THE EFFECTS OF PHYSICAL ACTIVITY AND MATURATION
ON BOYS' (8 TO 16 YEARS) RUNNING ECONOMY

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Graduate Studies and Research
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for the Degree of Masters of Science
in the College of Kinesiology
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By
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ABSTRACT

Previous reports have demonstrated that running economy (RE), a measure of efficiency of locomotion, is superior in adults than in children; however, it is unclear how these differences come to be. **Purpose:** To identify the effect of maturity status, physical activity and various other anatomical and physiological factors on RE development in boys aged 8 to 16 years. **Methods:** Data were collected as part of the Saskatchewan Growth and Development Study (SGDS; 1964-1973). Using a pure longitudinal study design, anthropometric, maturity, physiological characteristics (treadmill run) and physical activity were assessed annually for nine consecutive years. Two-hundred and two eight year-old males were measured in 1965; by 1973, complete longitudinal data were available for 63 participants. During the treadmill run, a measure of submaximal oxygen consumption ($\dot{VO}_2$) was recorded, an index of RE. Four approaches of normalizing $\dot{VO}_2$ to body size were investigated. Maturity status was determined based upon chronological age at peak height velocity (PHV). Physical activity was assessed by two teacher ratings and two questionnaires. **Results:** Normalizing $\dot{VO}_2$ to body surface area was found to be the most appropriate body size adjustment. Submaximal $\dot{VO}_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ decreased with increasing chronological age (p<0.05). At common chronological age bands, late-maturing boys demonstrated superior RE than early-maturing boys from ages 10-14 years (p<0.05); average-maturing boys were also found to be more efficient than early-maturers at 12 and 13 years of age (p<0.05). Physical activity was not found to have any significant effect on the development of RE (p>0.05). A series of age-specific regression analyses identified body surface area and respiratory exchange ratio (RER) as variables which account for a significant portion of
the variance in absolute \( \dot{VO}_2 \) (0.619<R\(^2\)<0.903); RER was not significant (p>0.05) at all chronological ages. **Conclusion:** Determining an appropriate approach for normalizing \( \dot{VO}_2 \) values is essential to allow for reliable investigation into factors other than size that affect RE. Maturity status was found to significantly affect RE development; however, only during the circumpubertal years. No effect of physical activity was found on RE development in boys 8-16 years. The relative influence of maturity status and RER are variable across different ages.
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I thank my family for their complete support of my academic pursuits. Each of you contributed to this in some way, whether as a proof reader or an inspiration (or both), or at times a sounding board, I would be remiss if I did not acknowledge the contributions of Dad, Mom, Geoff, Kara and Chris.

Finally, I would like to acknowledge the contributions made by some special friends. It is difficult to imagine what this process would have been like if it were not for the support of both Beth and the Royals. It is my sincere hope that this endeavor has prepared me well for what lies ahead of us all.
DEDICATION

I would like to dedicate this thesis to my father, Ian S. Spencer whom I miss terribly. You taught me many important lessons; you taught me how to respect others; you taught me to seek the answer for myself; you taught me that there’s nothing wrong with taking the ‘scenic route’; you taught me the difference between ‘effect,’ the noun and ‘affect,’ the verb; but the lesson that I appreciate the most is that I should never do a job half-assed, or is that half-fast… don’t worry Dad, the thesis is okay and it took over three years to complete, so I’ve got you covered either way! I love you, I miss you and this one’s for you.
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<table>
<thead>
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<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>RE</td>
<td>Running economy</td>
</tr>
<tr>
<td>$\dot{V}O_2$</td>
<td>Oxygen consumption</td>
</tr>
<tr>
<td>PHV</td>
<td>Age at peak height velocity</td>
</tr>
<tr>
<td>$\dot{V}O_{130}$</td>
<td>Oxygen consumption at a heart rate of 130 beats per minute</td>
</tr>
<tr>
<td>CV&lt;sub&gt;BS&lt;/sub&gt;</td>
<td>Coefficient of variation for the body size</td>
</tr>
<tr>
<td>CV&lt;sub&gt;VO2&lt;/sub&gt;</td>
<td>Coefficient of variation for oxygen consumption</td>
</tr>
<tr>
<td>SGDS</td>
<td>Saskatchewan Growth and Development Study</td>
</tr>
<tr>
<td>VO&lt;sub&gt;max&lt;/sub&gt;</td>
<td>Maximal oxygen consumption</td>
</tr>
<tr>
<td>$\dot{V}O_{155}$</td>
<td>Oxygen consumption at a heart rate of 155 beats per minute</td>
</tr>
<tr>
<td>$\dot{Q}$</td>
<td>Cardiac output</td>
</tr>
<tr>
<td>RER</td>
<td>Respiratory exchange ratio</td>
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<tr>
<td>LSS</td>
<td>Longitudinal sub-sample</td>
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<td>CSS</td>
<td>Cross-sectional sub-sample</td>
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<td>LBASS</td>
<td>Longitudinal biological age sub-sample</td>
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<tr>
<td>NLSS</td>
<td>Non-longitudinal sub-sample</td>
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<tr>
<td>NCSS</td>
<td>Non-cross-sectional sub-sample</td>
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CHAPTER 1 ~ SCIENTIFIC FRAMEWORK

1.1. Introduction

1.1.1. Research Questions

Children's physiological responses to exercise are different from those observed in adults and reflect the child's immaturity. One such difference that has continually been demonstrated is that running economy (RE) is superior in adults than in children (Ariëns, van Mechelen, Kemper & Twisk, 1997; Åstrand, 1952; Daniels & Oldridge, 1971; Daniels, Oldridge, Nagle & White, 1978a; Maliszewski & Freedson, 1996; Montoye, 1982; Robinson, 1938; Rowland, Auchinachie, Keenan & Green, 1987; Unnithan & Eston, 1990). RE is a measure of efficiency of locomotion; specifically, RE improves as the rate of oxygen consumption (\(\dot{V}O_2\)) at a given submaximal running workload decreases (Morgan, 2000).

Though it seems clear that differences exist between children and adults with respect to RE, what remains unclear is how these differences come to be. Therefore, the purpose of the present study is to identify factors that affect the development of RE in a group of boys studied longitudinally. The specific research questions addressed by the present research are as follows: does the timing of maturation affect the development of RE, and is the effect the same at all chronological ages? Does physical activity level affect the development of RE, and is the effect the same at all chronological and
biological ages? And, what other factors affect the development of RE, and are the relative size of these effects the same at all chronological ages?

Before any of the major research questions can be examined, two potentially confounding issues must be addressed. First, since \( \dot{V}O_2 \) is known to be a size-dependent variable, an appropriate adjustment for size must be made (Rogers, Turley, Kujawa, Harper & Wilmore, 1995b). A number of approaches have been suggested, however, the most appropriate method of expressing \( \dot{V}O_2 \) has not been agreed upon. Thus, before proceeding with the three major research questions stated above, an appropriate adjustment for size must be identified. Second, the findings from previous studies that show RE improves with increasing age (chronological or biological) must be substantiated within the sample tested for the present study. Therefore, two additional research questions must be addressed prior to proceeding onto the major questions; they are as follows: what is the most appropriate method to adjust \( \dot{V}O_2 \) values for differences in body size among participants of various ages; and, does appropriately normalized RE change with increasing chronological and biological age? Answering these two questions will make it possible to investigate other factors that influence the development of RE in growing boys.

1.1.2. Rationale for Study

Detecting differences between adults and children is often accomplished by comparing the two groups using a cross-sectional study design, where every individual is measured only once, thus providing information regarding differences between the two groups in the variable of interest (Malina, 1975). However, uncovering how these
differences came to be often requires investigation of changes within an individual. Longitudinal studies, those in which participants are measured repeatedly over time, are of great value because they allow for investigation of individual variations in the tempo and timing of such changes (Malina, 1975). This approach is particularly important during childhood and adolescence because they are periods of vast increases in size (growth) and advanced progression towards the mature, adult state (maturation). Within the context of growth and maturation, the value of a longitudinal study design cannot be overlooked because this design allows for investigation of changes to variables within individuals.

The present study considers longitudinal changes to endurance-related physiological variables, as they relate to increased chronological age (elapsed time since birth), growth and maturation. The central construct examined by the present research is running economy (RE). RE is a measure of efficiency of locomotion as indicated by the rate of oxygen consumption (VO$_2$) during submaximal work. RE should not be confused with mechanical efficiency, which describes the ratio of mechanical work completed to the net metabolic cost of that work (Frost, Bar-Or, Dowling, & Dyson, 2002). Morgan, Martin, Krahenbuhl and Baldini (1991) describe how mechanical work can be quantified during horizontal treadmill running. Essentially what is required is high-speed video data which can be used to estimate potential and kinetic energies of various leg segments; these estimates are used in the calculation of total body mechanical work.

As mentioned, RE improves as the rate of VO$_2$ at a given submaximal running workload decreases (Morgan, 2000). VO$_2$ can be expressed as an absolute rate of liters
of oxygen consumed per minute (L/min), but is more often expressed as a relative rate that takes into account individual differences in body size. An appropriate adjustment for size must be made to permit valid comparisons among individuals (Rogers et al., 1995b). However, the most appropriate method of expressing VO2 has not been agreed upon. Common methods have included ratios using body mass, theoretical body mass exponents, body surface area or fat-free mass (Morgan, 2000; Rogers et al., 1995b). Each method has its merits and shortcomings, but there has not been universal acceptance of one specific methodology (Rogers et al., 1995b). Thus, this study addresses the issue of normalizing VO2 to body size to allow for meaningful comparisons among individuals and groups.

It has been demonstrated that RE improves with increasing chronological age after differences in body mass have been controlled for during childhood, adolescence and even into adulthood as late as 27 years of age (Ariëns et al., 1997; Daniels & Oldridge, 1971; Daniels et al., 1978a). In other words, to complete the same amount of work while running at a given submaximal workload, the oxygen cost per unit body mass is higher in children than in adults; that is, adults generally possess superior RE than children (Ariëns et al., 1997; Åstrand, 1952; Daniels & Oldridge, 1971; Daniels et al., 1978a; Maliszewski & Freedson, 1996; Montoye, 1982; Robinson, 1938; Rowland et al., 1987; Unnithan & Eston, 1990).

Many studies have examined the relationship between RE and chronological age, with participants in the studies ranging in age from six to 91 years (Ariëns et al., 1997; Åstrand, 1952; Daniels & Oldridge, 1971; Daniels et al., 1978a; Maliszewski & Freedson, 1996; Montoye, 1982; Robinson, 1938; Rowland et al., 1987; Unnithan &
Eston, 1990). Since, under normal circumstances, the body is growing and maturing with increasing chronological age during childhood and adolescence, consideration of the processes of growth and maturation in the study of RE are essential to gain insight into how and when chronological age-related changes to RE occur. Few of the studies, however, that have investigated age-associated RE improvements have addressed the issues of growth and maturation. Additionally, few have investigated whether the timing of maturation affects RE; this is an important consideration given that the timing of maturation has been shown to affect other physiological variables (Malina & Bouchard, 1991). It is unclear whether improvements to RE with increasing chronological age are the result of an increase in size (growth), advanced maturational status, training or, perhaps, physical activity. This study addresses these omissions in previous research.

Even after controlling for differences in size, RE can vary greatly within a group of individuals. Researchers have proposed several explanations for variations in RE among individuals; they include, but are not limited to, differences in gait pattern, stride length, stride frequency, leg length, segmental mass distribution, ventilation, substrate utilization (Morgan, 2000), training (Daniels et al., 1978a), age and gender (Ariëns et al., 1997).

From this list of factors hypothesized to affect RE development, one that is likely of particular interest to people involved with middle- and long-distance running is training, since it is the only one that can be controlled by athletes or coaches. Research has demonstrated that RE, normalized using body mass (ml·kg$^{-1}$·min$^{-1}$) is related to better endurance running performance among children with comparable VO$_{2\max}$ (ml·kg$^{-1}$·min$^{-1}$) values (Daniels & Oldridge, 1971; Daniels et al., 1978a); when matched
for $\dot{V}O_{2\text{max}}$, greater performance is associated with superior RE (lower submaximal $\dot{V}O_2$). Furthermore, research has demonstrated that long-term exposure to run training can positively affect RE (Conley, Krahenbuhl, & Burkett, 1981; Conley, Krahenbuhl, Burkett, & Millar, 1984; Daniels, Yarbrough, & Foster, 1978b; Patton & Vogel, 1977; Svedenhag & Sjödin, 1985). Thus, it may be reasonable to suppose that regular physical activity over a long period of time may produce similar results, since training is a specialized division of physical activity. This study, therefore, investigates how varying levels of physical activity affect the development of RE in a group of growing boys.

Because RE has been linked with endurance running performance in children, adolescents (Daniels & Oldridge, 1971; Daniels et al., 1978a; Rowland, Aucharinachie, Keenan & Green, 1988) and adults (Conley & Krahenbuhl, 1980), a clear understanding of how and when changes to RE occur could provide an explanation, in part, for interindividual differences in endurance running performance. The potential for advanced knowledge of factors that affect RE and perhaps endurance running performance in children and adolescents makes the present study important in the field of child growth and maturation.

1.2. REVIEW OF LITERATURE

1.2.1. The Study of Growth and Maturation

Although the specific focus of the present study is the longitudinal development of RE in growing males, the participants were children and adolescents; thus, they were in the midst of great increases in size and physiological changes, during the period of data collection. Special consideration of the processes of growth and maturation must
be given when studying changes within children and adolescents (Malina, 1975). Therefore, an understanding of growth and maturation is essential before specific information regarding RE can be discussed. Specific examples in the opening sections relate to RE, thus, a brief overview of RE is provided.

RE is a measure of efficiency of locomotion. Specifically, lower values of \( \dot{VO}_2 \) at a given submaximal running workload are indicative of superior RE (Morgan, 2000). RE has been shown to improve with increasing chronological age after individual differences in body mass have been controlled for during childhood, adolescence and even into adulthood (Ariëns et al., 1997; Daniels & Oldridge, 1971; Daniels et al., 1978a). Therefore, it is generally accepted that adults possess superior RE than children (Ariëns et al., 1997; Åstrand, 1952; Daniels & Oldridge, 1971; Daniels et al., 1978a; Maliszewski & Freedson, 1996; Montoye, 1982; Robinson, 1938; Rowland et al., 1987; Unnithan & Eston, 1990). In order to understand the nature of these changes to RE with increasing chronological age, one must first have a firm grasp on principles of growth and maturation.

The terms ‘growth’ and ‘maturation’ are often used synonymously; however, they refer to two distinct processes (Claessens, Beunen & Malina, 2000). As mentioned, growth is an increase in the size of the body as a whole, or of specific parts. An example of growth would be an increase in leg length. Maturation, on the other hand, is described as progression towards the mature state; full maturity is attained once growth is no longer occurring. It is important to understand that the two components of maturation are timing and tempo. Timing is used to express when specific maturational events occur. Examples of such events include age of menarche (first menstruation) in females, age at the initial appearance of pubic hair, and age at maximum growth during
the adolescent growth spurt. Tempo refers to the rate at which maturation progresses; for example, how quickly or slowly a person passes from the beginning to the end of the adolescent growth spurt.

The value of a longitudinal study design in the study of child growth and maturation is that it allows for investigation of individual variations in the tempo and timing of growth and maturation (Malina, 1975). Longitudinal evaluations of growth patterns have been taking place since the mid-1700s, beginning with a single-subject analysis of growth in height in 1759 (Tanner, 1978). Count Philbert Guéneau de Montbeillard measured the height of his son every six months from birth to age 18 years to produce the oldest known longitudinal growth record in existence. Since that time, longitudinal studies of human growth and maturation during childhood and adolescence have become increasingly more common. During the 1920s, a number of longitudinal studies were begun in the United States of America, including the studies at the Harvard School of Public Health, the Fels Research Institute, the Child Research Council in Denver, the Adolescent Growth Study in Oakland, and the Guidance Study at the University of California at Berkley (Malina & Bouchard, 1991). Each of these studies measured growth and maturation in predominantly Caucasian children and adolescents. The Adolescent Growth Study in Oakland also included assessment of strength and motor performance (Malina & Bouchard, 1991).

For nearly 20 years, the United States led the world in longitudinal growth research; however, in the 1940s European countries became the predominant site for major longitudinal growth and maturation studies (Kemper, 1996). The Harpenden Growth Study, which began in 1948, was conducted in the suburbs of London, England. This study, which was among the first to include any substantial number of participants
of a race other than Caucasian, included a variety of measurements of size, physique, body composition and maturation. A later series of longitudinal studies, coordinated by the International Children’s Center (ICC), investigated growth and maturation, as well as physical activity patterns and measures of physical performance (Kemper, 1996). The ICC-coordinated longitudinal studies were completed in Paris, London, Brussels, Stockholm/Göteborg, and Zürich.

The Wroclaw Growth Study was begun in 1961 in Poland. A large cohort of boys and girls was followed longitudinally from age eight to 18 years. The major area of interest in the Wroclaw study was measurements of growth and maturation (Kemper, 1996). This was among the last longitudinal studies to focus specifically on growth and maturation, without investigating other factors such as physical activity patterns, nutrition status or physical performance. The Medford Boys’ Growth Study of the University of Oregon (1956), the Prague study (1961) and the Saskatchewan Growth and Development Study (1964) were among the early studies to longitudinally investigate the physiological effects of exercise in children and adolescents (Kemper, 1996). In fact, the Prague study and the Saskatchewan Growth and Development Study (SGDS) were the first to produce longitudinal data on $\text{VO}_2\text{max}$, the maximum rate at which oxygen can be consumed during exercise, of the same subjects over a considerable period of time.

During the past 40 years, a number of longitudinal studies of growth and maturation have been conducted in children, adolescents and even into adulthood. In 1969, two studies were begun in Belgium, while the Trois Rivières regional study at the University of Québec was initiated in Canada. The Leuven growth study was initiated in Belgium and designed to provide information on the physical fitness of normal boys
aged 12-20 years, while the Leuven longitudinal experimental study included participants aged three to 15 years (Kemper, 1996). The Trois Rivières regional study was unique in that it was the only longitudinal study of growth and maturation to include an intervention. The researchers investigated the effects of increasing physical education time from that of the normal school curriculum during primary school years.

As summarized by Kemper (1996), the Canada growth and development study at the University of Western Ontario was begun in 1975. The five-year longitudinal study investigated growth, maturation, physical activity and performance measures, as well as psychosocial/mental development in a group of participants aged ten years at study entry (Kemper, 1996). The Amsterdam Growth and Health study, which began in 1976, investigated similar variables as the Western Ontario study, however it included an assessment of participants’ nutrition, and the age span was different; participants in the Amsterdam Growth and Health study were as young as 13 years old at study entry, and were assessed at ages 13, 14, 15, 16, 21, and 27 years (Ariëns et al., 1997).

An important result of all of these studies has been the development of better methods to assess and control for growth and maturation. Growth and maturation are measured or observed at a single point in time, or over time. The point of reference for these measurements or observations is an individual’s chronological age (Malina, 1975). Although two individuals may share the same birthday and therefore the same chronological age, they are not necessarily equally mature; not all 12 year olds, for example, are at the same point in their progression towards adulthood. Therefore, in addition to chronological age, there exists a need to evaluate and express the timing and tempo of these biological processes (Claessens et al., 2000); hence, biological age can be calculated.
Age at peak height velocity (PHV) is the most commonly used indicator of somatic maturity in longitudinal studies of adolescents, and can be used to calculate biological age (Claessens et al., 2000). Age at PHV is the age at which height is increasing most rapidly during the adolescent growth spurt. This method of assessing maturation (PHV) requires serial measurements of height for a number of years surrounding the occurrence of peak velocity (Claessens et al., 2000). Fitting velocity curves to individual growth records of height derives age at PHV; this can be done either graphically or mathematically.

As mentioned, age at PHV can be used to calculate a biological age; by using age at PHV as a reference point (PHV = 0) the years surrounding this occurrence produce a biological timeline. For example, if an individual reaches PHV at 13.2 years of age, their biological age at 12.2 would be -1.0 year from PHV, and at a chronological age of 14.2 it would be 1.0 post PHV.

Malina and Bouchard (1991) point out that age at PHV correlates highly with other measures of maturation. The strongest correlations between skeletal maturity and the adolescent growth spurt occur after the age of PHV; and, in general, youngsters who are advanced or delayed in sexual maturation are, respectively, advanced or delayed in the timing of PHV. Since age at PHV has been used previously in studies investigating \( \text{VO}_2 \) (Cunningham, Patterson, Blimkie and Donner, 1984; Kobayashi, Kitamra, Miura, Sodeyama, Murae, Miyashita, & Matsui, 1978; Mirwald, Bailey, Cameron, & Rasmussen, 1981), and correlates highly with other measures of maturation, its use in the present study is well supported.
1.2.2. Normalizing Oxygen Consumption ($\dot{V}O_2$) to Body Size

A clear description of structural or functional changes that occur with increases in size is required in order for a study to contribute to an increased understanding of growth (Marshall, Bailey, Leahy, & Ross, 1978). Though changes associated with growth are anatomical and physiological, a clear description of these changes, by necessity, is mathematical. Thompson (1966) highlighted this claim by describing the relation between changes in height and changes in mass within an organism. Using a fish as an example, Thompson wrote, “in doubling its length, multiplies its weight by no less than eight times…” This simple example emphasizes the relation between growth in different dimensions (i.e. height = one dimension [$2^1 = 2$]; mass = three dimensions [$2^3 = 8$]) by using a mathematical description. Asmussen and Heebol-Nielson (1955) and Bouchard, Malina, Hollmann, and Leblanc (1977) provide excellent examples of how changes in anatomical characteristics can affect physiological processes.

The purpose of the 1955 paper was to analyze certain physiological performance in children aged seven to 17 years, in a dimensional way, correlating them to purely physical or anatomical characteristics such as height, surface area or body mass (Asmussen & Heebol-Nielson, 1955). Working on the assumption of geometric similarity between the younger and older participants, the researchers predicted theoretical differences in structural and functional characteristics of the approximately 400 participants (exact number not reported). By comparing the predicted values to actual values, Asmussen and Heebol-Nielson were able to determine whether qualitative changes (changes to physiological processes) were occurring within the growing bodies; if only quantitative changes (anatomical) were occurring, and the assumption of geometric similarity was valid, there would be no difference between theoretical and
observed values. For every variable measured, it was demonstrated that the observed values were significantly different from the predicted values. These findings led Asmussen and Heebol-Nielsen to conclude that anatomical changes were accompanied by physiological changes during growth during childhood and adolescence.

Bouchard et al. (1977) related submaximal $\dot{VO}_2$ to heart size and body size (anatomical characteristics) and chronological age. Heart diameters, heart volume, age, height and body mass were assessed in the cross-sectional sample consisting of 237 participants. Also, submaximal $\dot{VO}_2$ (L/min) at a heart rate of 130 beats per minute ($\dot{VO}_{130}$) was calculated by interpolation from two appropriate workloads on a cycle ergometer. Using correlation analyses, Bouchard et al. (1977) demonstrated that approximately 75% of the variation in $\dot{VO}_{130}$ was accounted for by heart volume, height, mass and age; and 20% by heart volume alone (holding the others constant). Although no causal relationship was demonstrated between changes in $\dot{VO}_{130}$ and the anthropometric variables, this study remains important because it highlights the need to study the interactions between age, size and physiological parameters. In other words, the study emphasized the fact that the growth process itself can have a significant relationship with other (physiological) measures. Therefore, when studying changes in physiological characteristics, including RE, it is important to consider changes to anatomical characteristics as well.

Since not all individuals are the same size, and $\dot{VO}_2$ is known to be associated with body size, normalizing oxygen consumption for body size to allow for meaningful comparisons among individuals is of paramount importance in physiology (Rogers et al., 1995b). Therefore, this section of the literature review examines several approaches to
normalizing $\dot{V}O_2$ to body size to permit valid comparisons among individuals.

Šprynarová, Pařízková and Bunc (1987) demonstrated that changes to submaximal $\dot{V}O_2$ with increasing chronological age were most closely related to increases in body mass in a group of boys studied longitudinally. The sample consisted of 90 boys, aged $10.8 \pm 0.35$ (SD) years at entry, who were tested annually for six consecutive years; data were collected on 39 of the participants until the age of 18 years. $\dot{V}O_2$ was recorded during ten minutes of submaximal work (various treadmill speeds based upon chronological age) on a horizontal treadmill. Anthropometric data, including height, total body mass, lean body mass and percentage body fat, were also gathered. The results of correlation analyses revealed that submaximal $\dot{V}O_2$ (L/min) correlated most strongly with body mass, and to a lesser extent to fat-free mass and/or height. In fact, significant correlations between submaximal $\dot{V}O_2$ (L/min) and body mass were detected even when height and lean body mass were held constant (Šprynarová et al., 1987). The researchers therefore concluded that inter-individual differences in submaximal $\dot{V}O_2$ were primarily conditioned by differences in body mass. What is important to note is that even the strongest correlation between body mass and submaximal $\dot{V}O_2$ accounted for only 36% of the variation when height and lean body mass were held constant, thus leaving 64% of the variance unaccounted for. These results seem to indicate that dividing $\dot{V}O_2$ by body mass would perhaps not be the best approach to normalizing these data to body size.

