation, the replacement drinks used during the marathon are unlikely to have been related to the higher R values, as higher values were also observed during the submaximal portion of the $\dot{V}O_2$ max tests, during which no fluids were ingested. Further, during the simulated marathon,

the black runners tended to drink less carbohydrate containing drinks like "Coca Cola" than the white runners. Therefore, any influence this may have had on the R values would have been an increased R value in the white runners rather than the black runners. The higher R values may be related, however, to the diet of the subjects. If the black runners consumed a diet higher in carbohydrates than the whites, this may be partly responsible for the higher values in these runners.

The R value can increase as a result of buffering. Lactic acid (HLa) that begins to accumulate in the blood is buffered by the sodium bicarbonate system in order to maintain acid-base homeostasis (McDougall 1977). During this process, carbonic acid is formed which breaks down to CO_2 and water in the pulmonary capillaries and the CO_2 exits via the lungs. This buffering process thus adds additional non-metabolic CO_2 to that quantity normally released during energy metabolism. As R is the volume of CO_2 produced/volume of O_2 consumed, buffering will result in an increase in R. (Brooks and Fahey 1984) This process can be written in the form of chemical reactions:

$$HLA \rightarrow La^- + H^+ \text{ (lactic acid dissociates almost completely due low pK) (Davis 1985)}$$

$$La^- + H^+ + NaHCO_3 \rightarrow H_2CO_3 + NaLa \text{ (Buffering by } NaHCO_3\text{)}$$

$$\Downarrow$$

$$H_2O + CO_2 \text{ (non-metabolic } CO_2 \text{ produced).}$$

The data presented concerning the % $\dot{V}O_2$ max utilised at 16 $km.hr^{-1}$, during a simulated marathon, and when racing, as well as the blood lactic acid levels and R values of the black and white runners, suggests that some metabolic process is occurring which differs between the two groups as regards lactic acid clearance or production. A lower lactic acid production in the black runners is a possibility, but was not measured in this study. Lactic acid production may be reduced if a shift occurs from carbohydrate

towards FFA utilisation (Donovan and Brooks 1983). This is an unlikely explanation of the lower lactic acid levels of the black runners (Table II, Page 65) in the present study as the R value of these runners were higher than the values observed in the white group during the marathon (Figure 6, Page 78), indicating less FFA metabolism in the black runners. Another reason that lower lactic acid production is unlikely is related to findings by Donovan and Brooks (1983) that training improves lactic acid clearance, while production is unaffected.

Assuming that clearance of lactic acid by oxidation (which can account for 78% of lactic acid clearance) was equally effective in both the black and white groups, the blood lactic acid remaining after oxidation may have been reduced to a greater extent in the black than the white runners if black runners possess a particularly effective bicarbonate buffering system. McDougall (1977) states that as soon as lactic acid starts accumulating in the blood (which occurs at the OBLA during submaximal exercise) buffering of HLA occurs. Mazzeo et al (1986) reported changes in blood bicarbonate levels during the transition from rest to exercise. A 20% reduction in bicarbonate concentration occurred during 65 minutes of exercise at 75% $\dot{V}O_2$ max compared to rest, while during exercise for 140 minutes at 50% $\dot{V}O_2$ max a 7% reduction in bicarbonate concentration from rest was observed. Donovan and Brooks (1983) attribute some unexpected results in a study conducted by them to excess CO_2 as a result of bicarbonate buffering. In the Donovan and Brooks study, rats were running submaximally at 69.6% $\dot{V}0_2$ max and had R values of 0.93. It is therefore indicated that buffering of lactic acid does occur to a certain extent during submaximal exercise. Any increase in the rate or effectiveness of bicarbonate buffering in the black runners, besides reducing the blood lactic acid concentration, would also cause the production of non-metabolic $C0_2$ as previously shown. This would result in the higher

R values observed during both the submaximal (Table V , Page 68) and simulated marathon runs (Figure 6 , Page 78), as well as the lower blood lactic acid levels of the black runners at the end of the simulated marathon (Table II , Page 65).

In order to maintain the lower blood lactic acid levels observed at the end of the simulated marathon, the bicarbonate buffering system would have to be of adequate capacity. Mazzeo et al (1986) indicate that lactic acid clearance by methods other than oxidation is approximately 50 $mg.kg^{-1}.min^{-1}$ at 75% $\dot{V}O_2$ max. The mean mass of the black runners was 59.5 kg and running time 173 minutes, therefore approximately 8580 mg (95 mmol) of lactic acid would have been cleared during the time-course of the marathon by means other than oxidation. Åstrand and Rodahl (1977) indicate that the bicarbonate system can buffer approximately 170 mmol of a strong acid. The potential buffering capacity is therefore in excess of what would be required.

The recent work of Brooks (1985b) and the postulated "lactate shuttle" provides a second, and possibly more likely explanation of the lower blood lactic acid levels (Table II , Page 65) and higher R values observed in the black runners during both submaximal running (Table V , Page 68) and the marathon (Figure 6 , Page 78). The postulate of the "lactate shuttle" involves the following: Lactic acid produced by a muscle from the degradation of muscle glycogen (for example in FTb muscle fibres) during exercise is released from the site of glycogenolysis and transported through the interstitium and vasculature. It can then serve as a source of oxidisable substrate at other active muscles (for example the heart and ST muscle fibres) with high rates of cellular respiration (Figure 7 , Page 83). Some lactic acid released could be combusted by ST fibres without leaving the tissue, while that which escapes and is not oxidised at other sites could be

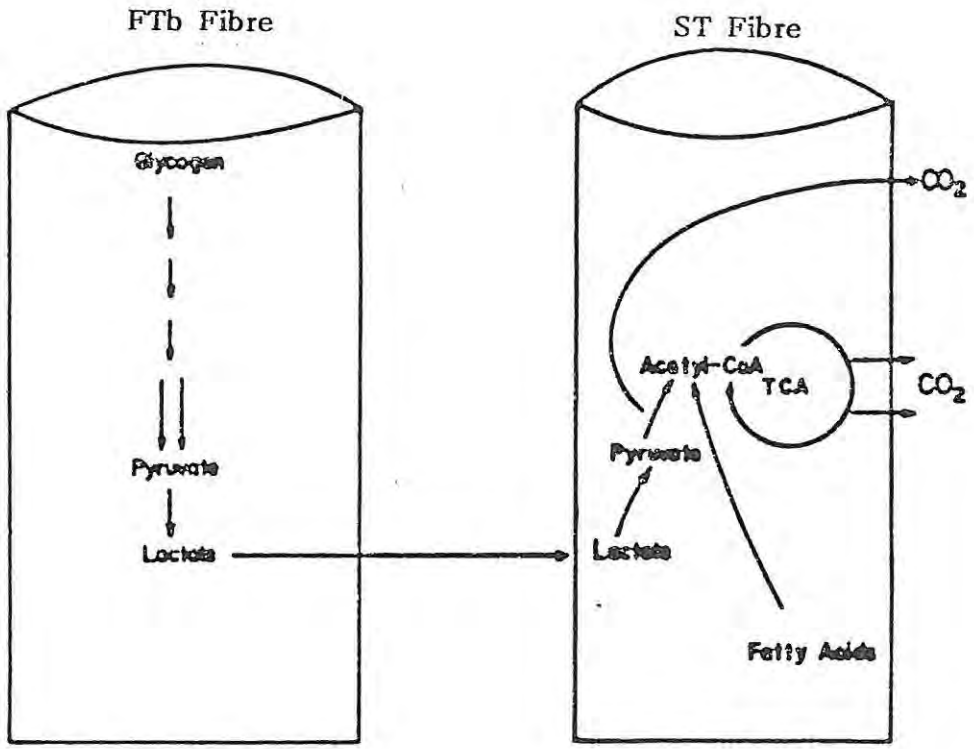

Figure 7: Lactic acid formed and released in FTb fibres when they are recruited is combusted by the ST fibres as part of the postulated "lactate shuttle". (Adapted from Brooks (1985b)).

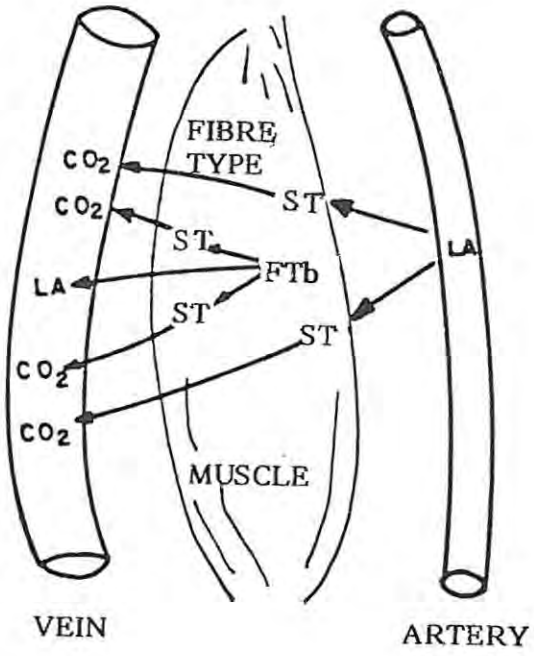

Figure 8: A contracting muscle can simultaneously release, consume and oxidise lactic acid as part of the postulated "lactate shuttle". Some lactic acid released from FTb fibres could be combusted by ST fibres without leaving the tissue, while some escapes and could be consumed on recirculation to the tissue. (Adapted from Brooks (1985b)).

consumed on recirculation to the tissue (Figure 8 , Page 84). Lactic acid release from muscle compares in importance with glucose release from the liver for supplying oxidisable substrate (Brooks 1985b). The reason for this statement is apparent when the rate of oxidation of lactic acid is expressed as a percentage of the total carbohydrate combusted. Oxidation accounts for 78% of the lactic acid turnover rate during exercise peformed at 75% $\dot{V}O_2$ max. At this intensity of exercise it appears that 11% of the total carbohydrate combusted passes through the lactate pool (Mazzeo et al 1986). If the "lactate shuttle" pathway occurred to a greater extent in the black runners than the whites, a greater percentage of lactic acid would have been oxidised in the case of the black runners, which may account for the lower blood lactic acid levels measured in these runners (Table II , Page 65). The percentage contribution by oxidation of lactic acid to the total carbohydrate utilisation would also have increased. This is substantiated by the R values (Table V , Page 68 ; Figure 6 , Page 78) which indicates that carbohydrate oxidation was higher in the black runners than the white runners. There was no difference, however, in the gross energy cost during the marathon ($KJ.kg^{-1}$) between the groups (Table III , Page 65). The observed higher R values may thus represent a true shift in substrate utilisation to greater carbohyrate oxidation by the black runners. Mazzeo et al(1986) attributed the decline that occurs in lactic acid clearance rate during hard exercise to blood flow redistribution away from the lactic acid removing areas of the kidney and liver. Therefore, if the redistribution of blood away from these organs is reduced in the black runners, a greater clearance of lactic acid may occur. Such an effect could be achieved by a decreased autonomic response in the black runners, resulting in less norepinephrine (a catecholamine) release from sympathetic nerve endings. Less vasoconstriction would therefore occur, the result of which would be a reduced shunting of blood away from the liver and kidney. Training results in a dampened catecholamine response (Brooks 1985b). Besides the effects described above, the

catecholamines may also result in less "overstimulation" of pyruvate production by glycogenolysis and glycolysis (Brooks 1985b). As pyruvate leads to lactic acid formation, lower catecholamine levels may be related to lower levels of circulating lactic acid (Brooks 1985b).

It might be expected that the trend, although not significant, of a higher $\dot{V}CO_2$ in the blacks (Table IV, Page 67) would result in a higher $\dot{V}_I$ in the black runners. This is because carbon dioxide provides a strong stimulus for an increase in pulmonary minute ventilation ($\dot{V}_I$) and alveolar minute ventilation ($\dot{V}_A$) (McArdle et al 1981, Davis 1985). Davis (1985) reports that H^+ from lactic acid in the blood is a major cause of the non-linear increase observed in $\dot{V}_I$ during exercise above the VT/OBLA, probably via a stimulation of the carotid bodies. Subjects who have had their carotid bodies surgically removed have a smaller increase in $\dot{V}_I$ during exercise above the VT/OBLA. The non-linear increase in $\dot{V}_I$ that does occur in these subjects is due to stimulation by the excess CO_2 from buffering of lactic acid (Davis 1985). It therefore appears that H^+ is responsible for most of the non-linear increase in $\dot{V}_I$. Although there was no significant difference in $\dot{V}_I$ between the black and white runners during submaximal running (Table IV, Figure 9, Page 87) and simulated marathon running (Figure 10, Page 88), at $\dot{V}O_2$ max (Figure 9, Page 87) the black runners had a significantly lower $\dot{V}_I$ (94.63 ± 7.31 l.min^{-1}) than the white runners (116.44 ± 11.47 l.min^{-1}) ($p < 0.05$). Although $\dot{V}_I$ is not a perfect correlate of $\dot{V}O_2$, it is well documented that $\dot{V}_I$ increases linearly with increases in $\dot{V}O_2$ and power output up to the point at which the VT/OBLA occurs. There is then a non-linear increase in $\dot{V}_I$ untill $\dot{V}O_2$ max is reached (McArdle et al 1981, Brooks 1985a, Davis 1985). Figure 9 (Page 87) shows that although such a non-linear increase in $\dot{V}_I$ occurred in the white runners, the increase remained closer

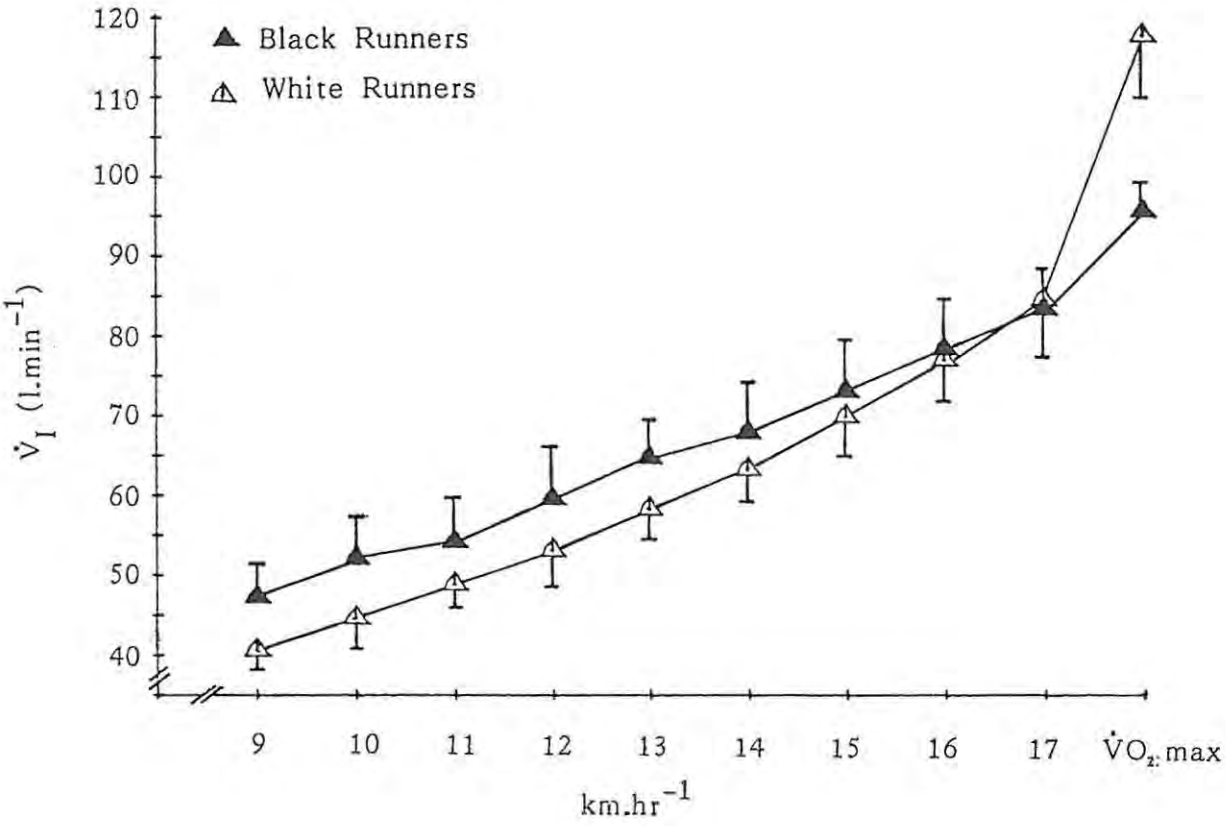

Figure 9 : No significant differences existed in $\dot{V}_I$ between the black and white runners during submaximal running up to 17 km.hr^{-1} ($p < 0.05$). At $\dot{V}O_2$ max the white runners had a significantly higher $\dot{V}_I$ than the black runners ($p < 0.05$).

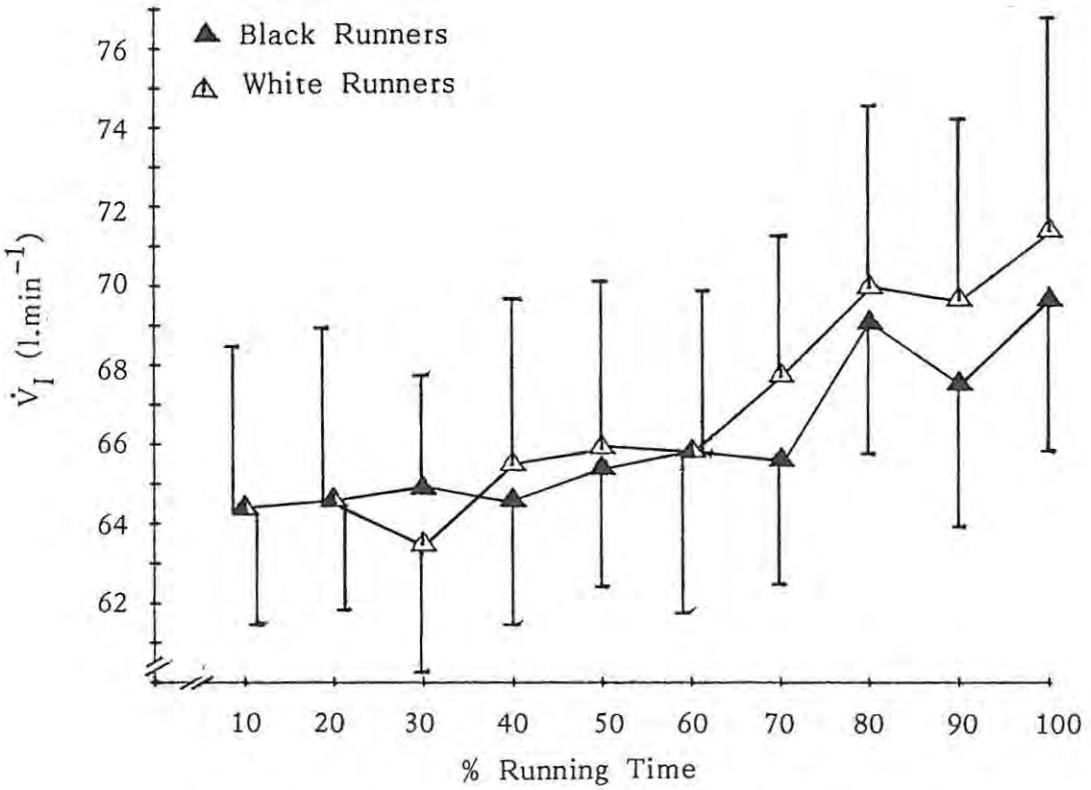

Figure 10: No significant differences existed in $\dot{V}_I$ between the black and white runners during a simulated marathon. The minute volume increased during the time-course of the marathon so that $\dot{V}_I$ from 70-100% running time was significantly greater than the 10% value. The values from 80-100% running time were significantly greater than those between 10-60% time.($p < 0.05$).

to linear in the black runners. It is possible to explain the smaller increase in $\dot{V}_I$ near $\dot{V}O_2$ max in the black runners using the information presented concerning the stimulatory effect of H^+ and CO_2 on $\dot{V}_I$. It was suggested previously that blood lactic acid levels may be lower in black runners. If this hypothesis is correct, the lower blood H^+ concentration in the black runners may result in a reduced ventilatory response during exercise above the VT/OBLA due to the reduced stimulatory effect from H^+. The $\dot{V}_I$ of the black runners did, however, show a small non-linear increase at $\dot{V}O_2$ max (Figure 9, Page 87). This may be due to the stimulatory effect of excess CO_2 from the buffering of lactic acid on $\dot{V}_I$.

It is possible that the differences observed in $\dot{V}_I$ between the groups may have been due entirely to differences in body size of the blacks and whites. The black runners were significantly shorter (171.1 $\pm$ 6.2 cm) than the white runners (180.7 $\pm$ 6.4 cm), while the mass of the black runners (59.5 $\pm$ 4.3 kg) was significantly less than that of the whites (71.1 $\pm$ 8.6 kg) (Table I, Page 62). Åstrand and Rodahl (1977) state that $\dot{V}_I$ is proportional to $(\text{stature})^2$ and $(\text{mass})^{\frac{2}{3}}$. In order to factor out differences in body size when considering $\dot{V}_I$, the absolute values ($l.min^{-1}$) were divided by $(\text{mass})^{\frac{2}{3}}$ and expressed as $l.kg^{-\frac{2}{3}}.min^{-1}$ (Table IV, Page 67). When this was done, however, the same similarities and differences existed between the groups in $\dot{V}_I$ as when this was expressed in absolute values.

