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CONTENTS

PHYSICAL ACTIVITY, PEAK OXYGEN UPTAKE AND PERFORMANCE ON THE WINGATE ANAEROBIC TEST IN 12-YEAR-OLDS

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ABSTRACT

This study examined 12-year-olds' physical activity (PA) patterns and investigated relationships between habitual PA and either peak VO_2 or Wingate anaerobic test performance. Subjects were 60 boys and 63 girls. PA was estimated from continuous heart rate (HR) monitoring over 3 schooldays. Criterion measures were % time and 5, 10 and 20 min periods with HR >139 bpm (moderate activity) and >159 bpm (vigorous activity). Boys spent a significantly higher % time than girls with HR >139 bpm (7.9% vs 6.5%, $p < 0.05$) and >159 bpm (3.7% vs 2.5%, $p < 0.001$). Boys recorded significantly more 5, 10 and 20 min sustained periods above each of the thresholds than girls ($p < 0.05$ to $p < 0.001$). Five minute periods of moderate physical activity were relatively common but longer periods of sustained moderate or vigorous activity were not characteristic of either sex. No significant correlations ($p > 0.05$) between PA and either aerobic or anaerobic exercise variables in boys ($r = -0.127$ to 0.160) or girls ($r = -0.017$ to 0.041) were detected. The lack of relationship between habitual PA and aerobic and anaerobic performance reflects the fact that few young people experience PA of the intensity, duration and frequency likely to improve aerobic or anaerobic performance.

Key words: mean power, peak power, peak VO_2, physical activity, young people

INTRODUCTION

Peak oxygen uptake (peak VO_2), the highest oxygen uptake elicited during an exercise test to exhaustion, is widely recognised as the best single indicator of young people's aerobic fitness [7]. The determination and interpretation and peak VO_2 in relation to growth and maturation has been extensively researched [8, 24]. The anaerobic performance of children and adolescents has received less research attention than the development of aerobic performance. This may in part be attributed to the lack of standardised, valid and reliable testing protocols with which to assess young people's anaerobic performance [28]. The Wingate Anaerobic Test (WanT), which allows the determination of peak power (PP) over 1 to 5s and mean power (MP) over its 30s duration, has emerged as the most popular test of young people's anaerobic performance and data on PP and MP in relation to growth and maturation are accumulating [18, 28].

The assessment and interpretation of young people's habitual physical activity is complex and a clear understanding of methodology is required to interpret adequately the extant literature. The technique used must be socially acceptable, it should not burden the child or adolescent with cumbersome equipment and it should minimally influence habitual physical activity [8]. The most accurate estimates of young people's physical activity patterns come from objective measures used over several days [17] and a minimum monitoring period of 3 days has been recommended [12, 16]. All currently available methods have weaknesses and no single technique is universally accepted as the gold standard for estimating children's and adolescents' level of habitual physical activity [6].

A recent review of the literature located only 10 studies of European children and adolescents which had used objective techniques and monitored physical activity over at least three full days [6]. Heart rate monitoring was the principal technique used in nine of the published studies. The data are remarkably consistent and indicate that significant numbers of European young people do not regularly engage in sustained periods of moderate to vigorous physical activity. Boys are generally more active than girls and

whereas the level of activity of both sexes declines with age the decline is more marked in girls [6].

Whether habitual physical activity impacts on the ability to perform aerobic or anaerobic exercise during growth and maturation is not well-documented. The evidence relating habitual physical activity to peak VO_2 in children and adolescents is equivocal, perhaps because most studies have involved small sample sizes, limited periods of monitoring physical activity or predictions of peak VO_2 from field tests or submaximal data [1]. Young people's anaerobic performance in relation to their habitual physical activity does not appear to have been investigated despite the fact that the short bursts of physical activity which appear to typify the physical activity patterns of children and adolescents may be more appropriate to the promotion of anaerobic fitness rather than aerobic fitness.

Data on the physical activity patterns of European children and adolescents are relatively sparse, and understanding of the relationship between habitual physical activity and anaerobic or aerobic performance is clouded [8, 24]. As part of an on-going longitudinal study we have collected data on the peak VO_2, PP, MP and physical activity patterns of a large sample of 12-year-old children. The purposes of this paper are to use these data to examine young peoples physical activity patterns and to enhance understanding of the relationship between habitual physical activity and aerobic and anaerobic performance.

MATERIAL AND METHODS

All the children in year six (age 11.1 ± 0.4 y) of the 15 state schools in the city of Exeter (UK) were invited to participate in a longitudinal study of physical activity patterns, aerobic and anaerobic fitness, and body composition. Seventy percent of the eligible children volunteered, and written informed consent was obtained from both the children and their parent/guardian. The study received ethical approval from the Exeter District Health Authority Ethical Committee. In an attempt to detect sample bias, the stature

and body mass of the volunteers were compared with the stature and body mass of those who declined to participate. No significant difference ($p > 0.05$) was detected in either sex. Twenty five percent of the eligible children in each school were randomly selected from those who volunteered. The data reported here are from the 63 girls and 60 boys who satisfactorily completed the appropriate procedures during the second year of the study.

The children had visited the laboratory on several occasions, and they were habituated to both the general environment and the specific experimental procedures. Body mass was determined using an Avery balance beam and stature was measured with a Holtain stadiometer.

Aerobic performance

Peak VO_2 was determined during an exercise test which involved running on a motorised treadmill using a discontinuous, incremental protocol. The subjects warmed-up by running on the belt at a speed of 1.67 $m \cdot s^{-1}$ for 3 min. The test commenced with four 3 min bouts at belt speeds of 1.94 $m \cdot s^{-1}$, 2.22 $m \cdot s^{-1}$, 2.50 $m \cdot s^{-1}$ and 2.78 $m \cdot s^{-1}$. For the remainder of the test the belt speed was held constant at 2.78 $m \cdot s^{-1}$ and the gradient elevated by 2.5% at the end of each 3 min period. The subjects were allowed a 1 min rest between stages but continued exercising until the test was terminated at the point of voluntary exhaustion. If the subjects showed signs of intense effort (hyperpnoea, facial flushing, unsteady gait, sweating) and if their heart rate had levelled off before the final exercise intensity, or had reached a value near or above 200 bpm, or if their respiratory exchange ratio (RER) was at least unity, peak VO_2 was accepted as a maximal index. All subjects reported here satisfied these criteria.

Throughout the test inspired and expired gases were monitored continuously using an Oxycon Sigma on-line analysis system which was recalibrated before each test with gases of known concentration. Heart rate was monitored using an electrocardiograph. Duplicate finger tip blood samples were taken immediately the exercise terminated and assayed for lactate concentration using a

YSI 2300 Stat Plus whole-blood analyser. The analyser self calibrated with a known concentration of lactate every five samples and the calibration was checked regularly against commercially prepared standards of verified concentration.

Anaerobic performance

Anaerobic performance was estimated using the WanT conducted on a friction-loaded cycle ergometer (Monark 8141) interfaced with a microcomputer. The same ergometer was used for all tests. The seat height and handlebars were adjusted appropriately for each subject, and the resistance was set at 0.74 $N \cdot kg^{-1}$ body mass [18]. Following a standardised 3 min warm-up, involving pedalling at 60 rpm interspersed with three 2 to 3 s a11-out sprints, the subject rested on the ergometer. The WanT commenced from a rolling start, at 60 rpm against minimal resistance (weight-basket supported). When a constant pedal rate of 60 rpm was achieved, a countdown of "3-2-1-go" was given, and the resistance was applied and the computer activated. Subjects were verbally encouraged throughout the test, and the power output was calculated each second for the 30s duration of the test. The peak power (PP) in 1 s and the mean power (MP) over the 30s period were recorded. Duplicate finger tip blood samples were taken 3 min following the WanT and immediately assayed for lactate concentration.

Physical activity

The frequency, intensity and duration of physical activity was estimated from continuous monitoring of heart rate. A self contained, computerised telemetry system (Polar, Sport Tester 3000) was used to record heart rates minute by minute. Each child was monitored from about 0900 to 2100 over three schooldays during the same week.

Data analysis

Data were stored and analysed using SPSS-PC+ statistical package. In order to interpret heart rate estimates of physical activity we have, in a series of studies (see Table 2), invited over 100 young people to visit the Centre to walk and run at various speeds on a horizontal treadmill. We noted that brisk walking and jogging elicit steady state heart rates of about 140 and 160 bpm respectively. We have therefore defined moderate physical activity (equivalent to brisk walking) as generating a heart rate >139 bpm and vigorous physical activity (equivalent to jogging) as generating a heart rate >159 bpm. Accordingly we have computed for the present data % of time with heart rate >139 and 159 bpm and number of 5 min, 10 min and 20 min sustained periods with heart rate >139 and 159 bpm.

Sex differences in stature, mass, exercise variables and heart rate estimates of physical activity were examined using one-way ANOVA. The relationship between exercise variables and body mass were examined further by computing ANCOVA on log-transformed data with mass as the covariate. As well as producing adjusted means for exercise variables, i.e. means from which the influence of body mass has been removed, the ANCOVA enables identification of the parameters a and b in the allometric equation, $y = a \cdot (mass^b)$. We have discussed in detail elsewhere [8, 30] the theoretical principles underlying the use of log-linear models to partition out the effect of body size for measures of physiological function during growth.

Relationships between heart rate estimates of physical activity and aerobic and anaerobic exercise variables were examined by sex using Pearson product moment correlation coeffcients. Sample specific mass exponents were used to calculate power function ratios ($y/mass^b$) for mass-adjusted correlations. Exponents, common to boys and girls, were 0.64 for peak VO_2, 1.08 for PP and 0.91 for MP.

RESULTS

There was no significant sex difference ($p > 0.05$) in age (12.2 ± 0.4 y vs 12.2 ± 0.4 y) or stature (boys, 1.52 ± 0.07 m vs girls, 1.53 ± 0.08m) but girls were significantly ($p < 0.05$) heavier than boys (44.5 ± 9.0 kg vs 41.0 ± 7.2 kg).

Aerobic and anaerobic exercise data are presented in Table 1. Boys' peak VO_2 was significantly higher than girls' ($p < 0.001$) whether expressed in $L \cdot min^{-1}$, or in ratio with body mass ($mL \cdot kg^{-1} \cdot min^{-1}$). The boys adjusted mean for peak VO_2 derived from the log-linear ANCOVA was significantly higher than the girls' ($p < 0.001$). There were no significant differences between boys and girls in heart rate, RER or blood lactate concentration at peak VO_2 ($p > 0.05$).

Table 1. Exercise variable by sex

	Boys (n = 60)	Girls (n = 63)
Aerobic exercise		
Peak VO_2 ($L \cdot min^{-1}$)	2.13±0.34	1.93±0.27***
Peak VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$)	52±5	44±6***
Adj. Peak VO_2 ($L \cdot min^{-1}$)	2.16	1.87***
Heart rate at peak VO_2 (bpm)	201±7	202±7
RER at peak VO_2	1.05±0.04	1.05±0.05
Lactate at peak VO_2 ($mmol \cdot L^{-1}$)	5.5±1.2	5.7±1.5
Anaerobic exercise		
Peak power 1s (W)	327.1±89.0	330.9±86.9
Peak power 1s ($W \cdot kg^{-1}$)	7.9±1.2	7.5±1.4
Adj. Peak power 1s (W)	329.0	307.0*
Mean power 30s (W)	276.9±69.5	278±65.6
Mean power 30s ($W \cdot kg^{-1}$)	6.7±1.1	6.3±1.4
Adj. Mean power 30s (W)	277.5	261.6
Lactate following WanT ($mmol \cdot L^{-1}$)	6.3±1.6	5.9±1.3

Values are mean ± standard deviation
Significant differences between groups * $p < 0.05$, *** $p < 0.001$

There were no significant ($p > 0.05$) sex differences in PP or MP whether expressed in W or in ratio with body mass ($W \cdot kg^{-1}$). The

boys adjusted mean for PP derived from the log-linear ANCOVA was significantly higher than the girls' ($p < 0.05$) but the adjusted means for MP were not significantly different ($p > 0.05$). No significant sex difference in post WanT blood lactate concentration was detected ($p > 0.05$).

Boys spent a significantly higher percentage of time than girls with heart rate > 139 bpm (7.9 ± 4.2 vs 6.5 ± 3.2, $p < 0.05$) and >159 bpm (3.7 ± 2.5 vs 2.5 ± 1.7, $p < 0.001$). The number of sustained periods with heart rate >139 bpm and >159 bpm is presented in Tables 2 and 3. Boys recorded significantly more 5 min, 10 min and 20 min sustained periods above each of the heart rate thresholds than girls ($p < 0.05$ to $p < 0.001$).

Table 2. Number of sustained periods with heart rate >139 bpm over 3 days of monitoring

		Boys (n = 60)	Girls (n = 63)
5 min:	0	1(1.7)	5(7.9)
	1	5(8.3)	6(9.5)
	2	9(15.0)	2(3.2)
	3 or more	45(75.0)	50(79.4)
10 min:	0	11(18.3)	27(42.9)
	1	13(21.7)	14(22.2)
	2	8(13.3)	5(7.9)
	3 or more	28(46.7)	17(27.0)
20 min:	0	35(58.3)	50(79.4)
	1	15(25.0)	7(11.1)
	2	7(11.7)	4(6.3)
	3 or more	3(5.0)	2(3.2)

Values are numbers (percentages)

There were no significant correlations ($p > 0.05$) between heart rate estimates of physical activity and either aerobic or anaerobic exercise variables in boys ($r = -0.127$ to 0.160) or girls ($r = -0.017$ to 0.041).

Table 3. Number of sustained periods with heart rate >159 bpm over 3 days of monitoring

		Boys (n = 60)	Girls (n = 63)
5 min:	0	10(16.7)	20(31.7)
	1	9(15.0)	10(15.9)
	2	6(10.0)	10(15.9)
	3 or more	35(58.3)	23(36.5)
10 min:	0	26(35.0)	43(68.3)
	1	26(35.0)	14(22.2)
	2	9(15.0)	2(3.2)
	3 or more	9(15.0)	4(6.3)
20 min:	0	44(73.3)	58(92.1)
	1	12(20.0)	4(6.3)
	2	1(1.7)	1(1.6)
	3 or more	3(5.0)	–

Values are numbers (percentages)

DISCUSSION

The aerobic and anaerobic exercise data reported here are in general agreement with values reported by other investigators [8, 28]. They will not, however, be discussed further in this paper as we have recently examined the aerobic and anaerobic performance of 12-year-olds using larger samples drawn from the same population and including the subjects reported herein [9, 10].