A specific criterion, known as “Tanner’s Special Circumstance,” has been described to identify appropriate ratio standards (Tanner, 1949). The coefficient of variation (CV), calculated as: the standard deviation divided by the mean, multiplied by...
100, is a standardized index of variance (Winter, 1992). In order for a simple ratio standard to be valid (i.e. satisfy Tanner’s Special Circumstance), the coefficient of variation for the body size (CV\text{BS}) measure divided by the coefficient of variation for the physiological measure (CV\text{VO2}) must be equal to Pearson’s product moment correlation coefficient (r) for the two variables; otherwise, the data will be inappropriately skewed (Winter, 1992). A Pearson’s product moment correlation coefficient is a number between -1.0 and +1.0; the value of the coefficient indicates the extent to which two variables are related or associated (Vincent, 1999). Therefore, Tanner’s Special Circumstance relates the strength of a relationship between two variables (r) to the variance around the mean of each variable.

Another approach to testing whether a ratio has effectively eliminated any influence of body size is to calculate the strength of the relationship, using Pearson’s correlation analysis, between normalized \( \text{VO}_2 \) and the body size measurement used to normalize (e.g. body mass, fat-free mass, etc.). If the correlation coefficient is non-significant, one may conclude that any body size-related bias has been effectively eliminated. However, if Pearson’s r is significant, then the \( \text{VO}_2 \) value is still being influenced by an individual’s body size (Welsman & Armstrong, 2000).

With regards to the relationship between body mass and absolute \( \text{VO}_2 \) (L/min), Tanner (1949) demonstrated that \((\text{CV}_{\text{BS}})/(\text{CV}_{\text{VO2}})\) was not equal to the correlation coefficient of mass and \( \text{VO}_2 \) in 34 young men (age not reported). In short, Tanner produced evidence over 50 years ago that normalizing \( \text{VO}_2 \) to body mass is not always appropriate. The evidence and conclusions offered by Tanner (1949) have been supported by several other papers (Katch, 1972; Katch, 1973; Katch & Katch 1974).
Despite this evidence, researchers persist in using the simple ratio standard of \( \dot{VO}_2 \) and body mass to normalize oxygen consumption. This is done, presumably for two reasons; first, because body mass is what must be moved during treadmill running, and therefore normalizing using body mass makes intuitive sense, and second because of the ease of measuring body mass and the fact that this approach allows for simpler comparisons to other studies.

As an alternative to the simple ratio using body mass, physiologists have used allometric scaling to express \( \dot{VO}_2 \) as a ratio standard using theoretical body mass exponents. The simple ratio of \( \dot{VO}_2 \) to body mass assumes that the relationship between the two variables is a linear relationship (i.e. a body mass exponent equal to one). Allometric scaling allows for a non-linear relationship between \( \dot{VO}_2 \) and body mass by adjusting the exponent of the denominator (i.e. body mass).

Traditionally, the two exponents of body mass most commonly used for normalizing \( \dot{VO}_2 \) values are 0.67 and 0.75 (Schmidt-Nielsen, 1984). However, it is possible to calculate sample-specific mass exponents for individuals who are measured repeatedly or for cross-sectional groups. This calculation is accomplished by transforming the data into logarithmic values and generating the regression line for the variables of interest. The slope of that regression line (\( \beta \)-coefficient) represents the exponent used in the subsequent ratio (Rogers et al., 1995b). In the example using body mass and \( \dot{VO}_2 \), the calculations would follow the general formula:

\[
\log_{10} \dot{VO}_2 = \beta \left( \log_{10} \text{mass} \right) + b + \varepsilon
\]

Where: 
\( \beta \) = slope of regression equation  
\( b \) = intercept of regression equation  
\( \varepsilon \) = error associated with regression equation
Consequently, the β-coefficient would be the mass exponent, and the resulting equation for scaling VO₂ would follow the formula:

\[ \frac{\text{VO}_2}{\text{mass}^\beta} \]  \hspace{1cm} (1.2)

Using allometric scaling, Bergh, Sjödin, Forsberg and Svedenhag (1991) were able to demonstrate that submaximal VO₂ did not increase in linear proportion to body mass in a group of participants aged 17-44 years. A cross-sectional sample of 134 participants, which actually consisted of six groups of athletes and one group of trained non-athletes, had VO₂ assessed during treadmill running at various workloads. The relationship between body mass and VO₂ was evaluated for each of the groups by calculating an allometric mass exponent after controlling for chronological age. As indicated, a mass exponent equal to one would indicate that the two variables increased in direct linear proportion to one another. The mass exponents for the seven groups ranged from 0.60-0.83 with a mean value of 0.76 ± 0.06 (SD). Bergh et al. therefore concluded that changes in submaximal VO₂ were not in direct linear proportion to changes in body mass.

Though convenient, the widely recognized values of 0.67 and 0.75, which are based upon theoretical models, may not be appropriate for all populations. Simple principles of isometric geometry indicate that surface area is related to volume to the power \( \frac{2}{3} \), or 0.67 (Schmidt-Nielsen, 1975). Tanner (1949) concluded that a proportional relationship exists between VO₂ and surface area in adults that allows one variable to be substituted for the other. In the case of normalizing oxygen consumption, by substituting VO₂ for surface area and body mass for volume, the same exponential relationship should exist between VO₂ and body mass. One could therefore conclude
that $\dot{V}O_2$ increases as body mass to the power 0.67 increases (Rogers et al., 1995b). A problem, however, with normalizing $\dot{V}O_2$ for body mass to the power 0.67 is that this equation was derived from principles of isometric geometry; real organisms of different size usually are not isometric (Schmidt-Nielsen, 1975).

The second exponential value of 0.75 was originally derived from curves generated in comparative zoology studies on the $\dot{V}O_2$ to body mass relationship in adult animals (Rogers et al., 1995b). A mass exponent equal to 0.75 has also been demonstrated in studies of growing humans during submaximal exercise (Rogers, Olson & Wilmore, 1995a; Sjödin & Svedenhag, 1992). Eight young runners and four untrained controls were studied every six months for an eight year period to investigate the effects of endurance training on physiological characteristics during circumpubertal growth (Sjödin & Svedenhag, 1992). Submaximal $\dot{V}O_2$ at 15 km·h$^{-1}$ on a horizontal treadmill was assessed at each measurement occasion. As demonstrated in previous longitudinal and cross-sectional studies, running economy (RE) significantly improved with increasing chronological and biological age when expressed relative to body mass. Intra-individual mass coefficients were calculated for each participant to describe the non-linear relationship between the development of RE and body mass; the mean value for all of the participants within each group was used. The body mass scaling factor for both the control and experimental groups was equal to 0.75 (Mean ± SEM: ± 0.02 and 0.09, respectively). Sjödin and Svedenhag (1992) were among the first to demonstrate a mass exponent of 0.75 in a group of growing humans.

Rogers et al. (1995a) lent support to this approach to normalizing when they offered evidence that a mass exponent of 0.75 provided an appropriate adjustment of
body mass in 90 participants of various maturational statuses. All of the participants, which included 30 prepubertal, 30 circumpubertal and 30 adult males and females (15 of each in all three groups), were required to complete a maximal treadmill test and submaximal treadmill testing at two separate workloads: walking at 4.8 km·h⁻¹ and running at eight km·h⁻¹. Linear regression analysis was used to determine the slope of the regression equation between log-transformed \( \dot{V}O_2 \) (L/min) and body mass (kg) for the entire sample. The two mass exponents (slopes) generated for the submaximal workloads were 0.60 and 0.75 for the slower and faster speeds, respectively. However, the \( \dot{V}O_2 \) values at the higher speed (eight km·h⁻¹) were significantly less variable than those at the lower speed. This is likely due to the fact that running has been shown to be most economical at a self-selected stride length and therefore running speed (Cavanagh & Williams, 1982); the faster of the two speeds (running at eight km·h⁻¹) would more closely approximate this speed. This finding, in addition to the fact that Tanner’s Special Circumstance was not satisfied by the \( \dot{V}O_2 : \text{body mass ratio} \), led Rogers et al. (1995a) to conclude that the exponent of body mass to the 0.75 power was a more appropriate basis for analysis than body mass alone.

A cross-sectional study examined the use of allometric scaling during exercise in children by verifying that Tanner’s Special Circumstance \( ((CV_{BS})/(CV_{O2}) \approx \tau) \) had been satisfied (Rogers et al., 1995b). The sample used in the study consisted of 21 males and 21 females aged 7-9 years. Oxygen consumption was measured while participants ran at two treadmill speeds, eight and 9.6 km·h⁻¹ respectively. Regardless of the treadmill speed, the results indicated that Tanner’s Special Circumstance had, indeed, been satisfied when \( \dot{V}O_2 \) values were expressed using allometric transformations (0.67) of
body mass. However, the \( \dot{V}O_2 \) to body mass ratio did not satisfy the criteria described by Tanner. Therefore, Rogers et al. (1995b) concluded that submaximal \( \dot{V}O_2 \) expressed as “ml·kg\(^{-0.67}\)·min\(^{-1}\)” is more appropriate statistically and physiologically, than a simple ratio standard using body mass, regardless of the treadmill speed. Unfortunately, the statistics and results presented by the authors were insufficient for readers to draw conclusions of their own because the coefficients of variation and the correlation coefficient for \( \dot{V}O_2 \) and body mass to the exponent 0.67 were not published.

When dealing with longitudinal data, special consideration must be given to the inter-correlations between separate measurements within an individual because the assumption of independence of samples is no longer valid (Vincent, 1999). Therefore, when calculating an allometric exponent within a longitudinal sample, some approach other than pooling all of the data into one log-transformed regression equation must be employed, otherwise, a basic assumption of linear regression analysis would be violated. Beunen, Baxter-Jones, Mirwald, Thomis, Lefevre, Malina and Bailey (2002) demonstrated the use of intra-individual allometric mass exponents in a sample of children and adolescents measured longitudinally. In order to calculate intra-individual allometric mass exponents, a regression line of log-transformed body mass and \( \dot{V}O_2 \) values was fit for each participant, thus producing a \( \beta \)-coefficient for each participant. The mean \( \beta \)-coefficient was then used as the exponent for the entire sample. In the Beunen et al. study, which used participants from the Saskatchewan Growth and Development Study (SGDS), the intra-individual allometry coefficients ranged from \( k' = 0.555 \) to \( k' = 1.178 \), with a mean value of \( k' = 0.855 \). As described in the following paragraph, the wide range of individual \( k' \) values is of particular interest.
The use of the intra-individual approach is not without its problems. In preliminary analyses of the data, Tanner’s Special Circumstance was used to determine the most appropriate method of calculating an allometric mass exponent for submaximal $\dot{V}O_2$ in a longitudinal sample of 63 boys. Of three approaches tested (pooled data, theoretical value 0.67 and intra-individual), the approach that had the largest difference between the quotient of CVs (0.818) and Pearson’s r (0.918) was the intraindividual allometric approach. Due to the wide range of individual k’ values (0.410 - 0.962), however, it was concluded that the importance of biological variability among individuals cannot be overlooked (Spencer, Baxter-Jones & Drinkwater, 2003; abstract). In other words, it is unlikely that one single exponent can be used for all individuals during childhood and adolescence. Clearly, more research is needed which investigates allometric scaling of submaximal $\dot{V}O_2$ values within a longitudinal sample.

The rationale for normalizing $\dot{V}O_2$ values to fat-free mass is that fat is a relatively avascular tissue, and therefore would not contribute significantly to oxygen consumption. However, some have argued that since total body mass is what is being lifted, lowered and supported during running, then eliminating fat for the purposes of normalizing to body size, is inappropriate (Morgan, 2000). Janz, Burns, Witt and Mahoney (1998) provided evidence of the suitability of using fat-free mass to normalize $\dot{V}O_2$ values for body size.

The relationship between submaximal $\dot{V}O_2$ and body mass and/or fat-free mass was investigated longitudinally in a group of 126 children, aged 10.3 + 1.0 (SD) years at entry (Janz et al., 1998). Participants had submaximal $\dot{V}O_2$ during stationary cycling, sexual maturation, height, mass and six skinfolds assessed annually for five years.
Using generalized estimating equations, regression coefficients were estimated to describe the relationship between log transformed mass or log transformed fat-free mass and log transformed \( \dot{V}O_2 \). Janz et al. elected to use the comparison based on Pearson’s correlation (r) between normalized \( \dot{V}O_2 \) and body mass or fat-free mass to assess whether the effects of body size had been effectively eliminated. Fat-free mass exponents of 0.82 and 0.71 for boys and girls, respectively, were found to satisfactorily normalize submaximal \( \dot{V}O_2 \) to body size, as the correlation coefficients were non-significant in both cases (values not reported). Janz et al. therefore concluded that allometric models using fat-free mass were most effective at eliminating the effects of body size on submaximal \( \dot{V}O_2 \). This finding supports further investigation into the use of fat-free mass to normalize \( \dot{V}O_2 \) to body size.

Another approach to normalizing \( \dot{V}O_2 \) values is a ratio using body surface area as the denominator. It has long been recognized that oxygen consumption and heat production per unit weight (ml·kg\(^{-1}\)·min\(^{-1}\) and °C·kg\(^{-1}\) respectively) are greater in small animals compared to large ones at rest (Von Bertalanffy, 1957). However, when these resting metabolism rates are expressed relative to body surface area the differences seem to disappear. During physical activity, however, an organism is required to move body mass rather than surface area. Therefore, animals with the greatest surface area to body mass ratio (i.e. the smaller animals) are at a metabolic disadvantage during exercise. This could, in part, explain why children possess poorer RE than adults.

In general, children demonstrate higher resting energy expenditure values than adults (Puhl, 1989). Rowland (1990) suggested that it is not unreasonable to expect that this relationship may persist during exercise. In fact, two separate studies conducted by
Rowland (Rowland et al., 1987; Rowland & Green, 1988) both support this concept. In the 1987 study 20 prepubertal boys, aged nine to 13 years, and twenty adult male subjects aged 23-33 years, all completed maximal treadmill tests (Rowland et al., 1987). The participants were all required to complete four separate stages of submaximal running (7.2, 8.0, 8.8 and 9.6 km·h\(^{-1}\) respectively). The 20 adults required approximately 23% less oxygen per kilogram of body mass per minute than the 20 prepubescent boys at 9.6 km·h\(^{-1}\). The differences in RE, however, seemed to disappear when \(\dot{V}O_2\) rates were expressed relative to body surface area, as no significant differences were noted between the two groups.

In an investigation of physiological responses to treadmill exercise in females, Rowland and Green (1988) provided support for their earlier findings. The sample consisted of 18 premenarchal girls (11.3 ± 1.1 years) and an equal number of adult females aged 28.7 ± 3.6 years. Oxygen consumption was assessed during incremental treadmill tests to exhaustion. Except at the lowest workload, the girls consumed significantly more oxygen per kilogram of body mass per minute than the women. However, similar to the previous study by Rowland et al. (1987), the differences in submaximal \(\dot{V}O_2\) disappeared when the values were expressed relative to body surface area. Both studies demonstrated that child-adult differences in submaximal \(\dot{V}O_2\) disappeared when expressed relative to body surface area rather than mass.

The previously discussed Rogers et al. (1995b) study tested the appropriateness of using 0.67 as an allometric mass exponent to normalize submaximal \(\dot{V}O_2\) to body size. Rogers et al. also tested the appropriateness of using body surface area to normalize \(\dot{V}O_2\) values. Based upon satisfaction of Tanner’s criteria, Rogers et al.
(1995b) concluded that a ratio using body surface area (ml·m$^{-2}$·min$^{-1}$) is also more appropriate than a simple ratio standard using body mass (Rogers et al., 1995b).

What is most evident throughout this section is that agreement regarding the single most appropriate approach to normalizing $\dot{\text{VO}}_2$ to body size has yet to be made. Therefore, using both Tanner’s Special Circumstance and the association between normalized $\dot{\text{VO}}_2$ and a measure of body size, the appropriateness of several approaches were tested in the present study.

### 1.2.3. Importance of Running Economy to Endurance Running Performance

Athletes, coaches and researchers involved with middle- and long-distance running would have a deep appreciation for a greater understanding of factors that contribute to athletic performance. Research has demonstrated that RE, normalized using body mass (ml·kg$^{-1}$·min$^{-1}$) is related to better endurance running performance among children with comparable $\dot{\text{VO}}_2$:max (ml·kg$^{-1}$·min$^{-1}$) values (Daniels & Oldridge, 1971; Daniels et al., 1978a). Rowland et al., (1988) also demonstrated a significant positive relationship between RE (ml·kg$^{-1}$·min$^{-1}$) and endurance running performance in a group of boys who were heterogeneous with respect to $\dot{\text{VO}}_2$:max.

Twenty-eight prepubertal boys, aged nine to 14, completed an incremental treadmill test to determine submaximal $\dot{\text{VO}}_2$ and $\dot{\text{VO}}_2$:max as well as a measure of treadmill running time (Rowland et al., 1988). Two definitions of RE were used for this investigation. The first was $\dot{\text{VO}}_2$ per kilogram of body mass at a running speed of 9.6 km·h$^{-1}$. The second was the $\dot{\text{VO}}_2$ costs for increasing running speed by 1.6 km·h$^{-1}$.

Pearson’s correlation analysis revealed a significant negative relationship ($r =$
-0.44; p<0.05 for $\dot{V}O_2$ at 9.6 km·h$^{-1}$; $r = -0.58$; p<0.05 for $\Delta \dot{V}O_2$) between treadmill running time and RE, regardless of the definition used to describe RE. This result indicates that those who demonstrated better RE were capable of running for a longer period of time before reaching exhaustion. Although $\dot{V}O_2$max was also significantly positively correlated with endurance running performance ($r = 0.85$, p<0.05), no significant relationship was demonstrated between $\dot{V}O_2$max and RE. The researchers therefore concluded that RE in prepubertal boys bears a strong negative relationship to treadmill running time.

Unnithan, Timmons, Paton and Rowland (1995) conducted an investigation to describe the relationship between running performance and selected physiological measures (including RE) in run-trained prepubertal children. Thirteen male participants, who had a mean age of 11.7 ± 1.1 (SD) years, completed a 3000m time trial, four stages of submaximal treadmill running (eight, 9.6, 11.2 and 11.8 km·h$^{-1}$) and a peak $\dot{V}O_2$ test, which assessed the highest (peak) value of $\dot{V}O_2$ attained during an exhaustive treadmill run. The results of Pearson’s correlation analyses revealed a significant negative correlation ($r = -0.60$; p<0.05) between 3000m time and $\dot{V}O_2$ at eight km·h$^{-1}$ (ml·kg$^{-1}$·min$^{-1}$); in other words, the poorer the economy, the better the running performance. At the other three submaximal speeds, RE was not significantly correlated with running performance. Unnithan et al. point out, however, that these data contradict the majority of the work in this area. They further suggested that RE is perhaps more important in contributing to performance in longer races.

Fourteen boys, aged ten to 15, participated in a longitudinal study that investigated changes in $\dot{V}O_2$ during growth and training (Daniels & Oldridge, 1971).
The participants, who trained regularly using distance running over a 22-month period, were tested semi-annually for height, mass, submaximal $\dot{V}O_2$ at 202 m/min (12.12 km·h$^{-1}$) on a treadmill, and peak $\dot{V}O_2$. Additionally, the researchers recorded the participants’ competitive endurance running performances throughout the course of the study. When $\dot{V}O_2$ values were expressed relative to body mass (ml·kg$^{-1}$·min$^{-1}$), $\dot{V}O_2$ at 202 m/min decreased significantly with increasing chronological age, while peak $\dot{V}O_2$ showed no significant change over the duration of the study. The decreases in submaximal $\dot{V}O_2$ (increased economy) were accompanied by significant improvements in 1- and 2-mile run times of the participants. Daniels and Oldridge therefore concluded that improvements to endurance running performance were, in part, the result of improved efficiency of locomotion. One advantage of this study over the previously mentioned Rowland et al. study (1988) is that the longitudinal design of this study allowed researchers to monitor changes within individuals, rather than comparing two groups.

Daniels et al. (1978a) used a mixed-longitudinal design, a combination of cross-sectional and longitudinal designs where some participants are measured on all occasions, and others only on some occasions, to investigate differences and changes in $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) among young runners. The sample comprised 20 boys, aged 10-18 years. Participants were tested semi-annually for between two and five years. Despite the fact that $\dot{V}O_2$max (ml·kg$^{-1}$·min$^{-1}$), remained constant throughout the duration of the study, submaximal $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) at 12 km·h$^{-1}$ on a treadmill, and 1- and 2-mile run times decreased significantly. Therefore, Daniels et al. concluded that the improvements in distance running performance were due to improvements in RE. The
two studies presented above are similar; nonetheless they provide reliable evidence of a positive association between RE and endurance running performance in children and adolescents.

Three of the four studies presented above provide evidence of a positive association between RE and running performance (Daniels & Oldridge, 1971; Daniels et al., 1978a; Rowland et al., 1988). Since RE has been linked with endurance running performance in children and adolescents, it could be argued that a clear understanding of how and when chronological age-related changes in RE occur may provide an explanation, in part, for interindividual differences in endurance running performance. Also, since growth and maturation have been neglected somewhat in previous research on RE and chronological age, and the present study addresses these omissions, this study is potentially important for generating a better understanding of the development of RE during childhood and adolescents.

1.2.4. Age and Running Economy

1.2.4.1. Chronological Age and Running Economy

Chronological age is defined as the elapsed time since birth. Although two children may share the same chronological age, they are not necessarily equally mature. For example, one 14 year old male could have surpassed the age of PHV, while another may be a year or more away from attaining PHV. Biological age, which was discussed previously, can be calculated and used to evaluate maturation, or progression towards the mature state. Evidence from several studies indicates that RE improves with increasing chronological age when expressed relative to body mass and that adults possess greater RE than children at comparable workloads (Ariëns et al., 1997; Åstrand,
More than 65 years ago, Robinson provided evidence that younger children use greater quantities of oxygen per kilogram of body mass than older children and adults during submaximal exercise (Robinson, 1938). Using a cross-sectional sample of 92 healthy non-athletic males, ranging in age from six to 91 years, Robinson assessed oxygen consumption during treadmill walking at a speed of 5.6 km·h\(^{-1}\) at a grade of 8.6\%. It should be noted that the same treadmill protocols are not used in all studies that investigate the same variables; this can make direct comparisons among studies somewhat cumbersome. For comparison purposes, participants were grouped based upon chronological age. When expressed relative to body mass (ml·kg\(^{-1}\)·min\(^{-1}\)), \(\dot{V}O_2\) was approximately 22\% higher in the two groups of youngest boys as compared to the older groups. The boys in these two groups had a mean age of six and 10.5 years, respectively; the next oldest group had a mean age of 14.1 years. Similar results have consistently been demonstrated in subsequent studies.

Åstrand (1952) demonstrated that younger children possess poorer RE than older children and adolescents. Åstrand assessed \(\dot{V}O_2\) (ml·kg\(^{-1}\)·min\(^{-1}\)) of children and adolescents, aged four to 18 years, during flat treadmill runs at various speeds ranging from eight to 16 km·h\(^{-1}\). When submaximal \(\dot{V}O_2\) was expressed relative to body mass (ml·kg\(^{-1}\)·min\(^{-1}\)), older participants consumed significantly less oxygen per minute than their younger counterparts at common workloads. Åstrand concluded that the running of younger participants was less economical that that of the older participants; a finding which supported the work of Robinson from 14 years earlier.
More recently, Montoye demonstrated that submaximal oxygen consumption rates declined with increasing chronological age in a large sample of male participants (Montoye, 1982). One thousand and fifty-eight males, aged ten to 69, walked on a treadmill, at 4.8 km·h\(^{-1}\), with increasing grade (three percent every three minutes). Oxygen consumption was recorded at each grade. When expressed relative to body mass (ml·kg\(^{-1}\)·min\(^{-1}\)), younger participants consumed more oxygen than their older counterparts at all workloads. Montoye therefore concluded that RE was related to chronological age. This study is important because it verified the findings of previous researchers; however, it did so using a larger sample. The use of a large sample is essential because the probability of obtaining a biased sample decreases as the sample size decreases; in other words, larger samples are more likely to be representative of the population than smaller samples (Vincent, 1999).

MacDougall, Roche, Bar-Or and Moroz (1983) provided further evidence of an association of RE and chronological age in their attempt to develop a formula to predict \(\dot{V}O_2\)\textsubscript{max} in Canadian schoolchildren. A cross-sectional sample of 134 male and female participants, aged seven to 16 years, participated in this study. \(\dot{V}O_2\) was measured at two submaximal running speeds, ranging from 122-265 m·min\(^{-1}\), on a horizontal treadmill. Since not all participants ran at the same speeds, regression equations were derived to describe the relationship between running speed (workload) and \(\dot{V}O_2\) for four groups, based upon age. The four groups were seven to nine year-olds, ten to 12, 13-14, and 15-16 year-olds. When \(\dot{V}O_2\) values were expressed relative to body mass (ml·kg\(^{-1}\)·min\(^{-1}\)), and the four groups were compared at common treadmill speeds, the youngest group demonstrated significantly higher \(\dot{V}O_2\) values (poorer RE) than the
older participants. For example, at 180 m·min\(^{-1}\), the youngest group was consuming approximately 18.5% more oxygen per kilogram of body mass per minute than the oldest group. Although their primary intent was not to investigate the relationship between chronological age and submaximal \(\dot{V}O_2\), MacDougall et al. provided clear evidence of such an association.