The composition of the higher $\dot{V}_I$ in the white group of runners at the $\dot{V}O_2$ max can be examined. The $\dot{V}_I$ depends on the breathing frequency per minute (f) multiplied by the tidal volume ($\bar{V}_T$). An examination of the data showed that at $\dot{V}O_2$ max f was 60 $\pm$ 6 breaths.min^{-1} in the black subjects and 57 $\pm$ 8 breaths.min^{-1} in the white runners, a difference which was not statistically significant ($p < 0.05$) (Table VI, Page 72). These values are

similar to the 50-60 breaths .min^{-1} reported by De Vries (1980). Although f was similar, the $\bar{V}_T$ was significantly ($p < 0.05$) greater in the white subjects (2.04 $\pm$ 0.27 l.$breath^{-1}$) than in the blacks (1.54 $\pm$ 0.13 l.$breath^{-1}$), accounting for the higher $\dot{V}_I$ of the white runners (116.44 $\pm$ 11.47 l.min^{-1}) compared to the black runners (94.63 $\pm$ 7.31 l.min^{-1}) at $\dot{V}O_2$ max. When the $\bar{V}_T$ was expressed per (kilogram)$^{\frac{2}{3}}$ body mass to factor out differences in body size, it was found that the whites still had a significantly ($p < 0.05$) greater $\bar{V}_T$ (119 $\pm$ 11 ml.$kg^{-\frac{2}{3}}$.$breath^{-1}$) than the blacks who were found to have a $\bar{V}_T$ of 101 $\pm$ 8 ml.$kg^{-\frac{2}{3}}$.$breath^{-1}$ (Table VII, Page 72). The higher $\dot{V}_I$ of the white runners at $\dot{V}O_2$ max was achieved by a greater $\bar{V}_T$ in that group. It thus becomes necessary to explain the similar f of the two groups, but lower $\bar{V}_T$ of the black runners at VO_2 max. The similar f may be related to the mechanical properties of the breathing musculature, such as the time taken for elastic recoil and/or the high energy cost of breathing during maximal exercise, which may be 10% of the total amount of oxygen consumed (Åstrand and Rodahl 1977). An explanation for the lower $\bar{V}_T$ of the black runners at $\dot{V}O_2$ max (Table VII, Page 72) may be related to the smaller increase in $\dot{V}_I$ that occured at the $\dot{V}O_2$ max in the black runners. A lower $\dot{V}_I$ requirement would result in a lower $\bar{V}_T$ (as f was similar). The lower $\bar{V}_T$ may, however, be related to the body size of the runners. As the white runners were of greater mass and stature than the black group, the vital capacity (VC) of the whites would have been larger than that of the black runners, as VC is related to body size (Åstrand and Rodahl 1977). Each breath of the white runners would utilise X% of the VC. If the black group also utilised X% of their smaller VC, then this would dictate a smaller $\bar{V}_T$ in the case of the blacks. At 16 km.hr^{-1} the f of the black runners (59 $\pm$ 12 breaths.min^{-1}) was significantly ($p < 0.05$) higher than the whites (45 $\pm$ 8 breaths.min^{-1}) (Table V , Page 68).

The absolute $\bar{V}_T$ of the black runners at 16 km.hr^{-1} (1.33 $\pm$0.16 l.breath^{-1}) was significantly ($p < 0.05$) less than that of the white runners (1.75 $\pm$ 0.36 l.breath $^{-1}$) (Table V , Page 68). This difference also existed between the groups when differences in body size were accounted for. The smaller $\bar{V}_T$ of the black runners may be attributable to a smaller VC, as described previously. The $\dot{V}O_2$ required by both groups was the same at submaximal running speeds including the reference speed of 16 km.hr.$^{-1}$ (Table IV, Page 67), which may explain the similar $\dot{V}_I$ of both groups at submaximal speeds (Table IV , Page 67). With $\dot{V}_I$ the same in both groups, but a lower $\bar{V}_T$ in the black runners (Table IV), the f of the black runners would have to be higher than of the whites. The same responses were observed during the simulated marathon. Figure 11 (Page 92) shows that f was significantly ($p < 0.05$) higher in the black runners, while Figure 12 (Page 93) shows that $\bar{V}_T$ was significantly ($p < 0.05$) lower in the blacks during the simulated marathon. The $\dot{V}_I$, however,was the same in both groups(Figure 10 , Page 88). Summarising, it appears that during submaximal running (including the marathon), a lower $\bar{V}_T$ in the black runners, possibly due to their smaller body size, forces a higher f in order to attain a similar $\dot{V}_I$. These results are in agreement with Cerny et al (1982) who found that during cycle ergometry, American blacks had a higher f and lower $\bar{V}_T$ for any given $\dot{V}_I$ than did whites. Greater lung elastic recoil in the blacks was suggested as a possible reason by these authors.

Post-hoc analysis revealed that the increase in f became significant after 70% running time (Figure 11, Page 92) and the decrease in $\bar{V}_T$ after 60% running time (Figure 12, Page 93). A possible explanation is that fatigue of the respiratory musculature causes a decrease in $\bar{V}_T$, and thus forces the subsequent rise observed in f in order to maintain $\dot{V}_I$ during the time-course of the marathon (Figure 10, Page 88).

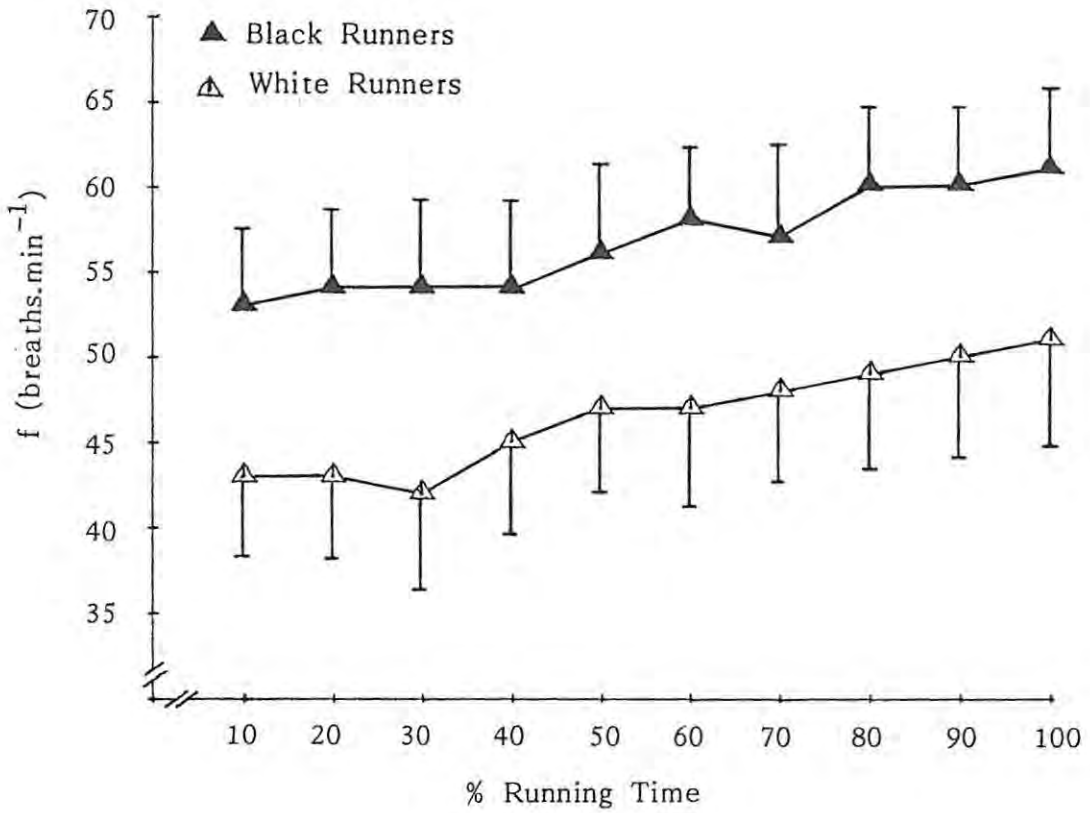

Figure 11: Black runners had a significantly higher f than white runners during a simulated marathon. Breathing frequency increased during the time-course of the marathon so that f from 70-100% running time was significantly higher than from 10-30%. The mean values of f between 80-100% running time was significantly greater than from 10-40% time. ($p < 0.05$).

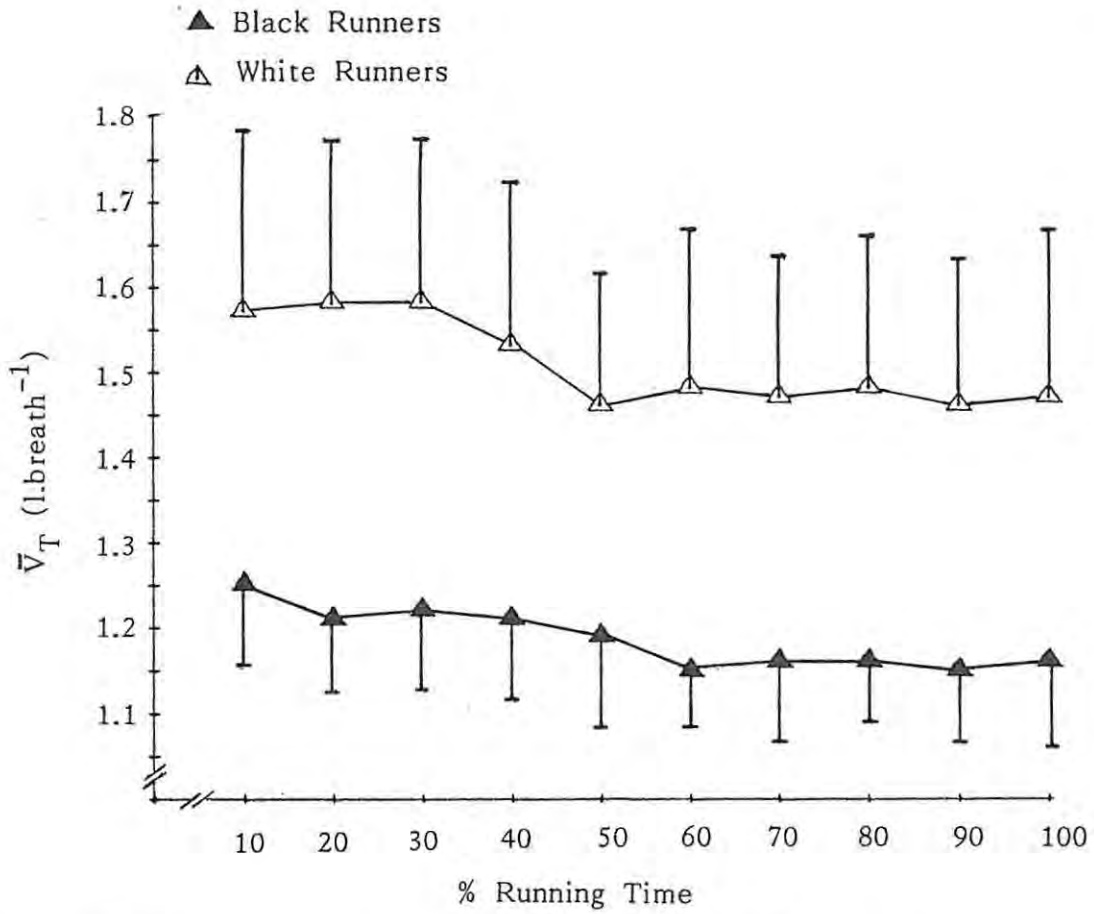

Figure 12: Black runners had a significantly lower $\bar{V}_T$ than white runners during a simulated marathon. Tidal volume decreased during the time-course of the marathon so that $\bar{V}_T$ was significantly lower from 60-100% running time than from 10-30% time. ($p < 0.05$).

The increase in $\dot{V}_I$ during the time-course of the simulated marathon (Figure 10 , Page 88) coincided with an increase in rectal temperature (Tr) at 70% running time (Figure 13 , Page 95). The correlation between these two variables, however, was not significant. This result differs from that of Adams (1977) who reported that an increased Tr was related to an increase in $\dot{V}_I$. This discrepancy may be due to differences in ambient temperature at the time of testing or the fitness of the subjects. Correlation analysis showed that there was a significant relationship, however, between Tr and f in both black ($r = 0.86$) and white ($r = 0.67$) runners. This result is in agreement with Dempsey et al (1977) who also showed that f increased as Tr increased. It is therefore possible that a rising Tr (Figure 13 , Page 95) was partly responsible for the observed increase in f (Figure 11 , Page 92). The increase in f may have been compounded by the decrease in $\bar{V}_T$ due to fatigue of the respiratory musculature, as previously suggested.

Tr was the same in both the black and white runners and increased significantly ($p < 0.05$) during the time-course of the simulated marathon from 38.6 ± 0.4 to 39.0 ± 0.7°C as shown in Figure 13 (Page 95). The percent mass loss, due mainly to sweating, was 2.7% in both the black and white runners, and therefore differences in dehydration between the groups would not have influenced thermal responses. The thigh skin temperature (Tth), although also similar in both groups, decreased from 32.4 ± 1.0 to 30.8 ± 1.4°C as shown in Figure 14 (Page 96), while the pectoral temperature (Tp) decreased from 30.2 ± 2.3 to 29.0 ± 2.1°C as shown in Figure 15 (Page 97). The latter Figure also shows that (Tp) was significantly ($p < 0.05$) higher in the black runners. This discrepancy may be due to differences in regional sweating and/or cutaneous vasomotor activity.

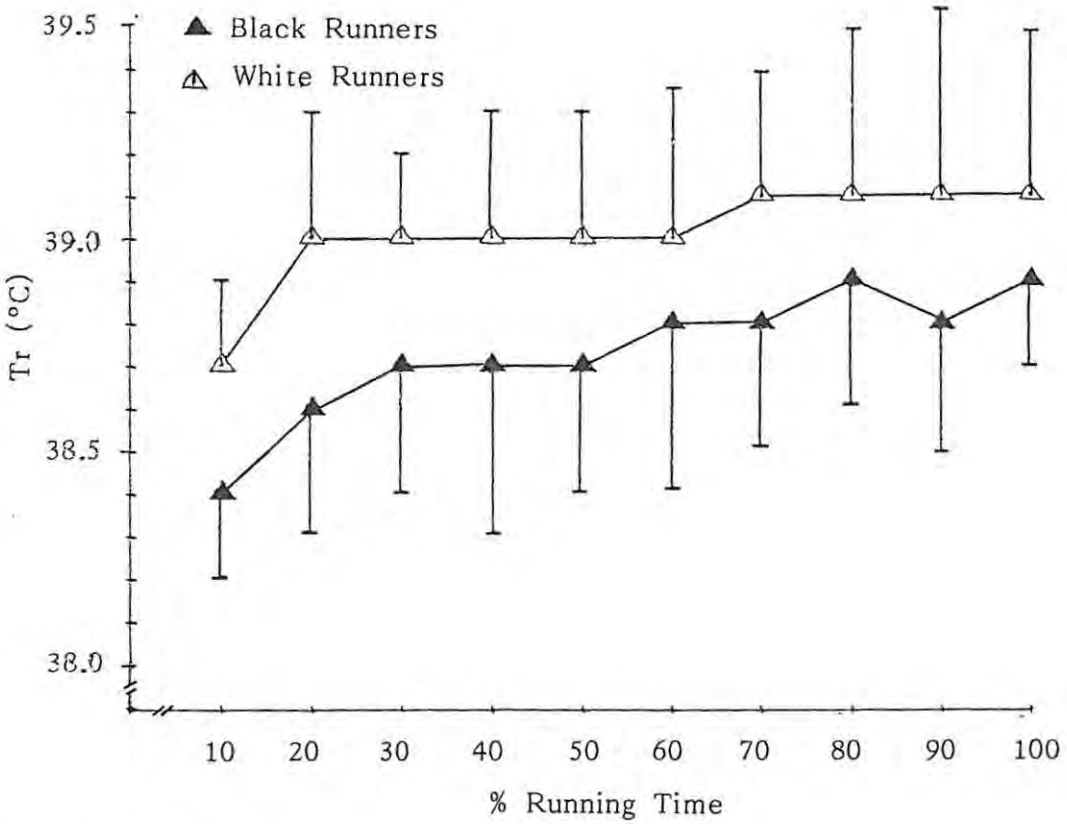

Figure 13: No significant differences existed in Tr between the black and the white runners during a simulated marathon. Rectal temperature increased during the time-course of the marathon so that Tr from 70-100% was significantly greater than at 10% running time. ($p < 0.05$).

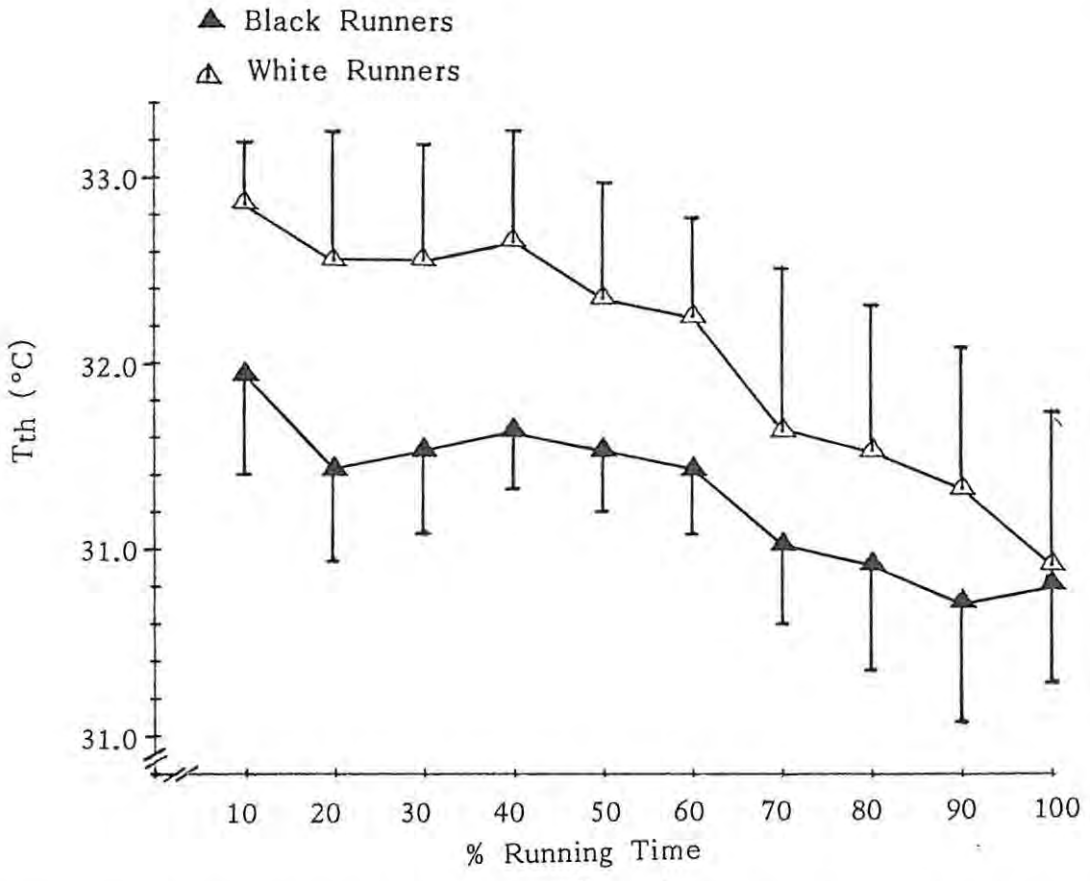

Figure 14: No significant differences existed in Tth between the black and white runners during a simulated marathon. Thigh temperature decreased during the time-course of the marathon so that Tth from 70-100% running time was significantly less than at 10% time. Tth from 10-60% running time was significantly greater than at 100%. ($p < 0.05$).

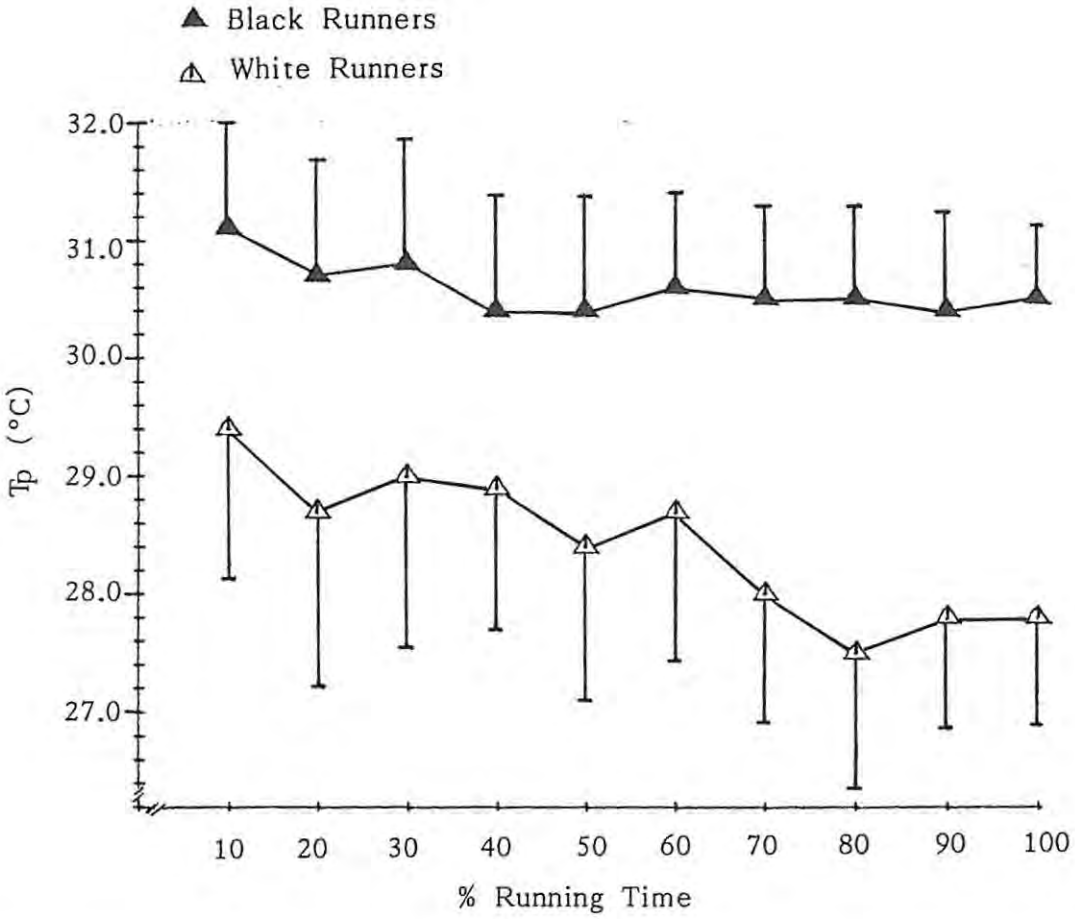

Figure 15: Black runners had a significantly higher T_p than white runners during a simulated marathon. Pectoral temperature decreased during the time-course of the marathon so that T_p from 70-100% running time was significantly less than at 10%. ($p < 0.05$).