Heart rate monitoring provides an attractive, objective method of estimating physical activity in real-life situations over several days [20]. A number of sophisticated, self contained, computerised telemetry systems have been developed for the unobtrusive monitoring of heart rate. These systems are socially acceptable, they permit freedom of movement, they are not immediately noticeable, and they therefore should not unduly influence children's habitual physical activity. Continuous heart rate monitoring has been extensively tested and found to be both reliable and valid with young people [26, 27]. However, the interpretation of heart rate in the

context of physical activity is complex. Heart rate not only reflects the posture and metabolism of the subject but also the transient emotional state, the prevailing climatic conditions, and the specific muscle groups which perform the activity. Heart rate is not a direct measure of physical activity but an indicator of the relative stress being placed on the body by the activity. The relationship between heart rate and physical activity is therefore more secure at moderate to vigorous levels of physical activity than during low levels of activity [23].

The percentage of time heart rate is maintained above preselected thresholds during the period studied is normally used as an estimate of physical activity [14]. In addition to noting percentage (or total) time above thresholds the number and length of sustained periods above threshold heart rates may offer a more informative picture of physical activity patterns [3]. With this type of analysis, heart rate monitoring provides an attractive means of distinguishing physical activity patterns and provides an indication of the intensity, duration and frequency of physical activity. However, heart rate estimates of physical activity must be interpreted cautiously.

The present data reflect previous studies from both Europe [3, 5] and elsewhere [13, 19]. Boys appear to be more active than girls regardless of how the data are analysed. Boys engaged in vigorous, sustained physical activities more often than girls but sustained periods of physical activity do not appear to be characteristic of young people's physical activity patterns. Five minute periods of moderate physical activity were relatively common in both girls and boys although many more boys than girls experienced the equivalent of a daily 5 min period of vigorous activity. Longer periods of sustained physical activity were sparse and more likely to be experienced by boys than girls.

Whether young people are classifed as "active" or "inactive" depends upon the criteria implemented. There are no universally accepted criteria of activity/inactivity for 12-year-olds but the guidelines established by the International Consensus Conference on Physical Activity Guidelines for Adolescents (11 to 21-year-olds) are widely recognised. Two important recommendations emerged from this conference,

- adolescents should be physically active daily, or nearly every day, as part of play, games, sports, work transportation, recreation, physical education, or planned exercise, in the context of family, school and community activities
- adolescents should engage in three or more sessions per week of activities that last 20 minutes or more at a time and that require moderate to vigorous levels of exertion [25, p 308].

The present data enable an analysis of the number of young people who satisfy these recommendations. Only 5% of boys experienced the equivalent of a daily 20 min period of moderate or vigorous physical activity. Three percent of girls maintained their HR >139 bpm on at least three occasions during the 3 days of monitoring but no girl exhibited the equivalent of a daily 20 min period of vigorous activity. Ninety two percent of girls did not experience a single 20 min period with their heart rate >159 bpm. The data reinforce our earlier report that over a 10 year period in which we have monitored the physical activity patterns of over 500 girls, aged 5 to 16 years, we have yet to observe a single girl who experienced the equivalent of a daily 20 min period of vigorous activity [8]. These findings reinforce the extant literature which suggests that sustained bursts of moderate to vigorous physical activity do not appear to characterise the physical activity patterns of either children [11] or adolescents [3]. Few young people currently achieve the recommendations of the International Consensus Conference on Physical Activity Guidelines for Adolescents [25].

Our findings of no significant relationship between heart rate indicators of physical activity and peak VO_2 supports the majority of previous studies although few other studies have utilized an objective estimate of physical activity over at least 3 days and directly determined peak VO_2. Morrow and Freedson [21] reviewed 17 studies which investigated the relationship between aerobic fitness and habitual physical activity. They included studies which used performance measures (eg 1 mile run/walk) and predictions of peak Vo_2 from submaximal data as criterion measures of aerobic fitness and concluded that the majority of reports suggested no relationship between physical activity and aerobic fitness. The median correlation from all reviewed studies was $r = 0.17$.

Armstrong [1] identified 12 studies which had utilized laboratory-determined peak VO_2 as their criterion of aerobic fitness and concluded that, the findings of the better controlled studies indicate habitual physical activity makes only a very limited contribution, if any, to aerobic fitness. In the largest study to date, Armstrong *et al.* [4] monitored 111 girls and 85 boys, aged 11–16 y, using similar techniques to the present study and reported no significant relationships between physical activity and peak VO_2. In a similar study, Welsman and Armstrong [29] reported no relationship between heart rate indicators of physical activity and submaximal indices of cardiorespiratory fitness, in 73 13-year-old children.

No previous study, to our knowledge, has examined the relationship between young people's habitual physical activity and anaerobic fitness. It is, however, conceivable that the short bursts of activity which appear to characterise children's and adolescents' physical activity patterns may be more conducive to the improvement of anaerobic than aerobic performance. The present data suggest no relationship between habitual physical activity and either PP or MP.

Intuitively it may be expected that the most active youngsters are the fittest (aerobically or anaerobically) or that high levels of aerobic or anaerobic fitness may encourage physical activity. The present data support previous studies which indicate that any relationship between habitual activity and fitness is tenuous. The issue may be clouded by the methodological problems associated with the assessment of peak VO_2, anaerobic performance and physical activity. However, the simple explanation for the lack of relationship between habitual physical activity and laboratory measures of anaerobic and aerobic performance may be that few children and adolescents experience physical activity of the intensity, frequency and duration which has been shown necessary to improve aerobic and/or anaerobic performance.

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BLOOD LACTATE CONCENTRATION VERSUS ANAEROBIC ENERGY RELEASE DURING EXHAUSTING AND NON-EXHAUSTING TREADMILL RUNNING

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ABSTRACT

High intensity exercise depends on anaerobic energy release and leads to lactate production in the working muscles. Some of the lactate produced is released to the blood where it is easily measured. However, it is not known to what extent the blood lactate concentration reflects the anaerobic energy release during exercise. Our aim was to relate the peak postexercise blood lactate concentration to the accumulated 0_2 deficit, a measure of the anaerobic energy release during exercise. In 113 experiments, 21 subjects ran for 15 s to 8 min to exhaustion on the treadmill. In addition, 28 nonexhausting runs were carried out at a speed causing exhaustion in 2 min but stopped after 15–90 s. The peak postexercise blood lactate concentration rose by the accumulated 0_2 deficit, but for the same accumulated 0_2 deficit the blood lactate concentration for the nonexhausting exercise was only 20–60% of the value of the exhausting bouts. The peak blood lactate concentration appeared later after the exhausting than after the nonexhausting exercises. Regression analyses showed systematic differences between the subjects. One likely explanation for the high lactate concentrations after the exhausting bouts is that the released lactate may be distributed in a smaller extracellular volume, perhaps because of a higher sympathetic activity after the exhausting exercises. Consequently, the blood lactate concentration may not be an adequate measure of the anaerobic energy release during exercise.

Key words: accumulated O_2 deficit, anaerobic capacity, blood lactate concentration, exhausting exercise, physical performance test.

INTRODUCTION

High intensity exercise depends on energy from both aerobic and anaerobic processes. Aerobic energy release is conventionally expressed by the O_2 uptake, which is easily measured in the laboratory. The anaerobic energy release is on the other hand not readily measured, and therefore a number of different approaches have been tried for indirect determination. The simplest and probably most widely used approach is to measure the blood lactate concentration after strenuous exercise [6, 9]. The idea behind this method is that breakdown of glycogen to lactate is the main anaerobic source of ATP in muscle during high intensity dynamic exercise [2, 5, 21, 23, 33]. Some of the lactate produced is released to the blood. Blood is easily sampled, and the blood lactate concentration is readily measured by conventional methods.

At least two preconditions must be fulfilled if the blood lactate concentration shall be a reliable measure of the anaerobic energy release during exercise. A constant and preferentially large fraction of all lactate produced should be released to the blood, and the distribution volume should be constant and thus not vary between experiments and subjects. These preconditions do not seem to be fulfilled since the extracellular distribution volume varies by time [11, 26]. Moreover, during one-leg knee extensor exercise leading to exhaustion in about 3 min, around 35% of all lactate produced is released to the blood during the exercise, and another 35% is released in the recovery [2]. The situation is different when a large muscle mass is engaged since during bicycling most of the glycogen broken down reappears as lactate and other glycolytic intermediates within the same muscle [15], and only 10% of the muscle lactate may be released in the recovery after repeated bouts of exhausting bicycling [8]. We have recently found that the extracellular distribution volume of lactate released from the working leg may vary considerably and be larger after nonexhausting than after exhausting exercise [22]. These findings suggest that the blood lactate concentration may not reflect the anaerobic energy release accurately, particularly if exhausting and nonexhausting exercises are compared.

This study relates the peak postexercise blood lactate concentration to the accumulated O_2 deficit, which has been shown to be an adequate measure of the anaerobic energy release [16, 17, 19, 12, 33], during exercise. The subjects carried out exhausting treadmill running at different speeds and durations. Additional bouts of exercise stopped before exhaustion were also included to examine a possibly different effect of exhausting versus nonexhausting exercise on the blood lactate concentration. Finally, the time from the end of exercise to the appearance of the peak blood lactate concentration was recorded since this value may provide additional information about the release of lactate from muscle to blood and its extracellular distribution.

MATERIAL AND METHODS

Subjects

Altogether 21 healthy men with the following characteristics (mean ± SD): age, 25 ± 5 yr; height, 1.83 ± 0.07 m; weight, 74 ± 8 kg; maximal O_2 uptake, 47 ± 6 $\mu mol\ kg^{-1}\ s^{-1}$ (63 ± 8 $ml_{STPD}\ kg^{-1}\ min^{-1}$); anaerobic capacity (maximal accumulated O_2 deficit): 3.0 ± 0.5 $mmol\ kg^{-1}$, served as subjects. Most of the subjects were physically active students. The subjects were thoroughly informed about the purpose of the experiments and its practical details before they gave their written consent to volunteer as subjects. The experiments were approved by the Institute's Ethics Committee.

Procedures

All treadmill running was done on a motordriven treadmill at 6° (10.5%) inclination.

Pretests. First the subjects were trained in treadmill running before any testing or experiments were done. Then the maximal O_2 uptake was measured. Thereafter individual relationships between

the treadmill speed and the O_2 uptake were established as follows. For each subject the steady state O_2 uptake, taken as the value at the end of 10 min running at constant speed, was measured at 10 to more than 30 different running speeds below the maximal O_2 uptake. The anaerobic contribution is negligible during those conditions, and the measured O_2 uptake thus equals the O_2 demand or the ATP turnover rate expressed in O_2 units [17, 19]. For each subject the regression of the steady state O_2 uptake on the treadmill speed was calculated. The mean (±SD) regression parameters were: Y-intercept, 4 ± 1 μmol O_2 kg^{-1} s^{-1} (6 ml_{STPD} kg^{-1} min^{-1}); slope, 13 ± 1 μmol O_2 kg^{-1} m^{-1} (0.29 ml_{STPD} $kg^{-1}min^{-1}$); scatter around the regression line, 0.8 ± 0.2 μmol O_2 $kg^{-1}s^{-1}$ (1.1 ml_{STPD} kg^{-1} min^{-1}); correlation coefficient, 0.994 ± 0.004. Data from two other subjects tested were excluded since we were unable to obtain reliable relationships between the treadmill speed and the O_2 demand for these two subjects.

Experiments. Before each experiment the subject warmed up for at least 10 min at around 50% of his maximal O_2 uptake.

In the exhausting exercise the subjects ran five times to exhaustion. The treadmill speed was chosen so that exhaustion was reached in about 15 s, 30 s,1 min, 2 min, and 4 min. Data from two experiments were lost because the blood lactate concentration or accumulated O_2 deficit was not obtained from these two experiments. Eight subjects did additional exhausting runs lasting ≈1.5 min (n = 4) or more than 5 min (n = 6). Thus, data from altogether 113 runs to exhaustion have been included. Each subject did only one exhausting run each day, and at least one day of rest separated each experiment.

In additional nonexhausting exercises seven of the subjects repeated runs at the speed leading to exhaustion in about 2 min, but this time the exercise was stopped after 15 s, 30 s, 1 min, and 1.5 min, respectively. On two different days one shortlasting bout (15 s or 30 s) preceded one longlasting bout (1 or 1.5 min) the same day with 1 h rest in-between.

Before exercise, immediately after exercise, and 1, 3, 6, 9, and 12 min postexercise 20 or 25 μl of arterialized capillary blood was taken from a prewarmed finger and precipitated in 0.4 mol L^{-1} per-

chloric acid (PCA) for later measurement of the blood lactate concentration. Expired air was continuously collected in Douglas bags during each experiment for measuring the O_2 uptake and determining the accumulated O_2 deficit. The sequence of each experiment was randomized.

Analyses

Fractions of O_2 and CO_2 were measured on a Scholander gas analyzer [29] or on an automatic system (O_2: S3A/I O_2 analyzer, Ametek, Pittsburgh, PA, USA; CO_2 on a CO_2-analyzer from Simrad Optronics, Oslo, Norway). The expired volume was measured in a wet spirometer, and the maximal O_2 uptake was established by the levelling-off criterion [31]. The anaerobic capacity was taken as the maximal accumulated O_2 deficit, the value for a 2–4 min run to exhaustion [19]. The blood lactate concentration was measured enzymatically [13] with an imprecision of O_2 mmol L^{-1} or less in the single measurements. The lactate standard used (L 1759 L(+) lactic acid, purity 99%, Sigma Chemical Company, Saint Louis, MO, USA) was calibrated by measuring the increase in NADH by the LDH-reaction in a spectrophotometer. Repeated measurements of the lactate concentration using new standard curves for each series of measurements showed a day-to-day variation (SD) of 0.4 mmol L^{-1} or less.

Calculations and statistics

The accumulated O_2 deficit was determined as described elsewhere [19]. In short, the O_2 demand was estimated by linear extrapolation of the individual relationships between the treadmill speed and the steady state O_2 uptake established during the pretests, and the accumulated O_2 demand was taken as the O_2 demand times the exercise duration. The accumulated O_2 uptake is the O_2 uptake integrated over the whole exercise period. The accumulated O_2 deficit was taken as the difference between the accumulated O_2 demand and the accumulated O_2 uptake. The statistical imprecision in the estimated accumulated O_2 deficit is 4% or less [16, 17, 19].