Twenty prepubertal boys, aged nine to 13 years, and twenty adult male subjects aged 23-33 years, all completed maximal treadmill tests in a study comparing physiological responses to treadmill running between the two groups (Rowland et al., 1987). The participants were all required to complete four separate stages of submaximal running (7.2, 8.0, 8.8 and 9.6 km·h\(^{-1}\) respectively). Once oxygen consumption rates were adjusted for individual body mass differences (ml·kg\(^{-1}\)·min\(^{-1}\)), the children were found to consume greater \(\dot{V}O_2\) than adults, even when running at slightly slower speeds. At 9.6 km·h\(^{-1}\), for example, the children were consuming approximately 23% more oxygen per kilogram of body mass per minute than their adult counterparts. The authors therefore concluded that the children possessed poorer RE than adults at the same workload.

Unnithan and Eston (1990) suggested a drawback to the Rowland study was that the prepubertal group (\(\dot{V}O_2\)max = 57.9 ml·kg\(^{-1}\)·min\(^{-1}\)) possessed significantly greater aerobic fitness than the adult group (\(\dot{V}O_2\)max = 48.3 ml·kg\(^{-1}\)·min\(^{-1}\)). It was postulated that the differences in RE between the two groups could have been due to differences in the degree of aerobic fitness between the two groups. Unnithan and Eston (1990) used the same treadmill protocol as Rowland to investigate the effects of different stride frequencies on RE. The sample in this study consisted of ten boys from a school cross...
country team and ten healthy and trained male university students (mean ages: 10.4 \( \pm \) 0.5 years and 20.8 \( \pm \) 1.2 years respectively). No significant difference between the two groups’ aerobic fitness (\( \dot{VO}_{2\text{max}} \); ml·kg\(^{-1}\)·min\(^{-1}\)) was demonstrated; however, the adult group did exhibit greater RE than the child group. For example, at 9.6 km·h\(^{-1}\) the children were consuming approximately 19% more oxygen per kilogram of body mass per minute than their adult counterparts. This number is comparable to the 23% difference demonstrated by the participants in the Rowland et al. study (1987). A significant finding from the Unnithan and Eston study is that differences in submaximal \( \dot{VO}_{2} \) do not appear to be the result of differences in maximal aerobic power; this finding is in opposition to the researchers’ hypothesis. The strength of the relationship between \( \dot{VO}_{2\text{max}} \) and RE appears to be most significant when both are used to describe endurance performance, as they both appear to contribute to overall performance.

Each of the previously mentioned studies that examined adult – child differences were cross-sectional in design. The findings from all of these studies are similar in that they provide evidence of greater economy being possessed by older participants, when expressed relative to body mass. However, a longitudinal design is preferable to investigate the development of RE relative to chronological age (Ariëns et al., 1997) as it allows the determination of intra-individual variability and possibly allows for an assessment of maturational status. As mentioned, a longitudinal study design is essential for assessing and controlling for change within individuals.

Two previously mentioned studies are relevant here; Daniels et al. (1978a) conducted a study of RE development using a mixed-longitudinal design. The results of their study demonstrated that \( VO_{2} \) (ml·kg\(^{-1}\)·min\(^{-1}\)) during horizontal treadmill running at
202 m·min\(^{-1}\) decreased significantly with increasing chronological age. Daniels et al. therefore concluded that RE significantly improves with increasing chronological age. Daniels and Oldridge (1971) completed a longitudinal investigation of changes in \(\dot{V}O_2\) of young boys during growth and training. The results of this study, which have been described previously, support Daniels et al. (1978) and all of the aforementioned cross-sectional studies investigating RE and age. RE, as measured by flat treadmill running at 202 m·min\(^{-1}\), was found to decrease with increasing chronological age, when expressed relative to body mass.

One hundred eighty-two participants (84 males, 98 females) completed submaximal treadmill tests at a constant speed of eight km·h\(^{-1}\), with increasing slopes (0%, 2.5% and 5% grade), in a longitudinal examination of RE development in males and females (Ariëns et al., 1997). Testing was carried out on the same individuals on six separate occasions, at the ages of 13, 14, 15, 16, 21, and 27 years. At all three treadmill grades, a significant decrease in mass-relative \(\dot{V}O_2\) (ml·kg\(^{-1}\)·min\(^{-1}\)) was found with increasing chronological age in both males and females. As expected, the researchers concluded there were significant improvements in RE with increasing chronological age. A drawback to this study was that the researchers failed to account for individual differences in maturity status. As mentioned, individuals of the same chronological age may not share the same biological age. Thus, by failing to account for maturational differences between participants, Ariëns et al. may have grouped participants inappropriately. The importance of assessing and controlling for individual differences in maturation is emphasized in the next section (1.2.4.2. Biological Age, Maturity Status and Running Economy).
Improved endurance running performance (Daniels & Oldridge, 1971; Daniels et al., 1978a) and improved RE (Ariëns et al., 1997; Daniels & Oldridge, 1971; Daniels et al., 1978a), when expressed relative to body mass, have been demonstrated with increasing chronological age within individuals. Furthermore, cross-sectional studies investigating RE and chronological age have provided strong evidence of an association between the two (Åstrand, 1952; Maliszewski & Freedson, 1996; Montoye, 1982; Robinson, 1938; Rowland et al., 1987; Unnithan & Eston, 1990). Many researchers have discussed possible explanations why these changes occur; yet they remain largely untested in a longitudinal study spanning the adolescent years. A longitudinal study which spans the adolescent years is essential for a deeper understanding of the development of RE from childhood through adolescence, and finally, to adulthood.

1.2.4.2. Biological Age, Maturity Status and Running Economy

Endurance running performance steadily improves throughout childhood. As mentioned, this appears to be, at least partly, the result of improvements in RE (Daniels & Oldridge, 1971; Daniels et al., 1978a; Morgan, Martin, & Krahenbuhl, 1989; Rowland et al., 1988). However, it remains unclear whether these improvements in RE are the result of training, physiological adaptations, advanced maturational status, or simply an increase in size. There is considerable variability in physical characteristics (mass, height, etc.) among individuals of the same chronological age, especially during the adolescent years (Claessens et al., 2000). Therefore, studies examining improvements in RE, should control for the normal growth and maturational processes that occur during childhood and adolescence.

Cunningham et al. (1984) used age at PHV to account for maturity differences
among boys in a longitudinal study that investigated the development of cardiorespiratory function in young males. The sample tested in this study consisted of 62 boys aged nine and ten at entry. Chronological age, height and mass were measured annually for six consecutive years. \( \dot{VO}_2 \) at a heart rate between 145-155 beats per minute on a cycle ergometer was also assessed. To make direct comparisons between individuals, the researchers predicted \( \dot{VO}_2 \) at a heart rate of 155 beats per minute (\( \dot{VO}_{155} \)) by interpolation of surrounding measures. Participants were grouped into three maturity groups (early, average and late) based upon age at PHV. The researchers demonstrated that \( \dot{VO}_{155} \) (L/min) was significantly greater in late-maturing boys than early- or average-maturing boys when compared at common biological ages. For example, when compared at the age of PHV, the late-maturing boys were consuming approximately 20% and 24% more oxygen per minute than the early- and average-maturing boys, respectively.

A possible drawback to the Cunningham et al. (1984) study was that the researchers chose to present data on absolute \( \dot{VO}_2 \) (L/min), rather than some type of relative approach (i.e. normalized to body size). Although maturity differences between the individuals were addressed, size differences were not. The importance of adjusting \( \dot{VO}_2 \) values for size cannot be overlooked; \( \dot{VO}_2 \) is a size-dependent variable. The authors reported that there were no significant height and mass differences among the three groups; however, these values were group means and therefore do not necessarily indicate that the group differences for absolute \( \dot{VO}_2 \) (L/min) would persist when normalized to body size. It is worth noting that this may, in fact, not be a flaw, as participants’ mass was supported during cycling, and therefore some may argue that it is
more appropriate to report absolute values in L/min. Another possible fault with the Cunningham et al. study is that participants’ \( \dot{VO}_2 \) values were compared at a common heart of 155 beats per minute. Wirth, Trager, Scheele, Mayer, Diehm, Reischle and Weicker (1978) produced evidence to suggest that working at “a given percentage of \( \dot{VO}_2 \max \) as a reference unit, is more reliable than a certain HR to obtain comparable results in subjects with different ages.” Despite these perceived shortcomings, the evidence presented by Cunningham et al. indicates one of two things: that late-maturing boys are less economical than early or average maturers, or that the late-maturing boys were different in size during adolescence.

Bouchard, Malina, Hollmann and Leblanc (1976) attempted to assess the relation between maturity (skeletal) and submaximal \( \dot{VO}_2 \) in boys aged eight to 18 years. All 237 participants had skeletal age, chronological age, height and body mass (among other variables) assessed on one occasion. \( \dot{VO}_2 \) during stationary cycling was also assessed. In order to make comparisons at a common heart rate, the researchers calculated \( \dot{VO}_2 \) at a heart rate of 130 beats per minute (\( \dot{VO}_{130} \)) by interpolation.

Correlations between skeletal age and \( \dot{VO}_{130} \) (\( r = 0.795 \)) and chronological age and \( \dot{VO}_{130} \) (\( r = 0.767 \)) were not significantly different from one another (\( p>0.05 \)), indicating that both skeletal and chronological age were equally well associated with \( \dot{VO}_{130} \). Also, multiple regression analyses were performed to predict \( \dot{VO}_{130} \) from height, mass, chronological age and skeletal age. Results revealed that body mass (\( \Delta R^2 = 0.641 \)) and chronological age (\( \Delta R^2 = 0.036 \)) contributed more to the explained variance (\( \Delta R^2 = 0.680 \)) than skeletal age (\( \Delta R^2 = 0.003 \)) or height (\( \Delta R^2 = 0.000 \)). When skeletal age was removed from the analysis, \( R^2 = 0.678 \), not tremendously different from
$R^2 = 0.680$ when skeletal age was included in the model. Bouchard et al. (1976), therefore, concluded that skeletal age was not a significant factor in explaining submaximal $\dot{V}O_2$ rate, beyond chronological age and body mass.

Bouchard et al. (1976) is among the few studies that have investigated how maturity status affects submaximal $\dot{V}O_2$. A drawback to the Bouchard et al. (1976) study, however, is the failure to report co-linearity statistics, as height, mass, chronological age and skeletal age are likely highly related. It would be interesting to know how well chronological age and skeletal age predicted $\dot{V}O_{130}$ in the absence of body mass. All the same, the results of this investigation support the conclusion drawn by the authors; that skeletal age was not a significant factor, beyond height, mass and chronological age, in explaining $\dot{V}O_2$ during submaximal exercise.

Despite the conclusions drawn from the 1976 paper, Bouchard, Leblanc, Malina and Hollmann (1978) further investigated the relationship between skeletal age and submaximal oxygen consumption ($\dot{V}O_{130}$), based upon the possibility of a closer association between the two variables among circumpubertal participants (i.e. those in the midst of puberty). The same data were used in this study as already described (Bouchard et al., 1976). Of note is the use of partial correlations in this paper. Bouchard et al. clearly recognized the close association (i.e. co-linearity) between the anthropometric variables and age during adolescence, and therefore addressed these concerns in this latter paper where they had been missed in the 1976 paper.

Chronological age-specific correlations showed that $\dot{V}O_{130}$ (L/min) was significantly (p<0.05) associated with skeletal age only from the ages of 12-16 years. Therefore, Bouchard et al. (1978) grouped the participants into three subsets based upon
age, 8-11 (n = 77), 12-16 (n = 118) and 17-18 (n = 42). Partial correlations, the correlation between the dependent variable and an independent variable when the linear effects of the other independent variables in the model have been removed from both, were used in the analyses in this study. Partialing out skeletal age resulted in significant decreases in the correlations of either height or mass with $\dot{V}O_{130}$ (L/min) only in the 12-16 year-old subset. Additionally, the total amount of variance explained in $\dot{V}O_{130}$ (L/min), by multiple linear regression using chronological age, height and mass as predictors, was significantly affected by the presence of skeletal age only in the 12-16 year-old subset. By the authors’ own admission, however, the bulk of the variance in $\dot{V}O_{130}$ was explained by differences in height and mass between individuals. Nonetheless, these results indicate that skeletal age, a marker of maturity, has a greater association with submaximal oxygen consumption during puberty, rather than before or after puberty, in males.

The conclusion drawn by Bouchard et al. (1978) seems straightforward; however, the interpretation and application of the results is not. In order to apply the knowledge gained from this study, one must understand the nature of the relationship between submaximal $\dot{V}O_2$ and skeletal age, as it is presented. Based upon previous studies, one might expect that the correlation coefficients for the two variables to be negative, indicating improved economy with advanced skeletal maturity. However, this expectation is based upon the assumption that economy would be normalized to body size; yet, Bouchard et al. failed to account for differences in body size in the initial correlations. $\dot{V}O_2$ is known to be a size-dependent variable, and advanced skeletal age is obviously associated with increases in body size during adolescence, the implications
of the conclusion drawn by the researchers are therefore difficult to understand. Perhaps the best lesson learned is that the relationship between maturity and submaximal VO₂ during adolescence is worthy of further study.

Armstrong, Welsman, and Kirby (1999) conducted a study that investigated the effects of maturation on RE. The cross-sectional sample consisted of 97 males and 97 females, aged 12.2 ± 0.4 years. Participants’ maturation was assessed using Tanner’s indices of pubic hair development; a gender-specific, subjective assessment of maturity. Submaximal VO₂ was assessed during a discontinuous, incremental exercise test on a horizontal treadmill. The running speeds were eight, nine, and ten km·h⁻¹ respectively. Results showed that when size differences were not controlled for, either by body mass or allometric ratios, there was a significant main effect of maturation in both sexes; that is, higher levels of VO₂ (L/min) were associated with advanced maturational status. This is logical since advanced maturation is often associated with increased size. When the participants’ size was accounted for, using either a per body mass or an allometric ratio, there was no longer a significant main effect of maturation on submaximal VO₂ (ml·kg⁻¹·min⁻¹).

Armstrong et al. (1999) demonstrated that the effects of maturation were non-significant once size was accounted for. This study, therefore, is among the few that has shed light on whether the timing of maturation affects the development of RE. The evidence from this study does not support an effect of timing of maturation, since all of the participants were of similar chronological age, but not of the same maturity status. As mentioned, however, a longitudinal design is preferable to investigate the development of RE relative to chronological or biological age (Ariëns et al., 1997).
Welsman and Armstrong (2000) followed-up on the 1999 study with a three-year longitudinal investigation into changes in submaximal VO₂ during treadmill running in a group of 11-13 year-olds. The participants, 118 boys and 118 girls, had a mean age of 11.2 ± 0.4 years at study entry. Submaximal VO₂ was measured at eight km·h⁻¹. Selected anthropometric variables were recorded annually for three years. Sexual maturation was assessed using Tanner’s indices of pubic hair development.

The researchers used two separate approaches to normalize VO₂ for size differences; per body mass ratios and allometric ratios. Data from male and female participants were analyzed together using a multi-level modeling approach. Regardless of the approach used to control for size differences, the effects of maturity on RE were non-significant in males and females. These findings are in agreement with those of the previously mentioned cross-sectional study by two of the same authors, but are in opposition to the findings of Cunningham et al. (1984).

In summary, the bulk of the evidence regarding the effects of maturity status on RE indicates that there is no significant relationship between the two variables once size differences have been accounted for (Armstrong et al., 1999; Bouchard et al., 1976; Welsman & Armstrong, 2000); however, the findings from Cunningham et al. (1984) and Bouchard et al. (1978) support further investigation into the relationship between maturity status and RE.

1.2.5. Other Factors That May Explain Individual Differences in RE

As discussed, RE has been shown to be related to body size and chronological age, and may be associated with maturity; however, even after controlling for
differences in size, age and maturity status, RE can vary greatly within a group of individuals. Researchers have suggested several explanations for variations in RE among individuals. Such explanations include, but are not limited to, greater resting energy expenditure (Frost et al., 2002), differences in gait pattern, stride length and frequency, leg length, segmental mass distribution, ventilation, substrate utilization (Morgan, 2000; Unnithan & Eston, 1990), training (Daniels et al., 1978), age and gender (Ariëns et al., 1997). In order to explain how RE affects endurance running performance, one must first be able to explain individual variations in RE. Therefore the following section examines several suggested explanations for inter-individual differences in RE.

Although they were not the only group to attempt to explain differences in RE, Frost et al. (2002) were the most recent group to attempt this. \( \dot{VO}_2 \) was assessed in 30 participants during submaximal treadmill exercise at six speeds; two walking and four running. The participants were aged seven and eight years \((n = 10)\), ten to 12 years \((n = 10)\) and 15-16 years \((n = 10)\). Kinematic variables such as total body mechanical power, energy transfer rates and stride rates, as well as electromyographic data on co-contraction of agonist and antagonist muscles in the thigh and leg segments were also recorded and analyzed. Multiple linear regression analyses were performed with net \( \dot{VO}_2 \) (exercising - resting \( \dot{VO}_2 \) values) or efficiency (work performed divided by \( \dot{VO}_2 \)) as the dependent variable and mechanical power, thigh and leg co-contraction, stride rate and age as the independent variables. Frost et al. demonstrated the ability to explain 77% and 62% of the variance in net \( \dot{VO}_2 \) and efficiency, respectively. Chronological age was the single best predictor of net \( \dot{VO}_2 \) and efficiency. Another important finding
to come out of this study was that when co-contraction indices for the thigh and leg were
the sole independent variables in the prediction of net VO2 and efficiency, more of the
variance in these dependant variables was explained for the younger participants than
the older ones. This finding suggests that an interaction between co-contraction (thigh,
leg or both) and age affect both net VO2 and efficiency (Frost et al., 2002).

Although much of the variance in RE can be explained, a significant proportion
remains unexplained. Therefore, there exists a need to continue to investigate the
predictive effects of different independent variables on RE.

The following sections examine selected variables hypothesized to relate to
interindividual differences in RE. The rationale for including each of the following
variables, and not others, is that only those that can be addressed using the SGDS data
are discussed.

1.2.5.1. Training and Physical Activity

Several review articles which focus on RE have discussed the possibility that
training status may contribute to inter-individual differences in economy. Most
researchers agree that the effects of training on RE are still relatively unknown, and the
effects of physical activity have not been widely investigated. The purpose of this
section is to discuss the possible chronic effects of physical activity on RE.

Exercise training is the systematic use of exercise of specific intensities,
durations, and frequencies to attain a desired fitness effect (Baranowski, Bouchard, Bar-
Or, Bricker, Heath, Kimm, Malina, Obazanek, Pate, Strong, Truman & Washington,
1992). This term is often restricted to a subset of physical activity with higher levels of
intensity of exertion. According to Malina (1996), physical activity refers to any body movement produced by the skeletal muscles and resulting in a substantial increase over the resting energy expenditure. Therefore, training refers to voluntary body movement of specific intensity, duration and frequency to attain a desired fitness effect.

As previously discussed, Daniels et al. (1978b) demonstrated that RE improved significantly between the ages of 10 and 18 in a group of run-trained boys. The initial cohort in the mixed-longitudinal study began at age ten and was tested semi-annually for a minimum of two years. Following two years of run training, the submaximal $\dot{V}O_2$ values from these boys were compared to those of a group of 12 year-old boys who were entering the study, and therefore beginning run training. The initial cohort demonstrated a significant decrease in submaximal $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) over a two-year period. They also demonstrated significantly lower submaximal $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) values than the newly introduced 12 year-old cohort. Based upon the comparison of the two cohorts, Daniels et al. (1978b) concluded that training was partly responsible for the RE differences between the two groups. However, this conclusion does not appear to be completely supported by the results. Since data at age ten were not available for the second cohort, any conclusion regarding the effectiveness of training within the first cohort should be interpreted cautiously. Nevertheless, the possibility of an effect of training on RE that is raised by the authors warrants further investigation.

Patton and Vogel (1977) demonstrated the effectiveness of a training program on RE in 60 untrained and 60 trained military personnel aged 17-35. Following a six-month conditioning program, the participants from both groups demonstrated significant improvements to RE when compared with baseline values. The untrained personnel also
demonstrated significant improvements to \( \dot{\text{VO}}_{\text{max}} \) during the course of the study. These results provide some support for the claim that training can improve RE.

Svedenhag and Sjödin (1985) also provided support of a positive effect of training on RE in a group of elite male runners. Ten members of the Swedish men’s national track team participated in treadmill tests on four separate occasions over a one-year period. Submaximal \( \dot{\text{VO}}_2 \) was assessed during flat treadmill running at 15 and 20 km\( \cdot \)h\(^{-1} \). The authors noted slightly lower values of submaximal \( \dot{\text{VO}}_2 \) (ml\( \cdot \)kg\(^{-1} \)\( \cdot \)min\(^{-1} \)) at each of the two treadmill speeds throughout the testing period, with significant changes occurring at the higher speed after one year. These findings led Svedenhag and Sjödin to conclude that slow, but steady improvements in RE are associated with training, even in highly trained adult runners.

A 14-week intervention study of changes in onset of blood lactate accumulation within eight well-trained runners added to the evidence that suggests an association between training and RE. The training intervention consisted of adding one 20-minute run at a treadmill velocity calculated to elicit a blood lactate concentration of 4 mmol\( \cdot \)L\(^{-1} \) (Sjödin, Jacobs & Svedenhag, 1982). Submaximal \( \dot{\text{VO}}_2 \) was assessed at the beginning and end of the 14-week training period at a treadmill speed of 15 km\( \cdot \)h\(^{-1} \). The results revealed a significant decrease in submaximal \( \dot{\text{VO}}_2 \) (ml\( \cdot \)kg\(^{-1} \)\( \cdot \)min\(^{-1} \)) from baseline values; the additional training resulted in a three percent decrease in submaximal \( \dot{\text{VO}}_2 \), from 50.6 to 49.2 ml\( \cdot \)kg\(^{-1} \)\( \cdot \)min\(^{-1} \). Although this was not the primary focus of the paper, Sjödin et al. produced evidence of an effect of training on RE.

Two separate case studies contributed evidence of improved RE as a result of training (Conley et al., 1981; Conley et al., 1984). Conley et al. (1981) assessed the
physiological changes in a 31-year-old elite runner who was engaged in an 18-week run training program. Body mass, percentage body fat, RE and $\dot{V}O_2\text{max}$ were assessed weekly for the duration of the study. The results indicated that, when expressed relative to body mass, there was a 9-18% decrease in submaximal $\dot{V}O_2$ over the course of 18 weeks, depending upon the workload. These gains in RE were accompanied by significant decreases in body mass and percentage body fat; thus, the rate of $\dot{V}O_2$ (L/min) at any given workload must have been decreasing at a faster rate than decreases in body mass or percentage body fat. Conley et al. (1981) therefore produced evidence of a training effect on RE in an elite, adult runner.

Conley followed up the 1981 study with a similar investigation in 1984. The results of the subsequent study supported the earlier results. Champion runner Steve Scott was studied for a period of nine months, including part of an off-season, the complete preseason and indoor track season, and part of the outdoor track season. During most of the study’s duration Scott was engaged in some form of run training. Submaximal $\dot{V}O_2$ was assessed on three separate occasions during horizontal treadmill running at speeds of 14.4, 16 and 17.6 km·h$^{-1}$. Results indicated that Scott’s RE had improved by five percent over the study’s duration, while his body mass varied less than one kilogram during the same time. Therefore, Conley et al. (1984) concluded that participation in run training could lead to improvements in RE (ml·kg$^{-1}$·min$^{-1}$).

Mahon and Vaccaro (1994) highlighted a likely mechanism responsible for changes in submaximal $\dot{V}O_2$ with training. They demonstrated significant increases in arterial-venous oxygen difference at submaximal treadmill workloads corresponding to 50 and 75% of $\dot{V}O_2\text{max}$ following 14 weeks of training. Since $\dot{V}O_2\text{max}$ significantly
increased during the 14-week period, the absolute workloads corresponding to 50 and 75% were obviously higher, thus causing the participants to consume more oxygen. The Fick equation (Wilmore & Costill, 1999) states that $\dot{V}O_2$ is the product of cardiac output ($\dot{Q}$; the volume of blood pumped from the heart in one minute) and arterial-venous oxygen difference. Changes to arterial-venous oxygen difference would indicate a greater or lesser ability of working skeletal muscle to extract oxygen from capillaries; these changes may be the result of training. With regards to changes in $\dot{Q}$, Armstrong and Welsman (2002) demonstrated that they are essentially in direct proportion to body surface area in males and females aged 11-13s.