When skin temperature was expressed as a single variable ($\bar{T}s$), this also decreased significantly during the time-course of the marathon, becoming significant from 70% running time (Figure 16 , Page 99). No difference existed in $\bar{T}s$ between the black and white runners during the marathon. The increase in Tr at 70% running time, and the corresponding decrease in $\bar{T}s$ at the same % time, suggests that the increase in Tr and the reduction in $\bar{T}s$ may have been related. A reduction in blood flow to the skin (reasons for which will be discussed subsequently) would result in a lower $\bar{T}s$, but would also result in a reduction in cooling of the blood at the skin surface. This would in turn result in the observed increase in Tr (Figure 13 , Page 95) due to a reduction in cooling of the blood via sweating. The increase in Tr may have precipitated changes in a number of other variables. The correlation between Tr and f was 0.86 in the case of the black runners and 0.67 in the case of the white runners, between Tr and H/R 0.70 and 0.67 respectively, and between Tr and "local" (leg) RPE 0.84 and 0.82 in the black and white runners respectively. There appeared to be no correlation, however, between Tr and either $\dot{V}_I$ or $\dot{V}0_2$.

During the time-course of the marathon there was a progressive rise in H/R (Figure 17 , Page 100) and a fall in stroke volume (SV) (Figure 18 , Page 101), which is in accordance with the literature (Costill 1979). This combination resulted in an increase in cardiac output ($\dot{Q}$) as shown in Figure 19 (Page 102). When body size differences were accounted for there was no difference in $\dot{Q}$ between the groups at either 16 $km.hr^{-1}$ (Table IV , Page 67) or during the simulated marathon (Figure 19 , Page 102). The rise in $\dot{Q}$ during the simulated marathon suggests that the increase in H/R (Figure 17 , Page 100) overcompensated for the decrease observed in SV (Figure 18 , Page 101). It is possible, however, that as fatigue occurs, possibly due to lowered muscle glycogen, additional muscle motor units are recruited as the depleted ST fibres are unable to generate sufficient

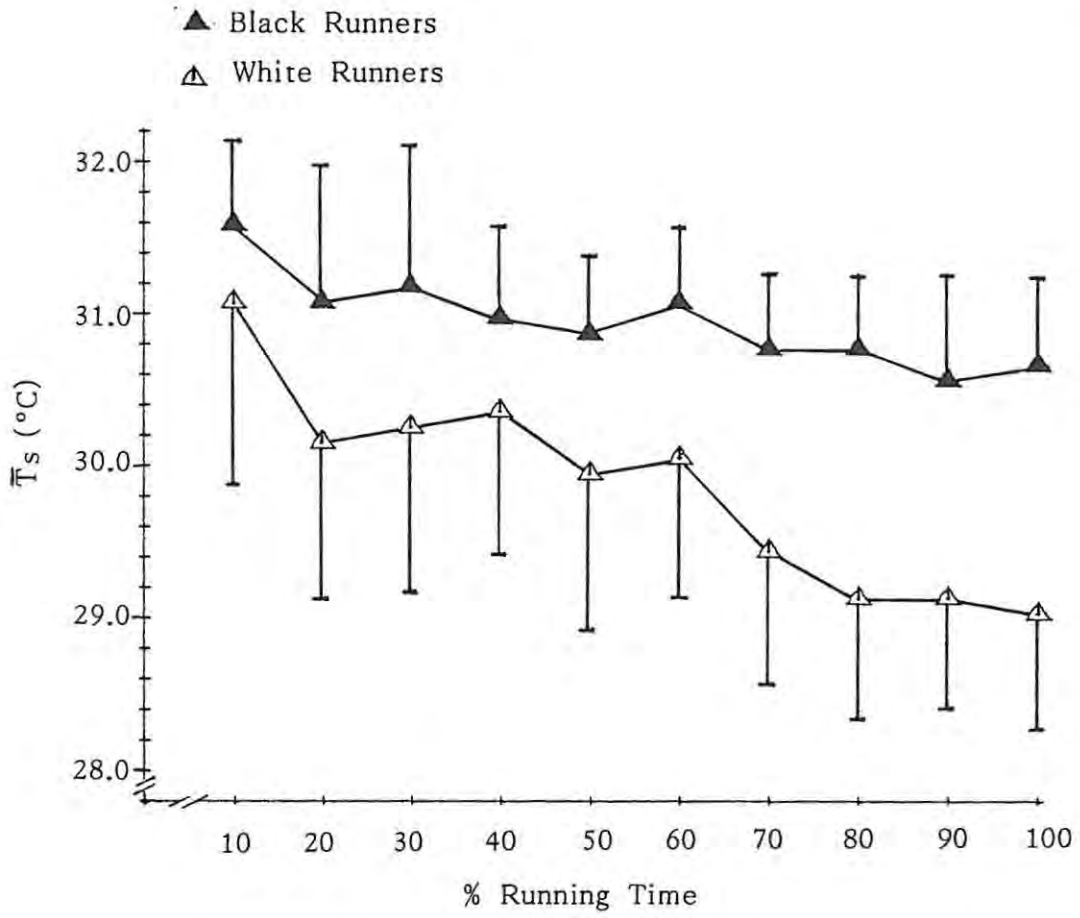

Figure 16: No significant differences existed in $\bar{T}s$ between black and white runners during a simulated marathon. Skin temperature decreased during the time-course of the marathon so that $\bar{T}s$ from 70-100% running time was significantly less than at 10%. ($p < 0.05$).

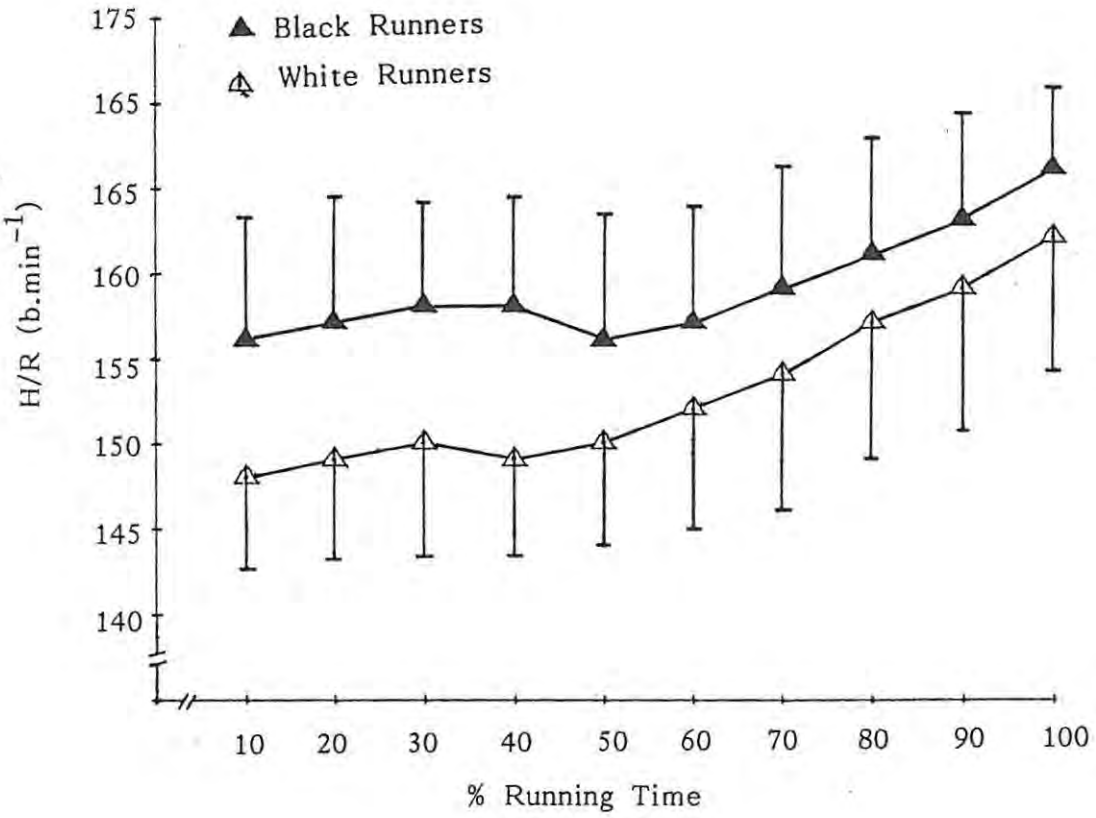

Figure 17: No significant differences existed in H/R between the black and white runners during a simulated marathon. Heart rate increased during the time-course of the marathon so that H/R from 70-100% running time was significantly higher than at 10%. Other significant differences were: 10-60% < 80-100% running time; 10-80% < 100% running time. ($p < 0.05$).

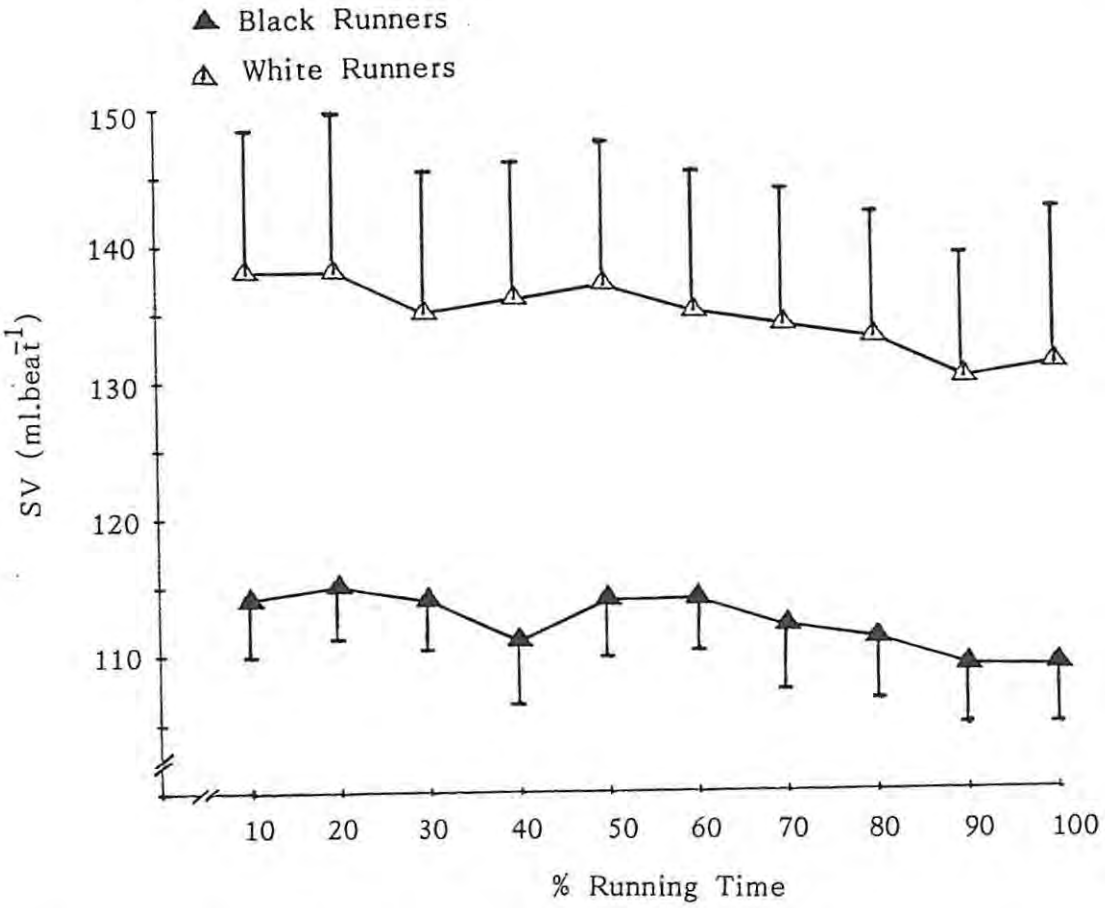

Figure 18: Black runners had a significantly lower stroke volume than white runners during a simulated marathon. Stroke volume decreased during the time-course of the marathon so that SV at 100% running time was significantly less than at 10, 20 and 50% time. ($p < 0.05$).

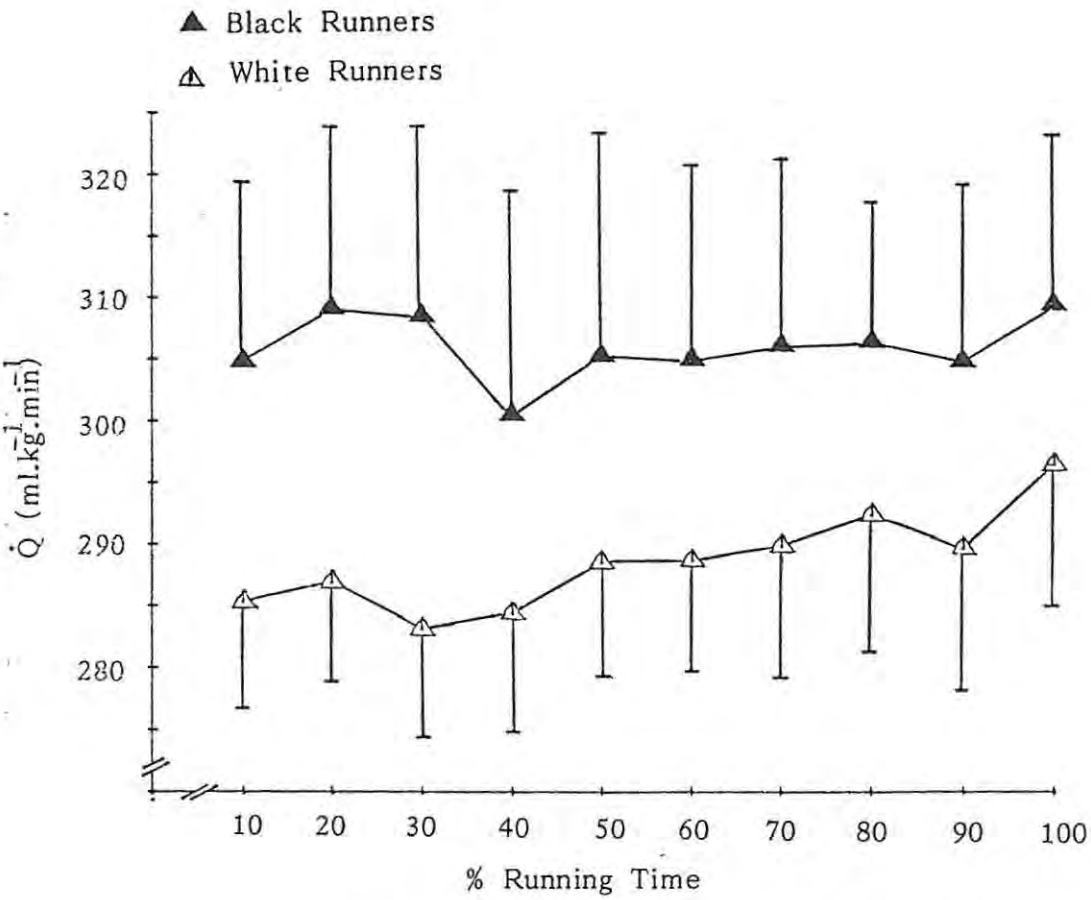

Figure 19: No significant differences existed in relative $\dot{Q}$ between the black and white runners during a simulated marathon. The increase in $\dot{Q}$ during the time-course of the marathon was significant only between the 40 and 100% running time intervals. ($p < 0.05$).

tension to maintain running speed (Costill et al 1973a), dictating an increase in $\dot{V}O_2$ (and thus $\dot{Q}$). The increase observed in H/R was significant at 70% running time. This corresponded to approximately 2 hours - 2 hours 15 minutes of running, which is the time at which muscle glycogen depletion occurs (Noakes and McArthur 1985) and therefore the time at which an increase in $\dot{V}O_2$ requirement may occur due to additional fibre recruitment. The increased $\dot{V}O_2$ requirement would explain the upward drift in $\dot{Q}$ (Figure 19, Page 102), achieved by the observed elevation in H/R. The increase in $\dot{V}O_2$ in the current study was very small and does not appear physiologically significant (44.2 $ml.kg^{-1}.min^{-1}$ at the start of the marathon to 45.7 $ml.kg^{-1}.min^{-1}$ at the end) but represents a 3% increase, similar to the value of 5% reported by Saltin and Stenberg (1964) which occurred during 195 minutes of running at 75% $\dot{V}O_2$ max. Besides the increased $\dot{V}O_2$ requirement necessitating an increase in H/R, another factor would be the decrease that occurred in SV (Figure 18, Page101). SV may decrease due to a reduced circulating blood volume (Nadel et al 1977, Costill 1979) and a progressive decrease in venous return of blood to the heart due to a breakdown in sympathetic blood flow control mechanisms (Brooks and Fahey 1984), or myocardial fatigue (Saltin and Stenberg 1964). To compensate for a reduction in circulating blood volume, cutaneous circulation may decrease as suggested by the decrease in $\bar{T}s$ (Figure 16, Page 99), increasing circulation to the muscle vasculature. The decrease in cutaneous circulation, however, would result in less cooling of the blood, which is reflected in the observed simultaneous increase in Tr (Figure 13, Page 95) and decrease in $\bar{T}s$. It should be pointed out that Costill (1979) reports an increased blood flow to the skin as Tr rises, which is in conflict with the results of this study. It is possible that a "hunting" response exists between the need for increased blood flow to the periphery (to increase cooling to prevent a progressive rise in Tr) and the need for an increased blood supply to the working musculature, as described.

Summarising and as shown schematically in Figure 20 (Page 105), it appears that due to muscle glycogen depletion additional motor units are recruited in order to maintain running speed, causing a subsequent greater $\dot{V}0_2$ requirement (Figure 4 , Page 74). This demand is met by an upward drift in $\dot{Q}$ (Figure 19 , Page 102) via an increase in H/R (Figure 17 , Page 100). The heart, however, may already have additional demands placed on it by a reduced blood volume and/or myocardial fatigure causing a decreased SV. A decrease in cutaneous blood flow may therefore occur in order to increase the central blood volume and blood flow to the muscle vasculature. Less cutaneous blood flow would result in the observed decrease in $\overline{T}s$ (Figure 16 , Page 99), less cooling of the blood via sweating, which in turn would result in the rise in Tr (Figure 13 , Page 95). The rise in Tr, in turn, may be related to the observed increase in f and RPE.

RPE Responses

As expected, "local" (leg) RPE (Figure 21, Page 106) and "general" RPE (Figure 22 , Page 107) both increased during the time-course of the marathon, but there was no difference between the black and white runners. It should be noted, however, that the blacks tended to be somewhat erratic in their rating of RPE, which may be due to them not interpreting the Borg Scale in the same way as the white runners. This may be due to cultural and language differences between the two groups. When translating the Borg RPE Scale from English to Xhosa, the concept may not have been translated with the equivalent English language meaning. This problem may be magnified as wider variations exist as to the meaning of words within the Xhosa language, than English. The erratic nature of the RPE rating of black runners is shown by the coefficient of variation which varied from 18.2 - 26.7 % for "local" RPE and 20 - 38.5% for "general" RPE. The corresponding figures for the white runners were 7.7 - 20.0% and 7.7 - 21.4% respectively. Correlation analysis revealed that in the case of the white runners there was a high relationship between "local" RPE and Tr ($r = 0.82$), "local" RPE and $\dot{V}_I$ ($r = 0.91$) "local" RPE and

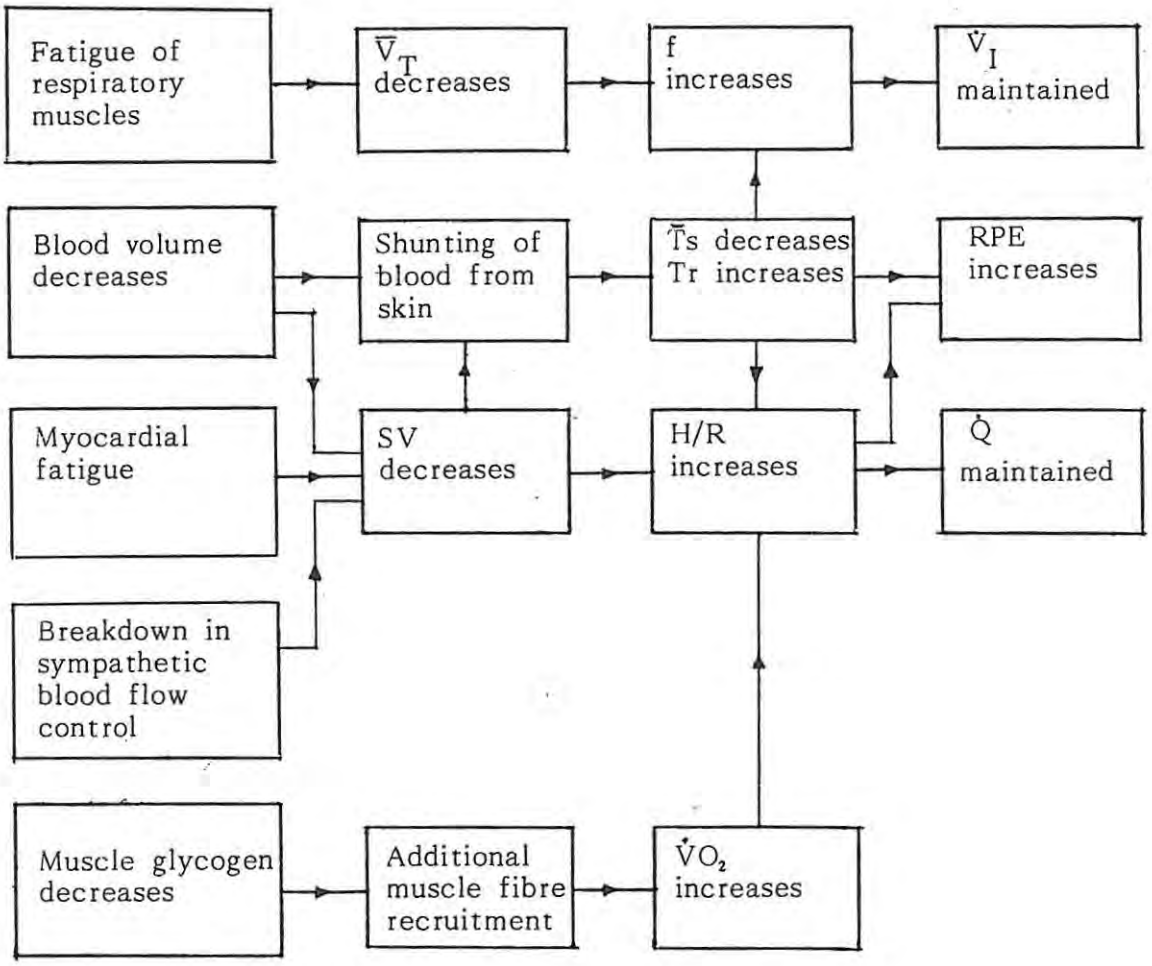

Figure 20: Flow-diagram of cardiopulmonary responses during a marathon.