The data are presented as individual values or as mean ± SEM unless otherwise stated. Linear regressions between the accumulated O_2 deficit and the blood lactate concentration were calculated by the procedure of geometric mean to account for between-subject variations in both entities [4, 27]. Since several measurements from each subject were included in the regression, the measurements were in formal terms not independent. However, dependence has probably not influenced the conclusions drawn in this study since the number of measurements from each subject was small and the number of subjects was large. In addition, the established relationships are not used for linear extrapolation. Analyses of the regression lines (in practice an analysis of covariance) for comparing different regression lines and models were calculated according to Weisberg [32]: First the residuals of fits of one common versus individual regression lines were compared by an ordinary analysis of variance. If individual fits appeared superior, possible differences in the individual slopes and the levels were examined.

RESULTS

Exhausting bouts

The subjects were exhausted in 15 s running at a treadmill speed of 6.6 ± 0.2 m s^{-1} (mean ± SEM). To enable the subjects run for more than 15 s, the treadmill speed had to be reduced, and the speed used for bouts lasting 4 min was 3.9 ± 0.1 m s^{-1} (Table 1). The accumulated O_2 deficit rose from 1.1 mmol kg^{-1} for the exhausting 15 s sprints to 3.0 mmol kg^{-1} for the 4 min exhausting bouts. The blood lactate concentration rose during exercise and continued to increase for 4–7 min after exercise, reaching individual peak values between 4 and 21 mmol L^{-1}. The peak post-exercise blood lactate concentration rose linearly by the accumulated O_2 deficit (Fig 1; $r = 0.85$), and in average a 1 mmol kg^{-1} increase in the accumulated O_2 demand was accompanied by a 4.2 mmol L^{-1} increase in the peak postexercise blood lactate con-

centration. The Y-intercept was nearly 5 mmol L^{-1}, and the error of regression (scatter around the regression line) was 1.9 mmol L^{-1} or 5–10 times the analytical error in single measurements of the blood lactate concentration.

The data for some subjects were all above the common regression line, while for other subjects all values were below the line. Examination of the individual curves showed that subjects with a low slope had particularly high peak lactate concentrations after the shortest bouts with the smallest accumulated O_2 deficits. Subjects with particularly high slopes had peak blood lactate concentrations around 20 mmol L^{-1} for the exhausting bouts lasting 2 min or more. Regression analyses showed significantly larger residuals when one common regression line was fit to the data compared with fits of individual relationships to each subject's data ($F_{40,71}$ = 3.34; $P < 0.001$). The systematic difference was caused both by individual differences in the slopes ($F_{20,71}$ =1.90; $P = 0.025$) and by differences in the individual levels for a given accumulated O_2 deficit ($F_{20,71} = 5.07$; $P < 0.001$).

Table 1. Summary of the main data from the exhausting exercises

	Exercise bout				
	15 s	30 s	1 min	2 min	4 min
Duration (s)	16±3	29±4	60±7	131±16	238±35
Speed ($m \cdot s^{-1}$)	6.6±0.7	6.0±0.7	5.3±0.6	4.4±0.6	3.8±0.6
O_2 demand ($\mu mol\ s^{-1}\ kg^{-1}$)	89±7	82±7	72±6	61±6	53±5
O_2 demand (relative to the $VO_{2\text{-max}}$)	1.91±0.24	1.76±0.20	1.54±0.18	1.30±0.10	1.14±0.07
Peak $[La]_{bl}$ (mmol L^{-1})	9±2	12±2	15±2	17±2	16±2
Time to peak $[La]_{bl}$ (s after exercise)	256± 68	337±88	389±80	381±128	318±120
Accumulated O_2 deficit (mmol kg^{-1})	1.1±0.2	1.5±0.3	2.3±0.3	2.9±0.5	3.0±0.5

The data are mean ± SD from 21 subjects. Speed is the treadmill speed at 6° (10.5%) inclination required to exhaust the subjects in the time shown. Peak $[La]_{bl}$, is the peak postexercise blood lactate concentration, and time to peak $[La]_{bl}$, is the time from the end of exercise to the peak value was found.

Nonexhausting bouts

Seven subjects repeated the treadmill runs at a speed of 4.5 ± 0.4 m s^{-1} (mean ± SD) leading to exhaustion in around 2 min but this time stopped after 15–90 s. The accumulated O_2 deficit and the peak post-exercise blood lactate concentration rose by each increase in the exercise duration (open symbols in Figure 1). When excluding the values for the 15 s run, the two parameters were roughly linearly related (r = 0.93), and in average the peak blood lactate concentration rose by 7.2 mmol L^{-1} for a 1 mmol kg^{-1} increase in the accumulated O_2 deficit; that slope is 72% larger than for the exhausting exercise bouts. The Y-intercept was –6 mmol lactate L^{-1}, and the X-intercept was 0.8 mmol O_2 kg^{-1}, suggesting that for exercise with an accumulated O_2 deficit less than about 1 mmol kg^{-1} (≈30% of the maximal) the blood lactate concentration did not increase much above the rest level. The scatter around the regression line was 2.0 mmol L^{-1}.

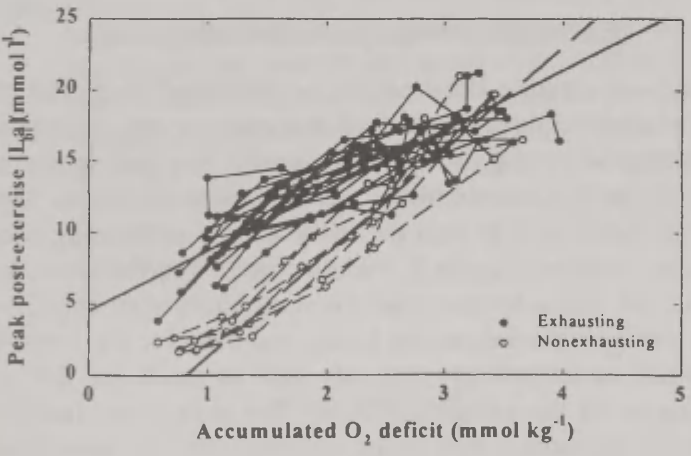

Figure 1. Peak postexercise blood lactate concentration versus the accumulated O_2 deficit for exhausting exercise lasting between 15 s and 7 min (filled symbols and solid lines), and nonexhausting exercise lasting 15–90 s (open symbols and short dashed lines). Datapoints for the same subject and serie are connected with lines.

The parameters of the two regression lines are (exhausting bouts, thick solid line): $Y = 4.5 + 4.2\ x$, $S_{Y|x} = 1.9$ mmol L^{-1}, $r = 0.85$, $n = 113$; nonexhausting bouts (the 15 s bouts were excluded while 2-min exhausting bouts for the seven subjects in question were included, thick dashed line): $Y = -5.9 + 72\ x$, $S_{Y|x} = 2.0$ mmol $L^{-1} = 0.93$, $n = 28$. The two regression lines were calculated as the geometric mean.

The data for two subjects were all above the common regression line, while for two other subjects all values were below the line. A regression analysis showed significantly larger residuals when one common regression line was fit compared with separate fits to each subject's data ($F_{12, 14} = 2.78$; $P < 0.05$). There were no differences between the individual slopes ($F_{6, 14} = 2.25$; $P = 0.10$), but the analysis showed systematic differences in the peak blood lactate concentration between the subjects when the accumulated O_2 deficits were the same ($F_{6, 14} = 3.20$; $P = 0.03$).

Exhausting versus nonexhausting bouts

For a given exercise duration the accumulated O_2 deficit of the nonexhauting bouts was ≈80% of the value of the corresponding exhausting bouts (Figure 2, upper panel). The peak postexercise blood lactate concentrations of the nonexhausting bouts were on the other hand far less than the value for the exhausting bouts of the same duration (Figure 2, middle panel). For the bouts lasting 15–30 s the values for the nonexhausting bouts were only 20–30% of the value for the exhausting bouts, and even for the 1 min bouts the lactate concentration rose only half as much for the nonexhausting as for the exhausting bouts. The peak blood lactate concentration appeared earlier in the recovery after the nonexhausting than after the exhausting bouts (Figure 2, lower panel).

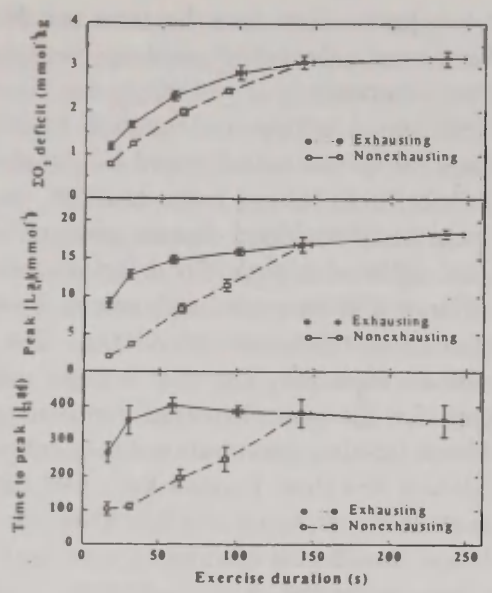

Figure 2. Accumulated O_2 deficit, (top panel), peak postexercise blood lactate concentration (middle panel), and time from the end of exercise to the peak blood lactate concentration appeared (lower panel) versus the exercise duration for the exhausting exercises lasting between 15 s and 4 min (filled symbols) and for the nonexhausting exercises lasting 15–90 s (open symbols). The data are mean ± SEM of seven subjects except for the exhausting bout lasting 1.5 min which was carried out by only four subjects.

DISCUSSION

The main result in this study was first that the peak postexercise blood lactate concentration rose by the accumulated O_2 deficit. However, the lactate concentration was around twice as high after the exhausting than after the nonexhausting exercise with the same accumulated O_2 deficit. There were in addition systematic differences between the subjects. Finally, the peak lactate concentration appeared later after the exhausting than after the nonexhausting

exercises. The two parameters were therefore not closely related, which would have been expected if they both measure the anaerobic energy release accurately.

The mean peak blood lactate concentration for the exhausting bouts in this study varied between 9 mmol L^{-1} for the 15 s sprints and 17 mmol L^{-1} for bouts lasting 2 min or more. Osnes and Hermansen [24] measured the blood lactate concentration on elite track runners and reported a peak blood lactate concentration of 16 mmol L^{-1} after a 100 m sprint and values between 20 and 24 mmol L^{-1} after competitions on 200–5000 m. Thus others have found higher values, especially for very intense and shortlasting sprints. Our data for the nonexhausting bouts suggest little increase in the blood lactate concentration for exercise with an accumulated O_2 deficit less than 1 mmol kg^{-1}. Our data agree well with the results of Margaria and coworkers [14] who found no increase in the blood lactate concentration for an estimated anaerobic ATP-formation equivalent to 1 mmol O_2 kg^{-1}. Hermansen on the other hand found that the peak blood lactate concentration rose linearly by time during a 3 min run to exhaustion [7]. The blood lactate concentrations in this study are therefore in fair agreement with others' data.

Since the accumulated O_2 deficit and the peak blood lactate concentration were not closely related, each method should be examined to unravel possible causes of the differences found. When adequately determined, the accumulated O_2 deficit has an experimental error of 4% or less [16, 17, 19]. The accumulated O_2 deficit has been compared with direct measures of the anaerobic energy release during bicycling [21, 28, 33], and no major deviations were found. The method has given reasonable values for treadmill running too [18–20, 30]. Systematic errors of 10–20% or more in the accumulated O_2 deficit is not likely [16, 17]. Possible errors in the accumulated O_2 deficit as a measure of the anaerobic energy release is therefore not large enough to explain the different relationships between the accumulated O_2 deficit and the peak blood lactate concentration for exhausting and nonexhausting exercise. The accumulated O_2 deficit is therefore here used as a quantitative measure of the anaerobic energy release.

If the blood lactate concentration is an accurate measure of the anaerobic energy release during exercise, the concentration should reflect a constant and preferably large fraction of all ATP regenerated anaerobically. Lactate accounts for around 75 % of all ATP produced anaerobically in muscle during exhausting bicycling lasting between 30 s and 4 min [5, 21, 23, 33], and possibly varying use of for example phosphocreatine should not be a big problem for the interpretation of these data. What may be a major problem is that the amount of lactate transferred from muscle to blood differs between experimental conditions, at least in relative terms. Bangsbo and coworkers [2] reported that during 3 min of oneleg knee extensor exercise 35% of all lactate produced was released during the exercise, and another 35% was released in the recovery period. We have on the other hand found that during bicycling when large muscle groups are engaged more than 90% of the glycogen lost in the quadriceps muscle can be accounted for by accumulation of lactate and other glycolytic intermediates in the muscle and by oxidation [15]. The lactate release may be relatively small during bicycling as judged from others' data on blood flow and arteriovenous lactate differences. Poole *et al* [25] found a blood flow of 0.004 L s^{-1} kg^{-1} body mass through both legs during bicycling, and Lindinger and coworkers [12] found an a-v difference for lactate across the leg of 1.5–2 mmol L^{-1} during 30 s exhausting bicycling. Applying these data to 2 min treadmill running suggests that less than 1 mmol lactate kg^{-1} body mass, equivalent to an accumulated O_2 deficit of 0.2 mmol kg^{-1} or <7% of the anaerobic capacity, was released during the run. These calculations are supported by a recent study [22], while data by Jorfeldt and colleagues [10] suggest even lower values. The lactate release after exercise may also be relatively small according to Hermansen and Vaage [8]. Their data suggest that during the first 30 min of the recovery after 3·1 min exhausting bicycling less than 10% of the muscle lactate was released to the blood. The blood lactate concentration thus seems to reflect a small and perhaps also varying part of the anaerobic energy release during exercise.

The peak postexercise blood lactate concentration was less for and appeared earlier after the nonexhausting than after the exhausting exercise. This could be caused by a larger uptake of lactate from the blood to other tissues after nonexhausting exercise, but in view of the discussion above the lactate release to the blood and thus uptake in other tissue may have been quite small in all experiments. This is supported by data of Bangsbo and coworkers [3] showing an uptake of lactate in other muscles of ≈3 μmol s^{-1} kg^{-1} muscle. For 2 min intense running that value is only 1 % of all produced. The splancnic uptake of lactate seems also quantitatively unimportant since Åstrand et al [1] found and uptake of around 0.8 μmol s^{-1} kg^{-1} body mass or 3–4 mmol min^{-1} after exhausting exercise, a value similar to the estimated uptake in inactive muscles.