Though some studies have provided support for an effect of training on RE (Conley et al., 1981; Conley et al., 1984; Daniels et al., 1978b; Patton & Vogel, 1977; Svedenhag & Sjödin, 1985), others have demonstrated no relation between training and RE (Daniels et al., 1978b; Petray & Krahenbuhl, 1985; Unnithan, Timmons, Brogan, Paton & Rowland, 1996; Wilcox & Bulbulian, 1984). Daniels et al. (1978b) found no change in RE in 15 trained runners (21.8 ± 3.1 years) following an eight-week training period. The 15 male participants increased the volume and intensity of their training during an eight week period between baseline and final testing sessions. Submaximal $\dot{V}O_2$ was determined during incremental treadmill running with increasing treadmill grade. Despite the fact that running performances improved during the training period, maximal and submaximal $\dot{V}O_2$ values showed no significant changes. Daniels et al. (1978b) therefore concluded that exposure to run training does not affect RE.

Similarly, an investigation using seven female college runners, aged 19.9 ± 1.1 (SD) years, demonstrated no significant differences in RE following an eight-week
cross-country training session (Wilcox & Bulbulian, 1984). At baseline, the participants had $\dot{V}O_2$ at 12.8 and 14.4 km$h^{-1}$, and $\dot{V}O_2$max using an incremental treadmill test assessed. No significant RE differences were demonstrated (at either treadmill speed) between baseline and after eight weeks of cross country run-training.

Petray & Krahenbuhl (1985) studied the effects of run training, instruction on running technique and a combination of instruction and training, on the RE of 50 ten-year-old children. No significant differences in RE (ml·kg$^{-1}$·min$^{-1}$) or running technique were demonstrated following the 12-week training/instruction intervention. Petray and Krahenbuhl therefore concluded that short-term exposure to instruction and training, alone or in combination, did not affect RE in young boys.

The research into the effects of training on RE are essentially equivocal. In fact, all of the review articles dealing with RE presented here agree that the effects of training on RE remain largely unknown (Bailey & Pate, 1991; Krahenbuhl & Williams, 1992; Morgan et al., 1989). Though the effects of training on RE may be equivocal, it seems as though long-term exposure to this stimulus may have a more profound effect than short-term exposure. In a review article, Krahenbuhl and Williams (p. 465, 1992) summarize the findings as, “…training, at least over a short term (2-3 months), results in little or no improvement in running economy during childhood and adolescence. Over the longer term (years), improvements in running economy may be augmented through participation in running training programs.” Since it appears that long-term exposure to training may affect RE, and training is a specialized type of physical activity, it seems reasonable to suppose that, or at least to investigate whether, regular exposure to physical activity over a long period of time may produce similar results; however, few
studies have investigated the effects of physical activity on RE development. Additionally, the relationship between physical activity and training is tenuous at best; since no measure of training was available in the data analyzed, the effects of physical activity were examined.

Using participants aged eight to 16 years from the SGDS, Mirwald and Bailey (1982) were among the few to investigate differences in submaximal aerobic power in active and inactive boys. Maximal aerobic power, submaximal $\dot{V}O_2$ at a treadmill speed of 9.6 km·h$^{-1}$, and a variety of anthropometric measurements were taken annually for 10 years. Additionally, physical activity level was assessed using a variety of techniques, and participants were categorized as either active ($n = 17$) or inactive ($n = 13$).

The results indicated that submaximal $\dot{V}O_2$ (L/min) for the two groups were not significantly different. The authors did not provide any information regarding body mass differences between the two groups, therefore the effects of size are unknown. Of note, however is the fact that when submaximal $\dot{V}O_2$ was expressed as a percentage of $\dot{V}O_2$ max, the active group demonstrated a lower required proportion of maximal aerobic power to perform submaximal work from ages eight to 16. The only age where this difference was significant, however, was at 15 years. The smaller percentage of $\dot{V}O_2$ max required by the active group is most likely the result of superior maximal aerobic power, rather than superior economy, since there were no differences between the two groups’ submaximal $\dot{V}O_2$. Another point that can be extracted from this study is that the regular physical activity completed by the active participants was of sufficient duration and intensity to elicit significant differences in maximal aerobic power; one
might therefore conclude that physical activity of similar intensity and duration could be sufficient to bring about changes in RE.

The measurement of physical activity, particularly in children, is problematic. At present, there is no single, valid, reliable and universally accepted “gold standard” for the assessment of physical activity either in children or adults (Baransoski et al., 1992). As the same authors point out, the absence of a gold standard makes it difficult to establish the validity and reliability of presumably simpler field measures.

Generally, the methods of assessing physical activity of children do not differ from those used in adults (Baransoski et al., 1992). There are, however, concerns that may limit the feasibility and validity of the use of certain methods within children. As Thompson (2001) points out, two considerations are: 1) that some children may be highly physically active outside of a sport setting, and a measurement technique may overlook this activity, therefore incorrectly assessing the amount of physical activity; 2) that some children and adolescents have difficulty recalling past events, particularly details such as the duration and intensity of previous bouts of physical activity. Given the problems associated with the assessment of physical activity, particularly in children and adolescents, any conclusions regarding the effectiveness of physical activity in bringing about change should be interpreted with caution. In other words, until an assessment technique is demonstrated to be suitably valid and reliable, data on physical activity patterns will be plagued with skepticism.

1.2.5.2. Stride Frequency, Stride Length and Leg Length

One group of proposed factors that could account for inter-individual differences in RE include differences in stride frequency, stride length, and leg length. Unnithan
and Eston (1990) investigated differences in RE between a group of trained university men and a group of healthy prepubertal boys who participated in regular run training. As mentioned previously, the study demonstrated that adults possess greater RE than children. $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) was measured in both groups at four submaximal running speeds. Also, stride frequency was monitored by video analysis. The researchers used the following formula to estimate the energy cost per stride:

$$\dot{V}O_2 \text{ per stride (ml·kg}^{-1} \cdot \text{stride}^{-1}) = \frac{\dot{V}O_2 \text{ (ml·kg}^{-1} \cdot \text{min}^{-1})}{\text{strides·min}^{-1}} \quad (1.3)$$

The results of a comparison revealed that the energy cost per stride was similar between the children ($0.45 \pm 0.04 \text{ ml·kg}^{-1} \cdot \text{stride}^{-1}; \pm 95\% \text{CI}$) and the adults ($0.47 \pm 0.04 \text{ ml·kg}^{-1} \cdot \text{stride}^{-1}$). Also, the video analysis demonstrated that as the treadmill speed increased, the children were forced to increase stride frequency rather than stride length, as a result of their shorter legs. Thus, since the stride frequency was higher in the children (as a result of shorter legs) one could conclude that the adult-child differences in RE could be partially explained by differences in stride frequency and leg length.

Although it would seem to make sense that leg length and stride length during running would be correlated, a study of elite, male, adult distance runners provided evidence that this may not be the case (Brisswalter, Legros & Durand, 1996). Several anthropometric measures, including leg length, were taken on 28 highly competitive adult male runners (national and international 800m) who had similar $\dot{V}O_2\text{max}$ values. Submaximal $\dot{V}O_2$ at treadmill speeds of 9 km·h$^{-1}$ and 15 km·h$^{-1}$ was also measured. The results of the investigation indicated no significant correlation between leg length and step length at either 9 ($r = 0.29; \text{df} = 26$) or 15 km·h$^{-1}$ ($r = 0.32; \text{df} = 26$). However, it should be noted that leg length was significantly negatively correlated with $\dot{V}O_2$ at
15 km·h\(^{-1}\) (r = -0.33; df = 26); in other words, those with longer legs consumed less oxygen at the same speed as those with shorter legs. The results of this particular study should be interpreted cautiously though, since the participants were all highly trained, elite adult runners, and therefore may not be representative of non-elite (or perhaps even elite) children. Additionally, a larger sample might have provided different results, as the significance of a correlation coefficient is influenced by sample size.

Maliszewski and Freedson (1996) examined RE using a slightly different approach. The researchers investigated adult-child differences in RE using a group (n = 25) of young men aged 25.0 ± 4.6 (SD) years and a group of boys aged 9-11 years (n = 21). Participants were required to complete two seven-minute runs. One run was at a predetermined absolute velocity of 9.6 km·h\(^{-1}\), while the other run was a relative speed adjusted for body size. The relative speed was equal to 3.71 leg lengths per second. As expected, the researchers demonstrated that at the same absolute submaximal treadmill speed, the adults possessed superior RE; however, at the relative treadmill speed of 3.71 leg lengths per second, the two groups consumed similar amounts of oxygen relative to body mass. These results support the notion that leg length, stride length and stride frequency are related to submaximal \(\dot{V}O_2\), and may be partly responsible for interindividual differences in RE.

To summarize, the exact nature of the relationship between leg length, stride length, stride frequency and \(\dot{V}O_2\) is not clearly understood. It appears as though there may be some benefit to investigating this relationship further.

1.2.5.3. Substrate Utilization
Bailey and Pate (1991) discussed the possible alterations to RE by altering the type of fuel (carbohydrate or fat) used during submaximal exercise. When carbohydrates are used for energy production 5.03 kcal of energy are produced for every liter of oxygen consumed, whereas only 4.61 kcal of energy are produced (per liter of oxygen) when fat is used (Plowman & Smith, 1996). Therefore, the amount of oxygen required to liberate a given amount of energy is lower when derived from carbohydrates than fats. Accordingly, the respiratory exchange ratio (RER), the ratio of the volume of CO₂ produced divided by the volume of O₂ consumed, is lower when fats are used as the primary fuel source, since more oxygen is required and less carbon dioxide is produced (Plowman & Smith, 1996).

Investigations into adult-child differences in acute responses to exercise have provided evidence that children rely on different fuel sources than adults during submaximal treadmill running (Martinez & Haymes, 1992; Rowland et al., 1987; Maliszewski & Freedson, 1996). This section of the literature review focuses on differences in substrate utilization during physical activity between adults and children.

Martinez and Haymes (1992) examined differences in substrate utilization between prepubertal girls and women during treadmill exercise of moderate intensity. Ten prepubertal girls (9.1 ± 0.6 years of age; mean ± SD) and ten women (24.4 ± 5.2) had oxygen and carbon dioxide consumptions, along with blood samples monitored during two treadmill tests each; one at an intensity corresponding to 70% of peak \( \dot{\text{V}} \text{O}_2 \) and the other at a treadmill speed of 7.2 km·h\(^{-1}\). The girls demonstrated significantly (p<0.05) lower RER values than women during work; while the women demonstrated significantly (p<0.05) greater elevations than girls in lactate concentrations following the treadmill tests. Eriksson, Karlsson and Saltin (1971) demonstrated in a group of 13 year
old males that elevated blood lactate concentration was associated with advanced pubertal development during submaximal cycling. Based upon the RER and lactate concentration differences between the two groups tested, Martinez and Haymes concluded that the younger participants relied more on fat utilization and less on carbohydrate metabolism than women during moderate intensity exercise (1992).

Rowland et al. (1987) investigated the physiologic responses to treadmill running in adults and prepubertal males. At a treadmill speed of 9.6 km·h$^{-1}$, the researchers observed significantly lower RER values in the 20 children (0.99 ± 0.09; SD) as compared to the 20 adults (1.06 ± 0.1) described previously. The authors concluded that children rely more heavily on fat as their primary fuel source during submaximal treadmill running than adults.

Maliszewski and Freedson (1996) demonstrated somewhat similar results to those already mentioned. As described previously, this study required participants to run at two treadmill speeds. One was an absolute speed of 9.6 km·h$^{-1}$, while the other run was a relative speed equal to 3.71 leg lengths per second. The RER values were similar for 21 boys and 25 men at the absolute speed, however they were significantly lower in boys (0.93 ± 0.03; SD) than men (0.98 ± 0.06) at the relative speed, thus indicating a greater reliance on fat in children.

An alternate explanation for adult-child differences in RER during submaximal exercise is the immaturity of the children’s anaerobic lactic system (Rowland et al., 1987). As Rowland et al. point out, less buffering of lactic acid would generate smaller amounts of excess carbon dioxide, thereby decreasing RER values. Macek, Vavra and Novosadova (1976) provided evidence to support this viewpoint as they demonstrated significantly lower lactate concentrations in a group of children than adults during
exercise of varying intensity.

Evidence exists that, in comparison to adults, children rely more heavily on fat during similar submaximal treadmill running (Rowland et al., 1987; Maliszewski & Freedson, 1996). Since the utilization of fat for energy requires a greater volume of oxygen than does the utilization of carbohydrate, one could conclude that children’s greater reliance on fat could partly explain the poorer RE (i.e. if the energy requirements are the same for both groups, the path to liberating that energy may lead to differences in \( \dot{VO}_2 \)). Therefore, the predictive effect of RER on submaximal \( \dot{VO}_2 \) deserves attention when attempting to explain individual differences in RE.

1.2.6. Saskatchewan Growth and Development Study

The Saskatchewan Growth and Development Study (SGDS) began in the summer of 1964. Two hundred and seven seven-year-old boys were tested on a variety of anthropometric, physiological, and performance tests (Mirwald, 1978). The purpose of the study was “to longitudinally evaluate the changes that occur in measures of anthropometry, flexibility, strength, reaction-movement time, fitness performance, and physiological response to exercise, and to study whether or not these changes are related to physical activity, physiological performance, and sociological and cultural factors.” (Mirwald, p. 289, 1978)

Findings from the SGDS have been used to publish over 15 book chapters and over 24 papers in academic journals, not to mention the 37 presentations at national and international conferences. Data collected as part of the SGDS have also been reported in at least 11 Master’s theses, and three Ph.D. Dissertations.
1.3. STATEMENT OF THE PROBLEM

At present there exists a paucity of knowledge regarding the relationship between biological maturation and the development of RE. Therefore, the primary purpose of this research project is to examine, longitudinally, the effects of growth and maturation on RE in a group of male children and adolescents.

The most appropriate method of expressing oxygen consumption has not been agreed upon. Therefore, a second purpose is to examine the appropriateness of using different approaches to scaling oxygen consumption values to account for individual differences in size.

The third, and final, purpose of the study is to assess the independent and combined effects of factors purported to account for some individual differences in running economy, specifically these include: leg length, RER as an indicator of substrate utilization and physical activity.
1.4. HYPOTHESES

1. Oxygen consumption expressed relative to (1.a.) body surface area, (1.b.) an allometric exponent of body mass or (1.c.) fat-free mass will be more appropriate than a simple ratio of $\dot{V}O_2$ and body mass.

2. Running economy, as measured at a treadmill speed of 9.6 km·h$^{-1}$ will not improve with increasing chronological and biological age once an appropriate adjustment for size has been made.

3. The timing of maturation will not have any significant effect on the development of running economy, as measured at a treadmill speed of 9.6 km·h$^{-1}$ at each chronological age.

4. Boys who are more physically active will demonstrate similar running economy to those who are inactive, once an appropriate adjustment for size has been made at each chronological age.

5. Chronological age, an appropriate measure of body size (as determined in Hypothesis 1), age at PHV, leg length, RER and physical activity will all be significant predictors of running economy (L/min) as measured at 9.6 km·h$^{-1}$. 
1.5. ASSUMPTIONS

1. The treadmill test is a valid and reliable method of assessing the participants’ running economy.

2. The methods used to assess physical activity (Annual Physical Activity Questionnaire, Sports Inventory, teachers’ assessments) are valid and reliable.

1.6. LIMITATIONS

1. The longitudinal nature of the SGDS and the fact that continued participation was voluntary made it difficult to maintain sample consistency. Therefore, the participants who remained in the study through its completion no longer represented a stratified random sample; thus possibly limiting the generalizability of the results.

2. The longitudinal design of the SGDS also made it susceptible to habituation of participants as a result of repeated testing. This represents a potential threat to internal validity.

3. The formula used to estimate percent body fat was generated from regression analyses on adults, not children and/or adolescents. Therefore, its use in the present study could potentially have introduced a bias.

1.7. DELIMITATIONS

1. The findings from this study are delimited to males of similar cultural and ethnic backgrounds as the participants.
2.1. STUDY DESIGN

The data analyzed in this study were collected as part of the Saskatchewan Growth and Development Study (SGDS). The data analyzed in the present study were collected using a longitudinal study design. The SGDS as a whole was an investigation of physiological response to exercise relative to growth and development. Data collection, which occurred annually, began in the summer of 1964. The SGDS spanned ten years, until 1973. For complete details on the procedures used in the SGDS, refer to Bailey (1968) or Mirwald (1978).

2.2 PARTICIPANTS

In 1964, 207 seven-year-old boys were recruited to participate in the SGDS. By 1973, the number of participants remaining in the SGDS sample had shrunk to 130. Though tested at age seven, observations taken on the participants at this age were not included in the analysis of the exercise data. This was to allow the participants an opportunity to become familiar with the test protocol.

Participants were randomly selected on a stratified socioeconomic basis from the elementary school system in the city of Saskatoon, Saskatchewan, Canada. The stratification was based upon father’s occupation, as some forms of employment are more stable than others; those with stable, long-term employment in Saskatoon were the most likely to remain in Saskatoon for a long period of time. The stratification was an attempt to limit subject attrition over the course of the longitudinal study.
The SGDS sample consisted of 202 eight-year-old male participants in 1965. For the purposes of the present study a smaller sub-sample, consisting of 63 participants, was defined from the original SGDS sample and was included in the longitudinal analyses at a treadmill speed of 9.6 km·h\(^{-1}\). A separate sub-sample, consisting of 139 participants, was also defined from the SGDS sample and was used in the cross-sectional analyses. For future reference, the sub-sample of 63 participants will be referred to as the longitudinal sub-sample (LSS), while the larger sub-sample of 139 participants will be known as the cross-sectional sub-sample (CSS). A final sub-sample tested, the longitudinal biological age sub-sample (LBASS), will also be described. All remaining participants were excluded from the analyses due to incomplete or missing data that will be identified within the text and tables of Chapter 3.

The LSS used in this study consisted only of participants who had completed the sixth minute of exercise on all nine measurement occasions (age eight to 16 years). Therefore, the total number of measurement occasions for this sub-sample was 567. In addition to having completed the sixth minute of exercise on all nine measurement occasions, participants must have had an accurate assessment of age at PHV to be included in the LSS. The reason that all nine occasions were required to be included in these analyses, is that the longitudinal analyses techniques used (repeated measures ANOVA procedure) do not analyze individuals with missing values. Similarly, the LBASS consisted of all participants who had complete data at all biological ages from -3 years from PHV to two-years post-PHV (n=40).

### 2.3. ANTHROPOMETRY

Annual anthropometric measurements were taken using the protocol of the
International Biological Programme (Weiner & Lortie, 1969). The measurements used in the present study included: chronological age, stature, sitting height, body mass and some skinfolds. Assessment techniques are described in the following sections.

2.3.1. Chronological Age

Chronological age was defined as the elapsed time between birth and measurement occasions; it was recorded to the nearest day and expressed as a decimal age to three decimal places (e.g. 8 years and 124 days = 8.340 years). Chronological age groups were generated by rounding chronological age values to the nearest whole year value. Therefore, anyone aged between 7.50 and 8.49 at a given measurement occasion was included in the eight-year-old age group, and so on.

2.3.2. Stature

During measurement of stature the participant stood, with shoes and socks removed, with his back to a wall-mounted stadiometer. The participants’ feet were together and flat on the floor throughout the test. The heels, buttocks, upper back and head touched the wall. When the rider was in contact with the head, which was in the Frankfort plane, the subject stepped out from under it, taking care not to change its location. The results were read from the bottom of the rider, added to 100cm and recorded to the nearest millimeter (Bailey, 1968).

2.3.3. Sitting Height

During testing for sitting height, participants sat on the bench with hips, back and head against the wall-mounted stadiometer. The head was positioned so the participant
naturally looked straight ahead. The feet were supported so that the upper surfaces of
his thighs were horizontal and the participant sat as tall as possible. To make sure
accurate measurements were taken, it was important to ensure that the hips were against
the wall, the thighs were horizontal, and that the participants’ hair did not add to the
result. The result was read from the bottom of the rider.

2.3.4. Body Mass

During the measurement of body mass, participants were wearing no shoes, and
were attired only in swim trunks. The tester read the results off of the Toledo scale to
the nearest pound, and converted this reading to kilograms by dividing the pound value
by a constant 2.205 (Bailey, 1968). The converted value was recorded in kilograms.

2.3.5. Skinfolds

Skinfold thicknesses were measured annually at six sites (iliac crest, abdominal,
front thigh, rear thigh (females only), chest (males only), triceps and sub-scapular) using
Harpenden skinfold calipers. Only the measurements from the triceps and sub-scapular
sites were required for the present study; therefore, only these two sites are described in
detail. The triceps skinfold was located on the back of the right arm, halfway between
the elbow and the level where the arm contacted the body (acromion process). The sub-
scapular skinfold was located just below and medial to the inferior angle of the right
scapula. The triceps and sub-scapular skinfolds were lifted parallel to the long axis of
the arm and body, respectively. One measurement was taken at each site annually.
2.3.6. Calculations

Some values of interest could not be or were not measured directly. Therefore, certain variables had to be calculated based upon the anthropometric measurements taken. The calculation of these variables is described in the following sections.

2.3.6.1. Leg Length

Leg length was determined by calculating the difference between sitting height from standing height.

2.3.6.2. Body Density

Body density was estimated using the age-, and gender-specific equations generated by Parizkova (1961). Two equations were used for the present study; they were:

**Boys 8-12 years:**

\[
\text{Body density} = -0.027(\log_{10}\text{TRICEPS}) - 0.0388(\log_{10}\text{SUB-SCAPULAR}) + 1.108 \quad (2.1)
\]

**Boys 13-16 years:**

\[
\text{Body density} = -0.055(\log_{10}\text{TRICEPS}) - 0.026(\log_{10}\text{SUB-SCAPULAR}) + 1.130 \quad (2.2)
\]

2.3.6.3. Body Fat

Percentage fat was estimated using a formula established by Brozek, Grande, Anderson and Keys (1963). It should be noted that in the determination of percentage fat, children’s values were used in a formula derived for adults; this constitutes a limitation of the methodologies employed. The formula was as follows:

\[
\text{Percentage fat} = \frac{4.570}{\text{Body Density (from Equations 2.1 & 2.2)}} - 4.14 \quad (2.3)
\]
2.3.6.4. Fat-free Mass

Fat-free mass was estimated using the following formula:

\[
\text{Fat-free mass} = (100 - \text{percentage fat [from Equation 2.3]}) \times \text{body mass} \tag{2.4}
\]

2.3.6.5. Body Surface Area

Although body surface area was calculated as part of the original SGDS using the Dubois and Dubois (1916) formula, body surface area was calculated for each participant in the present study using the Haycock formula (Haycock, Chir, Schwartz & Wisotsky, 1978), which uses height and weight to predict surface area. The formula is as follows:

\[
\text{Body Surface Area} = \text{mass (kg)}^{0.5378} \times \text{height (cm)}^{0.3964} \times 0.024265 \tag{2.5}
\]

The fact that both mass and height are being adjusted exponentially makes this formula at allometric calculation. This formula was selected over the more commonly used Dubois and Dubois formula (1916) due to its validation in infants, children and adults. Furthermore, the Haycock formula reportedly offers the ability to discriminate differences between small participants with low body surface areas (Haycock et al., 1978).

2.4. SEXUAL MATURATION

2.4.1. Peak Height Velocity

In the determination of age at PHV, velocity curves of height versus chronological age were derived for all participants. A velocity curve provides information regarding the rate of change in a given variable over a period of time; in the case of 'height velocity,' the change in height from one measurement occasion to the
next is divided by the age increment between the two measurement occasions to produce a rate of change. The velocity curves were fitted using the Preece-Baines model; this is described in Mirwald and Bailey (1986). Once individual curves were fitted, age at PHV was determined as the precise chronological age at which height was increasing most rapidly within an individual.

2.4.2. Biological Age

Biological age was calculated by constructing a timeline, with age at PHV as the reference. Since age at PHV is the reference value for this timeline, it is given a value of 0. Therefore, exactly one year prior to the occurrence of PHV would be given a value of -1.0 years from PHV and so on. For example, if an individual's age at PHV occurred at 13.04 years, their biological age at 14.45 years would be +1.41 years from PHV.

2.4.3. Maturity Status

Maturity status, defined as early, average or late, was determined from chronological age at PHV as compared to the sample mean age at PHV. The mean age of PHV for male participants in the SGDS was 14.03 ± 1.05 (SD) years. The ‘average’ maturity group was defined as any participant whose age at PHV was within one year of the sample mean. The ‘early’ maturity group consisted of participants whose age at PHV occurred more than one year prior to the sample mean (i.e. earlier than a chronological age of 13.03 years). Finally, the ‘late’ maturity group consisted of participants whose age at PHV occurred more than one year later than the sample mean (i.e. later than 15.03 years). This approach to classifying participants into maturity groups is similar to that used in previous studies (Cunningham et al., 1984).
2.5. AEROBIC POWER

2.5.1. Treadmill Testing Procedures

Each participant completed an incremental maximal treadmill test during the annual examination. The test, known as the Saskatchewan Treadmill Run, involved minute-to-minute gas collection and analysis of data (Figure 2.1). Following five minutes of standing prior to exercise, the speed of the treadmill was increased to 4.8 km·h\(^{-1}\) for 3 minutes. Participants were permitted to hold onto the treadmill rails just before and after speed changes. Following three minutes of walking at 4.8 km·h\(^{-1}\), the treadmill speed was then increased by 4.8 km·h\(^{-1}\) every 3 minutes until a speed of 19.2 km·h\(^{-1}\) was reached. The treadmill remained at this maximum speed until the participant reached exhaustion. The entire test was conducted at a treadmill grade of 0° (horizontal). Once a participant signified that he had reached the point of exhaustion (by ringing a bell) the treadmill was immediately stopped. Following the treadmill test, the participants were required to stand at rest on the treadmill for ten minutes while recovery data were collected (Bailey, 1968).