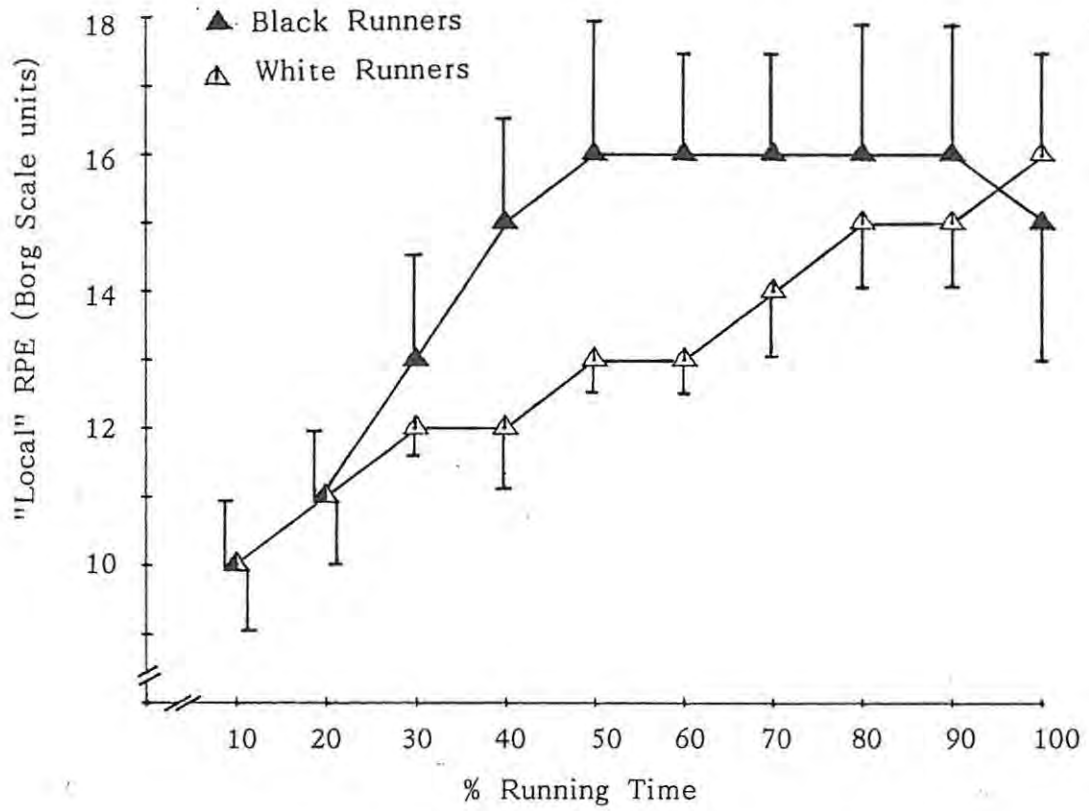

Figure 21: No significant differences existed in "local" (leg) RPE between the black and white runners during a simulated marathon. "Local" RPE increased during the time-course of the marathon so that the values from 40-100% running time were significantly greater than at 10% time. Other significant differences were: 10-20% < 40% < 90-100% running time. ($p < 0.05$).

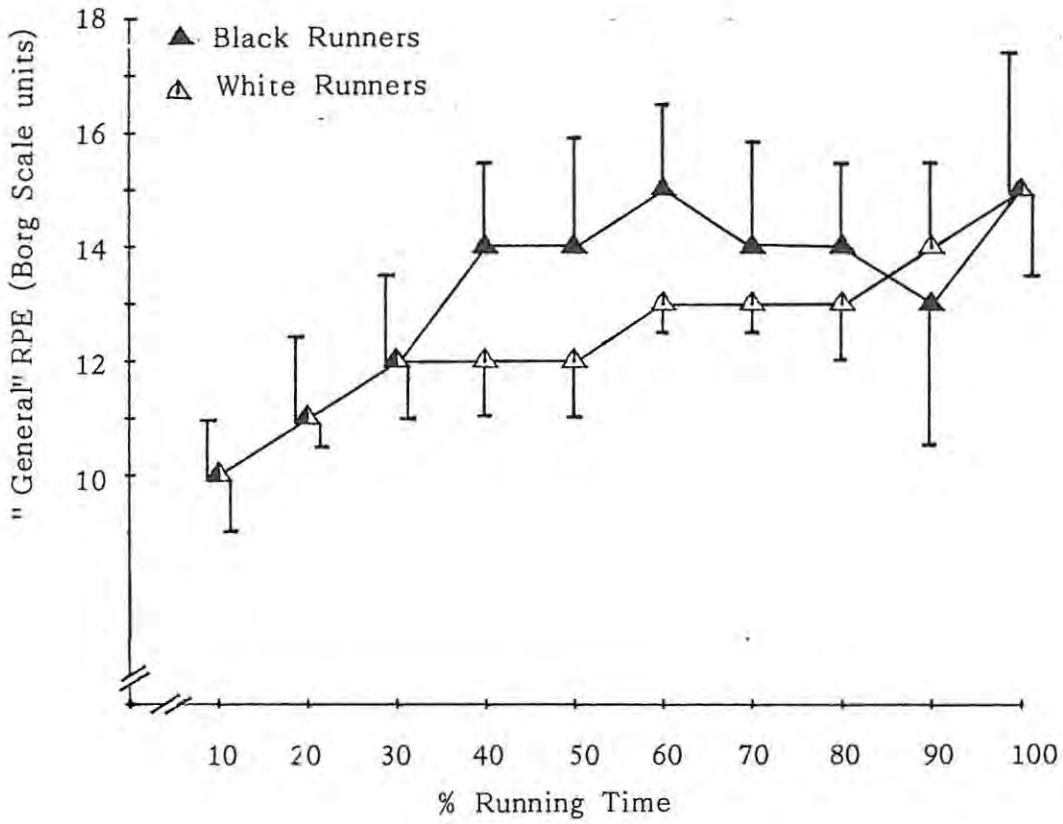

Figure 22: No significant differences existed in "general" RPE between the black and white runners during a simulated marathon. "General" RPE increased during the time-course of the marathon so that the values from 40-100% running time were significantly greater than at 10% time. Other significant differences were: 10-30% < 100%. ($p < 0.05$).

H/R (r=0.94) and "local" RPE and R(r=0.94). Although all these variables were strongly related to the "local" RPE in the white runners, in the case of the black runners only Tr was significantly correlated with "local" RPE, there being no correlation between RPE and the variables of $\dot{V}_I$, H/R, and R in these runners. This may be due to the erratic RPE responses of the black runners as shown by the coefficients of variation presented.

Gait and Anthropometrical Factors

When gait and anthropometrical factors were examined it was found that the white runners were taller (180.7 $\pm$ 6.4 cm) than the black runners (171.1 $\pm$ 6.2 cm) and had a greater trochanterion height (94.8 $\pm$ 4.7 cm) compared to the black runners (90.0 $\pm$ 3.6 cm). When trochanterion height was expressed as a percentage of stature, however, no difference was found to exist between the groups (Table XV , Page 109). No relationship was found to exist between stature and step rate, but there was a significant ($p < 0.05$), correlation ($r = 0.67$ in both groups) between trochanterion height and step rate in both black and white runners. These findings are in agreement with Cavanagh et al (1977) who also found a correlation of 0.67 between step rate and leg length. Step length was the same in both groups, but when expressed as a percentage of stature, it was found that the white runners took relatively shorter steps (166.0 $\pm$ 12.7% of stature) than the black runners (180.0 $\pm$ 7.0% of stature). Therefore, relative to stature, the black runners were taking longer steps (at 16 $km.hr^{-1}$). Similar results for the two groups would be expected as Cavanagh and Williams (1982) found that decreasing or increasing stride length increased $\dot{V}O_2$, and Cavanagh et al (1977) demonstrated that international class runners tended to take shorter steps than elite runners.

Table VIII (Page 109) shows that the stature of the black runners was less than that of the white runners ($p < 0.05$). In a study by Pollock et al (1977) it was shown that the stature of international class marathon runners was

Table VIII: Descriptive statistics of anthropometric data of subjects.

CATEGORY		Mass (kg)	Stature (cm)	Trochanterion Height (cm)	Trochanterion Ht. as % Stature	% fat	Step rate (steps.min^{-1})
Black (n=9)	$\bar{X}$	59.5	171.1	90.0	52.6	11.0	180
	SD	4.3	6.2	3.6	1.2	1.4	6
White (n=10)	$\bar{X}$	71.1	180.7	94.8	52.3	11.1	182
	SD	8.6	6.4	4.7	1.1	1.4	8
		*	*	*			

* $p < 0.05$

176.8 cm, slightly greater than the 171.1 cm of the black runners who participated in this study. In the same study by Pollock et al (1977), elite runners were found to have a stature of 181.1 cm, which is similar to that of the white runners (180.7 cm) in the present study (Table VIII, Page 109). Costill et al (1971a) have pointed out, however, that fast times have been recorded by marathon runners who are much taller than the mean. Even in the present study, the fastest black runner (WM), who was also the fastest runner in the entire sample, was one of the taller black runners (Table I , Page 62). No significant relationship was found between stature and marathon race time. Costill (1979) is of the opinion that success in distance running is unrelated to body height. It is thus unlikely that the smaller stature of the black runners is contributing to their superiority in the marathon. Although Costill (1979) discounts stature as a factor contributing to success in the marathon, he suggests that the mass of an individual may be of importance. In this regard, it was found that international class runners tested (Pollock 1977b) had a lower mean body mass (62.1 kg) than elite runners (67.5 kg). The black runners in this study were lighter in mass than the international runners in the Pollock (1977b) study , while the mean mass of the white runners was greater than that of the elite runners studied by Pollock (1977b). The lower body mass of the black compared to the white runners is to be expected, however, due to the smaller stature of the blacks (Table VIII, Page 109). Costill (1979) states that excessive body mass would add to the energy required to run, which would suggest that blacks may require less energy to run than whites. This was not found to be the case, however, there being no statistically significant difference between the groups in the gross energy cost of running the marathon ($KJ.kg^{-1}$) (Table III, Page 65).

Although body mass was greater in the case of the white runners, the percentage body fat was identical in the black and white groups. This

was 11.0 $\pm$ 1.4% in the case of the black group and 11.1 $\pm$ 1.4% in the white group (Table VIII, Page 109). As Schutte et al (1984) have shown that the observed body density of blacks was significantly greater than the predicted density using anthropometric techniques and standard formulae, it was suggested by the authors that a separate formula should be used when converting density to percent fat in the case of black subjects. It was this formula which was used when calculating the percent body fat of the black runners (Appendix G). The values for percent body fat reported in this study are higher than reported in previous studies. Pollock et al (1977) reports values for percent body fat ranging from 4.7 - 5.1% in the case of international class runners studied and 6% for elite runners. Costill et al (1970a) have reported values ranging from 6 - 8%. It is suggested that the difference between these values and those reported for the present study are due to the group of runners studied not being of the same performance standard as those studied by Pollock et al (1977) and Costill et al (1970a), or that the training distance and/or intensity of the runners in this study was less than the runners participating in the studies of the authors cited above.

CHAPTER 5

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

The purpose of the study was to determine to what extent black and white runners may differ in physiological, gait and RPE characteristics, and whether any changes occur in the variables examined during the time-course of a marathon.

Summary of Procedures

The variables investigated were measured during a progressive $\dot{V}O_2$ max test and simulated marathon on the treadmill. The data from the $\dot{V}O_2$ max tests were analysed at $\dot{V}O_2$ max and at 16 $km.hr^{-1}$ as physiological data have most often been reported at this speed, and because it was close to the average race speed of the subjects. Data from the simulated marathons were analysed at 10% intervals from 10 to 100% of the total running time of the subjects. The variables examined included cardiopulmonary, blood lactic acid and glucose, thermal, gait and RPE responses as well as body compositional factors. These variables were examined in 9 black and 10 white experienced male marathon runners, who had marathon best times of 2 hours 45 minutes or faster. This was chosen as it represents a fast time for the marathon, being close to the qualifying standard of the South African marathon championships.

Each subject was informed of the nature and purpose of the study, and gave signed, informed consent to act as a subject. Anthropometric data of stature, biacromial diameter, mass, trochanterion height and skinfolds were obtained, the skinfolds being triceps , biceps, subscapular, supra-iliac, abdominal, front thigh and medial calf. Physiological data, which included oxygen consumption, carbon dioxide production, respiratory exchange ratio, inspired volume, heart rate, breathing frequency, ventilatory equivalent for oxygen, ventilatory equivalent for carbon dioxide, estimated cardiac output,

estimated stroke volume, oxygen pulse, tidal volume, % $\dot{V}O_2$ max and treadmill speed were obtained using a computer-aided on-line data acquisition system designed by B.R. Goslin of the Human Movement Studies and Physical Education Department, Rhodes University (Goslin et al 1984). Gas analysis was performed via Beckman LB-2 and OM-14 analysers for carbon dioxide and oxygen respectively. Later in the study, these were replaced by the Ametek S-3AI oxygen analyser with N-22 oxygen sensor, and Godart Capnograph Mark III carbon dioxide analyser.

Each subject was informed of the $\dot{V}O_2$ max test protocol and then allowed time to habituate to treadmill running. The test was started with the treadmill speed at 8 $km.hr^{-1}$ and the treadmill horizontal. Every 1.5 minutes the speed was increased by 1 $km.hr^{-1}$ until a speed 2 $km.hr^{-1}$ faster than the subjects' best race speed was reached. At that point the speed was kept constant and the grade increased by 1% every 60 seconds until the subject was unable to continue. Subjective exhaustion, a plateau in $\dot{V}0_2$ where there was less than a 50 ml increase in $\dot{V}0_2$ from one sample to the next (Morehouse 1972), a decrease in $\dot{V}0_2$ after a further increment in power output and a respiratory exchange ratio greater than one were used as criteria that $\dot{V}0_2$ max had been reached. Heart rate was monitored during the $\dot{V}0_2$ max test and simulated marathon using a modified "Exersentry" heart rate monitoring system. After a minimum of 4 hours rest following the $\dot{V}0_2$ max test, each subject returned to the laboratory to run a simulated marathon. The speed at which this was run was determined from the VT of the subject. The VT was used as a marathon can be run at a speed corresponding to this (Farrell et al 1979). The main criteria for the determination of VT were a secondary increase in $VE-0_2$ and a smaller increase in $VE-C0_2$ (Dawson and Pyke 1984). Once the speed corresponding to the VT was determined, 92.5% of that speed was calculated and used as the speed for the simulated marathon run. This enabled most subjects to

complete the full marathon distance.

YSI model 408 surface skin temperature probes were used to measure pectoral and thigh surface temperature during the marathon and were attached using mesh screening covered by "Elastomesh". Core temperature was monitored using a YSI model 401 rectal temperature probe. Ratings of perceived exertion (RPE) for "general" and "local" (leg) RPE were obtained using the Borg RPE Scale (Borg 1973). Step rate per minute was obtained by counting the number of times the left foot made contact with the treadmill belt and multiplying by two.

During the run a fan was used to aid cooling and subjects were allowed to drink ad libitum of any replacement fluid they normally used when running a marathon. Subjects ran until 42.2 km had been completed, or until too tired to continue. A 5 ml venous blood sample was drawn from the antecubital vein before and after the marathon and analysed for lactic acid and glucose.

Data were recorded every 10 minutes during the marathon, later categorised into 10% temporal intervals from 10 - 100% of total running time and analysed using a 2 x 10 ANOVA with repeated measures on one factor, this being % running time. Where the ANOVA showed significant differences during the time-course of the marathon, the Sheffé or Tukey test was used for a post-hoc analysis. For data at 16 $km.hr^{-1}$ and $\dot{V}O_2$ max the groups were compared by using a t-test for independent samples. In all statistical analyses the level of significance chosen was 0.05.

Summary of Results

The subjects participating in the study were characterised by a high $\dot{V}0_2$ max. This was 60.4 $\pm$ 6.5 $ml.kg^{-1}.min^{-1}$ in the black runners and 63.2 $\pm$ 2.9 $ml.kg^{-1}.min^{-1}$ in the whites, but this difference was not statistically significant. The

correlation between $\dot{V}0_2$ max and marathon race time was 0.86 and 0.85 in the black and white runners respectively. These data suggest that a high $\dot{V}0_2$ max was partly responsible for the subjects being capable of running a fast marathon, but did not discriminate between the black and white runners.

There was no significant difference in submaximal $\dot{V}0_2$ of the groups, the $\dot{V}0_2$ at 16 $km.hr^{-1}$ of the black and white groups being 49.9 $\pm$ 1.7 and 51.0 $\pm$ 5.3 $ml.kg^{-1}.min^{-1}$ respectively. No significant relationships existed between $\dot{V}0_2$ at 16 $km.hr^{-1}$ and $\dot{V}0_2$ max, or $\dot{V}0_2$ at 16 $km.hr^{-1}$ and race performance. The correlation between % $\dot{V}0_2$ max at 16 $km.hr^{-1}$ and marathon race performance was 0.68 in the case of the white runners, but no correlation between these variables existed in the case of the black runners. The % $\dot{V}0_2$ max utilised at marathon race speed was significantly higher in the black (89.0 $\pm$ 5.5%) than the white runners (81.5 $\pm$ 3.1%) although the marathon race times of the groups were not significantly different. The ability of the black runners to run at a very high % $\dot{V}0_2$ max may be related to lower blood lactic acid levels in these runners at a given % $\dot{V}0_2$ max, as indicated by a higher % $\dot{V}0_2$ max during the simulated marathon (Figure 3 , Page 64) but lower post-run blood lactic acid levels than measured in the white runners (Table II , Page 65). A significant relationship was found to exist between $\dot{V}0_2$ max and VT in both groups ($r = 0.84$ and 0.60 in the black and white runners respectively).

The mean R value was significantly higher in the black than the white runners during the simulated marathon (Figure 6 , Page 78) and at submaximal running speeds. As there was no difference in the % $\dot{V}0_2$ max utilised between the groups at submaximal speeds, this was not the cause of higher R values observed.

An explanation is necessary to account for the higher % $\dot{V}0_2$ max at which the black runners were running during both the simulated marathon and their best marathon race, as well as the lower blood lactic acid levels and higher R values in these compared to the white runners. It is well established that within a range that occurs due to differences in running economy, a linear relationship exists between running speed and $\dot{V}0_2$ requirement. Therefore, as both groups had similar best times for the marathon, it would be expected that the $\dot{V}0_2$ of the two groups would be similar when running at a speed equivalent to that of their best marathon races. The black runners tended to have a higher $\dot{V}0_2$ at this speed than the white runners, but this was not significant. When $\dot{V}0_2$ was combined with the $\dot{V}0_2$ max to give % $\dot{V}0_2$ max, however, it was found that the combination of a higher $\dot{V}0_2$ but lower $\dot{V}0_2$ max in the black runners, resulted in a % $\dot{V}0_2$ max at race speed which was significantly higher in the black group. It is therefore suggested that the black runners in the current study were able to run marathons at a similar speed to the white runners because of their ability to tolerate running at a high % $\dot{V}0_2$ max, which enabled them to meet the necessary $\dot{V}0_2$ requirement. This implies that if the best black runners in South Africa have the same running economy and $\dot{V}0_2$ max as the top white runners, black runners would be capable of running faster marathons because of their ability to run at a greater % $\dot{V}0_2$ max. This ability can be explained in terms of the "lactate shuttle", as can the lower lactic acid levels and higher R values measured in the black runners. If the "lactate shuttle" pathway operates at a higher rate in the black runners, greater clearance of lactic acid by oxidation would occur, and a greater percentage contribution by lactic acid to the total carbohydrate oxidised. The effects would be reduced lactic acid levels, enabling the utilisation of a larger % $\dot{V}0_2$ max, and higher R values due to increased carbohydrate oxidation.

No significant differences were observed in $\dot{V}_I$ or $\dot{V}_A$ between the groups

during either submaximal or simulated marathon running, even when differences in body size were accounted for. At $\dot{V}O_2$ max, however, the black runners had a significantly lower $\dot{V}_I$ than the white runners, but f was similar in both groups. At submaximal speeds and during the simulated marathon, f was higher in the black runners and the $\bar{V}_T$ was lower than that observed in the whites (Table V , Page 68). If the white and black runners both utilised the same % VC for each breath, this would necessitate a lower $\bar{V}_T$ in the black runners as their VC was probably smaller due to their smaller body size.

During the simulated marathon, the increase in $\dot{V}_I$ became significant after 70% running time. Tr also increased at 70% running time and may be related to an increase in f (r = 0.86 and 0.67 in the black and white runners respectively) at the same time. Tr was similar in both groups. The increased Tr at 70% running time and corresponding decrease in $\bar{T}s$ suggests that a reduction in skin blood flow may have occurred. This would result in less cooling of the blood via sweating and the observed increase in Tr, which may have acted as a stimulus for the increase in H/R in the black and white runners (r = 0.70 and 0.67 respectively) and "local" (leg) RPE (r = 0.84 and 0.82 respectively).

"Local" (leg) and "general" RPE both increased during the time-course of the marathon, but no significant differences existed between the black and white runners. In the case of the white runners, "local" RPE was highly correlated with Tr (r = 0.82), $\dot{V}_I$ (r = 0.91), H/R (r = 0.94) and R (r= 0.94), but in the black runners the only correlation was with Tr (r = 0.94). The RPE ratings of the black runners may have been influenced by cultural differences between blacks and whites influencing interpretation of the wording of the Borg RPE Scale. The opportunities of blacks for social and economic mobility in South Africa are limited, but being successful in

races can provide a channel through which social and economic goals can be realised, including sponsorship, employment and enhanced prestige. This would be a powerful motivating factor for black runners compared to whites during racing.

When examining gait and anthropometrical factors, it was found that when trochanterion height was expressed as a percentage of stature, no difference was found to exist between the groups. The correlation between trochanterion height and step rate was the same in both groups ($r = 0.67$), but the black runners took longer steps when this was expressed as a percentage of stature (180 ± 7 and $166 \pm 13\%$ stature in the black and white groups respectively). This may be related to a minimisation of $\dot{V}O_2$. The black runners were shorter (171.1 ± 6.2 cm) and lighter in mass (59.5 ± 4.3 kg) than the white runners (180.7 ± 6.4 cm and 71.1 ± 8.6 kg respectively) but both had a similar percentage body fat ($11.0 \pm 1.4\%$ and $11.1 \pm 1.4\%$ for the black and white groups respectively). Gait and anthropometric factors are unlikely to account for differences in performance between the groups.

