THE LINKS BETWEEN ADOLESCENT BIOLOGICAL MATURITY, PHYSICAL ACTIVITY AND FAT MASS DEVELOPMENT, AND SUBSEQUENT CARDIOMETABOLIC RISK IN YOUNG ADULTHOOD

A Thesis Submitted to the College of

Graduate Studies and Research

in Partial Fulfillment of the Requirements

for the Degree of Doctorate of Philosophy

in the College of Kinesiology

University of Saskatchewan

Saskatoon

By

Lauren B Sherar

Permission to Use

In presenting this thesis in partial fulfillment of the requirements for a Postgraduate degree from the University of Saskatchewan, I agree that the Libraries of this University may make it freely available for inspection. I further agree that permission for copying of this thesis in any manner, in whole or in part, for scholarly purposes may be granted by the professor or professors who supervised my thesis work or, in their absence, by the Head of the Department or the Dean of the College in which my thesis work was done. It is understood that any copying or publication or use of this thesis or parts thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of Saskatchewan in any scholarly use which may be made of any material in my thesis. Requests for permission to copy or to make other use of material in this thesis in whole or part should be addressed to:

Dean of the College of Kinesiology

University of Saskatchewan

87 Campus Drive

Saskatoon, Saskatchewan

S7N 5B2

Abstract

The metabolic syndrome has become a major public health challenge world-wide and, at least in the industrialized world, the prevalence of the metabolic syndrome is increasing. There is evidence to show that biological and lifestyle risk factors for metabolic syndrome are present in adolescence, which suggests that the antecedents of the disease may lie in early life. The period of adolescence is characterized by a decline in physical activity (PA; lack of PA is a lifestyle risk factor for metabolic syndrome) and an increase in fat mass deposition (a biological risk factor for metabolic syndrome). Therefore, investigating how the development of these two variables relates to adult cardiometabolic risk is important to fuel early intervention. A factor which has the potential to influence these two risk factors, and thus ultimately the metabolic syndrome, is the timing of biological maturity (i.e. whether an individual is early, average or late maturing when compared to peers of the same age). The influence of biological maturity has largely been overlooked in previous research; therefore, the general objective of this thesis was to investigate the associations between biological maturity, adolescent PA and fat mass development, and young adult cardiometabolic risk. Three studies were necessary to realize this objective, and together help to elucidate the role of biological maturity in the adolescent decline in physical activity, fat development, and the development of adult metabolic syndrome. Ultimately, this information will aid in the development and implementation of interventions to decrease prevalence of metabolic syndrome.

Study 1: The purpose of study 1 was to investigate whether observed gender differences in objectively measured PA in children (8 to 13 years) are confounded by biological maturity differences. **Methods:** Four hundred and one children (194 boys and 207 girls) volunteered for this study. An Actigraph accelerometer was used to obtain 7 consecutive days of minute-by-minute PA data on each participant. Minutes of moderate to vigorous PA per day (MVPA), continuous minutes of MVPA per day (CMVPA), and minutes of vigorous PA per day (VPA) were derived from the accelerometer data. Age at peak height velocity (APHV), an indicator of somatic maturity, was predicted and individuals aligned by this biological age (years from APHV). Gender differences in the PA variables were analyzed using a two-way (gender X age) ANOVA. **Results:** Levels of PA decreased with increasing chronological ages in both genders ($p<0.05$). When aligned on chronological age, boys had a higher MVPA at 10 through 13 years, a higher CMVPA at 9 through 12 years, and a higher VPA at 9 though 13 years ($p<0.05$). When aligned on biological age, PA declined with increasing maturity ($p<0.05$); however gender differences between biological age groups disappeared. **Conclusion:** The observed age-related decline in adolescent boys and girls PA is antithetical to public health goals and as such is an important area of research. In order to fully understand gender disparities in PA, consideration must be given to the confounding effects of biological maturity.

Study 2: Understanding the influence of biological age (BA) on the decline in PA would better inform researchers about the effective timing of intervention. The purpose of study 2 was to describe the PA levels and perceived barriers to PA of adolescent girls grouped by school grade and biological maturity status (i.e., early or late maturing) within grades.

Methods: 221 girls (aged 8-16 years; grades 4-10) wore an Actical accelerometer for 7 days and then completed a semi-structured, open ended questionnaire on perceived barriers to PA over the 7 day period. Predicted APHV and recalled age at menarche were used to assess maturity among the elementary and high school girls, respectively. Maturity and grade group differences in PA were assessed using MANCOVA and independent sample t-test, and barriers to PA using chi squared statistics. **Results:** Daily minutes spent in MVPA decreased by 40% between grades 4 to 10. Within grade groupings, no differences in PA were found between early and late maturing girls (p>0.05). Grades 4-6 participants cited more interpersonal (i.e., social) barriers. Grades 9-10 participants cited more institutional barriers to PA, primarily revolving around the institution of school. No differences were found in types of barriers reported between early and late maturing girls. **Conclusion:** Since PA and types of perceived barriers to PA were dependent on grade, future research should work to identify the most salient (i.e., frequent and limiting) barriers to PA by chronological age in youth.

Study 3: Although the metabolic syndrome is thought to be mainly a consequence of obesity, the mechanisms underpinning its development are not that well understood. The purpose of study 3 was to examine total body fat mass (FM), trunk FM and PA developmental trajectories (aligned to BA; years from APHV) of individuals categorized as low and high for cardiometabolic risk at 26 years, while investigating biological and lifestyle risk factors. **Methods:** The sample were 55 males and 76 females from the Saskatchewan Pediatric Bone Mineral Accrual Study (1991-2007) who were assessed from childhood to young adulthood and had a measure of cardiometabolic risk at young

adulthood (26.0 $\pm$ 2.3 yrs). Height was measured biannually. Total body FM and trunk FM was assessed annually by dual energy-X-ray absorptiometry. PA and dietary intake was evaluated two to three times annually using surveys. Individuals were grouped into maturity status groups (early, average or late) depending on their APHV. Two composite cardiometabolic risk scores were calculated for males and females separately. The first was derived for a sub-sample (N=48) by summing the standardized residuals of inverted high-density lipoprotein cholesterol, homeostasis model assessment for insulin resistance, mean arterial pressure (MAP) and fasting triglyceride levels. A second score was derived for the whole sample by summing the standardized residuals for MAP. Scores for both samples were regressed on to age and adult smoking status. High and low cardiometabolic risk groups were determined based on a sex- specific median split of risk scores. Data were analyzed using random effects models. Models were built in a stepwise procedure with predictor variables added one at a time, using the log likelihood ratio statistic to determine if one model was a significant improvement over the previous one. **Results:** The final model indicated that once the independent effects of maturity (years from APHV) and height were controlled, the high risk group males and females had significantly (p<0.05) greater total body FM and trunk FM development at all ages. No association was found between young adult cardiometabolic risk and development of PA. Furthermore, in general, timing of biological maturity was not associated with development of PA or FM. **Conclusion:** Young adults at higher cardiometabolic risk have greater body fat as early as 8 years of age, which lends support to early intervention.

General Conclusions: Adolescence has been highlighted as a critical period for the development of adult disease, such as the metabolic syndrome. Results from this thesis support this contention by showing a decrease in PA (by both chronological and biological age) in males and females across adolescence. It further showed that an increase in total and central fatness during adolescence may be critical for the development of the metabolic syndrome in adulthood. Timing of biological maturity, in general, was not shown to have an independent impact on adolescent or young adult PA, adolescent perceived barriers to PA, fat mass development, or young adult cardiometabolic risk. However, further research is required before definitive conclusions can be made about the short and long term impacts of timing of biological maturity on health.

Acknowledgements

Foremost, I would like to thank my advisor, Dr. Adam Baxter-Jones, for his mentorship and guidance throughout my graduate studies. It has been a privilege to work with him. I also could not have maintained an active academic career throughout my PhD without his unwavering support and flexibility regarding my ever changing family situation. Babysitting my newborn at a conference goes beyond the duties expected of a supervisor!

I would like to thank my committee members: Drs. Robert Faulkner, Nazeem Muhajarine, Phil Chilibeck and Susanna Martin for their contributions and detailed proofing of my thesis. I would further like to thank Drs. Louise Humbert and Nancy Gyurcsik for their wisdom, time, and input into data collection for my second study; and Dale Esliger for his extensive advice on accelerometry data collection, cleaning and reduction. I would also like to express appreciation for the expert advice supplied by Dr. Joey Eisenmann regarding data collection and analysis of study 3. I am very thankful to Marta Erlandson and Stefan Jackowski for the many errands they, ever so obligingly, ran for me when I moved back to the UK. I would also like to thank Dr. Mark Tremblay for allowing me to use his data for study 1 and to the Saskatoon Public School Board for their excitement and support of the research presented in study 2. The biggest recognition and respect goes to the participants of the Saskatchewan Pediatric Bone Mineral Study (PBMAS), who have remained dedicated to the study for 25 years! Thank you!

Lastly, I would like to acknowledge CPHR and the College of Kinesiology for the funding support I received throughout the duration of my Ph.D. program.

Dedication

My thesis is dedicated to my children, Olivia, Jack and the soon-to-be new arrival; to my husband Dale; and, to my parents Jane and Terry. To my children, Olivia and Jack, you have made being a Mother and conducting my PhD studies a joy. To my husband, Dale, thank you for your love, support and your academic advice. You constantly help me to see 'the bigger picture'. Lastly, to Terry and Jane, thank you for your love and encouragement over my extended education and for helping care for my children. I could not have done it without you!

Table of Contents

Permission to Use i

Abstract ii

Acknowledgements vii

Dedication viii

List of Figures xviii

Glossary xx

List of Abbreviations xxii

Chapter 1: Introduction and review of literature 1

1.1 Introduction 1

1.2 Review of literature 8

1.2.1 Adolescence 8

1.2.3 Body dimensions and proportions 10

1.2.4 Stature 13

1.2.5 Body Mass 16

1.2.6 Fat mass 17

1.2.7 Biological maturity 19

1.2.7.1 Maturity indicators 20

1.2.7.1.1 Age at peak height velocity 20

1.2.7.1.2 Menarcheal status 22

1.2.7.1.3 Other indicators of biological maturity 23

1.2.7.2 Sequence of pubertal events 24

1.2.7.2.1 Relationship between indicators 27
1.2.7.3 Maturity associated variation in body size and composition 27
1.2.7.3.1 Stature and body mass 27
1.2.7.3.2 Body composition 29
1.2.8 Adolescent physical activity 30
1.2.9 Adolescence as a critical period for adult health 30
1.2.9.1 Tracking of fat mass from adolescence to adulthood 33
1.2.9.2 Tracking of physical activity from adolescence to adulthood 34
1.2.10 Metabolic Syndrome 36
1.2.10.1 A composite cardiometabolic risk score 38
1.2.10.2 Prevalence of the metabolic syndrome 38
1.2.10.3 Risk factors for metabolic syndrome 40
1.2.10.4 Adolescent antecedents of metabolic syndrome. 41
1.2.11 PA and risk for metabolic syndrome 41
1.2.11.1 Youth PA and youth risk for metabolic syndrome 41
1.2.11.2 Adult PA and risk for metabolic syndrome 42
1.2.11.3 Youth physical activity and adult risk for metabolic syndrome 43
1.2.12 Obesity and risk for metabolic syndrome 45
1.2.12.2 Abdominal obesity 46
1.2.12.2.1 Pathophysiology of abdominal obesity and metabolic syndrome risk. 48
1.2.13 Biological maturity and risk for metabolic syndrome 50
1.2.13.1 Biological maturity and the adolescent: Two hypotheses 51
1.2.13.2 Biological maturity and barriers to PA 53

1.2.13.3 Timing of biological maturity and physical activity 53
1.2.13.4 Timing of biological maturity and overweight/obesity 55
1.2.13.4.1 Biological maturity and regional fat deposition 58
1.2.13.5 Biological maturity and the metabolic syndrome 59
1.2.14 Other metabolic syndrome risk factors 60
1.2.16 Aims/Hypotheses 63
1.2.16.1 Study 1: 63
1.2.16.2 Study 2: 63
1.2.16.3 Study 3: 63
Chapter 2: Study 1: 65
2.1 Introduction 65
2.2 Methods 67
2.2.1 Participants 67
2.2.2 Chronological age 68
2.2.3 Anthropometry 68
2.2.4 Physical Maturity (Biological age) 69
2.2.5 Physical Activity 69
2.3 Results 71
2.4 Discussion 75
2.5 Conclusion 79
2.6 Acknowledgements 79
Chapter 3: Study 2 80
3.1 Introduction 80

3.1.1 Biological age 81
3.1.2 Perceived barriers to physical activity 81
3.2 Methods 84
3.2.1 Participants 84
3.2.2 Anthropometry 84
3.2.3 Physical Maturity (Biological age) 84
3.2.4 Physical Activity 86
3.2.5 Barriers to physical activity 87
3.2.6 Data analyses 88
3.3 Results 89
3.3.1 Physical activity by grade groupings 89
3.3.2 Physical activity by biological maturity groupings 90
3.3.3 Barriers to physical activity by grade groupings 91
3.3.4 Barrier categories by grade groupings 92
3.3.5 Barriers to physical activity by biological maturity groupings 96
3.3.5.1 Barrier categories by biological maturity groupings 96
3.4 Discussion 97
3.4.1 Physical activity by grade and maturity groupings 97
3.4.2 Barriers to physical activity by CA and BA 99
3.5 Considerations 102
3.6 Conclusion 103
3.7 Acknowledgements 104
Chapter 4: Study 3 105

4.1 Introduction 105
4.2 Methods 108
4.2.1 Participants 108
4.2.2 Chronological age 111
4.2.3 Anthropometry 111
4.2.4 Maturational Assessment 111
4.2.5 Body Composition 112
4.2.6 Assessment of Physical Activity 113
4.2.7 Nutritional Assessment 113
4.2.8 Adult smoking status 114
4.2.9 Markers of Metabolic Syndrome 114
4.10 Cardiometabolic risk score 115
4.2.11 Data Analysis 116
4.2.11.1 Modeling strategy 117
4.3 Results 117
4.3.1 Model 1: Total body Fat Mass (TBFM) 122
4.3.1.1 Males 122
4.3.1.2 Females 123
4.3.2 Model 2: Trunk FM 123
4.3.2.1 Males 123
4.3.2.2 Females 123
4.3.3 Model 3: Physical Activity 124
4.3.3.1 Males 124

4.3.3.2 Females 124
4.4 Discussion 128
4.4.1 Fat Mass Development 129
4.4.2 Lifestyle Variables 129
4.4.3 Biological Maturity 131
4.5 Conclusion 134
4.6 Acknowledgements 134
Chapter 5: General Discussion 136
5.1. Summary of major findings 137
5.2 The importance of controlling for biological age 139
5.3 Timing of biological maturity and fat mass development 140
5.3.1 Females 140
5.3.1.1 Controlling for biological maturity 140
5.3.1.2 Homogeneity of the sample 142
5.3.2 Males 143
5.4. Timing of biological maturity and physical activity 144
5.4.1 Choice of maturity indicator 145
5.4.2 Self-report versus objectively assessed biological maturity 148
5.4.3 Interacting factors 149
5.4.4 Cultural context 150
5.4.5 Chronological age/Grade influences 150
5.5 Childhood/adolescent antecedents of adult metabolic syndrome 151
5.5.1 Fat mass development and cardiometabolic risk 151

5.5.2 PA and fat mass development 153
5.5.3 PA and Cardiometabolic Risk 154
5.6 Limitations 156
5.6.1 Study 1 156
5.6.2 Study 2 156
5.6.3 Study 3 157
Chapter 6: Conclusions and future research 159
6.1. Conclusions and public health implications 159
6.2 Future research 159
Reference List 162
APPENDIX A: Certificate of Approval for Study 1 191
APPENDIX B: Certificate of Approval for Study 2 193
APPENDIX C: Certificate of Approval for Study 3 194
APPENDIX D: Participant Assent Form for Study 2 195
APPENDIX E: Parent and Guardian Consent Form for Study 2 196
APPENDIX F: Consent Form for Study 3 199
APPENDIX G: Accelerometry Methodology 202
APPENDIX H: Questionnaires and Participant Information Sheets for Study 2 207
APPENDIX I: Questionnaires and Participant Information Sheets for Study 3 225
APPENDIX J: Published Letters to the Editor-in-Chief (re. Study 1) 254
APPENDIX K: Published Manuscript from Study 1 257
APPENDIX L: Accepted Manuscript from Study 2 264

List of Tables

Table 1: Comparison of the metabolic syndrome definitions 37

Table 2: Subject characteristics by gender, chronological age (CA), and biological age (BA) 72

Table 3: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for girls in different grade groups 90

Table 4: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for early and late maturing elementary and high school girls 91

Table 5: Types of intrapersonal barriers listed by the participants 94

Table 6: The interpersonal barriers to physical activity 95

Table 7: Institutional barriers to physical activity 95

Table 8: Community barriers to physical activity 95

Table 9: Number of visits (i.e. DXA scans) for sample 1 (the sub sample) 110

Table 10: Number of visits (i.e. DXA scans) for sample 2 (whole sample) 110

Table 11: Descriptive (mean + SD) statistics for all male (N=55) and female (N=76) participants at age 26 119

Table 12: The risk score for males and females by maturity group (early, average and late) 120

Table 13: Multilevel regression models for total body fat mass development of males and females aligned by biological age 125

Table 14: Multilevel regression models for trunk fat mass development of males and females aligned by biological age 126

Table 15: Multilevel regression models for physical activity development of males and females aligned by biological age 127

Table 16: Outline of the methods used in all research examining the relationship between biological maturity and PA 147

Table 17: The intra-and inter- instrument reliability for the six shaking conditions 204

List of Figures

Figure 1: A schematic representation of an average girl's development of physical activity and fat mass from 8 to 26 years of age and how it may relate to metabolic syndrome risk 3

Figure 2: Schematic diagram showing the possible links between adolescent PA, fat mass, biological maturity and metabolic syndrome/CVD risk.. 6

Figure 3: Scammon growth curves of different parts and tissues of the body..... 12

Figure 4: Growth in height of a girl. Figure 4a shows a plot of the height for age data (distance data). Figure 4b shows the yearly increments in height (velocity curve)..... 15

Figure 5: Growth in body mass of a girl between 3 and 18 years old. Figure 5a shows a plot of the body mass for age data (distance data). Figure 5b shows the yearly increments in body mass (velocity curve).. 17

Figure 6: Growth curves for fat-free mass and fat mass and for boys and girls.. 18

Figure 7: Two boys photographed at the same chronological age (14 years).. 20

Figure 8: Average age of attainment of pubic hair (PH) stages 3-5 and peak height velocity (PHV). In boys only, axillary hair and facial hair growth and in girls only, menarche.. 26

Figure 9: Objectively measured physical activity (PA) variables (mean + SD) of boys and girls by chronological and biological ages..... 74

Figure 10: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by grade groupings.. 92

Figure 11: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the elementary school girls.. 96

Figure 12: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the high school girls. 97

Figure 13: Total body percent fat (a and b), trunk percent fat (c and d) and physical activity (e and f) by biological maturity age (years from APHV) for individuals classified as high and low for cardiometabolic risk at age 26 121

Figure 14: Mechanical set-up for reliability testing..... 203

Figure 15: Flow chart illustrating the criteria for accepting accelerometer data.. 205

Glossary

Accelerometer: A mechanical instrument that measures acceleration

Barriers to physical activity: Factors that make physical activity difficult or completely inhibit it

Biological maturation: Process of being mature, or the progress towards the mature state

Cardiometabolic Risk: Cluster of risk factors which predisposes individuals to cardiovascular disease and type 2 diabetes

Chronological age: The age of a person counted from birth by standard units, as months or years

Community barriers to physical activity: Occur between organizations, institutions, and informal networks within defined boundaries that may affect physical activity

Critical period: Periods of growth during which the antecedents of chronic degenerative disease may be initiated

Cross sectional research: Studies that are carried out at one period of time

Development: The acquisition of behavioural competence and/or differentiation and specialization of the embryo during prenatal life

Ecological Model: Focuses attention on both individual and social environmental factors

Growth: Changes in size of an individual, as a whole or in parts

Institutional barriers to physical activity: Occur within social institutions with organizational characteristics that may prevent physical activity

Interpersonal barriers to physical activity: Formal and informal social networks and support systems that may prevent physical activity

Intrapersonal barriers to physical activity: Characteristics of the individual that may prevent physical activity

Insulin resistance: The diminished ability of cells to respond to the action of insulin in transporting glucose from the bloodstream into muscle and other tissues

Lifespan perspective: An approach to studying health which considers accumulating and interacting risks that are manifest from prenatal life onward

Longitudinal research: Studies in which data are obtained on the same individual three or more times during a period of time

Metabolic Syndrome: A collection of health risks that increase the chance of developing heart disease, stroke and diabetes

Risk Factor: Something that's likely to increase the chances that a particular event (e.g. a disease) will occur

List of Abbreviations

AE	Albumin excretion
ANOVA	Analysis of variance
APHV	Age at peak height velocity
B	Breast
BA	Biological age
BP	Blood pressure
BMI	Body mass index
CA	Chronological age
CARDIA	Coronary artery risk development in young adults
CHHS	Canada heart and health survey
CMVPA	Continuous minutes of moderate to vigorous physical activity
CT	Computed tomography
CVD	Cardiovascular disease
CV_{intra}	Coefficient of variation
DBP	Diastolic blood pressure
DXA	Dual energy x-ray absorptiometry
FM	Fat mass
FPG	Fasting plasma glucose
G	Genital
GEE	Generalized estimating equation
HDL-c	High density lipoprotein cholesterol
HR	Resting heart rate
HOMA-IR	Homeostasis model assessment for insulin resistance
HPA	Hypothalamo-pituitary-adrenal
LDL-c	Low density lipoprotein cholesterol
MANCOVA	Multiple analysis of covariance
MAP	Mean arterial pressure
Mat Cat	Maturity category
MET	Metabolic equivalent
MRI	Magnetic resonance imaging
MVPA	Moderate to vigorous physical activity
NCEP ATP III	National cholesterol education program's adult treatment panel III
NEFA	Non-esterified fatty acids
NHANES	National health and nutrition examination survey
PA	Physical activity
PAI-1	Plasminogen activator inhibitor 1
PAQ-A	Physical activity questionnaire for adolescents
PAQ-AD	Physical activity questionnaire for adults
PAQ-C	Physical activity questionnaire for children
PBMAS	Pediatric bone mineral accrual study
PH	Pubic hair
PHV	Peak height velocity
Project FAB	Project fitness and bone
Q	Quartiles

SEE	Standard error of the estimate
SEM	Standard error of the mean
SD	Standard deviation
SBP	Systolic blood pressure
SPSS	Statistical package for the social sciences
TAAG	Trial of activity for adolescent girls
TBFM	Total body fat mass
TG	Triglycerides
US	United States
VPA	Vigorous physical activity
WC	Waist circumference
WHO	World Health Organization
WHR	Waist to hip ratio

Chapter 1: Introduction and review of literature

1.1 Introduction

The metabolic syndrome is defined as a clustering of cardiovascular disease (CVD) risk factors; including elevated blood pressure and triglycerides, lowered high-density lipoprotein, insulin resistance (the diminished ability of cells to respond to the action of insulin) and obesity (Eckel *et al.*, 2005). The metabolic syndrome has become a major public health challenge world-wide (Eckel *et al.*, 2005) and, at least in the industrialized world, the prevalence of the metabolic syndrome is increasing (Ford *et al.*, 2004). Based on criteria proposed by the World Health Organization (Alberti & Zimmet, 1998) and the Third Report of the National Cholesterol Education Program's Adult Treatment Panel, (NCEP 2001), an estimated 47 million individuals (≥ 20 years) in the United States have the syndrome (Ford *et al.*, 2002).

There is evidence to show that biological and lifestyle risk factors for metabolic syndrome are present in adolescence, which suggests that the antecedents of the disease may lie in early life. The period of adolescence is characterized by sexual maturation (timing of biological maturity), a decline in physical activity (PA) (lack of PA is a lifestyle risk factor for metabolic syndrome), and an increase in fat mass deposition (a biological risk factor for metabolic syndrome). Therefore, investigating how the development of these three variables relates to each other and links to adult risk for metabolic syndrome is of the utmost importance.

Although typically regarded as a middle- to late-adulthood disorder, the metabolic syndrome is now present in smaller numbers in adolescents. For example, among a sample (n=4,450) of US adolescents (12-19 years), 3.5% overall and 14.5% of

overweight adolescents had the metabolic syndrome, suggesting that nearly 1 million adolescents in the United States are currently affected (Pan & Pratt, 2008). Furthermore, one or more lifestyle (e.g. regular smoking, high dietary fat intake, high simple sugar intake, no regular PA etc.) and/or biological (e.g. high cholesterol, high triglycerides, glucose intolerance, insulin resistance, overweight etc.) risk factors for the metabolic syndrome have been observed among adolescents (Berenson *et al.*, 1998). All these provide compelling evidence to suggest that the antecedents of the metabolic syndrome exist during the growing years.

Adolescence is a volatile period where physiology is changing dramatically; sexual maturation is occurring and social and peer pressures dominate. Adolescence is also a period where healthy lifestyle often deteriorates, for example: sugar and fat intake increases (Story *et al.*, 2002), experimentation with tobacco begins (Brown *et al.*, 1996); there is a decline in PA (Dietz, 1994; Strauss *et al.*, 2001; Trost *et al.*, 2002), and an increase in fat deposition (especially in females). Figure 1 shows the development of PA and fat mass of a 'normal' female over adolescence and young adulthood. The grey bar shows a hypothetical 'metabolic syndrome risk area'. Although the decline in habitual PA and the increase in fat deposition from 8 to 16 years of age is normal and is, to a certain extent, 'to be expected', it is possible that an individual will enter into the 'risk area' if there is a) high fat mass and/or low PA upon entry into adolescence and/or b) a steep decline in PA and/or incline in fat mass deposition during adolescence. A factor which may have the potential to 'shift' these lines of development into the 'risk area' is the timing of biological maturity (i.e. puberty).

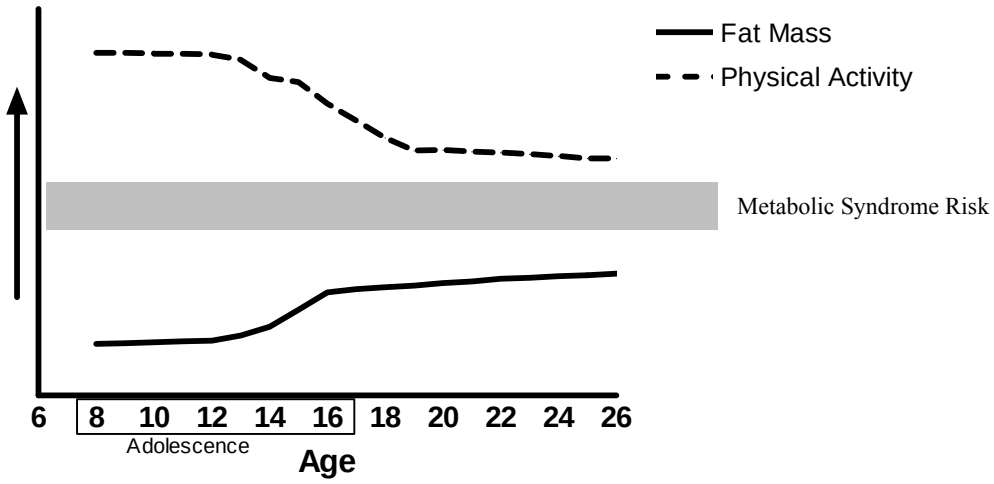


Figure 1: A schematic representation of an average girl's development of physical activity and fat mass from 8 to 26 years of age and how it may relate to metabolic syndrome risk

Puberty occurs during adolescence and is the process by which sexual maturation occurs and reproductive capacity is attained (Tanner, 1962). Puberty in girls usually starts with the development of the breasts at about 11 years of age. However, there is wide variation in the timing of normal puberty, with the onset varying by as much as 4 to 5 years among normal healthy boys and girls (Tanner, 1962). When considering the impact of pubertal development on PA, fat mass and general risk for metabolic syndrome, the *timing* of pubertal or biological maturity may be of vital importance. Timing of biological maturity refers to an individual's biological status when compared to peers of the same chronological age (CA) (Malina *et al.*, 2004). Using a classification of timing of pubertal development related to CA, an adolescent can be referred to as early, average, or late maturing for his/her CA. Biological maturity likely influences the behaviours of an

adolescent, including participation in PA. Thus it is imperative that researchers investigate the links between CA, biological maturity and PA.

When a child matures and develops secondary sexual characteristics at an earlier age than his or her peer group, there are clearly recognizable differences in physical appearance. Although these may be "normal," an early maturing adolescent may be more sensitive to these physical changes. In fact, previous research has shown that early biological maturation is associated with negative psychological well-being, especially among girls (Ge *et al.*, 1996; Graber *et al.*, 1997; Graber *et al.*, 1999; Kaltiala-Heino *et al.*, 2003; Laitinen-Krispijn *et al.*, 1999; Motl *et al.*, 2004; Ruble & Brooks-Gunn, 1982; Siegel *et al.*, 1999). These maturity related influences on psychological well-being have been shown to be related to the adoption of unhealthy behaviours such as smoking, alcohol, and substance abuse (Costello *et al.*, 2007; Deardorff *et al.*, 2005 ; Dick *et al.*, 2000; Graber *et al.*, 2004; Lanza & Collins, 2002; Michaud *et al.*, 2006; Tschann *et al.*, 1994; Waylen & Wolke, 2004). Although it is intuitive that early maturity is associated with adolescent disengagement from PA, thus likely increasing the risk of metabolic syndrome, an examination of the direct relationship between timing (i.e. if an individual is early, average or late maturing) of biological maturity, CA and participation in PA has been largely overlooked.

The relationship between timing of biological maturity and adolescent fat mass development is fairly well researched, with the majority of studies showing that early maturing girls have a greater body mass index (BMI; body mass (kg)/height $(m)^2$) and sum of skinfold measurements than average or late maturing girls (Biro *et al.*, 2001; Himes *et al.*, 2004) of the same CA. To my knowledge, only two longitudinal studies

have looked at the long term impact of early maturity on BMI and sum of skinfolds development. Results from the Amsterdam Growth and Health Study (1977-1991) showed that early maturing boys' BMI was significantly higher than for late maturing boys at all ages between 13 and 27 years (van Lenthe *et al.*, 1996a). Likewise, early maturing girls had greater BMI and sum of skinfold thickness than late maturing girls. In the Leuven Growth Study of Belgian Boys, early maturing boys had a consistently higher BMI during adolescence, but not at age 30 (Beunen *et al.*, 1994); however, importantly, adjustment for prepubertal BMI was not performed in these two studies, and thus the independent predictive role of pubertal timing on BMI and/or fatness could not be evaluated. Furthermore, only one longitudinal study has explored the relationship between biological maturity and abdominal fat mass (a particularly atherogenic form of obesity) and showed that among 579 males, age at maximum adolescent growth spurt in height (age at peak height velocity; APHV), independent of prepubertal BMI, was an independent negative predictor of central, but not peripheral, fat mass (Kindblom *et al.*, 2006). Thus, there is a need to investigate the relationship between timing of biological maturity and longitudinal development of fat mass (total and central), while assessing other lifestyle covariates (e.g. PA and dietary intake).

Figure 2 shows the possible pathways that link biological maturity and PA to metabolic syndrome/CVD risk in adulthood. The thickness of the arrows indicates how well established the relationship is; for example, an inverse relationship between adolescent PA and fat mass is fairly well established. However, the relationship among biological maturity, PA, and metabolic syndrome/CVD risk is less well known. Each relationship is posited as bi-directional, however some directions are more plausible than

others (which is represented by the size of the arrow head in figure 2). For example, it is still yet to be determined whether early biological maturity causes greater fat mass, or whether greater fat mass causes early maturity. Likewise, there is more evidence to suggest that inactivity causes metabolic syndrome/CVD risk, than the alternative (i.e. a genetic predisposition for CVD risk causing a decrease in PA). All of the aforementioned relationships are likely dependent on sex.

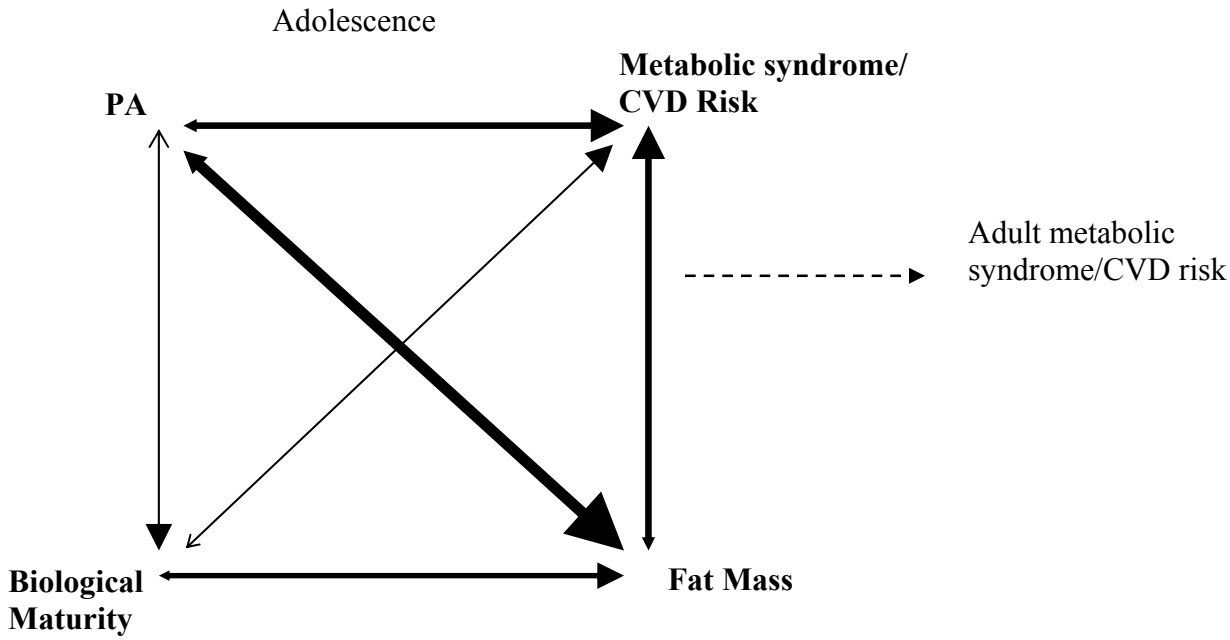


Figure 2: Schematic diagram showing the possible links between adolescent PA, fat mass, biological maturity and metabolic syndrome/CVD risk. A thick line represents strong evidence in support of the association and a large arrow head represents strong evidence in support of the direction of the association.

In summary, evidence suggests that biological maturity may be linked to PA levels and that early maturity is likely associated with an earlier decline in PA and an increase in fat mass development, two adolescent risk factors for metabolic syndrome. Furthermore, two historical prospective studies of Chinese women revealed that early

menarche, a biological maturational milestone, was associated with a higher risk of the metabolic syndrome (Feng *et al.*, 2008; Heys *et al.*, 2007). However, the relationships among early maturity, PA, fat mass (in childhood and adulthood) and early adult CVD/metabolic syndrome risk have not been studied.

The overall goal of this thesis was to investigate the associations among biological maturity, adolescent PA and fat mass development, and young adult risk for metabolic syndrome. Three studies were necessary to realize this objective. The first study explored whether the pervasive documentation of adolescent girls being less active than adolescent boys, was confounded by biological maturity differences between sexes, thereby shedding light on the potential association between biological maturity and adolescent PA. The second study explored the PA and barriers to PA (i.e. factors that make PA difficult or completely inhibit it (Bandura, 1997)) of adolescent girls by grade and maturity groups (i.e. early and late maturing). Information derived from this study will help to determine if girls of different maturity status participate in different levels of PA and, if so, what are the reasons for maturity related differences in PA. The final paper related the longitudinal development of adolescent and young adult PA and body fat to cardiometabolic risk at 26 years of age, whilst controlling for normal growth and biological development. This paper also investigated the links between the timing of maturity and subsequent cardiometabolic risk, whilst controlling for body fat and PA.

The following chapters include a general review of the literature, followed by three papers presented in a manuscript format, and a discussion of thesis findings. Study 1 has been published in a peer reviewed scientific journal (see Sherar *et al.,* 2007b; Appendix K) and study 2 is in press to be published (see Sherar *et al., In Press*; Appendix

L). Study 1 and 2, as presented in the main body of the thesis, differ slightly in content (e.g. removal of repetitive description of methodology among studies) and format from the published manuscripts.

1.2 Review of literature

1.2.1 Adolescence

The period of adolescence is often referred to as the transitional years between childhood and adulthood, and hosts momentous physiological, psychological, social and behavioural changes. During adolescence, there is a sudden increase in velocity of growth (growth spurt), second only to the rate of growth during the prenatal period (Schroeder, 1992). This acceleration of growth affects almost all parts of the body, including the long bones, vertebrae, skull and facial bones, heart, lung and viscera, and muscle mass. In addition to somatic growth, there are changes in reproductive tissues (including secondary sex characteristics), in body size and shape, in the relative proportions of muscle, fat and bone, and in a variety of physiological functions (Tanner, 1989). The term 'puberty' is often used interchangeably with adolescence. However, technically (from an auxological perspective) puberty refers to the specific period of sexual maturation, and extends from the first appearance of secondary sexual changes until the achievement of adult sexual maturity (Cameron & Demerath, 2002).

The following section outlines the basic concepts of growth, maturation and development during adolescence, and reviews some of the indicators that can be used to assess biological maturation.

1.2.2 Growth, maturation and development

The terms growth, biological maturation and development are often used synonymously. Although inter-related, the concepts have fundamental as well as semantic differences. Growth refers to changes in size of an individual, as a whole or in parts (Malina *et al.*, 2004). As children grow, they become taller and heavier, they increase their lean and fat tissues, and their organs increase in size. Changes in size are a result of three cellular processes: (1) an increase in cell number, or hyperplasia, (2) an increase in cell size, or hypertrophy, and (3) an increase in inter-cellular substances, or accretion. All three occur during growth but the predominance of one process over another varies with chronological age (CA) and the tissue involved (Malina *et al.*, 2004).

Maturation has been described as the process of being mature, or progress toward the mature (adult) state (Malina *et al.*, 2004). The process of maturing has two components, timing and tempo. The former refers to when specific maturational events occur (e.g. age when menarche is attained, age at the beginning of breast development, age at the appearance of pubic hair, or age at maximum growth in height during the adolescent growth spurt (peak height velocity; PHV)). Tempo refers to the rate at which maturation progresses (i.e. how quickly or slowly an individual passes from the initial stages of sexual maturation to the mature state). Maturation occurs in all biological systems in the body but at different rates. Furthermore, the timing and tempo of maturity vary considerably among individuals, with children of the same CA differing dramatically in their degree of biological maturity (Tanner, 1962).

Development refers to the acquisition of socio-behavioural and cognitive competence (the learning of appropriate behaviors expected by society) and is culture-

specific (Malina *et al.*, 2004). As children experience life at home, school, church, and engage in sports, recreation, and other community activities, they develop cognitively, socially, emotionally, morally, and so on. Children and adolescents learn to behave in a culturally appropriate manner. Development can also be thought of within the biological context. Here development refers to the processes of differentiation and specialization occurring during the prenatal life.

It is important to recognize that growth, maturation, and development occur simultaneously and these processes interact; however, they may not follow the same time line. A young person could be advanced in terms of social and emotional development but delayed in biological maturation, or vice versa.

1.2.3 Body dimensions and proportions

During adolescence, different parts of the body grow at different rates and different times. It has been proposed (Scammon, 1930) that all tissues and systems follow four patterns of growth: (1) neurological (e.g. brain and head), (2) genital (e.g. reproductive organs), (3) general (e.g. stature, heart size), and (4) lymphoid (e.g. lymph glands, tonsils, appendix). These patterns of growth are shown in figure 3. The data shown are relative, as size attained by each type of tissue at each age is expressed as a percentage of the total increment between birth and 20 years of age (100%).

The brain and head growth (neurological curve) shows the least growth during adolescence with the most rapid growth occurring between birth and 7 years of age. In fact, the brain and head reaches its full adult size between 8 to 10 years of age. The genital organs, which include primary sex characteristics (e.g. uterus, vagina, fallopian

tubes in females; prostate and seminal vesicles in males) and secondary sex characteristics (e.g. breasts in females, facial hair in males, and axillary and pubic hair in both sexes) shows some growth during infancy, but the most rapid development occurs during adolescence. The general curve of growth includes many tissues and systems in the body, such as skeletal tissue, the respiratory system and the digestive system to name a few. The general curve follows an 'S', or sigmoidal curve of growth. The 'S' shape reflects a rapid growth during infancy and early childhood, steady growth during mid-childhood, rapid growth during early adolescence and leveling off in late adolescence. At 10-12 years of age children are roughly 84% of their adult height. The lymphoid tissues are involved with the immunological capacities of the child and show a different growth curve from the rest of the body. There is a remarkable increase in size of the lymphoid tissue until the early adolescent years (approximately 11 to 13 years). The relative size of the tissue then steeply declines during puberty, probably a result of the up-regulation of sex hormones during this period. Thus the period of adolescence sees dynamic general, lymphoid and genital growth and development.

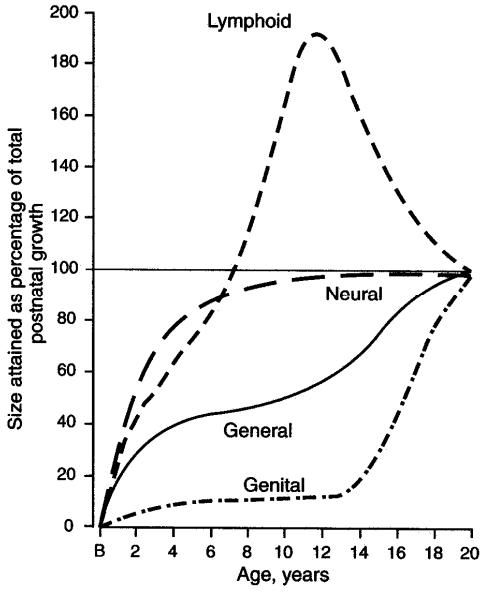


Figure 3: Scammon growth curves of different parts and tissues of the body. All curves are of size attained plotted as percentage of total gain from birth to 20 years. Size at 20 years is 100 % on the vertical scale.

Growth during childhood and adolescence occurs distal to proximal. For example, the hand and feet experience accelerated growth first, followed by the calf and the forearm, the hips and the chest, and lastly the shoulders. However, once the adolescent spurt has ended, hands and feet are a little smaller in proportion to arms, legs and stature. Most body dimensions, with the exception of subcutaneous adipose tissue and the dimensions of the head and face follow a growth pattern similar to that of stature; however, there are wide variations in the timing of growth spurts. From childhood to adolescence, the lower extremities (legs) grow faster than the upper body (trunk), which results in sitting height contributing less to stature as age progresses (Tanner, 1962). During the adolescent growth spurt the legs experience a growth spurt earlier than the trunk (Malina *et al.*, 2004). Thus, for a period during early adolescence, a youth will have

relatively long legs, but the appearance of long-leggedness disappears with the later increase in trunk length. Sex differences in leg length and sitting height are small during childhood. For a short time during the early part of adolescence, girls, on average, have a slightly longer leg length than boys. Boys' leg length exceeds girls by about 12 years of age, but boys do not catch up in sitting height until about 14 years of age (Malina *et al.*, 2004). The longer period of preadolescent growth in boys is largely responsible for the fact that men's legs are longer than women's in relation to trunk length (Malina *et al.*, 2004).

1.2.4 Stature

Standing stature is a linear measurement of the distance from the floor, or standing surface, to the top of the skull and is the most widely used indictor of somatic growth because of its relative ease in measurement. Stature varies during the course of the day, with greater readings in the morning that decrease throughout the day. Shrinkage during the day occurs because the intervertebral disks become compressed as result of weight bearing. The diurnal variation may be as much as 1cm or more (Malina *et al.*, 2004).

By linking together an individual's height data at successive ages, a distance graph is produced that describes the height achieved at any age. An example is shown in figure 4a. This type of graph has been named a height distance curve, or a height-for-age curve. Also, growth is not a linear process; individuals do not grow the same amount in each calendar year. For example, during infancy there is a relative rapid growth, steady growth during childhood, rapid growth in adolescence and slow growth as an individual

reaches maturity (sigmoidal curve). These patterns of rates of growth are better reflected when the velocity (or rate) of growth is plotted and a curve fitted. An example of a height velocity curve is shown in figure 4b. A velocity curve better reflects the child's state of growth at any particular time than does the distance curve (figure 4a). During the first year of life, infants grow at a fast rate, approximately 25cm/year. Throughout the second year of life there is growth of another 12-13 cm in stature so that by the age of two years the child has attained about 50% of adult stature (Tanner, 1962). From then on there is a steady deceleration in growth, dropping to a rate of about 5-6cm per year before the initiation of PHV (Malina *et al.*, 2004). Peak height velocity refers to the maximum rate of growth in stature during the adolescent growth period. Girls, on average, attain PHV approximately two years earlier than boys with their onset of peak height velocity occurring between 8.2 and 10.3 years. On average PHV is reached between 11.3 and 12.2 years. Corresponding ages for boys are 10.0-12.1 years (for onset) and 13.3-14.4 years (for attaining PHV) (Malina *et al.*, 2004).

Males, on average, are 13cm taller than females upon reaching their final adult height (Tanner, 1962). Up until the initiation of PHV the sex differences in height are small. Therefore, boys achieve their height advantage during the adolescent period. Specifically, on average, boys experience about 2 years more preadolescent growth, approximately 5cm/year, than girls (Malina *et al.*, 2004). This is roughly 10cm of growth that girls do not experience. Boys also achieve a slightly greater (on average 2cm) magnitude of height at PHV. Both of these growth differences cause males, on average, to have a greater adult stature. Girls stop growing in stature by about 16 years of age and boys about 18 or 19 years of age. However, these ages may be artificially low as many

growth studies stop measuring youth at 17 or 18 years of age. Limited longitudinal studies which track youth into adulthood have found that some late maturing youth continue to grow into their early- to mid-twenties (Hagg & Taranger, 1991).