It could be that the disproportionately high blood lactate concentration after the exhausting bouts was caused by a larger and more longlasting release of lactate than after the nonexhausting bouts. However, lactate is release from muscle to blood even after the peak concentration in blood is found [8, 22]. The extracellular distribution volume expands by time [11, 22, 26], and it may be twice as large 15 min postexercise compared with 5 min after exercise [11]. The disproportionately high blood lactate concentrations after the exhausting bouts may be a consequence of a smaller extracellular distribution volume, much less the extracellular space, rather than of a much larger release [22]. Thus, when the blood lactate concentration starts to fall some minutes after exercise, it may not reflect an uptake in other tissues but rather be a consequence of an expanding extracellular distribution volume [22]. It could be that a possibly larger sympathetic activity after exhausting than after nonexhausting exercise results in a smaller extracellular volume available after exhausting exercise. This possiblity remains to be examined.

In conclusion, the blood lactate concentration is higher and the peak value appears later after exhausting than after nonexhausting exercise and varied in addition between subjects with the same accumulated O_2 deficit. This suggests that if the anaerob energy

release is adequately measured by the accumulated O_2 deficit, the peak blood lactate concentration after exercise seems to be a poor measure of the anaerob energy release.

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INFLUENCE OF ESTROGEN ON MUSCLE FUNCTION AND POST-EXERCISE INFLAMMATORY RESPONSE: A REVIEW

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ABSTRACT

Recent research has focused interest on the potential for estrogen to influence muscle function and to protect muscle from exercise induced damage. Experimental evidence appears to be divided on whether estrogen can influence muscle force production and fatiguability, with some studies suggesting positive results while others report no effects. The potential mechanisms by which estrogen may exert it influence on physiological factors which may limit muscle function, force production and fatigue are also still unknown. Evidence for estrogenic efficacy as a protective agent during *in vivo* exercise induced muscle damage while emerging, it is still limited and usually indirect. The potential physiological mechanisms by which estrogen might influence skeletal muscle function and post-exercise muscle damage are also not yet clarified. This paper reviews the current understanding of the influence of estrogen on skeletal muscle and suggests directions for future research.

Key words: estrogen, skeletal muscle, strength, inflammatory response

INTRODUCTION

Recent research has focused interest on the potential for estrogen to influence muscle function and to protect muscle from exercise induced damage. Practical knowledge of the potential for estrogen to

influence skeletal muscle function and susceptibility to damage draws both theoretical and practical interest. Further knowledge of practical applications for estrogen replacement therapy in post-menopausal females, exercise prescription for females on oral contraceptives as well as gender differences in post-exercise muscle damage could all benefit by more information regarding the influence of estrogen on skeletal muscle and its mechanisms of action. Experimental evidence appears to be divided on whether estrogen can influence muscle force production and fatiguability, with some studies suggesting positive results [22, 26] while others reported no effects [7, 14]. The potential mechanisms by which estrogen may exert it influence on physiological factors which may limit muscle function, force production and fatigue are also still unknown. On the other hand, there is strong *in vitro* evidence that estrogen can act as an antioxidant and membrane stabilizer [30, 31]. However, while evidence to its efficacy as a protective agent during *in vivo* exercise induced muscle damage is emerging, it is still limited and usually indirect [31, 35]. Hence, this paper will critically review; (i) the current evidence for the ability of estrogen to influence muscle function and exercise induced muscle damage, (ii) suggest possible mechanisms by which estrogen may exert its influence on skeletal muscle, (iii) discuss future research directions in this field.

ESTROGEN AND MUSCLE FUNCTION

Estrogen has been demonstrated to directly influence contractility and function of both smooth and cardiac muscle [12, 27]. While the mechanisms by which estrogen can influence muscle smooth and cardiac muscle function have not been fully elucidated, it has been suggested that it may involve mediation of calcium movement and/or direct hormonal influence on myosin ATPase activity, myosin isoforms and protein content [12, 27]. Estrogen has also been reported to have anabolic effects on skeletal muscle in female cattle [15].

Recent studies from several English laboratories have heightened interest in the possible influence of estrogen on human skeletal muscle function. In 1993, Phillips *et al.* [23] reported differences in

age related changes to voluntary adductor pollicis muscle force between males and females. By measuring maximal voluntary force (MVF) as a function of adductor pollicis cross-sectional area (CSA) in 298 subjects aged 17–90 years, Phillips *et al.* [23] demonstrated that no significant gender difference existed in MVF/CSA until female menopause, when a significant decline occurred in females relative to males. In women taking estrogen for post-menopause hormone replacement therapy no such decline in MVF/CSA relationship was noted with increased age. Phillips *et al.* [22] further reported that 20–30 year old females exhibited greatest adductor pollicis MVF around ovulation than at other times during the normal menstrual cycle. They suggested that MVF may have been maximized due to the high circulating estrogen levels which peak just prior to ovulation.

Sarwar *et al.* [26] also examined the effects of different phases of the menstrual cycle on quadriceps and hand grip strength in 20 year old females. They noted significantly elevated hand grip and quadriceps strength as well as a lower quadriceps muscle fatigue rate at mid-cycle compared to luteal or follicular cycle phases and attributed this to the surge in estrogen preceding ovulation at mid-cycle. More recently Skelton *et al.* [29] found significantly increased adductor pollicis muscle strength in post-menopausal women given estrogen replacement therapy for up to one year. A control group administered placebo showed no improvement in strength over that year.

In contrast to these studies, based on a national survey, others have reported no differences in muscle strength between pre- and post-menopausal women [7]. In addition, there is little evidence for altered athletic or exercise performance in women taking oral contraceptives or at different stages of the menstrual cycle [10, 19]. In a more controlled study, Greeves *et al.* [14] reported no effect on first dorsal interosseus muscle strength or fatiguability in young women exposed to supra-physiological estrogen levels during in vitro fertilization. In addition, two recent studies from our laboratory using an *in vitro* mouse muscle model and an *in situ* rat soleus muscle model failed to demonstrate any significant effects of estrogen administration or gender on muscle strength, fatiguability or rate of force generation [9, 33]. Warren *et al.* [38] have also found no dif-

ference in extensor digitorum longus muscle peak torque following 150 eccentric contractions in ovariectomized mice, with or without estrogen replacement. Hence the potential efficacy of estrogenic action on skeletal muscle function is still unresolved.

If estrogen does indeed influence muscle strength and fatiguability, the potential mechanisms of action are on skeletal muscle not yet known. It appears unlikely that induction of muscle hypertrophy was a factor in those studies that reported a positive effect of estrogen on muscle function. Phillips *et al.* [23] noted that estrogen prevented a decline in mean force/cross sectional area in adductor pollicis muscle but no evidence of hypertrophy. Similarly Skelton *et al.* [29] did not see any muscle hypertrophy in their estrogen replaced subjects. The transitory nature of strength and fatiguability changes noted by Sarwar *et al.* [26] over the course of the menstrual cycle also speak against a hypertrophy based mechanism. Phillips *et al.* [23] and Sarwar *et al.* [26] have speculated that it intramuscular changes in inorganic phosphate (Pi) or pH are influenced by estrogen, this may be able to influence actin-myosin weak to strong binding transitions and thereby influence the number of cross-bridges in the force producing state at any given time. No measures of intramuscular effects of estrogen on Pi or pH are currently available, nor is it known how changes in estrogen concentration might be able to influence these variables.

Another possible mechanism by which estrogen might be able to influence muscle function might be via its potential to act as an antioxidant and membrane stabilizer [6, 31]. Since oxidative stress can potentially influence rate of fatigue in skeletal muscle [4], it is theoretically possible that estrogen could diminish the rate of strength loss over repeated muscular contractions. However, it is less clear how estrogen could influence maximal muscle strength through these mechanisms. It is also well documented that estrogen can increase fat utilization and preserve muscle glycogen stores during endurance exercise in rats [16]. However, it is unlikely that this mechanism would have played a significant role in the changes in strength and fatigue rates over the relatively short time periods reported in the positive studies.

It is possible that some of the aforementioned discrepancies in estrogenic effect might be due to the fact that estrogen may need to

exert its influence in concert with other hormones such as progesterone [14], or that cyclical fluctuations in estrogen, as occur during the normal menstrual cycle are necessary for its effects on skeletal muscle to be manifested [26]. These possibilities need to be investigated with a controlled model such as oophorectomized animals with varying degrees of estrogen/progesterone replacement.

ESTROGEN, POST-EXERCISE MUSCLE DAMAGE AND INFLAMMATION

A series of studies by Bär and Amelink and co-workers involving both *in vitro* and *in vivo* animal models established the influence of estrogen on post-exercise muscle damage [1, 2, 3, 5]. Gender differences, largely influenced by estrogen were critical in the degree of creatine kinase enzyme (CK) release from muscles following intense exercise. In both ovariectomized female and normal male rats estrogen significantly reduced the amount of CK loss from muscle following exercise [5]. CK release from isolated rat soleus muscle was also reduced by prior estrogen administration to ovariectomized female and normal male animals [2]. Post-exercise muscle CK leakage has been widely used as a marker of exercise induced muscle and muscle membrane damage [11, 37]. However, the level of post-exercise serum CK activity does not always correlate exactly with other indices of exercise muscle damage or strength loss [25]. Nevertheless, it remains a simple and useful indicator of the degree of muscle membrane disruption induced by an exercise regimen [1, 6, 11]. Amelink *et al.* [3] have also reported that, possibly due to their greater estrogen levels vitamin E deprived female rats had lower morphological and histochemical indices of muscle damage following exercise than vitamin E deprived males. Human females also have been reported to have lower resting and post-exercise serum CK activity [28, 37].

Again, the mechanisms by which estrogen may influence exercise induced muscle or membrane damage are not yet fully elucidated. It seems that the most likely mechanism by which estrogen can exert a protective influence on muscle is by acting directly as a

membrane stabilizer [5, 6, 18]. It has been suggested that estrogen can exert an antioxidant effect via direct interaction with membrane phospholipid side chains, thereby reducing their susceptibility to oxidative attack [31, 40]. Since estrogen is also a potent in vitro antioxidant [30] it is also possible that it may directly interact with oxidant radicals during and following exercise to diminish resultant muscle damage [31]. However, a recent study in our laboratory failed to demonstrate a significant antioxidant effect of estrogen administration on *in vivo* markers of exercise induced oxidative stress skeletal or cardiac muscle immediately following exercise in male rats [35]. Alternatively, it is possible that estrogen may also protect muscle from exercise induced muscle damage via direct interaction with muscle estrogen receptors [18]. This seems less likely since there are relatively few estrogen specific receptors on skeletal muscle and studies with the estrogen receptor antagonist, Tamoxifan have found it to have agonistic effects on post-exercise skeletal muscle damage in the rat model [6, 18].

Following damaging muscular exercise, a well documented post-exercise muscle inflammatory response ensues over several days [13]. This is characterized by, among other reactions, neutrophil and macrophage invasion of the muscle and cytokine release which via a number of interrelated mechanisms help promote and regulate the inflammatory response, removal of damaged tissue and subsequent repair of muscle [32]. Initial exercise induced muscle damage may be one of the factors associated with the degree of neutrophil adhesion and influx into muscle following exercise [24, 32]. Neutrophils and macrophages have the capacity to generate oxygen radicals via the NADPH oxidase reaction as well as generation of the strong oxidant, hypochlorous acid via the myeloperoxidase reaction [32, 39]. McCord [20] has suggested that neutrophil infiltration has the potential to destroy healthy as well as damaged tissue during the inflammatory response. Hence, if the production of neutrophil and macrophage generated oxidants is not closely controlled, they can theoretically propagate further post-exercise muscle damage and inflammation, secondary to the initial exercise induced damage [24, 32]. Belcastro *et al.* [8] have reported significant elevations in muscle neutrophil content (as determined by

myeloperoxidase activity) immediately following exhaustive running exercise in rats.

Therefore if estrogen does have the capacity to protect muscle from exercise induced damage via its activity as an antioxidant and/or membrane stabilizer, it is possible that it could also act as a post-exercise muscle anti-inflammatory and help minimize subsequent inflammation induced muscle damage. While there is currently little information on the ability of estrogen to influence post-exercise muscle inflammation, preliminary studies from our laboratory show some encouraging results. Male rats injected who were injected with estrogen for 2 weeks prior to an acute exercise bout, showed significantly reduced 24 hour post-exercise neutrophil invasion, as determined by myeloperoxidase activity, when compared to sham injected controls [34]. This suggested that estrogen administration may have diminished some aspects of the post-exercise muscle inflammatory response in male rats. In addition, the estrogen injected male rats exhibited a significantly lower 24 hour post-exercise muscle heat shock protein 72 (HSP 72) response, when compared to sham injected controls [21]. Heat shock protein response is believed to be an important reactive adaptation to muscle damage, which helps initiate subsequent tolerance to heat, chemical or exercise induced insult to tissues [17]. Thus the reduced 24 hour post-exercise heat shock protein response in estrogen administered male rats may be interpreted as indicative of reduced post-exercise inflammation associated muscle damage [21].

Much more research needs to be done to confirm these preliminary results. In particular, an exercised ovariectomized female animal model with varying degrees of estrogen replacement would be helpful. Measures of several indicators of post-exercise muscle inflammatory response such as pro- and anti-inflammatory cytokines, neutrophil adhesion factors as well as direct histochemistry of muscle would help confirm these preliminary findings.

It should be noted that one unexpected side effect of estrogen administration to male animals in particular is the loss of the antioxidant vitamin C from most tissues including heart and skeletal muscle [35, 36]. This occurs with or without exercise and in animals which synthesize their own vitamin C such as rats as well as in guinea pigs who like humans require vitamin C as part of their

diet [35, 36]. Other antioxidant vitamins such as vitamin E do not appear to be affected [35, 36]. Again, the mechanisms and physiological significance of this effect of estrogen administration on tissue vitamin C remain unclear. However, estrogen thus appears to have both positive and negative effects on aspects of tissue antioxidant protection.

At this stage it is hard to draw solid conclusions as to the influence of estrogen on skeletal muscle function and post-exercise damage and inflammatory response. Contrary findings on the efficacy of estrogen in influencing human and animal skeletal muscle function await clarification by a more definitive research model. The effectiveness of estrogen as an *in vitro* antioxidant and its ability to affect post-exercise serum CK activities are fairly well established. However, the mechanisms for the latter effect have yet to be conclusively defined. The ability of estrogen to diminish exercise induced muscle damage and inflammatory response, while theoretically compelling, also await definitive experimental verification and the elucidation of its potential mechanisms of action in skeletal muscle. The potentially exciting practical applications of further knowledge of estrogenic influence on skeletal muscle await further research clarification.