Conclusions

A number of variables have been considered important in discriminating between athletes of similar performance ability and it was thought that the same variables might discriminate between black and white marathon runners. It was therefore hypothesised that no differences would be found to exist between elite black and white marathon runners in a number of physiological, gait and RPE variables investigated.

1. Lower lactic acid levels at a given % $\dot{V}O_2$ max allow black runners to utilise a larger % $\dot{V}O_2$ max than white runners. Therefore, if the top black runners in South Africa have the same running economy and $\dot{V}O_2$ max as the top whites, the ability to utilise a larger % $\dot{V}O_2$ max by the blacks would enable them to run faster marathons.

2. Although the $\dot{V}_I$ was similar in both the black and white groups during the simulated marathon, f was higher and $\bar{V}_T$ lower in the case of the black runners. These ventilatory differences are unlikely to account for differences in performance between black and white marathon runners.

A second hypothesis of the study was that no changes would occur in the variables investigated during the time-course of a marathon.

1. During the time-course of the simulated marathon SV decreased and H/R increased, resulting in a slight increase in $\dot{Q}$. Although f increased, $\bar{V}_T$ decreased during the time-course of the marathon, resulting in a small increase in $\dot{V}_I$. Cardiopulmonary adjustments therefore occur during the time-course of a marathon which keep $\dot{Q}$, $\dot{V}_I$ and $\dot{V}O_2$ constant.
2. The decrease in R value during the time-course of the simulated marathon confirms the progressive importance of fats as a fuel substrate during marathon running.

Recommendations

Based on the findings reported it is suggested that future studies include the following:

1. The taking of blood samples for lactic acid analysis at a range of sub-maximal running speeds and relating these to the R values. It is hypothesised that blood lactic acid levels would be lower in black runners at all submaximal speeds, and the R values higher.
2. In order to further elucidate the fate of lactic acid in black runners, it is suggested that blood bicarbonate also be measured in order to assess the extent to which increased buffering may be occurring in the case of black runners. Isotope tracer techniques would also be of value. It is hypothesised that bicarbonate levels would differ between black and white runners.
3. Lactate turnover rates should also be measured, by means of isotope tracer techniques, to gain information regarding lactate production and clearance

in black compared to white runners. It is hypothesised that lactic acid production is the same in both black and white runners, but clearance might be greater in black runners.

4. Catecholamine levels should be measured during both submaximal running and the running of a marathon. It is hypothesised that catecholamine levels will be lower in black runners, which may ultimately permit greater oxidation of lactic acid.
5. It would be of interest to determine whether the difference in lactate kinetics of black runners is entirely genetic in nature, or whether this is as a result of differences in life-style of young blacks. This question may be answered by studying selected variables in blacks of various ages to determine if there is an age determined onset of the differences observed in this study or whether differences exist across all age groups.

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APPENDIX A

RHODES UNIVERSITY

DEPARTMENT OF HUMAN MOVEMENT STUDIES AND PHYSICAL EDUCATION

INFORMATION SHEET

The research in which you are involved aims to investigate any changes that may occur in running economy and other physiological, biomechanical and psychophysiological variables during the progression of a simulated marathon. A further aspect of the study is the determination of any differences that may exist in these parameters between black and white marathon runners.

You will be required to do the following:-

1. Complete the "Subject Consent" form.
2. Complete a $\dot{V}O_2$ max test, which involves running on the treadmill until you cannot continue.
3. Run on the treadmill at close to marathon race speed for the equivalent of the marathon distance, during which various physiological, gait and exertion parameters will be determined.

The following possible risks and discomforts exist:-

1. Normal hazards and discomforts associated with:
   a) Working up to maximum capacity
   b) Running a marathon

2. These normal hazards and discomforts include, as you know, the possibility of abnormal blood pressure, disorders of heartbeat, fainting and exhaustion.

Benefits to you include the assessment of your $\dot{V}O_2$ max, which is an important component in determination of success in the marathon; the establishment of the percentage of your maximum at which you race, as well as any changes that may occur during a race; an estimate of the fastest time in which you could currently complete a marathon, as well as the fastest time you are ever likely to achieve, if you desire this information.

Generally, the study will enable you to discover more about yourself as a runner.

Thank you for your participation in this research.

ANDY BOSCH

APPENDIX B

RHODES UNIVERSITY

DEPARTMENT OF HUMAN MOVEMENT STUDIES AND PHYSICAL EDUCATION

SUBJECT CONSENT FORM

I, _____ ,having been fully informed of the nature of the research project entitled "Physiological differences between black and white marathon runners", do hereby give my consent to act as a subject in the above-mentioned project. I am fully aware of the procedure involved, which includes:-

1. A maximal oxygen uptake test in which the level of work will be advanced in stages to exhaustion.
2. A simulated marathon on the treadmill in which running speed will approximate marathon race speed, and duration as close to marathon distance as possible. Blood samples will be taken before and at the completion of the marathon. Skin and core temperatures will be taken during the marathon.

The potential risks and benefits have been explained to me verbally and in writing. I understand that I may terminate my participation in the study at any time.

SIGNATURE OF SUBJECT

DATE: _____

SIGNATURE OF INVESTIGATOR

DATE: _____

SIGNATURE OF SUPERVISOR

DATE: _____

APPENDIX C

FORM LETTER FOR FEEDBACK TO SUBJECTS

Department of Human Movement Studies
Rhodes University
Grahamstown
6140

Dear

Thank you for your participation in the research project on the physiological responses to marathon running. Some of the data obtained will be of interest to you and are detailed below:-

The maximal amount of oxygent that the muscles can utilise is termed the $\dot{V}O_2$ max and is an important determinant of success in endurance activities such as marathon running. The possession of a high $\dot{V}O_2$ max is largely genetically determined and cannot be changed very much by training. Another important component of successful marathon running is the ability to require only a small amount of oxygen at any given running speed. The less oxygen required at any given speed, the more economical you are as a runner. Running economy can be improved to a certain extent with training, especially over a period of years. Another component that improves with training is the ventilatory threshold (VT). This is an indicator of the accumulation of lactic acid in the blood and is the major determinant of the speed at which you can run a marathon. Speedwork consisting of 3 - 5 minutes fast running with an equal recovery interval appears at present to be the best way of improving the ventilatory threshold.

If you train very hard, you can expect to run a marathon in a time of _____. It would appear from the results of your test that you would benefit most from _____. You should aim to drink _____ ml every hour during a marathon as too great a water deficit can result in a decrease in performance as well as cramping.

If you have any queries, please contact me.

Attached is a list of your results, as well as typical values for a sedentary person and a 2:20 marathoner. Thanks again for your participation.

Yours in running

ANDY BOSCH

	Your value	Sedentary male	Sedentary female	Typical 2:20 Marathoner
$\dot{V}O_{2}$ max ($ml.kg.^{-1}.min^{-1}$)		45	35	68
VT ($ml.kg.^{-1}.min^{-1}$)		23	18	53
VT as % $\dot{V}O_{2}$ max		50	50	80 - 85
Sweat loss ($ml.hr^{-1}$)		-	-	1500
% mass loss in run		-	-	3
Economy at 16 $km.hr^{-1}$ ($ml.kg.^{-1}min^{-1}$)		15 - 53	50 - 53	49
% fat		15	25	8.5
Core temp (°C)		-	-	38.2 - 39.2
Heart rate at 70 - 80% $\dot{V}O_{2}$ max (Most training at this rate)		-	-	160 - 168

APPENDIX D

ON-LINE DATA ACQUISITION SYSTEM EQUATIONS

The computer programme developed for the on-line data-acquisition system (Goslin et al 1984) and used in the present study was designed to calculate oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ration (R), ventilatory equivalents for oxygen (VE - O_2) and carbon dioxide (VE - CO_2), oxygen pulse (O_2 Pulse) (Consolazio et al 1963), and an estimate of cardiac output($\dot{Q}$) and stroke volume (SV). (Faulkner et al 1977).

The following equations form the computational package used in the on-line computer-aided data acquisition system

1. Partial Pressure of Water Vapour (PH_2O) (mmHg):

   PH_2O = EXP (2.303* (8.10765-(1750.286/(235+T))))

   Where T = gas temperature (°C)

2. Correction factor to reduce ambient conditions to standard temperature and pressure, dry (STPD):

   STPD FACTOR = (273/273+T)*(PB-FRH*PH_2O))/760)

   Where PB = barometric pressure (mm Hg)

   FRH = fractional relative humidity of inspired air

3. Correction of Inspired ambient volume ($\dot{V}_I$) for sample duration and STPD:

   $\dot{V}_I$STPD ($l.min^{-1}$) = $\dot{V}_I$ (l)*STPD FACTOR*(60/Time)

   Where Time = sample duration (s)

4. Fraction of nitrogen inspired (F_IN_2) and expired (F_EN_2):

   $F_IN_2 = 1-(F_IO_2 + F_ICO_2)$

   $F_EN_2 = 1-(F_EO_2 + F_ECO_2)$

5. Oxygen Consumption ($\dot{V}O_2$):

   $\dot{V}O_2$ ($l.min^{-1}$) = V_ISTPD*(F_IO_2 -((F_IN_2 /F_EN_2)*F_EO_2))

6. Carbon Dioxide Production ($\dot{V}CO_2$):

$\dot{V}CO_2\,(l.min^{-1}) = \dot{V}_I STPD*(((F_I N_2 / F_E N_2) * F_E CO_2) - F_I CO_2)$

7. Respiratory exchange ration (R):

$R = \dot{V}CO_2 / \dot{V}O_2$

8. Oxygen consumption per kilogram of body mass ($\dot{V}O_2$):

$\dot{V}O_2\,(ml.kg^{-1}.min^{-1}) = 1\,000*\dot{V}O_2\,(l.min^{-1}))/\text{Body mass (kg)}$

9. Breathing frequency (f):

$f\ (br.min^{-1}) = f\ (br)*(60/\text{sample duration (s)})$

10. Average tidal volume ($\bar{V}_T$):

$\bar{V}_T\ (l.br^{-1}) = V_I STPD(l.min^{-1})/f(br.min^{-1})$

11. Ventilatory Equivalent for oxygen (VE - O_2):

$VE - O_2\ (l.100\ ml O_2^{-1}) = \dot{V}_I STPD(l.min^{-1})/\dot{V}O_2\,(l.min^{-1}))/10$

12. Ventilatory Equivalent for carbon dioxide (VE - CO_2):

$VE - CO_2\ (l.100\ ml CO_2^{-1}) = (\dot{V}_I STPD(l.min^{-1})/\dot{V}CO_2\,(l.min^{-1}))/10$

13. Oxygen pulse (O_2 Pulse):

$O_2\ \text{Pulse}\ (ml O_2.bt^{-1}) = (1\,000*\dot{V}O_2(l.min^{-1}))/\text{heart rate}\ (b.min^{-1})$

14. Estimated cardiacoutput ($\dot{Q}$):

$\dot{Q}(l.min^{-1}) = (ZZ+(5.2*\dot{V}O_2\,(ml.kg^{-1}.min^{-1}))) * (BM/1000)$

Where ZZ = 66 if male subject

ZZ = 75 if female subject

BM = Body mass (kg)

15. Stroke volume (SV):

$SV\ (ml.bt^{-1}) = (1\,000*\dot{Q}(l.min^{-1}))/\text{heart rate}\ (b.min^{-1})$

APPENDIX E

Example of computer print-out of one-line data

EXPERIMENT:
CONDITION:

SUBJECT NAME: (M) AGE: 26 MASS (KG): 64.7

MAXIMAL OXYGEN UPTAKE (ML/KG/MIN): 76

DATE: TIME OF DAY: 14:30

BAROMETRIC PRESSURE (MM HG): 716.6 RELATIVE HUMIDITY (%): 82

THE COLUMN HEADED 'TIME' SHOWS THE ELAPSED TIME FROM THE START OF THE EXPERIMENT TO THE START OF THE CURRENT SAMPLE PERIOD.
THE NUMBER IN BRACKETS IS THE SAMPLE DURATION IN SECONDS.

NO	TIME	R VO2 ML/KG	VO2 L/M	VCO2 L/M	R	VI(STPD) L/M	HR B/M	F BR/M	TEMP	FEO2	FECO2	VE-O2 L/100ML	VE-CO2	Q L/M	SV ML/B	O2 PU ML/B	VT L/BR	SPEED KM/HR
1	00:00:30(25)	25.2	1.63	1.39	.85	33.71	46	28	30.3	.1621	.0419	2.07	2.43	12.8	276	35.3	1.17	9.95
2	00:01:00(25)	30.9	2	1.73	.86	41.65	46	36	30.3	.1623	.0423	2.08	2.4	14.7	317	43.34	1.16	10.91
3	00:01:30(25)	27.1	1.75	1.52	.87	37.38	46	33	30.3	.1634	.0414	2.13	2.45	13.4	289	37.91	1.11	10.96
4	00:02:01(25)	33.1	2.14	1.86	.87	45.11	46	36	30.4	.1628	.0419	2.1	2.42	15.4	333	46.39	1.25	11.99
5	00:02:31(25)	33.1	2.14	1.93	.9	47.05	46	33	30.4	.1645	.0415	2.2	2.44	15.4	333	46.39	1.4	12.01
6	00:03:01(25)	33.3	2.16	1.94	.9	47.84	46	40	30.3	.165	.0411	2.22	2.47	15.5	335	46.65	1.17	13.02
7	00:03:31(25)	36.7	2.37	2.05	.86	51.33	46	40	30.3	.1641	.0406	2.16	2.5	16.6	359	51.36	1.26	12.99
8	00:04:01(25)	36.7	2.38	2.08	.88	52.31	46	43	30.3	.1648	.0405	2.2	2.51	16.6	359	51.43	1.21	13.96

APPENDIX F

Computer programme listing for the computation of a variety of derived performance data

```
]PRINT CHR$(9);"100N"

]LIST

100  REM  "MARATHON"
110  REM  COMPUTES VARIETY OF DERIVED PERFORMANCE DATA
120  REM  FROM A MAX TEST AND A SIMULATED MARATHON
130  REM  MAX TEST - 8 KM/HR PLUS 1 KM/H TO 17 THEN 1% EACH MINUTE TO MAX
140  REM  MARATHON - AT 92,5%OF VELOCITY ON MAX TEST AT VENT THRESHOLD
145 AA = 0:BB = 0:CC = 0:DD = 0:EE = 0
150  DEF  FN R1(X) =  INT (10 * X + 0.5) / 10
160  DEF  FN R2(X) =  INT (100 * X + 0.5) / 100
170  DEF  FN R3(X) =  INT (1000 * X + 0.5) / 1000
175 D$ =  CHR$ (4)
180  HOME
200  PRINT "INPUT THE FOLLOWING INFORMATION"
210  PRINT : PRINT "FROM THE MAX TEST: "
220  PRINT : INPUT "MAX VO2 (ML/KG/MIN): ";RMVO2
230  PRINT : INPUT "MAX HEART RATE (B/MIN): ";MHR
240  PRINT : INPUT "SUBJECT NAME? ";NAME$
250  PRINT : INPUT "SAMPLE(KM/HR): ";SP
260  PRINT : INPUT "STRIDE RATE(ST/MIN): ";SR
270  PRINT : INPUT "VO2 AT THIS SPEED(ML/KG/MIN): ";VO2
280  PRINT : INPUT "HEART RATE AT THIS SPEED(B/MIN): ";HR
290 PMVO2 = (VO2 / RMVO2) * 100
300 PHR = (HR / MHR) * 100
310 SL = ((SP * 1000) / 60) / SR
315 MO2 = VO2 / ((SP * 1000) / 60)
320  PRINT D$;"PR#1"
330  PRINT : PRINT "SUBJECT NAME: ";NAME$
340  PRINT "TREADMILL SPEED(KM/HR): "; INT (SP)
350  PRINT "%MAX VO2 = "; FN R1(PMVO2)
360  PRINT "% MAX HR = "; FN R1(PHR)
370  PRINT "STRIDE RATE = "; INT (SR)
380  PRINT "STRIDE LENGTH (M) = "; FN R3(SL)
385  PRINT "RELATIVE ECONOMY (VO2 PER VELOCITY)(ML/KG/M) = "; FN R3(MO2)
390  PRINT D$;"PR#0"
395  HOME
400  PRINT : INPUT "ANOTHER SAMPLE?(Y/N): ";A$
410  IF A$ = "Y" THEN 250
420  PRINT : INPUT "WHAT IS 100% OF MARATHON RUNNING TIME(MIN): ";RT
425  PRINT : INPUT "TRUE (MORNING) INITIAL BODY MASS(KG): ";IBM
430  PRINT : INPUT "PRE MARATHON (AFTERNOON)INITIAL BODY MASS(KG): ";MIM
432  PRINT D$;"PR#1"
435  PRINT : PRINT "10% OF TOTAL TIME = "; INT (RT * 0.1 + 0.5)
440  PRINT : PRINT "20% OF TOTAL TIME = "; INT (RT * 0.2 + 0.5)
445  PRINT : PRINT "30% OF TOTAL TIME = "; INT (RT * 0.3 + 0.5)
450  PRINT : PRINT "40% OF TOTAL TIME = "; INT (RT * 0.4 + 0.5)
455  PRINT : PRINT "50% OF TOTAL TIME = "; INT ((RT * 0.5) + 0.5)
460  PRINT : PRINT "60% OF TOTAL TIME = "; INT (RT * 0.6 + 0.5)
465  PRINT : PRINT "70% OF TOTAL TIME = "; INT (RT * 0.7 + 0.5)
470  PRINT : PRINT "80% OF TOTAL TIME = "; INT (RT * 0.8 + 0.5)
475  PRINT : PRINT "90% OF TOTAL TIME = "; INT (RT * 0.9 + 0.5)
480  PRINT : PRINT "100% OF TOTAL TIME = ";RT
490  PRINT D$;"PR#0"
500  HOME
510  PRINT "NOW WILL YOU PLEASE INPUT DATA AT PERCENTS OF TOTAL TIME?"
511  PRINT : PRINT "AT 10% OF TOTAL TIME: "
512  GOSUB 5000
513  PRINT : PRINT "10% OF TOTAL TIME AT SAMPLE ";SN
514  GOSUB 5100
```

```
520  PRINT : PRINT "AT 20% OF TOTAL TIME: "
530  GOSUB 5000
540  PRINT : PRINT "20% OF TOTAL TIME AT SAMPLE ";SN
550  GOSUB 5100
551  PRINT : PRINT "AT 30% OF TOTAL TIME: "
552  GOSUB 5000
553  PRINT : PRINT "30% OF TOTAL TIME AT SAMPLE ";SN
554  GOSUB 5100
560  PRINT : PRINT "AT 40% OF TOTAL TIME: "
570  GOSUB 5000
580  PRINT : PRINT "40% OF TOTAL TIME AT SAMPLE ";SN
590  GOSUB 5100
591  PRINT : PRINT "AT 50% OF TOTAL TIME: "
592  GOSUB 5000
593  PRINT : PRINT "50% OF TOTAL TIME AT SAMPLE ";SN
594  GOSUB 5100
600  PRINT : PRINT "AT 60% OF TOTAL TIME: "
610  GOSUB 5000
620  PRINT : PRINT "60% OF TOTAL TIME AT SAMPLE ";SN
630  GOSUB 5100
631  PRINT : PRINT "AT 70% OF TOTAL TIME: "
632  GOSUB 5000
633  PRINT : PRINT "70% OF TOTAL TIME AT SAMPLE ";SN
634  GOSUB 5100
640  PRINT : PRINT "AT 80% OF TOTAL TIME: "
650  GOSUB 5000
660  PRINT : PRINT "80% OF TOTAL TIME AT SAMPLE ";SN
670  GOSUB 5100
671  PRINT : PRINT "AT 90% OF TOTAL TIME: "
672  GOSUB 5000
673  PRINT : PRINT "90% OF TOTAL TIME AT SAMPLE ";SN
674  GOSUB 5100
680  PRINT : PRINT "AT 100% OF TOTAL TIME: "
690  GOSUB 5000
700  PRINT : PRINT "100% OF TOTAL TIME AT SAMPLE ";SN
710  GOSUB 5100
720  PRINT "NOW INPUT THE FOLLOWING DATA"
730  PRINT : PRINT "FOR ANTHROPOMETRIC ASSESSMENT:"
740  PRINT : INPUT "STATURE(CM): ";ST
750  PRINT : INPUT "TRICEP SKINFOLD(MM): ";TS
760  PRINT : INPUT "BICEP SKINFOLD(MM): ";BS
770  PRINT : INPUT "SUBSCAPULAR SKINFOLD(MM): ";SSS
780  PRINT : INPUT "SUPRAILIAC SKINFOLD(MM): ";SIS
790  PRINT : INPUT "ABDOMINAL SKINFOLD(MM): ";AS
800  PRINT : INPUT "THIGH SKINFOLD(MM): ";THS
810  PRINT : INPUT "CALF SKINFOLD(MM): ";CS
820  PRINT : INPUT "SHOULDER DIAMETER(CM): ";SD
830  PRINT : INPUT "TOTAL RUN LIQUID INTAKE(ML): ";LI
835  PRINT : INPUT "TOTAL URINE VOIDED(ML): ";UV
850  PRINT : INPUT "FINAL BODY MASS(KG): ";FBM
860 BD = 1.05637 - (0.00344 * THS) + (0.00121 * SD)
870 BV = (IBM * 1000) / BD
880 PF = (495 / BD) - 450
890  PRINT : INPUT "IS THE SUBJECT WHITE(Y/N):";B$
900  IF B$ = "Y" GOTO 915
910 PF = (437.4 / BD) - 392.8
915 W = 170.18 / ST
920 Z1 = (1 / 4.67) * ((CS * W) - 16)
930 Z2 = (1 / 4.47) * ((TS * W) - 15.4)
940 Z3 = (1 / 5.07) * ((SSS * W) - 17.2)
950 Z4 = (1 / 4.47) * ((SIS * W) - 15.2)
960 Z5 = (1 / 7.78) * ((AS * W) - 25.4)
970 Z6 = (1 / 8.33) * ((THS * W) - 27)
980 FM = ((((Z1 + Z2 + Z3 + Z4 + Z5 + Z6) / 6) * 3.25) + 12.13) / W ^ 3
990 FP = (FM * 100) / IBM
1000 DM = MIM - FBM
```

```
1005 DH = DM * 2430
1010 BSA = 0.007184 * ((IBM ^ 0.425) * (ST ^ 0.725))
1020 SV = BSA / (BV / 1000)
1030 SWR = ((((DM * 1000) + LI - UV) / 1.003) / 1000) * (60 / RT)
1040 RSR = SWR / BSA
1050  PRINT D$;"PR#1"
1060  PRINT : PRINT "BODY DENSITY(G/ML) = "; FN R3(BD)
1070  PRINT "BODY VOLUME(ML) = "; INT (BV)
1080  PRINT "BODY SURFACE AREA(SQ.M) = "; FN R3(BSA)
1090  PRINT "BODY SURFACE AREA TO VOLUME RATIO(SQ.M/L) = "; FN R3(SV)
1100  PRINT "FAT MASS(KG) = "; FN R1(FM)
1110  PRINT "PERCENT FAT = "; FN R1(PF)
1120  PRINT "PERCENT FAT(DRINKWATER) = "; FN R1(FP)
1130  PRINT "CHANGE IN MASS DURING RUN(KG) = "; FN R1(DM)
1140  PRINT "ABSOLUTE SWEAT RATE(L/HR) = "; FN R3(SWR)
1150  PRINT "RELATIVE SWEAT RATE(L/SQ M/HR) = "; FN R3(RSR)
1155  PRINT "DELTA BODY HEAT(KJ) = "; FN R1(DH)
1160  HOME
1170  PRINT D$;"PR#0"
1180  PRINT "NOW ENTER THE FOLLOWING DATA FOR"
1190  PRINT : PRINT "EVERY 10 MIN COLLECTION PERIOD:"
1200  GOSUB 5500
1210  GOSUB 5700
1220 AA = AA + E
1230 BB = BB + UC
1240 CC = CC + CG
1250 DD = DD + UF
1260 EE = EE + FG
1270  PRINT : PRINT "ACCUMULATED TOTALS TO MINUTE :";ET
1280  PRINT "ENERGY INPUT(KJ) = "; FN R1(AA)
1290  PRINT "CARBOHYDRATE ENERGY(KJ) = "; FN R1(BB)
1300  PRINT "CARBOHYDRATE USED (G) = "; FN R1(CC)
1310  PRINT "FAT ENERGY(KJ) = "; FN R1(DD)
1320  PRINT "FAT USED (G) = "; FN R1(EE)
1330  PRINT D$;"PR#0"
1340  HOME
1350  PRINT : INPUT "ANOTHER 10 MIN SAMPLE?(Y/N): ";C$
1360  IF C$ = "Y" GOTO 1200
1370  PRINT : PRINT : INPUT "CALCULATE DATA FOR ANOTHER SUBJECT?(Y/N): ";E$
1380  IF E$ = "Y" GOTO 100
1390  PRINT : PRINT : PRINT "THATS ALL FOR NOW,IM QUITING!"
1400  END
5000  PRINT : INPUT "SAMPLE NUMBER: ";SN
5010  PRINT : INPUT "VO2(ML/KG/MIN) AT THAT SAMPLE: ";V2
5020  PRINT : INPUT "HR(B/MIN) AT THAT SAMPLE: ";H2
5030  PRINT : INPUT "CHEST TEMP(DEG C): ";TC
5040  PRINT : INPUT "THIGH TEMP(DEG C): ";TT
5050  PRINT : INPUT "CORE TEMP(DEG C): ";TR
5060  PRINT D$;"PR#1
5070  RETURN
5100 PVO2 = (V2 / RMVO2) * 100
5110 RHR = (H2 / MHR) * 100
5120 MST = (0.6 * TC) + (0.4 * TT)
5130 MBT = (MST / 3) + ((2 * TR) / 3)
5140 BHC = 3.47 * MIM * MBT
5150  PRINT "%VO2 MAX = "; FN R1(PVO2)
5160  PRINT "%HR MAX = "; FN R1(RHR)
5170  PRINT "MEAN SKIN TEMPERATURE(DEG C) = "; FN R1(MST)
5180  PRINT "MEAN BODY TEMP(DEG C) = "; FN R1(MBT)
5190  PRINT "BODY HEAT CONTENT(KJ) = "; FN R1(BHC)
5200  PRINT D$;"PR#0"
5210  HOME
5220  RETURN
5500  PRINT : INPUT "ELAPSED TIME(MIN): ";ET
5510  PRINT : INPUT "VO2(L/MIN): ";VL
5520  PRINT : INPUT "RESP. EXCHANGE RATIO: ";R
```

```
5530  PRINT D$;"PR#1"
5540  RETURN
5700 CV = 19.616 + (((R - 0.707) / 0.293) * 1.511)
5710 GEC = VL * CV
5720 CU = ((R - 0.7) / 0.01) * 3.33
5730 FU = 100 - CU
5740 E = GEC * 10
5750 UC = E * (CU * 0.01)
5760 CG = UC / 17.5812
5770 UF = E * (FU * 0.01)
5780 FG = UF / 39.3484
5790  PRINT : PRINT "FOR THE 10 MINUTES UP TO MINUTE "; INT (ET)
5800  PRINT "THE ONE MINUTE GROSS ENERGY COST(KJ/MIN) = "; FN R1(GEC)
5810  PRINT "ENERGY EQUIVALENT(KJ/L O2) = "; FN R2(CV)
5820  PRINT "% CARBOHYDRATE UTILIZED = "; FN R1(CU)
5830  PRINT "%FAT UTILIZED = "; FN R1(FU)
5840  PRINT "ENERGY FOR 10 MIN(KJ) = "; FN R1(E)
5850  PRINT "CARBOHYDRATE ENERGY IN 10 MIN (KJ) = "; FN R1(UC)
5860  PRINT "CARBOHYDRATE USED IN 10 MIN(G) = "; FN R1(CG)
5870  PRINT "FAT ENERGY IN 10 MIN (KJ) = "; FN R1(UF)
5880  PRINT "FAT USED IN 10 MIN (G) = "; FN R1(FG)
5890  RETURN
```