During the exercise test the participants were required to wear a large rubber mouthpiece that was connected to a two-way breathing valve. The purpose of the valve was to separate inspired and expired air. The valve used in the SGDS had a large inner diameter, and an angled inlet and outlet that allowed for low dead air space (dead space: 120 ml) and a minimum flow turbulence (Bailey, 1968). The volume of air inspired per minute (minute volume) was measured on the intake side of the breathing valve by a calibrated dry gasometer (Parkinson-Cowan CD4). To ensure that all expired air was collected, the nose of the participant was taped and a nose clip was put into place. Temperature, barometric pressure and relative humidity were recorded prior to each
treadmill test and used to standardize the oxygen consumption values for each participant (Bailey, 1968).

Using a four-way directional valve (attached to the three Douglas Bags and originating at the participant) expired air was diverted to one of three Douglas bags in a three-way rotating Douglas Bag system. While a sample of expired air to be analyzed was being drawn through a four-way valve from the first Douglas Bag, the second bag was collecting expired air, while the third Douglas Bag was being evacuated to prepare for the next minute. A pump on the carbon dioxide analyzer pumped air that was about to be analyzed through silica gel in order to dry it, thus preventing corrosion in the oxygen analyzer. A Beckman E2 Oxygen Analyzer measured the oxygen content of the expired air. The carbon dioxide content of the expired air was measured by an infra-red carbon dioxide analyzer (from ages eight through 12 years, a Godart Capnograph; from ages 13 through 16 years, a Beckman Model 215A). Both carbon dioxide analyzers were based upon the same measuring principle of absorption of infra-red rays by carbon dioxide.

The oxygen analyzer was calibrated at the start of each testing day and before the first test of the afternoon. By using a zero gas, 100% nitrogen and therefore no oxygen, technicians could adjust the oxygen analyzer to read zero. Then, by removing the zero gas supply and allowing room air to be pumped through the apparatus, the analyzer could be set to read 83.7, which corresponds exactly to the known concentration of oxygen in normal air. The calibration of the carbon dioxide analyzer followed a similar procedure to that of the oxygen analyzer. This apparatus was calibrated at the same time.
Figure 2.1. Schematic diagram of metabolic circuit
as the oxygen analyzer (Bailey, 1968).

The speed of the treadmill was calibrated before testing. The length of the treadmill belt was measured and the counting of the number of revolutions per minute of the belt at appropriate tachometer readings made possible the calculation of the speed of the treadmill in miles per hour (subsequently converted to kilometers per hour). This is an important consideration, as the treadmill speed of 9.6 km·h⁻¹ was used to draw comparisons across the age span tested.

2.5.2. Peak Oxygen Consumption (peak \( \dot{V}O_2 \))

Peak aerobic power was defined as the highest value of oxygen consumption recorded during the duration of the maximal treadmill test.

2.5.3. Submaximal Oxygen Consumption (submaximal \( \dot{V}O_2 \))

Submaximal oxygen consumption was defined as the value of oxygen consumption recorded for the sixth minute of exercise during the treadmill test. A potential concern with this definition was that some participants might not have demonstrated a steady state of oxygen consumption in the three-minute timeframe at each treadmill speed. Steady state is defined as a condition in which the energy expenditure during exercise is balanced with the energy required to perform that exercise (Plowman & Smith, 1996). Since the body takes time to adjust to increased workloads, achievement of steady state condition is an important consideration to ensure that recorded values are reflective of the actual oxygen (energy) requirements. Jones and Campbell (1982) suggest that steady state has been demonstrated when the oxygen
consumption and heart rates in the final minute of exercise are not greater than ten
percent different from the previous minute. Similar criteria (ignoring heart rate) have
been used in other investigations of energy requirements for treadmill walking and
running (Rogers, Turley, Kujawa, Harper & Wilmore, 1994; Walker, Murray, Jackson,
Morrow & Michaud, 1999). Therefore, steady state is operationally defined as an
oxygen consumption rate in the final minute of exercise (at a given intensity) that is not
greater than ten percent different from the previous minute.

2.6. ASSESSMENT OF PHYSICAL ACTIVITY

Physical activity was assessed in the SGDS through the use of two different
questionnaires (Rasmussen, 1978). Between 1964 and 1970, the Annual Physical
Activity Questionnaire was used, while from 1971-1973, the Sports Inventory was used.
Additionally, elementary and secondary school teachers rated the participants’ physical
activity levels relative to their peers.

The Annual Physical Activity Questionnaire was administered in the
participants’ homes in the presence of their parents using an interview style (Rasmussen,
1978). The scores on the Annual Physical Activity Questionnaire ranged from three to
61, with greater scores indicating more physical activity.

There were essentially two reasons for the change to the Sports Inventory
questionnaire in 1971. The researchers wanted to provide a comprehensive mechanism
to assess sport participation, which, it was felt, the Annual Physical Activity
Questionnaire failed to do effectively. Second, the principal SGDS researchers felt that
the presence of the parents during the interview may have biased the data obtained.
Specifically, the parents had a tendency to over-report their child’s physical activity
(Rasmussen, 1978). Scores for the Sports Inventory questionnaire ranged from zero to an indefinite amount (not definable since the scores were dependent upon the number of sports played) with greater scores indicative of more physical activity.

The decision to change assessment techniques in 1971 does not invalidate data collected from that point onward. For the purposes of the present study, an overall classification of physical activity was generated using information collected from both questionnaires; these questionnaires were scored independently of one another and therefore do not affect the validity of the physical activity classification.

The physical activity assessments provided by the elementary classroom teachers in 1971 required teachers to rate participants’ physical activity levels relative to their peers. Specifically, the teachers were asked:

<table>
<thead>
<tr>
<th>Within the realm of your observation, how well would you rate ________________ as to the amount of physical activity he gets compared to other children of similar age?</th>
</tr>
</thead>
<tbody>
<tr>
<td>The response choices were:</td>
</tr>
<tr>
<td>1. much less active than others</td>
</tr>
<tr>
<td>2. somewhat less active than others</td>
</tr>
<tr>
<td>3. about the same as others</td>
</tr>
<tr>
<td>4. somewhat more active than others</td>
</tr>
<tr>
<td>5. much more active than others</td>
</tr>
</tbody>
</table>

After the completion of the SGDS, secondary school physical education teachers were asked to rate the physical activity of the participants who were in Grade 12 at the time. This assessment used the same question and possible responses as the elementary school teachers. The scoring for these two assessments (elementary and secondary school) was the same; individuals whose rating was either one or two were considered inactive. Those with a teacher rating of three were considered to be of an average level of physical activity, and those who were given a rating of four or five were considered active (Thompson, 2001).
The procedures used to classify SGDS participants into physical activity groups were originally described by Teece (1969) and Rasmussen (1978). The procedures used to classify participants into activity groups in the present study, however, follow the approach described by Thompson (2001). Essentially, each participant was classified into one of the three activity groups (active, average and inactive) following each year’s testing. Combining the annual classifications generated an overall classification for the duration of the study. Each participant was then categorized as being active, average or inactive.

The annual physical activity categorization for participants aged eight to 13 years considered the Annual Physical Activity Questionnaire. From age 14 to 16 participants’ annual physical activity categorization was based upon the Sports Inventory. Additionally, the two teachers’ surveys were included in the overall categorization.

For both participant surveys, from age eight to 16, the physical activity scores for each year that data was obtained were standardized. Quartiles for each year of physical activity scores were established. The quartile cut-off points were determined using a rank order method (distribution free) of individual data rather than the more common distribution dependent method (mean and standard deviation). With the individual data sorted from lowest to highest values, the equivalent standardized scores at the 25th and 75th percentile were determined for each age. Using these scores (from the 25th and 75th values) as the cut-off points, each individual’s physical activity scores from each year was categorized as ‘active’ (upper quartile), ‘average’ (middle 50%) or ‘inactive’ (bottom quartile).

The overall childhood and adolescent physical activity categorization was a little more intricate. The following four components were used:
1. Annual Physical Activity Questionnaire (completed at ages 8-13). An average activity level was determined for each participant, with the predominant activity categorization used to reflect overall activity level during these years.

2. Teachers’ rating of activity level (obtained at age 13 from the participants’ Grade 8 classroom teacher). Those who were rated as “much less active than others” and “somewhat less active than others” were classified as inactive. Those who were rated as “about the same as others” were classified as average. Those who were rated as “much more active than others” and “somewhat more active than others” were rated as active.

3. Sports Inventory (completed at ages 14-16). Similar to the Annual Physical Activity Questionnaire, an average activity level was determined for each participant, with the predominant activity categorization used to reflect overall physical activity level during these years.

4. Teachers rating of activity level (at age 18 – by the secondary school physical education teachers two years after the completion of the SGDS). Those who were rated as “much less active than others” and “somewhat less active than others” were classified as inactive. Those who were rated as “about the same as others” were classified as average. Those who were rated as “much more active than others” and “somewhat more active than others” were rated as active.

To be classified in an activity category reflective of the participants’ overall physical activity level, the participant had to be classified similarly on at least two of the four different assessments (i.e. Annual Physical Activity Questionnaire, Teachers’ rating of physical activity at age 13, the Sports Inventory and the Teachers’ rating of physical activity at age 18).
2.7. ANALYSES

2.7.1. Normalizing Oxygen Consumption

2.7.1.1. Intraindividual Allometric Exponent

Sample-specific intraindividual allometric scaling factors for body mass were
determined using the procedures outlined in Chapter 1 (pg. 16). Body mass, age at PHV
and oxygen consumption rates were used in these calculations. The body mass and the
absolute $\dot{V}O_2$ (L/min) values were log-transformed and individual regression line
between these values were fit (Equation 1.1) for each participant. Therefore, a separate
slope ($\beta$ coefficient) was calculated for each participant. The mean $\beta$ coefficient was
then used as the mass exponent used for the entire sample (Beunen et al., 2002). The
resulting equation for normalizing oxygen consumption followed the formula outlined in
Equation 1.2 (pg. 16).

\[
\log_{10} \dot{V}O_2 = \beta (\log_{10} \text{mass}) + b + \epsilon \tag{1.1}
\]

Where: $\beta =$ slope of regression equation
       $b =$ intercept of regression equation
       $\epsilon =$ error associated with regression equation

2.7.1.2. Appropriateness of Normalizing Technique

To address the first hypothesis, which stated that, “Oxygen consumption
expressed relative to body surface area an allometric exponent of body mass or fat-free
mass will be more appropriate than a simple ratio of $\dot{V}O_2$ and body mass,” two
approaches were undertaken in order to determine the most appropriate method for
expressing oxygen consumption. Firstly, an approach similar to that described by
Tanner (1949) and Winter (1992) was taken. Tanner (1949) demonstrated that if a
physiological variable is divided by an anthropometric variable, its use is only valid when the quotient of the coefficient of variation for the anthropometric variable (CV_{BS}) and the coefficient of variation for the physiological measure (CV_{VO2}) is equal to the Pearson product moment correlation coefficient: \( CV_{BS}/CV_{VO2} \approx r \). Therefore, ratios that failed to satisfy this criterion were deemed inappropriate. This concept is more easily understood when presented graphically; the ratio line and the regression line are plotted on the same axes. For any normalizing approach that is appropriate, the ratio line will virtually lie directly atop the regression line.

The second approach used to assess Hypothesis 1 was to calculate the strength of the relationship, using Pearson’s correlation analysis, between normalized oxygen consumption and the body size measurement used to normalize (e.g. body mass, fat-free mass, etc.). A non-significant correlation indicated that any body size-related bias had been effectively eliminated. However, if Pearson’s r was significant, then the oxygen consumption value was still being influenced by an individual’s body size, and the ratio was deemed inappropriate (Welsman & Armstrong, 2000).

2.7.2. Statistical Analyses

2.7.2.1. Descriptive

To ensure that the sub-samples tested were representative of the entire SGDS sample, means and standard deviations were calculated for height, body mass and submaximal \( \dot{VO}_2 \). Independent sample t-tests were used to draw comparisons between the participants who were included in the analyses and the SGDS participants not included in analyses (not in either sub-sample) at each chronological age for the aforementioned variables. Independent sample t-tests were also used to test for
significant group differences, at each chronological age category, for \( \dot{V}O_2 \) (L/min) between participants who satisfied the steady state criteria and those who had not. The purpose of these tests was to ensure that all data included in subsequent analyses were valid measures of submaximal oxygen uptake.

To begin to describe the relationship between submaximal \( \dot{V}O_2 \) and chronological age, separate Pearson's product-moment correlation coefficients were calculated for chronological age and absolute \( \dot{V}O_2 \), \( \dot{V}O_2 \) normalized to body mass, an allometric exponent of body mass, body surface area and fat-free mass.

2.7.2.2. Hypotheses Testing

Hypothesis 2 stated that, “running economy, as measured at a treadmill speed of 9.6 km-h\(^{-1}\) will not improve with increasing chronological and biological age once an appropriate adjustment for size has been made.” This hypothesis was effectively tested within the longitudinal sub-sample (LSS) and the biological sub-sample (LBASS). One-factor analyses (age) of variance (ANOVA) with repeated measures were used to identify any main effect of either chronological or biological age on the development of RE within these sub-samples. This hypothesis was not tested within the cross-sectional sub-sample (CSS) because the available data were insufficient to allow for the repeated measures ANOVA procedure to be used.

The third hypothesis related to the possibility of an effect of maturity status on RE. This hypothesis was tested within the CSS and LSS, but not the LBASS because the independent variable was maturity status and therefore, testing within the LBASS would essentially be accounting for maturity twice. To test this hypothesis, between
group comparisons of RE at each chronological age were made among the three maturity
groups (early, average and late) using ANOVA and/or ANOVA with repeated measures
procedures, depending upon the sub-sample tested. One-factor (maturity status)
ANOVA procedures were performed at each chronological age group within the CSS to
identify significant differences. A two-factor (3x9) ANOVA with repeated measures
was performed on the LSS. Bonferroni’s post-hoc tests were used to identify specific
between group differences for the ANOVA procedures. These comparisons were made
using only the normalizing approach identified in Hypothesis 1 as the most appropriate.

The tests of significance for Hypothesis 4 were similar to those described for the
third hypothesis; however analyses were completed in all three sub-samples. Between
group comparisons (active, average and inactive) of RE at each chronological and
biological age were made using either one-factor (physical activity) factorial ANOVA or
two-factor (physical activity by age; 3x9 and 3x6) repeated measures ANOVA,
depending upon the sub-sample tested. Again, these comparisons were made using only
the normalizing approach identified in Hypothesis 1 as the most appropriate.

The analyses used to address Hypothesis 5 relied on the use of nine separate
forward stepwise linear regression analyses (one for each chronological age category) to
determine which variables were significant predictors of $\text{VO}_2$ (L/min). The only sub-
sample included in these analyses was the LSS because of the uniformity of participants
at each age and the higher sample size than the LBASS. The forward stepwise
procedure was selected over other approaches primarily because it identifies which
independent variables account for the largest amount of variance in the dependent
variable. The potential predictor variables included in each of the models were
chronological age, age at PHV, body surface area, leg length, respiratory exchange ratio
(RER) and physical activity category, as it has been suggested that each of them may account for inter-individual differences in RE. The level of significance for all tests was set at 0.05.
CHAPTER 3 ~ RESULTS

3.1. PARTICIPANT CHARACTERISTICS

In 1965, 202 boys were recruited into the SGDS. By the end of the study, eight years later, a total of 130 remained. Thirty-six percent of the sample dropped out during the course of the study. Sixty-three individuals (31% of the original sample) were measured annually at all nine measurement occasions (Table 3.1); in 1973 they represented 48% of the total children measured. These 63 individuals represent the longitudinal sub-sample (LSS).

When participants were aligned on biological age categories (years from PHV), only 40 of the 63 participants had sufficient data to be included in the longitudinal analysis aligned by biological age. The biological age range was from –3 to +2 years from PHV. These 40 individuals represent the longitudinal biological age sub-sample (LBASS).

The cross-sectional sub-sample (CSS) consisted of a total of 1110 measurement from 139 participants who were measured on up to nine separate occasions. To be included in the CSS, participants had to have completed the sixth minute of exercise and must have had an accurate assessment of age at PHV, to allow for categorization into one of the three maturity groups. The difference between the criteria for this CSS and the LSS was that the CSS did not require participants to have completed the sixth minute on all nine occasions. Table 3.2 presents a comparison of the number of SGDS participants tested, grouped by chronological age, to the number of participants analyzed in the CSS. All individuals from the LSS were also included in the CSS.
Table 3.1. Number of participants tested at each measurement occasion categorised as those in the longitudinal sub-samples (LSS) and those who were excluded (NLSS)

<table>
<thead>
<tr>
<th>Year of Test</th>
<th>n</th>
<th>Number of subjects not included in the longitudinal sub-sample (NLSS) in each age category</th>
<th>(n)</th>
<th>Number of subjects in longitudinal sub-sample (LSS) in each age category</th>
<th>n</th>
<th>Total number of subjects in each age category</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973</td>
<td>67</td>
<td>(63)</td>
<td>130</td>
<td>48.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1972</td>
<td>77</td>
<td>(63)</td>
<td>140</td>
<td>45.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1971</td>
<td>94</td>
<td>(63)</td>
<td>157</td>
<td>40.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1970</td>
<td>103</td>
<td>(63)</td>
<td>166</td>
<td>38.0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1969</td>
<td>106</td>
<td>(63)</td>
<td>169</td>
<td>37.3%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1968</td>
<td>118</td>
<td>(63)</td>
<td>181</td>
<td>34.8%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1967</td>
<td>127</td>
<td>(63)</td>
<td>190</td>
<td>33.2%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1966</td>
<td>133</td>
<td>(63)</td>
<td>196</td>
<td>32.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1965</td>
<td>139</td>
<td>(63)</td>
<td>202</td>
<td>31.2%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Chronological Age Category (years)

n = Number of subjects not included in the longitudinal sub-sample (NLSS) in each age category
(n) = Number of subjects in longitudinal sub-sample (LSS) in each age category
n = Total number of subjects in each age category
Table 3.2. Number of participants tested at each measurement occasion categorised as those in the cross-sectional sub-samples (CSS) and those who were excluded (NCSS)

<table>
<thead>
<tr>
<th>Year of Test</th>
<th>% CSS of Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td>1973</td>
<td>85.4%</td>
</tr>
<tr>
<td>1972</td>
<td>87.1%</td>
</tr>
<tr>
<td>1971</td>
<td>82.8%</td>
</tr>
<tr>
<td>1970</td>
<td>80.7%</td>
</tr>
<tr>
<td>1969</td>
<td>76.9%</td>
</tr>
<tr>
<td>1968</td>
<td>71.8%</td>
</tr>
<tr>
<td>1967</td>
<td>66.8%</td>
</tr>
<tr>
<td>1966</td>
<td>60.7%</td>
</tr>
<tr>
<td>1965</td>
<td>53.0%</td>
</tr>
</tbody>
</table>

Chronological Age Category (years)

n  Number of subjects not included in the cross-sectional sub-sample (NCSS) at each age group
(n ) Number of subjects in cross-sectional analyses (CSS) for the present study
n  Total number of subjects in each age category
3.1.1. Representativeness of Submaximal $\dot{V}O_2$ and Anthropometric Data

Independent sample t-tests were used to test for significant group differences, at each chronological age category, for $\dot{V}O_2$ (L/min) between participants who satisfied the steady state criteria (modified from Jones & Campbell, 1982) and those who had not. The purpose of these tests was to ensure that all data included in subsequent analyses were valid measures of submaximal oxygen consumption. The t-tests revealed no significant (p>0.05) differences in $\dot{V}O_2$ (L/min) between the two groups at any chronological age category.

To ensure that the participants included in the longitudinal sub-sample (LSS) were representative of the overall SGDS sample, independent sample t-tests were used to test for significant group differences, at each chronological age category, between those in the LSS and those participants not included in the longitudinal analyses (NLSS). Table 3.3 presents the mean values (+ standard deviation) for height, body mass and submaximal $\dot{V}O_2$ at 9.6 km·h$^{-1}$ (L/min) for the LSS and the NLSS. There were no significant (p<0.05) height, body mass or submaximal $\dot{V}O_2$ (L/min) differences, at any chronological age category, between the groups.

Similarly, independent sample t-tests were used to test for group differences between those in cross sectional sub-sample (CSS) and those who were not included in cross-sectional sub-sample (NCSS). The tests assessed differences in height, body mass and $\dot{V}O_2$ at 9.6 km·h$^{-1}$ (L/min) at each chronological age category. The results indicated that there were no significant (p<0.05) height, body mass or $\dot{V}O_2$ (L/min) differences at any chronological age category except body mass at age 14 (p < 0.05), between the groups (Table 3.4).
Table 3.3. Comparison of height, body mass and submaximal \( \dot{V}O_2 \) of participants included in longitudinal sub-sample (LSS) and those excluded (NLSS)

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Height (cm)</th>
<th>Body Mass (kg)</th>
<th>( \dot{V}O_2 ) (L/min) at 9.6 km-h(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>LSS</td>
<td>NLSS</td>
<td>LSS</td>
</tr>
<tr>
<td>8</td>
<td>126.8 ± 5.6</td>
<td>127.89 ± 5.9</td>
<td>25.5 ± 3.7</td>
</tr>
<tr>
<td>9</td>
<td>132.2 ± 5.8</td>
<td>133.54 ± 6.3</td>
<td>28.5 ± 4.2</td>
</tr>
<tr>
<td>10</td>
<td>137.4 ± 6.1</td>
<td>138.96 ± 6.7</td>
<td>31.7 ± 5.0</td>
</tr>
<tr>
<td>11</td>
<td>142.3 ± 6.6</td>
<td>144.05 ± 6.9</td>
<td>34.7 ± 5.8</td>
</tr>
<tr>
<td>12</td>
<td>147.4 ± 7.4</td>
<td>149.70 ± 7.8</td>
<td>38.3 ± 6.8</td>
</tr>
<tr>
<td>13</td>
<td>153.4 ± 8.5</td>
<td>155.95 ± 9.3</td>
<td>42.8 ± 0.2</td>
</tr>
<tr>
<td>14</td>
<td>160.7 ± 9.1</td>
<td>163.28 ± 10.4</td>
<td>49.2 ± 9.7</td>
</tr>
<tr>
<td>15</td>
<td>168.0 ± 8.2</td>
<td>169.98 ± 9.7</td>
<td>55.3 ± 9.8</td>
</tr>
<tr>
<td>16</td>
<td>172.9 ± 7.3</td>
<td>173.94 ± 8.5</td>
<td>61.2 ± 10.1</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation
LSS - longitudinal sub-sample
NLSS- not included in the longitudinal sub-sample
* \( p < 0.05 \) between groups

Table 3.4. Comparison of height, body mass and submaximal \( \dot{V}O_2 \) of participants included in cross-sectional sub-sample (CSS) to those excluded (NCSS)

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Height (cm)</th>
<th>Body Mass (kg)</th>
<th>( \dot{V}O_2 ) (L-min(^{-1})) at 9.6 km-h(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CSS</td>
<td>NCSS</td>
<td>CSS</td>
</tr>
<tr>
<td>8</td>
<td>127.6 ± 5.9</td>
<td>127.99 ± 5.7</td>
<td>25.7 ± 3.6</td>
</tr>
<tr>
<td>9</td>
<td>133.0 ± 6.0</td>
<td>133.00 ± 6.2</td>
<td>28.9 ± 4.4</td>
</tr>
<tr>
<td>10</td>
<td>138.4 ± 6.5</td>
<td>138.39 ± 6.6</td>
<td>32.0 ± 5.3</td>
</tr>
<tr>
<td>11</td>
<td>143.4 ± 7.1</td>
<td>143.20 ± 5.8</td>
<td>35.2 ± 6.2</td>
</tr>
<tr>
<td>12</td>
<td>148.9 ± 8.1</td>
<td>147.97 ± 6.6</td>
<td>39.5 ± 7.7</td>
</tr>
<tr>
<td>13</td>
<td>154.9 ± 9.1</td>
<td>153.68 ± 7.6</td>
<td>44.0 ± 9.1</td>
</tr>
<tr>
<td>14</td>
<td>162.6 ± 10.0</td>
<td>159.08 ± 8.5</td>
<td>50.4 ± 10.6</td>
</tr>
<tr>
<td>15</td>
<td>169.4 ± 9.2</td>
<td>163.93 ± 9.0</td>
<td>56.4 ± 10.5</td>
</tr>
<tr>
<td>16</td>
<td>173.8 ± 7.9</td>
<td>169.33 ± 10.1</td>
<td>61.5 ± 10.5</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation
CSS – cross-sectional sub-sample
NCSS- not included in the cross-sectional sub-sample
* \( p < 0.05 \) between groups
3.1.2. Maturity Groups

The mean age of PHV for participants in the SGDS was calculated to be 14.03 years. Of the 63 boys in the LSS, 12 (19%) were categorized into the early maturity group; 33 (52%) were classified as average maturers; and 18 (29%) as late maturers. Of the 139 boys included in the CSS, 34 (24%), 70 (50%) and 35 (26%) were classified as early-, average- and late-maturing, respectively.