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PHYSIQUE OF NATIONAL ELITE FEMALE FILIPINO JUDO ATHLETES

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ABSTRACT

The purpose of this study was to describe elite Filipino female judo athletes competing for the national selection relative to their somatotype. They were also statistically compared with a sample of elite American female taekwondo athletes. The subjects for this study consisted of 19 (21.13 ± 0.9 years) adult female judo athletes who competed at a national tournament in Metro Manila. The anthropometric measurements taken included height, weight, two girths, two breadths and two skinfold thicknesses. ANOVA procedures were used to determine group differences collapsed over body weight and within weight division. The judo group as a whole was more endomorphic ($p < 0.001$) and mesomorphic ($p = 0.002$) than the taekwondo group, but less ectomorphic ($p < 0.001$). Within weight category, the judo athletes in the lightweight group (< 60 kg) were more endomorphic ($p < 0.001$) and less ectomorphic ($p = 0.003$) than the taekwondo athletes. The heavyweight judo athletes (≥ 60 kg) were more endomorphic ($p < 0.001$) and mesomorphic ($p < 0.001$), but less ectomorphic ($p < 0.001$) than their taekwondo counterparts ($p < 0.01$). The heavyweight judo athletes were comparable in their somatotype to sedentary females of various ethnicity.

Key words: judo, female, taekwondo, somatotype

INTRODUCTION

Judo may be considered one of the more popular Asian combative sports. It was the first Oriental martial sport that acquired Olympic status when it was introduced in the regular program of the 1964 Olympic Games in Tokyo (the second being taekwondo, which achieved full Olympic status in 1994 to be on the program of the Olympic Games in Sydney, Australia in 2000). Although it was absent in the 1968 Mexico City Olympic Games, judo has been a permanent Olympic feature ever since 1972. However, it was not until the 1992 Olympic Games in Barcelona, Spain, that the program also included female judo athletes.

Despite this popularity of the sport, scientific research on judo is scarce. Physiological correlates of judo performance were reported by Callister *et al.* [2] and Little [16]. Structural features of male judo athletes were described by Carter *et al.* [6] and Maas [18], who concluded that elite male judoka (judo athletes) are heavy for their height and endomesomorphic.

Recent reviews of research in martial sports have shown that the availability of information depends on the scientific discipline and the martial sport studied [11, 19]. Somatotyping and other kinanthropometric analyses in martial sports in general were primarily conducted by Claessens *et al.* [8] in judo and karate, by Pieter [19] and Taaffe and Pieter [23] in taekwondo and by Claessens *et al.* [10] in judo, demonstrating the lack of data in this scientific discipline relative to athletes in martial sports. Data on female athletes in martial sports are practically absent. To the best of our knowledge, only a few studies in a Western language have reported somatotype of female judoka [7, 12].

Data on Filipino combative athletes in general and female judoka in particular are absent. Therefore, the purpose of this study, which is part of an ongoing project on male and female martial sport athletes, the International Combative Sports Project (ICSP), under the leadership of the first author (WP), was to describe elite Filipino female judo athletes competing for the national team to assess their somatotype. In addition, the judo athletes were statistically compared with a sample of elite American female taekwondo participants to signify the specific requirements of each sport as well as

elucidate any potential deficiencies the judo athletes may have that may inhibit the full utilization of their biological potential.

MATERIAL AND METHODS

Subjects were 19 out of 21 adult female judo athletes who competed at the team trials in Metro Manila, Philippines. They came from all over the country and may be considered the present top in their respective weight divisions in the Philippines. The coaches of these athletes were asked for permission to assess their athletes during the weigh-in or shortly thereafter, but prior to the start of the actual competition. Consent was denied for two competitors. In addition, 15 elite American taekwondo athletes, who were used for statistical comparison, were tested at the United States Olympic Training Center in Colorado Springs, CO, USA, as part of the Oregon Taekwondo Research Project (OTRP) that was initiated by the first author (WP) [19].

The anthropometric measurements taken consisted of stature, weight, tensed arm and calf girths, humerus and femur breadths as well as the triceps and medial calf skinfolds. Standing height was measured with a wall-mounted wooden stadiometer to the nearest 0.5 cm. Body weight was assessed with a calibrated electronic digital scale to the nearest 0.01 kg. A Lafayette skinfold caliper was used to assess skinfold thicknesses. All measurements were taken according to the specifications provided by Ross and Marfell-Jones [21]. Each measurement was taken three times and the median used for statistical analysis. Somatotype rating was derived by means of the Heath-Carter method [7]. Somatotype was derived for each combative group as a whole as well as for those weighing less than 60 kg and those weighing 60 kg or more. Research has shown that differences in body build of judo athletes are dependent on weight category [9, 10, 13]. To determine the differences between the Filipino female elite judo and the American elite female taekwondo athletes, one-way ANOVAs were used. A two-way ANOVA (Sport * Weight) was employed to determine differences between judo and taekwondo athletes within weight divi-

sion, i.e., < 60 kg and ≥ 60 kg. In case a significant interaction was found, post-hoc comparisons only consisted of simple effects [15], since the interest entered around group differences within weight division. The level of significance was set at a = 0.05 for all analyses except for somatotype components, where a Bonfezroni-adjustcd $\alpha = 0.025$ was in effect.

RESULTS

Table 1 shows the means and standard deviations of age, height, weight and body build of the judo and taekwondo athletes by group as well as by weight division (< 60 kg and ≥ 60 kg, respectively) within group. The judo athletes as a group were significantly younger ($p = 0.004$), had less experience in their sport ($p = 0.004$) and were shorter ($p < 0.001$) than their taekwondo counterparts, but were not different in body weight ($p > 0.05$). In terms of somatotype, the judo athletes as a group were more endomorphic ($p < 0.001$) and mesomorphic ($p = 0.002$), but less ectomorphic ($p < 0.001$) than their taekwondo colleagues.

Table 1. Bio-demographic data and somatotypes (x ± SE) of female Filipino judoka and American taekwondo athletes

Variable	Judo			Taekwondo		
	<60 kg (n=13)	≥60 kg (n=6)	Total (n=19)	<60 kg (n=6)	≥60 kg (n=9)	Total (n=15)
Age (year)	20.6±3.7	22.7±4.8	21.3±0.9	25.5±7.2	26.4±3.0	26.1±1.3
Height (cm)	155.5±2.2	155.8±2.5	155.6±1.7	163.6±2.0	173.8±2.3	169.7±2.0
Weight (kg)	51.3±1.2	74.5±5.1	58.6±3.1	50.5±1.5	64.8±1.5	59.1±2.2
Experience (yrs)	4.7±0.9	6.7±1.6	5.4±0.8	8.3±1.9	8.4±1.3	8.4±1.0
Endomorphy	4.0±0.3	6.6±0.6	4.8±0.4	2.1±0.2	2.7±0.2	2.5±0.2
Mesomorphy	3.8±0.3	7.1±0.4	4.8±0.4	3.0±0.2	2.9±0.3	3.0±0.2
Ectomorphy	2.5±0.3	0.3±0.2	1.8±0.3	4.3±0.2	3.6±0.3	3.8±0.2

When looking at somatotype per weight division, there were significant Sport and Weight interactions for endomorphy ($p = 0.012$), mesomorphy ($p < 0.001$) and ectomorphy ($p = 0.039$). In addition to the two-way interactions, there were main effects for

Sport ($p < 0.001$) and for Weight ($p < 0.001$). Follow-up simple effects analyses revealed that the lightweight Filipino judoka were more endomorphic ($p < 0.001$) than their American taekwondo colleagues, but less ectomorphic ($p = 0.003$). There was no difference in mesomorphy ($p > 0.05$). The heavyweight judoka were more endo- and mesomorphic ($p < 0.001$), but less ectomorphic ($p < 0.001$) than the taekwondo athletes.

DISCUSSION

The lighter Filipino judo athletes may be characterized as meso-endomorphs, while their heavier counterparts are more endo-mesomorphic. Compared to Junior Canadian high-performance female judoka [17], both the lighter and heavier Filipino female judo athletes were more endomorphic, whereas the Senior Canadian judoka were similar in endomorphy to the Filipino athletes as a group. The Senior Canadian judo athletes were slightly more mesomorphic than their Filipino colleagues and slightly less ectomorphic. On the other hand, the Junior Canadian judoka were similar to the Filipino judo athletes in both mesomorphy and ectomorphy. The somatotype ratings for the Canadian judo athletes were 3.83–3.89–2.56 (Junior women) and 4.06–4.19–2.12 (Senior women). South American elite female judoka were found to have a somatotype rating of 4.1–4.1–1.8 [7]. They were 19.4 years of age with a height of 158.0 cm and a weight of 55.4 kg, which make them more similar to the lighter Filipino judoka in physique, age, height and weight.

It is not surprising to find the heavier Filipino female judo athletes to be more endomorphic and mesomorphic than the lighter judoka. Similar findings were reported for male judo athletes by Farmosi [13] and Claessens *et al.* [10] as well as for elite female Bulgarian judoka by Toteva and Zacharieva [24], although heavier Belgian elite judo athletes were as endomorphic as their lighter colleagues but with a higher mesomorphic rating [9]. Elite female Bulgarian judoka showed an overall mean somatotype of 3.87–4.59–1.87 [24]. The Bulgarian heavy weight judoka (+72 kg) had a

somatotype of 5.69–6.40–0.75, while that of the Filipino judoka ≥ 60 kg was 6.6–7.1–0.3.

It is interesting to note that the lighter Filipino judoka exhibit a similar somatotype pattern to sedentary males from Nigeria [14]. The Nigerians are older (27.6 years), taller (175.8 cm) and heavier (64.5 kg), but they recorded an endomorphic rating of 4.0 with the mesomorphy and ectomorphy components being 3.1 and 2.1, respectively. Somatotype was assessed according to the same protocol as the one utilized for the present study. The lighter Filipino female judo athletes are also comparable to sedentary Canadian females (15–19 years old) relative to their somatotype [1]. The Canadians were slightly more endomorphic, with a rating of 4.33, less mesomorphic (3.69) and slightly less ectomorphic (2.41).

The ectomorphic component of the heavyweight Filipino female judo athletes may be considered extremely low. Elite male judoka recorded a rating of 1.3 in the > 86 kg weight category [10], while elite Belgian male judo athletes had an ectomorphic rating of 1.7 in the 71–86 kg group [9], which is the same rating the Canadian competitive male judo athletes recorded who weighed 79 kg [17]. Farmosi's [13] Hungarian elite male judo athletes had a rating of 1.4 in the heaviest weight category (> 71 kg). Olympic male weightlifters in the 80–89.9 kg division were found to have ectomorphic ratings of 0.8 (Rome), 0.7 (Mexico City) and 0.3 (Montreal), respectively, with the Montreal lifters in the > 100 kg category recording an ectomorphy rating of 0.1, which was similar to the Montreal wrestlers in the same weight group: 0.2 [4]. On the basis of the present study, the morphological identification of the Filipino female judo athletes may leave room for improvement when compared to elite judo athletes active at the world and Olympic levels. Although comparisons with elite American female taekwondo athletes may be more indicative of each sport's unique requirements for peak performance, a proportionally large contribution of the endomorphic component to the athlete's morphological make-up may prove to limit this performance rather than facilitate it [3]. It is true that endomorphy increases with increasing body mass [3, 10, 13, 24], but its relationship to the mesomorphic component as well as to relative total body fat should also be taken into account [7]. A high mesomor-

phic rating may be deceiving, since at the upper level of the distribution, large skinfold measurements will have an effect on both endomorphy and mesomorphy. In addition to other variables, both components are a function of skinfold thicknesses. On the other hand, the elite taekwondo athletes did not show the same pattern of increasing endomorphy with increasing body weight, although the composition of the heavier weight category may be related to the findings. In other words, the taekwondo heavyweights may have a positively skewed weight distribution compared to that of the judo group. The large endomorphic component of the Filipino female judo athletes in combination with the low ectomorphy rating for the heavyweights may have a certain biomechanical advantage, such as a lower center of gravity, but it is suggested that a prevalence of fat will most likely be detrimental to performance due to a higher weight-to-strength ratio, which is especially essential if speed of movement is called for [22]. Interestingly, the endomorphic and mesomorphic ratings of the judo athletes by weight or collapsed over weight division are comparable to those of sedentary females of various ethnicity: American (4.5–3.8–2.8), British (4.3–3.6–3.4), Japanese (4.5–4.0–2.7), Mexican (5.7–4.3–1.8), and Eskimo (6.3–4.2–1.3) [5].

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CHANGES IN HORMONE LEVELS FOLLOWING LEG STRENGTH EXERCISE IN MALES

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ABSTRACT

The aim of our study was to investigate the hormonal adaptation during leg press exercise at individual physical working capacity (PWC) level in male students (n = 9). At first individual PWC was measured using a cycle ergometer test. Three progressive workloads for a period of four minutes were used. Individual PWC was calculated at the level of predicted HR_{MAX} (205 – ½ age). At the second series the individual PWC of leg press exercise was determined similarly to cycle ergometer test. The loads were 30%, 45% and 60% of one repetition maximum and subjects performed 15 repetitions per minute. Significant correlation between PWC values of cycle ergometer test and leg press exercise was found (r = 0.59). After a few days rest, the leg press exercise for maximal duration was performed at an individual PWC (15 repetitions per minute). Mean duration was 458.67 ± 105.09 sec and HR 136–162 beats·min^{-1}. Venous blood samples were obtained 3 minutes before and immediately after the test. Testosterone, cortisol, sex-hormone-binding-globuline (SHBG), creatine kinase, glycose and lactate (LA) concentrations were measured. Only LA (from 1.62 ± 0.74 to 5.31 ± 1.03 mmol·l^{-1}) and SHBG (from 32.03 ± 13.28 to 43.44 ± 9.81 mmol·l^{-1}) were significantly increased. Relatively short endurance leg press exercise at individual PWC level influenced moderately the blood anabolic/catabolic ratio.

Key words: testosterone, cortisol, physical working capacity, leg press, cycle ergometer test

INTRODUCTION

Total work of a resistance exercise is a key factor in the response of endocrine system during the acute recovery period following exercise [6]. The anabolic hormone testosterone (T), catabolic hormone cortisol (C) and the metabolite lactate (LA) have all been shown to be responsive to heavy-resistance exercise protocols consisting of short rest periods and multiple sets at near maximal intensity [9, 12]. However, there are few studies examining the impact of total work of resistance exercises to the hormonal balance of blood [11]. Häkkinen and Pakarinen [8] demonstrated that the intensity of the load (i.e. one repetition maximum, [1 RM] sets) alone could not compensate for the lack of the volume with regard to hormonal responses. Their data indicated that a threshold for the amount of total work and intensity interactions may excist for such hormonal changes.