APPENDIX G

BODY DENSITY AND PERCENT BODY FAT EQUATIONS

Percent body fat was estimated by using the skinfold technique. The equation to estimate body density was a population specific equation developed for use with marathon runners (Pollock et al 1977). Percentage body fat was estimated using the Siri formula in the case of the white runners (McArdle et al 1981) and a formula proposed for use with black subjects (Schutte et al 1984) in the case of the black runners.

the following equations were used:

1. Body density (BD) = 1.05637 - (0.00344 x thigh skinfold (mm))
   + (0.00121 x shoulder diameter (cm))

2. % body fat (white runners) = (495/BD) - 450

3. % body fat (black runners) = (437.4/BD) - 392.8

APPENDIX H

DETAILS OF METHODS USED FOR BLOOD ANALYSES

Venous blood samples (5 ml) were collected from the seated subject immediately prior to running the marathon and within five minutes of completion. These were drawn from an antecubital vein with a 21-gauge needle, identical techniques being used throughout the study. Blood samples collected were immediately placed in a refrigerator at 4°C until centrifuged at 4 000 revolutions.minute^{-1}. The supernatant (serum) was then stored in a freezer until analysis.

1. Blood Lactic Acid (Wroblewski and La Due (1956))

A 10 µg.ml^{-1} lactic acid standard solution was prepared by serially diluting 1 ml of a standard 85% lactic acid solution to 1 l and then further diluting 1.18 mls of the diluted solution to 100 ml in a volumetric flask. Standard lactic acid solutions ranging from 0.2 µg.ml^{-1} were then prepared by dilution of the 10 µg.ml^{-1} lactic acid standard with distilled water, as shown:

Serial dilution of 10 µg.ml^{-1} standard lactic acid solution showing volumes of standard solution and distilled water used.

Volume of 10 µg.ml^{-1} La Standard soln. (ml)	Volume of distilled H_2O (ml)
0.2	0.8
0.4	0.6
0.6	0.4
0.8	0.2
1.0	0

To the 0.1 ml of blood supernatant was added 2 mls distilled water, 1 ml of a previously prepared 20% copper sulphate ($CuSO_4$) solution, a further 1.9 mls distilled water and 1g Ca $(OH)_2$. The stoppered tubes were shaken

vigorously and then allowed to stand for 20 minutes. During this time they were shaken 3 - 4 times and then centrifuged for 10 minutes at 4 000 revolutions.minute^{-1}. One millilitre of supernatant with withdrawn. To this 1 drop of a previously prepared 4% $CuSO_4$ solution was added as well as to the six standard lactic acid solutions and a blank containing 1 ml of distilled water. A burette was used to add 6 ml concentrated sulphuric acid (H_2SO_4) to all the tubes. This was added slowly, mixing the contents during the addition. The tubes were then heated in a boiling water bath for 5 minutes and allowed to cool to below 20°C before adding 0.1 ml of p-hydroxydiphenyl reagent (prepared by dissolving 1g p-hydroxydiphenyl reagent in 10 ml 5% NaOH and diluting to 100 ml). This was added drop by drop, once again shaking the tubes during addition. The tubes were then placed in a water bath at 30°C for 30 minutes and shaken twice during this period. After 30 minutes had elapsed all tubes were reheated in a boiling water bath for 90 seconds and then allowed to cool. The absorbance of the standards and samples were read using a Spectronic 1000 spectrophotometer set at 540 nm. Standards were done in triplicate and the average absorbance used to plot a standard curve of absorbance vs lactic acid concentration. Serum samples were also in triplicate and the average absorbance used to determine the lactic acid concentration of the serum from the standard curve.

2. Blood Glucose (Parrott 1968))

A 100 $\mu g.ml^{-1}$ glucose standard solution was prepared by dissolving 10 mg of glucose in distilled water and making up to volume (100 ml). Standard glucose solutions ranging from 25 - 100 $\mu g.ml^{-1}$ were then prepared by dilution of the 100 $\mu g.ml^{-1}$ glucose standard with distilled water (shown on page 147. Preparation of 0.3 N barium hydroxide solution for use in analysis was by dissolving 9.5 g $Ba(OH)_2$ in distilled water and making up to volume (100 ml) with distilled water. Zinc Sulphate solution was prepared by dissolving 8.7 g $ZnSO_4$ and making up to volume (100 ml) with distilled

water. Somogyi-Nelson reagent "A" was prepared by dissolving 6.75 g Na_2CO_3, 6.75 g of K Na-tartrate, 5 g $NaHCO_3$ and 50 g Na_2SO_4 and making up to volume (250 ml) with distilled water. Somogyi-Nelson reagent "B" contained 7.5 g $CuSO_4$ made up to 50 ml with distilled water, to which was added 1 drop of concentrated H_2SO_4. Before use, "A" and "B" were mixed in the ratio of 25:1.

Serial dilution of a 100 $mg.ml^{-1}$ standard glucose solution showing volumes of standard solution and distilled water used.

Volume of 100 $\mu g.ml^{-1}$ Glucose standard soln. (ml)	Volume of distilled H_2O (ml)
0.25	2.75
0.50	2.50
0.75	2.25
1.00	2.00

A centrifuge tube was filled with 6.5 ml of water and 0.1 ml of blood supernatant added. To this was added 0.2 ml of the 0.3 N $Ba(OH)_2$ solution, the contents mixed and then 0.2 ml of the 0.3 N $ZnSO_4$ solution added before again mixing. Thirty minutes were allowed to elapse before the samples were centrifuged at 4 000 $revolutions.min^{-1}$ for 10 minutes. The supernatant was pipetted off and 3 ml of the mixed "A" and "B" Somogyi-Nelson reagent added to each tube of sample, standards and blank. All the tubes were then heated in a boiling water bath for 20 minutes, cooled, and 2 ml of arsenomolybdate reagent added to each. The absorbance of the standards (in triplicate) and samples (in duplicate) were read using a Spectronic 1000 spectrophotometer set at 540 nm. The average absorbance of the standards were used to plot a standard curve of absorbance vs glucose concentration. The average absorbance of the serum samples were then used to determine the blood glucose concentration from the standard curve.

APPENDIX I

BORG SCALE (English)

6.	
7.	very, very light
8	
9	very light
10	
11	fairly light
12	
13	somewhat hard
14	
15	hard
16	
17	very hard
18	
19	very, very hard
20	

APPENDIX J

BORG SCALE (Xhosa)

6	
7	Kancinci Kakhulu
8	
9	Kancini
10	
11	Kancinane noko
12	
13	nzinyana
14	
15	nzima
16	
17	nzima Kakhulu
18	
19	nzima Kakhulu Kanye
20	

APPENDIX K

SUBMAXIMAL AND $\dot{V}O_2$ MAX DATA FORM

Subject: _____

Marathon Time: _____ Hrs _____ Mins Age: ____ Mass: _____ kg

Sample No./ Remarks	Elapsed Time (Mins:Secs)	Steps $(.min^{-1})$	$km.hr^{-1}$	Sample No./ Remarks	Elapsed Time (Mins:Secs)	Steps $(.min^{-1})$	$km.hr^{-1}$; % grade
1.	0:30	X	X	24.	12:00	X	X
2.	1:00		8	25.	12:30	X	X
3.	1:30	X	X	26.	13:00		16
4.	2:00	X	X	27.	13:30	X	X
5.	2:30		9	28.	14:00	X	X
6.	3:00	X	X	29.	14:30		17
7.	3:30	X	X	30.	15:00	X	X
8.	4:00		10	31.	15:30	X	X
9.	4:30	X	X	32.	16:00	X	
10.	5:00	X	X	33.	16:30	X	
11.	5:30		11	34.	17:00	X	
12.	6:00	X	X	35.	17:30	X	
13.	6:30	X	X	36.	18:00	X	
14.	7:00		12	37.	18:30	X	
15.	7:30	X	X	38.	19:00	X	
16.	8:00	X	X	39.	19:30	X	
17.	8:30		13	40.	20:00	X	
18.	9:00	X	X	41.	20:30	X	
19.	9.30	X	X	42.	21:00	X	
20.	10:00		14	43.	21:30	X	
21.	10:30	X	X	44.	22:00	X	
22.	11:00	X	X	45.	22:30	X	
23.	11:30		15	46.	23:00	X	

APPENDIX L
ANTHROPOMETRIC DATA FORM

Subject:

Mass:

Stature: _____ cm

Skinfolds (mm)

Triceps: _____

Biceps: _____

Subscapular: _____

Supra-iliac: _____

Abdominal: _____

Front Thigh: _____

Medial Calf: _____

Trochanterion: _____ cm

Shoulder Diameter: _____ cm

APPENDIX M

SIMULATED MARATHON DATA FORM

Subject: _____ Best Time: ____ Hrs ____ Mins

TM Speed: ____ $km.hr^{-1}$: ____ $m.min^{-1}$ Counts/30 Sec: _____ VT: _____

Final Mass: _____ kg

Sample No/ Remarks	Elapsed Time (Mins)	Steps ($.min^{-1}$)	Temperature (°C) Rectal	Chest	Thigh	RPE G	RPE L	Fluid Intake (ml)
1.								
2.								
3.								
4.								
5.								
6.								
7.								
8.								
9.								
10.								
11.								
12								
13.								
14.								

APPENDIX N

Descriptive statistics of measured and derived cardiopulmonary and related data at the submaximal running speed of 16 $km.hr^{-1}$.

Table IX

Category	Subject	$\dot{V}O_2$ ($ml.kg.^{-1}min^{-1}$)	$\dot{V}CO_2$ ($l.min^{-1}$)	% $\dot{V}O_2$ max	R value	H/R ($b.min^{-1}$)	% H/R max	$\dot{Q}$ ($l.min^{-1}$)	$\dot{Q}$ ($ml.kg.^{-1}min^{-1}$)	Step Rate ($Steps.min^{-1}$)
BLACK (n = 9)	TG	48.8	2.94	90.0	1.05	-	-	18.4	319.4	184
	GN	53.1	3.15	86.9	1.01	-	-	20.2	341.8	186
	CM	50.4	2.87	84.3	1.01	155	85.2	18.4	328.0	176
	EM	43.9	3.34	89.2	1.13	-	-	19.8	294.6	168
	EN	61.6	4.25	98.4	1.15	-	-	23.1	385.6	184
	MN	52.2	2.90	74.6	0.96	174	84.3	19.4	336.8	184
	WM	44.8	2.38	67.3	1.00	-	-	15.9	298.3	180
	MB	54.6	3.15	84.3	0.96	167	89.3	21.1	350.5	180
	VK	49.6	3.25	88.6	1.01	170	96.6	21.0	323.6	176
	$\bar{X}$	51.0	3.14	84.4	1.03	167	88.9	19.7	331.0	180
	SD	5.3	0.48	9.1	0.07	8	5.6	2.0	27.6	6
WHITE (n = 10)	SG	48.4	3.76	76.3	0.93	170	96.0	26.6	317.8	176
	RB	50.0	3.29	81.2	0.99	181	100.0	21.7	326.8	188
	RS	51.0	3.70	79.6	1.02	158	84.5	23.5	331.0	192
	HE	48.9	3.05	71.2	0.98	152	83.5	20.5	320.3	188
	CG	50.0	2.82	78.0	0.99	171	88.1	18.5	326.3	192
	BE	53.6	4.14	89.3	0.95	162	89.5	28.0	344.0	184
	RJ	50.4	3.87	80.8	1.02	161	91.0	24.7	328.5	172
	KS	47.7	3.30	70.4	0.96	163	91.1	22.7	314.0	180
	SB	48.7	3.75	84.3	1.00	157	87.7	24.6	319.1	176
	AB	50.7	3.08	74.7	0.96	161	84.7	20.8	329.6	176
	$\bar{X}$	49.9	3.48	78.6	0.98	164	89.6	23.2	325.7	182
	SD	1.7	0.40	5.8	0.03	8	5.2	2.9	8.6	8
						*		*		

* $p < 0.05$

APPENDIX O

Descriptive statistics of measured and derived ventilatory data at the submaximal running speed of 16 $km.hr^{-1}$.

Table X

Category	Subject	$\dot{V}_I$ ($l.min^{-1}$)	$\dot{V}_I$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	$\dot{V}_A$ ($l.min^{-1}$)	$\dot{V}_A$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	f ($breaths.min^{-1}$)	$\bar{V}_T$ ($l.breath^{-1}$)	$\bar{V}_T$ ($ml.kg.^{-\frac{2}{3}}breath^{-1}$)
BLACK (n = 9)	TG	77.69	5.21	69.44	4.66	55	1.41	95
	GN	95.33	6.27	82.43	5.42	86	1.10	72
	CM	73.92	5.03	65.37	4.45	57	1.28	87
	EM	72.74	4.41	65.21	3.95	50	1.44	87
	EN	103.10	6.74	93.05	6.08	67	1.53	100
	MN	62.85	4.22	56.10	3.77	45	1.38	93
	WM	64.07	4.51	54.77	3.86	62	1.03	73
	MB	66.86	4.34	59.66	3.87	48	1.39	90
	VK	82.12	5.10	73.12	4.54	60	1.37	85
	$\bar{X}$	77.63	5.09	68.79	4.51	59	1.33	87
	SD	13.87	0.88	12.55	0.79	12	0.16	9
WHITE (n = 10)	SG	84.86	4.42	79.06	4.14	36	2.35	123
	RB	77.20	4.71	70.45	4.30	45	1.69	103
	RS	84.69	4.95	77.19	4.51	50	1.68	98
	HE	64.86	4.05	57.66	3.60	48	1.35	84
	CG	72.02	4.87	63.77	4.31	55	1.30	88
	BE	89.39	4.75	82.19	4.37	48	1.86	99
	RJ	80.22	4.51	74.82	4.20	36	2.23	125
	KS	61.39	3.55	56.74	3.28	31	1.97	113
	SB	76.59	4.23	69.84	3.86	45	1.68	93
	AB	72.51	4.59	64.71	4.10	52	1.37	87
	$\bar{X}$	76.33	4.46	69.64	4.07	45	1.75	101
	SD	8.88	0.42	8.82	0.38	8	0.36	14
						*	*	

* $p < 0.05$

APPENDIX P

Descriptive statistics of measured and derived cardiopulmonary and related data at $\dot{V}0_2$ max.

Table XI

Category	Subject	$\dot{V}O_2$ (ml.kg.$^{-1}$min^{-1})	$\dot{V}CO_2$ (l.min^{-1})	R value	H/R (b.min^{-1})	$\dot{Q}$ (l.min^{-1})	$\dot{Q}$ (ml.kg.$^{-1}$min^{-1})
BLACK (n = 9)	TG	55.9	3.47	1.10	193	20.5	355.9
	GN	61.1	3.91	1.08	183	22.7	384.1
	CM	59.8	3.71	1.11	174	21.1	376.1
	EM	49.2	4.68	1.41	185	21.6	321.4
	EN	62.7	4.34	1.16	188	23.5	392.3
	MN	70.0	4.33	1.07	186	24.8	430.6
	WM	66.6	3.81	1.07	186	22.0	412.8
	MB	64.8	4.19	1.07	185	24.3	403.7
	VK	56.0	3.94	1.08	176	23.2	357.5
	$\bar{X}$	60.7	4.04	1.13	186	22.6	381.6
	SD	6.0	0.37	0.11	6	1.5	33.3
WHITE (n = 10)	SG	63.4	5.94	1.12	177	33.1	395.5
	RB	61.6	4.53	1.11	185	25.7	387.0
	RS	64.1	5.50	1.21	186	28.3	398.6
	HE	64.7	4.71	1.14	179	25.7	401.6
	CG	64.1	4.08	1.12	194	22.7	400.4
	BE	60.0	5.27	1.08	181	30.8	378.4
	RJ	62.4	5.08	1.08	170	29.4	391.0
	KS	65.9	5.32	1.12	175	29.6	409.4
	SB	57.8	5.12	1.15	179	28.3	367.1
	AB	67.9	4.74	1.11	182	26.5	420.0
	$\bar{X}$	63.2	5.03	1.12	183	28.0	394.9
	SD	2.8	0.53	0.04	5	3.0	15.1

* $p < 0.05$ *

APPENDIX Q

Descriptive statistics of measured and derived ventilatory data at $\dot{V}O_2$ max

Table XII

Category	Subject	$\dot{V}_I$ ($l.min^{-1}$)	$\dot{V}_I$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	$\dot{V}_A$ ($l.min^{-1}$)	$\dot{V}_A$ ($l.kg.^{-\frac{2}{3}}min.^{-1}$)	f ($breaths.min^{-1}$)	$\bar{V}_T$ ($l.breath^{-1}$)	$\bar{V}_T$ ($ml.kg.^{-\frac{2}{3}}breath^{-1}$)
BLACK (n = 9)	TG	88.18	5.92	78.88	5.29	62	1.41	95
	GN	104.05	6.85	93.7	6.16	69	1.49	98
	CM	92.56	6.30	82.51	5.61	67	1.38	94
	EM	96.71	5.86	88.16	5.34	57	1.68	102
	EN	102.87	6.72	93.27	6.10	64	1.59	104
	MN	87.82	5.89	80.32	5.39	50	1.74	117
	WM	99.09	6.98	89.79	6.32	62	1.59	112
	MB	82.62	5.36	74.82	4.86	52	1.38	90
	VK	97.73	6.07	88.73	5.51	60	1.63	101
	$\bar{X}$	94.63	6.22	85.58	5.62	60	1.54	101
	SD	7.31	0.54	6.68	0.48	6	0.13	8
WHITE (n = 10)	SG	136.28	7.14	128.48	6.73	52	2.58	135
	RB	105.52	6.43	97.27	5.93	55	1.91	116
	RS	120.73	7.06	111.43	6.52	62	1.93	113
	HE	107.26	6.70	97.96	6.12	62	1.72	108
	CG	116.54	7.87	105.95	7.16	67	1.73	117
	BE	133.40	7.10	122.60	6.52	72	1.85	98
	RJ	113.57	6.38	106.07	5.96	50	2.25	126
	KS	106.21	6.14	98.41	5.69	52	2.01	116
	SB	104.21	5.76	97.46	5.38	45	2.29	127
	AB	120.70	7.64	112.15	7.10	57	2.10	133
	$\bar{X}$	116.44	6.82	107.78	6.31	57	2.04	119
	SD	11.47	0.66	11.00	0.59	8.3	0.27	11
		*	*	*	*		*	*

* $p < 0.05$

APPENDIX R

Descriptive statistics of submaximal performance characteristics.