3.1.3. Physical Activity Groups

Of the 63 boys in the LSS 8 (13%) were classified as inactive; 29 (46%) participants were classified as average, while the balance of the participants, 26 (41%), were given a classification of active. Of the 139 boys included in the CSS, 26 (19%), 72 (52%) and 41 (29%) were classified as inactive, average and active, respectively.

3.1.4. Allometric Coefficients

Intra-individual allometric coefficients (k’) for body mass were calculated for each of the participants in the LSS. The linear regression of the logarithmic transformations of body mass (kg) on submaximal $\dot{V}O_2$ (L·min$^{-1}$) calculated for each individual resulted in a mean adjusted determination coefficient ($R^2$) of 0.862 ± 0.103 (standard deviation). The individual allometric coefficients varied greatly across participants, ranging from k’ = 0.415 to k’ = 0.974, with a mean k’ = 0.611 and SD = 0.240.

The linear regression of the logarithmic transformations of body mass on submaximal $\dot{V}O_2$ (L/min) for each individual in the CSS resulted in a mean adjusted
determination coefficient ($R^2$) value of $0.866 \pm 0.103$ (standard deviation). Four participants’ linear regression equations were not significant ($p>0.05$), and were therefore excluded from the weighted mean coefficient. The individual allometric coefficients varied greatly across participants, ranging from $k' = 0.286$ to $k' = 1.063$, with a weighted mean $k' = 0.628$ and SD = 0.243.

Within each sub-sample, three separate allometric mass exponents were calculated; one for each maturity group. However, within each of the three sub-samples, the maturity-specific exponents were not significantly different from one another, as indicated by a one-way ANOVA within each subsample. Therefore, only one (rather than three) exponent was reported and used in subsequent analyses for each sub-sample. Similarly, including age at PHV within the intra-individual allometric regression did not significantly affect the magnitude of the exponents and therefore the simplest model (log transformed mass and $\dot{V}O_2$) was used.

3.1.5. General Data Patterns

The general patterns of submaximal $\dot{V}O_2$ development in the longitudinal sub-sample (LSS), normalized using different techniques are plotted against chronological age (Figures 3.1a-e). Figure 3.1a indicates there was a significant positive correlation ($r = 0.696; p<0.05$) between absolute $\dot{V}O_2$ (L/min) and chronological age. A significant negative correlation ($r = -0.761; p<0.05$) was found between mass-relative submaximal $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) and chronological age (Figure 3.1b). Figure 3.1c plots submaximal $\dot{V}O_2$ normalized to body surface area (ml·m$^{-2}$·min$^{-1}$) and chronological age; again a significant negative relationship was identified ($r = -0.181; p<0.05$). No significant
relationship \((r = 0.012; \ p > 0.05)\) was found between submaximal \(\dot{\text{VO}}_2\) normalized using an allometric expression of body mass \((\text{ml} \cdot \text{kg}^{-0.611} \cdot \text{min}^{-1})\) and chronological age (Fig. 3.1d). Figure 3.1e depicts the significant negative relationship \((r = -0.372; \ p < 0.05)\) between submaximal \(\dot{\text{VO}}_2\) normalized to fat-free mass \((\text{ml} \cdot \text{kg}_{\text{FFM}}^{-1} \cdot \text{min}^{-1})\) and chronological age.
**Figure 3.1a.** Absolute submaximal $\dot{V}O_2$ (L/min) at 9.6 km·h$^{-1}$ versus chronological age in the longitudinal sub-sample (LSS)

\[ r = 0.696 \]
\[ p < 0.05 \]

**Figure 3.1b.** Submaximal $\dot{V}O_2$ (ml·kg$^{-1}$·min$^{-1}$) normalized using body mass at 9.6 km·h$^{-1}$ versus chronological age in the LSS

\[ r = -0.761 \]
\[ p < 0.05 \]
Figure 3.1c. Submaximal $\dot{VO}_2$ (ml·m$^{-2}$·min$^{-1}$) normalized using body surface area at 9.6 km·h$^{-1}$ versus chronological age in the LSS

Figure 3.1d. Submaximal $\dot{VO}_2$ (ml·kg$^{-0.611}$·min$^{-1}$) normalized using an allometric transformation of body mass at 9.6 km·h$^{-1}$ versus chronological age in the LSS
Figure 3.1e. Submaximal $\dot{V}O_2$ (ml·kg\textsubscript{FFM}·min\textsuperscript{-1}) normalized using fat-free mass at 9.6 km·h\textsuperscript{-1} versus chronological age in the LSS.

$r = -0.372$

$p < 0.05$
3.2. TESTS OF HYPOTHESES

3.2.1. Hypothesis 1

Hypothesis 1 proposed that $\dot{V}O_2$ expressed relative to body surface area, an allometric exponent of body mass or fat-free mass would be more appropriate than a simple ratio of $\dot{V}O_2$ and body mass. Figure 3.2a-d graphically illustrates the appropriateness of various methods used to normalize $\dot{V}O_2$ to body size within the LSS using Tanner’s Special Circumstance. Ratio and regression lines were estimated and plotted for $\dot{V}O_2$ (L/min) against (a) mass, (b) body surface area, (c) mass raised to the power 0.611 and (d) fat-free mass. These graphs suggest that per body surface area (BSA) ratio is the most appropriate scaling factor for the LSS (Figure 3.2b; Table 3.5, 3.6).

Table 3.5 presents the descriptive statistics and correlation coefficients and Table 3.6 presents the correlation coefficients of normalized $\dot{V}O_2$. The method that best satisfied Tanner’s Special Circumstance as indicated in Table 3.5 (page 88) and the only method which resulted in a non-significant correlation coefficient of normalized $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) and body size (Table 3.6) within the LSS, was body surface area.
**Figure 3.2a.** Body mass versus absolute \( \dot{V}O_2 \) at 9.6 km\( \cdot \)h\(^{-1}\) for the longitudinal sub-sample (LSS): ratio versus regression plots

**Ratio line:**
\[ \dot{V}O_2 = 0.0399(\text{mass}) \]

**Regression line:**
\[ \dot{V}O_2 = 0.0286(\text{mass}) + 0.533 \]

---

**Figure 3.2b.** Body surface area versus absolute \( \dot{V}O_2 \) at 9.6 km\( \cdot \)h\(^{-1}\) for the LSS: ratio versus regression plots

**Ratio line:**
\[ \dot{V}O_2 = 1.266(\text{BSA}) \]

**Regression line:**
\[ \dot{V}O_2 = 1.293(\text{BSA}) \]
Figure 3.2c. Allometric transformation of body mass versus absolute $\dot{V}O_2$ at 9.6 km·h$^{-1}$ for the LSS: ratio versus regression plots

**Ratio line:**
$\dot{V}O_2 = 0.171(\text{mass}^{0.611})$

**Regression line:**
$\dot{V}O_2 = 0.191(\text{mass}^{0.611}) - 0.192$

Figure 3.2d. Fat-free mass versus absolute $\dot{V}O_2$ at 9.6 km·h$^{-1}$ for the LSS: ratio versus regression plots

**Ratio line:**
$\dot{V}O_2 = 0.0493(\text{FFM})$

**Regression line:**
$\dot{V}O_2 = 0.0256(\text{FFM}) + 0.781$
Table 3.5. Comparison of four approaches to normalizing VO$_2$ at 9.6 km·h$^{-1}$ to body size within the LSS

<table>
<thead>
<tr>
<th></th>
<th>Coefficient of Variation (CV$_{BS}$)</th>
<th>Quotient of Coefficient of Variations (CV$<em>{BS}$ / CV$</em>{VO2}$)</th>
<th>Pearson Product-Moment Correlation Coefficient (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body Mass (kg)</td>
<td>33.7</td>
<td>1.359</td>
<td>0.915</td>
</tr>
<tr>
<td>Body Surface Area (m$^2$)</td>
<td>22.1</td>
<td>0.891</td>
<td>0.910</td>
</tr>
<tr>
<td>Body Mass (kg$^{0.611}$)</td>
<td>20.4</td>
<td>0.823</td>
<td>0.918</td>
</tr>
<tr>
<td>Fat-Free Mass (kg)</td>
<td>33.1</td>
<td>1.335</td>
<td>0.695</td>
</tr>
</tbody>
</table>

Table 3.6. Correlation Coefficients for normalized VO$_2$ at 9.6 km·h$^{-1}$ and body size measurements within the LSS

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pearson Product-Moment Correlation Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>VO$_2$ per kg</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>-0.704*</td>
</tr>
<tr>
<td>Body Surface Area (m$^2$)</td>
<td>0.052</td>
</tr>
<tr>
<td>Body Mass (kg$^{0.611}$)</td>
<td>0.242*</td>
</tr>
<tr>
<td>Fat-Free Mass (kg)</td>
<td>-0.645*</td>
</tr>
</tbody>
</table>

* p > 0.05
Figure 3.3 graphically illustrates the appropriateness of various approaches to normalizing $\dot{V}O_2$ to body size within the CSS. Ratio and regression lines were estimated and plotted for $\dot{V}O_2$ against (a) mass, (b) body surface area, (c) mass raised to the power 0.628 and (d) fat-free mass; again the body surface area adjustment fulfilled the criteria most accurately (Figure 3.3b). Table 3.7 presents the descriptive statistics and correlation coefficients and Table 3.8 presents the correlation coefficients of normalized $\dot{V}O_2$ and the body size measure used. Though Tanner’s Special Circumstance appears to have been satisfied by normalizing using body surface area and allometry in Table 3.7, as shown in Table 3.8, the correlation coefficient for all of the approaches tested was significant.

A comparison of the approaches to normalizing oxygen consumption revealed that only body surface area satisfactorily normalized $\dot{V}O_2$ to body size; this was only demonstrated within the LSS. Table 3.6 identifies body surface area as the only approach to effectively eliminate any size-related bias, as indicated by the non-significant ($p>0.05$) correlation coefficient. All further analyses therefore used $\dot{V}O_2$ normalized to body surface area ($\text{ml} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$).
**Figure 3.3a.** Body mass versus absolute \( \dot{\text{VO}_2} \) at 9.6 km\( \cdot \)h\(^{-1} \) for the cross sectional sub-sample (CSS): ratio versus regression plots

**Ratio line:**
\[
\dot{\text{VO}_2} = 0.0398(\text{mass})
\]

**Regression line:**
\[
\dot{\text{VO}_2} = 0.0279(\text{mass}) + 0.493
\]

**Figure 3.3b.** Body surface area versus absolute \( \dot{\text{VO}_2} \) at 9.6 km\( \cdot \)h\(^{-1} \) for the CSS: ratio versus regression plots

**Ratio line:**
\[
\dot{\text{VO}_2} = 1.268(\text{BSA})
\]

**Regression line:**
\[
\dot{\text{VO}_2} = 1.346(\text{BSA}) - 0.102
\]
Figure 3.3c. Allometric transformation of body mass versus absolute \( \dot{V}O_2 \) at 9.6 km\( \cdot \)h\(^{-1}\) for the CSS: ratio versus regression plots

\[
\text{Ratio line:} \\
\dot{V}O_2 = 0.161(\text{mass}^{0.628})
\]

\[
\text{Regression line:} \\
\dot{V}O_2 = 0.182(\text{mass}^{0.628}) - 0.217
\]

Figure 3.3d. Fat-free mass versus absolute \( \dot{V}O_2 \) at 9.6 km\( \cdot \)h\(^{-1}\) for the CSS: ratio versus regression plots

\[
\text{Ratio line:} \\
\dot{V}O_2 = 0.0493(\text{FFM})
\]

\[
\text{Regression line:} \\
\dot{V}O_2 = 0.0256(\text{FFM}) + 0.781
\]
Table 3.7. Comparison of four approaches to normalizing $\dot{V}\text{O}_2$ at 9.6 km·h$^{-1}$ to body size within the CSS

<table>
<thead>
<tr>
<th></th>
<th>Coefficient of Variation</th>
<th>Quotient of Coefficient of Variations</th>
<th>Pearson Product-Moment Correlation Coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(CV$_{BS}$)</td>
<td>(CV$_{\dot{V}\text{O}_2}$)</td>
<td>(CV$<em>{BS}$ / CV$</em>{\dot{V}\text{O}_2}$)</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>33.6</td>
<td>25.7</td>
<td>1.307</td>
</tr>
<tr>
<td>Body Surface Area (m$^2$)</td>
<td>22.1</td>
<td>25.7</td>
<td>0.860</td>
</tr>
<tr>
<td>Body Mass (kg$^{0.628}$)</td>
<td>20.9</td>
<td>25.7</td>
<td>0.813</td>
</tr>
<tr>
<td>Fat-Free Mass (kg)</td>
<td>33.0</td>
<td>25.7</td>
<td>1.284</td>
</tr>
</tbody>
</table>

Table 3.8. Correlation Coefficients for normalized $\dot{V}\text{O}_2$ at 9.6 km·h$^{-1}$ and body size measurements within the CSS

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pearson Product-Moment Correlation Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$\dot{V}\text{O}_2$ per kg $\dot{V}\text{O}_2$ per m$^2$ $\dot{V}\text{O}_2$ per kg$^{0.628}$ $\dot{V}\text{O}_2$ per kg (FFM)</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>-0.675*</td>
</tr>
<tr>
<td>Body Surface Area (m$^2$)</td>
<td>0.124*</td>
</tr>
<tr>
<td>Body Mass (kg$^{0.628}$)</td>
<td>0.261*</td>
</tr>
<tr>
<td>Fat-Free Mass (kg)</td>
<td>-0.625*</td>
</tr>
</tbody>
</table>

* p > 0.05
3.2.2. Hypothesis 2

Hypothesis 2 predicted that $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ would not decrease with increasing chronological or biological age. Figure 3.4 presents the results of the one-factor (chronological age) ANOVA with repeated measures test on the LSS $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$). A significant ($F_{5.87, 363.7}=7.88; p<0.05$; Table A.1. – Appendix A) decrease in submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) with increasing chronological age was demonstrated. Post hoc pairwise comparisons of the adjusted means identify that $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) values at 16 years of age were significantly different ($p<0.05$; Table A.2. – Appendix A) from all other chronological ages except 15 years of age, and that $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) values at age 15 years were significantly different than at age nine years.

Figure 3.5 presents the results of the one-factor (biological age) ANOVA with repeated measures test on $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) within the longitudinal biological age sub-sample (LBASS). A significant ($F_{4.89, 190.6}=2.81; p<0.05$; Table A.3. – Appendix A) main effect of biological age on submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) was identified. Post hoc pairwise comparisons identified a significant ($p<0.05$; Table A.4. – Appendix A) difference in $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) only between the age at PHV and two years post attainment of PHV.
Figure 3.4. Changes in submaximal \( \dot{\text{VO}}_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) with increasing chronological age at 9.6 km·h\(^{-1}\) within the LSS

Figure 3.5. Changes in submaximal \( \dot{\text{VO}}_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) with increasing biological age at 9.6 km·h\(^{-1}\) within the LSS
3.2.3. Hypothesis 3

The third hypothesis proposed that the timing of maturation would not have any significant effect on the development of RE at any chronological age tested. Using the CSS, one-factor (maturity status) ANOVA tests at each chronological age identified significant differences (p<0.05) among maturity groups’ RE (submaximal \( \dot{V}O_2 \); ml·m\(^{-2}\)·min\(^{-1}\)) at all age categories after nine years of age (Figure 3.6; Table 3.9). Post hoc tests revealed that from 10-16 years of age, late maturing individuals had significantly (p<0.05; Table A.5. – Appendix A) lower \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) values than early maturers.

Secondary analysis was performed using the LSS. A two-factor (maturity status and chronological age) ANOVA with repeated measures revealed a significant interactive effect (F\(_{11.66, 349.9} = 7.26, p<0.05; \) Table A.6. – Appendix A) of maturity status and chronological age. The mean value for each age category for the three maturity groups is shown in Figure 3.7. Post-hoc tests showed that the early maturing group demonstrated significantly higher submaximal \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) than those in the late maturing group between 11-14 years of age (p<0.05; Table A.7. – Appendix A). Average maturing boys were found to be significantly more efficient (p<0.05; Table A.7. – Appendix A) than those in the early maturing group between 12 and 13 years.
Figure 3.6. Comparison of maturity groups’ $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ at each chronological age within the CSS.

Figure 3.7. Longitudinal comparison of maturity groups’ $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each chronological age within the LSS.
Table 3.9. ANOVA table for one-factor ANOVA of maturity group differences in VO₂ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹ for each chronological age

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>F₂, 104 = 0.04</td>
<td>ns</td>
</tr>
<tr>
<td>9</td>
<td>F₂, 116 = 2.71</td>
<td>ns</td>
</tr>
<tr>
<td>10</td>
<td>F₂, 124 = 10.17</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>11</td>
<td>F₂, 127 = 8.89</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>12</td>
<td>F₂, 127 = 13.89</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>13</td>
<td>F₂, 131 = 19.15</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>14</td>
<td>F₂, 127 = 16.08</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>15</td>
<td>F₂, 119 = 6.57</td>
<td>p &lt; 0.05</td>
</tr>
<tr>
<td>16</td>
<td>F₂, 108 = 3.25</td>
<td>p &lt; 0.05</td>
</tr>
</tbody>
</table>

ns = not significant (p > 0.05)
3.2.4. Hypothesis 4

The fourth hypothesis sought to determine whether exposure to physical activity had a significant main effect, or interactive effect with chronological or biological age, on submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$. One-factor (physical activity) ANOVA tests at each chronological age identified no significant differences (p>0.05; Table 3.10) between activity groups’ submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ at any age (Figure 3.8).

In the LSS, results of a two-factor repeated measures ANOVA of physical activity by chronological age (3x9; Figure 3.9) between groups show no significant interaction between submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) and physical activity ($F_{11.9, 357} = 1.01$, p>0.05; Table A.8. – Appendix A). There was, however, a significant chronological age effect ($F_{5.95, 357} = 5.99$, p<0.05; Table A.8. – Appendix A). When aligned on biological age (Figure 3.10) using the LBASS, no significant biological age effects were identified.
Figure 3.8. Comparison of physical activity groups’ VO₂ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹ at each chronological age within the CSS
Table 3.10. ANOVA table for one-way ANOVA of physical activity group differences in $\text{VO}_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each chronological age

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>$F_{2,103} = 0.25$</td>
<td>ns</td>
</tr>
<tr>
<td>9</td>
<td>$F_{2,115} = 0.11$</td>
<td>ns</td>
</tr>
<tr>
<td>10</td>
<td>$F_{2,123} = 0.11$</td>
<td>ns</td>
</tr>
<tr>
<td>11</td>
<td>$F_{2,126} = 1.23$</td>
<td>ns</td>
</tr>
<tr>
<td>12</td>
<td>$F_{2,126} = 0.34$</td>
<td>ns</td>
</tr>
<tr>
<td>13</td>
<td>$F_{2,130} = 0.48$</td>
<td>ns</td>
</tr>
<tr>
<td>14</td>
<td>$F_{2,125} = 0.66$</td>
<td>ns</td>
</tr>
<tr>
<td>15</td>
<td>$F_{2,117} = 0.50$</td>
<td>ns</td>
</tr>
<tr>
<td>16</td>
<td>$F_{2,106} = 1.22$</td>
<td>ns</td>
</tr>
</tbody>
</table>

ns = not significant (p > 0.05)
Figure 3.9. Longitudinal comparison of physical activity groups’ $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each chronological age within the LSS.

Figure 3.10. Longitudinal comparison of physical activity groups’ $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each chronological age within the LBASS.
3.2.5. Hypothesis 5

Hypothesis 5 proposed that a number of independent variables, namely, chronological age, an appropriate measure of body size (body surface area as identified in Hypothesis 1), age at PHV, leg length, respiratory exchange ratio (RER) and physical activity would all be significant predictors of absolute \( \dot{V}O_2 \) (L/min) at 9.6 km·h\(^{-1}\) for each chronological age group tested. Though not listed in the original hypothesis as an independent variable, maturity status was also included on the basis of the results of the third hypothesis. To test the fifth hypothesis, nine separate forward stepwise linear regression analyses (one at each chronological age category) were performed to determine which, if any variables were significant predictors of submaximal \( \dot{V}O_2 \) (L/min). The potential predictor variables included in each of the models were chosen based on the fact that it has been suggested that each of them may account for inter-individual differences in RE. The regression equations that were generated are presented in Table 3.11.

In all nine analyses, body surface area accounted for the greatest amount of variance of submaximal \( \dot{V}O_2 \) (L/min) at 9.6 km·h\(^{-1}\). In seven of the nine equations, RER was also a significant predictor of absolute \( \dot{V}O_2 \) at 9.6 km·h\(^{-1}\). The only other independent variable that had a significant predictive effect on \( \dot{V}O_2 \) (L/min) at 9.6 km·h\(^{-1}\) was age at PHV; this was significant only for the 12- and 14-year-old age groups.
Table 3.11. Chronological age-specific linear regression equations for $\dot{V}O_2$ (L/min)

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Linear Regression Equation [$\dot{V}O_2$ (L/min)]</th>
<th>$R^2$ (previous models)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>1.923 (BSA) – 0.777 (RER)</td>
<td>0.619 (0.585)</td>
</tr>
<tr>
<td>9</td>
<td>-0.377 + 1.677 (BSA)</td>
<td>0.729</td>
</tr>
<tr>
<td>10</td>
<td>-0.716 + 1.938 (BSA)</td>
<td>0.843</td>
</tr>
<tr>
<td>11</td>
<td>1.812 (BSA) – 0.592 (RER)</td>
<td>0.719 (0.688)</td>
</tr>
<tr>
<td>12</td>
<td>1.871 (BSA) – 0.705 (RER) – 0.039 (PHV)</td>
<td>0.838 (0.824; 0.805)</td>
</tr>
<tr>
<td>13</td>
<td>2.087 (BSA) – 0.909 (RER)</td>
<td>0.903 (0.838)</td>
</tr>
<tr>
<td>14</td>
<td>1.818 (BSA) – 0.829 (RER) – 0.040 (PHV)</td>
<td>0.870 (0.863; 0.839)</td>
</tr>
<tr>
<td>15</td>
<td>1.753 (BSA) – 1.077 (RER)</td>
<td>0.852 (0.801)</td>
</tr>
<tr>
<td>16</td>
<td>1.743 (BSA) – 1.116 (RER)</td>
<td>0.732 (0.658)</td>
</tr>
</tbody>
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Where: BSA = body surface area in m$^2$
RER = respiratory exchange ratio (no units)
PHV = age at peak height velocity (years)
CHAPTER 4 ~ DISCUSSION

4.1. DISCUSSION

The primary purpose of this research project was to longitudinally examine the effects of growth and maturation on the development of running economy (RE) in a group of male children and adolescents. Childhood and adolescence represent periods of tremendous growth and, in adolescence, sexual maturation; countless anthropometric and physiological characteristics are changing during these years. Adults have been shown to consistently demonstrate superior RE in comparison to children and adolescents; this project examined some possible explanations for why these differences occur.

RE is a measure of efficiency of locomotion; specifically, RE improves as the rate of oxygen consumption ($\dot{V}O_2$) at a given submaximal running workload decreases (Morgan, 2000). However, before the major hypotheses could be addressed, an appropriate method of controlling for intra- and inter-individual size differences in $\dot{V}O_2$ had to be determined. It was necessary to determine whether age-associated differences in RE persisted once size differences were appropriately controlled for.

The data used to investigate these problems were collected as part of the Saskatchewan Growth and Development Study (SGDS); a longitudinal examination of changes that occur in measures of anthropometry, flexibility, strength, reaction-movement time, fitness performance and physiological response to physical activity (Mirwald, 1978). Some of the SGDS participants’ data were excluded from analyses in the present study for various reasons. Therefore, prior to addressing any of the research
questions, it was first necessary to determine whether the data used were sufficiently representative of the overall SGDS data.

4.1.1. Representativeness of Analyzed Data

An important consideration, which has previously been discussed, was that $\dot{V}O_2$ values should be steady state values. The reason that this is important is that increased workloads (increase of 4.8 km·h$^{-1}$) after the third minute of exercise during a treadmill test are accompanied by an increase in oxygen consumption. A plateau in the $\dot{V}O_2$–time relationship will occur once the amount of oxygen being consumed is equal to the amount of oxygen required to perform that work; this plateau is known as steady state. Therefore, in theory, any value that does not satisfy the steady state criteria (modified from Jones and Campbell, 1982) may not accurately reflect the amount of oxygen required to perform a given workload. Because not all $\dot{V}O_2$ (L/min) values in the SGDS database satisfied the outlined criteria (Chapter 2, page 65), and because submaximal $\dot{V}O_2$ values typically plateau by the third minute of submaximal exercise (Rowland, 1996), those values that satisfied the steady state criteria were compared to those which did not. A series of independent sample t-tests at each chronological age demonstrated that there were no significant differences between $\dot{V}O_2$ (L/min) values that satisfied the modified steady-state criteria and those which did not. Since no differences were detected between $\dot{V}O_2$ (L/min) values that satisfied the steady-state criteria and those which did not, it was concluded that all values could be used in subsequent analyses.
Chronological age-specific comparisons of height, mass and $\dot{V}O_2$ (at 9.6 km·h$^{-1}$) were drawn between the sub-samples identified for inclusion in subsequent analyses and data that were excluded from analyses. Based on the results of independent sample t-tests (at each chronological age), it was concluded that there were no significant (p>0.05) differences at any chronological age in height, body mass or $\dot{V}O_2$ between the longitudinal sub-sample (LSS) and the SGDS participants excluded from analyses (NLSS). It was therefore concluded that the participants’ data that were included in the LSS were sufficiently representative of overall SGDS data.