These curves of growth in height (figure 4) reflect the growth patterns found in all healthy children who live in a normal environment. As mentioned, individuals will differ in absolute height of growth velocity (i.e. adult heights) and in the timing of the adolescent growth spurt; however, to reach their destined final height each individual will go through a similar pattern of growth.

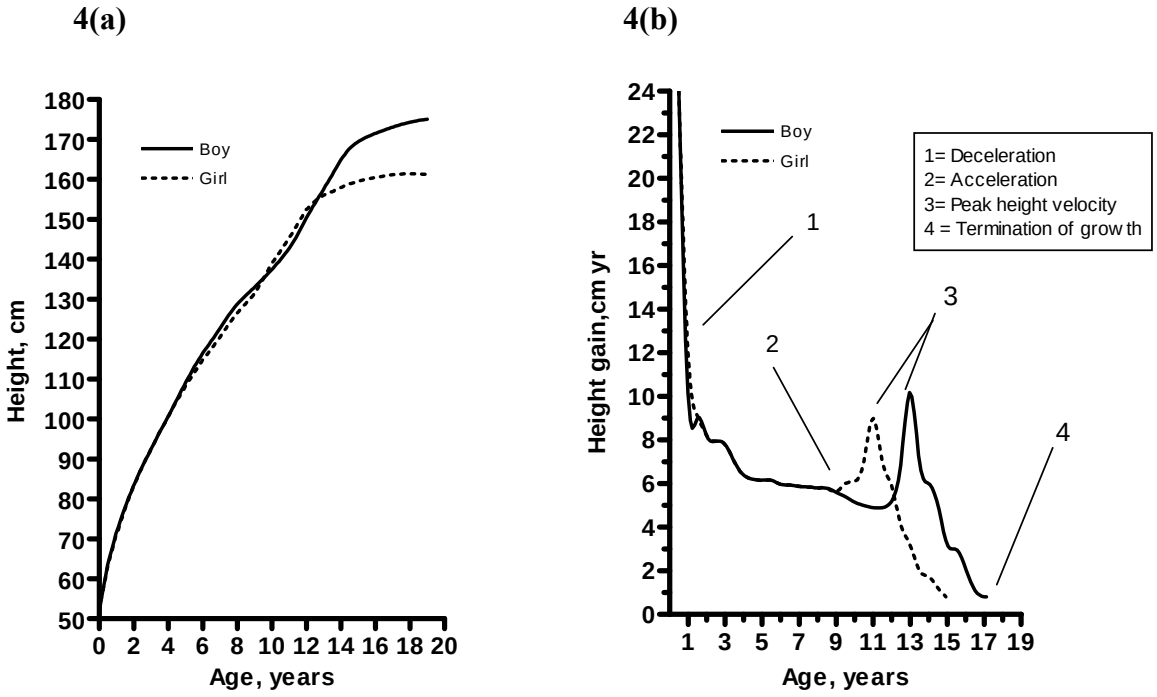


Figure 4: Growth in height of a girl. Figure 4a shows a plot of the height for age data (distance data). Figure 4b shows the yearly increments in height (velocity curve). Data from (Malina *et al.,* 2004).

1.2.5 Body Mass

Body mass is made up of a composite of tissues, including both fat and fat-free tissue, that accrue at different rates and times. Changes in body mass can thus be a result of changes in fat or fat-free mass, but also changes in body water (de-hydration or over hydration). The relative proportions and distributions of fat and fat-free components depend on age, sex, and other environmental and genetic factors. Body mass is a very sensitive and thus fluctuating measurement, in the sense that it can change from one day to another due to minor alterations in body composition.

The average distance and velocity curve for the development of body mass in males and females is shown in figure 5. As seen with the development of height (figure 4), body mass follows a four-phase growth pattern: rapid growth in infancy and early childhood, rather steady gain during mid childhood, rapid gain during adolescence, and usually a slower increase into adulthood. At the onset of adolescence there is a rapid gain in the velocity of body mass development. The precise timing of the adolescent growth spurt in body mass is generally less clear than it is for height. It has been estimated that peak velocity in body mass normally occurs 0.2-0.4 years after PHV in boys and 0.3-0.9 years after PHV in girls (Armstrong & Welsman, 1997).

Boys and girls follow the same pattern in body mass development. Before the adolescent growth spurt boys are slightly heavier than girls. Girls then experience an earlier growth spurt and thus for a short time are heavier. As soon as boys go through their adolescent growth spurt they catch up and thus become and remain heavier than girls (Malina *et al.*, 2004).

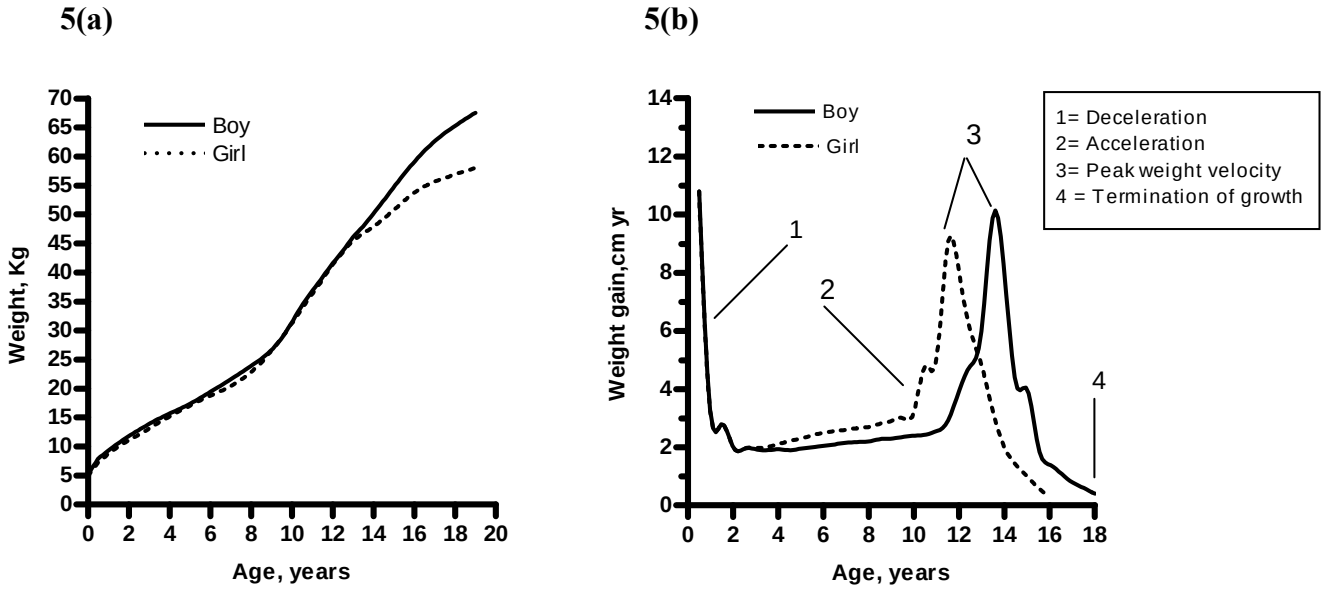


Figure 5: Growth in body mass of a girl between 3 and 18 years old. Figure 5a shows a plot of the body mass for age data (distance data). Figure 5b shows the yearly increments in body mass (velocity curve). Data from (Malina *et al.*, 2004).

1.2.6 Fat mass

Fat mass can be estimated through many different methods, such as dual-energy X-ray absorptiometry (DXA), under water weighing and sum of skinfolds. The development of total fat and fat free mass of boys and girls is shown in Figure 6. Estimated fat mass increases during the first 2-3 years of life and then shows little change up until approximately 6 years. During these years sex differences in fat mass are negligible. From 6 years onwards fat mass increases more rapidly in girls than in boys. Whereas fat mass increases during adolescence in girls, in boys fat mass reaches a plateau around the time of PHV (approximately 14 years). Subsequently, females have on average 1.5 times the fat mass of males in late adolescence and young adulthood (Malina *et al.*, 2004). In females, relative fatness (as a percentage of body mass) increases

gradually during adolescence, showing a similar trajectory to fat mass. In males percent fat mass also increases progressively until just before PHV (about 11 to 12 years) and then gradually declines reaching its lowest point at about 16 to 17 years. There is then a usual rise in percent fat mass into adulthood. The decline in males' percent fat mass during adolescence is caused by the rapid growth of fat free mass (in particular muscle mass) combined with slower accumulation of fat mass at this time (see figure 6).

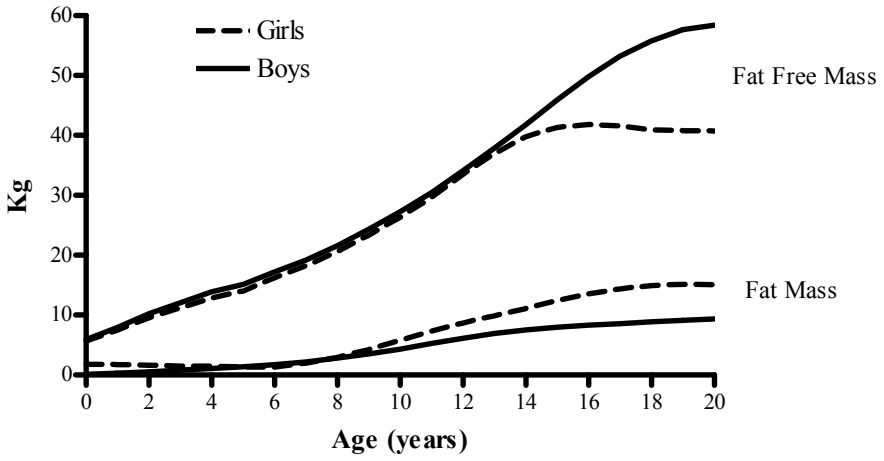


Figure 6: Growth curves for fat-free mass and fat mass and for boys and girls. Adapted from (Malina *et al.,* 2004).

Adolescence is also a period of redistribution of fat. Extremity subcutaneous fat (i.e. sum of 5 extremity skinfolds) is greater than trunk subcutaneous fat (sum of 5 trunk skinfolds) at all ages in females, and up until the onset of puberty in boys (Malina *et al.*, 2004). With the onset of puberty both sexes gain more fat on the trunk, but the gain is less in girls than in boys. In addition, boys experience a reduction in the thickness of subcutaneous fat on the extremities during adolescence, which accentuates the relative accumulation of subcutaneous fat on the trunk at this stage of growth. These subcutaneous fat growth trends are based on skinfold thickness, which does not take into

account the changes occurring in limb sizes and areas, which occurs with normal growth and development. Therefore, a better estimate of the growth changes in adipose tissue distribution may be obtained from other methods, such as DXA, magnetic resonance imaging (MRI) or computed tomography (CT). However, at present there are no longitudinal studies published of DXA, MRI or CT measured body fat that spans adolescence.

1.2.7 Biological maturity

When considering how to assess biological maturation, it is first important to understand that one year of chronological time does not equal one year of maturational time. Whilst every individual passes through the same stages of maturity they do so at differing rates, resulting in children of the same CA differing in their degree of maturity. This is reflected in Figure 7. Both boys are 14 years of age but differ considerably in their degree of maturity, with the boy on the left being an early maturer and the boy on the right being a late maturer. Furthermore, the size of an individual is not an accurate indicator of maturity. Certainly, in very general terms, size is associated with maturity, in that a bigger individual is likely to be chronologically older and thus be more mature than a smaller individual. However, it is well recognized that size does not play a part in the assessment of maturity.

Maturity indicators are used to assess maturity and can be any definable and sequential change in any part of the body that is characteristic of the progression of the body from immaturity to maturity (Cameron, 2002). The most commonly used methods

involve an assessment of skeletal age, secondary sex characteristics, menarcheal status and/or somatic characteristics. Each method will be briefly reviewed.

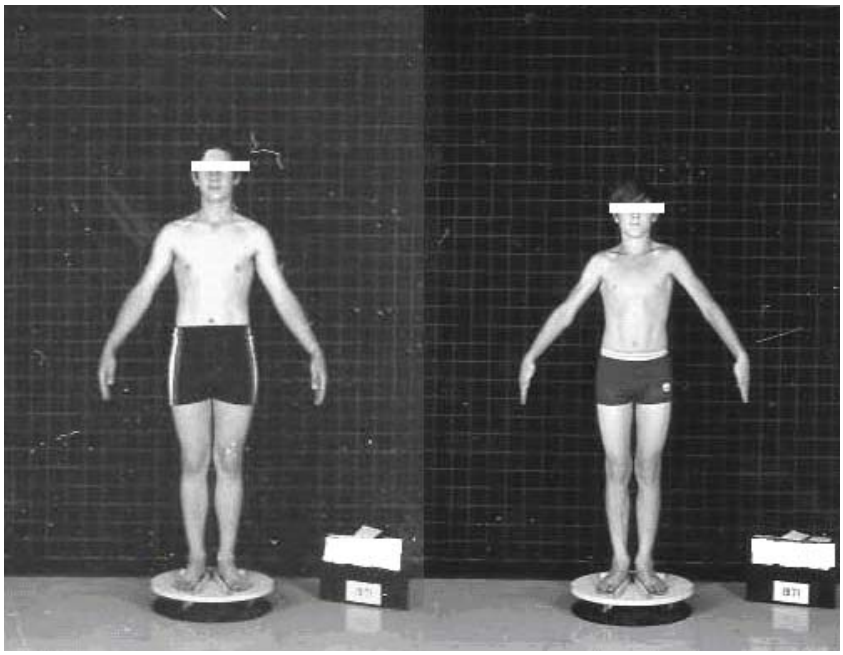


Figure 7: Two boys photographed at the same chronological age (14 years). The boy on the left is an early maturer and the boy of the right is a late maturer. Figure taken from (Baxter-Jones & Sherar, 2007).

1.2.7.1 Maturity indicators

1.2.7.1.1 Age at peak height velocity

Landmarks on an individual's height growth curve can be used as an indicator of maturity. The most commonly used somatic milestone in longitudinal studies of childhood growth is the age at PHV (APHV), although take off (or initiation of PHV) and cessation of growth have also been used. To obtain APHV, whole year height velocity (cm/year) increments are plotted and mathematical curve fitting procedures are used to identify the age when the maximum velocity in statural growth occurs. Girls usually

reach PHV around 12 years and boys around 14 years of age (Tanner, 1962). However, the timing of this event in relation to CA shows great variance. Data from a Swiss study revealed that girls reached PHV anywhere between 9.3 and 15.0 years and boys anywhere between 12.0 and 15.8 years (Malina *et al.*, 2004). Once APHV has been determined individuals can be aligned by biological maturity age (years from APHV) rather than CA. For example, APHV of an individual has a biological maturity age equal to 0 years. At 11.8 years an individual who reaches PHV at 13.8 years will have a biological maturity age of -2.0 years. Alternatively, individuals can be characterized as early, average or late maturers depending on the age at which PHV is attained. Age at PHV occurs, on average, close to 12 years in girls and 14 years in boys with a standard deviation of approximately 1 year (Malina *et al.*, 2004); therefore, individuals are often classified as early or late maturing if they fall 1 year outside of these ages.

To obtain APHV, time series data is required and therefore this indicator of maturity has previously been limited to longitudinal studies. Mirwald and colleagues (2002) developed sex-specific multiple regression equations, based on the growth patterns of the upper body and legs, which predicts years from PHV. When years from PHV are considered in relation to current CA, APHV can be estimated. The prediction equations require measures of stature and trunk length, as well as body mass and CA. Using these growth indicators the true APHV value can be predicted, within $\pm$ 1 year, 95 out of 100 times. This method of assessing maturity is quick, non-invasive, and inexpensive to administer and can be used in cross-sectional studies. The added advantage to this technique is that it predicts a maturity bench mark that exists in both boys and girls; therefore, it allows for comparisons of maturity between boys and girls.

1.2.7.1.2 Menarcheal status

Age at menarche (the first menstrual period) is the most commonly reported developmental milestone of female adolescence in both cross sectional and longitudinal studies. Three methods (prospective, status quo, and recall) are commonly used to establish age at menarche. The best and most reliable is to follow individuals and note the date menarche occurs. However, this method is limited in that longitudinal data is required. Alternatively, normative values can be established by the status quo method. This involves asking a large number of girls (usually aged between 8 and 18 years) when they were born and whether they have started their menstrual flow. From their ages and their answers (yes or no) it is possible to calculate mean and standard deviation values for age of menarche. The third method is the recall method. A simple questionnaire is used to establish if an individual has experienced menarche, if the answer is yes, they are asked to indicate the date or month. The retrospective method is useful for individuals after 17 years of age, when almost all girls have attained menarche (Tanner, 1962).

Although age at menarche is a widely used maturity indicator in studies involving females, its use is limited to later adolescence as menarche usually occurs after PHV (Tanner, 1962). Most studies, especially in athletes, use the recall method which has the limitation of recall error. Estimated mean ages are biased, since all subjects have not yet reached menarche. Furthermore, age of menarche has no use in gender comparison studies as no corresponding maturity indicator exists in males.

1.2.7.1.3 Other indicators of biological maturity

Two other common indicators of maturity used in research and clinical practice are skeletal age and secondary sex characteristics. A skeletal age assessment requires an x-ray, usually of the hand and wrist or knee, and is based on the observation that an individual more advanced in the maturity process will have greater bone development and a smaller amount of cartilage than a less mature child. The assessment of skeletal age is considered the best maturational index because it is the only indicator that spans the whole growth period (from birth to maturity) and it can be used to compare maturity levels of boys and girls. However, it is costly, requires specialized equipment and interpretation, and exposes subjects to low-dose radiation which are the reasons why it has not been adopted in the research outlined in this thesis. Furthermore, discrepancies of one or more years between skeletal ages of the knee and of the hand-wrist have been documented in individual youths (Roche *et al.*, 1975). This questions whether the skeletal maturity of the hand and wrist represents the maturity of the whole skeleton.

Secondary sex characteristics are one of the most frequently used maturity indicators in growth studies because they are a visible manifestation of sexual maturity at a given period of time. Secondary sex staging divides the process of breast development in girls, genitalia development in boys, and pubic hair development in both sexes, into 5 stages. The scale is usually used in conjunction with a series of photographs or drawings (Tanner, 1962). Stage 1 indicates the pre-pubertal state; the absence of visual signs of the development of each characteristic. Stage 2 indicates the initial overt development of each characteristic. Stages 3 and 4 indicate continued maturation of each characteristic and are somewhat difficult to evaluate. Stage 5 indicates the adult or mature state.

Determination of sexual maturity has been obtained through direct visual observation and child self assessment (comparing themselves to standardized photographs or drawings). Aligning individuals by secondary sex characteristics is used frequently because it does not require longitudinal observations, is easy to administer, cost effective, and non-invasive (with the replacement of physician assessment with self-assessment). However, problems arise when secondary sex characteristics stages are analyzed as if they are continuous variables. For example, an individual in the early phase of stage 3 of pubic hair development is rated the same as an individual in the late phase of this stage. This provides even less information when you consider that the length of time it takes to move through a stage varies considerably among individuals (Marshall & Tanner, 1969; Marshall & Tanner, 1970).

1.2.7.2 Sequence of pubertal events

The first overt sign of pubertal development in boys is usually the enlargement of the testes accompanied by changes in texture and colour of the scrotal skin. The penis then begins to enlarge and pubic hair appears. In females the first sign of sexual maturity is breast development, followed by pubic hair development. However, in about one-third of girls, pubic hair appears before the breast bud. A review of the usual age ranges of secondary sex characteristic development of boys and girls from European and North American countries have been published (Malina *et al.*, 2004). The average age of entering genital stage 2 (G2) in boys ranged anywhere between 9.2 and 12.4 years, depending on the sample. The onset of pubic hair development (PH2) on average occurs anywhere between 11.2 years and 13.4 years. In comparison, PHV normally occurs when

most boys are in G4 and PH4 (between 13.8-14.1 years). Elongation of the larynx (voice breaking) usually occurs late in puberty, about 1 year after the attainment of PHV. The first spontaneous ejaculation of seminal fluid during wakefulness has been reported to occur between 12.5 years and 16.5 years. Axillary hair appears usually after PH4; however, occasionally axillary hair may appear before the onset of pubic hair. Facial hair usually appears after the complete development of both pubic hair and genitalia. These wide age ranges illustrate the individual variation in entering, progressing through, and completing puberty.

Similar variability observed in males is seen in the onset, progression and completion of female sexual maturity. The advent of breast stage 2 (B2) is usually followed closely by the appearance of pubic hair (PH2). The progression of breast development and pubic hair development show considerable independence so, of girls in B3, 25% may be in PH1 and 10% in PH5 (Tanner, 1962). The range of average ages reported by Malina and colleagues (2004) for the onset of breast stage 2 (B2) ranged from 8.9 years to 11.6 years and for pubic hair stage 2 (PH2) from 8.8 years to 12.1 years. In comparison menarche occurs late in the sequence of events, an average of one year after PHV (between 12.8 and 13.5 years). Again there is considerable independence of menarche from pubertal characteristics; although most girls are in B4 some are in B3 and a small percentage may be in B2. Likewise most girls are in PH3 or PH4, while some may be in PH1. The majority of girls experience menarche at the time of maximum deceleration of growth in stature. Thus, menarche is closely associated with the timing of PHV, although the hormonal significance of this is unknown.

The various timings of these pubertal events are illustrated in figure 8, using data from the Saskatchewan Pediatric Bone Mineral Accrual Study (PBMAS) (Bailey, 1997). The figure shows that a number of pubertal events are occurring at the same time, all under the control of various endocrine systems and ultimately controlled by genetic expression. However, the timing of pubertal events varies between individuals of the same sex.

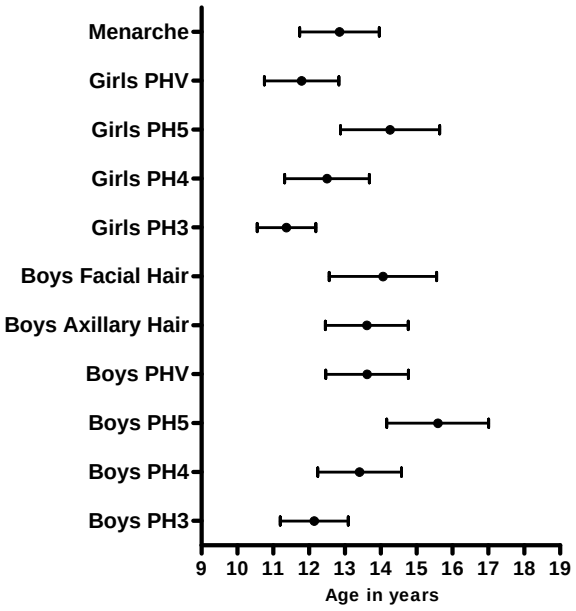


Figure 8: Average age of attainment of pubic hair (PH) stages 3-5 and peak height velocity (PHV). In boys only, axillary hair and facial hair growth and in girls only, menarche. Values are means (circles) and two standard deviation (bars). Data taken from the Saskatchewan Pediatric Bone Mineral Accrual Study (1964-1973) (Bailey, 1997). Figure taken from (Baxter-Jones *et al.,* 2005).

As well as individual variation there is also a marked sex difference in the timing of somatic and sexual maturation. Girls enter and end puberty approximately 2 years before boys (Tanner, 1962). Pubertal events do not occur in the same sequence between

the sexes. For example, when comparing pubic hair growth to statural growth, PHV is a relatively early event in girls and a relatively late event in boys. Boys' PHV occurs, on average, during PH4 and 5, whereas girls' PHV usually occur during PH 3 and 4 (Sherar *et al.*, 2004). This suggests that the timing of sexual and somatic maturation is not the same between girls and boys.

1.2.7.2.1 Relationship between indicators

Correlations between the timing of maturity indicators are generally moderate to high (Bielicki *et al.*, 1984) suggesting that there is a general maturity factor underlying the tempo of growth and maturation during adolescence in both boys and girls. However, there is sufficient variation to suggest that no single system (i.e. sexual, skeletal, or somatic) provides a complete description of the tempo of maturation during adolescence. Furthermore, although sexual maturation and skeletal development are associated, an individual in one stage of secondary sexual development cannot be assumed to be in a set stage of skeletal development (Bielicki *et al.*, 1984). The apparent discord among the aforementioned indicators reflects individual variation in the timing and tempo of sexual and somatic maturity, and the methodological concerns in the assessment of maturity that have been previously outlined.

1.2.7.3 Maturity associated variation in body size and composition

1.2.7.3.1 Stature and body mass

As previously mentioned, children of the same CA can vary considerably in their degree of biological maturity, or maturity status (Tanner, 1962). A child's maturity status

will influence measures of growth. Early maturing individuals of both sexes are taller and heavier than average maturing and late maturing individuals of the same CA (Beunen *et al.*, 1994; van Lenthe *et al.*, 1996a). If a youth's stature is expressed as a percentage of adult height, early maturing individuals are closest to their adult height at all ages during adolescence (Bayley, 1962). The height advantage of the early maturing individual is primarily due to an earlier attainment of PHV and also a greater magnitude of peak height gain (Iuliano-Burns *et al.*, 2001). Early maturing individuals also have a greater weight for height at each age (Beunen *et al.*, 1994; van Lenthe *et al.*, 1996a). However, if the average growth curves for height and weight for early, average and late maturing boys and girls are aligned on PHV, little differences in height and weight are found at PHV among maturity groups (Iuliano-Burns *et al.*, 2001).

Studies have repeatedly shown little or no correlation between the timing of the adolescent growth spurt (i.e. maturity status) and adult stature (Bielicki & Hauspie, 1994; Largo *et al.*, 1978; Malina *et al.*, 2004), suggesting that early, average and late maturing children reach, on average, the same adult height. However, early maturing girls from the Growth and Health Study were shorter in early adulthood (starting at approximately 15 years of age) despite having greater post PHV and menarcheal increment in height (Biro *et al.*, 2001). It was suggested that the shorter young adult stature of early maturing individuals was due to decreased growth pre PHV (because PHV is occurring earlier). In summary, there still appears to be controversy surrounding the timing of biological maturity and final adult stature. With regards to weight, one study showed no difference in adult (i.e. 30 years) body mass between maturity groups (Beunen *et al.*, 1994), whereas another study found early maturers to be heavier at 27 years (van Lenthe *et al.*, 1996a).

1.2.7.3.2 Body composition

The average age at which the velocity in growth of lean mass and fat mass peak occurs is earliest in early maturers, later in average maturers, and latest in late maturers (Iuliano-Burns *et al.*, 2001). In both sexes during adolescence, early maturing youngsters have, on average, larger measurements of muscle (Reynolds, 1946) and fat (Biro *et al.*, 2001; Reynolds, 1946; van Lenthe *et al.*, 1996a). The differences between children of contrasting maturity groups are primarily due to size differences, because early maturers are taller and heavier than late maturers of the same CA (Malina *et al.*, 2004). When muscle widths are expressed relative to height the differences between maturity groups are often eliminated (Reynolds, 1946). However, there is some evidence that during the later adolescent years early maturing boys have larger muscle widths even after taking into account height differences. On the other hand early maturing individuals of both sexes appear to have greater fat widths at all ages through adolescence, even when height differences are controlled (Reynolds, 1946).

In summary, adolescence is a period of dramatic change in growth and physiology. At any given CA during adolescence, early maturing boys and girls are on average taller, heavier, have greater fat free mass (especially in boys), total body fat, and percent body fat (especially in girls) than their less mature peers. The relationship between timing of biological maturity and fat mass development, especially as it relates to the development of obesity, will be discussed in more detail later in the chapter.

1.2.8 Adolescent physical activity

There is a significant decline in physical activity (PA) as children progress through adolescence, with a large proportion being inactive (Allison *et al.*, 2007; Sallis, 2000). Cross sectional studies reveal that activity levels drop by as much as 50% during this period (Aaron *et al.*, 1993; Heath *et al.*, 1994). There is also consistent evidence that boys are more active than girls during adolescence (Armstrong & VanMechelen, 1998; Sallis *et al.*, 1996; Trost *et al.*, 2002) and that the decline in PA over the adolescent period is more pronounced in girls than boys (Sallis *et al.*, 2000; Telama & Yang, 2000). Sallis and colleagues (1993) estimated that girls are, on average, approximately 25% less active than boys during the school year, and that activity declines at a rate of 7.4% per year in girls and 2.7% per year in boys. In confirmation, Livingstone and colleagues (2003) found that girls aged 7-15 years of age spent, on average, 8 to 10 minutes per day in vigorous PA, while boys of the same age spend approximately 30 minutes per day in vigorous PA. Girls' lower levels of activity appear to extend across numerous sports and forms of physical involvement (Sallis *et al.*, 1996). However, sex differences are greatest in strenuous sports and activities (Fuchs *et al.*, 1988). Although the adolescent decline and sex difference in PA (i.e. boys more active than girls) are some of the most frequently documented phenomena in PA epidemiology, the impact of variation in biological maturity on PA and its impact on sex differences in PA has yet to be studied.

1.2.9 Adolescence as a critical period for adult health

Traditionally, cardiovascular and metabolic diseases have been considered to be the result of behavioral risk factors in adulthood interacting with genetic predisposition to

disease. This paradigm was first questioned by findings from follow-up studies of men and women born in England between 1911–1930 which showed that the death rate from coronary heart disease was significantly higher in adults who had been in the lower end of the birth weight distribution (i.e. low birth weight) rather than the upper end (Barker *et al.*, 1989; Osmond *et al.*, 1993). Since this research, low birth weight has been associated with increased rates of coronary heart disease, stroke, hypertension and non-insulin-dependent diabetes, even after the effects of confounding variables have been controlled (Barker, 2004). Evidence is also accumulating which suggests that post natal growth is linked to adult health. For example, early age at adiposity rebound (the nadir of BMI and fat usually occurring ~ 6 yrs of age) has been related to adult obesity (Whitaker *et al.*, 1998).

There appears to be three 'critical periods' during human development when exposure to specific environmental stimuli are required in order to elicit the normal development of particular anatomical structures or their normal functioning. The periods are prenatal and early post-natal life, adiposity rebound and adolescence (Cameron & Demerath, 2002). The idea that individuals essentially "grow into" the diseases that they will have as older adults, has been coined "the lifespan perspective", a term which encompasses accumulating and interacting risks that are manifest from prenatal life onward (Cameron & Demerath, 2002). The theory that stems from this perspective is that to prevent adult diseases, one must scrutinize the growth and maturation process to find the early fetal/childhood/adolescence/adulthood variations that lead to later disease.

As previously mentioned, adolescence represents one of the most complex and interesting periods within human growth because rapid changes are occurring in growth

but also in maturation (i.e. skeletal, sexual and morphological). To date, research on adolescence as a critical period has focused largely on the accumulation of body fat (both absolute and its distribution; e.g. Serdula *et al.*, 1993) and in obtaining peak bone mass (e.g. Baxter-Jones *et al.*, 2003). However, research is also available which explores adolescence as a critical period in terms of behaviour (e.g. PA; Malina *et al.*, 2004). Specifically, the decline in PA coupled with the increase fat mass development (especially in females) over the adolescent period has highlighted adolescence as a pivotal (i.e. critical) time for youth and adult health. For example, the Surgeon General's report in the United States (Department of Health and Human Services, 1996) state that maintaining PA habits in youth helps prevent sedentary behaviour in adults. Similarly, the British Heart Foundation (2000) says that "physically active children are more likely to become active adults". Likewise adolescent overweight and obesity has also received much attention because it is generally believed that an overweight/obese adolescent will become an overweight/obese adult (Department of Health and Human Services, 2001). Much of the impetus for linking adolescent health with adult health and health behaviours has come from tracking studies.

Tracking is a term which is used to express the degree of consistency over time of an attribute, and is used to describe the ability to predict future values from early measurements (Twisk *et al.*, 1994). Generally, tracking is expressed in two ways: (1) by calculating the correlation between repeated measurements of the same attribute (e.g. PA) in the same individual, or (2) by calculating the proportion of individuals who, at the later measurement, maintain their relative position within the distribution of values. However, when interpreting results, it should be remembered that the strength – or weakness – of

tracking depends on how well the attribute can be measured and the degree of within-person variation. The following section outlines the evidence surrounding how well fat mass (and body mass index, a frequently used indicator of weight status) and PA track from adolescence to adulthood.

1.2.9.1 Tracking of fat mass from adolescence to adulthood

The period of adolescence is characterized by an increased deposit and altered distribution of body fat. A review of all prospective studies published between 1970 and July 1992 which had used anthropometric indices to assess fat mass/weight status (e.g. BMI, skinfold assessment), found moderate to high correlations of 0.4–0.8 between adolescent and adult obesity (Serdula *et al.*, 1993). Furthermore, the risk of becoming an obese adult was 3.9–6.5 times higher for obese adolescents than for non-obese adolescents. About half of obese school children (and a third of overweight pre-school children) were also obese as adults; however, the majority of overweight adults (about 75%) were not overweight as adolescents. Since the review by Serdula and colleagues (1993), several prospective studies have confirmed that overweight or obese adolescents are at greater risk of being obese adults. Furthermore, studies have shown that the probability increases according to the degree of overweight and the age at onset of overweight in adolescence (Freedman *et al.*, 2004 ; Guo *et al.*, 2000; Hulens *et al.*, 2001; Kemper *et al.*, 1999; Kvaavik *et al.*, 2003; Magarey *et al.*, 2003; Power *et al.*, 1997). For example, Guo and colleagues (2002), using data from the Fels Longitudinal Study, correlated BMI parameters taken at 2 to 25 y of age to BMI and fat mass values at adulthood (35-45 years). It was found that more than half of obese boys greater than 13

years of age became obese adults (at 35 years), compared to only a fifth of obese boys younger than 8 years and a third of obese boys between 8 and 13 years. A similar pattern was seen among females. There is, however, little evidence to indicate that an individual who becomes overweight during adolescence is likely to be more overweight in adulthood than an individual who first becomes overweight in young adulthood.

Although tracking of body weight, BMI, or fat mass between adolescence and adulthood is better than tracking between infancy and adulthood, it is important to remember that there will be a fraction of overweight and obese adolescents who become normal-weight adults, and likely a larger percentage of normal weight children and adolescents who will gain a substantial amount of weight and become obese adults (Malina *et al.*, 2004). Other factors, such as parental overweight, socioeconomic status, levels of PA or inactivity, high fat and sugar intake are believed to influence the tracking of overweight/obesity from adolescence to adulthood. Also, an early onset of puberty is accompanied by a higher BMI (Laitinen *et al.*, 2001), and a higher risk of the individual continuing to be overweight as an adult (Power *et al.*, 1997), thus the timing of biological maturity could also influence tracking of PA, and will be discussed later in the chapter.

1.2.9.2 Tracking of physical activity from adolescence to adulthood

In the Cardiovascular Risk in Young Finns Study, boys' and girls' leisure time PA (measured by a 5-item questionnaire) showed low-to-moderate tracking of coefficients between 12 and 18 years (Raitakari *et al.*, 1994). In both sexes tracking was better in the older (i.e. adolescent) age groups. When the participants of the study were followed-up 12 years later, correlations were reduced (Raitakari *et al.*, 1994), and after 21 years of

follow-up, when the subjects were 30–39 years old, no further reduction in correlations had occurred (Telama *et al.*, 2005). However, being persistently active (i.e. over 3 to 6 years) during the school years significantly increased the probability of being physically active as an adult. Furthermore, the probability of remaining physically inactive between adolescence and young adulthood was significantly stronger than the probability of remaining active (Raitakari *et al.*, 1994). In confirmation, other studies have reported higher tracking of inactivity than activity (Andersen & Haraldsdottir, 1993; Campbell *et al.*, 2001).

In the Amsterdam Growth and Health Longitudinal Study (in this study PA was measured by a semistructured interview) few individuals remained inactive or active from 13 to 21 years, and at 27 years (Van Mechelen & Kemper, 1995). However, when adolescent PA was estimated on the basis of a 4-year average (13–16 years), 38% of boys and 29% of girls were still active at 27 years, while 10% of boys and 42% of girls were inactive at the age of 27 years. Malina and colleagues (2004) reviewed longitudinal studies published before the mid-1990s, and concluded that levels of PA (mostly assessed on the basis of self-reported data on the frequency and intensity of PA in spare time and at school/work) indicated low-to-moderate stability.

In summary, physical inactivity and fat mass appear to track moderately well between adolescence and adulthood; thus providing modest evidence to support adolescence as a critical period for adult health. In addition, there has been some evidence to suggest that diseases which were typically regarded as a middle- to late-adulthood disorder, are present during adolescence, further highlighting adolescence as a critical period for adult health. One such ‘adult’ disease that has received attention over

the past decade is the metabolic syndrome, and will be the focus of following sections. Next I will discuss the relationship between adolescent growth and maturation, PA and fat mass (and obesity) and how they relate to adult metabolic syndrome and/or cardiometabolic risk.

1.2.10 Metabolic Syndrome

The metabolic syndrome is defined as the concurrence of abnormal glucose and insulin metabolism, overweight and abdominal fat distribution, mild dyslipidemia and hypertension (Eckel *et al.*, 2005). The reason that metabolic syndrome is important is that it helps identify individuals who are likely at high risk of CVD and type 2 diabetes. The concept of the metabolic syndrome has existed for at least 80 years. The syndrome was first observed by Kylin in 1923, who described the clustering of hyperglycemia, hypertension and gout, as a syndrome. In the 1940s upper-body obesity received attention as the obesity phenotype commonly associated with CVD and type 2 diabetes (Vague, 1947). The simultaneous presence, or 'clustering', of cardiovascular risk factors has also been named the insulin resistance syndrome, syndrome X, plurimetabolic syndrome, and the deadly quartet. However, the term "metabolic syndrome" is the preferred term at present and will be used throughout this thesis.

Just as the metabolic syndrome has many different names, it has many different definitions. The World Health Organization (WHO) was the first to formulate a definition for the metabolic syndrome (Alberti & Zimmet, 1998). Subsequently the NCEP ATP III (2001) and the European Study of Insulin resistance (Balkau & Charles, 1999) have put forward definitions. The most recent definition was proposed by the International

Diabetes Federation in Berlin at the 2005 International Meeting on Prediabetes and Metabolic syndrome (Alberti *et al.*, 2005). All of these definitions agree on the core components of the metabolic syndrome (obesity, hypertension, dyslipidemia and glucose intolerance) and classify individuals as either having or not having the syndrome, but differ in detail and cut-points (i.e. the threshold values that define diagnoses) (see Table 1).

Table 1: Comparison of the metabolic syndrome definitions

WHO, 1999	**European Group for the Study of Insulin Resistance, 1999**
Diabetes or impaired fasting gylycemia or impaired glucose tolerance or insulin resistance (hyperinsulinaemic, euglycaemic clamp-glucose uptake in lowest 25%) Plus ≥ 2 of the following: BMI> 30 or waist/hip ratio >0.9 (male) or > 0.85 (female) TG ≥ 1.7 mmol/L or HDL-c <0.9 (male) or <1.0 (female) mmol/L BP >140/90 mm Hg AE>20 µg/min	Insulin resistance-hyperinsulineamia: top 25% of fasting insulin values from non-diabetic population Plus ≥ 2 or more of the following: WC ≥ 94 cm (male) or ≥ 80 cm (female) TG >2.0 mmol/L or HDL cholesterol <1.0 BP ≥ 140/90 mm Hg and/or medication FPG ≥ 6.1 mmol/L
NCEP: ATP III, 2001	**IDF, 2005**
≥ 3 of the following: WC >102 cm (male), >88cm (female) TG ≥ 1.7mmol/L HDL-c <1.0 mmol/L (male), <1.3 mmol/L (female) BP ≥ 135/85 mm Hg or medication FPG ≥ 6.1 mmol/L	≥ 3 of the following: WC >94 cm (europid male), >80cm (europid female)* TG ≥ 1.7mmol/L HDL-c <1.03 mmol/L (male), <1.29 mmol/L (female) BP ≥ 130/85 mm Hg FPG ≥ 5.6 mmol/L or previously diagnosed type II diabetes

* South Asian and South-East Asian men > 90 cm, women >80 cm; Japanese men >85 cm, women >90 cm.
WC= Waist circumference; TG= Triglycerides; HDL-c= HDL-cholesterol, BP= Blood Pressure; AE= Albumin excretion; FPG= Fasting Plasma Glucose

1.2.10.1 A composite cardiometabolic risk score

A composite risk score has been used when investigating associations between variables (such as PA) and cardiometabolic risk, especially in young populations (DuBose *et al.*, 2007; Ekelund *et al.*, 2006). A quantitative composite score is often chosen over the traditional dichotomous (i.e. present/not present) method of diagnosing metabolic syndrome because it is statistically more sensitive and less error prone (Brage *et al.*, 2004; Ragland, 1992). All traits of the metabolic syndrome show a graded association with cardiovascular risk (Stratton *et al.*, 2000); therefore, a very minor improvement in one measure could result in an individual no longer being classified as having the syndrome, despite minimal change in presumed risk (Andersen *et al.*, 2006a). A composite score could also, to some extent, compensate for fluctuations in the single risk factors. Lastly, a continuously distributed variable will maximize statistical power (Ragland, 1992), which is especially important when the sample size is small and/or the study population is young and thus the prevalence of metabolic syndrome is expected to be low. Most often the average or sum of standardized scores (i.e. z scores) of metabolic syndrome risk variables (which are dependent on the definition adopted) are used to create a cardiometabolic composite risk score. A lower score is indicative of a better CVD risk factor profile.

1.2.10.2 Prevalence of the metabolic syndrome

The prevalence of the metabolic syndrome varies markedly between studies, most likely because of the lack of accepted criteria for the definition of the syndrome. Using the NCEP ATP III (2001) definition, the third National Health and Nutrition Examination

Survey (NHANES III) estimated the prevalence of metabolic syndrome in the US between 1988 and 1994 was 24% (Ford *et al.*, 2002). In this US national random sample (n=8,814), the metabolic syndrome was found to be highly prevalent, with little difference between men (24%) and women (23.4%) aged 20 to ≥70 years. The prevalence of the metabolic syndrome was shown to increase linearly with age. The authors suggested that the age specific prevalence could be estimated as a percentage by AGE-20 (i.e. in a sample of 30 year olds the prevalence would be 10%). Applying this estimation of age-specific prevalence of the metabolic syndrome to the US census counts from 2000 suggests that 47 million US residents, 25% of the population, have the metabolic syndrome (Ford *et al.*, 2002).

Using the NCEP ATP III criteria for metabolic syndrome, abdominal obesity was the most prevalent characteristic among participants of the NHANES survey, occurring in 38.6% of those with the metabolic syndrome (Ford *et al.*, 2002). Low HDL-c, high blood pressure (or medication use), and hypertriglyceridemia were also highly prevalent in 37.1%, 34.0%, and 30.0% of participants, respectively. Alarmingly, 71.2% of participants had ≥1 and 43.9% had ≥ 2 metabolic syndrome traits (Ford *et al.*, 2002).

To date, a population wide documentation of prevalence of metabolic syndrome, as provided by NHANES, in Canada is not available. However, there are some smaller studies which indicate that metabolic syndrome is not a condition which stops at the border. Using the NCEP ATP III criteria (2001), the Canada Heart and Health Survey (CHHS) indicate that the prevalence of the metabolic syndrome in Canada (1986 through 1992) were 17.0% in men (n=3872) and 13.2% in women (n=3503) (Ardern *et al.,* 2003). These estimates are substantially lower than those recently reported by NHANES III

(24.0% and 23.4% in men and women, respectively) (Ford *et al.*, 2002). It is unlikely that the disparity in the estimated was due to ethnic or racial differences, because the prevalence (23.8%) of the metabolic syndrome in the white cohort of NHANES is not unlike that of the entire sample (Ford *et al.*, 2002). The authors (Ardern *et al.*, 2003) suggested that the difference in the two national estimates of metabolic syndrome was due to differences in abdominal obesity (high waist circumference; WC) and high blood glucose/diabetes between the two surveys. Cholesterol, triglycerides and blood pressure were similar between the two surveys, whereas high blood glucose/diabetes was almost triple, and the prevalence of abdominal obesity was more than double in the NHANES III compared to the CHHS. However, it appears as if the problem is not subsiding with 25% of a more recent (1996–2000) sample of multi-ethnic Canadians (n =1276; average age 50.4 years), having the metabolic syndrome (Anand *et al.*, 2003).

1.2.10.3 Risk factors for metabolic syndrome

A risk factor is a condition that, when present over an extended period, significantly increases the probability of a common chronic disease, such as CVD. Many modifiable risk factors for metabolic syndrome are known. The most important biological risk factors are dyslipidemia, insulin resistance, high blood pressure, low cardiopulmonary fitness and high body fatness. In turn these biological risk factors are often associated with unhealthy lifestyle factors such as low PA, smoking and unhealthy dietary habits (Berenson *et al.*, 1998).

1.2.10.4 Adolescent antecedents of metabolic syndrome.

Although typically regarded as a middle-to-late adulthood disorder, increasingly the metabolic syndrome is present in childhood and adolescence. For example, among a sample (n=4450) of US adolescents (12-19 years), 3.5% overall and 14.5% of overweight adolescents had the metabolic syndrome, suggesting that nearly 1 million adolescents in the US are currently affected (Pan & Pratt, 2008). These results provide compelling evidence to support adolescent antecedents of the metabolic syndrome.

The following section(s) review the literature which explores the relationship between PA (a lifestyle risk factor) and metabolic syndrome, and fat mass (a biological risk factor) and metabolic syndrome. Specifically, the following will be discussed a) youth PA and youth metabolic syndrome b) adult PA and adult metabolic syndrome c) youth PA and adult metabolic syndrome. The role of timing of biological maturity in the development of metabolic syndrome will then be discussed; specifically with regards to the timing of biological maturity and PA, fat mass development and risk for metabolic syndrome.