Table XIII

Category	Subject	$\dot{V}O_2$ (est.)** in Best Race ($ml.kg^{-1}.min^{-1}$)	% $\dot{V}O_2$ max in Best Race	VT ($ml.kg^{-1}.min^{-1}$)	%VT During Sim.*** Marathon	H/R at VT ($b.min^{-1}$)	% H/R max at VT	MPQ	Gross Energy Cost ($KJ.kg^{-1}$)
BLACK (n = 9)	TG	48.8	90.0	44.3	91.6	-	-	1.00	152
	GN	56.4	92.3	53.0	94.0	-	-	1.06	175
	CM	50.4	84.3	41.8	98.8	152	83.5	1.00	158
	EM	43.4	88.2	38.5	98.4	-	-	0.99	128
	EN	60.7	96.8	57.3	92.1	183	97.3	0.98	177
	MN	54.7	78.1	52.2	85.1	174	87.9	1.05	122
	WM	58.5	87.8	53.7	97.8	-	-	1.30	138
	MB	60.9	94.0	60.4	81.5	173	92.5	1.12	114
	VK	50.0	89.3	49.0	91.6	169	96.0	1.01	-
	$\bar{X}$	53.8	89.0	50.0	92.3	170	91.4	1.06	146
	SD	6.0	5.5	7.3	5.9	11	5.7	0.10	24
WHITE (n = 10)	SG	50.2	79.2	44.8	92.4	162	91.5	1.04	151
	RB	48.6	78.9	47.4	93.5	170	91.9	0.97	162
	RS	53.0	82.7	45.9	93.2	150	80.2	1.04	167
	HE	49.3	76.2	44.1	85.5	137	75.3	1.07	131
	CG	53.9	84.1	50.0	91.6	171	88.0	1.08	162
	BE	52.3	87.2	47.4	87.1	158	87.3	0.98	153
	RJ	51.8	83.0	46.6	89.5	155	87.6	1.03	122
	KS	53.4	81.09	50.9	92.9	159	88.8	1.15	155
	SB	47.4	82.0	43.7	89.5	144	80.4	0.97	153
	AB	54.9	80.9	51.1	95.5	168	88.9	1.08	162
	$\bar{X}$	51.5	81.5	47.2	91.1	157	86.0	1.04	152
	SD	2.5	3.1	2.7	3.1	11.2	5.5	0.05	14

* $p < 0.05$
** estimated $\dot{V}O_2$ during best race
*** simulated

APPENDIX S

Descriptive statistics of pre/post marathon data:-
Blood lactic acid and glucose concentrations and sweat rates.

Table XIV

Category	Subject	Pre-run Lactic Acid ($mmol.l^{-1}$)	Post-run Lactic Acid ($mmol.l^{-1}$)	Pre-run Glucose ($mmol.l^{-1}$)	Post-run Glucose ($mmol.l^{-1}$)	Δ Mass During Run (kg)	Absolute Sweat Rate ($l.hr^{-1}$)	Rel. Sweat Rate ($l.m^{-2}.hr^{-1}$)	% Mass Loss
BLACK (n = 9)	TG	0.98	1.29	10.2	7.4	1.7	0.897	0.547	3.0
	GN	0.93	1.33	10.7	10.4	1.8	0.799	0.491	3.0
	CM	0.93	1.07	11.1	10.7	0.8	0.931	0.558	1.4
	EM	1.16	1.73	11.1	10.9	2.1	1.234	0.662	3.1
	EN	1.16	1.60	10.7	7.4	3.0	1.682	0.999	5.0
	MN	-	-	-	-	1.3	1.090	0.648	2.2
	WM	1.02	1.02	10.9	10.0	2.0	0.989	0.603	3.8
	MB	1.02	1.07	11.1	11.1	1.3	1.029	0.620	2.2
	VK	-	-	-	-	0.1	0.150	0.083	0.6
	$\bar{X}$	1.02	1.30	0.3	1.5	1.6	0.978	0.579	2.7
	SD	0.08	0.26	0.3	1.5	0.8	0.404	0.236	1.3
WHITE (n = 10)	SG	0.93	1.42	10.4	10.0	3.1	1.667	0.789	3.7
	RB	1.02	1.91	10.2	10.0	1.5	0.997	0.531	2.3
	RS	0.89	1.38	10.7	12.2	1.9	1.083	0.571	2.7
	HE	0.98	1.33	11.1	10.9	0.9	0.658	0.360	1.4
	CG	0.93	1.82	10.7	10.9	0.7	0.756	0.467	1.2
	BE	1.02	1.47	10.6	10.9	2.8	1.429	0.707	3.4
	RJ	1.11	1.60	10.7	10.9	2.0	1.457	0.730	2.6
	KS	1.16	1.78	10.4	12.6	2.5	1.638	0.860	3.4
	SB	0.93	1.73	10.2	9.8	2.3	1.511	0.747	3.0
	AB	1.11	1.47	8.9	10.4	2.0	1.208	0.675	3.1
	$\bar{X}$	1.01	1.59	10.4	10.9	2.0	1.240	0.644	2.7
	SD	0.09	0.20	0.6	0.9	0.7	0.358	0.157	0.8

* $p < 0.05$)

APPENDIX T

Descriptive statistics of anthropometric and related data.

Table XV

Category	Subject	Body Density ($g.ml^{-1}$)	Body Volume (ml)	BSA (m^2)	BSA/Volume	% fat	Trochanterion Height (cm)	Trochanterion Ht. as % Stature	Step Length as % Stature ($16km.hr^{-1}$)
BLACK (n = 9)	TG	1.082	53259	1.640	0.031	11.6	86.0	51.7	87.1
	GN	1.081	54690	1.627	0.030	12.0	85.1	52.5	89.5
	CM	1.080	51924	1.668	0.032	12.0	93.4	54.0	87.6
	EM	1.088	61745	1.864	0.030	9.1	93.0	51.3	87.6
	EN	1.086	55155	1.684	0.031	10.0	87.2	51.7	86.0
	MN	1.087	53010	1.682	0.032	9.8	89.9	52.2	84.2
	WM	1.084	49152	1.641	0.033	10.6	95.3	54.7	85.0
	MB	1.085	55506	1.659	0.030	10.5	87.9	53.4	90.0
	VK	1.077	60282	1.806	0.030	13.5	92.3	52.1	85.0
	$\bar{X}$	1.083	54969	1.697	0.031	11.0	90.0	52.6	86.9
	SD	0.004	3944	0.082	0.001	1.4	3.6	1.2	1.9
WHITE (n = 10)	SG	1.083	77291	2.114	0.027	7.1	101.6	53.6	79.9
	RB	1.074	61801	1.877	0.030	10.7	93.1	50.5	76.7
	RS	1.059	67015	1.897	0.028	17.2	94.5	52.5	77.2
	HE	1.081	59208	1.828	0.031	7.9	93.0	51.2	78.1
	CG	1.077	52627	1.619	0.031	9.4	85.0	51.5	84.2
	BE	1.076	75648	2.020	0.027	10.0	95.9	53.0	80.1
	RJ	1.066	70564	1.996	0.028	14.5	97.1	52.0	83.1
	KS	1.081	66894	1.904	0.028	8.0	94.0	52.5	82.8
	SB	1.060	72721	2.024	0.028	16.9	101.2	54.0	80.8
	AB	1.077	58587	1.789	0.031	9.6	92.9	52.2	85.2
	$\bar{X}$	1.073	66235	1.907	0.029	11.1	94.8	52.3	80.8
	SD	0.009	8065	0.142	0.002	1.4	4.7	1.1	2.8
		*	*	*	*		*		*

* $p < 0.05$

APPENDIX U

Descriptive statistics of measured and derived cardiopulmonary ventilatory, thermal, gait, and RPE responses during the time-course of a simulated marathon run.

Table XVI: $\dot{V}O_2$ ($ml.kg.^{-1}min^{-1}$) responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	41.0	40.2	41.6	37.2	37.7	40.3	41.3	42.0	41.6	43.1
	GN	47.7	48.2	47.8	49.4	50.0	52.6	51.5	51.2	52.0	53.4
	CM	40.1	42.5	41.6	37.5	39.4	42.0	40.1	42.2	43.8	44.0
	EM	39.4	38.5	37.4	36.8	36.7	37.3	38.3	40.3	36.1	38.3
	EN	56.3	52.8	53.0	52.9	53.6	49.7	51.1	50.7	53.3	54.4
	MN	45.0	45.6	44.0	45.6	46.2	42.1	42.6	43.1	45.7	44.3
	WM	50.1	54.7	54.4	53.3	53.7	52.6	54.2	50.2	49.5	50.2
	MB	47.4	50.7	52.6	47.6	50.0	50.2	49.4	48.1	48.6	47.6
	$\bar{X}$	45.9	46.7	46.6	45.0	45.9	45.9	46.1	46.0	46.3	46.9
	SD	5.8	5.9	6.3	7.0	7.1	6.1	6.1	4.5	5.7	5.5
WHITE (n = 10)	SG	43.4	44.3	41.2	41.2	41.1	41.3	39.8	39.5	38.5	43.5
	RB	42.6	42.2	41.8	45.3	47.8	44.0	41.6	45.7	45.0	46.9
	RS	42.1	43.7	43.4	42.4	40.9	42.7	41.5	44.4	43.5	43.6
	HE	37.3	36.5	37.9	37.8	39.7	37.7	38.4	37.5	36.4	37.7
	CG	43.8	43.3	41.5	44.4	45.5	45.7	49.3	48.7	48.2	47.6
	BE	40.1	40.4	39.5	40.2	41.0	42.0	42.4	43.8	41.3	41.9
	RJ	41.8	41.1	43.5	40.4	40.7	41.8	40.9	41.2	41.5	44.0
	KS	47.1	47.0	44.5	45.9	45.9	46.1	48.4	48.7	49.1	50.1
	SB	38.5	40.9	37.4	37.0	38.9	39.2	40.1	40.0	38.7	40.0
	AB	47.1	47.1	48.7	48.0	48.2	49.5	50.5	48.9	50.1	49.4
	$\bar{X}$	42.4	42.7	41.9	42.3	43.0	43.0	43.3	43.8	43.2	44.5
	SD	3.3	3.2	3.3	3.6	3.5	3.5	4.4	4.2	4.8	4.0

Table XVII: % $\dot{V}O_2$ max during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	75.6	74.2	76.8	68.6	69.6	74.4	76.2	77.5	76.8	79.5
	GN	78.1	78.9	78.2	80.9	81.8	86.1	84.3	83.8	85.1	87.4
	CM	67.1	71.1	69.6	62.7	65.9	70.2	67.1	70.6	73.2	73.6
	EM	80.1	78.3	76.0	74.8	74.6	75.8	77.8	81.9	73.4	77.8
	EN	92.6	86.8	87.2	87.0	88.2	81.7	84.0	83.4	87.7	89.5
	MN	64.3	65.1	62.9	65.1	66.0	60.1	61.6	65.3	62.0	63.3
	WM	74.8	81.6	81.2	79.6	80.1	78.5	80.9	75.4	74.3	75.4
	MB	73.1	78.2	81.2	73.5	77.2	77.5	76.2	74.2	75.0	73.5
	$\bar{X}$	76.0	77.0	77.0	74.0	76.0	76.0	76.0	77.0	76.0	78.0
	SD	8.8	6.8	7.3	8.3	7.9	8.0	7.9	6.6	8.0	8.4
WHITE (n = 10)	SG	68.5	69.9	65.0	65.0	65.0	65.1	62.8	62.3	60.7	68.6
	RB	69.2	68.5	67.9	73.5	77.6	71.4	67.5	74.2	73.1	76.1
	RS	65.7	68.2	67.7	66.1	63.8	66.6	64.7	69.3	67.9	68.0
	HE	54.3	53.1	55.2	55.0	57.8	54.9	55.9	54.6	53.9	54.9
	CG	68.3	67.6	64.7	69.3	71.0	71.3	76.9	76.0	75.2	74.3
	BE	66.8	67.3	67.0	67.0	68.3	70.0	70.7	73.0	68.8	69.8
	RJ	67.0	65.9	69.7	64.7	65.2	67.0	65.5	66.0	66.5	70.5
	KS	69.5	69.3	65.6	67.7	67.7	68.0	71.4	71.8	72.4	73.9
	SB	66.6	70.8	64.7	64.0	67.3	67.8	69.4	69.2	67.0	69.2
	AB	69.4	69.4	71.7	70.7	71.0	72.9	74.4	72.0	73.8	72.8
	$\bar{X}$	66.6	67.0	66.0	66.0	68.0	67.5	68.0	69.0	68.0	70.0
	SD	4.6	5.1	4.5	5.0	5.3	5.0	5.9	6.3	6.7	5.9

Table XVIII : R ratio responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	0.99	0.97	0.96	1.06	1.03	0.93	0.91	0.91	0.91	0.85
	GN	0.96	0.94	0.93	0.91	0.87	0.87	0.87	0.85	0.83	0.81
	CM	1.03	0.98	1.01	1.12	1.03	0.98	1.01	0.95	0.93	0.91
	EM	1.01	0.97	0.97	1.00	1.04	0.95	0.92	0.85	0.98	0.95
	EN	0.94	1.01	0.94	0.99	0.92	0.98	0.95	0.98	0.92	0.93
	MN	0.98	0.96	1.00	0.96	0.93	1.02	1.00	0.96	1.00	0.96
	WM	0.98	0.85	0.85	0.87	0.86	0.85	0.85	0.84	0.91	0.93
	MB	1.00	0.99	0.95	0.99	0.96	0.93	0.97	0.97	1.01	1.00
	$\bar{X}$	0.99	0.96	0.95	0.99	0.96	0.94	0.94	0.91	0.93	0.92
	SD	0.03	0.05	0.05	0.08	0.07	0.06	0.06	0.06	0.06	0.06
WHITE (n = 10)	SG	0.91	0.89	0.92	0.92	0.89	0.85	0.87	0.93	0.93	0.84
	RB	0.95	0.92	0.92	0.84	0.87	0.85	0.99	0.88	0.90	0.83
	RS	0.92	0.92	0.87	0.91	0.93	0.92	0.92	0.88	0.92	0.89
	HE	0.94	0.94	0.90	0.92	0.85	0.84	0.86	0.82	0.84	0.83
	CG	1.01	1.03	1.07	0.98	0.94	0.92	0.88	0.87	0.89	0.92
	BE	0.97	0.94	0.92	0.91	0.88	0.85	0.85	0.84	0.88	0.86
	RJ	1.04	1.01	0.94	1.00	0.99	0.94	0.95	0.98	0.94	0.95
	KS	0.94	0.93	0.93	0.93	0.88	0.88	0.90	0.90	0.88	0.87
	SB	0.92	0.85	0.90	0.95	0.92	0.91	0.89	0.91	0.86	0.86
	AB	1.00	0.97	0.95	0.94	0.93	0.92	0.88	0.91	0.89	0.92
	$\bar{X}$	0.96	0.94	0.93	0.93	0.91	0.89	0.90	0.89	0.89	0.88
	SD	0.04	0.05	0.05	0.04	0.04	0.04	0.04	0.05	0.03	0.04

Table XIX: H/R responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	150	154	154	153	149	148	154	154	160	160
	GN	166	166	167	168	168	172	172	182	179	181
	CM	131	137	138	139	137	142	143	147	150	158
	EM	145	143	149	149	145	143	145	147	150	153
	EN	168	172	164	163	160	155	160	164	168	168
	MN	153	150	149	149	147	147	147	149	152	157
	WM	178	176	178	178	177	178	185	176	178	180
	MB	158	161	164	165	166	167	168	170	167	170
	$\bar{X}$	156	157	158	158	156	157	159	161	163	166
	SD	15	14	13	13	14	14	15	14	12	11
WHITE (n = 10)	SG	140	137	137	133	133	133	135	137	138	139
	RB	157	158	155	153	152	156	158	158	160	161
	RS	150	154	155	159	157	161	162	174	169	176
	HE	136	138	143	140	143	137	137	141	144	151
	CG	168	167	171	165	169	170	174	177	183	183
	BE	149	150	146	149	152	158	165	165	170	174
	RJ	153	159	159	170	168	172	175	175	176	179
	KS	147	147	149	146	148	150	151	153	154	157
	SB	134	137	137	135	135	140	138	138	143	144
	AB	142	139	143	141	142	143	144	149	151	155
	$\bar{X}$	148	149	150	149	150	152	154	157	159	162
	SD	10	11	11	13	12	14	15	16	15	15

Table XX: % H/R max during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	78	80	80	79	77	77	80	80	83	83
	GN	91	91	91	92	92	94	94	100	98	99
	CM	72	75	76	76	75	78	79	81	82	87
	EM	78	77	81	81	78	77	78	80	81	83
	EN	89	92	87	87	85	82	85	87	89	89
	MN	77	76	75	75	74	74	74	75	77	79
	WM	96	95	96	95	96	96	100	95	96	97
	MB	85	86	88	88	89	89	90	91	89	91
	$\bar{X}$	83	84	84	84	83	84	85	86	87	89
	SD	8	8	7	7	8	8	9	9	7	7
WHITE (n = 10)	SG	79	77	77	75	76	75	76	77	78	79
	RB	87	87	86	85	84	86	87	88	88	89
	RS	80	82	83	85	84	86	87	87	93	94
	HE	75	76	79	77	79	75	75	78	79	83
	CG	87	86	88	85	87	88	90	91	94	94
	BE	82	83	83	82	84	87	91	91	94	96
	RJ	86	90	90	96	95	97	99	99	100	100
	KS	82	82	83	82	83	84	84	86	86	88
	SB	75	77	77	75	75	78	77	77	80	80
	AB	75	73	75	74	75	75	76	78	80	82
	$\bar{X}$	81	81	82	82	82	83	84	85	87	89
	SD	5	6	5	7	6	7	8	8	8	7

Table XXI: SV ($ml.beat^{-1}$) responses during a simulated marathon

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	107	102	105	96	100	106	104	105	101	103
	GN	112	113	112	114	115	117	115	108	108	110
	CM	116	117	114	104	110	112	107	108	109	104
	EM	125	124	117	116	119	121	121	125	113	116
	EN	127	118	124	124	128	124	124	119	121	123
	MN	112	116	113	116	119	111	113	116	110	108
	WM	98	106	105	103	103	103	100	97	97	97
	MB	118	122	123	114	117	117	115	111	114	110
	$\bar{X}$	114	115	114	111	114	114	112	111	109	109
	SD	9	8	7	9	9	7	8	9	8	8
WHITE (n = 10)	SG	173	180	170	175	176	177	168	164	162	174
	RB	122	120	121	131	138	126	118	127	124	128
	RS	133	134	133	126	124	126	122	120	121	116
	HE	121	117	117	119	121	121	123	118	113	111
	CG	98	98	93	101	101	100	105	102	97	96
	BE	149	149	151	149	149	146	141	144	135	131
	RJ	138	132	137	122	123	123	119	121	121	123
	KS	152	152	144	150	148	146	151	150	150	149
	SB	152	156	146	146	153	147	152	152	144	146
	AB	138	141	140	140	140	142	143	135	136	131
	$\bar{X}$	138	138	135	136	137	135	134	133	130	131
	SD	21	23	21	21	21	21	20	19	19	22

Table XXII: $\dot{Q}$ ($l.min^{-1}$) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	16.0	15.8	16.2	14.9	15.0	15.8	16.1	16.3	16.2	16.6
	GN	18.6	18.8	18.7	19.1	19.3	20.1	19.8	19.7	19.3	19.9
	CM	15.4	16.1	15.8	14.6	15.2	15.9	15.4	16.0	16.5	16.5
	EM	18.2	17.9	17.5	17.3	17.3	17.5	17.8	18.8	17.0	17.8
	EN	21.5	20.4	20.5	20.4	20.7	19.4	19.9	19.7	20.6	20.9
	MN	17.3	17.4	17.0	17.5	17.7	16.4	16.7	17.5	16.8	17.1
	WM	17.4	18.7	18.6	18.3	18.4	18.1	18.5	17.2	17.4	17.5
	MB	18.8	19.9	20.4	18.9	19.6	19.7	19.4	19.0	19.2	18.9
	$\bar{X}$	17.9	18.1	18.1	17.6	17.9	17.9	18.0	18.0	17.9	18.2
	SD	1.9	1.7	1.8	3.0	2.0	1.7	1.7	1.5	1.6	1.6
WHITE (n = 10)	SG	24.4	24.8	23.5	23.4	23.4	23.5	22.9	22.7	22.3	24.4
	RB	19.1	18.9	18.8	20.0	20.9	19.6	18.7	20.1	19.9	20.6
	RS	20.1	20.7	20.6	20.2	19.7	20.3	19.9	21.0	20.6	20.7
	HE	16.6	16.4	16.8	16.8	17.4	16.8	17.0	16.7	16.3	16.8
	CG	16.7	16.5	16.0	16.8	17.2	17.2	18.3	18.1	17.9	17.8
	BE	22.3	22.5	22.1	22.4	22.7	23.2	23.3	23.9	22.9	23.1
	RJ	21.3	21.1	22.0	20.7	20.9	21.3	20.9	21.1	21.2	22.2
	KS	22.5	22.5	21.5	22.0	22.0	22.1	23.0	23.1	23.2	23.6
	SB	20.5	21.5	20.1	19.9	20.7	20.8	21.2	21.1	20.6	21.1
	AB	19.6	19.6	20.1	19.9	20.0	20.4	20.7	20.2	20.6	20.4
	$\bar{X}$	20.3	20.5	20.2	20.2	20.5	20.5	20.6	20.8	20.6	21.1
	SD	2.5	2.7	2.4	2.2	2.0	2.3	2.1	2.2	2.1	2.4