Usually heart rate (HR) has not been used for the measurement of the intensity of weight exercises. In contrast, different cycle ergometer and/or treadmill tests using leg muscles, have been used for maximal and/or submaximal aerobic power estimation. World Health Organisation (WHO) recommended to use a simple cycle ergometer test for the measurement of physical working capacity (PWC) [13]. This test is also included to the EUROFIT for ADULTS [5] test battery. Subject pedals at three progressive workloads each for a period of four minutes during this test and the PWC at the age predicted maximum HR (205 – ½ age) is calculated. To our knowledge the effects of maximal duration leg press exercise at individual PWC calculated (similarly to cycle ergometer test) on peripheral hormone concentrations are unknown. Subsequently, it was our hypothesis that leg press exercise performed at the level of individual PWC for a relatively long period may well affect the level of anabolic-catabolic hormonal balance. Therefore, the main aim of our study was to investigate the hormonal adaptation during leg press exercise at individual PWC in male students.

METHODS

Nine male students of physical education faculty volunteered to participate in this study. None of them had a medical history of any endocrine disorder, orthopaedic problems, medical pathologies, or reported anabolic steroid use. Prior to participation, all subjects gave written informed consent, and completed a health history and physical activity questionnaires. None of them had participated in any resistance training for at least a year. This study was approved by the Medical Ethics Committee of University of Tartu.

The height (Martin anthropometer) and body mass (medical balance scale) of subjects were measured and body mass index (BMI, kg/m^2) calculated. Percentage of body fat was obtained using bioelectrical impedance analysis method (Bodystat-500, UK).

Individual PWC was measured at two times: using a (a) cycle ergometer test; and (b) leg press exercise. Three progressive workloads at the intensities of 150, 200 and 250 W each for a period of four minutes were used in a cycle ergometer test [13]. Heart rate [HR] at the end of each work load was measured using sporttester PE-3000 (Kempele, Finland). Individual PWC was calculated at the level of predicted HR_{MAX} (205 – ½ age) by extrapolation. After a few days rest, one repetition maximum (1 RM) of isoinertial leg press exercise was measured. Individual PWC of leg press exercise was determined similarly to cycle ergometer test [13]. Specifically, three sets of leg press exercise for a four minutes duration each were used. The loads were 30%, 45% and 60% of 1 RM and subjects performed 15 repetitions per minute. Heart rate at the end of each set was measured using sporttester PE-3000 (Kempele, Finland). Individual PWC at the age predicted HR_{MAX} (205 – ½ age) was measured by extrapolation. The leg press task was completed on a 45° leg press machine.

After a few days rest, the leg press exercise for maximal duration was performed at a individual PWC. Intensity was set to 15 repetitions per minute. Heart rate was measured continuously and stored at 15 second intervals (sporttester PE-3000, Kempele, Finland). Blood samples (5 ml) were obtained from the anticubital vein three minutes before and immediately after the test to deter-

mine T_{TOT}, C, sex-hormone-binding-globuline (SHBG), creatine kinase (CK), glucose (GL) and LA concentrations. Blood plasma was immediately stored in plastic Eppendorf tubes and frozen at -20°C until analyses were performed. All testing for each subject was conducted at the same time of day (from 9.00 to 12.00 a.m.) to reduce the within — individual effects of diurnal variations in hormonal concentrations. In addition, hematocrit was measured to monitor plasma volume changes.

Total T, C and SHBG were measured in duplicate by radioimmunoassays (Orion Diagnostica, Orion Corporation, Finland). All samples from an individual were run in the same assay to avoid any changes in inter-assay variability. The inter- and intra-assay coefficients of variation were less than 5%. Free T (T_{FREE}) was calculated as the ratio of T_{TOT} to SHBG and T_{FREE}:C as the ratio of T_{FREE} to C. Whole blood LA and GL concentrations were determined enzymatically with Lange (Germany) analyzer. While serum CK was determined using calorimetric assay method. Inter- and intra-assay variances were less than 8%.

Descriptive statistics (mean ± standard deviation [SD]) for each of the dependent variables were determined. Differences were estimated with Wilcoxon's signed rank test with an error of estimation set to 0.05. In addition, effect sizes (ES) were calculated to compare changes in biochemical parameters of blood as a consequence of maximal duration leg press exercise. Effect sizes of 0.2, 0.5 and 0.8 were characterized as small, moderate and large differences, respectively. The preintervention SD was used in ES computation. Kendall Rank Correlation coefficient was used to determine the relationships between hormone levels and functional characteristics. Again an alpha level of 0.05 was used.

RESULTS

Anthropometrical and PWC parameters of the subjects are presented in Table 1. Mean duration of the continuous leg press exercise at individual PWC level was seven minutes and 38 seconds. Mean HR stabilized after four minutes of work and was at the

level of 136–162 beats·min^{-1}. Mean PWC was about 41.6% from 1 RM. Total work (repetitions × weight lifted) was 6516.9 ± 3315.5 kg.

Table 1. The anthropometrical and physical working capacity parameters of subjects.

	Mean ± SD	Minimum	Maximum
Age (yrs)	22.33±2.78	19.00	28.00
Height (cm)	182.00±6.06	175.00	194.00
Weight (kg)	75.44±8.08	65.00	90.00
Percent body fat (%)	8.50±2.08	4.60	11.40
LBM (kg)	69.04±6.77	59.40	80.60
BMI (kg/m^2)	22.71±1.01	21.07	23.94
PWC cycle ergometer test (W)	348.78±95.80	240.00	480.00
1 RM leg press (kg)	151.33±49.47	105.00	270.00
PWC leg press (kg)	63.09±13.83	41.30	82.50
Maximal duration of leg press exercise (sec)	458.67±105.09	360.00	645.00

LBM — lean body mass; BMI — body mass index; PWC — physical working capacity; 1 RM — one repetition maximum.

Table 2 shows the results of a maximal duration leg press exercise test on biochemical parameters of blood. Only LA and SHBG concentrations were significantly increased ($p < 0.05$). However, the transformation of C values to ES demonstrated that leg press exercise highly influenced this hormone level (ES = 0.81). GL concentration (ES = 0.71) and T_{FREE}:C ratio (ES = 0.52) decreased moderately. While changes in T_{TOT} and T_{FREE} were low (ES < 0.27). Hematocrit was nearly constant before and after leg press exercise (45.9 ± 2.1 and 46.8 ± 2.0 respectively, $p > 0.05$).

Correlational analysis revealed a statistically significant correlation between PWC values of cycle ergometer test and leg press exercise ($r = 0.59$). In addition, the PWC measured by leg press exercise was highly related ($p < 0.05$) to the body height ($r = 0.75$), weight ($r = 0.78$), lean body mass ($r = 0.75$) and BMI ($r = 0.72$). In contrast, anthropometrical parameters did not influence PWC measured in cycle ergometer test. Furthermore, PWC value measured by cycle ergometer test did not correlate significantly with

resting blood biochemical parameters. However, PWC measured using leg press exercise correlated significantly with postexercise CK value ($r = 0.81$). While total work did not significantly correlate with any measured blood biochemical parameters.

Table 2. Changes in blood biochemical parameters during maximal duration leg press exercise at individual physical working capacity level (Mean ± SD).

	Before	After	ES
LA ($mmol \cdot l^{-1}$)	1.62±0.74	5.31±1.03*	4.99
GL ($mmol \cdot l^{-1}$)	5.27±0.70	4.77±0.68	0.71
T_{TOT} ($mmol \cdot l^{-1}$)	18.38±4.33	18.91±4.33	0.12
C ($mmol \cdot l^{-1}$)	451.89±139.44	564.56±146.04	0.81
SHBG ($mmol \cdot l^{-1}$)	32.03±13.28	43.44±9.81*	0.86
T_{FREE}	0.54±0.25	0.47±0.18	0.27
T_{FREE}:C ($x10^{-3}$)	1.33±0.86	0.88±0.40	0.52
CK ($u \cdot l^{-1}$)	167.56±109.96	230.56±224.51	0.57

LA — lactate; GL — glycose; T_{TOT} — total testosterone; C — cortisol; SHBG — sex-hormone-binding-globuline; T_{FREE} — free testosterone; CK — creatine kinase; ES — effect size. * — $p < 0.05$; ESs of 0.2, 0.5 and 0.8 are categorised as small, moderate and large differences, respectively.

DISCUSSION

The primary finding of this investigation was that the relatively short endurance leg press exercise at individual PWC level only moderately influenced the blood anabolic/catabolic ratio. Furthermore, the amount of total work was not a key factor in changes of hormone levels following endurance leg press exercise. However, the total amount of work was significantly related to the CK activity in the blood following leg press exercise at an individual PWC level.

The concentration of T_{TOT} in blood has been shown to increase immediately after heavy resistance exercise [11]. However, probably there exists a threshold of acute amounts of total work before the elevation of T_{TOT} in the circulating blood occurs [10]. Our re-

sults of the intensity of a prolonged leg press exercise at individual PWC level are contradictory. The LA concentration after the exercise was higher than lactate threshold of 4.0 $mmol \cdot l^{-1}$. (i.e. 5.31 ± 1.03 $mmol \cdot l^{-1}$, see Table 2). Similarly, shorter (40 s) leg press task has been reported to increase the LA concentration to 14 $mmol \cdot l^{-1}$ [1]. In contrast, the mean HR during the leg press exercise was relatively low (i.e. 136–162 $beats \cdot min^{-1}$). It is speculated that the amount of total work and the intensity of leg press exercise in our study were under the threshold which is needed to increase the concentration of T_{TOT} in blood [8]. The slight decrease in T_{FREE} level could be explained by the increase in T_{FREE} consumption during muscular work or by the significant increase in the concentration of SHBG (Table 2). This significant increase in SHBG concentration might be a delayed response of strength exercise to the physical stress.

It has been reported that a significant increase in the blood C level usually requires an exercise of more than 20 minutes duration with the intensity of at least 60% of maximal O_2 consumption [4]. The concentration of C in blood did not increase significantly ($p > 0.05$) in our study. However, the data may have been distorted by a low sample size. The large ES (ES = 0.81) supports this notion. Another explanation to nonsignificant changes in the C concentrations bases on the fact that blood samples were taken immediately after weight exercises and the concentrations of hormones might have been underestimated. For example there is evidence to suggest that peak values in hormone levels could be obtained between five and 30 minutes post exercise [12].

The results of several studies have been demonstrated that intense muscular work, especially eccentric actions, could cause muscle cell damage resulting in a significant increase in plasma activity of CK [2, 3]. In our study the activity of CK was not significantly increased (see Table 2). Total time of a leg press exercise at the individual PWC level was significantly related ($p < 0.05$) to the concentration of CK at rest ($r = 0.93$) and immediately after ($r = 0.84$) the test.

Similarly to other studies [7] the level of GL was unchanged following leg press exercise (see Table 2).

In summary, the relatively short endurance leg press exercise at individual PWC level only moderately influenced the blood anabolic/catabolic ratio. However, the addition of HR measure and the use of calculated PWC in leg press exercise during sport practice or recreational purposes need more investigations before any conclusions can be drawn.

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HEALTH-RELATED PHYSICAL FITNESS OF FILIPINO COLLEGE FRESHMEN

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ABSTRACT

The purpose of this study was to provide a profile of health-related physical fitness of Filipino freshmen. Subjects (189 males and 206 females) were incoming freshmen at a private university in Manila. In addition to age (males: 18.02 ± 1.36 years; females: 17.49 ± 0.96 years), height (males: 1.71 ± 0.07 m; females:1.58 ± 0.06 m) and weight (males: 65.92 ± 15.17 kg; females: 51.04 ± 9.17 kg), the following health-related physical fitness variables were assessed: sum of triceps and subscapular skinfolds, sit-and-reach, pull-ups (men), flexed-arm hang (women), 1.5-mile-run and sit-ups. Subjects were divided into three groups according to their body mass index and classified as lean, normal and obese. Differences between obesity levels within gender in any of the fitness variables were determined by MANOVA procedures. In the males, both the lean and normal males performed significantly more pull-ups and ran faster ($p = 0.016$ and $p = 0.012$, respectively) than their obese colleagues. In the females, the lean group performed better ($p = 0.000$) in the flexed-arm hang than the other two groups. The lean females performed more ($p = 0.021$) sit-ups than the obese females, as did the normal group ($p = 0.009$). Compared to American norms, the Filipino students rate poorly, generally below the 25th percentile.

Key words: health, fitness, youth

INTRODUCTION

The leading cause of mortality for Filipinos across all ages is heart disease, which accounted for 15.3% of the total deaths in 1992 [20]. In the same year, close to 5% of the total mortality in the Philippines comprised children between the ages of five and 19 years [20]. Regular physical activity is believed to help in reducing the risk of all-cause mortality [22], osteoporosis [17] and non-insulin dependent diabetes mellitus [11]. A shift downward in all-cause mortality of about 3–5% in the whole population through improved fitness and related aspects, such as nutrition, socio-psychological environment, hygiene, etc., has been estimated to be attainable and would be a major public health achievement [7]. Since the relationship between physical fitness and reductions in coronary heart disease as well as other risks has been established for adults but not yet clearly for children and adolescents, information on this group's health and fitness has become a major public health [30].

Recent studies among children and youth have shown that they are fatter compared to their counterparts in the 1960s [26, 27], which may be related to increased energy intake and/or decreased physical activity [4]. However, a general consensus about how fit today's children in the USA and possibly other western countries really are, has not yet been reached due to lack of data on physical activity and fitness in previous decades [25]. Longitudinal data seem to suggest that there is no difference in aerobic power between children tested in the 1980s compared to those in the 1930s and 1950s [13]. However, relative total body fat has been found to track best from the early teenage period to young adulthood as a cardiovascular disease risk indicator [14]. With an increased total body fat in children compared to previous decades [26, 27], the likelihood of incurring cardiovascular diseases in adulthood has also increased.

Investigations on health-related fitness in the Philippines are meager. A comparison of several fitness and anthropometric measurements between ethnic groups were also investigated [16]. Female students belonging to the highland Igorot tribe were heavier and shorter, as well as having shorter segmental lengths than low-

landers. The Igorots had superior leg power but had similar performances to those of the non-Igorots on the sit-and-reach, push-ups, squat thrusts, quadrant jump and Queen's step test. In general, among the comparable fitness data, it appears that the children of two decades ago fared better than those of recent days, although the disparity of the socio-economic status of the samples should be considered [6].

Research comparing Filipino children and youths to other samples is likewise scant. A study comparing the fitness of Filipino adolescents to that of analogous American and Japanese students [18] showed general deficiencies in arm and abdominal strength in the Filipinos, but they showed superior agility. Another study contrasted the physical fitness Filipino and Thai schoolchildren [21]. The Filipino boys were comparably poorer in arm and abdominal strength, as well as in aerobic endurance. The Filipino females did no better in arm and abdominal strength and in flexibility.