1.2.11 PA and risk for metabolic syndrome

1.2.11.1 Youth PA and youth risk for metabolic syndrome

Studies that have examined the relationship between single risk factors, such as blood pressure and lipids, and PA in youth have yielded inconsistent results (Froberg & Andersen, 2005). However, results from studies which used clustered risk factors as the outcome variable are clearer. Studies show an inverse relationship between youth PA and

youth risk for metabolic syndrome (Andersen *et al.*, 2006b; Andersen *et al.*, 2008; Brage *et al.*, 2004; Kelishadi *et al.*, 2007). Furthermore, among 4,811 school students aged 6–18 years randomly selected from six provinces in Iran, low levels of PA significantly increased the risk of having the metabolic syndrome, before and after adjustment for age and BMI (Kelishadi *et al.*, 2007). Data from the European Youth Heart Study showed that among children aged 9 and 15 (n=1732), severity of metabolic syndrome risk score was associated with objectively assessed PA (Andersen *et al.*, 2008), again irrespective of body weight. Specifically, a graded negative association between clustering of risk factors and PA was observed, with the least active quintile having an odds ratio of clustered risk around 3 compared with the most active quintile.

1.2.11.2 Adult PA and risk for metabolic syndrome

The majority of cross-sectional studies document a relationship between greater PA and a reduced risk of metabolic syndrome in adults (Brien & Katzmarzyk, 2006; Carroll *et al.*, 2000; Lakka *et al.*, 2003; Zhu *et al.*, 2004). For example, a Canadian sample of 6406 men and 6475 women aged 18–64 y from the CHHS (1986–1992) revealed that physically active (active at least once each week for at least 30 min) men were more than 50% less likely to have metabolic syndrome than their physically inactive counterparts (Brien & Katzmarzyk, 2006). However, the relationship between PA and the metabolic syndrome was less clear among females. Only physically active women between the ages of 35 and 49 years of age were less likely to have metabolic syndrome compared with physically inactive women of the same age. Other studies have noted differences between men and women in the relationship between PA and metabolic

syndrome (Boule *et al.*, 2005; Zhu *et al.*, 2004). For example, results from the NHANES III demonstrated a significant relationship between PA and metabolic syndrome in men, but not in women, after consideration of covariates including age, race, education, and income levels (Zhu *et al.*, 2004). It has been postulated that these inconsistencies are caused by gender differences in hormones, fat distribution, and types and intensity of PA (Boule *et al.*, 2005). However, these thoughts have yet to be verified.

The few prospective cohort studies investigating the relationship between PA and metabolic syndrome in adults present inconsistent findings. Byberg et al. (Byberg *et al.*, 2001) found in a 20 y follow-up of men (n=1860) aged 50 y that those who increased their leisure time PA (determined through self-reports) had lower BMI, reduced plasma triglycerides, increased HDL cholesterol, decreased fasting glucose, and decreased proinsulin levels. However, in contrast, baseline self report PA was not a significant predictor of metabolic syndrome 5 years later, after sex, study site, ethnicity and impaired glucose tolerance were controlled, among 714 black, white and Hispanic men and women (Palaniappan *et al.*, 2004).

1.2.11.3 Youth physical activity and adult risk for metabolic syndrome

Despite the substantial evidence supporting an inverse association between PA and metabolic syndrome in adults, little is known about the relationship between PA during childhood and adult risk for metabolic syndrome. Three longitudinal studies from Europe, the Amsterdam Growth and Health Longitudinal Study (AGHLS) (Twisk *et al.*, 2002), the Leuven Longitudinal Study on Lifestyle, Fitness and Health (Lefevre *et al.*, 2002), and the Danish Youth and Sports Study (Hasselstrom *et al.*, 2002), have investigated the direct relationship between PA in youth and adult CVD risk factors (e.g.

lipoprotein levels, blood pressure, body fatness and body fat distribution). These studies consistently failed to find a significant relationship between childhood and adolescent PA and adult risk for CVD. However, these studies did not track PA from adolescence through to early adulthood. For example, The AGHLS (1977-1997) carried out eight measurements over a period of 20 years. However, due to low subject number, participants were included in the analysis if they had at least three adolescent measurements and an adult (at 32 years) measurement. The Leuven study (1969-1996) conducted 9 measurements over 27 years and the Danish Youth and Sports Study (1983-1991) conducted 2 measurements over 8 years. A possible reason for the lack of association between PA and CVD risk in adulthood is that unlike physical fitness, which has a large genetic component, PA is a behaviour which is characterized by a dramatic decline following adolescence. The possibility exists, therefore, that those who are most active as adolescents may exhibit the greatest relative decline in activity by the time they reach young adulthood. For example, it was shown that the girls with the greatest increases in sports participation during the three year adolescent period displayed lower diastolic blood pressure, some seven years later (Boreham *et al.*, 2002). Therefore, there is a need for longitudinal assessment of PA through adolescence but also continued assessment into the third decade of life. Two other longitudinal studies, the Northern Ireland Young Hearts Project (Boreham *et al.*, 2002) and the Muscatine Study (Janz *et al.*, 2002), also failed to find a relationship between childhood PA and adult CVD risk factors; however, they stopped measuring PA when participants were 15 and 16 years of age, respectively.

A later analysis on the AGHLS (Ferreira *et al.*, 2005) investigated the longitudinal development (13-36 years) of PA of individuals with and without the metabolic syndrome at 36 years of age. Using generalized estimating equations (GEE) to examine growth trajectories of PA, it was found that with aging, individuals with the metabolic syndrome tended to spend more time in self-reported light-to-moderate intensity activities than those without the metabolic syndrome. However, the decline in vigorous PA was greater amongst those with the metabolic syndrome (Ferreira *et al.*, 2005).

1.2.12 Obesity and risk for metabolic syndrome

Obesity increases the risk for the metabolic syndrome in children, adolescents (Kopelman & Albon, 1997; Must, 1996) and in adults (Calle *et al.*, 1999; Janssen *et al.*, 2004a). For example, studies in adolescent populations have shown that the metabolic syndrome is largely confined to the overweight population. Across increasing body-weight categories in children and adolescents there is a rising and significant trend for risk variables for metabolic syndrome, including plasma glucose and insulin concentrations, increasing triacylglycerol levels, decreasing HDL levels, increased impaired glucose tolerance test and increased systolic blood pressure (Weiss *et al.*, 2004). Likewise, among 4450 US adolescents enrolled in the NHANES survey (1999-2002), compared to normal weight youth, the prevalence of metabolic syndrome was 16 times higher among adolescents who were overweight (Pan & Pratt, 2008). Other investigations have demonstrated that overweight in youth persists into adulthood and may be associated with adverse health outcomes in later life, including metabolic syndrome (DiPietro *et al.*, 1994; Must *et al.*, 1992; Vanhala *et al.*, 1999). For example, a population

based sample (n=712) of Finnish males and females found that half of the males and females who were obese (determined via BMI) at 7 years of age (n=75) had become obese adults (36-46 years of age) and had a high risk for the metabolic syndrome (Vanhala *et al.*, 1998). The risk of the metabolic syndrome was, however, lower among the obese adults who had not been obese as children (n=71). This finding suggests that obesity in adulthood that became established in childhood may be more harmful than obesity that has appeared in adulthood, highlighting the need to investigate the relationship between longitudinal development of fat mass (between adolescence and adulthood) and risk for metabolic syndrome.

1.2.12.2 Abdominal obesity

It has been known for some time that a central distribution of body fat, particularly an excess accumulation of fat intra-abdominally rather than a more peripheral distribution, carries a higher risk for metabolic syndrome. In 1947, Professor Jean Vague, from the University of Marseille, proposed that fat distribution was more highly correlated with diabetes, hypertension and CVD than overall obesity (Vague, 1947). Vague noted that women were more likely to accumulate fat around the hips and thighs (gynoid obesity) and men were more likely to accumulate fat over the trunk (android obesity). He further proposed that the gynoid obesity was seldom associated with the common complications of obesity. However, it took many decades before these observations were fully supported in the research and medical fields. Now, some 60 years later, it is well established that central or abdominal adiposity is a significant predictor of morbidity (Janssen *et al.*, 2004b) and mortality (Bigaard *et al.*, 2005) in adults,

independently of age, race, sex and BMI. Further, there is substantial evidence supporting the notion that, in adults, too much abdominal fat is predictive of insulin resistance and of the presence of metabolic syndrome (Despres *et al.*, 1990; Lemieux *et al.*, 2001).

Similar to adults, children's abdominal fat distribution, particularly visceral or intra-abdominal fat deposition, appears more strongly related to cardiovascular and diabetes risk than does whole-body fat. For example, Kelishadi and colleagues (2008) compared the prevalence of CVD risk factors and metabolic syndrome in a large (n=4811) nationally representative sample of children and adolescents aged 6 to 18 years from Iran. Children were grouped by BMI and WC into one of four groups: a) normal weight b) generalized obesity only c) central obesity only, d) combined obesity. The authors found that the prevalence of dyslipidemia, high BP, and metabolic syndrome was higher in those children with combined obesity than in those with the other two types of obesity. Furthermore, a higher prevalence of dyslipidemia, high BP, and metabolic syndrome was seen in the central than in the generalized obesity group. This study used WC as a surrogate measure of central adiposity. It is important to remember that intra-abdominal adipose mass is only one component of the WC, and WC measurement in children relates to both subcutaneous abdominal fat and intra-abdominal (or visceral) fat. However, numerous studies have now shown, using computed tomography (CT), that individuals with more visceral fat have a worse CVD risk profile (Bacha *et al.*, 2003; Kim & Park, 2008), even within normal weight populations (Tanaka *et al.*, 2004).

Studies investigating the relationship between adolescent development of central body fat and adult risk for metabolic syndrome are limited. However, among 48 men and women from the Aerobics Center Longitudinal Study, a high WC at one time period

during adolescence was related to a higher BP in young adulthood (average age 26.6 years) (Eisenmann *et al.*, 2005).

In summary, central obesity appears to play a more prominent role in metabolic syndrome/CVD. However, there are children, adolescents (Kelishadi *et al.*, 2008) and adults (St Onge *et al.*, 2004) with CVD risk factors and metabolic syndrome who are not obese according to BMI and WC. Even though the percentage is fairly small compared with overweight or centrally obese populations, it does suggest that fat mass (overall and abdominal) is not the sole determinant of CVD/metabolic syndrome.

1.2.12.2.1 Pathophysiology of abdominal obesity and metabolic syndrome risk

Controversy exists surrounding the specific mechanisms by which visceral fat confers greater CVD risk than subcutaneous fat (Bergman *et al.*, 2006). For example, at present, it is not known whether the storage of an absolute or relative excess amount of triacylglycerols in visceral fat depots is directly responsible for increased CVD risk or whether the central deposition is simply a marker of other processes that cause CVD. Furthermore, whether these mechanisms are apparent during the growing years has yet to be established. Although the pathophysiology behind abdominal obesity and CVD has yet to be elucidated, several hypotheses have been proposed.

An early hypothesis, proposed by Bjorntorp and colleagues (1997), suggested that activation of the hypothalamo-pituitary-adrenal (HPA) axis by environmental stressors, such as depression, anxiety, alcohol and smoking, caused the preferential deposition of fat to the abdominal area and the CVD disorders (e.g. insulin resistance) associated with that deposition. More recently it was suggested (Lemieux, 2004) that an increase in intra-

abdominal adipose tissue is a marker of the limited ability of subcutaneous fat depots to store excess fat when an individual has to handle a calorie surplus (due to excess energy intake and/or reduced energy expenditure). Such a relative deficit in the capacity of subcutaneous fat to store excess energy would result in increased accumulation of fat at other undesirable sites, such as the intra-abdominal area, liver, skeletal muscle, heart and even the pancreatic β-cells, a phenomenon that has been described as ectopic fat deposition. It is this ectopic fat disposition which is believed to cause metabolic dysfunctions. In support of this hypothesis, increased intrahepatic fat has been associated with dyslipidemia and hepatic insulin resistance (Seppala-Lindroos *et al.*, 2002), and increased intramyocellular fat has been associated with skeletal muscle insulin resistance (Sinha *et al.*, 2002).

A third hypothesis, sometimes termed the 'portal theory', proposes a direct effect of intra-abdominal fat depots on insulin resistance, lipoprotein metabolism, and blood pressure (Bergman *et al.*, 2006). Lipolysis of visceral adipose tissue triacylglycerols causes a release of non-esterified fatty acids (NEFA). The resultant flood of NEFAs to the liver and the systemic circulation could be a stimulus for insulin secretion and thus impair hepatic glucose production and induce hepatic insulin resistance (Bergman *et al.*, 2006). Although there is a correlation between visceral fat accumulation and portal delivery of NEFAs to the liver, most portal NEFAs originate from the systemic circulation (and not from visceral fat per se) (Jensen, 2006). This suggests that other factors might explain the altered metabolic profile of viscerally obese patients. There is ample evidence to suggest that visceral adipose tissue not only stores and mobilizes lipids, but also acts as an endocrine organ, secreting 'offensive' adipocytokines, such as

PAI-1, tumor necrosis factor-α or visfatin, and under secretes defensive adipocytokines, such as adiponectin (Despres & Lemieux, 2006). These cytokines likely contribute to conditions, such as diabetes mellitus, hyperlipidemia, hypertension and atherosclerosis, which comprise the metabolic syndrome.

A fourth hypothesis proposes that a genetic predisposition independently causes preferential deposition of fat in abdominal depots and CVD risk (Katzmarzyk *et al.*, 1999). The visceral fat phenotype has been investigated in two family studies and several intervention studies (Katzmarzyk *et al.*, 1999). The studies indicate a strong genetic component for the amount of abdominal visceral fat even after adjustment for age, sex, and total amount of body fat.

It is likely that some, or all of the aforementioned mechanisms, plus other unknown mechanisms, are involved in the association between abdominal fat mass and adverse metabolic consequences.

1.2.13 Biological maturity and risk for metabolic syndrome

Research studying the relationship between biological maturity and metabolic syndrome is scarce; however, there is a wide array of research investigating the relationship between biological maturity and fat mass and limited research on biological maturity and central body fat. Likewise, there is limited research on biological maturity and PA. Because obesity, and in particular central obesity, and inactivity confers greater risk for metabolic syndrome, findings from these studies can be used to shed light on the role that biological maturity may play in child and adult risk for metabolic syndrome. Therefore, the following section discusses the literature linking biological maturity and a)

PA b) overweight/obesity c) visceral/abdominal fat mass and lastly d) metabolic syndrome risk.

1.2.13.1 Biological maturity and the adolescent: Two hypotheses

With regards to the relationship between timing of biological maturity and health behaviour and/or psychosocial functioning, two basic hypotheses have been prominent in the literature. The stage termination hypothesis, or early maturation hypothesis, (Petersen & Taylor, 1980) posits that only early maturing adolescents are at particular risk for psychosocial problems and adoption of unhealthful behaviours (such as smoking). This hypothesis is based on the assumption that early maturation interrupts the regular course of development so that early maturers have less time, and are less prepared, to resolve the normal developmental tasks of adolescence. In addition, based on their apparent physical maturity, early developers might be pressured by expectations from adults and peers that are not appropriate with regard to their actual cognitive and social-emotional development (Petersen & Taylor, 1980). The maturational deviance hypothesis (Alasker, 1995) proposes that any deviation from the norm relative to chronological aged peers (i.e. early and late maturation) heightens the risk for psychosocial problems. This hypothesis is based on the assumption that normative events that are occurring earlier or later than anticipated are stressful for individuals. More specifically, off time maturers are at risk because their physical development is incongruent with their peer group (Alasker, 1995).

With regards to the impact of the timing of biological maturity and psychosocial outcomes in females, research generally supports the early maturational hypothesis. With evidence to suggest that early biological maturation is associated with poorer body image

(Graber *et al.*, 1999), negative initial experiences to puberty (e.g. inconvenience, ambivalence and confusion; (Ruble & Brooks-Gunn, 1982) and increased distress, anxiety, depression, and psychosomatic symptoms (Graber *et al.*, 1997; Kaltiala-Heino *et al.*, 2003; Laitinen-Krispijn *et al.*, 1999). For example, among 178 US girls, early maturity at age 11 predicted lower psychological well-being at age 13 including depression (e.g. I hate myself, I have difficulty sleeping), global self-worth (e.g. I am often disappointed with myself), and weight related maturity fears (e.g. I don't like changes in my body because they make me feel fat) (Davison *et al.*, 2007). Early maturity has been further linked with early substance abuse (Dick *et al.*, 2000), alcohol abuse (Costello *et al.*, 2007) and early sexual initiation (Brown *et al.*, 2005).

The literature examining timing of biological maturity in boys appears a little less conclusive. Some research indicates that early-maturing boys are more vulnerable to psychological problems (Petersen & Crockett, 1985) and unhealthy behaviours (e.g. smoking) (Simon *et al.*, 2003) than their later-maturing peers. Conversely, early-maturing boys have been found to have greater self-esteem, confidence and popularity than later-maturing boys (Dubas *et al.*, 1991b). Research has also shown late maturation, rather than early maturation, is most risk-laden in terms of psychological symptoms and psychosocial problems in males (Dorn *et al.*, 2003; Graber *et al.*, 1997). Lastly, consistent with the "deviance hypothesis," research suggests that both early and late maturation may be risk factors for negative consequences. For example, Williams and Dunlop (1999) reported that both early- and late-maturing boys exhibited higher rates of delinquency than boys that were 'on-time'.

1.2.13.2 Biological maturity and barriers to PA

Given the research linking timing of biological maturity, psychology and adoption of unhealthful behaviours, it appears intuitive that timing of biological maturity may be related to adolescent PA behaviour. More specifically, new and distinct perceived barriers to PA may be experienced during adolescence and barriers may differ depending on timing of biological maturity. Perceived barriers to PA are defined as factors that make PA difficult or completely inhibit it (Bandura, 1997). Barriers can be personal or situational in nature (Bandura, 1997). In general, barriers have been identified as a consistent inverse correlate of PA across a diverse range of populations, including adolescents (Allison *et al.*, 1999). Previous research has shown that adolescents experience unique barriers to PA (Gyurcsik *et al.*, 2006). However, to date there has been no research which has considered the role that biological maturity, or more specifically timing of biological maturity, may have on perceived barriers to PA.

1.2.13.3 Timing of biological maturity and physical activity

Until recently the specific relationship between levels of PA and timing of biological maturity has not been addressed. Of the limited research that has been undertaken, most has focused on adolescent girls and produced mixed results. Niven and colleagues (2007) found no difference in self-reported PA among early, average and late maturing 11-year old girls (N=208). Likewise, no significant difference in pedometer steps were found among early, average and late 13-14 year old girls (N=86) (Wickel & Eisenmann, 2007). In contrast, a longitudinal study of 2247 adolescents found that among grades 7 and 8 (11-13 years), early maturing girls participated in more minutes of

self-reported vigorous PA than late maturing girls (van Jaarsveld *et al.*, 2007). However, no differences in PA were noted among maturity groups in grades 9-10 (13-16 years). Baker and colleagues (2007) showed that early maturing girls (assessed via a combination of estradiol, breast development and mother's report on pubertal development using the pubertal development scale at age 11) participated in less accelerometer measured PA at age 13 when compared to late maturing girls. Likewise, a study by Riddoch and colleagues (2007) found that parental reported pubic hair and breast development was inversely related to PA among 2933 eleven year old girls.

With regards to the timing of biological maturity and PA behaviour and boys, even less research has been conducted. Among 13-14 year old boys (N=81), the average number of pedometer steps per day did not differ between early, average and late maturing boys (Wickel & Eisenmann, 2007). In contrast, a study which followed a cohort of 2982 boys from ages aged 11-12 years, for 5 years, found that early maturity was consistently associated with higher levels of self-reported vigorous PA (van Jaarsveld *et al.*, 2007). However, early maturing boys also reported more sedentary behaviour than their average, or late maturing counterparts. A study of two hundred and sixty five 11-14 year old boys showed that pubertal stage (as assessed via the pubertal development scale) was not related to self report PA (Bradley *et al.*, 2000). Lastly, pubertal development was shown to be inversely related to minutes spent in accelerometer assessed moderate-to vigorous PA among 11-year old boys, however to a lesser extent that that observed by the same age girls (Riddoch *et al.*, 2007).

There has been only one study to date which has examined the persistent association of timing of biological maturity on PA behaviour in males. Among males

(*N*=166) from the Leuven Longitudinal Study on Lifestyle, Fitness and Health, greater sports participation at age 40 was associated with a later APHV (Beunen *et al.*, 2004).

1.2.13.4 Timing of biological maturity and overweight/obesity

Puberty has been identified as a critical period for the development of overweight (Dietz, 1994); however, the specific role of puberty and variations within the normal range in pubertal timing for the development of obesity has yet to be elucidated. Age at pubertal onset is decreasing, and although girls are more extensively studied in this respect, there are reports of secular trends in pubertal onset for boys as well (Liu *et al.*, 2000; Parent *et al.*, 2003). Among girls, the decreased age at menarche over the last century has paralleled the increasing incidence of obesity (Muinich Keizer & Mul, 2001; Parent *et al.*, 2003), thus researchers have speculated that the secular trend for menarcheal age is a part of the obesity epidemic in the western world.

Evidence from several different epidemiologic studies (Anderson *et al.*, 2003; Biro *et al.*, 2001; Himes *et al.*, 2004; Wang, 2002) over the past 30 years indicates a relationship between earlier puberty in girls and increased BMI. For example, the National Heart, Lung, and Blood Institute's National Growth and Health Study is a cohort study of 2379 girls divided approximately equally between white and black girls who were recruited in 1987 at age 9 from schools in the US. Across the entire 9- to 18-year age range, the white and black girls with an early age at menarche, had consistently higher BMIs than the mid onset girls, who had higher BMIs than the late-onset girls (Biro *et al.*, 2001). Similar findings were reported when the sum of skinfold thickness was examined in relation to the timing of menses; however, the group of early maturing girls

had a significantly greater WC (surrogate for visceral adiposity) than mid-onset or late maturers but had no difference in the waist to hip ratio (regional distribution of adipose tissue). Analysis from the Girls Health Enrichment Multi-site Studies, which included 147 black girls between the ages of 8 and 10 yrs, found that increasing stages of breast development (but not pubic hair) were positively related to BMI and WC, as well as DXA derived fat mass and percentage body fat and that pubertal girls were 8 times as likely to have a BMI ≥95th percentile, as were age-matched prepubertal girls (Himes *et al.*, 2004).

Although evidence is mounting which suggest that early maturity in girls may be related to a greater BMI and possibly adiposity, the question of whether earlier puberty is the cause or the result of increased body fat has not been resolved. However, two longitudinal studies suggested that increased body fat or a rapid increase in BMI during childhood predicts earlier onset of puberty (Davison *et al.*, 2003; Lee *et al.*, 2007). Further, there are emerging data suggesting that the rate of weight increase in childhood determines the age at puberty through complex mechanisms involving leptin (Dunger *et al.*, 2005).

The studies investigating the relationship between pubertal timing and body fat among boys are limited, and are likely due to the lack of a convenient and obvious milestone of puberty, such as age at menarche in girls. Nevertheless, in contrast to the findings with adolescent girls, few studies have found a link between body fat and earlier puberty in boys. An Italian study found that obese boys (N=141) did not mature earlier than normal, in fact 19% had later genital development and 16% had later pubic hair development (Vignolo *et al.*, 1988). A more recent study of Spanish boys between 11 and

14 years found a positive relationship between age of pubertal onset (entry into genital stage 2) and BMI; however, the sum of skinfolds and the percentage body fat did not differ according to the age of pubertal onset (Vizmanos & Marti-Henneberg, 2000). Furthermore, a study of Israeli adolescents (n= 136) found that obese boys were taller up to age 14, but there was no difference in the age of pubic hair and facial hair onset or of testicular and genital enlargement (Laron, 2004). However, many of these studies used the BMI to assess body fat; therefore, when interpreting these findings one must remember that during male puberty, the normal increase in muscle mass related to the anabolic effect of rising testosterone levels will cause an increase in weight and BMI independent of any increase in body fat. Therefore, more studies which use direct measures of body fat are needed to accurately assess the relationship between biological maturity and fat mass in adolescent boys.

Several longitudinal studies have looked at the long term impact of early maturity on body composition. Van Lenthe and colleagues (1996a) investigated the impact of early or late maturity on the development of BMI and sum of skinfolds among 79 males and 89 females enrolled in the Amsterdam Growth and Health Longitudinal Study (AGHLS). Based on skeletal age or APHV, early maturing boys' BMI was significantly higher than for late maturing boys between 13 and 27 years. Based on skeletal age only, early maturing boys also showed higher mean sum of skinfold thickness over this period. Likewise, early maturing girls (based on skeletal age or age at menarche) had greater BMI and sum of skinfold thickness than late maturing girls. In a similar manner, Beunen and colleagues (1994) assessed somatic characteristics of 149 boys from 13-18 years and at 30 years of age. They found that an early age at PHV was related to a consistently

higher BMI during adolescence but not at age 30. However, importantly, adjustment for pre-pubertal BMI was not performed in these two studies, and thus the independent predictive role of pubertal timing on BMI and/or fatness could not be evaluated.

1.2.13.4.1 Biological maturity and regional fat deposition

The timing of the adolescent growth spurt is an important factor influencing the distribution of subcutaneous abdominal fat in males and females. However, only a few studies have considered biological maturity in the development of regional body fat, and the persisting effects of maturity status (i.e. early, average and late maturing). Malina and colleagues (1999) examined changes in three individual skinfolds (triceps, subscapular, abdominal) and ratios of skinfolds of children of the Wroclaw Growth Study (193 boys and 197 girls), who were followed longitudinally from 8 to 18 years of age. Relative to CA, the abdominal/triceps skinfold ratio was greater in early maturing boys and girls (top quartile for APHV). However, when plotted relative to biological age (years APHV) the difference between boys of contrasting maturing status was negligible, while that for early and late maturing girls was more apparent at and after PHV.

In the Leuven Growth Study, Beunen and colleagues (1994), demonstrated that early maturers had increased skinfold thickness in the abdominal area. A similar pattern of increased central adiposity was seen in early maturing girls in the Fels Study (Remsberg *et al.*, 2005); however, neither of these studies controlled for baseline or prepubertal body composition. A more recent longitudinal study of Norwegian girls (n=844) found that an early age at menarche was associated with overweight (assessed via BMI) in late adolescence, but only among girls with relatively high WC in early

adolescence (Bratberg *et al.*, 2007). The authors thus concluded that it is the combination of early central adiposity and early age at menarche which increases the risk of being overweight in late adolescence. Likewise, Kindblom and colleagues (2006) demonstrated that among 579 males, age at PHV, independent of prepubertal BMI, was a negative predictor of young adult (18.9 $\pm$ 0.5 years) BMI and DXA derived total body fat mass. Furthermore, APHV was an independent negative predictor of central, but not peripheral, fat mass. Lastly, a study of 1092 white and 1164 black girls showed that early maturers had significantly greater WC than average or late maturers from 9 through 19 years of age, but no difference were noted in waist to hip ratio (distribution of adipose tissue) (Biro *et al.*, 2001).

1.2.13.5 Biological maturity and the metabolic syndrome

Timing of maturity (i.e. whether an individual is early, average or late maturing) has also been associated with risk for metabolic syndrome/CVD in adulthood. Most of the literature on the relationship between physical maturity and adulthood development of CVD risk factors has focused on age at menarche in girls.

Precocious puberty (i.e. puberty occurring before 8 years of age) in girls is a clear indicator of increased risk for development of metabolic syndrome in adulthood (Ibanez *et al.*, 2000). With regards to boys, one study showed that boys with precocious adrenarche exhibited decreased insulin sensitivity, independent of BMI; (Denburg *et al.*, 2002) whereas another study found no association (Potau *et al.*, 1999).

The impact of pubertal timing, within the 'normal' range, on metabolic syndrome risk is yet to be fully elucidated. Early menarche (as defined as the lowest quartile of menarcheal age in NHANES III (Chumlea *et al.*, 2003)) is associated with

hyperinsulinemia, insulin resistance, (Frontini *et al.*, 2003) elevated blood pressure and glucose intolerance (Remsberg *et al.*, 2005) in young and mid-adulthood when compared with later maturing girls. However, inconsistent findings have been found on the relation between age at menarche and adult blood pressure (Frontini *et al.*, 2003; Heys *et al.*, 2007; Remsberg *et al.*, 2005) and lipid levels (Frontini *et al.*, 2003; Heys *et al.*, 2007). A retrospective historical cohort study of 7349 Chinese women (≥50 years) revealed that a young age of menarche (<12.5 years) was associated with the presence of the metabolic syndrome (Heys *et al.*, 2007). The associations remained after adjustment for adult central obesity which means that central obesity but did not fully explain the association between early menarche and adult metabolic syndrome risk. In confirmation, among a separate Chinese female cohort (n=9090), aged 25-64 years, age at menarche was inversely associated with number of metabolic syndrome components (Feng *et al.*, 2008).

The relationship between early maturity and adult CVD risk in males is yet to be elucidated. Furthermore, the relationships among early maturity, PA (in childhood and adulthood) and early adult CVD risk have not been studied.

1.2.14 Other metabolic syndrome risk factors

Smoking and dietary intake are possible risk factors for the metabolic syndrome. There is overwhelming evidence for an adverse effect of smoking on health (US Department of Health and Human Services, 2004). Specifically, the number of cigarettes smoked per day is positively related to the metabolic syndrome (Lee *et al.*, 2005, Wilsgaard & Jacobsen, 2007) and the prevalence of metabolic syndrome is higher in

both current and past smokers compared with individuals who have never smoked (Wada *et al.*, 2007).

In general dietary intakes rich in whole-grain foods, dairy products, fruit, vegetables (Esmaillzadeh *et al.*, 2005; Lutsey *et al.*, 2008; Pereira *et al.*, 2002; Sahyoun *et al.*, 2006) and/or, low in refined grain, fried foods, meat and regular and diet soda (Dhingra *et al.*, 2007; Esmaillzadeh *et al.*, 2005; Lutsey *et al.*, 2008), have been associated with lower incidence of the metabolic syndrome in adults. Studies examining metabolic syndrome and its association with diet in adolescents are limited. Using NHANES data (1999-2002), Pan and colleagues (2008) observed a lower prevalence of metabolic syndrome with higher overall diet quality (measured as Healthy Eating Index overall score) among 12-19 year old adolescents (n=4450).

1.2.15 Statement of problem

The metabolic syndrome has become a major public health challenge world-wide (Eckel *et al.*, 2005), and in the industrialized world the prevalence of the metabolic syndrome is increasing. There is evidence to show that biological and lifestyle risk factors for metabolic syndrome are present in adolescence, which suggests that the antecedents of the disease may lie in early life. The period of adolescence is characterized by a decline in PA (a lack of PA is a lifestyle risk factor for metabolic syndrome) and an increase in fat mass deposition (a biological risk factor for metabolic syndrome). Therefore, investigating how the development of these two variables relates to adult metabolic syndrome is important to fuel early intervention.

A factor which has the potential to influence these two risk factors, and thus ultimately the metabolic syndrome, is the timing of biological maturity (i.e. whether an individual is early, average or late maturing when compared to peers of the same age). However, the influence of biological maturity has largely been overlooked in previous research. Therefore, the general objective of this thesis was to investigate the associations between biological maturity, adolescent PA and fat mass development and young adult cardiometabolic risk. Three studies were necessary to realize this objective. The first study explored whether the pervasive documentation of adolescent girls being less active than adolescent boys, is confounded by biological maturity differences between genders; thereby shedding light on the potential association between biological maturity and adolescent PA. The second study explored the PA and barriers to PA (i.e. factors that make PA difficult or completely inhibit it (Bandura, 1997)) of adolescent girls by grade and biological maturity status. Information derived from this study will help to determine if girls of different maturity status participate in different levels of PA and if so, what are the reasons for maturity related differences in PA. The final paper related the longitudinal development of adolescent and young adult PA and body fat to cardiometabolic risk at 26 years of age, while investigating the role of biological maturity. The three studies together will help elucidate the role of biological maturity in the adolescent decline in physical activity, fat development and in the development of adult metabolic syndrome. Ultimately, this information will aid in the development and implementation of interventions to decrease prevalence of metabolic syndrome.

1.2.16 Aims/Hypotheses

1.2.16.1 Study 1:

To investigate whether observed gender differences in objectively measured PA in children (8 to 13 years) are confounded by physical maturity differences. I hypothesized that a) when considering PA aligned by CA, boys would be more active than girls at each age b) when considering PA aligned by biological age (years from APHV), the sex differences observed in PA would disappear.

1.2.16.2 Study 2:

To describe the PA levels of adolescent girls (i.e., aged 8-16 years) by school grade level and by maturity status (i.e., early or late maturing) within grades. Secondly, to identify perceived barriers to PA among adolescent girls by grade and by maturity status within grades. I hypothesized that: (a) PA would decrease with increasing grade and (b) within grade groups, early maturing girls would participate less in PA than late maturing girls. Given the expected decline in PA with CA and biological age, I further hypothesized that: (a) the average number of barriers to PA would increase as grade in school increased and (b) within grade groups, early maturing girls would experience a greater number of barriers to PA than late maturing girls.

1.2.16.3 Study 3:

To examine total body fat mass (TBFM), trunk FM and PA developmental trajectories (aligned to biological maturity age) of individuals categorized as low and high for cardiometabolic risk at 26 years, while investigating biological and lifestyle risk

factors. I addressed the following hypotheses. First, early maturity in males and females would be associated with higher cardiometabolic risk at 26 years. Second, males and females with greater fat mass development (TBFM and trunk FM) during childhood, adolescence and young adulthood would have higher cardiometabolic risk at 26 years. Furthermore, early biological maturity, greater dietary fat intake and less PA would be associated with greater FM development. Third, males and females who participate in less PA during childhood, adolescence and young adulthood would be at higher cardiometabolic risk at 26 years. Furthermore, early biological maturity and greater TBFM would be associated with less PA.

Chapter 2: Study 1:

Age and gender differences in youth physical activity: Does physical maturity matter?

2.1 Introduction

One of the most pervasive findings in epidemiological studies of physical activity (PA) is the decline in PA with age. Although PA is shown to decline throughout the lifespan (Troiano *et al.*, 2008), both cross-sectional and longitudinal studies have shown that the decline in self-reported PA is greatest during the adolescent years. For example, a Finnish longitudinal study showed that in boys the greatest overall decline in PA was between 12 and 18 years, and in girls between 12 and 15 years (Telama & Yang, 2000). Additionally, data from the Amsterdam Longitudinal Growth and Health Study documented the greatest decline in PA between 13 and 16 years of age (Van Mechelen *et al.*, 2000). Lastly, in a cross-sectional U.S. sample, Caspersen and colleagues found that the greatest reduction in self-report PA occurred between 15 and 18 years of age (Caspersen *et al.*, 2000).

In addition to the consistent documentation of an age related decline in PA in both boys and girls, it is also well established that boys are more active than girls at all ages during adolescence (Kimm *et al.*, 2000; Riddoch *et al.*, 2004; Sallis, 1993; Trost *et al.*, 2002). For example, an investigation of accelerometer assessed PA in 375 students (grade 1 through to grade 12), showed that boys participated in more minutes of daily moderate to vigorous PA (MVPA) (with the exception of grades 1-3) and more minutes of daily vigorous PA (VPA) (Trost *et al.*, 2002). Further evidence using a large sample

(*N*=2185) from the European Youth Heart Study showed that 9- and 15-year old boys spent 20% and 36% more time than girls in daily moderate PA (Riddoch *et al.*, 2004). In a Canadian study of 1057 normal weight children, it was again found that boys spent more time in PA than girls. Specifically, boys in grades 3, 7 and 11, spent 9%, 22% and 27%, respectively more time in MVPA than girls (Thompson *et al.*, 2005).

The observation that at a given age boys are more active than girls may be well established, but at least one group believes the finding is confounded. A longitudinal study by Thompson *et al.,* (2003) found that during adolescence the gender differences in self-reported PA disappeared when differences in physical maturity were controlled. The main concern regarding confounding is the fact that, on average, girls mature approximately two years before boys (Malina *et al.*, 2004; Tanner, 1989). For example, on average, boys reach PHV, a somatic indicator of physical maturity, at approximately 14 years of age whereas girls, on average, reach the same maturity milestone at approximately 12 years of age. Adolescence is recognized as a period of great physical, psychosocial, cognitive and emotional change. Each of these changes (which may be linked to physical maturity) influences adolescent participation in PA (Sallis *et al.*, 2000). Therefore, the consistent observation that boys are more active than girls during adolescence may purely be an artifact of boys maturing later than girls, and as a result altering their PA behaviour later.

Thompson *et al.,* (2003) determined that gender differences in PA disappear when aligned on maturational age, suggesting that physical maturity may be intricately involved in the adolescent decline in PA. This has important public health implications. For example, adolescent PA interventions may need to target biological maturity groups,

rather than chronological age groups (i.e. grades in school) to effect positive change. However, to the authors' knowledge, the study by Thompson *et al.,* (2003) is the only one to date to consider biological maturation in the study of adolescent PA and has yet to be replicated using an objective assessment of PA. Therefore, the purpose of the present study was to investigate whether observed gender differences in objectively measured PA in children (8 to 13 years) are confounded by physical maturity differences. It was hypothesized that: a) when aligned by CA, boys would be more active that girls at each age b) when aligned by biological age (years from APHV) the gender difference in PA would disappear.

2.2 Methods

2.2.1 Participants

Data were from 194 boys and 207 girls, aged 8 to 13.9 years. Participants at ages 8-13 years were chosen as they are likely to be predominately pre-PHV (boys on average reach PHV at 14 years of age and girls at 12 years of age). This age group is important to study because most of the variation in physical maturity occurs during the years approaching PHV (Mirwald *et al.*, 2002; Tanner, 1962). Participants were comprised of four groups of children residing in Canada: 1. Rural living children from Saskatchewan (*N*=127), 2. Urban living children from Saskatchewan (*N*=91), 3. Old-Order Mennonite children from southwestern Ontario (*N*=119) 4. Old- Order Amish children from southwestern Ontario (*N*=64). Information on the recruitment of participants can be found elsewhere (Bassett, Jr. *et al.*, 2007; Tremblay *et al.*, 2005). For each child written assent

and parental consent were obtained. All procedures were approved by the Institutional Research Ethics Board.

2.2.2 Chronological age

Age, expressed in decimal form, was calculated by subtracting date of birth from the measurement date. Chronological age categories were constructed using 1-year intervals, for example, individuals between 8.00 and 8.99 years of age were grouped in the 8-year old category. These age categories are consistent with the age-specific accelerometer cut-points (Trost *et al.*, 2002). The decision to use whole year age categories in truncated format (e.g., 8.00-8.99 = 8) rather than centered on whole year age midpoint (e.g., 7.50-8.49 = 8) was taken out of necessity. Because the PA data was calculated using whole year age categories in truncated format according to the age-specific cut-points described by Trost *et al.,* (2002) we were obligated to present the data in this format. This is important as it ensures that children grouped in the same age category are held to the same count intensity standard when determining how many minutes of PA they accumulate.

2.2.3 Anthropometry

Measurements were taken for stature, sitting height and body mass. All measurements were performed by a Professional Fitness and Lifestyle Consultant certified by the Canadian Society for Exercise Physiology. Leg length was calculated by subtracting sitting height from stature.

2.2.4 Physical Maturity (Biological age)

A common maturity assessment technique in longitudinal studies is the determination of years from attainment of PHV (Malina, 1978). Peak height velocity is an indicator of somatic maturity and reflects the age at maximum growth rate in stature during adolescence (age at PHV, APHV). In the present cross-sectional study, each individual's years from APHV was predicted using a gender specific multiple regression equation that included height, sitting height, leg length, chronological age, and their interactions (Mirwald *et al.*, 2002). Years from APHV results in a continuous measure of biological age. Biological age categories were constructed using 1-year intervals such that -1 APHV age group included observations between -0.49 and -1.50 years from (i.e., before) APHV.

2.2.5 Physical Activity

Physical activity levels were directly measured for seven consecutive days using the Actigraph 7164 accelerometer (Actigraph, LLC, Fort Walton Beach, FL). The Actigraph is a uniaxial accelerometer that detects vertical acceleration in the magnitude of 0.05-2.00*g* with a frequency response of 0.25-2.50 Hz (Tyron & Williams, 1996). A 7 day protocol was chosen because it provides reliable estimates of usual PA behavior in children and adolescents and accounts for potentially important differences in weekend versus weekday activity behavior as well as differences in activity patterns within a given day (Trost *et al.*, 2000). All accelerometers underwent a calibration check on a hydraulic shaker plate at varying accelerations and frequencies before use in the study (Esliger & Tremblay, 2006). Only accelerometers with intra- and inter- instrument reliability values

below 5% coefficient variation were used. The monitors were attached with an adjustable, elasticized nylon belt. The device was positioned on the right hip, above the iliac crest. Participants were asked to record when the monitor was put on in the morning and removed in the evening before bed for the purpose of distinguishing between activity time and sleep time. Upon completion of the data collection, the data were electronically downloaded resulting in a file containing minute-by-minute movement counts (or epoch) for each child. After data were scanned for spurious measures, sleep time was determined from the log sheets and activity counts were added to the data file for unworn daytime periods for which the activity was included on the log sheet (using the compendium of physical activities and MET-to-count conversion values derived via regression equations published by Trost *et al*., (2002). Files with minimal levels of incomplete data underwent imputation procedures to ensure seven days of data were available for analysis. A more complete description of the data reduction procedures can be found elsewhere (Esliger *et al.*, 2005; Tremblay *et al.*, 2005). In brief, all files were retained for analysis if it included at least five full days of data, with at least one of those being a weekend day. A non-monitored weekday was replaced by the mean data of the other four weekdays. The monitored weekend day was used to represent the non-monitored weekend day.

The average number of minutes of moderate to vigorous PA (MVPA; accumulated minutes $\geq$ 3 METs), continuous minutes of MVPA (CMPVA; accumulated minutes $\geq$ 3 METs clustered in bouts $\geq$ 10 minutes) and minutes of vigorous PA (VPA; accumulated minutes $\geq$ 6 METs) per day were calculated using age specific cut-points (Trost *et al.*, 2002).

2.2.6 Statistical analysis

Independent t-tests were used to investigate the gender difference in stature and body mass at each chronological and biological age. Gender and age (chronological and biological) differences in PA were tested using a two-way ANOVA. Included in each model were the main effects for age category and gender, and the interaction of gender and age. Physical activity data were log transformed due to the positive skewness of MVPA, VPA and CMVPA. The alpha level was set at 0.05. SPSS (version 11.5) was used to analyze the data.

2.3 Results

The subjects' physical characteristics, by chronological and biological age category, are shown in Table 2. There were no significant differences between boys and girls in body mass or stature, except at age 12 where girls were significantly ($p<0.05$) heavier than the boys. When the data were aligned on biological age boys were significantly taller and heavier than girls at all ages pre-and post- PHV that were common between the groups.

Table 2: Subject characteristics by gender, chronological age (CA), and biological age (BA)

CA Group (yrs)	*N*	Stature (cm)	Body Mass (kg)	BA Group (yrs)	*N*	Stature (cm)	Body Mass (kg)
Boys							
8	11	129.2 (7.8)	26.8 (4.7)	-5	12	127.1 (4.4)	25.3 (2.2)
9	39	137.8 (7.2)	34.6 (7.2)	-4	23	134.6 (5.6)	31.0 (5.8)
10	42	142.8 (7.2)	37.4 (7.2)	-3	62	141.7 (5.8)*	36.7 (4.2)*
11	43	148.0 (7.2)	44.0 (8.7)	-2	55	149.5 (4.8)*	43.3 (5.9)*
12	43	153.8 (8.0)	44.8 (8.3)*	-1	36	158.1 (5.6)*	50.7 (8.5)*
13	16	157.9 (8.5)	48.3 (8.0)				
Girls							
8	8	127.2 (5.2)	25.3 (3.6)	-3	15	130.3 (4.1)	27.7 (1.8)
9	32	135.8 (6.4)	33.0 (6.6)	-2	39	138.2 (6.7)	33.4 (5.9)
10	56	143.3 (6.3)	38.9 (8.3)	-1	64	145.4 (4.4)	40.6 (6.4)
11	46	149.0 (7.1)	43.2 (9.6)	0	47	151.6 (5.4)	45.6 (8.7)
12	56	155.8 (6.6)	49.6 (9.6)	1	36	160.2 (4.6)	54.3 (7.1)
13	9	159.9 (4.2)	52.5 (6.2)				

Means (SD); biological age calculated in years from estimated age at peak height velocity (APHV) *significantly ($p<0.05$) greater than girls. Note: *N*=401 when aligned by CA and *N*=389 when aligned by BA because observations that fell outside -5 to -1 for girls and -3 to 1 for boys were excluded because of the small number.

For ease of interpretation, Figure 9 shows the arithmetic means and standard deviations (i.e., non-transformed) for MVPA, CMPVA and VPA. When aligned on chronological age, PA showed the characteristic decrease with increasing age; however, not all decreases between age categories were significant ($p>0.05$) (figure 9A, C, E). The boys were found to have a higher MVPA at 10 through 13 years, a higher CMVPA at 9 through 12 years, and a higher VPA at 9 though 13 years compared to the girls ($P<0.05$). For MVPA the gender difference was the least at 8 years of age (9.7%) and the greatest at 13 years of age (30.5%). For CMPVA the gender difference ranged from 20.9% at 9 years to 52.1% at 11 years. For VPA the gender difference ranged from 20.8% at 9 years to 46.3% at 11 years.

When aligned on biological age, PA decreased with increasing biological age;

however, not all decreases between biological age categories were significant ($p>0.05$) (figure 9B, D, F). In contrast, there were no significant differences in MVPA, CMVPA or VPA between girls and boys when grouped by biological age ($p<0.05$).