Table XXIII: $\dot{Q}$ ($ml.kg^{-1}.min^{-1}$) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	278	274	281	259	260	274	280	283	281	288
	GN	315	318	316	323	327	340	335	333	327	337
	CM	275	287	282	260	271	283	275	285	294	294
	EM	271	266	260	257	257	260	265	275	253	265
	EN	359	341	342	341	346	324	332	329	344	349
	MN	300	302	295	304	307	288	290	304	292	297
	WM	327	351	349	343	345	340	347	323	327	328
	MB	312	331	339	314	326	327	322	316	319	314
	$\bar{X}$	305	309	308	300	305	305	306	306	305	309
	SD	30	31	33	37	37	32	32	23	30	28
WHITE (n = 10)	SG	292	296	281	280	280	281	271	266	266	292
	RB	288	285	283	301	315	295	285	303	300	310
	RS	283	292	291	285	278	286	280	296	290	292
	HE	259	256	263	263	272	263	266	261	255	263
	CG	295	291	282	296	303	303	323	319	316	314
	BE	266	268	263	267	270	276	277	285	273	275
	RJ	283	281	293	275	278	283	278	281	282	295
	KS	311	311	297	304	304	306	318	320	321	326
	SB	266	279	261	258	269	270	275	274	267	274
	AB	311	311	319	315	317	323	328	320	327	323
	$\bar{X}$	285	287	283	284	289	289	290	292	290	296
	SD	18	17	18	19	19	18	24	23	25	22

Table XXIV: $\dot{V}_I$ ($l.min^{-1}$) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	62.1	59.3	62.6	62.7	61.0	59.4	60.3	65.4	62.5	64.4
	GN	68.8	70.5	71.3	73.5	73.1	77.3	76.6	76.8	77.6	76.7
	CM	60.2	65.9	65.5	65.2	65.3	66.0	65.6	66.4	68.0	67.8
	EM	62.3	58.7	56.8	56.1	61.5	61.5	60.3	61.2	60.3	60.7
	EN	72.8	72.3	69.3	71.5	73.2	77.4	70.0	78.1	75.8	79.8
	MN	55.8	57.0	56.3	56.5	55.0	53.1	57.1	60.9	59.1	60.7
	WM	71.7	68.6	71.1	68.0	68.6	67.7	68.0	71.9	71.3	73.7
	MB	60.2	63.7	65.5	62.8	64.5	64.1	66.4	70.7	64.9	72.2
	$\bar{X}$	64.2	64.5	64.8	64.5	65.3	65.8	65.5	68.9	67.4	69.5
	SD	6.1	5.8	5.9	6.4	6.2	8.4	6.3	6.1	7.0	7.3
WHITE (n = 10)	SG	73.9	76.4	77.2	80.6	77.5	71.6	73.6	79.3	78.1	79.4
	RB	59.1	55.1	54.7	56.5	56.4	55.5	61.3	59.9	64.3	61.3
	RS	64.8	65.6	64.2	66.5	64.6	70.8	70.3	73.6	78.1	75.6
	HE	45.9	46.6	47.7	49.0	47.6	47.5	51.3	49.0	49.0	49.5
	CG	66.4	68.7	68.9	69.4	70.3	71.8	75.4	77.0	77.5	81.7
	BE	73.1	70.0	67.1	69.9	72.2	72.0	73.5	77.2	75.8	76.3
	RJ	68.3	71.4	66.2	70.4	71.9	71.2	72.1	77.5	76.3	87.8
	KS	64.8	66.5	62.1	65.8	63.2	62.8	66.3	69.9	68.3	67.6
	SB	58.1	56.7	57.2	58.7	65.8	63.2	64.7	65.6	59.0	62.9
	AB	68.6	67.7	68.7	67.7	68.1	70.5	67.5	69.0	68.9	71.3
	$\bar{X}$	64.3	64.5	63.4	65.4	65.8	65.7	67.6	69.8	69.5	71.3
	SD	8.3	9.0	8.4	8.8	8.6	8.4	7.3	9.5	9.3	11.3

Table XXV: f (breaths.min^{-1}) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	57	59	57	60	58	60	61	63	65	68
	GN	59	63	67	66	70	73	72	75	74	75
	CM	51	54	56	57	59	58	57	59	58	60
	EM	44	44	42	43	48	52	48	51	48	48
	EN	45	48	44	46	45	55	46	53	49	51
	MN	48	47	50	48	47	47	49	51	56	53
	WM	69	69	70	69	73	69	72	74	73	75
	MB	47	48	48	46	49	50	53	56	57	59
	$\bar{X}$	53	54	54	54	56	58	57	60	60	61
	SD	9	9	10	10	11	9	10	10	10	11
WHITE (n = 10)	SG	37	38	40	44	42	37	42	45	45	47
	RB	44	40	38	40	42	40	43	39	43	43
	RS	51	45	50	56	55	56	57	59	60	59
	HE	44	47	46	49	45	53	57	56	56	56
	CG	63	64	64	66	68	70	71	76	74	81
	BE	45	44	38	42	47	42	41	44	44	44
	RJ	35	34	32	34	38	36	40	42	42	45
	KS	37	36	39	42	40	38	41	43	46	42
	SB	25	28	26	27	37	37	35	34	29	33
	AB	49	50	48	51	52	56	53	56	57	57
	$\bar{X}$	43	43	42	45	47	47	48	49	50	51
	SD	10	10	11	11	10	12	11	12	12	13

Table XXVI: $\bar{V}_T$ (l.breath^{-1}) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	1.09	1.00	1.10	1.04	1.05	0.99	0.99	1.04	0.96	0.95
	GN	1.17	1.12	1.06	1.11	1.04	1.06	1.02	1.02	1.05	1.02
	CM	1.18	1.22	1.17	1.14	1.11	1.14	1.15	1.13	1.17	1.13
	EM	1.42	1.33	1.35	1.30	1.28	1.18	1.26	1.20	1.26	1.26
	EN	1.62	1.51	1.58	1.55	1.63	1.41	1.52	1.47	1.55	1.56
	MN	1.16	1.21	1.13	1.18	1.17	1.13	1.17	1.19	1.06	1.14
	WM	1.04	0.99	1.02	0.99	0.94	0.98	0.94	0.97	0.98	0.98
	MB	1.28	1.33	1.36	1.37	1.32	1.28	1.25	1.26	1.14	1.22
	$\bar{X}$	1.25	1.21	1.22	1.21	1.19	1.15	1.16	1.16	1.15	1.16
	SD	0.19	0.18	0.19	0.19	0.22	0.15	0.19	0.16	0.19	0.20
WHITE (n = 10)	SG	2.00	2.01	1.93	1.83	1.85	1.94	1.75	1.76	1.74	1.69
	RB	1.34	1.38	1.44	1.41	1.34	1.39	1.42	1.54	1.50	1.43
	RS	1.27	1.46	1.28	1.19	1.18	1.26	1.23	1.25	1.30	1.28
	HE	1.04	0.99	1.04	1.00	1.06	0.90	0.90	0.88	0,88	0.88
	CG	1.05	1.07	1.08	1.05	1.03	1.03	1.06	1.01	1.05	1.01
	BE	1.62	1.59	1.76	1.66	1.54	1.71	1.79	1.75	1.72	1.73
	RJ	1.95	2.10	2.07	2.07	1.89	1,98	1.80	1.85	1.65	1.95
	KS	1.75	1.85	1.59	1.57	1.58	1.65	1.62	1.63	1.48	1.61
	SB	2.32	2.02	2.20	2.17	1.78	1.71	1.85	1.93	2.03	1.91
	AB	1.40	1.35	1.43	1.33	1.31	1.26	1.27	1.23	1.21	1.25
	$\bar{X}$	1.57	1.58	1.58	1.53	1.46	1.48	1.47	1.48	1.46	1.47
	SD	0.43	0.40	0.40	0.41	0.32	0.37	0.34	0.37	0.35	0.37

Table XXVII: Tr (°C) responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	38.3	38.4	38.4	38.4	38.4	38.4	38.4	38.5	38.5	38.6
	GN	39.0	39.2	39.2	39.6	38.8	38.8	38.8	39.0	38.8	38.8
	CM	38.1	38.0	38.0	38.0	38.0	38.0	38.1	38.2	38.2	38.5
	EM	38.7	38.7	38.8	38.8	38.8	38.6	38.6	38.7	38.6	38.7
	EN	37.6	37.6	37.4	36.9	37.8	38.1	38.2	38.2	38.3	38.5
	MN	38.7	39.2	39.2	39.0	39.0	39.1	39.0	39.2	39.0	39.0
	WM	38.4	38.5	38.6	38.8	38.9	38.9	39.0	38.8	39.0	39.0
	MB	38.4	39.0	39.6	39.8	40.0	40.1	40.1	40.2	40.0	40.0
	$\bar{X}$	38.4	38.6	38.7	38.7	38.7	38.8	38.8	38.9	38.8	38.9
	SD	0.4	0.6	0.7	0.9	0.7	0.7	0.6	0.7	0.6	0.5
WHITE (n = 10)	SG	38.8	38.9	38.8	38.6	38.5	38.6	38.6	38.7	38.7	39.0
	RB	38.4	38.5	38.5	38.4	38.4	38.5	38.6	38.6	38.5	38.6
	RS	38.5	38.8	39.0	39.0	39.1	39.2	39.2	39.3	38.4	38.6
	HE	38.4	38.5	38.5	38.5	38.4	38.3	38.1	37.6	37.4	37.6
	CG	39.6	40.0	39.8	40.0	40.2	40.4	40.6	40.6	40.6	39.4
	BE	39.1	39.3	38.8	38.8	38.8	38.9	39.2	39.3	39.6	40.0
	RJ	38.6	39.3	39.4	39.8	39.9	40.0	40.2	40.2	40.3	40.6
	KS	38.7	38.8	38.8	38.8	39.0	39.0	39.0	39.0	39.1	39.1
	SB	38.4	38.7	38.7	38.6	38.4	38.4	38.6	38.6	38.6	38.6
	AB	38.9	39.1	39.2	39.2	39.2	39.1	39.2	39.2	39.3	39.4
	$\bar{X}$	38.7	39.0	39.0	39.0	39.0	39.0	39.1	39.1	39.1	39.1
	SD	0.4	0.5	0.4	0.5	0.6	0.7	0.8	0.8	0.9	0.8

Table XXVIII: Tp (°C) responses during a simulated marathon

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	29.8	28.4	27.7	27.0	27.4	28.3	29.2	29.3	28.4	30.0
	GN	34.2	33.4	33.1	30.2	32.5	32.6	32.7	32.0	31.8	31.7
	CM	29.5	29.4	29.4	29.6	29.0	29.6	28.8	29.4	29.8	29.8
	EM	30.6	29.2	30.9	30.8	31.1	31.3	31.0	30.8	31.2	30.8
	EN	33.4	33.8	33.7	33.6	33.2	32.6	32.6	32.9	33.0	32.6
	MN	31.2	31.2	31.6	32.0	30.6	30.6	31.3	31.8	32.0	31.4
	WM	29.6	28.4	28.4	28.5	28.6	28.5	28.3	28.0	28.6	28.4
	MB	30.8	31.4	31.8	31.8	30.6	31.6	30.0	29.8	28.4	29.3
	$\bar{X}$	31.1	30.7	30.8	30.4	30.4	30.6	30.5	30.5	30.4	30.5
	SD	1.8	2.1	2.2	2.1	2.0	1.7	1.7	1.7	1.8	1.4
WHITE (n = 10)	SG	26.0	26.4	27.2	27.0	26.8	28.8	28.2	28.2	27.8	29.4
	RB	26.0	26.0	27.2	27.0	25.0	25.6	26.4	25.7	26.8	25.8
	RS	31.0	31.0	31.4	31.0	29.2	29.4	29.4	29.5	28.0	27.0
	HE	33.4	33.1	33.4	33.2	33.2	33.2	32.8	32.2	31.8	31.8
	CG	30.4	31.8	32.5	28.7	29.4	29.7	25.8	24.2	25.6	26.8
	BE	29.2	26.4	26.6	26.7	26.5	26.4	25.8	25.9	25.2	26.1
	RJ	28.2	27.8	28.6	31.4	31.6	30.8	29.2	28.3	29.0	27.4
	KS	31.1	29.6	27.5	27.8	27.3	28.2	27.4	26.8	27.4	26.6
	SB	27.2	24.1	24.6	26.0	26.1	24.9	25.8	26.2	28.4	29.4
	AB	31.6	31.1	30.6	30.6	29.1	30.2	28.8	27.9	28.1	27.7
	$\bar{X}$	29.4	28.7	29.0	28.9	28.4	28.7	28.0	27.5	27.8	27.8
	SD	2.5	3.0	2.9	2.4	2.6	2.5	2.2	2.3	1.8	1.9

Table XXIX: Tth (°C) responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	30.8	30.4	29.8	31.2	31.7	31.5	31.2	31.1	28.8	29.2
	GN	32.1	31.0	31.2	31.3	31.1	31.1	31.0	31.0	30.9	30.8
	CM	32.1	32.0	32.0	32.0	31.8	32.0	32.0	32.0	32.0	31.6
	EM	31.2	30.4	31.4	31.6	31.9	32.1	32.1	32.2	32.1	32.2
	EN	33.0	32.8	32.2	32.0	31.6	31.0	31.1	31.3	31.2	31.0
	MN	33.5	31.4	32.2	31.0	30.2	30.2	30.4	30.2	31.2	31.6
	WM	30.8	30.4	30.6	30.7	31.4	31.0	30.4	30.3	30.4	30.4
	MB	32.0	32.8	32.6	32.6	32.0	32.5	29.6	28.9	28.6	29.6
	$\bar{X}$	31.9	31.4	31.5	31.6	31.5	31.4	31.0	30.9	30.7	30.8
	SD	1.0	1.0	0.9	0.6	0.6	0.7	0.8	1.1	1.3	1.0
WHITE (n = 9)	SG	33.5	33.2	32.7	32.1	32.1	32.4	32.5	32.4	32.3	32.5
	RB	–	–	–	–	–	–	–	–	–	–
	RS	31.5	32.6	32.6	32.6	33.0	32.8	32.7	32.4	32.4	29.6
	HE	33.4	33.4	33.8	33.7	33.4	33.2	32.2	32.0	31.7	31.4
	CG	32.7	32.4	32.5	33.0	32.8	32.8	32.9	32.8	32.3	32.6
	BE	32.2	29.0	29.1	29.9	29.0	29.4	27.4	28.2	28.1	28.4
	RJ	32.5	33.3	33.5	34.0	33.7	33.0	32.2	32.4	32.2	31.6
	KS	32.3	32.0	31.6	31.7	31.5	31.4	31.0	30.2	29.6	29.4
	SB	34.1	34.1	33.7	33.6	33.2	33.2	33.0	33.2	32.8	32.9
	AB	32.6	32.3	32.6	32.7	32.3	31.8	30.6	30.3	30.1	29.5
	$\bar{X}$	32.8	32.5	32.5	32.6	32.3	32.2	31.6	31.5	31.3	30.9
	SD	0.8	1.5	1.4	1.3	1.4	1.2	1.8	1.6	1.6	1.7

Table XXX: $\bar{T}s$ (°C) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 8)	TG	30.2	29.2	28.5	28.7	29.1	29.6	30.0	30.0	28.6	29.7
	GN	33.4	32.4	32.3	30.6	31.9	32.0	32.0	31.6	31.4	31.3
	CM	30.5	30.4	30.4	30.6	30.1	30.6	30.1	30.4	30.7	30.5
	EM	30.8	29.7	31.1	31.1	31.4	31.6	31.4	31.4	31.6	31.4
	EN	33.2	33.4	33.1	33.0	32.6	32.0	32.0	32.3	32.3	32.0
	MN	32.1	31.3	31.8	31.6	30.4	30.4	30.9	31.2	31.7	31.5
	WM	30.1	29.2	29.3	29.4	29.7	29.5	29.1	29.3	29.2	29.2
	MB	31.3	32.0	32.1	32.1	31.2	32.0	29.8	29.4	28.7	29.4
	$\bar{X}$	31.5	31.0	31.1	30.9	30.8	31.0	30.7	30.7	30.5	30.6
	SD	1.3	1.6	1.6	1.4	1.2	1.1	1.1	1.1	1.5	1.1
WHITE (n = 10)	SG	29.0	29.1	29.4	29.0	28.9	30.2	29.9	29.9	29.6	30.6
	RB	27.7	27.7	28.4	28.3	27.1	27.5	28.0	27.5	28.2	27.6
	RS	31.2	31.6	31.9	31.6	30.7	30.8	30.7	30.7	29.8	28.0
	HE	36.4	33.2	33.6	33.4	33.3	33.2	32.6	32.1	31.8	31.6
	CG	31.3	32.0	32.5	30.4	30.8	30.9	28.6	27.6	28.3	29.1
	BE	30.4	27.4	27.1	28.0	27.5	27.6	26.4	26.8	26.4	27.0
	RJ	29.9	30.0	30.6	32.4	32.4	31.7	30.4	29.9	29.2	29.1
	KS	31.6	30.6	29.1	29.4	29.0	29.5	28.8	28.2	28.3	27.7
	SB	30.0	28.1	28.1	29.0	28.9	28.2	28.7	29.0	30.2	30.8
	AB	32.0	31.6	31.4	31.4	30.4	30.8	29.5	29.1	28.9	28.4
	$\bar{X}$	31.0	30.1	30.2	30.3	29.9	30.0	29.4	29.1	29.1	29.0
	SD	2.3	2.0	2.1	1.8	2.0	1.8	1.7	1.6	1.4	1.5

Table XXXI: Step rate (steps.min^{-1}) during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 7)	TG	176	172	172	172	170	168	168	168	170	168
	GN	-	-	-	-	-	-	-	-	-	-
	CM	168	172	176	172	172	172	172	176	172	168
	EM	158	160	160	156	160	160	160	160	160	160
	EN	180	180	180	180	180	176	176	176	176	176
	MN	184	184	186	188	188	184	188	188	184	184
	WM	180	180	176	176	180	180	180	176	176	176
	MB	192	188	188	188	180	180	180	180	180	180
	$\bar{X}$	176	176	176	176	176	174	174	174	174	174
	SD	12	10	10	12	10	8	10	8	8	8
WHITE (n = 10)	SG	176	176	172	174	172	172	174	168	172	168
	RB	168	168	168	172	178	174	176	172	174	174
	RS	188	184	184	180	180	180	178	180	180	180
	HE	176	176	180	180	178	180	180	176	176	176
	CG	196	192	196	192	192	192	192	192	196	192
	BE	180	176	180	180	180	182	180	180	176	180
	RJ	172	172	170	172	164	166	180	176	172	176
	KS	176	180	180	184	184	184	184	184	184	184
	SB	176	176	176	172	172	172	172	172	172	168
	AB	172	176	172	172	172	170	168	172	170	172
	$\bar{X}$	178	178	178	178	178	178	176	178	178	178
	SD	8	6	8	6	8	8	8	8	8	8

Table XXXII: "General" RPE responses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 7)	TG	11	13	13	13	13	13	9	11	9	9
	GN	-	-	-	-	-	-	-	-	-	-
	CM	13	11	13	13	15	15	17	17	17	19
	EM	9	11	9	9	9	11	11	13	13	18
	EN	9	9	11	11	9	11	11	13	8	13
	MN	11	17	17	19	20	18	14	13	8	7
	WM	9	9	13	15	17	19	19	19	19	19
	MB	7	8	10	15	16	18	19	19	20	20
	$\bar{X}$	10	11	12	14	14	15	14	14	13	15
	SD	2	3	3	3	4	3	4	3	5	5
WHITE (n = 10)	SG	10	10	11	11	12	12	13	13	13	13
	RB	11	12	12	14	15	15	15	15	16	16
	RS	11	12	15	15	15	14	15	15	12	15
	HE	10	11	11	13	14	14	14	17	20	20
	CG	9	12	12	12	12	12	13	14	16	17
	BE	7	9	11	11	11	11	11	11	11	11
	RJ	9	10	11	11	11	12	14	14	16	17
	KS	9	9	9	9	11	11	12	12	12	12
	SB	8	10	11	11	11	12	12	12	13	13
	AB	12	12	12	12	11	13	12	11	11	12
	$\bar{X}$	10	11	12	12	12	13	13	13	14	15
	SD	2	1	2	2	2	1	1	2	3	3

Table XXXIII: "Local" (leg) RPE reponses during a simulated marathon.

Category	Subject	% Running Time									
		10	20	30	40	50	60	70	80	90	100
BLACK (n = 7)	TG	9	11	11	11	11	11	11	9	11	11
	GN	-	-	-	-	-	-	-	-	-	-
	CM	9	13	13	15	15	17	17	17	19	19
	EM	9	9	11	11	11	13	13	15	15	15
	EN	11	11	13	13	15	15	15	15	15	15
	MN	13	13	16	19	20	19	15	14	11	9
	WM	13	13	17	17	19	19	19	19	19	19
	MB	7	8	10	16	18	20	19	20	20	20
	$\bar{X}$	10	11	13	15	16	16	16	16	16	15
	SD	2	2	3	3	4	3	3	4	4	4
WHITE (n = 10)	SG	10	11	12	12	13	13	13	13	13	13
	RB	11	13	14	15	15	16	17	17	17	17
	RS	8	8	12	13	13	12	17	15	13	15
	HE	11	11	11	13	14	14	15	18	20	20
	CG	9	12	12	12	12	13	13	15	16	17
	BE	7	11	11	11	13	13	13	13	15	15
	RJ	12	12	11	12	13	13	15	15	17	19
	KS	7	7	9	9	11	12	12	12	12	12
	SB	10	12	12	13	13	14	15	15	15	15
	AB	12	12	12	13	12	13	13	13	13	13
	$\bar{X}$	10	11	12	12	13	13	14	15	15	16
	SD	2	2	1	2	1	1	2	2	2	3