Similar findings were generated when the cross-sectional sub-sample (CSS) was compared to the excluded data (NCSS). Independent sample t-tests identified no significant differences between height and $\dot{V}O_2$ values between those included in the CSS and the NCSS at any chronological age. There was a significant difference in body mass between those included in the CSS and those not included, however it only occurred at age 14. Because of the similarities between the two groups compared, it was concluded that the CSS was sufficiently representative of overall SGDS data.

4.2. TESTS OF HYPOTHESES

4.2.1. Hypothesis 1: Normalizing Oxygen Consumption ($\dot{V}O_2$) for Body Size

Normalizing oxygen consumption values for body size is essential to allow for meaningful comparisons between individuals or groups (Rogers et al., 1995). This is particularly true of submaximal $\dot{V}O_2$ because absolute values (L/min) increase with increasing age during childhood and adolescence. However, depending upon the method used to normalize $\dot{V}O_2$ values, relative values typically decrease with increasing
age. This pattern produces a paradox when trying to interpret submaximal \( \dot{V}O_2 \) values that are not adjusted for body size. The most appropriate method of expressing (submaximal) \( \dot{V}O_2 \), however, has not yet been agreed upon. A review of relevant literature quickly reveals this fact and suggests that the best reason for using some approaches is convenience, rather than statistical appropriateness. The biological and statistical rationale for normalizing \( \dot{V}O_2 \) values using allometric exponents of body mass, fat-free mass or normalizing to body surface area led to the hypothesis that all three of these techniques would prove to be more appropriate than normalizing to body mass.

Using the criteria described by Tanner (1949) it appeared as though using body surface area was a satisfactory approach to normalizing \( \dot{V}O_2 \) values within the CSS. However, a small but significant \((r = -0.124)\) correlation coefficient was identified for the relationship between \( \dot{V}O_2 \) \((\text{ml} \cdot \text{m}^{-2} \cdot \text{min}^{-1})\) and body surface area \((\text{m}^2)\) in the CSS; thus indicating that the effects of body size had not totally been removed from the \( \dot{V}O_2 \) measure. Though the coefficient was significant, it was relatively small, indicating that any size-related bias would be small as well. Within the LSS, normalizing \( \dot{V}O_2 \) values to body size using body surface area was found to be statistically more appropriate than normalizing to body mass, as this approach satisfied both sets of outlined criteria. This finding is not only consistent with the stated hypothesis, but also with previous findings (Rogers et al., 1995b; Rowland et al., 1987; Rowland & Green, 1988). Of note is the fact that normalizing using body surface area is the only technique of those tested that effectively eliminated any size-related bias according to the objective criteria described by Welsman and Armstrong (2000); this was only the case within the LSS. Also, no
known study has demonstrated that normalizing submaximal $\dot{V}O_2$ values using body surface area is inappropriate. It was therefore concluded that it is appropriate to use body surface area as the denominator when normalizing $\dot{V}O_2$ values to body size.

The biological rationale for normalizing $\dot{V}O_2$ values to body size using body surface area is that both oxygen consumption and heat production per unit weight (ml·kg$^{-1}$·min$^{-1}$ and ºC·kg$^{-1}$ respectively) are greater in small animals compared to large ones at rest (Von Bertalanffy, 1957). However, when these resting metabolism rates are expressed relative to body surface area the differences disappear. Rowland (1990) suggests that it is not unreasonable to expect that this relationship may persist during exercise. When performing physical work in the form of running, an organism is required to move body mass rather than surface area; therefore, animals with the greatest surface area to body mass ratio (i.e. the smaller animals) are at a metabolic disadvantage during exercise only if $\dot{V}O_2$ values are normalized using body mass. A problem with normalizing $\dot{V}O_2$ values using body surface area is that the effects of mass, which is what must be moved during treadmill exercise, have not been totally eliminated. This may help explain why the early maturing participants would have demonstrated greater oxygen demands than average or late maturing participants when compared at common chronological ages.

Despite the significant correlation between $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) and body surface area (m$^2$) in the CSS, it was decided that further analyses should be carried out using these data in order to maintain a logical progression from descriptive statistics, to basic inferential statistical analyses (one-way ANOVA), to true longitudinal analyses.
(repeated measures ANOVA). All further results generated in the CSS were confirmed within the LSS before conclusions were drawn.

None of the other three methods tested, per body mass ratios, per an allometric exponent of body mass or per fat-free mass, were shown to satisfactorily normalize \( \dot{V}O_2 \) measures to account for differences in body size. Similar to previous reports dating as far back as 1949 (Tanner), a ratio of \( \dot{V}O_2 \) to body mass was found to be inappropriate regardless of the criteria used for evaluation. These findings essentially confirm what has been documented for the last 50 years (Bergh et al., 1991; Katch, 1972; Katch, 1973; Katch & Katch, 1974; Rogers et al., 1995a; Rogers et al., 1995b; Sjödin & Svedenhag, 1992); which is that changes in \( \dot{V}O_2 \) are not in direct linear proportion to changes in body mass. The present findings, however, contradict the conclusions drawn by Šprynarová et al. (1987), which stated that \( \dot{V}O_2 \) (L/min) values correlated most strongly with body mass as opposed to fat-free mass and/or height. Volpe Ayub and Bar-Or (2003) also produced evidence to suggest that total body mass is what affects the energy cost of locomotion. Though the results offered by Šprynarová et al. cannot be discounted, these researchers failed to use a statistically sound approach to draw their comparisons; in other words, correlation analyses are not sufficient to deem one method of normalizing more or less appropriate than another method.

Volpe Ayub and Bar-Or assessed \( \dot{V}O_2 \) in two groups of boys aged 11-18 years who were matched for body mass, however, differed in adiposity. They demonstrated that the variance in \( \dot{V}O_2 \) (l/min) explained by total body fat ranged between 2.1% and 16% depending upon the treadmill speed. Volpe Ayub and Bar-Or concluded that the energy cost of locomotion in boys aged 11-18 is affected most by body mass (2003).
Although the researchers took a novel approach to examining the influence of size on \( \dot{\text{VO}}_2 \), they too failed to use a recognized approach to validating the use of body mass as an appropriate adjustment for \( \dot{\text{VO}}_2 \).

Based upon the results presented in Chapter 3, it was concluded that it is inappropriate to normalize submaximal \( \dot{\text{VO}}_2 \) values using a body mass ratio. The practice of normalizing \( \dot{\text{VO}}_2 \) values using body mass, however, will undoubtedly continue because of the fact that body mass is what must be moved during treadmill work and the relative ease of the procedure combined with the added convenience of being able to compare results to other published works; this practice can only encourage the production of misleading results and conclusions and should be avoided.

Contrary to the evidence presented by Janz et al. (1998), in both sub-samples tested, normalizing \( \dot{\text{VO}}_2 \) values using fat-free mass failed to effectively eliminate any influence of body size. Regardless of the criteria used to determine the appropriateness of this approach, normalizing \( \dot{\text{VO}}_2 \) values using fat-free mass proved to be ineffective at eliminating any size related bias. A difference between the Janz et al. paper and the present study, however, is that the former used an allometric transformation of fat-free mass, while the present paper simply divided \( \dot{\text{VO}}_2 \) values by unadjusted fat-free mass values. The decision to use unadjusted fat-free mass values was based on two factors; first, Winter and Hamley (1976) demonstrated a significant linear relationship \((p<0.01)\) between submaximal \( \dot{\text{VO}}_2 \) and fat-free mass in a sample of 32 male middle- and long-distance runners, and second, adjusting estimates such as fat-free mass (which was estimated not measured) is statistically undesirable. The biological rationale for normalizing \( \dot{\text{VO}}_2 \) values using fat-free mass as the denominator is that fat is a relatively
anaerobic tissue, and therefore its elimination (from total body mass) would more accurately reflect oxygen requirements of more aerobic tissues during work. It appears, however, as though the argument (Morgan, 2000) which states that since it is the entire body’s mass (i.e. fat-mass included) that is being transported during locomotion, oxygen consumption values would reflect this ‘extra work,’ may hold some truth. Whatever the case, normalizing using fat-free mass did not satisfy the described criteria, and therefore further analyses was not completed using this approach to normalizing $\dot{V}O_2$ values.

Somewhat surprisingly, intra-individual allometric coefficients of body mass did not satisfactorily normalize $\dot{V}O_2$ values to body size. This finding opposes many previous findings (Bergh et al., 1991; Rogers et al., 1995a; Rogers et al., 1995b; Sjödin & Svedenhag, 1992). As mentioned, sample-specific allometric exponents were calculated for each of the sub-samples. The mass exponents calculated for the two sub-samples were $0.611 \pm 0.059$ (95% confidence interval) and $0.628 \pm 0.040$ (95% confidence interval) for the LSS and CSS, respectively. Neither of the calculated exponents included the theoretical exponent 0.75 within their respective 95% confidence intervals; however, the more popular of theoretical body mass exponents, 0.67 (Rogers et al., 1995b), was included within both of their respective 95% confidence intervals’. The sample-specific mass exponents were therefore considered to be reliable estimates.

The biological rationale for allometric scaling is similar to that described for fat-free mass in that the goal is to eliminate a bias of relatively anaerobic mass. Though this biological theory may remain valid, the statistical techniques used to arrive at the final mass exponents (0.611 and 0.628) may be the downfall. In other words, a possible drawback associated with using intra-individual allometric exponents to normalize
longitudinal submaximal \( \dot{VO}_2 \) data is that the mean value may not necessarily be representative of all individuals at all ages; although the mean exponent (k’) used for the LSS was 0.611, the range of individual values was 0.415-0.974. An even wider range (0.286-1.063) was calculated for the CSS. The wide ranges in individual allometric coefficients highlight the importance of biological variability and ultimately lead to the conclusion that more research needs to be done in this area. Whereas a single mass exponent was used for all participants within given sub-samples at all ages, this was not the case with body surface area. The fact that body surface area was age-specific and reflective of individual biological variations is likely the most important factor in its appropriateness and the inappropriateness of allometric mass exponents in normalizing \( \dot{VO}_2 \) values. It may be that one single exponent does not exist that can satisfactorily eliminate any size-bias at all ages during childhood and adolescence. In any case, the mass exponents calculated, 0.611 and 0.628, failed to satisfy the defined criteria for appropriateness when used to normalize \( \dot{VO}_2 \) values.

The finding that division of participants into three maturity groups did not result in significantly different allometric exponents is in opposition to the conclusions drawn by Beunen et al. (2002). Beunen et al. provided evidence that maturity status affects the magnitude of allometric coefficients; early maturers may have different coefficients than average or late maturers, for example. This may, however simply be a result of small groups; once divided into the three maturity groups, the sample sizes would have been 12, 33 and 18 participants, respectively. These relatively small groups would lead to large standard error (of the mean) statistics and therefore increase the likelihood of overlap.
The more subjective Tanner’s Special Circumstance did not immediately identify intraindividual allometry as being inappropriate. As a matter of fact, judging solely by Figure 3.2.c and Figure 3.3.c, it would appear that the ratio and regression lines, which are plotted on the same axes, virtually overlap. However, the more objective criteria (correlation coefficients) demonstrated the inappropriateness of this technique in both sub-samples tested.

The results of the present study contradict previous work that identified allometry as an appropriate technique to normalize submaximal \( \dot{VO}_2 \) values to body size (Rogers et al., 1995b; Welsman & Armstrong, 2000). One possible explanation for why intraindividual allometry failed to satisfy the described criteria is the statistically inefficient method used to generate the exponents 0.611 and 0.628. The two-step process, linear regression analyses and calculation of group mean, is cumbersome and could constitute a drawback to this method. The results of the present study concur with previous research (Rogers et al., 1995b) that normalizing \( \dot{VO}_2 \) values using body surface area is the most appropriate method of those tested to use during adolescents; the present study is the first to draw this conclusion.

### 4.2.2. Hypothesis 2: Changes in Oxygen Consumption (\( \dot{VO}_2 \)) with Increased Age

It was hypothesized that once \( \dot{VO}_2 \) values were appropriately normalized, any previously reported age-associated differences would disappear; in other words, RE, as measured at a treadmill speed of 9.6 km·h\(^{-1}\), would not improve with increasing chronological and/or biological age once an appropriate adjustment for size had been made. The results generally did not support these hypotheses. Significant \( \dot{VO}_2 \)
(ml·m⁻²·min⁻¹) differences were identified within the LSS across chronological ages using a one-factor (chronological age) ANOVA with repeated measures. Post-hoc results highlighted that this difference was between age 16 years and all other ages except age 15 years, and between 15 years and nine years (Fig. 3.4, Table A.1.; A.2. – Appendix A). Because the ANOVA test identified a significant main effect of chronological age on \( \dot{V}O_2 \), it is difficult to conclude that RE does not improve with increasing chronological age. Similarly, within the biological sub-sample, a significant main effect of age on \( \dot{V}O_2 \) (ml·m⁻²·min⁻¹) was identified (see Fig. 3.5, Table A.3.; A.4. – Appendix A); however the post-hoc test showed a significant difference only between PHV and two years post-PHV. This indicates that there were no RE differences among other biological age groups; the exact relationship between RE and maturity therefore is still unclear. Although RE improved after PHV, no differences were found between pre- or post-PHV groups (Figure 3.5). The interpretation is further complicated because it was also found that early, rather than late maturers, were less economical (Figure 3.7), a finding that would seem counter-intuitive. The latter finding may be due in part to small sample size, measurement error or sampling, among other possibilities, all of which could have caused a type I error leading to the rejection of a null hypothesis that was indeed true. Further work in this area is therefore warranted.

Much of what is thought to be known about the relationship between RE and age is based on studies that normalized \( \dot{V}O_2 \) values using body mass ratios. Since body mass was shown to be an inappropriate denominator, these previous conclusions must carefully reviewed. Previous cross-sectional studies have demonstrated that once \( \dot{V}O_2 \) values were normalized using body surface area, no significant differences exist between
adults and children (Rowland et al., 1987; Rowland & Green, 1988). Both of these papers identified \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) differences between a group of adults and a group of children; in both cases, when the \( \dot{V}O_2 \) values were expressed relative to body surface area (ml·m\(^{-2}\)·min\(^{-1}\)) the group differences disappeared. However, as indicated, both of these studies were cross-sectional in design and compared adults to children. Therefore, the present study is among the first studies that clearly demonstrates that (chronological) age-associated differences in submaximal \( \dot{V}O_2 \) values persist even when normalized using body surface area (ml·m\(^{-2}\)·min\(^{-1}\)), albeit only at ages 15 and 16 years. This finding, therefore, opens the door to exploration of factors other than size that might influence age associated changes to RE. The focus of the three remaining hypotheses is to examine why these differences might occur.

The results of the post-hoc tests subsequent to one-factor repeated measures ANOVA tests in the LSS demonstrate that decreases in appropriately normalized \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) occur only at the highest ages tested (15 and 16 years). These findings are indicative that the timing of these changes may be related to maturity. A better understanding of why age-associated RE differences occur only at higher ages is obtained following sections 4.2.3.-4.2.5; a discussion of possible physiological and functional mechanisms responsible for the timing of these age-associated changes to RE is therefore included at the end of section 4.2.5.

The post-hoc tests performed within the LBASS identified a significant difference in appropriately normalized \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) only between the age of PHV and two-years post-PHV. A possible explanation for this is the increased muscle mass associated with maturity, coupled with an interaction between motor skill
development (co-contraction of agonist and antagonist muscles in thigh and leg) and age discussed by Frost et al. (2002). The increased muscle mass near the time of PHV would lead to increases in $\dot{V}O_2$; however, this increased oxygen demand would likely diminish as motor skills developed. This could explain why the only significant biological-age associated difference in submaximal $\dot{V}O_2$ ($\text{ml} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$) occurred between PHV and two-years post-PHV.

4.2.3. Hypothesis 3: Timing of Maturation

As young people grow, they also mature, but few studies have investigated the influence of maturity, independent of body size, on submaximal $\dot{V}O_2$; particularly values normalized using body surface area. Those which have investigated the role of maturity status on changes to RE have provided conflicting results (Armstrong et al., 1999; Bouchard et al., 1976; Bouchard et al., 1978; Cunningham et al., 1984; Welsman & Armstrong, 2000); however, consistent with the majority of previous findings, it was hypothesized that the timing of maturation would not have any significant effect on the development of RE, as measured at a treadmill speed of $9.6 \text{ km} \cdot \text{h}^{-1}$ at each chronological age. Once again, however, the findings from the present study are in opposition to most previous results. The analyses presented in Chapter 3 indicate that maturity status significantly affects RE at certain ages; however, these findings must be explained against the backdrop of sound reasoning and physiological rationale.

A series of one-factor (maturity status) ANOVA tests were performed to test for $\dot{V}O_2$ ($\text{ml} \cdot \text{m}^{-2} \cdot \text{min}^{-1}$) differences among the three maturity groups within the CSS at each chronological age. These tests revealed that early maturing boys required more oxygen
per unit body surface area than late maturing boys at all ages from 10 years to 16 years; in terms of RE, late maturing boys appear to be more economical than early maturing boys from ages 10-16 years. Also of note is the fact that early maturing boys were shown to have greater oxygen requirements than average maturing boys at chronological ages of 12, 13, 14 and 15 years. Similar results were produced when the data were analysed using the two-factor (maturity status and chronological age; 3x9) repeated measures ANOVA procedure within the LSS. Pairwise comparisons of adjusted means showed that early maturing boys demonstrated higher $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) values than late maturers at all chronological age groups between 11-14 years; the results indicated that late maturers are more economical than early maturers from ages 11-14 years. Again, average maturing boys’ $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) values were lower (greater economy) than early maturers’ at 12 and 13 years of age.

These results provide some evidence that the timing of maturation is significantly related to the development of RE, with the general pattern being that average and late maturing boys demonstrate superior RE values than early maturing boys during the circumpubertal years. A problem associated with this conclusion is that, when considering the results of the second hypothesis, it is counter-intuitive to conclude that early-maturing individuals could demonstrate poorer economy than average- and late-maturing individuals. As previously stated, these findings may be due in part to small sample size, measurement error or sampling, all of which could have caused a type I error (inappropriately rejecting a null hypothesis that is true). Therefore, the findings regarding the effects of the timing of maturation on RE development in boys 8-16 years are relatively equivocal.
The results of statistical tests in the present study support the conclusions drawn by Bouchard et al. (1978) which demonstrated that the impact of maturity was most evident during the circumpubertal years. This magnified impact during the circumpubertal years is perhaps not a surprise, since participants were being compared at common chronological ages. Prior to ten years of age, virtually none of the participants would have begun the pubertal growth spurt; however, following this, the three maturity groups would all pass through this stage of maturation at different chronological ages. Following the circumpubertal period, all participants would be of more similar maturity, therefore muting any maturity status effect. This conclusion can be used to help explain why age-associated differences in $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) did not occur until ages 15 and 16 years and two years post-PHV; the timing of maturation affects the development of RE in growing males. Most importantly, however, is to understand that maturation simply describes the timing and tempo of changes and that physiological and/or structural changes, other than increases in size, are occurring during the circumpubertal years that significantly affect RE. A discussion of possible mechanisms of change follows shortly, and will include: the unstable body mass to body surface area ratio during this period; alterations to the oxygen transport system (e.g. increased hemoglobin concentration); and changes to preferred substrate utilization among others.

The conclusions drawn in the present study oppose the findings of Cunningham et al. (1984) who concluded that late-maturing boys demonstrated poorer RE than early or average maturing boys. As mentioned, however, a major drawback to the findings presented by Cunningham et al. was that their results were based on absolute $\dot{V}O_2$ values (L/min), rather than relative (to body size) values. A difference between the
present study and the Cunningham et al. study was that the 1984 study compared maturity groups at similar biological ages whereas the present study compared maturity groups at common chronological ages. This major difference in approaches makes direct comparison of the two studies very difficult.

Because the participants used in the present study were all males, and because the effects of maturity status were most pronounced during the circumpubertal years, it is difficult to compare these results to those produced by Armstrong et al. (1999) and Welsman and Armstrong (2000). The participants used in these studies ranged in age from 11-13 years; therefore, it may be that the participants were not sufficiently mature to experience the impact of maturity demonstrated in the present study. Had these two papers used participants who where in the midst of puberty, the results might have mirrored those presented here.

Several factors could potentially explain the discrepancy in economy demonstrated amongst the three maturity groups within the present study. One such factor is the unstable relationship between body surface area and body mass during puberty. This unstable relationship is the product of these two variables changing at different tempos. According to Von Bertalanffy (1957) those with the greatest body surface area to body mass ratios (the smaller animals) are at a metabolic disadvantage. Logic dictates that individuals who are relatively late maturing will be smaller at a younger, but common, chronological age than those who are early maturing. Since puberty is a period of tremendous growth and maturation, it seems reasonable to suppose that maturity status may play a role in the relationship between body surface area and body mass. Future research should examine the effects of maturity status on the body
surface area to body mass ratio to determine if this may be influencing submaximal
\(\dot{VO}_2\) (ml·m\(^{-2}\)·min\(^{-1}\)) values during adolescence.

The post hoc pairwise comparisons of \(\dot{VO}_2\) (ml·m\(^{-2}\)·min\(^{-1}\)) and biological age
within the LBASS described in Table A.4. (Appendix A) identify a significant (p<0.05)
difference in RE between the age at PHV and two years post attainment of PHV; since
changes in body size have already been accounted for appropriately, one can only
conclude that structural and/or functional changes are occurring which cause these
differences. Therefore, it is also reasonable to conclude that these same structural and/or
functional changes are partly responsible for the maturity group differences which have
been demonstrated. Examples of structural and functional changes that occur during this
period include alterations to the oxygen transport system (e.g. increased haemoglobin
concentration) and muscle hypertrophy.

The main focus of the present study is to identify factors that help explain age-
associated differences in RE. After body size was appropriately controlled for, maturity
status was shown to significantly affect the development of RE at certain ages;
specifically, the effects of maturity status seem to be more pronounced during the
circumpubertal years.

4.2.4. Hypothesis 4: Effects of physical activity level on RE

The possibility that engaging in run training may lead to improved RE has been
discussed. Several studies have demonstrated a relationship between long-term
exposure to training and RE (Conley et al., 1981; Conley et al., 1984; Daniels et al.,
1978b; Patton & Vogel, 1977; Svedenhag & Sjödin, 1985). Since training is a specific
form of physical activity, it was hypothesized that increased physical activity would be associated with superior RE in the children and adolescents tested. Therefore, the fact that there was no evidence of any effect of physical activity level on the development of RE in the sub-samples tested is surprising. Tested in the cross-sectional (CSS), longitudinal (LSS) and biological sub-samples (LBASS), there were no independent or interactive (with chronological/biological age when using repeated measures procedure) effect of physical activity exposure on \( \text{VO}_2 \) (ml\( \cdot \text{m}^{-2} \cdot \text{min}^{-1} \)) values at 9.6 km\( \cdot \text{h}^{-1} \). Interestingly, the main effect of biological age that was demonstrated within the entire LBASS (Fig. 3.5; Table A.3. – Appendix A) disappeared when the sample was divided into three physical activity groups. This was likely the result of the distribution of participants among the three groups; the lower sample size within each of the three activity groups would create greater standard error of the mean statistics, and therefore a greater likelihood of overlap.

Several explanations exist which could possibly contribute to why physical activity exposure was not found to affect RE. The most obvious, of course, is that there is actually no relationship between the volume of physical activity completed and the development of RE in growing males. As mentioned, some studies have shown that short-term exposure to run training has had no significant impact on RE in male and female adults (Daniels et al., 1978b; Petray & Krahenbuhl, 1985; Unnithan et al., 1996; Wilcox & Bulbulian, 1984); one could therefore conclude that these results confirm what has been demonstrated in the past. However, since others have shown that training has been linked with improved RE in adults, it seems as though another explanation might be required.
As mentioned in Chapter 1, training is a specialized form of physical activity, normally associated with specific duration, intensity and type. The physical activity completed by the participants in this study may have lacked a sufficient amount of any one of the three of these characteristics (duration, intensity and type), thus making it insufficient to induce change. In other words, there may be a minimum threshold that must be achieved before the effects of physical activity become apparent. For example, in the cited studies, those which were of longer duration generally yielded conclusions that training is associated with improved RE (Conley et al., 1981; Conley et al., 1984; Daniels et al., 1978b; Patton & Vogel, 1977; Svedenhag & Sjödin, 1985). It has been suggested in the past that simple “physical activity,” rather than “training,” may lack the intensity required to bring about changes to physiological variables such as peak and/or submaximal \( \dot{V}O_2 \) (ml·m\(^2\)·min\(^{-1}\)).