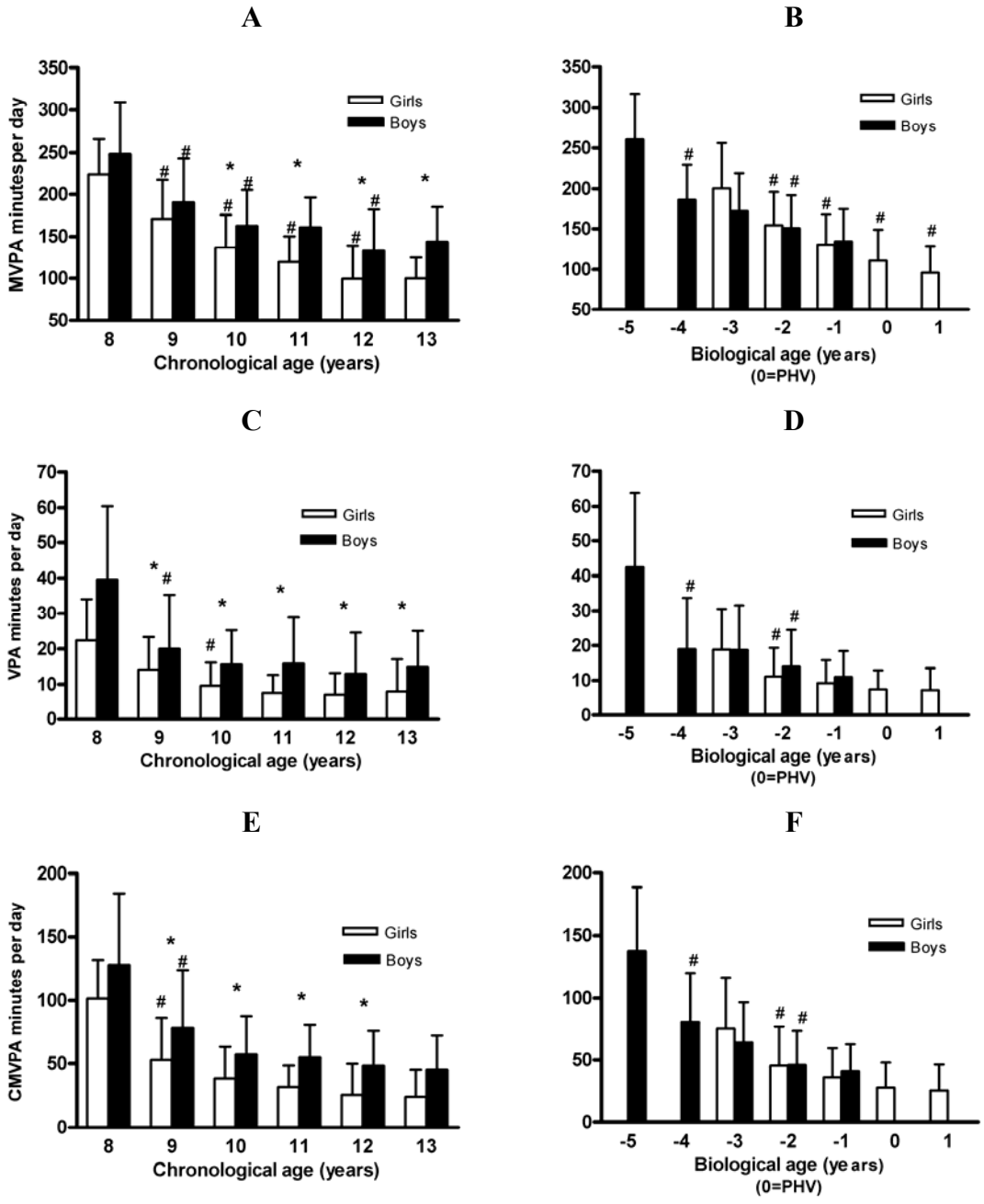


Figure 9: Objectively measured physical activity (PA) variables (mean + SD) of boys and girls by chronological and biological ages. Figures 9A, minutes of moderate to vigorous PA (MVPA); 9C, minutes of vigorous PA (VPA); 9E, continuous minutes of MVPA (CMVPA) by chronological age. Figures 9B, MVPA; 9D, VPA; 9F, CMVPA by biological age. *Significant gender difference within age category ($p < 0.05$); # significant difference from previous age category ($p < 0.05$)

Among the boys the largest chronological age difference (relative to the previous chronological age category) in MVPA, CMVPA and VPA occurred between 8 and 9 years of age (28.4%, 38.8% and 49.5%, respectively). Among the girls the largest chronological age difference in MVPA, CMVPA and VPA occurred between 8 and 9 years of age also (23.6%, 47.9% and 37.1%, respectively). Among the boys the largest biological age difference (relative to the previous biological age category) in MVPA, CMVPA and VPA occurred between -5 and -4 years from APHV (28.4%, 41.3% and 55.4%, respectively). Among the girls the largest biological age difference in MVPA, CMVPA and VPA occurred between -3 and -2 years from APHV (23.0%, 39.8% and 40.8% respectively).

2.4 Discussion

The finding that accelerometer assessed PA declined in both genders with increasing chronological age from 8 to 13 years supports previous research (Klasson-Heggebo & Anderssen, 2003; Riddoch *et al.*, 2004; Trost *et al.*, 2002). As expected, when comparing boys to girls on the basis of chronological age, boys accumulated significantly more MVPA, CMVPA, and VPA at most ages between 8 and 13 years. The fact that boys are more active than girls is a consistent finding; however, the magnitude of the gender differences appear to depend on the type and intensity of the activity (Telama & Yang, 2000; Trost *et al.*, 2000). For example, Trost and colleagues (2000) showed that for VPA the average gender gap was 45%, whereas the average gender gap for MVPA was only 11%. Results from the present study also demonstrate that the gender difference is greater for VPA than MVPA. The greatest decrease in MVPA, VPA

and CMVPA occurred between 8 and 9 years of age in both boys and girls.

My results also support the findings of Thompson and colleagues (2003) who demonstrated that the gender differences in PA disappeared when aligned on physical maturity. This finding suggests that maturity differences between genders (i.e. on average, girls mature earlier than boys) may account for conclusions from previous research that girls are less active than boys of the same chronological age. To my knowledge this is the first study to demonstrate this phenomenon using accelerometer assessed PA.

The confounding effect of biological maturity in adolescent PA may help to understand findings from previously published work. For example, Klasson-Haggebo and Anderssen (2003) noted that the rate of longitudinal decline in PA was similar for both boys and girls (25.9% and 27.2%, respectively), but that the age that PA levels began to decline was younger for girls than boys. The authors provided no explanation for this finding; however, I speculate that the earlier decline in PA shown in girls was due to their earlier age of maturation since on average, girls reach every maturity milestone earlier than boys (Malina *et al.*, 2004). Consistent with previous research (Malina *et al.*, 2004; Sherar *et al.*, 2004), the average APHV in my study was 1.65 years younger in girls compared to boys. The average APHV for boys (13.54 $\pm$ 0.81 years) and girls (11.89 $\pm$ 0.58 years) falls within the range of ages reported in previous literature (Malina *et al.*, 2004; Sherar *et al.*, 2004).

The age related decline in PA is well accepted, but not well understood (Livingstone *et al.*, 2003). My results provide evidence to suggest that, at least in 8-13 year olds, the decline in PA is associated with biological maturity. Further supporting the

importance of biological maturity, Finnish studies have shown that the beginning of the decline in PA is associated with pubertal stage, with early maturing girls being slightly less active than late maturing girls (Telama & Yang, 2000). There is evidence, from both cross sectional and longitudinal studies, to suggest that the annual rate of decline in PA is greater during adolescence than during late childhood and adulthood (Caspersen *et al.*, 2000; Kimm *et al.*, 2000; Telama & Yang, 2000; Van Mechelen *et al.*, 2000); however, most research exploring the age-related decline has not included children younger than 9 years. The inclusion of younger children in the present study showed that the greatest decrease in MVPA, VPA and CMVPA was between 8 and 9 years (pre-adolescence) in both boys and girls. However, longitudinal PA data including young children is required in order to make a definitive determination of the age at which the marked decline in PA occurs.

The consistent finding of an age related decline in PA, with boys being more active than girls, provides the primary rationale for many intervention programs targeting adolescent girls. Three examples of these programs are the Trial of Activity in Adolescent Girls (TAAG) (Stevens *et al.*, 2005), Project FAB (Jamner *et al.*, 2004) and 'Girls on the Move' (Robbins *et al.*, 2006). My results showing that controlling for biological maturity seems to nullify gender differences in PA, suggest that it may be erroneous to target adolescent females over males when designing interventions. However, prior research support gender targeted PA interventions, as adolescent boys and girls prefer different activities, participate in PA for dissimilar reasons, and may face different barriers to PA (Vu *et al.*, 2006). Interventions may also need to target girls at an earlier chronological age than boys; considering that on average girls mature two years

earlier than boys.

Previous research has explored barriers (and correlates) to adolescent PA in an effort to reduce the decline in PA over time. Yet to be addressed, however, is the relationship between barriers to PA and biological maturity. Identifying maturity related barriers to PA during adolescence may provide valuable information that can be used to design interventions to help boys and girls of varying maturation overcome these barriers. In the future, the consideration of biological maturity may also cause us to re-think the implementation of adolescent PA interventions. The majority of interventions are based on chronological age groups (i.e. grades) in schools; but, within one grade there may be considerable variation in biological age or the level of biological maturity attained. For example, some 12 year old girls maybe sexually mature, others are in the process of maturing, and others may not begin the process for several more years. Yet, all the girls have the same chronological age and are typically in the same grade in school. This wide variation in physical maturity was evident in my sample of adolescents, with boys APHV spanning 4.78 years and girls spanning 4.84 years. Therefore, in the design and implementation of school based PA interventions there may be a need to consider the variation in physical maturity within gender and grades. That said, one must acknowledge the potential hurdles in conducting a PA intervention based on maturity. For example, adolescents may be more comfortable participating in interventions with their peers in the same grade. Also considering that most school scheduling is based on chronological age grades, organizing maturity-based intervention that span many grades may be problematic.

The present sample of boys and girls spanned 8.0 to 13.9 years of age; therefore,

some of the participants (especially the boys) were likely pre-pubertal. To completely document the confounding effects of biological maturity on age and gender differences in adolescent PA, there is a need for an objective assessment of PA and an assessment of biological maturity on children older then 13 years of age. The present sample is also cross-sectional and thus APHV was predicted and as such, is likely less accurate than when observed in a longitudinal study.

2.5 Conclusion

The observed age-related decline in adolescent boys and girls PA is antithetical to public health goals and as such is an important area of research. In order to fully understand gender disparities in PA consideration must be given to the confounding effects of biological age.

2.6 Acknowledgements

This research was supported by a grant from the Canadian Population Health Initiative of the Canadian Institute for Health Information; and the Community and Population Health Research (CPHR) Training Program delivered by the Saskatchewan Population Health and Evaluation Research Unit (SPHERU).

Chapter 3: Study 2

Activity and barriers in girls (8-16 yrs) based on grade and maturity status

3.1 Introduction

Regular participation in physical activity (PA) during childhood and adolescence is important for health and well-being. Furthermore, this type of positive health behavior may track into adulthood (Hallal *et al.*, 2006). One of the most ubiquitous findings in PA research, however, is that PA declines with increasing chronological age (CA), and the decline is most marked during the adolescent period (Allison *et al.*, 2007; Armstrong *et al.*, 1990; Armstrong & VanMechelen, 1998; Fuchs *et al.*, 1988; Sallis *et al.*, 1996; Stone *et al.*, 1998). Data from cross-sectional studies reveal that activity levels can decrease by as much as 50% during adolescence (Troiano *et al.*, 2008; Trost *et al.*, 2002).

Little research has been directed towards understanding whether the decline in PA among adolescent girls is associated with their CA or biological age (BA). Whereas CA is determined by the date a child is born, a child's BA is how close the child is to reaching the mature adult state. Children of the same age can differ by several years in BA (Malina *et al.*, 2004). Understanding the influence of BA on the decline in PA would better inform researchers about the most effective timing of interventions. In a similar vein, understanding whether perceived barriers, defined as personal or situational factors that slow or prevent one from engaging in regular PA (Bandura, 1997), differ by CA and/or BA would provide valuable information for future intervention work aimed at reducing or negating salient barriers as a way to impact adolescent girls' regular participation in PA.

3.1.1 Biological age

The major growth process occurring during adolescence is puberty. Puberty is a period of rapid hormonal, physiological, and physical changes (Malina *et al.*, 2004). One cause of the decline in PA among adolescent girls may be related to the experience of pubertal development (i.e., BA or maturity). Results from previous studies showed that with increasing BA there was a decrease in self-report (Thompson *et al.*, 2003) and objectively measured (Sherar *et al.*, 2007b) PA among adolescent girls. These studies, however, did not assess the *timing* of pubertal or biological maturity on PA; and these factors may be of more importance. Timing of biological maturity refers to an individual's biological maturity status when compared to peers of the same CA (Malina *et al.*, 2004). In this manner a girl can be referred to as early, average, or late maturing for her CA. Because an early maturing girl is entering puberty at a younger CA than her peers, she may be more sensitive to the physical changes occurring during puberty. Previous research has shown that early biological maturation is associated with poorer body image and increased eating disorders, distress, anxiety and depression (Graber *et al.*, 1997; Kaltiala-Heino *et al.*, 2003; Laitinen-Krispijn *et al.*, 1999). As a result of these aforementioned outcomes associated with the timing of biological maturity, new and distinct perceived barriers to PA may be experienced.

3.1.2 Perceived barriers to physical activity

Perceived barriers to PA are defined as factors that make PA difficult or completely inhibit it (Bandura, 1997). Barriers can be personal or situational in nature (Bandura, 1997). Using an ecological approach (McLeroy *et al.*, 1988), personal barriers

are typically defined as intrapersonal barriers, which are factors within an individual that prevent activity (e.g., lack of motivation) (Brittain *et al.*, 2006; Gyurcsik *et al.*, 2006). Situational barriers can be further delineated into the categories of interpersonal, institutional, community, or public policy. Interpersonal barriers revolve around informal and formal social support networks (e.g., no friend to be active with). Institutional barriers occur within social institutions (e.g., school-based barrier of not having sufficient playground equipment). Community barriers occur between organizations and institutions within defined boundaries (e.g., lack of community-based activities). Public policy barriers revolve around policies and laws impacting activity (e.g., a law prohibiting road hockey). The importance of identifying barriers within the aforementioned categories is that distinct intervention strategies may be required for barrier alleviation in the different categories (Brittain *et al.*, 2006; Gyurcsik *et al.*, 2006; McLeroy *et al.*, 1988).

In general, barriers have been identified as a consistent inverse correlate of PA across a diverse range of populations, including adolescents (Allison *et al.*, 1999); however, recent research suggests that although some barriers, such as a lack of motivation, are common across populations, other barriers are specific to the population under study (i.e., population-specific barriers) (Brawley *et al.*, 1998; Brittain *et al.*, 2006; Gyurcsik *et al.*, 2006). For example, among a study population of female and male students in grade 7 through the first year of university, barriers specific to participants in specific grades were found, such as social invitations during one's planned physical activity time (i.e., an interpersonal barrier) among first-year university students (Gyurcsik *et al.*, 2006). The institutional barrier of a heavy school workload was specific to grades

11-12 and first-year university students but not to students in grades 7-10. Further, of note, as grade in school increased, the number of reported barriers also increased.

To date, all prior research examining barriers to PA in youth and adolescents has focused on CA or school grade level, and there is no data on the role that puberty may have on perceived barriers to PA. As indicated previously, girls of the same age vary in their degree and timing of biological maturity (Malina *et al.*, 2004) and thus may experience different barriers to PA; for example, early maturing girls may be more likely to experience the barrier of concern over how one looks compared to less biologically mature girls (i.e. late maturers) (Allison *et al.*, 1999; O'dea, 2003; Tergerson & King, 2002).

The first purpose of my study was to describe the PA levels of adolescent girls (i.e., aged 8-16 years) by school grade and maturity status (i.e., early or late maturing) within grades. The second purpose was to identify perceived barriers to PA of adolescent girls by grade and maturity status within grades. I hypothesized that: (a) PA would decrease with increasing grade and (b) within grade groups, early maturing girls would participate in less PA than late maturing girls. Given the expected decline in PA with increasing CA and BA, I also hypothesized that: (a) the average number of barriers to PA would increase as grade in school increased and (b) within grade groups, early maturing girls would experience a greater number of barriers to PA that were specific to the timing of biological maturity, such as embarrassment over pubertal development, when compared to late maturing girls.

3.2 Methods

3.2.1 Participants

Anthropometric and barriers to PA data were available on 221 girls in grades 4 (n=21; 9-10 yrs), 5 (n=22; 10-11 yrs), 6 (n=19; 11-12 yrs), 7 (n=24; 12-13 yrs), 8 (n=22; 13-14 yrs), 9 (n=55; 14-15 yrs) and 10 (n=58; 15-16 yrs), at 5 different schools (3 elementary and 2 high schools). All schools were publicly funded and located in Saskatoon, Saskatchewan, Canada. Each child gave written assent and parental written informed consent was also obtained. All procedures were approved by the Institutional Research Ethics Board.

3.2.2 Anthropometry

Measurements were taken for stature, sitting height and body mass. Skinfold thickness at five sites of the body (subscapular, triceps, biceps, iliac crest and medial calf) were used to assess body fat. All measurements were performed by a Professional Fitness and Lifestyle Consultant certified by the Canadian Society for Exercise Physiology. Leg length was calculated by subtracting sitting height from stature. Girls' stature and body mass was used to calculate their BMI ($kg/(m^2)$).

3.2.3 Physical Maturity (Biological age)

A common maturity assessment technique in longitudinal studies is the determination of years from attainment of peak height velocity (PHV), (Malina *et al.*, 2004). Peak height velocity is an indicator of somatic maturity and reflects the age at maximum growth rate in stature during adolescence (age at PHV or APHV). In the

present cross-sectional study, measurement of BA was attained by predicting years from reaching PHV. This was achieved using a gender specific multiple regression equation that included measures of stature, sitting height, leg length, CA, and their interactions to predict how many years a girl is from APHV (Mirwald *et al.*, 2002) (Equation 1). Subtracting years from APHV from CA yields a predicted APHV.

Equation 1:

Years from reaching PHV = -9.376 + (0.0001882 * Leg Length and Sitting Height interaction) + (0.0022 * CA and leg length interaction) + (0.005841 * CA and Sitting Height interaction) + (0.002658 * Weight to Height ratio)

The prediction of APHV is most accurate around the attainment of PHV (approximately 12 years of age in girls (Malina *et al.*, 2004)). The elementary school girls' ages ranged from 9.3-14.3 (11.7 $\pm$ 1.4) years; thus, the regression equation was appropriate. The regression equation was deemed inaccurate for the high school girls' because their ages ranged from 14.1-16.7 (15.3 $\pm$ 0.6) years. Therefore, their biological maturity was estimated using a BA scale related to age of attainment of menarche. The girls were asked if they had experienced menses and if so, what month and year it had occurred. Age at menarche was calculated from date of menarche (month and year) and date of birth. A total of 4 girls (5%) had not attained menarche. For the maturity status assessment elementary school girls were grouped based on APHV quartiles and the high school girls on age at menarche quartiles. The top quartile was classified as 'early maturers' and the bottom quartile as 'late maturers'. Consequently, these two groups

indicate timing of biological maturity relative to the sample and are not intended to indicate either precocious or delayed puberty.

3.2.4 Physical Activity

Physical activity was objectively measured on 182 girls (82% of the sample) for seven consecutive days using calibrated Actical accelerometers (Mini Mitter Co., Inc., Bend, OR). Actical is an omnidirectional accelerometer which is sensitive to movement in all directions and is a valid and reliable measure of PA in children and adolescents (Puyau *et al.*, 2004). The monitors were worn on the hip as outlined in study 1. When worn on the hip the Actical is most sensitive to vertical movements of the torso and thus is designed for measurement of whole body PA. The Actical is sensitive to movements in the 0.5- to 3-Hz range which allows for detection of sedentary as well as high-energy movements (Puyau *et al.*, 2004); however, it will filter high-frequency movements. The filtered acceleration signal is digitized and integrated over a user-specified epoch interval. At the end of each epoch, the summed value or ‘‘activity count’’ is stored in memory. For the current study, 15 second epochs were used.

Similar to study 1, participants were asked to record when the monitor was put on in the morning and removed in the evening before bed. Upon completion of the data collection, the data were electronically downloaded resulting in a file containing 15 second movement counts for each girl. After data were scanned for spurious measures, sleep time was determined from the log sheets. Files with minimal levels of missing data underwent imputation procedures to ensure seven days of data were available for analysis. For more information on procedures see Esliger *et al.*, (2005) and Appendix G.

Physical activity data were presented as average accelerometer counts per day and average minutes spent in moderate to vigorous PA (MVPA) per day. Accelerometer counts per day were used to evaluate the raw data without influence of cut-points. The child/adolescent specific intensity cut-point of ≥ 1500 counts per minutes (Puyau *et al.*, 2004) were used to calculate minutes spent in MVPA.

3.2.5 Barriers to physical activity

Barriers to PA were assessed through a semi-structured open-ended survey. The survey was previously piloted for readability and understandability in a sample of grade 4-8 students (N=78) from a school not recruited for participation in the study. Minor modifications in wording were made to the survey for use in the actual study based on the pilot testing. The survey was not pilot tested in the high school students since the format, including the wording, was based on a similar survey used in previously published research with high school students (Gyurcsik *et al.*, 2006).

After the participants wore the accelerometers for one week (i.e., 7 days), the girls returned the accelerometers and data on barriers to PA were collected on all participants (n=221) through the semi-structured, open-ended survey. An investigator distributed surveys to the students during class and read the instructions on how to complete the questions. The grades 4-8 participants completed the survey during regularly scheduled class day and the grades 9-10 participants during a required physical education class. Participants were first asked "*Were there any physical activities that you would have liked to do but didn't over the last 7 days*?" If answered affirmatively, girls were then asked in an open fashion to list up to five barriers that prevented participation over the

last 7 days. This open-ended barrier measure was consistent with those used in other research (Gyurcsik *et al.*, 2002). Confidentiality of responses was assured.

3.2.6 Data analyses

Independent t-tests and one-way ANOVA were used to investigate the difference in descriptive statistics between grade (i.e. grades 4-6, grades 7 and 8, and grades 9 and 10) and maturity status groups (i.e. early and late), respectively. Differences in PA variables across the same grade and maturity groupings were obtained using a MANCOVA. Body fat (sum of five skinfolds) and CA (in the maturity group comparison only) were added as covariates. It was necessary to control for body fat in the analyses because higher body fat or overweight status is linked with both the independent (i.e. BA) and dependent (i.e. PA) variables (Davison *et al.*, 2003; Trost *et al.*, 2001). Physical activity data were log transformed due to the positive skewness of accelerometer counts and MVPA.

In line with previously published research (Brittain *et al.*, 2006; Gyurcsik *et al.*, 2006), two steps were followed to classify the barriers to PA listed by the participants. First, three researchers independently coded each barrier into one of four categories: intrapersonal, interpersonal, institutional or community. Using the ecological model (McLeroy *et al.*, 1988), the following definitions were provided for use in the classification: (a) Intrapersonal barriers are characteristics of the individual that may prevent PA, (b) Interpersonal barriers are formal and informal social networks and support systems that may prevent PA, (c) Institutional barriers occur within social institutions with organizational characteristics that may prevent PA, and (d) Community

barriers occur between organizations, institutions, and informal networks within defined boundaries that may prevent PA. The ecological group 'public policy' was not included in the present study as the majority of the girls were deemed too young to recognize barriers associated with public policy. This is supported by previous research (Gyurcsik *et al.*, 2006), where no public policy barriers were reported by adolescent girls. After classification, the three researchers discussed all barriers that were not similarly characterized (initially the researchers reached agreement on the categorization of 88% of all barriers), until agreement was reached.

To obtain descriptive statistics on the number of barrier categories and specific types of barriers within each category, percentages of reported barriers were calculated within the three grade groupings: (a) grades 4-6, n=62 (b) grades 7-8, n=46 (c) grades 9-10, n=113. Collapsing the grade groupings increased sample size and was used to investigate the whole adolescent period, while also considering the transition from elementary to high school. An ANOVA and independent t-tests were used to determine if the average number of barriers identified by girls differed among grade and maturity groupings, respectively. A chi-squared statistic was used to investigate if the number of responses in each barrier category differed by grade or maturity grouping. The alpha level was set at 0.05. SPSS (version 11.5) was used to analyze the data.

3.3 Results

3.3.1 Physical activity by grade groupings

Height and body mass were within reference standard ranges for all chronological ages (US Department of Health and Human Services, 2002). Girls in grades 7-8 and 9-10

were significantly taller and heavier, and had a greater BMI and sum of five skinfolds than girls in lower grade groupings (Table 3). Results from MANCOVA showed that accelerometer counts ($F[2,179] = 27.8$, $p < .05$) and MVPA ($F[2,179] = 32.6$, $p < .05$) significantly decreased with an increase in grade grouping. Bonferroni post-hoc tests revealed a significant difference in accelerometer counts and MVPA among all grade groupings (Table 3). Between grade 4 and grade 10, average counts decreased by 44.0% and MVPA decreased by 40.4%.

Table 3: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for girls in different grade groups

	Grade Grouping		
Measure	Grades 4-6 (n=58)	Grades 7-8 (n=43)	Grades 9-10 (n=81)
Age (yrs)	10.7 (0.9)	13.1 (0.6)	15.3 (0.6)
Weight (kg)	39.4 (9.7)	51.7 (10.0)	62.5 (13.2)
Height (cm)	145.1 (9.0)	159.6 (7.3)	164.9 (6.2)
BMI (kg/m^2)	18.5 (3.2)	20.3 (3.5)	22.9 (4.4)[a]
Sum 5 SF (mm)	53.5 (24.0)	55.4 (20.2)	77.5 (30.9)
Accelerometer Counts/day	758297.8 (306646.4)	568840.4 (302541.3)	424430.4 (191799.4)
MVPA (average min/d)	152.0 (57.8)	113.6 (57.1)	84.5 (34.7)

NB= A significant ($p<.05$) grade effect was noted for all variables except BMI ([a] = significantly ($p<.05$) difference between grades 7-8 and grades 9-10 only).

3.3.2 Physical activity by biological maturity groupings

The early maturing elementary school girls were taller and heavier ($p<.05$) when compared to late maturing girls (Table 4). In contrast there was no significant difference in the height or weight of early and late maturing high school girls. This represents the 'catch-up growth' of late matures. In terms of PA variables, a MANCOVA (controlling for body fat and CA) revealed no significant differences in accelerometer counts or

MVPA between early and late maturing elementary school girls. Likewise, there were no significant differences in accelerometer counts or MVPA between early and late maturing high school girls.

Table 4: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for early and late maturing elementary and high school girls

School	Measure	Maturity Status	
		Early Maturers (n=25)	Late Maturers (n=25)
Elementary (Grades 4-8)	Age (yrs)	11.5 (1.4)	12.2 (1.3)
	Weight (kg)	49.2 (10.7)*	38.7 (9.9)
	Height (cm)	158.0 (10.2)*	145.7 (10.2)
	BMI (kg/m^2)	19.7 (3.6)	17.9 (2.6)
	Sum 5 SF	55.4 (24.6)	46.9 (16.1)
	Accelerometer counts/day	803623 (318242)	685021 (357441)
	MVPA (average min/d)	158.9 (58.1)	138.6 (68.4)
		Early Maturers (n=19)	Late Maturers (n=19)
High (Grades 9-10)	Age (yrs)	15.3 (0.6)	15.4 (0.6)
	Weight (kg)	66.7 (12.6)	60.4 (14.2)
	Height (cm)	166.9 (5.1)	166.1 (4.7)
	BMI (kg/m^2)	23.9 (4.2)	21.8 (4.7)
	Sum 5 SF	82.1 (26.8)	66.1 (29.8)
	Accelerometer counts/min	385946 (183137)	455391 (257523)
	MVPA (average min/d)	75.0 (30.7)	89.2 (47.2)

* = significant ($P < .05$) difference between early and late maturing girls

3.3.3 Barriers to physical activity by grade groupings

Out of 221 girls, 160 (72.4%) listed a PA they would have liked to do but did not do over the previous seven days. In total, 374 barriers were listed by the 160 participants, with an average of 2.3 $\pm$ 1.4 barriers being reported per participant. When examined by grade group, the average number of barriers identified by girls in grades 4-6 was 2.2 $\pm$ 1.1, grades 7-8 was 2.4 $\pm$ 1.4 and grade 9-10 was 2.4 $\pm$ 1.4. A one way ANOVA

examined the grade effect. There were no differences in the frequency of barriers listed among the grade groups (p>.05).

3.3.4 Barrier categories by grade groupings

Figure 10 displays the overall percentage of barriers that were reported in each specific barrier category (intrapersonal, interpersonal, institutional and community) by grade groupings. A 4 (barrier category) x 3 (grade grouping) chi square test was significant (χ^2 (6; N=77) =20.4, p<.05), indicating that overall the number of responses in specific barrier categories was related to grade groupings. Follow-up tests using adjusted residuals showed that grades 4-6 participants cited significantly more interpersonal barriers (adjusted residuals=3.2, p<.05) and grades 9-10 participants cited significantly more institutional barriers (adjusted residuals = 2.7, p<.05).

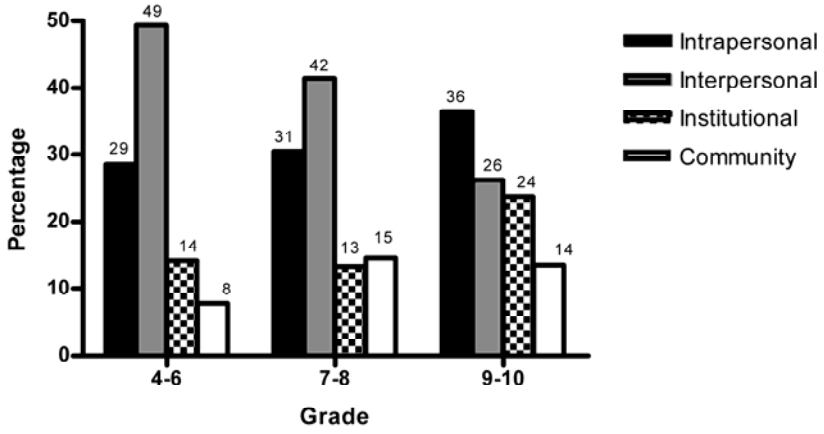


Figure 10: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by grade groupings. Note. Percentages for each grade group may not equal 100 due to rounding error.

3.3.4.1 Specific type of barriers by grade groupings

Tables 5 through 8 contain example descriptors of specific barrier types within the categories of intrapersonal, interpersonal, institutional or community. Some of the most apparent examples among these barriers are outlined below.

As shown in Table 5, the two specific intrapersonal barrier types of having an illness/injury (e.g. “I wanted to go to my hockey practice but I got sick”) and a lack of time (e.g. “[I] didn't have enough time to go running”) comprised 69% of all responses by grades 4-6, 52% of all responses by grade 7-8 participants, and 34% of all responses by grade 9-10 participants. Interestingly the intrapersonal barrier of lack of motivation/lazy (e.g. “I did not feel very motivated this week”) began to emerge, comprising 16% and 29% of all responses in grades 7-8 and grades 9-10, respectively. Also, the barrier of paid work (e.g. “I guess working evenings and weekends makes a lot less time for physical activity”) was a prominent barrier (27% of all responses) among participants in grades 9-10.

As seen in Table 6, the two specific types of interpersonal barriers, friends did not want to go with me (e.g. “I wanted to go swimming but my friends bailed”) and a person in authority said no to activity (e.g. “My mom said no because she didn't want to drive me”) comprised the majority of all responses by grades 4-6, grades 7-8 and grades 9-10 participants (85%, 71% and 89%, respectively). The intrapersonal barrier of coaches/teachers altering scheduling of activity (e.g. “I wanted to play soccer but my soccer practice was cancelled”) was cited among all grades but was more prominent among the elementary school participants (grade 4-8).

In terms of institutional barrier category, school physical education or extra-curricular activity scheduling (e.g. “I wanted to do physical activity at lunch but had drivers ed.”) and the timing of other organized activities conflicted (e.g. “I had to baby-sit at church instead of swimming”) encompassed 80% of all responses by grades 4-6, 50% of all responses by grades 7-8 and 34% of all responses by grades 9-10 (Table 7). Too much homework (e.g. “When I have too much homework I can't go skating”) emerged in grade 9-10 as the most prominent manifestation of this type of barrier (55% of all responses).

Some consistency existed across grades in terms of community barriers, with weather is too cold/snowing (e.g. “Too much snow, so I couldn't go for my usual dog walk”) comprising 83% of all grade 4-6, 68% of all grade 7-8 and 75% of all grade 9-10 responses (see Table 8). Although cited less frequently, the community barrier ‘the facility is too far away’ (e.g. “We didn't have a [swimming] pool close by) was reported consistently across grades (grades 4-6, 17%, grades 7-8, 17% and grades 9-10, 18%).

Table 5: Types of intrapersonal barriers listed by the participants

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Illness/Injury	46% (n=10)	24% (n=6)	25% (n=19)
Lack of time	23% (n=5)	28% (n=7)	9% (n=7)
Lack of motivation/Lazy	5% (n=1)	16% (n=4)	29% (n=22)
No Money to buy equipment or access facilities	14% (n=3)	16% (n=4)	15% (n=11)
Was doing something else	14% (n=3)	12% (n=4)	14% (n=3)
Paid Work	-	-	27% (n=2)
Didn’t want to/feel like it	-	-	7% (n=5)
[a]**Other**	-	4% (n=1)	-
Total Reported Frequency	22	25	75

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Table 6: The interpersonal barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Friends did not want to go with me	18% (n=7)	17% (n=6)	33% (n=18)
A person in authority (e.g. parent or older sibling) said no to activity	67% (n=26)	54% (n=19)	56% (n=30)
Coaches/teachers altered the scheduling of the activity	13% (n=5)	11 (n=4)	4% (n=2)
I could not get a ride	-	9 (n=3)	7% (n=4)
[a]Other	3% (n=1)	9 (n=3)	-
Total reported frequency	39	35	54

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Table 7: Institutional barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
School phys ed or extra-curricular activity scheduling	60% (n=6)	20% (n=2)	20% (n=10)
The timing of other organized activities conflicted	20% (n=2)	30% (n=3)	14% (n=7)
Too much homework to do activity	-	20% (n=2)	55% (n=27)
[a]Other	20% (n=2)	30% (n=3)	10% (n=5)
Total	10	10	49

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Table 8: Community barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Weather is too cold/Snowing	83% (n=5)	68% (n=8)	75% (n=21)
The community facility is too far away	17% (n=1)	17% (n=2)	18% (n=5)
[a]Other	-	17% (n=2)	7% (n=2)
Total	6	12	28

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

3.3.5 Barriers to physical activity by biological maturity groupings

There were no differences between maturity groups in the average number of barriers identified by early maturers (2.4 ± 1.2) and late maturers (2.5 ± 1.4).

3.3.5.1 Barrier categories by biological maturity groupings

Figures 11 and 12 display the overall percentage of barriers that were reported in each specific barrier category (intrapersonal, interpersonal, institutional and community) by maturity groupings for elementary and high school girls, respectively. The group was split by school (i.e. elementary and high school) because previous analyses showed that older girls experienced different barriers to PA than younger girls. Results from a 4 (barrier category) x 2 (maturity grouping) chi square test showed no significant associations. Thus, the overall frequency of responses in specific barrier categories was not related to maturity groupings for either elementary or high school students. No specific maturity-related barriers to PA were found.

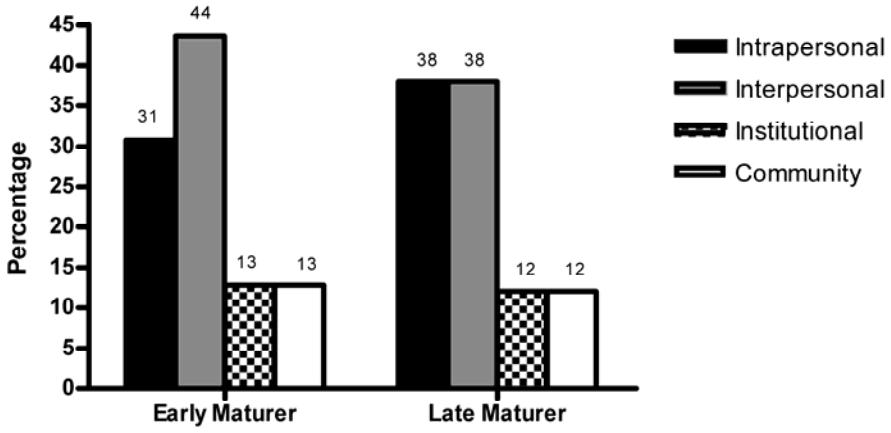


Figure 11: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the elementary school girls. Note. Percentages for each grade group may not equal 100 due to rounding error.

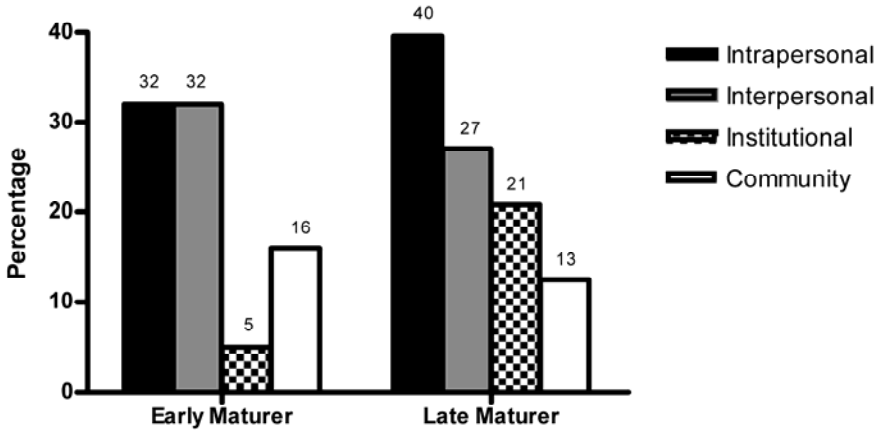


Figure 12: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the high school girls. Note. Percentages for each grade group may not equal 100 due to rounding error.

3.4 Discussion

3.4.1 Physical activity by grade and maturity groupings

The first objective of the study was to examine the levels of PA across grades in school and maturity groups. My finding that accelerometer assessed MVPA declined with increasing grade, supports previous research (Klasson-Heggebo & Anderssen, 2003; Riddoch *et al.*, 2004; Sherar *et al.*, 2007b; Thompson *et al.*, 2003; Troiano *et al.*, 2008; Trost *et al.*, 2002).

Until recently the specific relationship between levels of PA and biological maturity status had not been addressed. In the last year (i.e., January to December, 2007) five studies have been published which compare PA levels among maturity groups; these studies have mixed results. Niven and colleagues (2007) found no difference in self-reported PA among early, average and late maturing 11-year old girls. In contradiction, a longitudinal study of 2247 adolescent girls reported that early maturing girls in grades 7 and 8 participated in more minutes of self-reported VPA than late maturing girls;

however this association was not seen among girls in grades 9 through 11 (van Jaarsveld *et al.*, 2007). Baker and colleagues (2007) showed that early maturing girls (assessed via a combination of estradiol, breast development and mother's report on pubertal development using the pubertal development scale at age 11) participated in less accelerometer measured PA at age 13 when compared to late maturing girls. Likewise, a study by Riddoch and colleagues (2007) showed that parental reported pubic hair and breast development was inversely related to PA among 2933 eleven year old girls.

My results showing no significant differences in participation in MVPA between early and late maturing girls contradict findings from two other studies (Baker *et al.*, 2007; Riddoch *et al.*, 2007) that used an accelerometry. The contradictory finding could be related to the indicator(s) used to assess maturity. Maturity indicators (e.g. pubic hair stage, genital stages, APHV, age at menarche, skeletal age, etc.) are not equivalent. I used predicted APHV and recalled age at menarche as indicators of maturity. It is possible that development of secondary sexual characteristics, the maturity indicator featured in the aforementioned studies, is more closely related to the girls' disengagement from PA than timing of peak velocity in height and onset of menstruation. Secondary sex characteristics are the first overt sign of puberty, whereas PHV and menarche are later occurring pubertal events (Sherar *et al.*, 2004). The appearance of secondary sex characteristics, in particular breast development, may cause a girl to feel self conscious, or even uncomfortable about participating in PA (however there are no studies available to support or refute this suggestion). Although, in the present study, the elementary school girls with earlier predicted APHV and the high school girls with younger recalled age at menarche are likely more sexually mature, the direct relationship between development

of secondary sex characteristics and participation in PA could not be assessed. Wickle and Eisenmann (2007) also used predicted APHV to create maturity groups and found no relationship between maturity status and pedometer determined PA in 14-15 year old girls.

3.4.2 Barriers to physical activity by CA and BA

The hypothesis that the average number of barriers to PA would increase as grade increased was not supported. There were however, fluctuations in the types (i.e., diversity) of the ecologically-based barrier categories that were reported across grade groupings. Interpersonal barriers were the primary type of barrier category reported by elementary students in grades 7-8. Among the high school students, mainly intrapersonal and institutional barriers were reported. The latter finding is consistent with findings from a previous study that used an ecological framework to examine the pattern of barriers among students from grade 7 through first year of university (Gyurcsik *et al.*, 2006).

Fluctuations in the specific types of barriers within each ecological category across the grade groupings were also found. The finding that elementary school students did not report the institutional barrier of too much homework as a barrier, in contrast to the high reporting of this barrier among high school students, may be a reflection of the increased academic demands that occur during high school. Similar findings have been reported in other research (Allison *et al.*, 1999; Gyurcsik *et al.*, 2006). The intrapersonal barrier of a lack of motivation/lazy was frequently reported by participants in grades 9-10, which again parallels finding from previous research (Gyurcsik *et al.*, 2006). The mounting consistency of the reporting of these types of barriers by high school students

suggests that an effective intervention strategy may be one which helps high school students learn and become confident in their self-regulatory skills and abilities to cope with such barriers (Maddux & Gosselin, 2003; Woodgate *et al.*, 2005). For example, self-regulatory skills that may help students cope with homework and a lack of motivation barriers may include: a) setting goals in regards to amount of time spent doing regular PA and homework, b) self-monitoring of these behaviors and thus whether goals are being achieved, and c) the re-establishment of weekly goals on a continuous basis.

The reporting of the interpersonal barrier of a person in authority not permitting activity is noteworthy. Over half of the participants in grades 4-6, 7-8, and 9-10 reported this as a barrier. This finding suggests that opportunities to participate in PA may not be completely under the direct control of youth and adolescents of these particular chronological ages. As such, intervention strategies to make authority figures, such as parents and teachers, aware of their responsibility in creating PA opportunities for youth and adolescents may be effective.

My study is the first to consider a girl's maturity status (i.e. early or late maturing) in concert with perceived barriers to PA. The hypothesis that early maturing girls would experience more barriers to PA was not supported. No significant differences were evident in the frequency of reported barriers or in the specific types of barriers listed between early and late maturing girls. This finding was surprising considering that previous research has found that early pubertal maturation is associated with negative psychological well-being among girls (Graber *et al.*, 1997; Kaltiala-Heino *et al.*, 2003; Laitinen-Krispijn *et al.*, 1999); furthermore, a longitudinal study specifically designed to explore the relationships among maturity status, psychological well-being and PA in 178

adolescent girls found that early maturity at age 11 predicted lower psychological well-being at age 13 including depression (e.g. I hate myself, I have difficulty sleeping), global self-worth (e.g. I am often disappointed with myself), and weight related maturity fears (e.g. I don't like changes in my body because they make me feel fat) (Davison *et al.*, 2007). As such, population-specific barriers to PA that revolve around maturity should also be expected to be reported.

One plausible explanation for the lack of support for my hypothesis surrounding PA, barriers to PA and maturity status is the measure of BA used in my study versus perceptions of BA that may have existed by the study participants. That is, previous research (Michaud *et al.*, 2006) identifying the psychological traits and health behaviours of early and late maturing girls and boys have advocated the use of a subjective, self-report measure of BA. In such a measure, adolescents rate their pubertal development in relation to their peers. A subjective, self-report measure of physical maturity may be more closely related to the identification of specific maturity-related barriers to PA than an objective measure of physical maturity (as used in my study). An objective measure of maturity may have no personal meaning to the adolescent; an early maturing girl who does not perceive herself as early maturing will likely not experience/report maturity-related barriers to PA as a girl who does perceive herself as early maturing. Future research would benefit from examining the discourse between a subjective, self-report measure and an objective measure of maturity and their association with PA and barriers to PA.

3.5 Considerations

When interpreting these results it is necessary to consider the limitations of identifying early and late maturing girls through within-sample comparisons (i.e. maturity quartiles (Q); Q1=early maturity, Q4=late maturity). Many studies (Baker *et al.*, 2007; Davison *et al.*, 2007; Riddoch *et al.*, 2007; Niven *et al.*, 2007; Wickel & Eisenmann, 2007) exploring the characteristics of early and late maturing adolescents use within sample comparison to classify individuals into maturity groups. This is likely because, in general, adolescent girls are average maturing, leaving only a minority presenting extreme maturity. For example, 97% of the present elementary school sample's APHV fell within the range of 11.0-12.0 years (mean = 11.80, SD= 0.48 years). It may be that within a grade at school it is only the few girls presenting a degree of physical maturity which is extremely different from their peers who are at heightened risk for disengagement from PA. Future research examining the PA and barriers to PA of extreme early and late maturing girls is warranted.

I applied an ecological approach in the study of barriers to PA. The advantage of an ecological approach is that a range of personal and situation barriers are captured, which may require different intervention strategies (McLeroy *et al.*, 1988). For example, to alleviate the impact of salient intrapersonal barriers, which may be of particular relevance to high school students, one strategy might be to target an improvement in the management of extra curricular time (e.g. paid work and homework). An additional advantage of my study is the use of objective monitoring of PA. The limitations of self report measures of PA, in particular biases to do with accurate recalling of PA participation, are well recognized (Sallis & Saelens, 2000; Shephard, 2003).