Recent research on selected health-related fitness parameters on Filipinos has shown that both college male and female students perform below the 25th percentile for American college students. For instance, Filipino male students scored at the 25th percentile of American college student norms in the sit-and-reach. Their female counterparts scored below the 25th percentile for the 50-yard dash [6]. The same Filipino college students also showed a preponderance of central fat accumulation in the males, which is believed to be implicated in coronary heart disease [24].

A disturbing pattern is apparent from the literature concerning the health-related fitness of Filipino children and youth. This, along with the absence of a definite policy in the Philippines in regard to the relationship of physical activity to health, necessitate further investigations in this matter. Hence, the aim of this study was to provide a profile of health-related physical fitness variables of Filipino freshmen.

MATERIAL AND METHODS

Subjects (189 males and 206 females) were incoming freshmen at a private university in Manila. Standing height was measured with a wall-mounted wooden stadiometer to the nearest 0.5 cm. Body weight was assessed with a calibrated electronic digital scale to the nearest 0.01 kg. The health-related physical fitness parameters included: sum of triceps and subscapular skinfolds, sit-and-reach, pull-ups (men), flexed-arm hang (women), 1.5-mile-run and 60-sec timed sit-ups [5]. In addition, subjects were divided into three groups according to their body mass index (BMI) [9] and classified as lean (BMI $\leq$ 21.150 and $\leq$ 21.766 for males and females, respectively), normal (males: 21.151–24.266; females: 21.767–25.767) or obese (males: $\geq$ 24.267, females: $\geq$ 25.768).

Descriptive statistics included means and standard deviations, while differences between obesity levels within gender in any of the fitness variables were determined by one-way MANOVA followed by univariate analyses. In case significant differences were found, T-tests were conducted to determine the exact location of these differences. Since the pull-ups for the males and flexed-arm hang for the females were not normally distributed and since all scores (including those who failed to perform either the pull-ups or the flexed-arm hang) were to be analysed, the Kruskal-Wallis test was used to determine any differences in these two components between BMI groups within each gender. All analyses were performed with $\alpha = 0.05$.

RESULTS

Tables 1 (males) and 2 (females) show the mean and standard deviations of the health-related fitness components. Among the lean males, 69.57% reported to be physically active, which was defined as engaging in physical activity for at least one day per week. In the normal and obese groups, this was 70.37% and 81.40%, respectively. Among the females, the lean group recorded 40.13% as being active, with 41.18% and 13.33% for the normal and obese

groups, respectively. More males (72.49% of all males) were active than females (38.35%). In the males, significant differences were found between BMI groups in age ($p = 0.002$), weight ($p = 0.000$), pull-ups (Kruskal-Wallis statistic = 23.30, df = 2, $p = 0.000$) and run time ($p = 0.023$). There was no difference between the lean and normal males in age ($p = 0.330$). However, the obese males were older than both the lean ($t = 3.415$, $p = 0.001$) and their normal ($t = 2.198$, $p = 0.030$) counterparts. As expected, weight increased with BMI level with no difference in height ($p > 0.05$).

Table 1. Means and standard deviations of demographic data and health-related fitness variables of male Filipino freshmen

Variable	Lean	Normal	Obese	Total
Age (year)	17.76±1.13 (n=92)	17.96±1.21 (n=54)	18.63±1.79 (n=43)	18.02±1.36 (n=189)
Height (cm)	1.71±0.07 (n=92)	1.72±0.07 (n=54)	1.72±0.06 (n=43)	1.71±0.07 (n=189)
Weight (kg)	55.72±6.27 (n=92)	66.53±6.14 (n=54)	86.97±14.69 (n=43)	65.92±15.17 (n=189)
BMI	19.08±1.47 (n=92)	22.56±0.90 (n=54)	29.33±4.06 (n=43)	22.40±4.62 (n=189)
Sum of 2 skin-folds (mm)	19.83±6.06 (n=92)	26.48±6.26 (n=54)	41.33±11.63 (n=43)	26.62±11.45 (n=189)
Run (min)	18.71±4.25 (n=86)	18.29±4.59 (n=52)	20.73±4.41 (n=40)	19.04±4.46 (n=178)
Pull-ups (reps)	3.85±3.43 (n=92)	3.42±3.46 (n=53)	1.38±1.96 (n=40)	3.19±3.31 (n=185)
Sit-and-Reach (cm)	29.25±8.45 (n=92)	28.81±8.55 (n=54)	27.56±8.10 (n=43)	28.74±8.38 (n=189)
Sit-ups (reps)	30.65±8.64 (n=91)	31.48±7.39 (n=54)	30.35±7.19 (n=43)	30.82±7.95 (n=188)

Table 2. Means and standard deviations of demographic data and health-related fitness variables of female Filipino freshmen

Variable	Lean	Normal	Obese	Total
Age (year)	17.47±0.97 (n=157)	17.41±0.99 (n=34)	17.80±0.78 (n=15)	17.49±0.96 (n=206)
Height (cm)	1.59±0.06 (n=157)	1.57±0.07 (n=34)	1.59±0.07 (n=15)	1.58±0.06 (n=206)
Weight (kg)	47.43±4.50 (n=157)	66.53±6.14 (n=34)	72.48±12.28 (n=15)	51.04±9.17 (n=206)
BMI	18.89±1.49 (n=157)	23.61±1.31 (n=34)	28.54±3.10 (n=15)	20.37±3.31 (n=206)
Sum of 2 skin-folds (mm)	29.88±4.33 (n=157)	40.85±8.16 (n=34)	51.13±12.53 (n=15)	33.24±9.79 (n=206)
Run (min)	24.80±4.33 (n=148)	26.18±2.96 (n=32)	26.19±2.66 (n=14)	25.13±4.06 (n=194)
Flexed arm hang (sec)	6.77±7.49 (n=155)	2.04±3.19 (n=32)	0.66±1.53 (n=15)	5.57±7.05 (n=202)
Sit-and-Reach (cm)	32.27±6.89 (n=156)	33.75±5.70 (n=33)	33.63±9.84 (n=15)	32.61±9.96 (n=204)
Sit-ups (reps)	21.03±8.87 (n=155)	20.88±6.03 (n=34)	15.53±7.01 (n=15)	20.60±8.43 (n=204)

Both the lean (Mann-Whitney U = 2773, p = 0.000) and normal males (Mann-Whitney U =1531, p = 0.000) performed significantly more pull-ups than their obese colleagues. The lean males did not differ in run time from their normal counterparts (t = 0.538, p = 0.591). However, they ran faster than their obese colleagues (t = 2.453, p = 0.016). The normal males were also faster (t = 2.565, p = 0.012) than their obese counterparts. No differences ($p > 0.05$) were apparent in flexibility or sit-ups between BMI groups.

In the females, significant differences were found in weight (p = 0.000), flexed-arm hang (Kruskal-Wallis statistic = 38.52, df = 2, p = 0.000) and sit-ups (p = 0.025) between BMI groups. Again as expected, body weight increased with increasing BMI level, while no difference was found in height ($p > 0.05$) or age ($p > 0.05$).

The lean females performed better ($p = 0.000$) in the flexed-arm hang than the other two groups. The normal females also performed better (Mann-Whitney $U = 322$, $df = 1$, $p = 0.051$) than the obese group. No difference was found in sit-ups between the lean and normal females ($p > 0.05$). However, the lean females performed more ($t = 2.326$, $p = 0.021$) sit-ups than the obese females, as did the normal group ($t = 2.722$, $p = 0.009$). No differences were found in flexibility and run time between the BMI groups.

DISCUSSION

Improved physical fitness through regular physical activity has continually been shown to have prophylactic effects against the risk of all-cause mortality [22]. The relationship has been recently officially recognized by the American government as evidenced by the Surgeon General's report [31]. Hence, the importance of health-related fitness must be espoused to the general public, specially in developing countries like the Philippines where health services are prohibitive. It is likewise essential to improve the physical fitness of children since there is evidence suggesting associations between childhood fitness and adult-onset risk factors for cardiovascular disease [1].

The results of this study seem to indicate that the present sample has generally poor health-related physical fitness. In abdominal strength, the Filipino females, regardless of BMI group, scored lower than the 10th percentile of the NCYFS (National Children and Youth Fitness Study) norms [26], while only the normal males from this study barely reached the 10th percentile of the same set of norms, while the rest scored lower.

All males also fared poorly in trunk flexibility, scoring slightly better than the 11th percentile, while the females placed lower than the 10th percentile. The lean and normal males landed below the 20th percentile of the NCYFS norms for pull-ups, while the obese group scored below the 10th. All male groups scored lower than the 25th percentile of the AAHPER norms [28]. The lean females scored lower than the 50th percentile of the AAHPER (American

Alliance for Health, Physical Education and Recreation) norms for flexed-arm hang, while the normal group landed lower than the 25th and the obese group lower than the 5th percentile of the same norms.

The best performance among the females in the 1.5-mile run (24.8 ± 4.33 minutes for the lean group) rate "very poor" in their age category on the scale developed by Cooper [5]. In fact, they had slower times than what is considered "very poor" for 60+ year-old women. Similarly, the males rated "very poor" as well, with the obese group scoring lower than the lower limit for men between 50 and 59 years old.

Most Filipino studies have used different test batteries, hence comparisons between them and the present study would be limited. Abarientos [2] compared fitness and sport-related skills of in- and out-of-school youth of similar age as those in the present study. The pull-up score of the male in-school youths (3.3 repetitions) was similar to those of the present lean and normal groups. However, the out-of-school subjects did noticeably better (4.5 repetitions). The lean females of the present study had better arm strength than their compatriots of two decades ago (3.2 and 5.2 seconds, for the in- and out-of school subjects, respectively), but the normal females did obviously worse. In developing fitness norms for the students of a state university, Sebastian [29] tested 367 females and 152 males. Females of comparable age to the present sample averaged 1.9 seconds for the flexed-arm hang, better only than the present obese group's performance. However, the corresponding male group from the same study recorded better pull-up scores, with a mean of 5.28 repetitions.

In trunk flexibility, both groups of both genders from the 70's [2] had better scores than the present sample. For females, the in-school sample scored 58.0 cm, while the out-of-school group averaged 49.6 cm. The male groups' means are 48.9 cm and 51.5 cm, respectively. The trend is the same with Sebastian's study [29], where the females scored 59.51 cm and the males, 61.61 cm. A more recent study that compared highland tribeswomen to lowlanders likewise reported superior sit-and-reach scores than the present sample's: 59.99 cm and 59.48 cm, respectively [16].

The contrasts of the preceding results may be due partly to the difference in the characteristics of the different samples. Most of the students from the past studies were recruited from public schools in non-cosmopolitan provinces in the Philippines, as opposed to those of the present study who came from an exclusive private university in Metro Manila. Variances in socio-economic status of the samples may invariably affect the physical activity patterns of the children, subsequently influencing the physical fitness scores [21]. Differences may exist between socio-economic status of those tested in the 70's [2, 29] and those for this study. Further research may be focused on the examination of the different variables that may exert influence, and the extent of this influence, on physical activity patterns as related to health-related fitness.

Compared to final-year secondary school children in Hong Kong [10], the lean an normal Filipino males scored slightly lower than the 50th percentile of norms (4 repetitions) in the pull-ups, while their obese counterparts scored around the 10th percentile (1 repetition). The total Filipino male sample scored around the 40th percentile. No comparison can be made with the Hong Kong females, for they did not do the flexed-arm hang. In the sit-and-reach, the lean and normal Filipino males scored around the 40th percentile of Hong Kong final-year secondary school boys, with the obese Filipinos between the 30th and 40th percentiles. The total Filipino male sample scored around the 40th percentile [10]. The females scored between the 20th and 30th percentiles (lean group) and at the 30th percentile (normal and obese groups). The total Filipino female group scored around the 30th percentile of norms for Hong Kong secondary school girls [10].

The lean and normal Filipino males scored around the 30th percentile of norms for Hong Kong final-year secondary school boys, while the obese Filipinos scored around the 20th percentile. The total Filipino male sample scored around the 30th percentile [10]. The Filipino females scored between the 10th and 20th percentiles for the lean and normal groups, and around the 5th percentile for the obese group. The total female Filipino sample scored between the 10th and 20th percentile for sit-ups compared to the Hong Kong girls [10]. In summary, when compared to other Asian

youth, the Filipinos also score poorly in selected fitness components, which seems to support earlier findings [18, 21].

The sum of two skinfolds in the lean males is around the 40th percentile of NCYFS norms for 18-year-olds, while those of the normal and obese groups were around the 20th and less than the 10th percentile, respectively [26]. The female groups all scored below the 10th percentile. Socio-economic status and dietary habits of the present sample may be related to the poor scores of the Filipino students. Research seems to indicate that modernisation of previously traditional societies have led, among other things, to an increase in obesity levels [e.g.,12, 15]. Since the present sample was drawn from a private university in an urban area, it stands to reason to suggest that, similar to university students in the Gulf countries, for instance, the Western lifestyle of the subjects may have been a contributory factor to their high fat levels [3, 19]. Future research should include Filipino youth from both urban and rural areas as well as younger children.

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ESTIMATION OF BODY COMPOSITION IN 9 TO 11 YEAR OLD CHILDREN FROM DIFFERENT SKINFOLD THICKNESS MEASUREMENTS

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ABSTRACT

The purpose of this investigation was to study whether meaningful differences occurred when percent body fat (%BF) values were estimated for prepubertal children using four different regression equations found in the literature. Skinfold (SF) thickness measurements were taken on 107 boys (10.30 ± 0.62 yrs; 144.09 ± 5.83 cm; 35.37 ± 5.36 kg) and 105 girls (10.18 ± 0.50 yrs; 143.30 ± 7.29 cm; 34.98 ± 6.92 kg). All children were in pubertal stage 1 according to Tanner, except two 11 year old girls, who were in stage 2. Analyses of variance showed that there was a significant main effect of the regression equations on %BF values for boys ($F = 50.6$; $p < 0.001$) and girls ($F = 55.4$; $p < 0.001$). The post hoc comparisons indicated significant differences between almost all equations for boys and girls. Mean %BF calculated using different SF and BIA equations ranged from 11.0 ± 3.9 to 16.7 ± 4.0% for boys and from 14.7 ± 4.3 to 19.6 ± 5.0% for girls. In addition, the calculated sums of SF were highly correlated ($p < 0.001$) with the Slaughter *et al.* and Boileau *et al.* age-specific SF regression equations in boys and girls ($r = 0.93–0.97$). The correlation coefficients with other SF prediction formulas were also significant but somewhat lower (boys: $r = 0.44–0.57$; girls: $r = 0.63–0.73$). Our results demonstrate the need for caution when using this field technique to estimate %BF of prepubertal children. According to the results of this investigation, it is suggested that the most appropriate regression equations for use with this group of children are the Boileau *et al.* SF formulas. In addition, the calculated sums of SF, especially the sum of two SF (triceps and

calf) could be used to monitor changes in body composition in 9–11 year old children.