Another possible explanation of why no significant effect of physical activity was detected is that the approach used to collect data and classify participants into physical activity groups may not have been sensitive enough to accurately separate the most from the least active participants. For example, rather than using quartiles as the cut-points for each of the three groups, it might have been more appropriate to use deciles (include those below the tenth percentile in the inactive group, and those above the ninetieth percentile in the active group). This would have the desired effect of producing more extreme groups.

Very few of the studies that investigated a relationship between training and improved RE use children or adolescents as participants. Those which did include children or adolescents as participants either suffered from some serious methodological
flaws (Daniels et al., 1978a) or demonstrated no effect of training on RE (Petray & Krahenbuhl, 1985). Therefore, another possible explanation of why no significant effect of physical activity was identified is that perhaps adults are more sensitive to the stimulus than children; in other words, it could be that increased levels of physical activity may affect RE in adults, but not in youth. The pattern of a “sensitive period” was demonstrated with RE differences between maturity groups (i.e. the effects of maturity were more pronounced during the circumpubertal years); therefore the effects of physical activity on RE may be worth investigating at ages other than those tested in the present study.

Future research in this area is not likely to advance the understanding of the role of physical activity in the development of RE until a valid, reliable “gold standard” for assessing physical activity is agreed upon. Until such time, it will remain difficult to have confidence in the conclusions drawn regarding the relationship between RE and physical activity exposure.

4.2.5. Hypothesis 5: Effects of other factors on RE

Previous reports have identified a number of factors, other than size, that may partially explain the age associated changes in submaximal $\dot{V}O_2$ (L/min). Such factors include, but are not limited to, greater resting energy expenditure, differences in gait pattern, stride length and frequency, leg length, segmental mass distribution, ventilation, substrate utilization, training, age and gender. Once the independent effects of maturity status and physical activity were known, it remained important to identify other variables that may help explain intra- and inter-individual differences in RE. Therefore,
those variables for which data were available within the SGDS database were included in age-specific multiple-linear regression equations to determine their predictive effect on the development of RE. It was therefore hypothesized that chronological age, an appropriate measure of body size (body surface area as determined in Hypothesis 1), leg length, RER and physical activity would all be significant predictors of RE (L/min) as measured at 9.6 km·h\(^{-1}\).

The most obvious trend presented in Table 3.20 was that body surface area, a measure of body size, was the most significant predictor of submaximal \(\dot{V}O_2\) (L/min) in each of the nine age groups tested. This result does not come as a major surprise, since \(\dot{V}O_2\) (L/min) is known to be a size dependent variable. This finding simply reiterates the importance of normalizing absolute \(\dot{V}O_2\) values to reflect body size. This topic has been discussed at length and, therefore, will not be revisited.

Another trend was the significant contribution of the respiratory exchange ratio (RER), the ratio of the volume of CO\(_2\) produced divided by the volume of O\(_2\) consumed, within most of the age groups tested. At ages eight and 11-16 years, RER was a significant predictor of submaximal \(\dot{V}O_2\) (L/min). Previous studies have demonstrated that children display significantly lower RER values than adults when working at common workloads (Rowland et al., 1987; Maliszewski & Freedson, 1996). The importance of RER is likely tied to substrate utilization (Rowland et al., 1987; Maliszewski & Freedson, 1996). As discussed previously, when carbohydrates are used for energy production 5.03 kcal of energy are produced for every liter of oxygen consumed, whereas only 4.61 kcal of energy are produced (per liter of oxygen) when fat is used (Plowman & Smith, 1996). Therefore, the amount of oxygen required to liberate
a given amount of energy is lower when derived from carbohydrates than fats. Accordingly, RER is lower when fats are used as the primary fuel source, since more oxygen is required and less carbon dioxide is produced (Plowman & Smith, 1996).

The findings of the present study are among the first to identify RER as a significant predictor of submaximal $\dot{V}O_2$ (L/min) within a longitudinal sample of children and adolescents. It is important to note, however, that although the data analyzed were derived from a longitudinal sample, the analyses themselves were cross-sectional. A limitation of the repeated measures ANOVA procedure is that it does not allow for the inclusion of age-specific covariates. For example, it is not possible to investigate the combined effects of body surface area and RER on $\dot{V}O_2$ (L/min) using the repeated measures ANOVA procedure. For this reason, it is recommended that future research into factors that affect the development of RE using a longitudinal study design make use of more advanced statistical techniques such as multi-level modeling.

Further support of the conclusions drawn regarding the effects of the timing of maturity on RE development was provided by these tests. Only during the adolescent years, at ages 12 and 14 years, was age at PHV a significant predictor of submaximal $\dot{V}O_2$ (L/min). In fact, these results indicate that even once size and RER are controlled for, maturity status remains an important predictor of RE. This finding strengthens the conclusions drawn regarding the third hypothesis and again demonstrates the importance of continued investigation of factors responsible for these changes. Again, this is among the first papers to demonstrate longitudinally that maturity status significantly impacts submaximal $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$), even when appropriately normalized to body size.

Similar to the investigation conducted by Frost et al. (2002), a majority of the
variance in $\dot{V}O_2$ (L/min) values could be explained by the independent variables included in the various models. The mean adjusted determination coefficient ($R^2$) for the nine models was 0.789 with a range of 0.619-0.903; this compares favorably with the work of Frost et al., who were able to account for 77% of the variance in net $\dot{V}O_2$ in 30 participants aged seven to 16 years. As previously stated, though much of the variance in RE can be explained, a significant proportion remains unexplained; this reinforces the need for continued research into the predictive effects of different independent variables. Also, the analyses for this future research should include multi-level modeling, to make full use of the longitudinal nature of the data.

Given the conclusions drawn regarding the influence of physical activity exposure on the development of RE, it is not surprising that no significant predictive effects on $\dot{V}O_2$ (L/min) were identified for physical activity. Similarly, none of the other variables included in the age specific models were found to be significant predictors of $\dot{V}O_2$ (L/min). Although they may still bear a significant relationship to RE, it is clear that their contribution is dwarfed by those made by size and RER. A number of other factors have been suggested as possible explanatory variables for differences in RE; however, in many cases data were not available to test these claims. Nonetheless, the potential predictive effect of these variables deserves attention.

One possible explanation for the differences in $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) among (and within) participants in the present study is the rise in hemoglobin concentration that has been demonstrated in adolescent males (Armstrong & Welsman, 2001). Hemoglobin is the protein portion of the red blood cell that binds with oxygen for transport throughout the body (Plowman & Smith, 1997). Armstrong and Welsman used a longitudinal study
design to demonstrate that there is a significant increase in hemoglobin concentration in males between the ages of 13 and 17 years (2001). As the physiological limit for oxygen carrying capacity is 1.34 ml of oxygen per gram of fully saturated hemoglobin, increased hemoglobin concentrations could potentially increase the oxygen carrying capacity of blood (Plowman & Smith, 1997). An increase in the oxygen carrying capacity of blood would translate into greater oxygen availability at needed sites. It should be noted, however, that Armstrong and Welsman found that hemoglobin concentration was not a significant explanatory variable for differences in peak \( \dot{V}O_2 \) values; one may therefore question the impact of increasing hemoglobin concentrations on the development of RE in growing males.

Another factor that might partly explain age-related differences in RE is a difference in resting energy expenditure. Puhl (1989) demonstrated that, in general, children demonstrate higher resting energy expenditure values than adults. It has been suggested that the higher resting values may, in fact, lead to higher working \( \dot{V}O_2 \) values (Rowland, 1990). In an attempt to avoid this concern, Frost et al. (2002) used net \( \dot{V}O_2 \) values (exercising - resting values) when attempting to identify independent predictive variables which can explain differences in RE.

Though body mass was included in the anthropometric measurements taken at each testing occasion, no measurements were taken of specific body segments; therefore, data were not available to test the claim that segmental mass distribution can affect RE. Bailey and Pate (1991) discussed the possibility that smaller individuals (i.e. children) possess a relatively greater amount of body mass in the extremities, and would thereby have to perform a relatively greater amount of work moving body segments during
running than a larger individual. This increased workload would, in theory, translate into greater oxygen demands and therefore higher $\overline{VO}_2$ values.

The energy cost of ventilation has also been introduced as a possible explanatory factor for differences in RE. Bar-Or (1983) concluded that the metabolic cost of respiration is greater in children than adults. Ventilatory equivalent (ventilation per unit oxygen consumed) is greater in children, thus indicating decreased breathing efficiency. To compensate for this decreased efficiency, children are required to breathe more rapidly; the energy cost of this increased work due to ventilation may account for some of the age-associated differences in RE.

A final group of factors that have been suggested as possible explanations for inter- and intra-individual differences in RE are factors associated with the immature gait possessed by younger individuals (Bailey and Pate, 1991). Rowland et al. (1987) and Rowland and Green (1988) both provided evidence that children possess greater vertical displacement, increased hip, ankle and knee extension at take-off, greater time in the non-support phase of the stride, and a decrease in the relative distance of the support foot ahead of the body’s centre of gravity at contact than adults during treadmill running. These biomechanical differences between children and adults have been shown to be partially responsible for the differences in RE between these two groups during submaximal running.

To date, no comprehensive longitudinal data set exists in the world that could be used to answer the question of what factors explain age-associated differences in RE. Though some valuable insights were provided (i.e. the relative impacts of body surface area, RER and maturity status) by the present study, it is important to identify
shortcomings. One such example in the present study was that the effects of chronological age were muted as a result of analyzing the participants after grouping them into chronological age categories. The fact that hypothesis 2 identified a significant main effect of chronological age would suggest that this effect should still be present in further analyses. As mentioned, the statistical techniques used in the present study to address this question fall short of providing an appropriate answer.

4.3. SUMMARY AND CONCLUSIONS

4.3.1. Summary of Hypotheses

Hypothesis 1 stated that oxygen consumption expressed relative to (1.a.) body surface area, (1.b.) an allometric exponent of body mass or (1.c.) fat-free mass will be more appropriate than a simple ratio of $\dot{V}O_2$ and body mass. Hypotheses 1.a. and 1.b. were accepted, while Hypothesis 1.c. was rejected.

Hypothesis 2, which stated that running economy, as measured at a treadmill speed of $9.6 \text{ km·h}^{-1}$ will not improve with increasing chronological and biological age once an appropriate adjustment for size has been made, was rejected.

The third hypothesis, which tested the claim that the timing of maturation would not have any significant effect on the development of running economy, as measured at a treadmill speed of $9.6 \text{ km·h}^{-1}$ at each chronological age was also rejected.

Hypothesis 4, which stated that boys who are more physically active will demonstrate similar running economy to those who are inactive, once an appropriate adjustment for size has been made at each chronological age was accepted.

The fifth and final hypothesis, which stated that chronological age, an
appropriate measure of body size (as determined in Hypothesis 1), age at PHV, leg
length, RER and physical activity will all be significant predictors of running economy
(L/min) as measured at 9.6 km·h\(^{-1}\) was rejected, as not all of these were found to affect
RE.

4.3.2. Conclusions

A number of findings from the present study are novel. First and foremost, this
is among the first studies to longitudinally demonstrate the appropriateness of
normalizing \(\dot{\text{VO}}_2\) values using body surface area. None of the other normalizing
techniques tested satisfied the described criteria; therefore no further analyses were
conducted using these approaches. Once an appropriate approach to normalizing \(\dot{\text{VO}}_2\)
(ml·m\(^{-2}\)·min\(^{-1}\)) values was identified, it was demonstrated that chronological age still had
a significant effect on the development of RE; albeit only at the highest ages tested (15
and 16 years). This was also a novel finding, as no previous longitudinal study had
demonstrated the effects of chronological age on appropriately normalized \(\dot{\text{VO}}_2\) values.
This finding allowed for further investigation into the underlying causes of these age-
associated differences in RE.

Maturity status was shown to be a significant factor in explaining RE differences
at certain ages; specifically, the effects of maturity status on RE were most pronounced
during the circumpubertal years. The general pattern was that those who matured early
were less economical than average or late maturers when compared at common
chronological ages during the circumpubertal years. This study supported evidence
from Bouchard et al. (1978) which suggested that there might be an effect of maturity
status between the ages of 12-16 years.

No relationship was identified between physical activity exposure and RE development during childhood and adolescence. The exact reason for this finding is difficult to identify, as it may simply be an accurate reflection of a non-relationship, or it could be that the measurement and classification tools used were not sensitive enough to separate the most from the least active. There also remains the possibility that the activity completed by the participants in the present study did not meet a certain threshold (duration, intensity and/or type) required to bring about changes in RE; or that adulthood may be a more sensitive period than childhood or adolescence for the effects of physical activity on influencing RE changes within an individual.

Finally, it was shown that the single best predictor of submaximal $\dot{V}O_2$ (L/min) was an appropriate measure of body size, in this case, body surface area. The relative effect of body size, however was not the same at all ages. Similarly, the relative impact of RER was not uniform at all ages tested, and, in fact, was not even significant at some ages. More evidence was produced that indicated that the effects of age at PHV (used as an index of maturity status) were only significant during the circumpubertal years. An important conclusion drawn regarding these findings was the need for appropriate statistical techniques in future investigations so as to take full advantage of the longitudinal nature of the data.
REFERENCES


## Appendix A – ANOVA and Post-hoc Result Tables

### Table A.1. ANOVA table for repeated measures ANOVA of $\dot{\text{VO}}_2$ (ml·m^{-2}·min^{-1}) by chronological age in the LSS

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**NOTE:** Greenhouse-Geisser epsilon value used due to violation of sphericity.
Table A.2. Pairwise comparisons of mean $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each chronological age

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<tr>
<th>Reference Age</th>
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<th>Mean Difference</th>
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### Table A.2. Pairwise comparisons of mean \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) at 9.6 km·h\(^{-1}\) for each chronological age (CONTINUED)

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<td>9</td>
<td>-0.066</td>
<td>0.015</td>
<td>0.002</td>
<td>0.014</td>
<td>-0.118</td>
</tr>
<tr>
<td>10</td>
<td>-0.039</td>
<td>0.013</td>
<td>ns</td>
<td></td>
<td>-0.084</td>
</tr>
<tr>
<td>11</td>
<td>-0.028</td>
<td>0.018</td>
<td>ns</td>
<td></td>
<td>-0.086</td>
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<tr>
<td>12</td>
<td>-0.041</td>
<td>0.015</td>
<td>ns</td>
<td></td>
<td>-0.093</td>
</tr>
<tr>
<td>13</td>
<td>-0.025</td>
<td>0.016</td>
<td>ns</td>
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<td>-0.079</td>
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<tr>
<td>14</td>
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<td>0.015</td>
<td>ns</td>
<td></td>
<td>-0.093</td>
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<tr>
<td>16</td>
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<td>0.017</td>
<td>ns</td>
<td></td>
<td>-0.007</td>
</tr>
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<td>16</td>
<td>8</td>
<td>-0.094</td>
<td>0.025</td>
<td>0.015</td>
<td>-0.179</td>
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<td>0.017</td>
<td>0.000</td>
<td>0.174</td>
<td>-0.060</td>
</tr>
<tr>
<td>10</td>
<td>-0.090</td>
<td>0.016</td>
<td>0.000</td>
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</tr>
<tr>
<td>11</td>
<td>-0.078</td>
<td>0.021</td>
<td>0.014</td>
<td>0.149</td>
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</tr>
<tr>
<td>12</td>
<td>-0.092</td>
<td>0.018</td>
<td>0.000</td>
<td>0.151</td>
<td>-0.033</td>
</tr>
<tr>
<td>13</td>
<td>-0.076</td>
<td>0.018</td>
<td>0.004</td>
<td>0.138</td>
<td>-0.014</td>
</tr>
<tr>
<td>14</td>
<td>-0.094</td>
<td>0.019</td>
<td>0.000</td>
<td>-0.158</td>
<td>-0.030</td>
</tr>
<tr>
<td>15</td>
<td>-0.051</td>
<td>0.017</td>
<td>ns</td>
<td></td>
<td>-0.108</td>
</tr>
</tbody>
</table>
Table A.3. ANOVA table for repeated measures ANOVA of \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) by biological age in the BSS

<table>
<thead>
<tr>
<th>Source</th>
<th>Epsilon</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 )</td>
<td>Huynh-Feldt</td>
<td>0.133</td>
<td>4.887</td>
<td>2.714E-02</td>
<td>2.816</td>
<td>0.019</td>
</tr>
<tr>
<td>Error(( \dot{V}O_2 ))</td>
<td>Huynh-Feldt</td>
<td>1.837</td>
<td>190.605</td>
<td>9.638E-02</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Huynh-Feldt epsilon value used due to violation of sphericity.
Table A.4. Pairwise Comparisons of mean $\dot{V}O_2$ (ml·m$^{-2}$·min$^{-1}$) at 9.6 km·h$^{-1}$ for each biological age in the biological sub-sample

<table>
<thead>
<tr>
<th>Reference Age</th>
<th>Comparative Age</th>
<th>Mean Difference</th>
<th>Standard Error</th>
<th>Significance</th>
<th>95% Confidence Interval for Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>-3</td>
<td>-2</td>
<td>-0.018</td>
<td>0.024</td>
<td>ns</td>
<td>-0.092 0.056</td>
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<tr>
<td>-1</td>
<td>-0.016</td>
<td>0.015</td>
<td>ns</td>
<td>-0.064 0.033</td>
<td></td>
</tr>
<tr>
<td>0</td>
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<td>0.021</td>
<td>ns</td>
<td>-0.079 0.047</td>
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</tr>
<tr>
<td>1</td>
<td>-0.016</td>
<td>0.020</td>
<td>ns</td>
<td>-0.039 0.109</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.035</td>
<td>0.024</td>
<td>ns</td>
<td>-0.064 0.033</td>
<td></td>
</tr>
<tr>
<td>-2</td>
<td>-3</td>
<td>0.018</td>
<td>0.024</td>
<td>ns</td>
<td>-0.056 0.092</td>
</tr>
<tr>
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<td>0.002</td>
<td>0.021</td>
<td>ns</td>
<td>-0.068 0.064</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>-0.025</td>
<td>0.025</td>
<td>ns</td>
<td>-0.074 0.077</td>
<td></td>
</tr>
<tr>
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<td>0.002</td>
<td>0.024</td>
<td>ns</td>
<td>-0.021 0.127</td>
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</tr>
<tr>
<td>2</td>
<td>0.053</td>
<td>0.024</td>
<td>ns</td>
<td>-0.033 0.064</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>-3</td>
<td>0.016</td>
<td>0.015</td>
<td>ns</td>
<td>-0.068 0.064</td>
</tr>
<tr>
<td>-2</td>
<td>-0.002</td>
<td>0.021</td>
<td>ns</td>
<td>-0.068 0.064</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>-0.027</td>
<td>0.019</td>
<td>ns</td>
<td>-0.087 0.033</td>
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</tr>
<tr>
<td>1</td>
<td>0.000</td>
<td>0.021</td>
<td>ns</td>
<td>-0.065 0.064</td>
<td></td>
</tr>
<tr>
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<td>0.051</td>
<td>0.024</td>
<td>ns</td>
<td>-0.023 0.124</td>
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</tr>
<tr>
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<td>0.021</td>
<td>ns</td>
<td>-0.024 0.109</td>
</tr>
<tr>
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<td>0.025</td>
<td>0.025</td>
<td>ns</td>
<td>-0.054 0.103</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>0.027</td>
<td>0.019</td>
<td>ns</td>
<td>-0.033 0.087</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0.026</td>
<td>0.018</td>
<td>ns</td>
<td>-0.031 0.084</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.077</td>
<td>0.022</td>
<td>0.018</td>
<td>0.008 0.147</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-3</td>
<td>0.016</td>
<td>0.020</td>
<td>ns</td>
<td>-0.047 0.079</td>
</tr>
<tr>
<td>-2</td>
<td>-0.002</td>
<td>0.024</td>
<td>ns</td>
<td>-0.077 0.074</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>0.000</td>
<td>0.021</td>
<td>ns</td>
<td>-0.064 0.065</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>-0.026</td>
<td>0.018</td>
<td>ns</td>
<td>-0.084 0.031</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>0.051</td>
<td>0.021</td>
<td>ns</td>
<td>-0.016 0.118</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>-3</td>
<td>-0.035</td>
<td>0.024</td>
<td>ns</td>
<td>-0.109 0.039</td>
</tr>
<tr>
<td>-2</td>
<td>-0.053</td>
<td>0.024</td>
<td>ns</td>
<td>-0.127 0.021</td>
<td></td>
</tr>
<tr>
<td>-1</td>
<td>-0.051</td>
<td>0.024</td>
<td>ns</td>
<td>-0.124 0.023</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>-0.077</td>
<td>0.022</td>
<td>0.018</td>
<td>-0.147 -0.008</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>-0.051</td>
<td>0.021</td>
<td>ns</td>
<td>-0.118 0.016</td>
<td></td>
</tr>
</tbody>
</table>
Table A.5. Bonferroni post hoc tests for maturity group differences in $\dot{V}O_2$ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹ for appropriate chronological ages within the CSS.

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Mean $\dot{V}O_2$ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Early</td>
<td>1.339</td>
</tr>
<tr>
<td>Average</td>
<td>1.262</td>
</tr>
<tr>
<td>Late</td>
<td>1.238</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Mean $\dot{V}O_2$ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Early</td>
<td>1.370</td>
</tr>
<tr>
<td>Average</td>
<td>1.266</td>
</tr>
<tr>
<td>Late</td>
<td>1.233</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Mean $\dot{V}O_2$ (ml·m⁻²·min⁻¹) at 9.6 km·h⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>14</td>
</tr>
<tr>
<td>Early</td>
<td>1.378</td>
</tr>
<tr>
<td>Average</td>
<td>1.264</td>
</tr>
<tr>
<td>Late</td>
<td>1.215</td>
</tr>
</tbody>
</table>
### Table A.6. ANOVA table for two-way (maturity group by chronological age) repeated measures ANOVA of \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) at 9.6 km·h\(^{-1}\)

<table>
<thead>
<tr>
<th>Source</th>
<th>Epsilon</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 )</td>
<td>Greenhouse-Geisser</td>
<td>0.519</td>
<td>5.831</td>
<td>0.089</td>
<td>7.256</td>
<td>0.000</td>
</tr>
<tr>
<td>Interaction</td>
<td>Greenhouse-Geisser</td>
<td>0.268</td>
<td>11.663</td>
<td>0.023</td>
<td>1.870</td>
<td>0.038</td>
</tr>
<tr>
<td>Error(( \dot{V}O_2 ))</td>
<td>Greenhouse-Geisser</td>
<td>4.292</td>
<td>349.884</td>
<td>0.012</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Greenhouse-Geisser epsilon value used due to violation of sphericity.

### Table A.7. Bonferroni post hoc tests for interaction of chronological age and maturity status for \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) at 9.6 km·h\(^{-1}\) for appropriate chronological ages within the longitudinal sup-sample.

<table>
<thead>
<tr>
<th>Chronological Age</th>
<th>Mean ( \dot{V}O_2 ) (ml·m(^{-2})·min(^{-1})) at 9.6 km·h(^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 years</td>
<td>1.368</td>
</tr>
<tr>
<td>12 years</td>
<td>1.401</td>
</tr>
<tr>
<td>13 years</td>
<td>1.372</td>
</tr>
<tr>
<td>14 years</td>
<td>1.362</td>
</tr>
</tbody>
</table>

### Table A.8. ANOVA table for two-way (physical activity group by chronological age) repeated measures ANOVA of \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) at 9.6 km·h\(^{-1}\)

<table>
<thead>
<tr>
<th>Source</th>
<th>Epsilon</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 )</td>
<td>Greenhouse-Geisser</td>
<td>0.439</td>
<td>5.951</td>
<td>0.074</td>
<td>5.966</td>
<td>0.000</td>
</tr>
<tr>
<td>Interaction</td>
<td>Greenhouse-Geisser</td>
<td>0.149</td>
<td>11.902</td>
<td>0.013</td>
<td>1.014</td>
<td>ns</td>
</tr>
<tr>
<td>Error(( \dot{V}O_2 ))</td>
<td>Greenhouse-Geisser</td>
<td>4.411</td>
<td>357.053</td>
<td>0.012</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Greenhouse-Geisser epsilon value used due to violation of sphericity.

### Table A.9. ANOVA table for two-way (physical activity group by biological age) repeated measures ANOVA of \( \dot{V}O_2 \) (ml·m\(^{-2}\)·min\(^{-1}\)) at 9.6 km·h\(^{-1}\)

<table>
<thead>
<tr>
<th>Source</th>
<th>Epsilon</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \dot{V}O_2 )</td>
<td>Huynh-Feldt</td>
<td>0.069</td>
<td>5.000</td>
<td>0.014</td>
<td>1.440</td>
<td>ns</td>
</tr>
<tr>
<td>Interaction</td>
<td>Huynh-Feldt</td>
<td>0.070</td>
<td>10.000</td>
<td>0.007</td>
<td>0.733</td>
<td>ns</td>
</tr>
<tr>
<td>Error(( \dot{V}O_2 ))</td>
<td>Huynh-Feldt</td>
<td>1.767</td>
<td>185.000</td>
<td>0.010</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE: Huynh-Feldt epsilon value used due to violation of sphericity.