My results are limited by several factors: First, due to the cross sectional study design, information on the stability and variability of barrier categories and specific barrier types was not obtained. Second, one must not assume that because certain barriers were reported by a greater number of participants, that such barriers are the most salient. Rather, to capture saliency, both the frequency with which a specific barrier occurs as well as the extent to which a specific barrier limits PA must be assessed (see Brawley *et al.*, (1998) for a review of this issue). Third, the results pertained to a select group of youth and thus may not be generalizable. Finally, biological maturity was assessed through predicting APHV among the elementary school girls and recalled age at menarche among the high school girls. Predicting APHV is likely less accurate than when observed in a longitudinal study and recalling age at menarche is subject to error.

3.6 Conclusion

Adolescent girls from grades 4-10 displayed a decrease in MVPA with increasing grade. However, when grouped by maturity status (early and late maturers) no differences were found in PA levels between maturity groups. Girls reported a multitude of barriers to PA within all grades. Types of barriers were dependent on grade but not maturity status. I therefore suggest that future research should aim to identify salient (i.e., frequent and limiting) personal and situational barriers to PA by CA in youth and adolescents. Once reliably identified, multi-pronged intervention strategies to help youth and adolescents cope with their salient barriers can be tested for effectiveness.

3.7 Acknowledgements

Sherar holds a Community and Population Health Research (CPHR) scholarship. The research was funded by a Saskatchewan Health and Research Foundation (SHRF) grant, a CPHR grant and a North American Society for Paediatric Exercise Medicine (NASPEM) grant.

Chapter 4: Study 3

Longitudinal development of fat mass and physical activity during adolescence and cardiometabolic risk in young adulthood

4.1 Introduction

The metabolic syndrome, defined as a clustering of adverse cardiovascular disease (CVD) risk factors; including elevated abdominal fat, blood pressure and triglycerides, lowered high-density lipoprotein, and insulin resistance, has become a major public health challenge world-wide (Eckel *et al.*, 2005). An aspect of the metabolic syndrome is that it identifies individuals at high risk of CVD and type 2 diabetes.

The concept of 'critical' or 'sensitive' periods of growth, during which the antecedents of 'adult' diseases may be initiated has been studied, mostly with regards to 'fetal life' (Barker, 2000). Recent research, however, has investigated how later periods of growth, such as the period of adolescence, might be also considered 'critical' in the development of chronic disease (Ovesen, 2006). It has been suggested that growth, maturation, and 'adult' diseases are intimately linked to each other in that environmental stimuli during adolescence (and other critical periods) can have a profound, permanent impact on growth and function (Cameron & Demerath, 2002). Thus, it is now recognized that CVD, and thus the metabolic syndrome, is partly a pediatric problem; that is, the antecedents of CVD lies in childhood and adolescence (Berenson *et al.*, 1998), even though the clinical symptoms of this disease do not become apparent until much later in life.

Although the metabolic syndrome is thought to be mainly a consequence of obesity, (Grundy, 2000; Sun *et al.*, 2008) the mechanisms underpinning its development are not that well understood. Other correlates for the metabolic syndrome include low levels of physical activity (PA) (Ferreira *et al.*, 2005; Lakka *et al.*, 2003), early biological maturity (Feng *et al.*, 2008; Heys *et al.*, 2007) and excessive energy and/or unbalanced nutrient intake (Ferreira *et al.*, 2005; Grundy *et al.*, 2002). Understanding the adolescent and young adult development of biological and lifestyle variables associated with adult cardiometabolic risk is important in determining optimum preventative measures (Lenfant & Savage, 1995) and ultimately should help reduce CVD mortality.

Previous cross sectional studies have examined the relationship between adolescent overweight/obesity (DiPietro *et al.*, 1994; Must *et al.*, 1992) and PA (Hasselstrom *et al.*, 2002; Lefevre *et al.*, 2002; Twisk *et al.*, 2002) and adult cardiometabolic risk, or presence of CVD/metabolic syndrome. Cross-sectional studies are limited, however, as assessing the independent impact of lifestyle behaviors on fat development in adolescence and relating it to adult cardiometabolic risk is problematic because normal growth-related body composition changes may mask or be greater than the lifestyle effects. Therefore, only longitudinal studies, which identify each individual's pattern of fat mass development (i.e. growth trajectories) from adolescence into young adulthood, can examine the independent contribution of closely interrelated lifestyle and biological risk variables.

During adolescence there is a dramatic increase in the velocity of growth in body composition (e.g. proportions of muscle, fat and bone). There is also variation in the rate and magnitude of linear growth both within- and between- sexes of the same

chronological age (CA). Thus, when assessing body composition during adolescence, one needs to control for differences in biological maturity within individuals of the same CA and sex. There is also evidence to suggest that behaviours such as PA may be more closely related to biological maturity than to CA (Sherar *et al.*, 2007b, Thompson *et al.*, 2003). One method, which has been used frequently in longitudinal studies of growth is to align fat mass and PA trajectories to a biological maturity age (such as years from age at peak height velocity; APHV), rather than CA.

In summary, it is necessary to control for individual variation in timing of growth and maturation by examining the effects of adolescent biological and lifestyle factors on growth at different biological ages (i.e. through adolescent-to-adult developmental trajectories), rather than only at one time point (i.e. cross-sectional analyses) to better understand how the period of adolescence contributes to cardiometabolic risk in early adulthood. The aim of this study was to conduct a retrospective analysis of prospective data to examine total body fat mass (TBFM), trunk FM and PA developmental trajectories (aligned to biological maturity age) of individuals categorized as low and high for cardiometabolic risk at 26 years, while investigating biological and lifestyle risk factors. Three hypotheses were addressed.

Hypothesis 1: Early maturity in males and females is associated with higher cardiometabolic risk at 26 years.

Hypothesis 2: Males and females with greater fat mass development (TBFM and trunk FM) during childhood, adolescence and young adulthood have greater cardiometabolic risk at 26 years. Furthermore, early biological maturity, greater dietary fat intake and less PA are associated with greater FM development.

Hypothesis 3: Males and females who participate in less PA during childhood, adolescence and young adulthood have greater cardiometabolic risk at 26 years. Furthermore, early biological maturity and greater TBFM is associated with less PA.

4.2 Methods

4.2.1 Participants

Participants were part of the University of Saskatchewan's Pediatric Bone Mineral Accrual Study (PBMAS; 1991-2006), which has been described in detail elsewhere (Bailey, 1997). In brief, the study utilized a mixed-longitudinal design and incorporated eight age cohorts. The cohorts were aged between 8 and 15 years at baseline. During the seven years of childhood and adolescent annual data collection, the composition of these clusters remained the same. Eligible children had no history of chronic disease or long-term medication use. Between 1991 and 1993, written informed consent was obtained from 251 parents and their children. Of the 94% of participants whose race was known, 95% were Caucasian. By 1997, 197 individuals had been assessed on two or more occasions. In 2002 participants were re-contacted and tested annually between 2003 and 2006. Of the 197 participants with longitudinal data, 169 individuals participated as young adults. For the present analysis two samples of the PBMAS data set were selected using specific inclusion criteria. Sample 1 (male n=21; female n=27) included all participants who had blood sampling at the last test occasion (approximately 26 years of age). Sample 2 included all participants who had a measurement of blood pressure (BP) at the last testing occasion (male n=55; female

n=76). Finally, for inclusion in the analysis participants required a measured APHV during adolescence.

Tables 9 and 10 provides a summary of the number of individuals assessed at each biological maturity age (years from APHV) for sample 1 and sample 2, respectively. For sample 1, the median number of testing occasions per female is 11 (min 6, max 12) and per male is 10 (min 7, max 11). For sample 2, the median number of testing occasions per female is 10 (min 7, max 12) and per male is 11 (min 4, max 12).

The study received approval from the University and Hospital Advisory Committee on Ethics in Human Experimentation

Table 9: Number of visits (i.e. DXA scans) for sample 1 (the sub sample)

	Biological Maturity Age (yrs from APHV)																									
	-6	-5	-4	-3	-2	-1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Total
Females	0	0	3	7	9	16	20	23	22	23	19	16	12	10	7	8	13	11	16	13	14	5	7	1	1	276
Males	1	1	4	7	8	13	16	20	19	16	13	12	9	8	6	7	9	11	14	9	10	4	4	1	0	222
Total	1	1	7	14	17	29	36	43	41	39	32	28	21	18	13	15	22	22	30	22	24	9	11	2	276	549

Sample 1 (N=48) has full metabolic syndrome information at the last testing occasion

Table 10: Number of visits (i.e. DXA scans) for sample 2 (whole sample)

	Biological Maturity Age (yrs (from APHV)																									
	-6	-5	-4	-3	-2	-1	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	Total
Females	0	1	13	22	28	41	56	63	64	62	53	46	35	29	25	20	31	30	38	39	36	22	15	5	1	775
Males	2	6	13	21	26	37	47	49	47	39	31	23	20	16	16	16	24	30	30	23	19	11	7	1	0	554
Total	2	7	26	43	54	78	103	112	111	101	84	69	55	45	41	36	55	60	68	62	55	33	22	6	1	1329

Sample 2 (N=131) has recorded blood pressure at the last testing occasion

4.2.2 Chronological age

Decimal age was calculated by subtracting date of birth from the measurement date. Chronological age categories were constructed using 1-year intervals from the midpoints of the age; for example, the 10-yr age group included subjects from 9.50 to 10.49 yr of age.

4.2.3 Anthropometry

Standing height was measured biannually as stretch stature, using a wall-mounted stadiometer (Holtain Ltd, Crymych, Dyfed, UK**),** and recorded to ± 1mm. Body mass was measured on a digital scale (Toledo Honest Weight, CA) accurate to ± 0.05 kg. Body mass index (BMI) was calculated (weight (kg) / height (m^2)).

4.2.4 Maturational Assessment

Peak height velocity in adolescence was determined by plotting individual's changes in height and establishing the peak growth period with a cubic spline procedure (GraphPad Prism version 3.00 for Windows, GraphPad Software, San Diego, CA) (Bailey *et al.*, 2000). A cubic spline employs interpolating cubic polynomials, which uses information from neighboring points to obtain a degree of global smoothness. The cubic spline procedure was chosen over other curve fitting protocols because it maintains the integrity of the data without transforming or modifying the underlying growth characteristics.

Biological maturity age was determined by subtracting the CA of an individual at the time of measurement from APHV in males and females. Years from APHV results in

a continuous measure of biological age. Biological age categories were constructed using 1-year intervals such that -1 APHV age group included observations between -0.49 and -1.50 years from (i.e., before) APHV. Individuals were grouped into maturity status groups depending on their APHV. Males were classified as an early maturer if APHV <13.0 yrs, as a late maturer is APHV > 15.0 years; all others were classified as average maturers. Females were classified as an early maturer if APHV <11.0 yrs, as a late maturer is APHV > 13.0 years; all others were classified as average maturers. These cut-points are used because APHV occurs, on average, close to 12 years in girls and 14 years in boys with a standard deviation of approximately 1.0 year (Malina *et al.*, 2004).

4.2.5 Body Composition

During the childhood and young adulthood testing periods, participants were scanned annually using dual energy X-ray absorptiometry (DXA) (Hologic 2000 QDR, Hologic, Waltham, MA) in the array mode, employing enhanced global software version 7.10. Standard procedures and positioning were adopted and strictly followed to maximize consistency (Hologic, 1993). Participants removed metal objects (e.g., jewelry, glasses) and shoes and wore loose-fitting clothes during the scanning procedure (Bailey, 1997). Whole-body scans were analyzed using software version 5.67A and all scans were analyzed by one of two qualified individuals to minimize operator-related variability. Data on total body fat mass (TBFM) and trunk fat mass (FM) were recorded in grams and as a relative value to total body weight (%). Short-term precision in vivo for TBFM, expressed as a coefficient of variation (%) was 2.95% and for trunk fat was 4.88%.

4.2.6 Assessment of Physical Activity

Physical activity levels were assessed a minimum of three times per year for the first two years of the study (1991-1992) and twice yearly thereafter (1993-1997) using the Physical Activity Questionnaire for Children (PAQ–C) and/or Adolescents (PAQ–A) (Crocker *et al.*, 1997). This instrument scores nine items on a 5- point scale, with a higher value indicating higher levels of PA. A mean composite score of activity was calculated from all measurement occasions. The PAQ–C questionnaire has consistently demonstrated good internal consistency and validity with moderate relationships to teacher evaluations of activity, Caltrac motion sensors, 7-d activity recalls, step tests of fitness, and leisure-time activity scales (Crocker *et al.*, 1997). More recently, the PAQ-C and PAQ-A were compared against an Actigraph accelerometer and were found to have good internal consistency and reliability (Janz *et al.*, 2008). In adulthood (2003-2006) the PAQ-AD, a 7-item version of the childhood questionnaires (PAQ-C and PAQ-A) was used.

4.2.7 Nutritional Assessment

Dietary intake and nutrition information was collected via a self-reported, 24-hour recall questionnaire. In the first 3 years of the study (1991-1993) the questionnaire was administered three times. Thereafter the questionnaire was administered twice a year (1994-1997, 2003-2006). For the analysis, yearly averages were calculated and aligned with the body composition measures. Each participant was given a 20-min training session on food portion sizes at baseline. At each subsequent recall session display, boards with life-size pictures of food and portion sizes were available. Nutrient

composition based on the 1988 Canadian nutrient file was used to analyze the 24-hour recall data. The data was then coded and the same individual checked all forms to ensure consistent interpretation (Whiting *et al.*, 2001). Average total energy intake per day ($kcal \cdot d^{-1}$) and average total fat intake per day ($g \cdot d^{-1}$) were calculated. A fat index variable was calculated to determine fat consumption per 1000 Kcal (total fat /1000 total kcal).

4.2.8 Adult smoking status

Because of the relationship between smoking behaviour and presence of metabolic syndrome risk and central distribution of body fat (Chiolero *et al.*, 2008), participants were asked about their past and current smoking history. Based on the information provided participants were classified as non-smokers, past smokers (if they had given up for ≥ 1 year), and smokers.

4.2.9 Markers of Metabolic Syndrome

Markers of metabolic syndrome were measured once in 2006. Resting BP and heart rate were assessed once using an automated cuff (Omron, M6 comfort). Mean arterial pressure (MAP) was calculated from diastolic blood pressure (DBP) and systolic blood pressure (SBP) using the following formula; ([(2*DBP)+SBP]/3). A fasting blood sample was also taken. Prior to blood tests a Medication History and Fasting Questionnaire was completed. The following serum markers were measured using automated techniques (Royal University Hospital): total cholesterol, high-density lipoprotein cholesterol (HDL-C), triglycerides (TG), fasting glucose and fasting insulin. Homeostasis model assessment for insulin resistance (HOMA-IR) was derived from

fasting glucose and insulin values ([fasting insulin (U/ml)] * [fasting glucose (mmol/l)]}/22.5) (Matthews *et al.*, 1985).

4.10 Cardiometabolic risk score

A quantitative composite score was chosen to represent adult cardiometabolic risk because the PBMAS participants are relatively young and healthy (i.e., the number of individuals with the metabolic syndrome will be low). To account for age and smoking, adult metabolic syndrome variables were regressed onto CA and smoking status (non, past [given up for ≥ 1yr], present) at the final adult testing occasion, and the standardized residuals saved. Two composite risk scores were calculated for males and females separately. The first cardiometabolic risk score was derived from the sub sample (sample 1) by adding the standardized residuals for TG, HOMA, MAP and inverted HDL-C. This method has been used previously to provide a continuous cardiometabolic risk score (Eisenmann *et al.*, 2004). These variables were chosen because they are consistent with the variables used in the NCEP (2001) definition of metabolic syndrome. However, because FM is the variable of interest, an obesity-independent metabolic syndrome score was developed (i.e. waist circumference was excluded). Due to the fact that a full metabolic syndrome risk profile was only available on a small sub sample, a second score was derived from the larger sample (sample 2) which had blood pressure measurements at the last testing occasion (whole sample). The score for sample 2 represents risk for high blood pressure (and thus a higher risk for the metabolic syndrome) and was calculated by summing the standardized residuals for MAP, regressed on to CA and smoking status. In both sample 1 and 2 a lower score is indicative of a better

cardiometabolic risk factor profile. High and low risk groups were determined based on a sex- specific median split of risk scores.

4.2.11 Data Analysis

Descriptive characteristics are expressed as mean $\pm$ standard error of the mean (SEM) (SPSS version 11.5, SPSS Inc., Chicago, IL). Sex differences were examined by independent t-tests ($p< 0.05$). Hypothesis 1 was addressed using a one-way analysis of variance (ANOVA) test. To test hypotheses 2 and 3, a prospective analysis of the longitudinal data was conducted using hierarchical (multilevel) linear modeling using random effects models (MlwiN version 1.0, Multilevel Models Project, Institute of Education, University of London, London, UK). This procedure has been described in detail previously (Goldstein *et al.*, 1998). Separate additive and gender-specific multilevel regression models were developed to describe the developmental changes in TBFM (g), Trunk FM (g) and PA (score 1 to 5) as follows;

$$y_{ij} = \alpha_j + \beta_j x_{ij} + k_1 z_{ij} + \ldots + k_n z_{ij} + \varepsilon_{ij}$$

where y is the modeled variable (i.e. TBFM, trunk FM or PA) on the measurement occasion i in the jth individual, and α_j is the constant for the jth individual, $\beta_j x_{ij}$ is slope of the modeled time variable (e.g. with biological age (years from APHV)) for the jth individual, and k_1 to k_n are the coefficients of various explanatory variables (e.g., risk group (0=low, 1=high), maturity category (0=early, 1=average, 2=late), PA (PAQ score 1–5) and dietary fat index) at assessment occasion i in the jth individual, and ε_{ij} is the level 1 residual (within individual variance) for the *i*th assessment of the modeled

variable in the *j*th individual and the level 2 residual variance (between individual variance).

4.2.11.1 Modeling strategy

Models were built in a stepwise procedure; that is, predictor variables (k-fixed effects) were added one at a time. Likelihood ratio statistics were used to determine whether the effects of independent variables were significant contributors to the model. The difference in likelihoods between two models follows a chi-square distribution; this difference was compared against the degrees of freedom lost to determine whether one model was a significant improvement over the other. In this way, predictor variables were added to the models and retained if deviance improved and/or if the variances at levels 1 and 2 were reduced. Predictor variables (k) were accepted as significant if the estimated mean coefficient was greater than twice the standard error of the estimate (SEE) $P<$ 0.05)). If the retention criteria were not met, the predictor variable was discarded. Biological age was added as both a fixed and random coefficient. To allow for the nonlinearity of growth, age power functions (biological age^2) were added to the models as fixed effects. Once biological age and height were modeled, PA (or TBFM in the PA model), dietary fat index, metabolic syndrome risk group and maturity groups (and their interactions) were incorporated into the models and their independent effects were tested. Alpha level was set at P=0.05.

4.3 Results

Average height and body mass approximated 50th percentile at all chronological ages in both sexes (US Department of Health and Human Services, 2002). There were no

significant (P>0.05) difference in CA, APHV, BMI, TBFM, Trunk FM, SBP, DBP or PA at age 26 between participants who had the full metabolic syndrome measures (i.e. sample 1; N=48) and those that did not (i.e. only had information on BP; N=76). Using NCEP ATP III guidelines (2002), 2 men and 2 women (8.8% of sample 1) were classified as having the metabolic syndrome.

Descriptive characteristics of subjects at age 26 are shown in Table 11. High risk (using the MAP risk scores) males and females were significantly heavier, had a greater BMI, TBFM percentage, trunk FM percentage, DBP, MAP and risk score. High risk females also had a significantly higher SBP than low risk females (table 5).

Table 11: Descriptive (mean + SD) statistics for all male (N=55) and female (N=76) participants at age 26

	Males	
	Low Risk (*N*=27)	**High Risk (*N*=28)**
Chronological age (yrs)	26.3 (2.3)	26.5 (2.1)
Age at PHV (yrs)	13.2 (1.1)	13.4 (1.0)
Height (cm)	177.6 (7.2)	180.2 (6.4)
Weight (kg)	77.3 (9.2)	94.0 (19.4)*
BMI (kg/m^2)	24.5 (2.8)	28.9 (5.4)*
Total Body Percent Fat	20.4 (6.0)	25.0 (7.9)*
Trunk Percent Fat	18.2 (8.0)	25.0 (10.1)*
Systolic Blood Pressure (mmHg)	112.7 (24.6)	123.6 (25.6)
Diastolic Blood Pressure (mmHg)	73.7 (7.9)	95.6 (21.3)*
Mean arterial pressure (mmHg)	86.7 (7.9)	104.9 (9.0)*
Risk Score	-0.7 (0.6)	0.7 (0.7)*
	Females	
	Low Risk (*N*=38)	**High Risk (*N*=38)**
Chronological age (yrs)	25.6 (2.3)	25.8 (2.3)
Age at PHV (yrs)	12.0 (0.9)	11.9 (0.9)
Height (cm)	165.9 (6.2)	167.0 (6.6)
Weight (kg)	63.3 (11.8)	79.5 (19.9)*
BMI (kg/m^2)	22.9 (3.7)	28.5 (6.9)*
Total Body Percent Fat	33.4 (7.6)	40.8 (8.8)*
Trunk Percent Fat	38.2 (9.3)	26.6 (12.3)*
Diastolic Blood Pressure (mmHg)	70.5 (7.8)	89.2 (15.8)*
Systolic Blood Pressure (mmHg)	104.7 (10.4)	115.4 (19.9)*
Mean arterial pressure (mmHg)	81.9 (5.1)	97.9 (6.7)*
Risk Score	-0.8 (0.5)	0.8 (0.7)*

* = significant (P<0.05) difference between groups; risk score = sum of standardized mean arterial pressure, regressed on to chronological age and smoking status.

Table 12 shows the young adult (aged 26 years) MAP risk scores for early, average and late maturing males and females. In males, ANOVA test revealed no significance difference in adult cardiometabolic risk score among maturity groups ($F[2,54] = 0.74$, $p > .05$). Likewise, in females there were no significance difference in the adult cardiometabolic risk score among maturity groups ($F[2,75] = 0.05$, $p > .05$).

Table 12: The risk score for males and females by maturity group (early, average and late)

	Males			Females		
Maturity group	*N*	Mean	SD	*N*	Mean	SD
Early	19	0.10	1.1	10	-0.31	1.08
Average	32	-0.12	0.93	60	-0.01	1.0
Late	4	0.44	1.06	6	0.12	0.73

Risk score = sum of standardized mean arterial pressure, regressed on to chronological age and smoking status.

Early maturing males =APHV <13.0 yrs, average maturing males = APHV 13.01-15.0 yrs, as a late maturer is APHV > 15.0 years; Early maturing females = APHV <11.0 yrs, average maturing female 11.01-13.0, late maturing female = APHV > 13.0 years.

Figure 13 shows the sex-specific developmental patterns of percent TBFM, percent trunk FM and PA of individuals at high and low cardiometabolic risk. Percent TBFM and trunk FM show the general age-related incline in both males and females following PHV (Figure 13a-d). Visual inspection of the graphs suggests that individuals at higher cardiometabolic risk at 26 have greater fat mass development (both total body and trunk) at all ages. Both males and females show the characteristic decline in PA with age (figure 13 e and 13f). Visual inspection of the graphs suggests that there is no difference in PA score between males and females at low and high cardiometabolic risk.

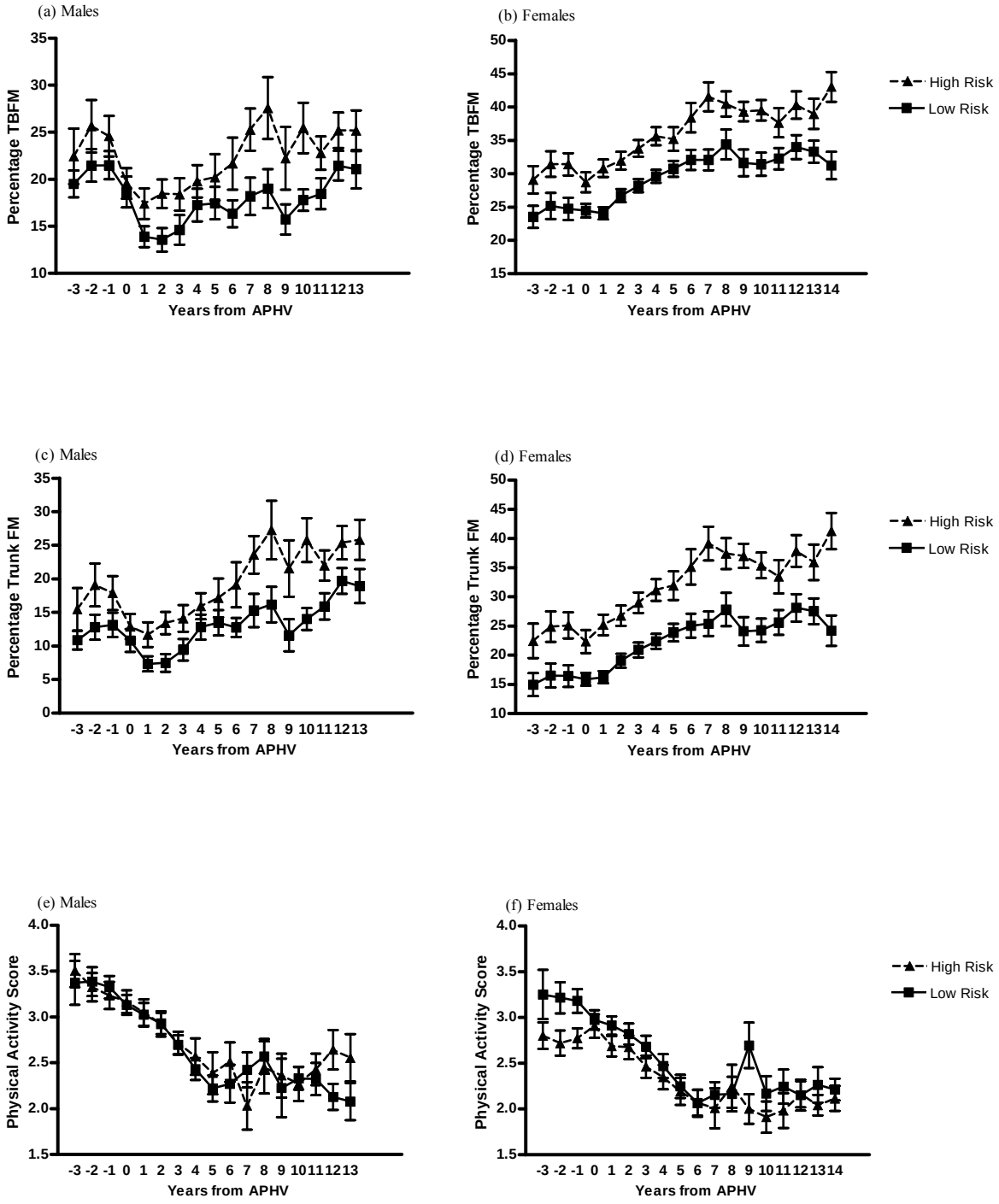


Figure 13: Total body percent fat (a and b), trunk percent fat (c and d) and physical activity (e and f) by biological maturity age (years from APHV) for individuals classified as high and low for cardiometabolic risk at age 26

Results from the multilevel models for males and females TBFM, trunk FM and PA, respectively are summarized in Tables 13-15. The random effects coefficients describe the two levels of variance [within individuals (level 1 of the hierarchy) and between individuals (level 2 of the hierarchy)]. For all 12 sex-specific models, the significant variances at level 1 indicate that FM (TBFM and trunk FM) or PA were significantly different at each measurement occasion within individuals (E > 2*SEE; $P<0.05$). The between-individuals variance matrix (level 2) for each model indicates that individuals had significantly different TBFM, trunk FM or PA curves both in terms of their intercepts (constant/constant, $P<0.05$) and the slopes of their lines (biological age/biological age, $P<0.05$)). The variance of these intercepts and slopes were significantly correlated (constant/biological age, $P > 0.05$) in some models. The fixed effects give the estimates for the coefficients of the various explanatory variables.

4.3.1 Model 1: Total body Fat Mass (TBFM)

4.3.1.1 Males

Once biological age (years from APHV) and height effects were controlled, there were no significant independent effect of maturity category or cardiometabolic risk group on TBFM development in sample 1 (Table 13a). In contrast, there was a significant independent PA and dietary fat effect, in that increasing PA significantly reduced TBFM development (-0.09 $\pm$ 0.03 log g) and dietary fat intake significantly increased TBFM independent of the other variables (0.005 $\pm$ 0.002 log g). Sample 2 revealed the same results as sample 1 (Table 13b); however, risk group was shown to have an independent

effect. Males in the high risk group had significantly greater (0.39 $\pm$ 0.1 log g) TBFM development.

4.3.1.2 Females

In sample 1, when biological age and height were controlled, neither PA nor dietary fat intake significantly affected TBFM development. However, females in the high risk group and of late maturity had greater TBFM (0.44 ± 0.09, 0.38 ± 0.09 log g, respectively). As in sample 1, high risk females of sample 2 had greater TBFM (0.32 ± 0.07 log g,); however, maturity group had no effect.

4.3.2 Model 2: Trunk FM

4.3.2.1 Males

In sample 1 (Table 14a), when biological age and height are controlled, PA significantly decreases (-0.16 ± 0.05 log g) trunk FM, and dietary fat significantly increases (0.009 ± 0.003 log g) trunk FM. Cardiometabolic risk or maturity group had no independent effect. Consistent with sample 1, in sample 2 (Table 14b) PA significantly decreases (-0.1 ± 0.03 log g) trunk FM and dietary fat significantly increases (0.005 ± 0.002 log g) trunk FM. However, risk group did have an independent effect; males in the high risk group had significantly greater trunk FM (0.61 ± 0.14 log g) at all ages.

4.3.2.2 Females

When biological age and height were controlled, females from sample 1 (Table 14c) who were in the high risk group and late maturers showed an increase in Trunk FM

(0.68 ± 0.14 and 0.57 ± 0.13 log g, respectively) at all ages. Physical activity and dietary fat showed no significant effect. In sample 2 (Table 14c) females in the high risk group had greater trunk FM development (0.53 ± 0.1 log g); however, maturity status was no longer shown to have an independent effect.

4.3.3 Model 3: Physical Activity

4.3.3.1 Males

In sample 1 (Table 15a), increased TBFM and dietary fat intake was shown to be significantly associated with decreased PA (-0.00002 ± 0.000008 and 0.01 ± 0.005, respectively). No independent effects were found for maturity or risk group. Only TBF was shown to be significantly related to PA in sample 2 (-0.000009 ± 0.000003; Table 15b).

4.3.3.2 Females

In sample 1 (Table 15c) TBFM and dietary fat were significantly related to PA (-0.007 ± 0.004 and -0.00001 ± 0.000006, respectively). No independent effects were found for maturity or risk group. In keeping with males from sample 2, only TBFM was shown to be significantly related to PA among females in sample 2 (-0.000007 ± 0.000004; Table 15d).

Table 13: Multilevel regression models for total body fat mass development of males and females aligned by biological age

	Male				Female			
	a) Sample 1		**b) Sample 2**		**c) Sample 1**		**d) Sample 2**	
Fixed Effect	Estimates		Estimates		Estimates		Estimates	
Constant	10.91 ± 0.63		11.15 ± 0.42		6.76 ± 0.38		6.9 ± 0.28	
Biological Age	0.11 ± 0.02		0.14 ± 0.01		0.07 ± 0.01		0.08 ± 0.008	
Biological Age ^{2}	-0.004 ± 0.001		-0.005 ± 0.0008		-0.002 ± 0.0007		-0.003 ± 0.0005	
Height	-0.01 ± 0.004		-0.01 ± 0.003		0.01 ± 0.002		0.02 ± 0.002	
Physical activity	-0.09 ± 0.03		-0.06 ± 0.02		NS		NS	
Fat Intake	0.005 ± 0.002		0.002 ± 0.002		NS		NS	
Mat Cat	NS		NS		0.38 ± 0.09		NS	
Risk Group	NS		0.39 ± 0.1		0.44 ± 0.09		0.32 ± 0.07	
Random Effects	**Level 1**		**Level 1**		**Level 1**		**Level 1**	
Constant	0.03 ± 0.003		0.04 ± 0.003		0.02 ± 0.002		0.02 ± 0.001	
	Level 2		**Level 2**		**Level 2**		**Level 2**	
	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age
Constant	0.29 ± 0.09	NS	0.19 ± 0.04	-0.008 ± 0.002	0.05 ± 0.02	NS	0.1 ± 0.02	NS
Biological Age	NS	0.001± 0.0003	-0.008 ± 0.002	0.001 ± 0.0002	NS	0.0005 ± 0.0002	NS	0.0005 ± 0.0001

Fixed effect values are Estimated Mean Coefficients ± SEE (Standard Error Estimate) of Total Body Fat Mass (log g)
Random effects values Estimated Mean Variance ± SEE [Total Body Fat Mass (log g)^2].
Biological Age (BA) is years from age at peak height velocity (PHV)(yrs); Height (cm); Physical Activity Score (1=low, 5=high); Total Fat Index (daily total fat intake (g)/1000 (Kcal)); Mat Cat (Maturity Category: 0=early, 1=average, 2=Late) Cardiometabolic Risk Group (0= low, 1=High)

Table 14: Multilevel regression models for trunk fat mass development of males and females aligned by biological age

	Male				**Female**			
	(a) Sample 1		**(b) Sample 2**		**(c) Sample 1**		**(d) Sample 2**	
Fixed Effect	Estimates		Estimates		Estimates		Estimates	
Constant	12.07 ± 0.97		11.9 ± 0.66		4.89 ± 0.56		5.08 ± 0.39	
Biological Age	0.22 ± 0.03		0.26 ± 0.02		0.1 ± 0.02		0.11 ± 0.01	
Biological Age ^{2}	-0.008 ± 0.002		-0.01 ± 0.001		-0.004 ± 0.001		-0.004 ± 0.0006	
Height	-0.03 ± 0.006		-0.03 ± 0.004		0.02 ± 0.004		0.02 ± 0.002	
Physical activity	-0.16 ± 0.05		-0.1 ± 0.03		NS		NS	
Fat Intake	0.009 ± 0.003		0.005 ± 0.002		NS		NS	
Mat Cat	NS		NS		0.57 ± 0.13		NS	
Risk Group	NS		0.61 ± 0.14		0.68 ± 0.14		0.53 ± 0.1	
Random Effects	**Level 1**		**Level 1**		**Level 1**		**Level 1**	
Constant	0.07 ± 0.008		0.1 ± 0.007		0.04 ± 0.004		0.04 ± 0.003	
	Level 2		**Level 2**		**Level 2**		**Level 2**	
	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age
Constant	0.66 ± 0.21	NS	0.44 ± 0.09	-0.02 ± 0.0007	0.12 ± 0.03	NS	0.23 ± 0.04	-0.005 ± 0.002
Biological Age	NS	0.002 ± 0.0007	-0.02 ± 0.0007	0.002 ± 0.0007	NS	0.001 ± 0.0003	-0.005 ± 0.002	0.001 ± 0.0002

Fixed effect values are Estimated Mean Coefficients ± SEE (Standard Error Estimate) of Trunk Fat Mass (log g)
Random effects values Estimated Mean Variance ± SEE [Trunk Fat Mass (log g)^2].
Biological Age (BA) is years from age at peak height velocity (PHV)(yrs); Height (cm); Physical Activity Score (1=low, 5=high); Total Fat Index (daily total fat intake (g)/1000 (Kcal)); Mat Cat (Maturity Category: 0=early, 1=average, 2=Late); Cardiometabolic Risk Group (0= low, 1=High)

Table 15: Multilevel regression models for physical activity development of males and females aligned by biological age

	Male				**Female**			
	(a) Sample 1		**(b) Sample 2**		**(c) Sample 1**		**(d) Sample 2**	
Fixed Effect	Estimates		Estimates		Estimates		Estimates	
Constant	2.8 ± 0.24		3.09 ± 0.06		1.14 ± 0.99		0.71 ± 0.7	
Biological Age	-0.2 ± 0.02		-0.14 ± 0.01		-0.12 ± 0.03		-0.19 ± 0.02	
Biological Age ^{2}	0.01 ± 0.001		0.007 ± 0.0006		0.006 ± 0.002		0.01 ± 0.001	
Height	NS		NS		0.01 ± 0.006		0.01 ± 0.005	
Total Body Fat	-0.00002 ± 0.000008		-0.000009 ± 0.000003		-0.00001 ± 0.000006		-0.000007 ± 0.000004	
Fat Intake	0.01 ± 0.005		NS		-0.007 ± 0.004		NS	
Mat Cat	NS		NS		NS		NS	
Risk Group	NS		NS		NS		NS	
Random Effects	**Level 1**		**Level 1**		**Level 1**		**Level 1**	
Constant	0.14 ± 0.02		0.19 ± 0.008		0.17 ± 0.02		0.2 ± 0.01	
	Level 2		**Level 2**		**Level 2**		**Level 2**	
	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age	Constant	Biological Age
Constant	0.4 ± 0.13	-0.02 ± 0.01	0.26 ± 0.04	-0.01 ± 0.003	0.13 ± 0.04	NS	0.26 ± 0.05	-0.01 ± 0.004
Biological Age	-0.02 ± 0.01	0.003 ± 0.001	-0.01 ± 0.003	0.003 ± 0.0004	NS	0.001 ± 0.0005	-0.01 ± 0.004	0.003 ± 0.0005

Fixed effect values are Estimated Mean Coefficients ± SEE (Standard Error Estimate) of Physical Activity Score (1=low, 5=high)
Random effects values Estimated Mean Variance ± SEE [Physical Activity Score ^{2}].
Biological Age (BA) is years from age at peak height velocity (PHV)(yrs); Height (cm); Total Body Fat Mass (log g); Total Fat Index (daily total fat intake (g)/1000 (Kcal)); Mat Cat (Maturity Category: 0=early, 1=average, 2=Late); Cardiometabolic Risk Group (0= low, 1=High)

4.4 Discussion

In this study I investigated the relationship between longitudinal development of FM and PA in adolescence with young adult cardiometabolic risk. The results indicate that young adult (at age 26) males and females classified as high cardiometabolic risk had a steeper FM trajectory (TBFM and trunk FM) from adolescence into young adulthood, when confounders were controlled. Greater consumption of dietary fat and lower levels of PA were also associated with less TBFM and Trunk FM at each biological age in males in sample 1 and sample 2. Among females there was no consistent association between TBFM development and dietary fat intake and PA. Timing of biological maturity (i.e. late maturity) was related to TBFM and Trunk FM development but only in the smaller sample of females with full measures of metabolic syndrome (i.e. sample 1). No association was found between young adult cardiometabolic risk and development of PA (model 3). Decreasing TBFM predicted increased PA in both samples and high dietary fat intake predicted less PA, in sample 1 only, for both males and females.

Previous studies have used cross sectional analysis to examine the association between adolescent FM (and other risk factors) and adult cardiometabolic risk (Hasselstrom *et al.*, 2002; Lefevre *et al.*, 2002; Twisk *et al.*, 2002; Yang *et al.*, 2008). However, the longitudinal design of my study allows for individual growth trajectories of FM and PA to be examined. Furthermore, previous longitudinal studies have been limited to indirect measures of adiposity (such as BMI and waist circumference). To my knowledge, this is the first study which has related serial DXA measures of body composition from adolescence into young adulthood to adult cardiometabolic risk.

4.4.1 Fat Mass Development

Trunk FM is of interest since it has been shown to be more closely related to CVD risk than TBFM in children (Gutin *et al.*, 2007; Teixeira *et al.*, 2001) and adults (Han *et al.*, 2006; Niederauer *et al.*, 2006), and is also a key feature of the metabolic syndrome. The atherogenic properties of trunk FM is likely due to an increased mobilization of non-esterified fatty acids (NEFA), over secretion of 'offensive' adipocytokines (e.g. PAI-1, tumor necrosis factor-α, visfatin) and under secretion of defensive adipocytokines (e.g. adiponectin) by intra-abdominal (i.e. visceral) fat (Despres & Lemieux, 2006).

My results show that individuals classified as high for cardiometabolic risk in young adulthood had greater TBFM and Trunk FM from adolescence through to young adulthood. This is consistent with results published by Ferreira *et al.* (2005) who found increased skinfold thickness and WC (13-36 years) was related to presence of metabolic syndrome presence at 36 years. The association between development of FM and young adult cardiometabolic risk is an important finding as it suggests that the antecedents of metabolic syndrome (i.e. increased fat mass) lies in late childhood/early adolescence and thus lends support for early intervention or identification of clinical tracking. Males from sample 1 showed no significant association between FM and cardiometabolic risk although there is an apparent trend. The lack of statistical significance is likely due to the small sample size of this group (N=21).

4.4.2 Lifestyle Variables

The results examining the relationship of self-report PA and fatness are less clear. Decreased PA was associated with increased TBFM and Trunk FM among males but not females. The lack of an inverse relationship between PA and TBFM in females may be a

function of natural female maturation (i.e. females laying down more fat during adolescence) and not because of PA levels per se (because there were no apparent difference in PA between males and females).

Cardiometabolic risk at 26 years was not associated with PA development from 8-26 years of age. Despite the substantial evidence supporting an inverse association between PA and metabolic syndrome in adults (Brien & Katzmarzyk, 2006; Carroll *et al.*, 2000; Lakka *et al.*, 2003; Zhu *et al.*, 2004), little is known about PA development during childhood/adolescence and adult cardiometabolic risk. This may be particularly important given the age related decline during this period. Four longitudinal studies from Europe - the Amsterdam Growth and Health Longitudinal Study (AGHLS) (Twisk *et al.*, 2002), the Leuven Longitudinal Study on Lifestyle, Fitness and Health (Lefevre *et al.*, 2002), the Danish Youth and Sports Study (Hasselstrom *et al.*, 2002), the Cardiovascular Risk in Young Finns Study (Yang *et al.*, 2008) - have investigated the direct relationship between PA in youth and adult CVD risk factors (e.g. lipoprotein levels, blood pressure, body fatness and body fat distribution). Only one of the four studies (Yang *et al.*, 2008) revealed a significant association between childhood and adolescent PA and adult CVD risk factors. These studies, however, used cross sectional analysis and thus were unable to examine growth trajectories or development of risk, as has been done here. Ferreira *et al*., (2005) investigated the longitudinal development (13-36 years) of PA of individuals with and without the metabolic syndrome at 36 years of age. They found that with increasing age, individuals with the metabolic syndrome, tended to spend more time in self-reported light-to-moderate intensity activities compared to those without the metabolic syndrome; however, the decline in vigorous PA was greater amongst those with the metabolic syndrome. The authors suggested that vigorous intensity PA may provide protection through a positive influence on body fatness and cardio-

respiratory fitness. Results from my study cannot confirm or refute these suggestions as cardiorespiratory fitness data was not available on the participants and PA was assessed via a composite score (1-5) of overall MVPA (i.e. no information is available on intensity).

4.4.3 Biological Maturity

An array of research has revealed an association between early biological maturity and increased adiposity (assessed via BMI and/or sum of skinfolds) in adolescent females (Himes *et al.*, 2004; Wang, 2002); however, research findings on the timing of biological maturity and central body fat in adolescence is inconclusive (Beunen *et al.*, 1994; Himes *et al.*, 2004; Malina *et al.*, 1999; Remsberg *et al.*, 2005). Previous research is limited largely to females, perhaps due to the lack of a convenient and obvious milestone of puberty in males, such as age at menarche in girls. The few studies that examined the timing of biological maturity and overweight/obesity in male adolescents have mixed results (Laron, 2004; Vignolo *et al.*, 1988; Vizmanos & Marti-Henneberg, 2000). Two other longitudinal studies have examined the long-term impact of early maturity on body composition (Beunen *et al.*, 1994; van Lenthe *et al.*, 1996a). Data from the AGHLS found early maturing boys and girls had greater BMI and sum of skinfold thickness between 13 and 27 years than late maturing individuals (van Lenthe *et al.*, 1996a). However, data from the Leuven Growth Study of Belgian Boys showed that an early age at PHV was related to a consistent higher BMI during adolescence (13-18 years) but not at age 30 (Beunen *et al.*, 1994). To my knowledge, the present study is the first to use longitudinal analysis to examine the trajectories of DXA- assessed fatness from childhood to adolescence and its relationship with the timing of biological maturity. My results, in general, showed that TBFM and trunk FM

development from adolescence into young adulthood did not differ between early, average or late maturing males or females.

Findings from studies investigating the relationship between the timing of biological maturity and PA are inconsistent: Some have found that early maturing girls (Baker *et al.*, 2007; Riddoch *et al.*, 2004; van Jaarsveld *et al.*, 2007) and boys (Riddoch *et al.*, 2004; van Jaarsveld *et al.*, 2007) participate in less PA than their later maturing counterparts; whereas other studies have found no association in girls (Niven *et al.*, 2007; Sherar *et al.*, 2009; Wickel & Eisenmann, 2007) and boys (Wickel & Eisenmann, 2007). To my knowledge, there has been only one longitudinal study examining the relationship between timing of maturity and adult PA. Among males (N=166) from the Leuven Longitudinal Study on Lifestyle, Fitness and Health greater sports participation at age 40 was associated with a later APHV (Beunen *et al.*, 2004). I found that timing of biological maturity was not associated with PA from 8-26 years.

Lastly, cross sectional analysis revealed no relationship between biological maturity status and adult MAP risk scores. Therefore, in this sample timing of biological maturity does not appear to be an independent risk factor for the development of cardiometabolic risk (including increased FM and decreased PA). However, it should be noted that the majority of the sample, especially females, were average maturers [i.e. 58.2% (*N*=32) of males and 78.9% (*N*=60) of females APHV fell between $\pm$ 1 year from normal APHV]. It may be that a larger sample of boys and girls of extreme maturity (i.e. early and late) is required for associations to be observed.