Key words: skinfold thickness, regression equations, prepubescent children

INTRODUCTION

The relative amount of body fat has important health and health-related fitness implications for children [3, 8]. Several field methods have been recommended to monitor changes in body composition of children. One of the more practical methods for predicting percent body fat (%BF) is the measurement of selected skinfold (SF) thicknesses. All SF prediction equations in general use are based on two-component model of body composition [3, 8]. In this case, human body is divided into fat and fat-free tissue and the assumption is made that the fat-free mass (FFM) has a constant composition in terms of water, protein and mineral [8].

It is well recognized that SF thickness equations for body composition assessment are very population-specific and estimates of body fat obtained from different regression equations may vary greatly among individuals [3, 8]. Over a hundred of different SF equations are currently available [9]. For example, Norgan and Ferro-Luzzi [10] compared five generalized equations in men and found that they all differed significantly from one another. Thus, there is a reason for concern when the SF equations are applied inappropriately in a homogeneous group of children. Furthermore, only few SF regression equations are specifically developed for children [1, 8, 13]. Unfortunately, these regression equations are developed on a heterogeneous sample of children ranging in sexual maturation from pre- to post-pubescent [1, 8, 13].

The purpose of this investigation was to study whether meaningful differences in estimated %BF occur when %BF values are calculated using different SF thickness equations found in the literature. In addition, the sums of different SF were computed to assess whether they could be used in the estimation of body fat. A homogeneous sample of 9 to 11 year old boys and girls was cho-

sen to assess the applicability of different regression equations for this age-group of children.

MATERIAL AND METHODS

Subjects

A total of 212 nine to 11 year old prepubertal children (107 boys and 105 girls) participated in this investigation. The children had no known chronic diseases and none of them were heavily involved in any sports (as assessed through self-reported questionnaire). Pubertal stages were determined according to the criteria of Tanner [15]. All children were in the Tanner stage 1 (except two 11 year old girls, who were in stage 2). The children were classified prepubertal as pubic hair and genitalia (boys), and breast (girls) ratings were both scored as stage 1. Informed parental consent was obtained prior to the children's participation in the experiment.

Anthropometric Measurements

The heights (Martin's metal anthropometer) and weights (medical balance scale) of the subjects were measured to the nearest 0.1 cm and 0.05 kg, respectively. The body mass index (BMI) was calculated as mass (kg) divided by height (m) squared. All anthropometric measurements were taken on the same day following a 12-h fast with an empty bladder between 8.00 to 10.00 a.m. Skinfold thickness measurements were made in triplicate using metal Holtain calipers (Crymmych, UK) and the mean of the three measurements at each site was used. The measurements were taken on the right side of the body with an accuracy of 0.2 cm at the triceps, biceps, subscapular, midaxillary, chest, suprailiac, supraspinale, abdominal, front thigh and medial calf sites as recommended by the O-Scale physique assessment system [16]. All measurements were done by the same investigator to minimize technical variability. The anthropometrist had previously shown test-retest reliability of $r > 0.90$ [7]. Body density

was calculated using Jackson and Pollock [4], and Jackson *et al.* [5] generalized equations developed on adult samples. Percent body fat was then calculated using Lohman's [8] age-specific constants. For comparison, %BF was calculated from body density using the adult equation developed by Siri [12]. Percent body fat was also derived using Slaughter *et al.* [13] and Boileau *et al.* [1] age-specific regression equations. In addition, the following sums of SF thicknesses were calculated: 1) sum of triceps and calf (Sum2) [13]; 2) sum of chest, abdominal and thigh (Sum3) [1, 13]; 3) sum of triceps, biceps, subscapular, suprailiac and calf (Sum5) [14]; 4) sum of triceps, subscapular, midaxillary, chest, suprailiac, abdominal and mid-thigh (Sum7) [4, 5].

Data Analysis

Descriptive statistics (mean, standard deviation [SD]) for each of the dependent variables were determined and independent t-tests were used to indicate sex differences. One way analyses of variance (ANOVAs) and paired t-tests post hoc procedures were used to examine differences for each regression equation among the mean estimated %BF values. The Pearson Product Moment Correlation analysis was used to examine the relationship among SF measurements of body composition. The level of $p < 0.05$ was considered significant.

RESULTS

The descriptive characteristics of the subjects are presented in Table 1. The boys and girls were not significantly different in terms of age, height, mass and BMI. The mean SF thicknesses as well as Sum2, Sum3, Sum5 and Sum7 were significantly lower ($p < 0.05$) in boys than girls (Table 1).

Table 2 presents the mean values of %BF for boys and girls as estimated using four SF regression equations. Analyses of variance showed that there was a significant main effect of the regression

equations on %BF values for boys ($F = 50.6$; $p < 0.001$) and girls ($F = 55.4$; $p < 0.001$). The post hoc comparisons indicated that with a few exceptions, almost all regression equations for boys and girls yielded significantly different results ($p < 0.05$). Mean %BF calculated using different SF thickness equations ranged from 11.0 ± 3.9 to $16.7 \pm 4.0\%$ and from 14.7 ± 4.3 to $19.6 \pm 5.0\%$ in boys and girls, respectively.

Table 1. Mean (±SD) age, height, weight and anthropometric characteristics of prepubertal boys and girls.

Variable	Boys (n = 107)			Girls (n = 105)		
	Mean ± SD	Minimum	Maximum	Mean ± SD	Minimum	Maximum
Age (yrs)	10.30±0.62	9.0	12.0	10.18±0.50	9.0	12.0
Height (cm)	144.09±5.83	132.2	157.4	143.30±7.29	125.7	160.5
Weight (kg)	35.37±5.36	25.5	53.0	34.98±6.92	23.0	63.0
BMI ($kg \cdot m^2$)	16.93±1.89	13.5	23.4	16.86±2.19	12.7	24.6
Skinfold thicknesses (mm):						
Triceps	8.67±2.91	4.0	20.2	10.54±3.76*	5.0	23.3
Biceps	4.43±2.37	2.0	16.2	5.97±2.91*	2.2	18.0
Subscapular	6.00±2.60	3.0	19.2	7.31±3.64*	4.0	30.2
Midaxillary	4.63±2.20	3.0	15.2	5.84±3.66*	2.2	29.0
Chest	4.70±2.60	2.2	16.2	5.83±3.03*	2.0	17.2
Suprailiac	5.34±2.86	2.6	17.6	6.65±3.86*	3.0	27.0
Supraspinale	5.56±3.67	2.6	21.2	6.82±4.32*	3.0	25.6
Abdominal	7.70±4.88	3.0	27.6	10.23±6.19*	3.0	38.0
Thigh	14.19±4.83	6.6	34.6	17.97±5.93*	7.2	42.0
Calf	10.41±4.03	3.0	23.2	13.23±4.79*	5.6	34.6
Sum2	19.09±6.66	9.0	43.6	23.84±8.22*	11.6	18.0
Sum3	26.49±11.64	12.0	78.6	33.53±14.23*	13.0	97.2
Sum5	34.83±13.55	18.2	91.6	43.57±17.42*	22.0	133.2
Sum7	51.16±21.56	25.2	149.2	63.93±27.31*	30.0	206.0

BMI, body mass index; Sum2 (sum of triceps and calf skinfolds); Sum3 (sum of chest, abdominal and mid-thigh skinfolds); Sum5 (sum of triceps, biceps, subscapular, suprailiac and calf); Sum7 (sum of triceps, subscapular, chest, midaxillary, suprailiac, abdominal and mid-thigh skinfolds).
* Significantly different from boys; $p < 0.05$.

Percent body fat in boys calculated using the Boileau *et al.* [1] SF equation did not differ ($p > 0.05$) from those obtained using the Jackson *et al.* [5] SF regression equation and Lohman [8] age-adjusted constants (Table 2). For girls, the mean difference be-

tween %BF obtained using the Siri [12] equation and Slaughter *et al.* [13] SF age-specific equation did not differ significantly.

Table 2. Mean (±SD) and ranges of percentage body fat (%BF) estimated from skinfolds (SF) regression equations.

Dependent Variable	Boys (n = 107)	Girls (n = 105)
SF (Lohman)[1]	11.0±3.9[a]	14.7±4.3
	(5.7–25.6)	(6.3–30.6)
SF (Siri)[2]	16.7±4.0	19.7±4.7[b]
	(8.3–30.6)	(8.3–35.9)
SF (Slaughter)[3]	15.0±4.9	19.6±5.0[b]
	(7.6–33.1)	(12.2–40.5)
SF (Boileau)[4]	12.5±4.6[a]	17.3±5.2
	(4.9–30.3)	(8.8–35.5)

[1]SF technique using Jackson and Pollock [4], and Jackson *et al.* [5] regression equations for boys and girls, respectively, to calculate body density (BD) and Lohman's [8] age-specific constants to calculate %BF.

[2]SF technique using Jackson and Pollock [4], and Jackson *et al.* [5] regression equations for boys and girls, respectively, to calculate body density (BD) and the Siri [12] equation to calculate %BF.

[3]SF technique using the Slaughter *et al.* [13] age-specific equation to calculate %BF.

[4]SF technique using the Boileau *et al.* [1] age-specific equation to calculate %BF.

[a,b]Means (±SD) in the same column with the same superscript are not significantly different ($p>0.05$). All other means (±SD) are significantly different ($p<0.05$).

The correlations between Sum2, Sum3, Sum5 and Sum7 SF thickness measurements and the %BF derived from four different regression equations in boys and girls are presented in Table 3. It appeared that all sums of SF were highly correlated ($p < 0.001$) with the Slaughter *et al.* [13] and Boileau *et al.* [1] age-specific SF regression equations in boys ($r = 0.93–0.99$) and girls ($r = 0.92–0.97$). The correlation coefficients with other SF prediction formulas were also significant ($p < 0.001$) but somewhat lower (boys: $r = 0.44–0.57$; girls: $r = 0.63–0.73$).

Table 3. Pearson Product Moment intercorrelations between percent body fat (%BF) values estimated from skinfolds (SF) regression equations and the sums of SF thickness measurements in prepubertal boys and girls (in brackets).

	Sum2#	Sum3	Sum5	Sum7
SF (Lohman)*	0.57	0.54	0.49	0.46
	(0.72)	(0.73)	(0.70)	(0.71)
SF (Siri)	0.50	0.50	0.47	0.44
	(0.66)	(0.67)	(0.63)	(0.65)
SF (Slaughter)	0.93	0.96	0.93	0.99
	(0.94)	(0.97)	(0.93)	(0.99)
SF (Boileau)	0.96	0.96	0.94	0.93
	(0.94)	(0.95)	(0.92)	(0.93)

All correlations are statistically significant ($p < 0.001$).
#Abbreviations used are the same as in Table 1.
*Abbreviations used are the same as in Table 2.

DISCUSSION

The results of this investigation demonstrated that great variation existed among %BF values calculated using different SF regression equations in prepubescent boys and girls. There was a significant main effect on the mean %BF values between the regression equations in both groups of subjects (Table 2). Furthermore, there was much more variation in the %BF values in the prepubescent girls than the boys, which is consistent with available data [6].

The accuracy of %BF estimation in children depends on several factors. According to the results of this study and that of others [3, 6, 17] a key factor appears to be the selection of an appropriate prediction equation. For example, when examing the difference between %BF values in prepubescent boys and girls using the SF technique of Jackson and Pollock [4], and Jackson *et al.* [5], the Siri [12] formula yielded significantly higher %BF values in comparison with those using Lohman's [8] age-specific constants (Table 2). This would suggest that the age-adjusted constant of Lohman [8] formula compensated the chemical immaturity in pre-

pubescent boys and girls, as prior to sexual maturation children have more water and less bone mineral content than adults [3, 8]. This means that the density of fat free mass (FFM) in prepubertal children is lower than the adult value of 1.1 $g.ml^{-1}$ [8, 13]. Thus, the previously established adult SF equations used in this study [i.e., 4, 5] appear to be inappropriate for use in prepubescent boys and girls because they systematically overestimate %BF [8, 13].

Percent body fat calculated from the age-adjusted SF equations of Slaughter *et al.* [13] and Boileau *et al.* [1] in boys was significantly lower than the %BF values obtained using the adult equation of Siri [12]. In contrast, the %BF values using the Siri [12] and Slaughter *et al.* [13] equations were similar ($p > 0.05$) in girls. These results are in accordance with Janz *et al.* [6] study, who reported that the triceps and calf SF formula of Slaughter *et al.* [13] tended to systematically overpredict %BF in girls.

According to the results of our study, the sum of SF thicknesses without conversion to %BF is another portable method and could also be used to track changes in body composition in prepubescent children. In accordance with our results, Gutin *et al.* [2] also found a high correlation between the sum of seven SF measurements and the %BF derived from the two SF equation of Slaughter *et al.* [13] in 9 to 11 year old children ($r = 0.97$). As all computed sums of SF thicknesses were highly related to %BF in this investigation (Table 3), it is suggested that the computation of Sum2 (i.e., triceps and calf) is a simple way to monitor changes in body fat of prepubescent boys and girls.

The comparison of regression equations to estimate body fat content in prepubescent boys and girls revealed the need for a "gold standard" against which the different formulas could be evaluated to find the most appropriate. However, such a "gold standard" may not exist [17]. The use of hydrodensitometry as a criterion method for children is limited because of the changes in the relative water and bone mineral content of the FFM that occur during growth and development [3, 8]. Furthermore, many children may have difficulties with the breathing maneuver involved in the determination of underwater weight [11]. The procedure of total submergence also presents difficulties for children who are inexperienced or highly anxious in water. Thus, the limitation in our

study was the fact that we did not have a "gold standard" against which the different methods and regression equations could be evaluated to assess which was the most appropriate.

In summary, the results of this study clearly indicate the importance of selecting a regression equation that is appropriate for the homogeneous group of children. Among the regression equations used in this investigation, we feel that the most appropriate ones for use are the Boileau *et al.* [1] SF prediction formulas for boys and girls. In addition, the sum of two SF (triceps and calf) thickness is also a simple and reasonable predictor of changes in body composition among prepubescent children.

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