4.4.4 Limitations

The difficulties in conducting longitudinal studies that span childhood/adolescence into adulthood are well recognized (Kemper *et al.*, 1997; Roche *et al.*, 1997). One of the major concerns with the nature of this research design is small sample size, which was realized here as well. Although the within-subject repeated measures increases statistical power, in some cases the sample sizes may have been too small to detect real associations.

DXA is a valid and reliable method to assess body fat (Glickman *et al.*, 2004), however it's ability to accurately assess body composition in children and adolescents has been questioned (Roemmich *et al.*, 1997) For example, the DXA software does not account for maturational differences in the proportional contribution of water to fat free mass, which can result in an over estimation of body fat in adolescents (Roemmich *et al.*, 1997). Furthermore, since intra-abdominal or visceral fat has been shown to be one of the most detrimental fat deposits, from a metabolic perspective, the inability of DXA to assess visceral fat independently of subcutaneous fat could be a limitation. However, high correlations between DXA assessed trunk fat and MRI-derived total visceral abdominal tissue have been noted among men (Park *et al.*, 2002), which provides support for DXA derived trunk fat as an appropriate reflection of intra-abdominal fat.

Physical activity was assessed via self reported survey (PAQ-C, A and Ad). Although the aforementioned questionnaires have demonstrated reasonable internal consistency and validity with several other evaluations of activity level (Copeland *et al.*, 2005; Crocker *et al.*, 1997; Janz *et al.*, 2008) it is a self-report assessment, and therefore has the associated limitations (e.g. inaccurate recalling/reporting of PA) (Shephard, 2003). Dietary intake was also assessed via self report techniques (24-hour recall). Although the food recall procedure is thought to offer the best

method of obtaining a dietary record (Goran *et al.*, 1998), it may have questionable reliability because of under recording, under eating, or both (Bingham, 1991; Goris & Westerterp, 1999).

Lastly, because of the young age and good health of the sample it was necessary to create a continuous cardiometabolic risk score. A limitation with the continuous score is that it is sample specific. Furthermore, risk groups (high and low) were created using a median split which means that information on the distribution of the data is lost (Ragland, 1992) and statistical power is reduced (Selvin, 1987).

4.5 Conclusion

Excess body fatness, in particular central fatness, is a well recognized trigger of the metabolic syndrome. However, information on the development of body fatness during childhood and adolescence and its relationship with cardiometabolic risk is sparse. This study suggests that the increase in total and central fatness from adolescence to young adulthood may be critical for the development of cardiometabolic risk, and thus the metabolic syndrome. Therefore, intervention strategies to prevent the metabolic syndrome should strive to prevent/reduce excessive weight gain and central fat deposition during this period.

4.6 Acknowledgements

Sherar holds a Community and Population Health Research (CPHR) scholarship. The Canadian National Health and Research Development Program (NHRDP), the Canadian Institute of Health Research (CIHR), and the Saskatchewan Health Research Foundation (SHRF) provided funding for this study. PBMAS Group members include D. A. Bailey, A. D. G. Baxter-

Jones, P. E. Crocker, K. S. Davison, D. T. Drinkwater, E. Dudzic, R. A. Faulkner, K. Kowalski, H. A. McKay, R. L. Mirwald, W. M. Wallace, and S. J. Whiting.

Chapter 5: General Discussion

A factor which has the potential to influence PA and fat mass development, and thus ultimately the metabolic syndrome, is the timing of biological maturity. However, the influence of biological maturity has largely been overlooked in previous research. Thus, the general objective of this thesis was to investigate the associations between biological maturity, adolescent PA and fat mass development and young adult cardiometabolic risk.

The purpose of study 1 was to investigate whether observed gender differences in objectively measured PA in children (8 to 13 years) were confounded by physical maturity differences. I hypothesized that a) when aligned by CA boys will be more active that girls at each age b) when aligned by biological age (years from APHV) the gender difference in PA will disappear.

The purpose of study 2 was to describe the PA levels and perceived barriers to PA of adolescent girls (i.e., aged 8-16 years) by school grade and maturity status (i.e., early or late maturing) within grades. I hypothesized that: (a) PA would decrease with increasing grade and (b) within grade groups, early maturing girls would participate in less PA than late maturing girls. Given the expected decline in PA with CA and biological age, it was further hypothesized that: (a) the average number of barriers to PA would increase as grade in school increased and (b) within grade groups, early maturing girls would experience a greater number of barriers to PA, when compared to late maturing girls.

The purpose of study 3 was to examine total body fat mass (TBFM), trunk FM and PA developmental trajectories (aligned to biological maturity age) of individuals

categorized as low and high for cardiometabolic risk at 26 years, while investigating biological and lifestyle risk factors. I addressed the following hypotheses. First, early maturity in males and females would be associated with higher cardiometabolic risk at 26 years. Second, males and females with greater fat mass development (TBFM and trunk FM) during childhood, adolescence and young adulthood would be at higher cardiometabolic risk at 26 years. Furthermore, early biological maturity, greater dietary fat intake and less PA would be associated with greater FM development. Third, males and females who participate in less PA during childhood, adolescence and young adulthood would be at higher cardiometabolic risk at 26 years. Furthermore, early biological maturity and greater TBFM would be associated with less PA.

5.1. Summary of major findings

Adolescence is a dynamic period of growth and development. In addition to other physiological and behavioural changes, adolescence is characterized by sexual maturation (puberty), a decline in PA and an increase in fat mass deposition. The period of adolescence is important from a public health perspective because it is believed that the antecedents of many adult diseases, such as CVD or metabolic syndrome, lie in childhood and/or adolescence.

Adolescents of the same sex and CA can differ considerably in their degree of biological maturity (Malina *et al.*, 2004), resulting in early, average and late maturers. It is plausible that timing of biological maturity may play a role in the development of adult disease (namely CVD and/or metabolic syndrome). In particular, timing of biological maturity may be intricately involved in the development of fat mass (a biological risk

factor for the metabolic syndrome) and PA behaviour (a lifestyle risk factor for metabolic syndrome).

In study 1, I found that when youths' (8-13 yrs) PA were aligned by biological age (years from APHV) the sex difference in activity (i.e. girls less active than boys) disappeared. In other words, sex differences in biological maturity (i.e. girls on average maturing earlier) explain, at least in part, why girls participate in less PA than boys throughout adolescence. It also highlights biological maturity as an important influence in the adolescent decline in PA. Based on this rationale, in study 2 I examined if early and late maturing girls a) participated in less minutes of PA b) experienced different barriers to PA. After controlling for CA, no differences in PA or barriers to PA were found among girls of differing maturity status. However, a significant grade (CA) effect was seen, with PA declining with increasing grade and the type of barrier differing across grade. Lastly, in study 3, I examined the interrelationships between biological maturity status, development (8-26 years) of fat mass and PA (while controlling for dietary fat intake) of young adults at high and low cardiometabolic risk. It was found that greater adolescent and young adult fat mass (both total body fat mass (TBFM) and trunk fat mass (FM)) development was related to higher cardiometabolic risk at 26 years. There was little evidence to link biological maturity status and cardiometabolic risk (or fat mass and PA development).

5.2 The importance of controlling for biological age

During adolescence there is a dramatic increase in the velocity of growth in body composition (e.g. proportions of muscle, fat and bone). There is also variation in the rate and magnitude of linear growth of both within- and between- sexes of the same CA (Malina *et al.*, 2004); therefore, when studying the change in body composition (and other physiological parameters) during adolescence, there is a need to separate the independent effects of growth and maturation from those of other environmental factors, such as PA or diet. This separation is important because often the effects of growth and maturation mask or are greater than the effects associated with such environmental exposures. Thus, when assessing body composition during adolescence it is common practice to control for differences in biological maturity. One method which has been used frequently in longitudinal studies of growth is to align data to a biological maturity age (such as years from APHV), rather than CA. This method was adopted in study 3. Less commonly practiced, however, is the alignment of adolescent behaviour (such as participation in PA) by BA. For example, the majority of adolescent PA literature aligns PA on CA and/or grade, and notes the age related decline and the sex difference in PA (i.e. boys more active than girls at each age/grade) (Kimm *et al.*, 2000; Klasson-Heggebo & Anderssen, 2003; Riddoch *et al.*, 2004; Sallis, 1993; Trost *et al.*, 2002). Research and public health decisions and recommendations are based on these findings; for example, the funding of research and/or interventions that target adolescent girls (rather than boys) (Jamner *et al.*, 2004; Robbins *et al.*, 2006; Stevens *et al.*, 2005). However, results from Study 1 suggest that the previous observations that girls are less active than boys (and thus warrant intervention) may be overly simplistic, as when adolescents' data was

aligned by BA there were no significant sex difference in PA. Thus the lower levels of PA of girls at each age are likely due to their greater biological maturity (i.e. maturing approximately 2 years before boys (Malina *et al.*, 2004). These findings support the research by Thompson *et al.,* (2002), and highlight the importance of considering BA when studying adolescent PA.

5.3 Timing of biological maturity and fat mass development

5.3.1 Females

An array of research in females has shown an association between early biological maturity and increased adiposity assessed via BMI (Biro *et al.*, 2001; Wang, 2002), sum of skinfolds (Biro *et al.*, 2001; Wang, 2002) and DXA assessed fat mass (Himes *et al.*, 2004) in adolescence. The limited data in regards to trunk/abdominal fat mass and timing of biological maturity in females is inconclusive (Beunen *et al.*, 1994; Himes *et al.*, 2004; Malina *et al.*, 1999; Remsberg *et al.*, 2005; van Lenthe *et al.*, 1996b). The results from study 3 appear to be inconsistent with the previous studies in this area, revealing no significant difference in TBFM between girls of early, average and late maturity status during adolescence (and also into young adulthood). The surprising results of study 3 could be due to the methodology adopted and/or the sample studied, both of which will be discussed in more detail.

5.3.1.1 Controlling for biological maturity

Previous literature investigating timing of biological maturity and body composition/weight status has either chosen a sample of adolescents with a narrow CA

range or used a sample of adolescents with a wider age range and controlled for CA differences in the analysis. Using these two methods it is perhaps not surprising that early maturing girls of the same age have greater fat mass. As girls progress though adolescence (i.e. become more mature) they lay down more fat (Malina *et al.*, 2004); thus a girl who is further along the road to maturity but of the same CA as her peers, would be expected to have greater fat mass. Maybe of more interest (and of greater public health importance) is investigating whether early maturing girls have greater fat mass development than their later maturing counterparts at the same time point on the road to maturity (i.e. at the same biological age). This question is hard to address as longitudinal data is required to align individuals to a biological age. The longitudinal data used in study 3 enabled individuals' fat mass development to be aligned to a biological maturity age, years from APHV. The results showed, when differences in biological age were controlled, early, average and late maturing girls did not differ in TBFM or trunk FM. Therefore, the inconsistent findings of study 3, in comparison to other research, could be due to differences in methodology (i.e. aligning results by CA rather than biological age).

Data from the Amsterdam Growth and Health Study found early maturing girls, in comparison to late maturing girls, had greater BMI and sum of skinfold thickness between 13 and 27 years (van Lenthe *et al.*, 1996a); which suggests that the association between timing of biological maturity and body composition is persisting even after maturity is reached. Furthermore, a longitudinal study of 200 Finnish girls found that high circulating concentrations of estradiol, a hormone that promotes the accumulation of body fat, persisted until the age of 25 y in early maturing individuals (Vihko & Apter,

1984). In the present study no differences in fat mass were found between maturity groups at age 26 (i.e. when full maturity is reached).

5.3.1.2 Homogeneity of the sample

The participants of study 3 (i.e. the PBMAS participants) were homogenous in terms of BMI classification and timing of maturity, which could also help explain the lack of association between timing of maturity and fat mass development.

Much of previous research exploring the relationship between timing of biological maturity and body composition has focused on the specific relationship between early maturity and prevalence of overweight and/or obesity (Adair & Gordon-Larsen, 2001; Bratberg *et al.*, 2007; Wang, 2002). A theory linking early maturity to greater fat mass is that obese females have relatively lower levels of sex hormone binding globulin which means that larger proportions of sex hormones (e.g. estradiol) are available for bioactivity, thus advancing maturity (MacDonald *et al.*, 1978). However, the mechanisms driving puberty still remain poorly understood (Bray *et al.*, 1998). The participants in sample 3 were, in general, of a normal weight status during adolescence. In fact at PHV, using Cole's age specific BMI cut-offs (Cole *et al.*, 2000), 93.6% of the boys were classified as normal weight and the remaining (6.4%) as overweight and 100% of girls were normal weight. It may be that early maturity only increases fat mass development in girls who are overweight or obese upon entry into puberty. In support, Bratberg and colleagues (2007) found that among 864 girls followed from 14 to 18 years of age, early age at menarche in girls was associated with overweight in late adolescence but not among girls that were relatively lean upon study entry, i.e. in early adolescence.

The majority of the participants in study 3 were of average maturity status. In fact, 58.2% (*N*=32) of males and 78.9% (*N*=60) of females APHV fell between $\pm$ 1 year from normal APHV, and thus were classified as average maturing. It may be that a larger sample of girls of extreme maturity (i.e. early and late) is required for associations to be observed.

5.3.2 Males

Previous research has concentrated on females, perhaps due to the lack of a convenient and obvious milestone of puberty, such as age at menarche in girls. Results from studies which examined timing of biological maturity and overweight/obesity in adolescence in males have been mixed (Laron, 2004; Vignolo *et al.*, 1988; Vizmanos & Marti-Henneberg, 2000; Wang, 2002). In study 3 I found no difference in TBFM and trunk FM among early, average and late maturing boys. Although more research is needed, one might expect a gender difference when studying the relationship between timing of biological maturity and fat mass development (especially if early maturity precedes adiposity rather than the alternative). In pre-adolescence, the proportions of adipose tissue and lean body mass in boys and girls are similar. However, during adolescence, on average, girls have relative greater increase in fat than fat-free mass, whereas boys gain more fat-free mass than fat mass; thus, an early maturing boy is likely to have relatively more lean mass than his later maturing counterparts, whereas an early maturing female is likely to have relatively more fat mass (Malina *et al.*, 2004). One may expect early maturity to either have no influence or a positive influence (i.e. late maturers having greater relative fat mass) on fat mass development. However, more research is

needed which examines the longitudinal relationship between timing of biological maturity and fat mass development among normal weight and overweight males before any definitive conclusions can be made.

In conclusion, study 3 is, to my knowledge, the first to use longitudinal analysis to examine the trajectories of DXA- assessed fatness from childhood to adolescence and its relationship with timing of biological maturity. More longitudinal research with serial measures of body fat (rather than BMI) from pre-adolescence, through adolescence and into young adulthood is needed (preferably accompanied by measurements of sex-hormones) to fully elucidate the impact of early biological maturity on long term body composition.

5.4. Timing of biological maturity and physical activity

Until recently, the relationship between timing of biological maturity and participation in PA had been largely overlooked. However, in 2007 five papers were published which studied the relationship between timing of biological maturity and self report (Niven *et al.*, 2007; van Jaarsveld *et al.*, 2007) and objectively assessed PA (Baker *et al.*, 2007; Riddoch *et al.*, 2004; Wickel & Eisenmann, 2007), with mixed results. I found no significant difference in levels of objectively assessed PA among 8-16 year old girls (study 2). Likewise, I found no significant difference in the longitudinal development (8-26 years) of self-report PA among early, average and late maturing males and females (study 3). The inconsistency between previous research and my findings could be due to methodological issues, which will be discussed in more detail.

5.4.1 Choice of maturity indicator

When relating timing of biological maturity to PA a range of maturity indicators are used. Table 16 outlines the methods used in studies 2 and 3, and also methods used in previous research. Prior research in this area has used the pubertal development scale (PDS), recalled age at menarche, hormonal (i.e. estradiol) assays, APHV (both predicted and assessed), and secondary sex characteristics to assess the biological maturity status of boys and girls; however, the maturity indicators are not equivalent. Sexual, somatic and skeletal maturity occur at the different times during adolescence (e.g. in females the appearance of pubic hair development is an early event whereas age at menarche is a late event (Marshall & Tanner, 1969; Sherar *et al.*, 2004). It may be that the development of certain maturity indicators is more closely related to PA than others. For example, appearance of secondary sex characteristics could be more closely related to the girls' disengagement from PA than timing of peak velocity in height and onset of menstruation. The appearance of secondary sex characteristics, in particular breast development, may cause a girl to feel self conscious, or even uncomfortable about participating in PA.

It also is plausible that timing of PHV may be more closely related to males PA levels than other maturity indicators; with the possibility that an earlier onset of PHV might exert a positive influence on PA behaviour. The timing of peak velocity in lean mass is tightly associated with the timing of APHV, with its peak occurring approximately one year post APHV (Malina *et al.*, 2004). As previously mentioned, males lay down a considerable amount of lean mass throughout the adolescent period; thus approximately one year after APHV early maturing boys will, on average, have greater lean mass (i.e. muscle mass) than their later maturing counterparts. The physical

benefits might translate into improvements in sport performance and thus, possibly greater participation in overall PA. Research within the sporting literature suggests that successful/elite adolescent male athletes are of early maturity status (Malina *et al.*, 2005; Sherar *et al.*, 2007a). How this perceived or actual maturity associated physical advantage relates to daily minutes spent in PA has yet to be elucidated.

It should be noted that all indicators of biological maturity have their associated limitations (Baxter-Jones *et al.*, 2005). For example, on one end of the spectrum is skeletal age assessment, which is still regarded as the gold standard for assessing biological maturity (Malina *et al.*, 2004). On the other end of the spectrum are measures such as parental report of child's pubertal development using the PDS, which likely has poor validity. In conclusion, the inconsistent findings from studies investigating timing of biological maturity and PA behaviour could be due to the methodology used to assess biological maturity.

Table 16: Outline of the methods used in all research examining the relationship between biological maturity and PA

Study	N	Gender	Age	Study Design	Maturity Indicators	Finding
(Niven *et al.*, 2007)	208	Female	11-12 years	Cross-sectional	PDS	No association
(van Jaarsveld *et al.*, 2007)	5863	Male and Female	11-12 yrs	Cross-sectional	PDS	EM associated with ↑ sedentary behaviour among boys and younger girls (grade 7 only) EM associated with ↑ VPA among boys and younger girls (grades 7 and 8 only)
(Baker *et al.*, 2007)	143	Female	11 and 13 yrs*	Longitudinal (2 time points)	Blood estradiol, secondary sex characteristics, parental report using PDS	EM associated with ↑ MVPA and VPA
(Riddoch *et al.*, 2004)	5595	Male and female	11 yrs	Cross-sectional	Secondary sex characteritics	Stage of pubertal development was inversely related to PA in girls and to a lesser extent in boys
(Wickel & Eisenmann, 2007)	167	Male and female	13-14 yrs	Cross-sectional	Predicted APHV	No association
Study 2	221	Female	8-16 yrs	Cross-sectional	Predicted APHV and recalled age at menarche	No assciation
Study 3	131	Male and Female	8-26 yrs	Longitudinal	Yrs from APHV	No association

EM= early maturity; VPA = vigorous physical activity (PA); MVPA = moderate to vigorous PA; PDS= self report pubertal development scale (Petersen *et al.*, 1988). 5 –item questionnaire which asks girls to rate degree of development with regards to growth spurt in height and development secondary sex characteristics; APHV = age at peak height velocity ;* biological maturity assessed at 11 years and physical activity assessed at 13 years.

5.4.2 Self-report versus objectively assessed biological maturity

Research (including study 2 and 3) investigating the relationship between timing of biological maturity and PA have used objective indicators to measure biological maturity. However, researchers investigating the relationship between other behaviours (e.g. substance abuse, delinquent behaviour etc.) and timing of biological maturity have advocated the use of a subjective measure of pubertal timing (i.e. measures of perception of pubertal timing rather than actual pubertal timing) (Dubas *et al.*, 1991a; Michaud *et al.*, 2006). In subjective assessments, individuals are asked to rate their level of pubertal development in relation to their peers on a five-point scale ranged from "matured much earlier" to "matured the same time" to "matured much later". Using these indicators the relative timing of biological maturity among adolescents within a cohort can be estimated. Dubas and colleagues (1991a) argue that although actual and perceived timing are overlapping, they are distinct measures that reflect different biological and psychosocial processes; thus two individuals with the same assessed biological age might have perceptions of their pubertal timing relative to their peer group which differ markedly. It may be that the perception of pubertal timing rather than actual pubertal timing is associated with disengagement from PA. Galambos *et al*., (1999) in a study of 226 9-17 year old males and females, found that biological age was related to self reported problem behaviour (e.g. substance abuse, disobeying parents and antisocial behaviour) in girls and boys; however, when CA was controlled, associations disappeared. On the other hand, when CA and biological maturity status were controlled, subjective maturity status was still associated with self report behaviours. These findings suggest that self-perceptions of maturity, such as subjective biological age, are more

proximal to adolescents' behaviour than are actual biological or chronological age. Future research which examines the discord between a subjective self-report measure and an objective measure of maturity and their association with PA is needed.

5.4.3 Interacting factors

The inconsistent findings surrounding the relationship between biological maturity and PA suggest there are other factors that interact with pubertal timing to increase risk for inactivity in some individuals. It is plausible that not all early maturing youth become inactive. An example can be drawn from the depression literature. It has been shown that rather than early maturity creating depression, early maturity accentuates pre-existing behavioural problems (Caspi & Moffitt, 1991). This finding is consistent with the diathesis-stress perspective that pre-existing individual differences interact with the pubertal transition, such that certain adolescents develop depression while others do not (Graber *et al.*, 2006). For example, low self-esteem in girls who mature early and in boys who mature late predicts depression in late adolescence (Ge *et al.*, 2001; Harter & Whitesell, 1996; Petersen, 1988). This finding suggests that there may be individual characteristics that differentiate early maturing adolescents who become inactive from those who do not. For example, it may be that individuals who participate in high levels of activity pre-adolescence are protective from the possible harming effect of early maturity. Likewise, it may be the early maturing girls and late maturing boys with low self-esteem who are more likely to disengage from PA during adolescence. It is probable that disengagement from PA during adolescence results from a multitude of behavioural,

social and biological factors interacting with timing of maturity; however, this conjecture requires more research to confirm.

5.4.4 Cultural context

Individuals are embedded in social contexts and there is a dynamic interaction between the social contexts and biological factors (such as timing of biological maturity). Existing research (including Study 2 and 3) has not simultaneously considered both contextual variables and timing of biological maturity when studying adolescent PA. Cigarette and alcohol involvement is related to the timing of biological maturity among 11-14 year old girls and boys and social context mediated most associations *(*Foshee *et al*., 2007). For example, the association between pubertal status and cigarette involvement was shown to be mediated by family context (e.g. early maturing individuals with less authoritative parents smoked more cigarettes). It may be that parental support of PA before and during adolescence may provide a protection from the harming effect of early biological maturity. Likewise biological maturity may exert different influences depending on the social context the adolescent lives in (e.g. the SES of the child's family). Thus, to fully understand how timing of biological maturity influences PA, there is a need to consider how psychological and social factors interact with biological maturity influences.

5.4.5 Chronological age/Grade influences

Finally, CA likely has a strong independent influence on the relationship between BA and PA. Chronological age represents the age/grade influences and transitions that

will likely impact the adolescent; thus it is possible that in terms of PA behaviour, CA/grade may exert a stronger influence than biological maturity. Although results from Study 1, suggest that biological maturity may be more tightly associated with the adolescent decline in PA, the results are limited as it was an observational study. In Study 2, I attempted to provide some explanation to how biological maturity influenced PA. These results showed that PA levels were largely related to grade (i.e. as grade increased, PA decreased) and not biological maturity status. Furthermore, the types of barriers to PA were more closely related to grade level (i.e. girls in grades 4-6 cited more interpersonal barriers and girls in grades 9-10 more institutional barriers). Thus, there is a possibility that among a normal sample of adolescents, CA/grade groups exerts a greater influence on PA behaviour than biological maturity status. However, more research is required to support or refute this suggestion.

5.5 Childhood/adolescent antecedents of adult metabolic syndrome

In Study 3, I investigated childhood/adolescent antecedents of adult metabolic syndrome. Specifically, the study focused on the impact of PA, fat mass and timing of biological maturity on adult risk for metabolic syndrome (as assessed via a composite score of a) general metabolic risk and b) risk for hypertension (mean arterial pressure). Each individual association will now be discussed.

5.5.1 Fat mass development and cardiometabolic risk

Abdominal or trunk FM is of interest when considering the relationship between fat mass development and cardiometabolic risk. It has been shown that central

accumulation of FM in children (Gutin *et al.*, 2007; Teixeira *et al.*, 2001) and adults (Han *et al.*, 2006; Niederauer *et al.*, 2006) is more closely related to CVD risk than TBFM. Study 3 revealed that individuals classified as high for cardiometabolic risk in young adulthood had greater TBFM and trunk FM from adolescence through to young adulthood. This is consistent with a previous longitudinal study which found that increased skinfold thickness and WC (13-36 years) was related to presence of metabolic syndrome presence at 36 years (Ferreira *et al.*, 2005).

I expected a stronger association between trunk FM and cardiometabolic risk than between TBFM and cardiometabolic risk. The associations (i.e. coefficients), however, were similar in both TBFM and trunk FM models for males and females. In addition, correlations (data not shown) between TBFM and trunk FM were high from 8-28 years (ranging from r=0.98-1.0 in males and r=0.97-0.99 in females). This result is consistent with a previous study which found that accumulation of DXA- assessed trunk FM was strongly related to TBFM in men and women, aged 18-96 years (Wu *et al.*, 2007).

Ultimately, finding an association between development of FM and young adult cardiometabolic risk is important as it suggests that the antecedents of metabolic syndrome (i.e. increase fat mass) lies in childhood and/or early adolescence. More research is required to inform intervention strategies to prevent/reduce excessive weight gain and central fat deposition during this period.

5.5.2 PA and fat mass development

In Study 3, PA was associated with fat mass development (TBFM or trunk FM) from adolescence to adulthood in males but not females. In confirmation, a previous longitudinal analysis of a larger sample (105 males and 103 females) of the PBMAS cohort (through the adolescent years only), revealed a significant negative relationship between PA and TBFM from 8-19 years in boys, but no association was found for girls (Mundt *et al.*, 2006). In both of these papers (i.e. study 3 and the paper by Mundt *et al.*, 2006) no difference in PA existed between males and females; therefore, the lack of an inverse relationship between PA and TBFM (and trunk FM) in females may be a function of natural female maturation (i.e. females laying down more fat during adolescence). However, this suggestion is not supported in the literature. Past longitudinal research has, in general, found that PA is inversely related to fat mass development in females (Kemper *et al.*, 1999; Kettaneh *et al.*, 2005; Must & Tybor, 2005; Must *et al.*, 2007; Stevens *et al.*, 2007); whereas research among males is less extensive and has produced inconsistent findings (Kemper *et al.*, 1999; Kettaneh *et al.*, 2005). The lack of association between PA and fat mass development (found in study 3 and in the research conducted by Mundt and colleagues (2006)) could be due to methodological limitations in the assessment of PA (discussed in more detail later in the chapter). Furthermore, it is possible that associations between fat mass and PA may have been evident if parental fatness/overweight had been considered. Must and colleagues (2007) found that among 8-12 year old girls followed until 4 years post menarche, PA was inversely related to body fat, particularly in girls whose parents were overweight. Furthermore, a twin study found that much of the variance in fat mass development was due to hereditary rather than

lifestyle (i.e. diet and PA) factors (Malis *et al.*, 2005). Although not conclusive, this research suggests that hereditary/parental influences explain some variance in body fat development. Lastly, past research largely did not consider differences in biological maturity of adolescents, which likely confounded results.

5.5.3 PA and Cardiometabolic Risk

I did not find a significant relationship between PA development from 8-26 years and young adult cardiometabolic risk (Study 3). Despite the substantial evidence supporting an inverse association between PA and metabolic syndrome in adults (Brien & Katzmarzyk, 2006; Carroll *et al.*, 2000; Lakka *et al.*, 2003; Zhu *et al.*, 2004), little is known about the relationship between PA during childhood/adolescence and adult cardiometabolic risk/metabolic syndrome. Three studies from Europe (the Amsterdam Longitudinal Study (Twisk *et al.*, 2002), the Leuven Longitudinal Study on Lifestyle, fitness and Health (Lefevre *et al.*, 2002), the Danish Youth and Sports Study (Hasselstrom *et al.*, 2002)), have investigated the direct relationship between PA in youth and adult CVD risk factors (e.g. lipoprotein levels, blood pressure, body fatness and body fat distribution). These studies failed to find a significant relationship between childhood and adolescent PA and adult risk for CVD. However, these studies were confined to a low number of observations during adolescence and young adulthood and cross sectional analysis. Assessing the independent impact of lifestyle behaviors on fat development in adolescence and relating it to adult cardiometabolic risk/metabolic syndrome is problematic because normal growth-related body composition changes may mask or be greater than the lifestyle effects. Therefore, only longitudinal studies, which identify each

individual's pattern of fat mass development (i.e. growth trajectories) from adolescence into young adulthood, can examine the independent contribution of closely interrelated lifestyle and biological risk variables.

One previous study has investigated the longitudinal development (13-36 years) of PA (and fat mass, nutrient intake and cardiorespiratory fitness) of individuals with and without the metabolic syndrome at 36 years of age (Ferreira *et al.*, 2005). Using generalized estimating equations (GEE) to examine growth trajectories of PA, they found individuals with versus those without the metabolic syndrome tended to spend more time in self-reported light-to-moderate intensity activities between 13 and 36 years. However, the decline in vigorous PA was greater amongst those with the metabolic syndrome (Ferreira *et al.*, 2005). The authors also found that individuals with the metabolic syndrome had greater fatness (at 21, 27, 32 and 36 years) and decreased cardiorespiratory fitness (at 27, 32 and 36) than those without the metabolic syndrome. Therefore, the authors suggested that vigorous intensity PA may provide protection through a positive influence on body fatness and cardio-respiratory fitness. Study 3 cannot comment on this as it is limited to the information obtained from the PA questionnaires (PAQ-C, A, AD) that were adopted at the onset of the study (i.e. 1991). The PAQs are comprised of questions structured to discern moderate through vigorous PA during the last 7 days. Therefore, it is not possible to detract information spent on specific intensities of PA (i.e. low to moderate and vigorous).

5.6 Limitations

5.6.1 Study 1

A strength of Study 1 and 2 is that I utilized an objective measurement of PA (i.e. accelerometry). Accelerometry is limited, however, because it does not provide information on the type or context of the activity. Other limitations include reactivity, mechanical failure and non-compliance; although, a delayed initialization and a technical reliability assessment of devices was conducted to mitigate these limitations (see appendix G for more detail). Lastly, previous studies have used different thresholds to convert raw accelerometer counts to minutes spent in certain intensities of activity. The lack of consistency in the literature hinders comparison of results between studies (Sherar *et al.*, 2008a; see appendix J).

5.6.2 Study 2

In Study 2 I also used an accelerometer to assess PA so the same accelerometer association limitations listed for study 1 applies for study 2. Furthermore, study 1 utilized a cross sectional study design to examine barriers to PA of girls by grade and biological maturity group. Therefore, information on the stability and variability (over time) of barriers categories and specific barrier types was not obtained.

Secondly, information was not collected on the extent to which a specific barrier limits PA. For example, one method of assessing how a specific barrier limits PA is to assess a youth belief in their capabilities to effectively manage the barriers they are encountering. This type of belief is called coping self-efficacy and has been defined as confidence in one's skills and abilities to overcome barriers to PA (Gyurcsik *et al.*, 2002).

If an individual perceives that barriers exist and must be overcome, coping self-efficacy likely will become an important determinant of the extent to which the barriers limits PA.

5.6.3 Study 3

The difficulties in conducting longitudinal studies spanning childhood/adolescence into adulthood are well recognized (Kemper *et al.*, 1997; Roche *et al.*, 1997). Study 3 included relatively small sample sizes. Although the within-subject repeated measures helps to increase statistical power, in some cases the sample sizes may have been too small to detect real associations. Longitudinal studies must use the same method of measurement at each time point; therefore, study 3 was limited to the data collections tools adopted at the beginning of the study. Physical activity was assessed via self reported survey (PAQ-C, A and Ad). Although the aforementioned questionnaires have demonstrated reasonable internal consistency and validity with several other evaluations of activity level (Copeland *et al.*, 2005; Crocker *et al.*, 1997; Janz *et al.*, 2008) it is a self-report assessment and therefore has the associated limitations (e.g. inaccurate recalling/reporting of PA) (Shephard, 2003).

Dietary intake also was assessed via self report techniques (24-hour recall). Although the food recall procedure is thought to offer the best method of obtaining a dietary record (Goran *et al.*, 1998), it is also a self-reported measure and has associated limitations. Measurements of food intake with use of dietary records, food-frequency questionnaires, dietary histories, or 24-h recalls are mostly unreliable because of under-recording, under-eating, or both (Bingham, 1991; Goris & Westerterp, 1999). Specifically, underreporting of habitual food intake was observed in both obese and lean

youth (Bandini *et al.*, 1990) and adults (Goris *et al.*, 2000; Goris & Westerterp, 1999). Furthermore, a recent longitudinal study of girls compared energy intake assessed via a self report dietary record with total energy expenditure assessed via doubly labeled water technique (gold standard) at 10, 12 and 15 yrs, and found that as the girls got older they tended to report their energy intake less accurately (Bandini *et al.*, 2003).

Lastly, because of the young age and good health of the PBMAS sample in study 3, it was necessary to create a continuous score for cardiometabolic risk. A limitation with the continuous score is that it is sample specific. Furthermore, risk groups (high and low) were created using a median split which means that information on the distribution of the data is lost (Ragland, 1992) and statistical power is reduced (Selvin, 1987).

Study 3 used body composition data derived from a DXA. Although a DXA is a valid and reliable method to assess body fat (Glickman *et al.*, 2004), its ability to accurately assess body composition in children and adolescents has been questioned (Roemmich *et al.*, 1997). Furthermore, DXA is unable to assess visceral fat depots independently of subcutaneous abdominal adipose tissue, which is a limitation. The trunk region as defined by DXA includes fat deposits other than those in the intra-abdominal or visceral cavity, including subcutaneous and intermuscular fat and epicardial and pelvic deposits (Lohman, 2005). Since intra-abdominal or visceral fat has been shown to be one of the most detrimental fat deposits from a metabolic perspective, the inability of DXA to assess visceral fat independently of subcutaneous fat could be a limitation. However, high correlations between DXA assessed trunk fat and MRI-derived total visceral abdominal tissue have been noted among men (Park *et al.*, 2002), which provides support for DXA derived trunk fat as an appropriate reflection of intra-abdominal fat.

Chapter 6: Conclusions and future research

6.1. Conclusions and public health implications

The metabolic syndrome is a major public health challenge world-wide. Adolescence is a volatile period where physiology is changing dramatically; sexual maturation is occurring, social and peer pressures dominate and healthy lifestyle often deteriorates. Therefore, adolescence has been highlighted as a critical period for the development of adult disease, such as the metabolic syndrome. Results from this thesis support this contention by showing a steep decrease in PA (by both chronological and biological age) in males and females across adolescence. It further showed that an increase in total and central fatness during adolescence may be critical for the development of the metabolic syndrome in adulthood. Timing of biological maturity was not shown to have an independent impact on adolescent or young adult PA or fat mass development, or young adult cardiometabolic risk. However, further research is required before definitive conclusions can be made about the short and long term impacts of timing of biological maturity on health. From a public health perspective, intervention strategies to prevent the metabolic syndrome should strive to prevent/reduce excessive weight gain and central fat deposition during childhood and adolescence.

6.2 Future research

1. There is a need to examine the association between the development of different intensities of PA and adult cardiometabolic risk. The advent of objective tools to assess PA (e.g. accelerometers) provides useful and reliable information on differing intensities of PA. Although, accelerometers are fairly new technology there are

longitudinal studies (such as the European Youth Heart Study (Poortvliet *et al.*, 2003)) which have collected serial accelerometer data on children and adolescents while assessing biological and lifestyle risk factors for CVD/metabolic syndrome. At present the oldest individuals in these studies are in late adolescence; therefore, in 5-10 years the relationship between the development of objectively assessed PA and young adult cardiometabolic risk will able to be addressed (dependent on continuation of these studies into young adulthood). Furthermore, because of the serial measures of individual components of CVD/metabolic syndrome, for the first time one will be able to investigate the effects of PA on development of cardiometabolic risk across time (rather that just investigating young adult cardiometabolic risk).

2. Future research should consider the interaction of timing of biological maturity and psychological and social context as they relate to adolescent PA. Conducting this research may help to better elucidate the relationship between biological maturity and PA, but also if associations are found, will help in the design of programs to alter the social context thereby decreasing the possible negative impact of timing of biological maturity. Furthermore, future research would benefit from examining the discord between a subjective, self-report measure and an objective measure of maturity and their association with PA and barriers to PA.
3. A fruitful study would be one which selectively samples boys and girls of extreme maturity (for example, girls with an APHV $\leq$ 11 years and boys with an APHV $\leq$ 13 years from APHV). This would provide an adequate sample size of girls and boys who are of more extreme maturity. Examining the PA (and barriers to PA) of this

group would help to shed more light on the impact of timing of biological maturity on PA and barriers to PA. Likewise, tracking of these individuals from adolescence into young adulthood would enable comment on the PA trajectories of early and late maturity how it relates to adolescent and young adult health.

Reference List

Aaron, D. J., Kriska, A. M., Dearwater, S. R., Anderson, R. L., Olsen, T. L., Cauley, J. A., & Laporte, R. E. (1993). The epidemiology of leisure physical activity in an adolescent population. Med.Sci.Sports Exerc. 25, 847-853.

Adair, L. S. & Gordon-Larsen, P. (2001). Maturational timing and overweight prevalence in US adolescent girls. Am.J.Public Health 91, 642-644.

Alasker, F. D. (1995). Timing of puberty and reactions to pubertal changes. In Psychosocial disurbances in young people, ed. Rutter, M., pp. 37-82. Cambridge University Press, Cambridge,UK.

Alberti, K. G., Zimmet, P., & Shaw, J. (2005). The metabolic syndrome--a new worldwide definition. Lancet. 366, 1059-1062.

Alberti, K. G. & Zimmet, P. Z. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabet.Med. 15, 539-553.

Allison, K. R., Adlaf, E. M., Dwyer, J. J., Lysy, D. C., & Irving, H. M. (2007). The decline in physical activity among adolescent students: a cross-national comparison. Can.J.Public Health. 98, 97-100.

Allison, K. R., Dwyer, J. J., & Makin, S. (1999). Perceived barriers to physical activity among high school students. Prev.Med. 28, 608-615.

Anand, S. S., Yi, Q., Gerstein, H., Lonn, E., Jacobs, R., Vuksan, V., Teo, K., Davis, B., Montague, P., & Yusuf, S. (2003). Relationship of metabolic syndrome and fibrinolytic dysfunction to cardiovascular disease. Circulation. 108, 420-425.

Andersen, L. B., Boreham, C. A., Young, I. S., Davey, S. G., Gallagher, A. M., Murray, L., & McCarron, P. (2006a). Insulin sensitivity and clustering of coronary heart disease risk factors in young adults. The Northern Ireland Young Hearts Study. Prev.Med. 42, 73-77.

Andersen, L. B. & Haraldsdottir, J. (1993). Tracking of cardiovascular disease risk factors including maximal oxygen uptake and physical activity from late teenage to adulthood. An 8-year follow-up study. J.Intern.Med. 234, 309-315.

Andersen, L. B., Harro, M., Sardinha, L. B., Froberg, K., Ekelund, U., Brage, S., & Anderssen, S. A. (2006b). Physical activity and clustered cardiovascular risk in children: a cross-sectional study (The European Youth Heart Study). Lancet. 368, 299-304.

Andersen, L. B., Sardinha, L. B., Froberg, K., Riddoch, C. J., Page, A. S., & Anderssen, S. A. (2008). Fitness, fatness and clustering of cardiovascular risk factors in children from Denmark, Estonia and Portugal: The European Youth Heart Study. Int.J Pediatr Obes. 3 Suppl 1:58-66.

Anderson, S. E., Dallal, G. E., & Must, A. (2003). Relative weight and race influence average age at menarche: results from two nationally representative surveys of US girls studied 25 years apart. BMC Cardiovasc Disord. 111, 844-850.

Ardern, C. I., Katzmarzyk, P. T., Janssen, I., & Ross, R. (2003). Discrimination of health risk by combined body mass index and waist circumference. Obes.Res. 11, 135-142.

Armstrong, N., Balding, J., Gentle, P., & Kirby, B. (1990). Patterns of physical activity among 11 to 16 year old British children. British Medical Journal 301, 203-205.

Armstrong, N. & VanMechelen, W. (1998). Are young people fit and active? In Young and active and health enhancing physical activity: Evidence and implications, eds. Biddle, S. J. H., Cavill, N., & Sallis, J. F., pp. 69-97. Health Education Authority, London.

Armstrong, N. & Welsman, J. (1997). Young People and Physical Activity, pp. 1-369. Oxford University Press, Oxford.

Bacha, F., Saad, R., Gungor, N., Janosky, J., & Arslanian, S. A. (2003). Obesity, regional fat distribution, and syndrome X in obese black versus white adolescents: race differential in diabetogenic and atherogenic risk factors. J Clin.Endocrinol.Metab. 88, 2534-2540.

Bailey, D. A. (1997). The Saskatchewan Pediatric Bone Mineral Accrual Study: bone mineral acquisition during the growing years. Int.J.Sports Med. 18 Suppl 3:S191-4.

Bailey, D. A., Martin, A. D., McKay, H. A., Whiting, S., & Mirwald, R. (2000). Calcium accretion in girls and boys during puberty: a longitudinal analysis. Journal of Bone and Mineral Research 15, 2245-2250.

Bailey, R. C., Olson, J., Pepper, S. L., Porszasz, J., Barstow, T. J., & Cooper, D. M. (1995). The level and tempo of children's physical activities: an observational study. Med Sci Sports Exerc. 27, 1033-1041.

Baker, B. L., Birch, L. L., Trost, S. G., & Davison, K. K. (2007). Advanced pubertal status at age 11 and lower physical activity in adolescent girls. J.Pediatr. 151, 488-493.

Balkau, B. & Charles, M. A. (1999). Comment on the provisional report from the WHO consultation. European Group for the Study of Insulin Resistance (EGIR). Diabet.Med. 16, 442-443.

Bandini, L. G., Must, A., Cyr, H., Anderson, S. E., Spadano, J. L., & Dietz, W. H. (2003). Longitudinal changes in the accuracy of reported energy intake in girls 10-15 y of age. Am J Clin.Nutr. 78, 480-484.

Bandini, L. G., Schoeller, D. A., Cyr, H. N., & Dietz, W. H. (1990). Validity of reported energy intake in obese and nonobese adolescents. Am J Clin.Nutr. 52, 421-425.

Bandura, A. (1997). Self-efficacy: The Exercise of Control WH Freeman, NY: New York.

Barker, D. J. (2000). In utero programming of cardiovascular disease. Theriogenology. 53, 555-574.

Barker, D. J. (2004). The developmental origins of chronic adult disease. Acta Paediatr.Suppl. 93, 26-33.

Barker, D. J., Winter, P. D., Osmond, C., Margetts, B., & Simmonds, S. J. (1989). Weight in infancy and death from ischaemic heart disease. Lancet. 2, 577-580.

Bassett, D. R., Jr., Tremblay, M. S., Esliger, D. W., Copeland, J. L., Barnes, J. D., & Huntington, G. E. (2007). Physical activity and body mass index of children in an old order Amish community. Med Sci Sports Exerc. 39, 410-415.

Baxter-Jones, A. D., Mirwald, R. L., McKay, H. A., & Bailey, D. A. (2003). A longitudinal analysis of sex differences in bone mineral accrual in healthy 8-19-year-old boys and girls. Ann.Hum.Biol. 30, 160-175.

Baxter-Jones, A. D. G., Eisenmann, J. C., & Sherar, L. B. (2005). Controlling for maturation in pediatric exercise science. Pediatric Exercise Science 17, 18-30.

Baxter-Jones, A. D. G. & Sherar, L. B. (2007). Growth and Maturation. In Paediatric Exercise Physiology, ed. Armstrong, N., pp. 1-26. Elsevier, Churchill, Livingstone.

Bayley, N. (1962). The accurate prediction of growth and adult height. Modern Problems in Paediatrics 7, 234-255.

Berenson, G. S., Srinivasan, S. R., Bao, W., Newman, W. P., III, Tracy, R. E., & Wattigney, W. A. (1998). Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. N.Engl.J.Med. 338, 1650-1656.

Bergman, R. N., Kim, S. P., Catalano, K. J., Hsu, I. R., Chiu, J. D., Kabir, M., Hucking, K., & Ader, M. (2006). Why visceral fat is bad: mechanisms of the metabolic syndrome. Obesity (Silver.Spring). 14 Suppl 1:16S-19S.

Beunen, G., Malina, R. M., Lefevre, J., Claessens, A. L., Renson, R., Simons, J., Maes, H., Vanreusel, B., & Lysens, R. (1994). Size, fatness and relative fat distribution of males of contrasting maturity status during adolescence and as adults. International Journal of Obesity 18, 670-678.

Beunen, G. P., Lefevre, J., Philippaerts, R. M., Delvaux, K., Thomis, M., Claessens, A. L., Vanreusel, B., Lysens, R., Vanden Eynde, B., & Renson, R. (2004). Adolescent correlates of adult physical activity: a 26-year follow-up. Med Sci Sports Exerc. 36, 1930-1936.

Bielicki, T. & Hauspie, R. C. (1994). On the independence of adult stature from the timing of the adolesecnt growth spurt. American Journal of Human Biology 6, 245-247.

Bielicki, T., Koniarek, J., & Malina, R. M. (1984). Interrelationships among certain measures of growth and maturation rate in boys during adolescence. Annals of Human Biology 11, 201-210.

Bigaard, J., Frederiksen, K., Tjonneland, A., Thomsen, B. L., Overvad, K., Heitmann, B. L., & Sorensen, T. I. (2005). Waist circumference and body composition in relation to all-cause mortality in middle-aged men and women. Int.J Obes.(Lond). 29, 778-784.

Bingham, S. A. (1991). Limitations of the various methods for collecting dietary intake data. Ann.Nutr.Metab. 35, 117-127.

Biro, F. M., McMahon, R. P., Striegel-Moore, R., Crawford, P. B., Obarzanek, E., Morrison, J. A., Barton, B. A., & Falkner, F. (2001). Impact of timing of pubertal maturation on growth in black and white female adolescents: The National Heart, Lung, and Blood Institute Growth and Health Study. J.Pediatr. 138, 636-643.

Bjorntorp, P. (1997). Hormonal control of regional fat distribution. Hum.Reprod. 12 Suppl 1:21-5., 21-25.

Boreham, C., Twisk, J., Neville, C., Savage, M., Murray, L., & Gallagher, A. (2002). Associations between physical fitness and activity patterns during adolescence and cardiovascular risk factors in young adulthood: the Northern Ireland Young Hearts Project. Int.J.Sports Med. 23 Suppl 1:S22-6.

Boule, N. G., Bouchard, C., & Tremblay, A. (2005). Physical fitness and the metabolic syndrome in adults from the Quebec Family Study. Can.J Appl.Physiol. 30, 140-156.

Bradley, C. B., McMurray, R. G., Harrell, J. S., & Deng, S. (2000). Changes in common activities of 3rd through 10th graders: the CHIC study. Med Sci Sports Exerc. 32, 2071-2078.

Brage, S., Wedderkopp, N., Ekelund, U., Franks, P. W., Wareham, N. J., Andersen, L. B., & Froberg, K. (2004). Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children: the European Youth Heart Study (EYHS). J R Soc Med. 27, 2141-2148.

Bratberg, G. H., Nilsen, T. I., Holmen, T. L., & Vatten, L. J. (2007). Early sexual maturation, central adiposity and subsequent overweight in late adolescence. a four-year follow-up of 1605 adolescent Norwegian boys and girls: the Young HUNT study. BMC.Public Health. 7:54.

Brawley, L. R., Martin, K. A., & Gyuresik, N. C. (1998). Problems in assessing perceived barriers to exercise: Confusing obstacles with attributions and excuses. In Advances in sport and exercise psychology, ed. Duda, J. L., pp. 337-350. Fitness Information Technology, Moragantown,WV.

Bray, G. A., Bouchard, C., & James, W. P. T. (1998). Handbook of obesity NY: Marcel Dekker, New York.

Brien, S. E. & Katzmarzyk, P. T. (2006). Physical activity and the metabolic syndrome in Canada. Appl.Physiol Nutr.Metab. 31, 40-47.

British Heart Foundation. Couch kids - the growing epidemic. 2000. London, British Heart Foundation. Ref Type: Report.

Brittain, D. R., Baillargeon, T., McElroy, M., Aaron, D. J., & Gyurcsik, N. C. (2006). Barriers to moderate physical activity in adult lesbians. Women Health. 43, 75-92.

Brown, J. D., Halpern, C. T., & L'Engle, K. L. (2005). Mass media as a sexual super peer for early maturing girls. J Adolesc.Health. 36, 420-427.

Brown, R. A., Lewinsohn, P. M., Seeley, J. R., & Wagner, E. F. (1996). Cigarette smoking, major depression, and other psychiatric disorders among adolescents. J Am Acad.Child Adolesc.Psychiatry. 35, 1602-1610.

Byberg, L., Zethelius, B., McKeigue, P. M., & Lithell, H. O. (2001). Changes in physical activity are associated with changes in metabolic cardiovascular risk factors. J Hypertens. 44, 2134-2139.

Calle, E. E., Thun, M. J., Petrelli, J. M., Rodriguez, C., & Heath, C. W., Jr. (1999). Body-mass index and mortality in a prospective cohort of U.S. adults. N.Engl.J Med. 341, 1097-1105.

Cameron, N. (2002). Assessment of Maturation. In Human Growth and Development, ed. Cameron, N., pp. 363-382. Academic Press, San Diego.

Cameron, N. & Demerath, E. W. (2002). Critical periods in human growth and their relationship to diseases of aging. Am.J.Phys.Anthropol. Suppl 35:159-84., 159-184.

Campbell, P. T., Katzmarzyk, P. T., Malina, R. M., Rao, D. C., Perusse, L., & Bouchard, C. (2001). Prediction of physical activity and physical work capacity (PWC150) in young adulthood from childhood and adolescence with consideration of parental measures. Am J Hum.Biol. 13, 190-196.

Carroll, S., Cooke, C. B., & Butterly, R. J. (2000). Metabolic clustering, physical activity and fitness in nonsmoking, middle-aged men. Med Sci.Sports Exerc. 32, 2079-2086.

Caspersen, C. J., Pereira, M. A., & Curran, K. M. (2000). Changes in physical activity patterns in the United States, by sex and cross-sectional age. Med Sci.Sports Exerc. 32, 1601-1609.

Caspi, A. & Moffitt, T. E. (1991). Individual differences are accentuated during periods of social change: the sample case of girls at puberty. J.Pers.Soc.Psychol. 61, 157-168.

Chiolero, A., Faeh, D., Paccaud, F., & Cornuz, J. (2008). Consequences of smoking for body weight, body fat distribution, and insulin resistance. Am J Clin.Nutr. 87, 801-809.

Chumlea, W. C., Schubert, C. M., Roche, A. F., Kulin, H. E., Lee, P. A., Himes, J. H., & Sun, S. S. (2003). Age at menarche and racial comparisons in US girls. BMC Cardiovasc Disord. 111, 110-113.

Cole, T. J., Bellizzi, M. C., Flegal, K. M., & Dietz, W. H. (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey. J Med Food. 320, 1240-1243.

Copeland, J. L., Kowalski, K. C., Donen, R. M., & Tremblay, M. S. (2005). Convergent validity of the physical activity questionnaire for adults: the new member of the PAQ family. Journal of Physical Activity and Health 2, 216-229.

Costello, E. J., Sung, M., Worthman, C., & Angold, A. (2007). Pubertal maturation and the development of alcohol use and abuse. Drug Alcohol Depend. 88 Suppl 1:S50-9.

Crocker, P. R., Bailey, D. A., Faulkner, R. A., Kowalski, K. C., & McGrath, R. (1997). Measuring general levels of physical activity: preliminary evidence for the Physical Activity Questionnaire for Older Children. Med Sci Sports Exerc. 29, 1344-1349.

Davison, K. K., Susman, E. J., & Birch, L. L. (2003). Percent body fat at age 5 predicts earlier pubertal development among girls at age 9. BMC Cardiovasc Disord. 111, 815-821.

Davison, K. K., Werder, J. L., Trost, S. G., Baker, B. L., & Birch, L. L. (2007). Why are early maturing girls less active? Links between pubertal development, psychological well-being, and physical activity among girls at ages 11 and 13. Soc.Sci Med. 64, 2391-2404.

Deardorff, J., Gonzales, N. A., Christopher, F. S., Roosa, M. W., & Millsap, R. E. (2005). Early puberty and adolescent pregnancy: the influence of alcohol use. BMC Cardiovasc Disord. 116, 1451-1456.

Denburg, M. R., Silfen, M. E., Manibo, A. M., Chin, D., Levine, L. S., Ferin, M., McMahon, D. J., Go, C., & Oberfield, S. E. (2002). Insulin sensitivity and the insulin-like growth factor system in prepubertal boys with premature adrenarche. J Clin.Endocrinol.Metab. 87, 5604-5609.

Department of Health and Human Services. Physical activity and health: A report of the surgeon general.1996. Atlanta, GA, DHHS & CDCP.Ref Type: Report.

Department of Health and Human Services. The Surgeon General's Call To Action To Prevent and Decrease Overweight and Obesity. 2001. Atlanta, GA, DHHS & CDCP. Ref Type: Report.

Despres, J. P. & Lemieux, I. (2006). Abdominal obesity and metabolic syndrome. Nature. 444, 881-887.

Despres, J. P., Moorjani, S., Lupien, P. J., Tremblay, A., Nadeau, A., & Bouchard, C. (1990). Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. Arteriosclerosis. 10, 497-511.

Dhingra, R., Sullivan, L., Jacques, P. F., Wang, T. J., Fox, C. S., Meigs, J. B., D'agostino, R. B., Gaziano, J. M., & Vasan, R. S. (2007). Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 116, 480-488.

Dick, D. M., Rose, R. J., Viken, R. J., & Kaprio, J. (2000). Pubertal timing and substance use: associations between and within families across late adolescence. Dev.Psychol. 36, 180-189.

Dietz, W. H. (1994). Critical periods in childhood for the development of obesity. Am.J.Clin.Nutr. 59, 955-959.

DiPietro, L., Mossberg, H. O., & Stunkard, A. J. (1994). A 40-year history of overweight children in Stockholm: life-time overweight, morbidity, and mortality. Int.J Obes.Relat Metab Disord. 18, 585-590.

Dorn, L. D., Susman, E. J., & Ponirakis, A. (2003). Pubertal timing and adolescent adjustment and behavior: Conclusions vary by rater. Journal of Youth and Adolescence 32, 157-167.

Dubas, J. S., Graber, J. A., & Petersen, A. C. (1991a). A longitudinal investigation of adolescents changing perceptions of pubertal timing. Developmental Psychology 27, 580-586.

Dubas, J. S., Graber, J. A., & Petersen, A. C. (1991b). The effects of pubertal development on achievement during adolescence. American Journal of Education 99, 444-460.

DuBose, K. D., Eisenmann, J. C., & Donnelly, J. E. (2007). Aerobic fitness attenuates the metabolic syndrome score in normal-weight, at-risk-for-overweight, and overweight children. BMC Cardiovasc Disord. 120, e1262-e1268.

Dunger, D. B., Ahmed, M. L., & Ong, K. K. (2005). Effects of obesity on growth and puberty. Best.Pract.Res.Clin.Endocrinol.Metab. 19, 375-390.

Eckel, R. H., Grundy, S. M., & Zimmet, P. Z. (2005). The metabolic syndrome. Lancet. 365, 1415-1428.

Eisenmann, J. C., Welk, G. J., Wickel, E. E., & Blair, S. N. (2004). Stability of variables associated with the metabolic syndrome from adolescence to adulthood: the Aerobics Center Longitudinal Study. Am.J.Hum.Biol. 16, 690-696.

Eisenmann, J. C., Wickel, E. E., Welk, G. J., & Blair, S. N. (2005). Relationship between adolescent fitness and fatness and cardiovascular disease risk factors in adulthood: the Aerobics Center Longitudinal Study (ACLS). Am.Heart J. 149, 46-53.

Ekelund, U., Brage, S., Froberg, K., Harro, M., Anderssen, S. A., Sardinha, L. B., Riddoch, C., & Andersen, L. B. (2006). TV viewing and physical activity are independently associated with metabolic risk in children: the European Youth Heart Study. PLoS.Med. 3, e488.

Esliger, D. W., Copeland, J. L., Barnes, J. D., & Tremblay, M. S. (2005). Standardizing and optimizing the use of accelerometer data for free-living physical activity monitoring. Journal of Physical Activity and Health 3, 366-383.

Esliger, D. W. & Tremblay, M. S. (2006). Technical reliability assessment of three accelerometer models in a mechanical setup. Med Sci Sports Exerc. 38, 2173-2181.

Esmaillzadeh, A., Mirmiran, P., & Azizi, F. (2005). Whole-grain consumption and the metabolic syndrome: a favorable association in Tehranian adults. Eur.J Clin.Nutr. 59, 353-362.

Feng, Y., Hong, X., Wilker, E., Li, Z., Zhang, W., Jin, D., Liu, X., Zang, T., Xu, X., & Xu, X. (2008). Effects of age at menarche, reproductive years, and menopause on metabolic risk factors for cardiovascular diseases. Atherosclerosis. 196, 590-597.

Ferreira, I., Twisk, J. W., Van Mechelen, W., Kemper, H. C., & Stehouwer, C. D. (2005). Development of fatness, fitness, and lifestyle from adolescence to the age of 36 years: determinants of the metabolic syndrome in young adults: the amsterdam growth and health longitudinal study. Arch.Intern.Med. 165, 42-48.

Ford, E. S., Giles, W. H., & Dietz, W. H. (2002). Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. J Hum Hypertens. 287, 356-359.

Ford, E. S., Giles, W. H., & Mokdad, A. H. (2004). Increasing prevalence of the metabolic syndrome among U.S. Adults. J R Soc Med. 27, 2444-2449.

Foshee, V. A., Ennett, S. T., Bauman, K. E., Granger, D. A., Benefield, T., Suchindran, C., Hussong, A. M., Karriker-Jaffe, K. J., & DuRant, R. H. (2007). A test of biosocial models of adolescent cigarette and alcohol involvement. Journal of Early Adolescence 27, 4-39.

Freedman, D. S., Khan, L. K., Serdula, M. K., Dietz, W. H., Srinivasan, S. R., & Berenson, G. S. (2004). Inter-relationships among childhood BMI, childhood height, and adult obesity: the Bogalusa Heart Study. Int.J Obes.Relat Metab Disord. 28, 10-16.

Froberg, K. & Andersen, L. B. (2005). Mini review: physical activity and fitness and its relations to cardiovascular disease risk factors in children. Int.J Obes.(Lond). 29 Suppl 2:S34-9., S34-S39.

Frontini, M. G., Srinivasan, S. R., & Berenson, G. S. (2003). Longitudinal changes in risk variables underlying metabolic Syndrome X from childhood to young adulthood in female subjects with a history of early menarche: the Bogalusa Heart Study. Int.J.Obes.Relat Metab Disord. 27, 1398-1404.

Fuchs, R., Powell, K. E., Semmer, N. K., Dwyer, J. H., Lippert, P., & Hoffmeister, H. (1988). Patterns of physical activity among German adolescents: the Berlin-Bremen Study. Prev.Med. 17, 746-763.

Galambos, N. L., Kolaric, G. C., Sears, H. A., & Maggs, J. L. (1999). Adolescents' subjective age: An indicator of perceived maturity. Journal of Research on Adolescence 9, 309-337.

Ge, X., Conger, R. D., & Elder, G. H., Jr. (1996). Coming of age too early: pubertal influences on girls' vulnerability to psychological distress. Child Dev. 67, 3386-3400.

Ge, X., Conger, R. D., & Elder, G. H., Jr. (2001). Pubertal transition, stressful life events, and the emergence of gender differences in adolescent depressive symptoms. Dev.Psychol. 37, 404-417.

Glickman, S. G., Marn, C. S., Supiano, M. A., & Dengel, D. R. (2004). Validity and reliability of dual-energy X-ray absorptiometry for the assessment of abdominal adiposity. J Appl.Physiol. 97, 509-514.

Goldstein, H., Rashbash, I., Plewis, I., Draper, D., Browne, W., Yang, M., Woodhouse, G., & Healy, M. (1998). A users's guide to MLwiN, pp. 1-140. Institute of Education, University of London, London.

Goran, M. I., Gower, B. A., Nagy, T. R., & Johnson, R. K. (1998). Developmental changes in energy expenditure and physical activity in children: evidence for a decline in physical activity in girls before puberty. Pediatrics 101, 887-891.

Goris, A. H., Westerterp-Plantenga, M. S., & Westerterp, K. R. (2000). Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. Am J Clin.Nutr. 71, 130-134.

Goris, A. H. & Westerterp, K. R. (1999). Underreporting of habitual food intake is explained by undereating in highly motivated lean women. J Nutr. 129, 878-882.

Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (1999). The vulnerable transition: puberty and the development of eating pathology and negative mood. Womens Health Issues. 9, 107-114.

Graber, J. A., Brooks-Gunn, J., & Warren, M. P. (2006). Pubertal effects on adjustment in girls: Moving from demonstrating effects to identifying pathways. Journal of Youth and Adolescence 35, 413-423.

Graber, J. A., Lewinsohn, P. M., Seeley, J. R., & Brooks-Gunn, J. (1997). Is psychopathology associated with the timing of pubertal development? J.Am.Acad.Child Adolesc.Psychiatry. 36 , 1768-1776.

Graber, J. A., Seeley, J. R., Brooks-Gunn, J., & Lewinsohn, P. M. (2004). Is pubertal timing associated with psychopathology in young adulthood. J.Am.Acad.Child Adolesc.Psychiatry. 43 , 718-726.

Grundy, S. M. (2000). Metabolic complications of obesity. Endocrine. 13, 155-165.

Grundy, S. M., Abate, N., & Chandalia, M. (2002). Diet composition and the metabolic syndrome: what is the optimal fat intake? Am.J.Med. 113 Suppl 9B:25S-29S.

Guo, S. S., Huang, C., Maynard, L. M., Demerath, E., Towne, B., Chumlea, W. C., & Siervogel, R. M. (2000). Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. Int.J.Obes.Relat Metab Disord. 24, 1628-1635.

Guo, S. S., Wu, W., Chumlea, W. C., & Roche, A. F. (2002). Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. Am.J.Clin.Nutr. 76, 653-658.

Gutin, B., Johnson, M. H., Humphries, M. C., Hatfield-Laube, J. L., Kapuku, G. K., Allison, J. D., Gower, B. A., Daniels, S. R., & Barbeau, P. (2007). Relationship of visceral adiposity to cardiovascular disease risk factors in black and white teens. Obesity (Silver.Spring). 15, 1029-1035.

Gyurcsik, N. C., Brawley, L. R., & Langhout, N. (2002). Acute thoughts, exercise consistency, and coping self-efficacy. Journal of Applied Social Psychology 32, 2134-2153.

Gyurcsik, N. C., Spink, K. S., Bray, S. R., Chad, K., & Kwan, M. (2006). An ecologically based examination of barriers to physical activity in students from grade seven through first-year university. J.Adolesc.Health. 38, 704-711.

Hagg, U. & Taranger, J. (1991). Height and height velocity in early, average and late maturers followed to the age of 25: a prospective longitudinal study of Swedish urban children from birth to adulthood. Ann.Hum.Biol. 18, 47-56.

Hallal, P. C., Victora, C. G., Azevedo, M. R., & Wells, J. C. (2006). Adolescent physical activity and health: a systematic review. Sports Med. 36, 1019-1030.

Han, T. S., Sattar, N., & Lean, M. (2006). ABC of obesity. Assessment of obesity and its clinical implications. J Med Food. 333, 695-698.

Harter, S. & Whitesell, N. R. (1996). Multiple pathways to self-reported depression and psychological adjustment among adolescents. Development and Psychopathology 8, 761-777.

Hasselstrom, H., Hansen, S. E., Froberg, K., & Andersen, L. B. (2002). Physical fitness and physical activity during adolescence as predictors of cardiovascular disease risk in young adulthood. Danish Youth and Sports Study. An eight-year follow-up study. Int.J.Sports Med. 23 Suppl 1:S27-31.

Heath, G. W., Pratt, M., Warren, C. W., & Kann, L. (1994). Physical activity patterns in American high school students. Results from the 1990 Youth Risk Behavior Survey. Arch.Pediatr.Adolesc.Med. 148, 1131-1136.

Heys, M., Schooling, C. M., Jiang, C., Cowling, B. J., Lao, X., Zhang, W., Cheng, K. K., Adab, P., Thomas, G. N., Lam, T. H., & Leung, G. M. (2007). Age of menarche and the metabolic syndrome in China. Epidemiology. 18, 740-746.

Himes, J. H., Obarzanek, E., Baranowski, T., Wilson, D. M., Rochon, J., & McClanahan, B. S. (2004). Early sexual maturation, body composition, and obesity in African-American girls. Obes.Res. 12 Suppl:64S-72S.

Hologic (1993). Hologic QDR 2000 Supine Lateral Bone Densitometer: Operator Manual and User's guide. pp. 5.1-5.28. Hologic Inc., Waltham, MA.

Hulens, M., Beunen, G., Claessens, A. L., Lefevre, J., Thomis, M., Philippaerts, R., Borms, J., Vrijens, J., Lysens, R., & Vansant, G. (2001). Trends in BMI among Belgian children, adolescents and adults from 1969 to 1996. Int.J Obes.Relat Metab Disord. 25, 395-399.

Ibanez, L., Dimartino-Nardi, J., Potau, N., & Saenger, P. (2000). Premature adrenarche--normal variant or forerunner of adult disease? Endocr.Rev. 21, 671-696.

Iuliano-Burns, S., Mirwald, R. L., & Bailey, D. A. (2001). Timing and magnitude of peak height velocity and peak tissue velocities for early, average, and late maturing boys and girls. Am.J.Hum.Biol. 13, 1-8.

Jamner, M. S., Spruijt-Metz, D., Bassin, S., & Cooper, D. M. (2004). A controlled evaluation of a school-based intervention to promote physical activity among sedentary adolescent females: project FAB. J.Adolesc.Health. 34, 279-289.

Janssen, I., Katzmarzyk, P. T., & Ross, R. (2004a). Duration of overweight and metabolic health risk in American men and women. Ann.Epidemiol. 14, 585-591.

Janssen, I., Katzmarzyk, P. T., & Ross, R. (2004b). Waist circumference and not body mass index explains obesity-related health risk. Am.J.Clin.Nutr. 79, 379-384.

Janz, K. F., Dawson, J. D., & Mahoney, L. T. (2002). Increases in physical fitness during childhood improve cardiovascular health during adolescence: the Muscatine Study. Int.J.Sports Med. 23 Suppl 1:S15-21.

Janz, K. F., Lutuchy, E. M., Wenthe, P., & Levy, S. M. (2008). Measuring activity in children and adolescents using self-report: PAQ-C and PAQ-A. Med Sci Sports Exerc. 40, 767-772.

Jensen, M. D. (2006). Is visceral fat involved in the pathogenesis of the metabolic syndrome? Human model. Obesity (Silver.Spring). 14 Suppl 1:20S-24S.

Kaltiala-Heino, R., Marttunen, M., Rantanen, P., & Rimpela, M. (2003). Early puberty is associated with mental health problems in middle adolescence. Soc.Sci Med. 57, 1055-1064.

Katzmarzyk, P. T., Perusse, L., & Bouchard, C. (1999). Genetics of abdominal visceral fat levels. Am J Hum.Biol. 11, 225-235.

Kelishadi, R., Cook, S. R., Motlagh, M. E., Gouya, M. M., Ardalan, G., Motaghian, M., Majdzadeh, R., & Ramezani, M. A. (2008). Metabolically obese normal weight and phenotypically obese metabolically normal youths: the CASPIAN Study. J Am Diet.Assoc. 108, 82-90.

Kelishadi, R., Razaghi, E. M., Gouya, M. M., Ardalan, G., Gheiratmand, R., Delavari, A., Motaghian, M., Ziaee, V., Siadat, Z. D., Majdzadeh, R., Heshmat, R., Barekati, H., Arabi, M. S., Heidarzadeh, A., & Shariatinejad, K. (2007). Association of physical activity and the metabolic syndrome in children and adolescents: CASPIAN Study. Horm.Res. 67, 46-52.

Kemper, H. C., Post, G. B., Twisk, J. W., & Van Mechelen, W. (1999). Lifestyle and obesity in adolescence and young adulthood: results from the Amsterdam Growth And Health Longitudinal Study (AGAHLS). Int.J.Obes.Relat Metab Disord. 23 Suppl 3:S34-40.

Kemper, H. C., Van Mechelen, W., Post, G. B., Snel, J., Twisk, J. W., van Lenthe, F. J., & Welten, D. C. (1997). The Amsterdam Growth and Health Longitudinal Study. The past (1976-1996) and future (1997-?). Int.J Sports Med. 18 Suppl 3:S140-50.

Kettaneh, A., Oppert, J. M., Heude, B., Deschamps, V., Borys, J. M., Lommez, A., Ducimetiere, P., & Charles, M. A. (2005). Changes in physical activity explain paradoxical relationship between baseline physical activity and adiposity changes in adolescent girls: the FLVS II study. Int J Obes.(Lond). 29, 586-593.

Kim, J. A. & Park, H. S. (2008). Association of abdominal fat distribution and cardiometabolic risk factors among obese Korean adolescents. Diabetes Metab. 34, 126-130.

Kimm, S. Y., Glynn, N. W., Kriska, A. M., Fitzgerald, S. L., Aaron, D. J., Similo, S. L., McMahon, R. P., & Barton, B. A. (2000). Longitudinal changes in physical activity in a biracial cohort during adolescence. Med Sci.Sports Exerc. 32, 1445-1454.

Kindblom, J. M., Lorentzon, M., Norjavaara, E., Lonn, L., Brandberg, J., Angelhed, J. E., Hellqvist, A., Nilsson, S., & Ohlsson, C. (2006). Pubertal timing is an independent predictor of central adiposity in young adult males: the Gothenburg osteoporosis and obesity determinants study. Ethn Dis. 55, 3047-3052.

Klasson-Heggebo, L. & Anderssen, S. A. (2003). Gender and age differences in relation to the recommendations of physical activity among Norwegian children and youth. Scand J.Med Sci.Sports. 13, 293-298.

Kopelman, P. G. & Albon, L. (1997). Obesity, non-insulin-dependent diabetes mellitus and the metabolic syndrome. Br.Med Bull. 53, 322-340.

Kvaavik, E., Tell, G. S., & Klepp, K. I. (2003). Predictors and tracking of body mass index from adolescence into adulthood: follow-up of 18 to 20 years in the Oslo Youth Study. Arch.Pediatr.Adolesc.Med. 157, 1212-1218.

Laitinen-Krispijn, S., Van der, E. J., Hazebroek-Kampschreur, A. A., & Verhulst, F. C. (1999). Pubertal maturation and the development of behavioural and emotional problems in early adolescence. Acta Psychiatr Scand 99, 16-25.

Laitinen, J., Power, C., & Jarvelin, M. R. (2001). Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. Am J Clin.Nutr. 74, 287-294.

Lakka, T. A., Laaksonen, D. E., Lakka, H. M., Mannikko, N., Niskanen, L. K., Rauramaa, R., & Salonen, J. T. (2003). Sedentary lifestyle, poor cardiorespiratory fitness, and the metabolic syndrome. Med Sci.Sports Exerc. 35, 1279-1286.

Lanza, S. T. & Collins, L. M. (2002). Pubertal timing and the onset of substance use in females during early adolescence. Prev.Sci. 3, 69-82.

Largo, R. H., Gasser, T., Prader, A., Stuetzle, W., & Huber, P. J. (1978). Analysis of the adolescent growth spurt, using smoothing spline functions. Annals of Human Biology 5, 421-434.

Laron, Z. (2004). Is obesity associated with early sexual maturation? BMC Cardiovasc Disord. 113, 171-172.

Lee, J. M., Appugliese, D., Kaciroti, N., Corwyn, R. F., Bradley, R. H., & Lumeng, J. C. (2007). Weight status in young girls and the onset of puberty. BMC Cardiovasc Disord. 119, e624-e630.

Lee, W. Y., Jung, C. H., Park, J. S., Rhee, E. J., & Kim, S. W. (2005). Effects of smoking, alcohol, exercise, education, and family history on the metabolic syndrome as defined by the ATP III. Diabetes Res.Clin.Pract. 67, 70-77.

Lefevre, J., Philippaerts, R., Delvaux, K., Thomis, M., Claessens, A. L., Lysens, R., Renson, R., Vanden Eynde, B., Vanreusel, B., & Beunen, G. (2002). Relation between cardiovascular risk factors at adult age, and physical activity during youth and adulthood: the Leuven Longitudinal Study on Lifestyle, Fitness and Health. Int.J.Sports Med. 23 Suppl 1:S32-8.

Lemieux, I. (2004). Energy partitioning in gluteal-femoral fat: does the metabolic fate of triglycerides affect coronary heart disease risk? Arterioscler.Thromb.Vasc.Biol. 24, 795-797.

Lemieux, I., Pascot, A., Prud'homme, D., Almeras, N., Bogaty, P., Nadeau, A., Bergeron, J., & Despres, J. P. (2001). Elevated C-reactive protein: another component of the atherothrombotic profile of abdominal obesity. Arterioscler.Thromb.Vasc.Biol. 21, 961-967.

Lenfant, C. & Savage, P. J. (1995). The early natural history of atherosclerosis and hypertension in the young: National Institutes of Health perspectives. Am.J.Med Sci. 310 Suppl 1:S3-7.

Liu, Y. X., Wikland, K. A., & Karlberg, J. (2000). New reference for the age at childhood onset of growth and secular trend in the timing of puberty in Swedish. Acta Paediatr. 89, 637-643.

Livingstone, M. B., Robson, P. J., Wallace, J. M., & McKinley, M. C. (2003). How active are we? Levels of routine physical activity in children and adults. Proc.Nutr.Soc. 62, 681-701.

Lohman, T. G. (2005). Dual energy X-ray absorptiometry. In Human Body Composition, eds. Roche, A. F., Heymsfield, S. B., & Lohman, T. G., pp. 63-78. Human Kinetics, Champaign, IL.

Lutsey, P. L., Steffen, L. M., & Stevens, J. (2008). Dietary intake and the development of the metabolic syndrome: the Atherosclerosis Risk in Communities study. Circulation. 117, 754-761.

MacDonald, P. C., Edman, C. D., Hemsell, D. L., Porter, J. C., & Siiteri, P. K. (1978). Effect of obesity on conversion of plasma androstenedione to estrone in postmenopausal women with and without endometrial cancer. Am J Obstet.Gynecol. 130, 448-455.

Maddux, J. F. & Gosselin, J. T. (2003). Maddux. In Maddux, eds. Leary, M. R. & Tangney, J. P., Guilford, New York.

Magarey, A. M., Daniels, L. A., Boulton, T. J., & Cockington, R. A. (2003). Predicting obesity in early adulthood from childhood and parental obesity. Int.J.Obes.Relat Metab Disord. 27, 505-513.

Malina, R. M. (1978). Adolescent growth and maturation: Selected aspects of current research. Yearbook of Physical Anthropology 21, 63-94.

Malina, R. M., Bouchard, C., & Bar-Or, O. (2004). Growth, maturation and physical activity, 2 ed. Human Kinetics, Illionois.

Malina, R. M., Cumming, S. P., Morano, P. J., Barron, M., & Miller, S. J. (2005). Maturity status of youth football players: a noninvasive estimate. Med.Sci.Sports Exerc. 37, 1044-1052.

Malina, R. M., Koziel, S., & Bielicki, T. (1999). Variation in subcutaneous adipose tissue distribution associated with age, sex, and maturation. Am J Hum.Biol. 11, 189-200.

Malis, C., Rasmussen, E. L., Poulsen, P., Petersen, I., Christensen, K., Beck-Nielsen, H., Astrup, A., & Vaag, A. A. (2005). Total and regional fat distribution is strongly influenced by genetic factors in young and elderly twins. Obes.Res. 13, 2139-2145.

Marshall, W. A. & Tanner, J. M. (1969). Variations in pattern of pubertal changes in girls. Arch.Dis.Child 44, 291-303.

Marshall, W. A. & Tanner, J. M. (1970). Variations in the pattern of pubertal changes in boys. Arch.Dis.Child 45, 13-23.

Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. J Hypertens. 28, 412-419.

McLeroy, K. R., Bibeau, D., Steckler, A., & Glanz, K. (1988). An ecological perspective on health promotion programs. Health Educ.Q. 15, 351-377.

Metcalf, B. S., Voss, L. D., & Wilkin, T. J. (2002). Accelerometers identify inactive and potentially obese children (EarlyBird 3). Arch.Dis.Child. 87, 166-167.

Michaud, P. A., Suris, J. C., & Deppen, A. (2006). Gender-related psychological and behavioural correlates of pubertal timing in a national sample of Swiss adolescents. Mol.Cell Endocrinol. 254-255:172-8.

Mirwald, R. L., Baxter-Jones, A. D., Bailey, D. A., & Beunen, G. P. (2002). An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc. 34, 689-694.

Motl, R. W., Birnbaum, A. S., Kubik, M. Y., & Dishman, R. K. (2004). Naturally occurring changes in physical activity are inversely related to depressive symptoms during early adolescence. Psychosom.Med. 66, 336-342.

Muinich Keizer, S. M. & Mul, D. (2001). Trends in pubertal development in Europe. Hum.Reprod.Update. 7, 287-291.

Mundt, C. A., Baxter-Jones, A. D., Whiting, S. J., Bailey, D. A., Faulkner, R. A., & Mirwald, R. L. (2006). Relationships of activity and sugar drink intake on fat mass development in youths. Med Sci Sports Exerc. 38, 1245-1254.

Must, A. (1996). Morbidity and mortality associated with elevated body weight in children and adolescents. Am J Clin.Nutr. 63, 445S-447S.

Must, A., Bandini, L. G., Tybor, D. J., Phillips, S. M., Naumova, E. N., & Dietz, W. H. (2007). Activity, inactivity, and screen time in relation to weight and fatness over adolescence in girls. Obesity (Silver.Spring). 15, 1774-1781.

Must, A., Jacques, P. F., Dallal, G. E., Bajema, C. J., & Dietz, W. H. (1992). Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N.Engl.J Med. 327, 1350-1355.

Must, A. & Tybor, D. J. (2005). Physical activity and sedentary behavior: a review of longitudinal studies of weight and adiposity in youth. Int.J.Obes.(Lond). 29 Suppl 2:S84-96.

NCEP (2001). Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection,Evaluation,And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). J Hum Hypertens. 285, 2486-2497.

Niederauer, C. M., Binkley, T. L., & Specker, B. L. (2006). Effect of truncal adiposity on plasma lipid and lipoprotein concentrations. J Nutr.Health Aging. 10, 154-160.

Niven, A. G., Fawkner, S. G., Knowles, A., & Stephenson, C. (2007). Maturational differences in physical self-perceptions and the relationship with physical activity in early adolescent girls. Pediatric Exercise Science 19, 472-480.

O'dea, J. A. (2003). Why do kids eat healthful food? Perceived benefits of and barriers to healthful eating and physical activity among children and adolescents. J.Am.Diet.Assoc. 103, 497-501.

Osmond, C., Barker, D. J., Winter, P. D., Fall, C. H., & Simmonds, S. J. (1993). Early growth and death from cardiovascular disease in women. J Med Food. 307, 1519-1524.

Ovesen, L. (2006). Adolescence: a critical period for long-term tracking of risk for coronary heart disease? Ann.Nutr.Metab. 50, 317-324.

Palaniappan, L., Carnethon, M. R., Wang, Y., Hanley, A. J., Fortmann, S. P., Haffner, S. M., & Wagenknecht, L. (2004). Predictors of the incident metabolic syndrome in adults: the Insulin Resistance Atherosclerosis Study. J R Soc Med. 27, 788-793.

Pan, Y. & Pratt, C. A. (2008). Metabolic syndrome and its association with diet and physical activity in US adolescents. J Am Diet.Assoc. 108, 276-286.

Parent, A. S., Teilmann, G., Juul, A., Skakkebaek, N. E., Toppari, J., & Bourguignon, J. P. (2003). The timing of normal puberty and the age limits of sexual precocity: variations around the world, secular trends, and changes after migration. Endocr.Rev. 24, 668-693.

Park, Y. W., Heymsfield, S. B., & Gallagher, D. (2002). Are dual-energy X-ray absorptiometry regional estimates associated with visceral adipose tissue mass? Int.J Obes.Relat Metab Disord. 26, 978-983.

Pereira, M. A., Jacobs, D. R., Jr., Van Horn, L., Slattery, M. L., Kartashov, A. I., & Ludwig, D. S. (2002). Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. J Hum Hypertens. 287, 2081-2089.

Petersen, A. C. (1988). Adolescent Development. Annual Review of Psychology 39, 583-607.

Petersen, A. C. & Crockett, L. (1985). Pubertal timing and grade effects of adjustment. Journal of Youth and Adolescence 14, 191-206.

Petersen, A. C., Crockett, L., Richards, M., & Boxer, A. (1988). A self-report measure of pubertal status: Reliability, validity, and initial norms. Journal of Youth and Adolescence 17, 117-133.

Petersen, A. C. & Taylor, B. (1980). The biological approach to adolescence: Biological change and psychosocial adaptation. In Handbook of adolescent psychology, ed. Aldelson, J., pp. 117-155. Wiley, New York.

Poortvliet, E., Yngve, A., Ekelund, U., Hurtig-Wennlof, A., Nilsson, A., Hagstromer, M., & Sjostrom, M. (2003). The European Youth Heart Survey (EYHS): an international study that addresses the multi-dimensional issues of CVD risk factors. Forum Nutr. 56:254-6.

Potau, N., Ibanez, L., Rique, S., Sanchez-Ufarte, C., & de Zegher, F. (1999). Pronounced adrenarche and precocious pubarche in boys. Horm.Res. 51, 238-241.

Power, C., Lake, J. K., & Cole, T. J. (1997). Body mass index and height from childhood to adulthood in the 1958 British born cohort. Am J Clin.Nutr. 66, 1094-1101.

Puyau, M. R., Adolph, A. L., Vohra, F. A., Zakeri, I., & Butte, N. F. (2004). Prediction of activity energy expenditure using accelerometers in children. Med Sci Sports Exerc. 36, 1625-1631.

Ragland, D. R. (1992). Dichotomizing continuous outcome variables: dependence of the magnitude of association and statistical power on the cutpoint. Epidemiology. 3, 434-440.

Raitakari, O. T., Porkka, K. V., Taimela, S., Telama, R., Rasanen, L., & Viikari, J. S. (1994). Effects of persistent physical activity and inactivity on coronary risk factors in children and young adults. The Cardiovascular Risk in Young Finns Study. Am.J.Epidemiol. 140, 195-205.

Remsberg, K. E., Demerath, E. W., Schubert, C. M., Chumlea, W. C., Sun, S. S., & Siervogel, R. M. (2005). Early menarche and the development of cardiovascular disease risk factors in adolescent girls: the Fels Longitudinal Study. J.Clin.Endocrinol.Metab. 90, 2718-2724.

Reynolds, E. L. (1946). Sexual maturation and the growth of fat, muscle and bone in girls. Child Development 17, 121-144.

Riddoch, C. J., Bo, A. L., Wedderkopp, N., Harro, M., Klasson-Heggebo, L., Sardinha, L. B., Cooper, A. R., & Ekelund, U. (2004). Physical activity levels and patterns of 9- and 15-yr-old European children. Med Sci.Sports Exerc. 36, 86-92.

Riddoch, C. J., Mattocks, C., Deere, K., Saunders, J., Kirkby, J., Tilling, K., Leary, S. D., Blair, S. N., & Ness, A. R. (2007). Objective measurement of levels and patterns of physical activity. Arch.Dis.Child. 92, 963-969.

Robbins, L. B., Gretebeck, K. A., Kazanis, A. S., & Pender, N. J. (2006). Girls on the Move Program to Increase Physical Activity Participation. Nurs.Res. 55, 206-216.

Roche, A. F., Guo, S. S., & Towne, B. (1997). Opportunities and difficulties in long-term studies of growth. Int.J Sports Med. 18 Suppl 3:S151-61.

Roche, A. F., Wainer, H., & Thissen, D. (1975). The RWT method for the prediction of adult stature. Pediatrics 56, 1026-1033.

Roemmich, J. N., Clark, P. A., Weltman, A., & Rogol, A. D. (1997). Alterations in growth and body composition during puberty. I. Comparing multicompartment body composition models. J Appl.Physiol. 83, 927-935.

Rowlands, A. V., Eston, R. G., & Ingledew, D. K. (1999). Relationship between activity levels, aerobic fitness, and body fat in 8- to 10-yr-old children. J.Appl.Physiol. 86, 1428-1435.

Ruble, D. N. & Brooks-Gunn, J. (1982). The experience of menarche. Child Dev. 53, 1557-1566.

Sahyoun, N. R., Jacques, P. F., Zhang, X. L., Juan, W., & McKeown, N. M. (2006). Whole-grain intake is inversely associated with the metabolic syndrome and mortality in older adults. Am J Clin.Nutr. 83, 124-131.

Sallis, J. F. (1993). Epidemiology of physical activity and fitness in children and adolescents. Crit Rev.Food Sci.Nutr. 33, 403-408.

Sallis, J. F. (2000). Age-related decline in physical activity: a synthesis of human and animal studies. Med Sci.Sports Exerc. 32, 1598-1600.

Sallis, J. F., Prochaska, J. J., & Taylor, W. C. (2000). A review of correlates of physical activity of children and adolescents. Med.Sci.Sports Exerc. 32, 963-975.

Sallis, J. F. & Saelens, B. E. (2000). Assessment of physical activity by self-report: status, limitations, and future directions. Res.Q.Exerc Sport. 71, S1-14.

Sallis, J. F., Strikmiller, P. K., Harsha, D. W., Feldman, H. A., Ehlinger, S., Stone, E. J., Williston, J., & Woods, S. (1996). Validation of interviewer- and self-administered physical activity checklists for fifth grade students. Med.Sci.Sports Exerc. 28, 840-851.

Scammon, R. E. (1930). The measurement of the body in childhood. In The measurement of man, eds. Harris, J. A., Jackson, C. M., Paterson, D. G., & Scammon, R. E., pp. 173-215. University of Minnesota Press, Minneapolis.

Schroeder, B. A. (1992). Human Growth and Development West Publishing Company, St. Paul.

Selvin, S. (1987). Two issues concerning the analysis of grouped data. Eur.J Epidemiol. 3, 284-287.

Seppala-Lindroos, A., Vehkavaara, S., Hakkinen, A. M., Goto, T., Westerbacka, J., Sovijarvi, A., Halavaara, J., & Yki-Jarvinen, H. (2002). Fat accumulation in the liver is associated with defects in insulin suppression of glucose production and serum free fatty acids independent of obesity in normal men. J Clin.Endocrinol.Metab. 87, 3023-3028.

Serdula, M. K., Ivery, D., Coates, R. J., Freedman, D. S., Williamson, D. F., & Byers, T. (1993). Do obese children become obese adults? A review of the literature. Prev.Med. 22, 167-177.

Shephard, R. J. (2003). Limits to the measurement of habitual physical activity by questionnaires. Br.J.Sports Med. 37, 197-206.

Sherar, L., Baxter-Jones, A., Esliger, D., & Tremblay, M. (2008a). Response. Med Sci Sports Exerc. 40, 980.

Sherar, L. B., Baxter-Jones, A. D., Faulkner, R. A., & Russell, K. W. (2007a). Do physical maturity and birth date predict talent in male youth ice hockey players? J.Sports Sci. 25, 879-886.

Sherar, L. B., Baxter-Jones, A. D., & Mirwald, R. L. (2004). Limitations to the use of secondary sex characteristics for gender comparisons. Ann.Hum.Biol. 31, 586-593.

Sherar, L. B., Esliger, D. W., Baxter-Jones, A. D., & Tremblay, M. S. (2007b). Age and gender differences in youth physical activity: does physical maturity matter? Med Sci Sports Exerc. 39, 830-835.

Sherar, L. B., Gyurcsik, N. C., Humbert, M. L., Dyck, R. F., Fowler-Kerry, S., & Baxter-Jones, A. D. G. (2009). Activity and barriers in girls (8-16 yrs) based on grade and maturity status. Med Sci Sports Exerc. In Press.

Siegel, J. M., Yancey, A. K., Aneshensel, C. S., & Schuler, R. (1999). Body image, perceived pubertal timing, and adolescent mental health. J.Adolesc.Health. 25, 155-165.

Simon, A. E., Wardle, J., Jarvis, M. J., Steggles, N., & Cartwright, M. (2003). Examining the relationship between pubertal stage, adolescent health behaviours and stress. Psychological Medicine 33, 1369-1379.

Sinha, R., Dufour, S., Petersen, K. F., LeBon, V., Enoksson, S., Ma, Y. Z., Savoye, M., Rothman, D. L., Shulman, G. I., & Caprio, S. (2002). Assessment of skeletal muscle triglyceride content by (1)H nuclear magnetic resonance spectroscopy in lean and obese adolescents: relationships to insulin sensitivity, total body fat, and central adiposity. Ethn Dis. 51, 1022-1027.

St Onge, M. P., Janssen, I., & Heymsfield, S. B. (2004). Metabolic syndrome in normal-weight Americans: new definition of the metabolically obese, normal-weight individual. Diabetes Care 27, 2222-2228.

Stevens, J., Murray, D. M., Baggett, C. D., Elder, J. P., Lohman, T. G., Lytle, L. A., Pate, R. R., Pratt, C. A., Treuth, M. S., Webber, L. S., & Young, D. R. (2007). Objectively assessed associations between physical activity and body composition in middle-school girls: the Trial of Activity for Adolescent Girls. Am J Epidemiol. 166, 1298-1305.

Stevens, J., Murray, D. M., Catellier, D. J., Hannan, P. J., Lytle, L. A., Elder, J. P., Young, D. R., Simons-Morton, D. G., & Webber, L. S. (2005). Design of the Trial of Activity in Adolescent Girls (TAAG). Contemp.Clin.Trials. 26, 223-233.

Stone, E. J., McKenzie, T. L., Welk, G. J., & Booth, M. L. (1998). Effects of physical activity interventions in youth. Review and synthesis. Am.J.Prev.Med. 15, 298-315.

Story, M., Neumark-Sztainer, D., & French, S. (2002). Individual and environmental influences on adolescent eating behaviors. J Am Diet.Assoc. 102, S40-S51.

Stratton, I. M., Adler, A. I., Neil, H. A., Matthews, D. R., Manley, S. E., Cull, C. A., Hadden, D., Turner, R. C., & Holman, R. R. (2000). Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. J Med Food. 321, 405-412.

Strauss, R. S., Rodzilsky, D., Burack, G., & Colin, M. (2001). Psychosocial correlates of physical activity in healthy children. Arch.Pediatr.Adolesc.Med. 155, 897-902.

Sun, S. S., Liang, R., Huang, T. T., Daniels, S. R., Arslanian, S., Liu, K., Grave, G. D., & Siervogel, R. M. (2008). Childhood obesity predicts adult metabolic syndrome: the Fels Longitudinal Study. J Pediatr. 152, 191-200.

Tanaka, S., Togashi, K., Rankinen, T., Perusse, L., Leon, A. S., Rao, D. C., Skinner, J. S., Wilmore, J. H., Despres, J. P., & Bouchard, C. (2004). Sex differences in the relationships of abdominal fat to cardiovascular disease risk among normal-weight white subjects. Int.J Obes.Relat Metab Disord. 28, 320-323.

Tanner, J. M. (1962). Growth at Adolescence, 2 ed. Blackwell Scientific Publications, Oxford.

Tanner, J. M. (1989). Foetus into man. Physical growth from conception to maturity, 2 ed. Castlemead Publications, London.

Teixeira, P. J., Sardinha, L. B., Going, S. B., & Lohman, T. G. (2001). Total and regional fat and serum cardiovascular disease risk factors in lean and obese children and adolescents. Obes.Res. 9, 432-442.

Telama, R. & Yang, X. (2000). Decline of physical activity from youth to young adulthood in Finland. Med.Sci.Sports Exerc. 32, 1617-1622.

Telama, R., Yang, X., Viikari, J., Valimaki, I., Wanne, O., & Raitakari, O. (2005). Physical activity from childhood to adulthood: a 21-year tracking study. Am J Prev.Med. 28, 267-273.

Tergerson, J. L. & King, K. A. (2002). Do perceived cues, benefits, and barriers to physical activity differ between male and female adolescents? J.Sch Health. 72, 374-380.

Thompson, A. M., Baxter-Jones, A. D., Mirwald, R. L., & Bailey, D. A. (2003). Comparison of physical activity in male and female children: does maturation matter? Med.Sci.Sports Exerc. 35, 1684-1690.

Thompson, A. M., Campagna, P. D., Rehman, L. A., Murphy, R. J., Rasmussen, R. L., & Ness, G. W. (2005). Physical activity and body mass index in grade 3, 7, and 11 Nova Scotia students. Med Sci.Sports Exerc. 37, 1902-1908.

Tremblay, M. S., Barnes, J. D., Copeland, J. L., & Esliger, D. W. (2005). Conquering childhood inactivity: is the answer in the past? Med.Sci.Sports Exerc. 37, 1187-1194.

Treuth, M. S., Sherwood, N. E., Butte, N. F., McClanahan, B., Obarzanek, E., Zhou, A., Ayers, C., Adolph, A., Jordan, J., Jacobs, D. R., & Rochon, J. (2003). Validity and reliability of activity measures in African-American girls for GEMS. Med Sci Sports Exerc. 35, 532-539.

Troiano, R. P., Berrigan, D., Dodd, K. W., Masse, L. C., Tilert, T., & McDowell, M. (2008). Physical activity in the United States measured by accelerometer. Med Sci Sports Exerc. 40, 181-188.

Trost, S. G., Kerr, L. M., Ward, D. S., & Pate, R. R. (2001). Physical activity and determinants of physical activity in obese and non-obese children. Int.J.Obes.Relat Metab Disord. 25, 822-829.

Trost, S. G., Pate, R. R., Freedson, P. S., Sallis, J. F., & Taylor, W. C. (2000). Using objective physical activity measures with youth: how many days of monitoring are needed? Med.Sci.Sports Exerc. 32, 426-431.

Trost, S. G., Pate, R. R., Sallis, J. F., Freedson, P. S., Taylor, W. C., Dowda, M., & Sirard, J. (2002). Age and gender differences in objectively measured physical activity in youth. Med.Sci.Sports Exerc. 34, 350-355.

Tschann, J. M., Adler, N. E., Irwin, C. E., Jr., Millstein, S. G., Turner, R. A., & Kegeles, S. M. (1994). Initiation of substance use in early adolescence: the roles of pubertal timing and emotional distress. Health Psychol. 13, 326-333.

Twisk, J. W., Kemper, H. C., & Mellenbergh, G. J. (1994). Mathematical and analytical aspects of tracking. Epidemiol.Rev. 16, 165-183.

Twisk, J. W., Kemper, H. C., & Van Mechelen, W. (2002). The relationship between physical fitness and physical activity during adolescence and cardiovascular disease risk factors at adult age. The Amsterdam Growth and Health Longitudinal Study. Int.J.Sports Med. 23 Suppl 1:S8-14.

Tyron, W. W. & Williams, R. (1996). Fully proportional actigraphy: a new instrument. Behav Research Methods Instruments Comput 28, 392-403.

US Department of Health and Human Services. 2000 CDC Growth charts for the United States: Methods and development. 246. 2002. Washington, DC, Department of health and human services. Ref Type: Report

US Department of Health and Human Services. The health consequencesof smoking: a report of the Surgeon General. 2004. Washington, DC. Ref Type: Report

Vague, J. (1947). Sexual differentiation: A factor affecting the forms of obesity. Presse Med 30, 339-340.

van Jaarsveld, C. H., Fidler, J. A., Simon, A. E., & Wardle, J. (2007). Persistent impact of pubertal timing on trends in smoking, food choice, activity, and stress in adolescence. Psychosom Med. 69, 798-806.

van Lenthe, F. J., Kemper, C. G., & Van Mechelen, W. (1996a). Rapid maturation in adolescence results in greater obesity in adulthood: the Amsterdam Growth and Health Study. Am.J.Clin.Nutr. 64, 18-24.

van Lenthe, F. J., Kemper, H. C., Van Mechelen, W., Post, G. B., Twisk, J. W., Welten, D. C., & Snel, J. (1996b). Biological maturation and the distribution of subcutaneous fat from adolescence into adulthood: the Amsterdam Growth and Health Study. International Journal of Obesity and Related Metabolic Disorders 20, 121-129.

Van Mechelen, W. & Kemper, H. C. G. (1995). Habitual physical activity in longitudinal perspective. In The Amsterdam Growth Study: A Longitudinal Analysis of Health, Fitness, and Lifestyle, ed. Kemper, H. C. G., pp. 135-158. Human Kinestics, Champaign.

Van Mechelen, W., Twisk, J. W., Post, G. B., Snel, J., & Kemper, H. C. (2000). Physical activity of young people: the Amsterdam Longitudinal Growth and Health Study. Med.Sci.Sports Exerc. 32, 1610-1616.

Vanhala, M., Vanhala, P., Kumpusalo, E., Halonen, P., & Takala, J. (1998). Relation between obesity from childhood to adulthood and the metabolic syndrome: population based study. J Med Food. 317, 319.

Vanhala, M. J., Vanhala, P. T., Keinanen-Kiukaanniemi, S. M., Kumpusalo, E. A., & Takala, J. K. (1999). Relative weight gain and obesity as a child predict metabolic syndrome as an adult. Int.J Obes.Relat Metab Disord. 23, 656-659.

Vignolo, M., Naselli, A., Di Battista, E., Mostert, M., & Aicardi, G. (1988). Growth and development in simple obesity. Eur.J Pediatr. 147, 242-244.

Vihko, R. & Apter, D. (1984). Endocrine characteristics of adolescent menstrual cycles: impact of early menarche. J Steroid Biochem. 20, 231-236.

Vizmanos, B. & Marti-Henneberg, C. (2000). Puberty begins with a characteristic subcutaneous body fat mass in each sex. Eur.J Clin.Nutr. 54, 203-208.

Vu, M. B., Murrie, D., Gonzalez, V., & Jobe, J. B. (2006). Listening to girls and boys talk about girls' physical activity behaviors. Health Educ.Behav. 33, 81-96.

Wada, T., Urashima, M., & Fukumoto, T. (2007). Risk of metabolic syndrome persists twenty years after the cessation of smoking. Intern.Med. 46, 1079-1082.

Wang, Y. (2002). Is obesity associated with early sexual maturation? A comparison of the association in American boys versus girls. Pediatrics 110, 903-910.

Waylen, A. & Wolke, D. (2004). Sex 'n' drugs 'n' rock 'n' roll: the meaning and social consequences of pubertal timing. Eur.J Endocrinol. 151 Suppl 3:U151-9., U151-U159.

Weiss, R., Dziura, J., Burgert, T. S., Tamborlane, W. V., Taksali, S. E., Yeckel, C. W., Allen, K., Lopes, M., Savoye, M., Morrison, J., Sherwin, R. S., & Caprio, S. (2004). Obesity and the metabolic syndrome in children and adolescents. N.Engl.J Med. 350, 2362-2374.

Whitaker, R. C., Pepe, M. S., Wright, J. A., Seidel, K. D., & Dietz, W. H. (1998). Early adiposity rebound and the risk of adult obesity. BMC Cardiovasc Disord. 101, E5.

Whiting, S., Healey, A., Psiuk, S., Mirwald, R., Kowalski, K. C., & Bailey, D. A. (2001). Relationship between carbonated and other low nutrient dense beverages and bone mineral content of adolescents. Nutrition Research 21, 1107-1115.

Wickel, E. E. & Eisenmann, J. C. (2007). Maturity-related differences in physical activity among 13-14- year old adolescents. Pediatric Exercise Science 19, 384-392.

Williams, D. E., Prevost, A. T., Whichelow, M. J., Cox, B. D., Day, N. E., & Wareham, N. J. (2000). A cross-sectional study of dietary patterns with glucose intolerance and other features of the metabolic syndrome. Br.J Nutr. 83, 257-266.

Williams, J. M. & Dunlop, L. C. (1999). Pubertal timing and self-reported delinquency among male adolescents. Journal of Adolescence 22, 157-171.

Wilsgaard, T. & Jacobsen, B. K. (2007). Lifestyle factors and incident metabolic syndrome. The Tromso Study 1979-2001. Diabetes Res.Clin.Pract. 78, 217-224.

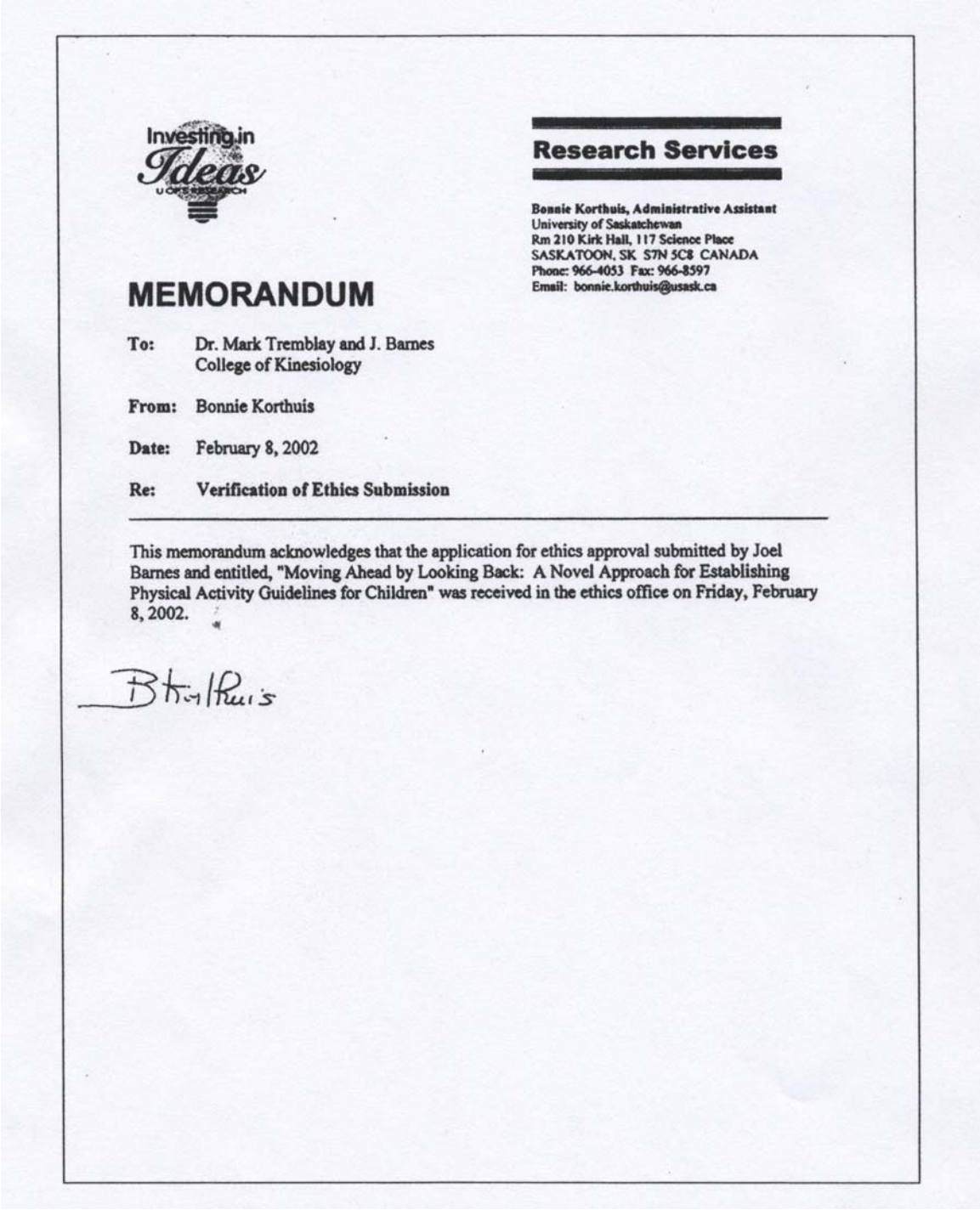
Woodgate, J., Brawley, L. R., & Weston, Z. J. (2005). Maintenance of cardiac rehabilitation exercise adherence: effects of task and self-regulatory self-efficacy. Journal of Applied Social Psychology 35, 183-222.

Wu, C. H., Heshka, S., Wang, J., Pierson, R. N., Jr., Heymsfield, S. B., Laferrere, B., Wang, Z., Albu, J. B., Pi-Sunyer, X., & Gallagher, D. (2007). Truncal fat in relation to total body fat: influences of age, sex, ethnicity and fatness. Int.J Obes.(Lond). 31, 1384-1391.

Yang, X., Telama, R., Hirvensalo, M., Mattsson, N., Viikari, J. S., & Raitakari, O. T. (2008). The longitudinal effects of physical activity history on metabolic syndrome. Med Sci Sports Exerc. 40, 1424-1431.

Zhu, S., St Onge, M. P., Heshka, S., & Heymsfield, S. B. (2004). Lifestyle behaviors associated with lower risk of having the metabolic syndrome. Metabolism. 53, 1503-1511.

APPENDIX A: Certificate of Approval for Study 1



Investing in Ideas

Research Services

Bonnie Korthuis, Administrative Assistant
University of Saskatchewan
Rm 210 Kirk Hall, 117 Science Place
SASKATOON, SK S7N 5C8 CANADA
Phone: 966-4053 Fax: 966-8597
Email: bonnie.korthuis@usask.ca

MEMORANDUM

To: Dr. Mark Tremblay and J. Barnes
College of Kinesiology

From: Bonnie Korthuis

Date: February 8, 2002

Re: **Verification of Ethics Submission**

This memorandum acknowledges that the application for ethics approval submitted by Joel Barnes and entitled, "Moving Ahead by Looking Back: A Novel Approach for Establishing Physical Activity Guidelines for Children" was received in the ethics office on Friday, February 8, 2002.

B Korthuis

UNIVERSITY OF SASKATCHEWAN
BEHAVIOURAL RESEARCH ETHICS BOARD
http://www.usask.ca/research/ethics.shtml

NAME: M. Tremblay (J. Barnes)
College of Kinesiology

BSC#: 02-344

DATE: March 8, 2002

The Behavioural Research Ethics Board has reviewed the revisions to the Application for Ethics Approval for your study "Moving Ahead by Looking Back: A Novel Approach for Establishing Physical Activity Guidelines for Children" (02-344).

1. Your study has been APPROVED.
2. Any significant changes to your proposed study should be reported to the Chair for Committee consideration in advance of its implementation.
3. The term of this approval is for 5 years.
4. This approval is valid for five years on the condition that a status report form is submitted annually to the Chair of the Committee. This certificate will automatically be invalidated if a status report form is not received within one month of the anniversary date. Please refer to the website for further instructions: http://www.usask.ca/research/ethics.shtml

I wish you a successful and informative study.

Dr. Valerie Thompson, Chair
Behavioural Research Ethics Board

VT/bk

APPENDIX B: Certificate of Approval for Study 2

UNIVERSITY OF SASKATCHEWAN

Behavioural Research Ethics Board (Beh-REB)

Certificate of Approval

PRINCIPAL INVESTIGATOR	DEPARTMENT	Beh #
Adam Baxter-Jones	Kinesiology	Beh 05-226

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT
University of Saskatchewan

SPONSORING AGENCIES
CANADIAN INSTITUTES OF HEALTH RESEARCH (CIHR)

TITLE
What prevents adolescent girls from being physically active? The effects and interactions of growth, and development

APPROVAL DATE	EXPIRY DATE
11-Oct-2005	11-Oct-2010

APPROVAL OF	APPROVED ON
Have added a physical activity questionnaire for children (PAQ-c) that includes questions on socio-economic status and ethnicity. Have extended the projected completion date.	09-Jan-2007

CERTIFICATION
The University of Saskatchewan Behavioural Research Ethics Board has reviewed the proposed revisions to your study. The revisions were found to be acceptable on ethical grounds. The principal investigator has the responsibility for any other administrative or regulatory approvals that may pertain to this research project, and for ensuring that the authorized research is carried out according to the conditions outlined in the original protocol submitted for ethics review. This Certificate of Approval is valid for the above time period provided there is no change in experimental protocol or consent process or documents.

Any significant changes to your proposed method, or your consent and recruitment procedures should be reported to the Chair for Research Ethics Board consideration in advance of its implementation.

ONGOING REVIEW REQUIREMENTS
In order to receive annual renewal, a status report must be submitted to the REB Chair for Board consideration within one month of the current expiry date each year the study remains open, and upon study completion. Please refer to the following website for further instructions: http://www.usask.ca/research/ethics_review/

John Rigby, Chair
University of Saskatchewan
Behavioural Research Ethics Board

Jan 11/07
Signature Date

Please send all correspondence to:

Ethics Office
University of Saskatchewan
Room 306 Kirk Hall, 117 Science Place
Saskatoon SK S7N 5C8
Telephone: (306) 966-2084 Fax: (306) 966-2069

APPENDIX C: Certificate of Approval for Study 3

UNIVERSITY OF SASKATCHEWAN

Biomedical Research Ethics Board (Bio-REB)

Certificate of Approval

PRINCIPAL INVESTIGATOR	DEPARTMENT	Bio #
Adam Baxter-Jones	Kinesiology	88-102

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT
College of Kinesiology
105 Gymnasium Place
Saskatoon SK S7N 5C2

SPONSORING AGENCIES
CANADIAN INSTITUTES OF HEALTH RESEARCH (CIHR)

TITLE
Protocol NHRDP #6608-126-OS: Relationship of Growth and Lifestyle to Peak Bone Mass

APPROVAL DATE	EXPIRY DATE	APPROVAL OF
20-Oct-1989	31-Oct-2007	Letter to Participant and Study Results Report (Oct-2007)

Full Board Meeting ☐

Delegated Review ☒

CERTIFICATION
The study is acceptable on scientific and ethical grounds. The principal investigator has the responsibility for any other administrative or regulatory approvals that may pertain to this research study, and for ensuring that the authorized research is carried out according to governing law. This approval is valid for the specified period provided there is no change to the approved protocol or consent process.

FIRST TIME REVIEW AND CONTINUING APPROVAL
The University of Saskatchewan Biomedical Research Ethics Board reviews above minimal studies at a full-board (face-to-face) meeting. Any research classified as minimal risk is reviewed through the delegated (subcommittee) review process. The initial Certificate of Approval includes the approval period the REB has assigned to a study. The Status Report form must be submitted within one month prior to the assigned expiry date. The researcher shall indicate to the REB any specific requirements of the sponsoring organizations (e.g. requirement for full-board review and approval) for the continuing review process deemed necessary for that project. For more information visit http://www.usask.ca/research/ethics_review/.

REB ATTESTATION
In respect to clinical trials, the University of Saskatchewan Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations and carries out its functions in a manner consistent with Good Clinical Practices. This approval and the views of this REB have been documented in writing.

Michel Desautels, Ph.D., Chair
University of Saskatchewan
Biomedical Research Ethics Board

OCT 15 2007
Signature Date

Please send all correspondence to:

Ethics Office
University of Saskatchewan
Room 305 Kirk Hall, 117 Science Place
Saskatoon SK S7N 5C8
Telephone: (306) 966-4053 Fax: (306) 966-2069

APPENDIX D: Participant Assent Form for Study 2

You are invited to participate in the research study titled **What prevents youth from being physically active?** We want to learn what makes youth physically active during later childhood and adolescence. This is not part of your regular class work and is an optional activity.

The testing will take approximately 30 minutes and will take place at school. You will be asked your age and grade at school. You will also have your height, sitting height, weight, skinfold thickness and waist girth measured. You will be asked if you have had your period. If you have you will be asked to recall when it happened

Physical activity monitor
After the testing session some of you will be loaned an activity monitor. The activity monitor tells the number of minutes spent in light, moderate, or strenuous exercise. The activity monitor is to be worn each day for 7 days following the testing session. You will also be given a log to record the time each day that the monitor was attached and removed for the purpose of calculating activity time and sleeping time. You can return the monitor to us at school after the 7 days.

Questionnaires
You will be asked to fill out a questionnaire that assess factors affecting physical activity.

Risks and Benefits
There are no risks to participating in this study. You have the right to refuse to answer any question. A donation will be given to your school's physical education fund as a thank you for your participation.

Keeping it Secret
Your test results will be kept secret and will not be shown to other people, unless you say so. The numbers for the entire group will be used in reports but your name will not be used. The data may also be published and presented at conferences; however, your identity will be kept confidential.

Right to Ask Questions and to Quit the Study
You can decide if you want to be in this study and you are free to get out of the study at any time and we will not be upset. Before you sign this form, do you have questions?

Consent
By signing this paper:
I understand the study and the consent form
I agree to be in the study

_____ _____
Your signature Date

APPENDIX E: Parent and Guardian Consent Form for Study 2

This Parent/Guardian Authorization form is being sent to you to obtain your permission in allowing your child to participate in the research study titled: **What prevents youth from being physically active?** This is an optional activity and is not part of the school curriculum. Please read this form carefully and feel free to ask any questions. The testing will be overseen by Dr. Adam Baxter-Jones and Dr. Louise Humbert. For more information contact Dr. Humbert at (306) 966-1070. Your participation in this project is greatly appreciated.

Purpose of the study
During childhood and adolescence physical activity levels dramatically decline. This study aims to explore some of the factors that may influence youth's participation in physical activity.

Procedures
The testing will take approximately 30 minutes and will take place at a time convenient to the girls and will not disrupt school. Students will be asked their age and grade at school. Height, sitting height, weight, skinfold thickness and waist girth will be measured. The girls will be asked if they have had their menstrual period. If they have they will be asked to recall when, to the month and year, their first period occurred

Physical activity monitor
After the testing session the female students only will be loaned an activity monitor. The activity monitor tells the number of minutes spent in light, moderate, or strenuous exercise. Your child (if selected) is asked to wear the activity monitor each day for 7 days following the testing session. Your child will also be given a log to record the time each day that the monitor was attached and removed for the purpose of calculating activity time and sleeping time. Your child can return the monitor to us at school after the 7 days.

Questionnaires
Your child will be asked to fill out a questionnaire that assess factors affecting physical activity.

Potential Risks
You child will not be subjected to any physical or psychological risk. You or your child may withdraw from the study for any reason, at any time, without penalty of any sort.

Potential Benefits

Participation of your child in this study may help researchers to better understand why youth become less active during later childhood and adolescence. Information from this study has the potential to tailor physical activity interventions in the future. A monetary donation will be given to the school's physical education fund as a thank you for taking part in the research.

Storage of Data

All research material will be securely stored in the office of Dr. Baxter-Jones, at the University of Saskatchewan, for a minimum of five years post publication of the findings.

Confidentiality

The data from this study will be written as a Doctoral thesis. The data may also be published and presented at conferences; however, your child's identity will be kept confidential.

Right to Withdraw

You child's participation is voluntary and he/she may withdraw from the study for any reason, at any time, without penalty of any sort. Should you decide to withdraw from the project, your child's information will be automatically deleted (erased) from the study and destroyed.

Questions

If you have any questions concerning the study, please feel free to ask the research assistants at any time; you are also free to contact the researcher (call collect) at the number provided above if you have any questions at a later date. This study has been approved by Saskatoon Public Schools and by the school's administration. This study has been also been approved on ethical grounds by the University of Saskatchewan Research Ethics board on Oct 1st, 2006. Any questions regarding your child's rights as a participant may be addressed to that committee on 966-2084. Out of town participants may call collect. You may contact the researcher to find out the results of the study and a copy of the published manuscript can also be requested.

Consent Form

My signature on this sheet indicates that I have received information regarding the nature of the study, its purpose, and procedures.

I will allow my child _____, to participate in the study entitled **What prevents youth from being physically active?** My signature indicates that I understand the following:

1. I received information regarding the nature of the study, its purpose, and procedures. This research project was reviewed and approved on ethical grounds by the University of Saskatchewan Advisory Committee on Ethics in Behavioral Science Research
2. Participation is totally voluntary; the participant (my child) has the right not to answer any or all of the questions, refuse any or all of the measurements and can withdraw from the study at any time without any fear of penalty.
3. If my child withdraws from the study then his/her data will be deleted.
4. There are no risks of psychological or physiological harm.
5. All individual data that is provided will remain confidential from sources outside of the study.
6. I will receive a summary of the project, upon request, following completion of the project.
7. I have read and understood the information provided in the cover letter, and have received a copy of that letter and the consent form for my records.

I _____ have read the above statements regarding the study and

understand the conditions of _____(my child's) participation in this study.

Parent/Guardian's Signature_____ Date:_____

Researcher's Signature _____ Date:_____

Lauren Sherar, MSc (306) 966-1123
Louise Humbert, PhD (306) 966- 1073
College of Kinesiology
University of Saskatchewan

APPENDIX F: Consent Form for Study 3

Investigators:
Dr. Adam Baxter-Jones, Dr. Bob Faulkner, Dr. Kent Kowalski, Dr. Don Bailey (kinesiology); Dr. Susan Whiting (Pharmacy and Nutrition) and Edyta Dudzic (Medical Imaging)

Purpose and Benefits of the Study

The maximum amount of bone mineral (peak bone mass) deposited into the skeleton is thought to occur by age 25-30; however, there is yet no longitudinal data to confirm this hypothesis. Peak bone mass is a major factor in determining lifetimes risk of osteoporosis; thus, accurate determination of when peak bone mass is achieved and of factors that affect its attainment are important in developing intervention strategies to optimize peak bone mass. The Bone Mineral Accrual Study is the only study internationally to date to have monitored yearly bone mineral accrual through childhood, adolescence and early adulthood. The continuation of testing into the adult years will answer the question of when peak bone mass occurs, and identify factors during the growing years that affect peak bone mass.

In addition to bone health of interest is the how childhood lifestyle factors relate to adult health. An indication of adult health can be obtained through assessing risk factors for diabetes (i.e. blood glucose) and cardiovascular disease (i.e. triglycerides and cholesterol).

Procedures
Procedures will be the same as previous testing. However, this year you have the option of taking part in another part of the study, which will involve a blood and urine test. Assessment of bone mineral density, nutrition, physical activity and lifestyle questionnaires and anthropometry will take place at the Physical Activity Complex. Assessment of blood and urine will take place at the Royal University Hospital. Certified technicians will administer all tests. The following procedures will be performed:

a) Assessment of Bone Mineral Density: Your total body, lumbar spine and proximal femur bone mineral density will be evaluated using dual-energy x-ray absorptiometry (DXA). This procedure is routinely used in clinical medicine. The radiation exposure of 1.24 millirems(mrems) is minimal – representing approximately the same radiation you would receive on a return air flight from Saskatoon to Toronto (1.62 mrems). The average annual background radiation in Saskatchewan due to natural sources is approximately 300 mrems per year. A typical chest x-ray is about 5 mrems of exposure. All bone density measures will be conducted and stored in the Department of Medical Imaging at the RUH and will be administered by certified technologists.
b) Nutrition, physical activity and lifestyle questionnaires. These are standard questionnaires that will ask you to identify your physical activity patterns and

typical nutritional intake. You will also be asked about other lifestyle behaviors such as tobacco and alcohol use.

c) Anthropometric assessment: Height, weight, waist circumference and skinfold thickness will be measured.
d) Blood assessment and urine: Approximately 5 mL of blood will be drawn from a catheter that is inserted into a vein in your arm and you will be asked to provide a urine sample. The purpose of the blood and urine collection is to measure indicators of health (i.e. glucose, triglycerides and cholesterol). There may be some discomfort during the drawing of blood. Although unlikely, there is a risk of bruising and infection with the drawing of blood but care will be taken to minimize these risks.
e) Resting blood pressure will be assessed.

Rights and Welfare of the Individual

It is understood that you will be free to withdraw from any or all of the study at any time without penalty. Your identity will remain confidential, only those directly involved in the study (that is investigators, project assistants) will have access to your records and results. All individual results will remain strictly confidential.

Please be assured that you may ask questions at any time. We will be glad to discuss your results with you when they become available and we welcome your comments and suggestions.

If you have any questions please contact our office at:

Dr. Adam Baxter-Jones (phone: _____email: _____)
Dr. Bob Faulkner (phone: _____email: _____)

Participant's Statement:

I, _____, understand the purpose and procedures of this study, as
(please print name)
I have read or have had described to me, and I voluntarily agree to participate. I understand that at any time during the study I will be free to withdraw without penalty. I understand the contents of the consent form, the proposed procedures and possible risks. I have had the opportunity to ask questions and have received satisfactory answers to all inquiries regarding the study.

I hereby acknowledge that the contents of the consent have been explained to me and that I have received a copy of the consent for my own records. Any questions regarding your rights as a participant may be addressed to the University of Saskatchewan Biomedical Research Ethics Board through the office of Research services call collect on 966-4053. This research was approved on May 2nd, 2006.

_____ _____
Signature of Participant Date

_____ _____
Signature of Witness Date

_____ _____
Signature of Investigator Date

APPENDIX G: Accelerometry Methodology

The protocol for collection and reduction of accelerometer data were similar between study 1 and study 2. However, the following methods section provides detail specifically on study 2, as study 1 used pre-collected data.

Reliability Check

All activity monitors underwent a reliability check before use in the study. A reliability check is important to ascertain whether intra- and inter accelerometer variability is within acceptable limits and to identify malfunctioning units. All reliability checks were carried out using a hydraulic shaker plate (figure 16). This mechanical set-up allows one to precisely control the magnitude of the acceleration as well as the frequency of the oscillations, thereby ascertaining the variability solely attributed to the accelerometers (Esliger & Tremblay, 2006).

Six different conditions, within the amplitude range of the shaker plate (approx. 6.5cm), of acceleration and frequency were chosen to produce a range of physiologically relevant accelerometer counts. The conditions can be described by the equation: acceleration ($m \cdot s^{-2}$)= (amplitude (m) · $frequency^2$ ($rad \cdot s^{-1}$).

Forty six Acticals were initialized to collect data using 15 second epochs at a synchronized time. All monitors were positioned vertically in a test jig and secured. The jig was then mounted to the surface of the shaker plate (surface area approximately 1500 cm^2) using industrial strength hook and loop material. The hydraulic shaker plate accelerated the monitors simultaneously in the vertical plane. All frequency/acceleration

conditions were carried out in random order and began at the turn of a new minute. After approximately 60 minutes of data collection (12-min warm-up + (six conditions x 7 minute per condition) + 6 x 1-min transitions between conditions) the accelerometers were removed from the shaker plate, downloaded, and analyzed using a custom analysis spreadsheet.

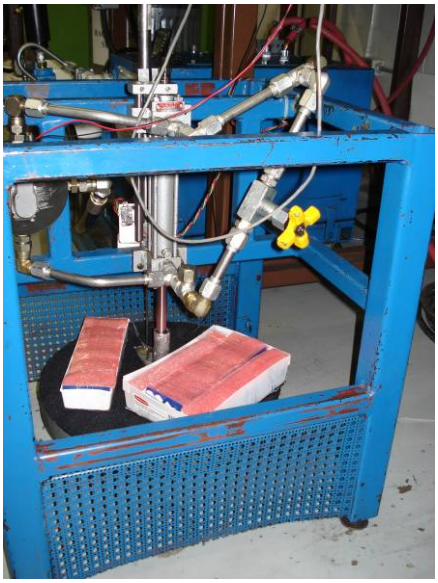


Figure 14: Mechanical set-up for reliability testing

To determine the variability within a given accelerometer (intra-instrument reliability), standard deviation (SD), standard error of the measurement (SEM) and coefficient of variation (CV_{intra}) were calculated from the replicate minutes (minutes 1-5) within each condition. This minute-by-minute variability characterizes the accelerometer‘s ability to consistently measure the given condition rendered by the shaker table. To determine the variability between units (inter-instrument reliability) SD, SEM and coefficient of variation (CV_{inter}) were calculated for each of the six testing conditions (table 17).

Ten monitors were deemed unacceptable for use; 1 malfunction and 9 fell outside of ±10% difference from the mean.

Table 17: The intra-and inter- instrument reliability for the six shaking conditions

Conditions				Intra-Instrument Reliability			Inter-Instrument Reliability		
Force (g)	Acceleration ($m \cdot s^{-2}$)	Frequency (Hz)	Counts	SD	SEM	CV	SD	SEM	CV
0.50	4.90	1.5	2348	11	5	0.48	154	23	6.55
0.50	4.90	2.5	2327	10	4	0.41	116	17	4.99
0.50	4.90	2.0	2404	70	31	2.94	132	20	5.48
0.75	7.36	2.0	4355	103	46	2.38	205	31	4.70
1.00	9.81	2.5	5933	13	6	0.23	246	37	4.15
1.25	12.26	2.5	6979	13	6	0.19	276	41	3.95
Overall Mean			**4058**	**37**	**16**	**1.10**	**188**	**28**	**4.97**

Sampling interval (Epochs)

The signal from an accelerometer is integrated over a given time interval, then summed and stored. The time interval is known as an epoch. An epoch of 15 seconds was chosen to capture the short bursts of rapidly changing activity which is often characteristic of children (Bailey *et al.*, 1995).

Number of days of monitoring

Participants were asked to wear the activity monitors for a full 7 days. Seven days of monitoring has been shown to provide an estimate of activity that is reliable for children and adolescents (Trost *et al.*, 2000) and allows for the consideration of a weekend and weekdays. Studies have shown that children and adolescents activity differs between weekend and weekdays (Metcalf *et al.*, 2002; Rowlands *et al.*, 1999; Treuth *et*

al., 2003; Trost *et al.*, 2000). Therefore, balancing the number of weekdays and weekend days measured may be important for ensuring an accurate estimate of the pattern of behaviour for a recorded week. All files were retained for analysis if it included at least five full days of data, with at least one of those be a weekend day (as represented in figure 15). A non-monitored weekday was replaced by the mean data of the other four weekdays. The monitored weekend day was used to represent the non-monitored weekend day (Esliger *et al.*, 2005). Of the 182 accelerometer files used in study 2, 51 (28%) had at least one unmonitored day that was imputed.

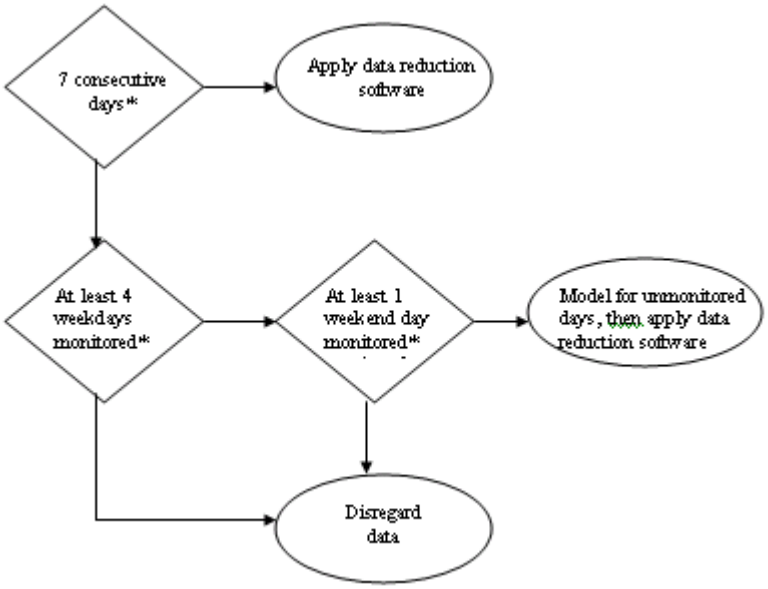


Figure 15: Flow chart illustrating the criteria for accepting accelerometer data. Adapted from (Esliger et al., 2005) * Each monitored day includes at least 10 hours of wear time.

Data Collection

Explicit instructions on where, when and how to wear accelerometers were provided to ensure quality data was collected. Furthermore, all participants were asked to

fill out a log sheet to fill out when the monitor was put on in the morning and when it was taken off in the evening. This was conducted for the purpose of determining wear time.

A delayed start time (i.e. 12am on the data following deployment of monitors) was used to reduce subject reactivity and to ensure that the same start time was used for all participants regardless of the time of day the activity monitors were deployed.

APPENDIX H: Questionnaires and Participant Information Sheets for Study 2

Physical Activity Monitor Information

Please:

- **Wear the activity monitor for 7 full days**
- **Remove the monitor when you go to bed and put it back on when you get up**
- **During the 7 days you are asked to record when you put the monitor on in the morning and when you take it off in the evening**

- **(If you take the monitor off for any reason during the day) record the length of time it was off and the activity you were doing while it was off.**
- **Do not alter your normal physical activity behaviour while wearing the activity monitor – we are interested in your normal level of activity**

- **Wear the monitor for all water activities (e.g. showering, swimming)**
- **Keep the monitor fastened on the belt to reduce the chance of loosing it, as these are expensive pieces of research equipment**

We will return to your school on Tuesday April 4th to collect the activity monitors. Please wear the monitor until we meet with you.

If you have questions about the monitor please call Lauren at _____ (daytime) and _____ (evenings)

Activity Monitor Log Sheet

NAME _____ **AGE**_____

MRS - ___ ___ ___ **SERIAL NUMBER: C 8 4**_____

	Friday	Saturday	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
	03-Oct-08	04-Oct-08	05-Oct-08	06-Oct-08	07-Oct-08	08-Oct-08	09-Oct-08	10-Oct-08	11-Oct-08
On at wake up (time you put monitor on for the day)									
Off at bedtime (time you took off monitor before bed)									Bring Log Sheet to school

The 7 full days of monitoring are finished on Saturday **11-Oct-08** . We will come and collect the monitors sometime Saturday
Please wear the monitor and bring the log sheet with you to school.

NOTES

1. The activity monitor is to be worn securely on the RIGHT hip using the belt provided.
2. The monitor can be worn over or under your clothing (your choice).
3. Although the monitors are fairly durable, care should be taken when wearing them as they are quite expensive.
4. Please wear your monitor everyday, even if you've stayed home from school (e.g., due to illness).

NOTE: If you have to take off the monitor for any reason please record the reason why below.

Example Activity Monitor Log Sheet

MRS - # # # **SERIAL NUMBER # # # # #**

	Friday	Saturday	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
	03-Oct-08	04-Oct-08	05-Oct-08	06-Oct-08	07-Oct-08	08-Oct-08	09-Oct-08	10-Oct-08	11-Oct-08
On at wake up (time you first put the monitor on for the day)	1:14 PM	7:05 AM	7:22 AM	7:09 AM	7:15 AM	8:02 AM	7:19 AM	8:30 AM	7:30 AM
Off at bedtime (time you take off the monitor before bed)	9:08 PM	9:13 PM	9:02 PM	10:33 PM	8:54 PM	10:00 PM	11:07 PM	9:07 PM	Bring Log Sheet to school

NOTES

1. The activity monitor is to be worn securely on the RIGHT hip using the belt provided.
2. The monitor can be worn over or under your clothing (your choice).
3. Although the monitors are fairly durable, care should be taken when wearing them as they are quite expensive.
4. Please wear your monitor everyday, even if you've stayed home from school (e.g., due to illness).

NOTE: If you have to take off the monitor for any reason please record the reason why below.

Physical Activity Survey

1) What is your first and last name::_____
First name Last name

2) How old are you? _____

3) What is your birth date?

Example: January / 1 / 2007

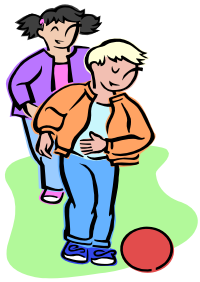
Now, you answer: _____ /_____/_____
Month Day Year

4) What grade are you in? _____

5) Who is your teacher? _____

6) Are you (circle your answer)? Female Male

We are trying to find out about your level of physical activity from ***the last 7 days*** (in the last week). This includes sports or dance that make you sweat or make your legs feel tired, or games that make you breathe hard, like tag, skipping, running, climbing, and others.



Remember:

1. There are no right and wrong answers — this is not a test.
2. Please answer all the questions as honestly and accurately as you can — this is very important.

---

1. <u>Physical activity in your spare time:</u> Have you done any of the following activities in the past 7 days (last week)? If yes, how many times? (Mark only one circle per row.)

	No	1-2	3-4	5-6	7 times or more
Skipping	❍	❍	❍	❍	❍
Rowing/canoeing	❍	❍	❍	❍	❍
In-line skating	❍	❍	❍	❍	❍
Tag	❍	❍	❍	❍	❍
Walking for exercise	❍	❍	❍	❍	❍
Bicycling	❍	❍	❍	❍	❍
Jogging or running	❍	❍	❍	❍	❍
Aerobics	❍	❍	❍	❍	❍
Swimming	❍	❍	❍	❍	❍
Baseball, softball	❍	❍	❍	❍	❍
Dance	❍	❍	❍	❍	❍
Football	❍	❍	❍	❍	❍
Badminton	❍	❍	❍	❍	❍
Skateboarding	❍	❍	❍	❍	❍
Soccer	❍	❍	❍	❍	❍
Street hockey	❍	❍	❍	❍	❍
Volleyball	❍	❍	❍	❍	❍
Floor hockey	❍	❍	❍	❍	❍
Basketball	❍	❍	❍	❍	❍
Ice skating	❍	❍	❍	❍	❍
Cross-country skiing	❍	❍	❍	❍	❍
Ice hockey/ringette	❍	❍	❍	❍	❍
Other:					
_____....	❍	❍	❍	❍	❍
_____....	❍	❍	❍	❍	❍

2. In the last 7 days, <u>during your gym classes</u>, how often were you very active (playing hard, running, jumping, throwing)? (Check one only)

I don't do gym..... ❍
Hardly ever ..... ❍
Sometimes ..... ❍
Quite often ..... ❍
Always ..... ❍

3. In the last 7 days, what did you do most of the time *<u>at recess</u>*? (Check one only)

Sat down (talking, reading, doing schoolwork)… ❍
Stood around or walked around ..... ❍
Ran or played a little bit ..... ❍
Ran around and played quite a bit ..... ❍
Ran and played hard most of the time ..... ❍

4. In the last 7 days, what did you normally do *<u>at lunch</u>* (besides eating lunch)? (Check one only)

Sat down (talking, reading, doing schoolwork)……. ❍
Stood around or walked around ..... ❍
Ran or played a little bit ..... ❍
Ran around and played quite a bit ..... ❍
Ran and played hard most of the time ..... ❍

5. In the last 7 days, on how many days *right after school*, did you do sports, dance, or play games in which you were very active? (Check one only.)

None ..... ❍
1 time last week ..... ❍
2 or 3 times last week ..... ❍
4 times last week ..... ❍
5 times last week ..... ❍

6. In the last 7 days, on how many *evenings* did you do sports, dance, or play games in which you were very active? (Check one only.)

None ..... ❍
1 time last week ..... ❍
2 or 3 times last week ..... ❍
4 or 5 last week ..... ❍
6 or 7 times last week ..... ❍

7. *On the last weekend*, how many times did you do sports, dance, or play games in which you were very active? (Check one only.)

None ..... ❍
1 time ..... ❍
2 — 3 times ..... ❍
4 — 5 times ..... ❍
6 or more times ..... ❍

8. <u>Which *one*</u> of the following <u>describes you best</u> for the last 7 days?

Read *all five* statements before deciding on the *one* answer that describes you.

A. All or most of my free time was spent doing things that involve little physical effort ..... ❍

B. I sometimes (1 — 2 times last week) did physical things in my free time (e.g. played sports, went running, swimming, bike riding, did aerobics) ..... ❍

C. I often (3 — 4 times last week) did physical things in my free time ..... ❍

D. I quite often (5 — 6 times last week) did physical things in my free time ..... ❍

E. I very often (7 or more times last week) did physical things in my free time ... ❍

9. Mark how often you did physical activity (like playing sports, games, doing dance, or any other physical activity) for each day last week.

	None	Little bit	Medium	Often	Very often
Monday	❍	❍	❍	❍	❍
Tuesday	❍	❍	❍	❍	❍
Wednesday	❍	❍	❍	❍	❍
Thursday	❍	❍	❍	❍	❍
Friday	❍	❍	❍	❍	❍
Saturday	❍	❍	❍	❍	❍
Sunday	❍	❍	❍	❍	❍

1. <u>Please think about the last 7 days</u>. Are there any physical activities that you would have liked to do but <u>didn't end up doing</u> in the last 7 days?

☐ YES (go to question 2 below)

☐ NO (raise your hand)

2. If you said YES, <u>write down</u> what physical activities you would have liked to do but <u>didn't end up doing</u> in the last 7 days?

_____

_____

_____

_____

_____

_____

Now, think about what stopped you from doing the physical activities you listed on the green piece of paper.

1) <u>Did someone say or do anything</u> that stopped you from doing the physical activities that you listed on the green piece of paper?

☐ YES (answer the questions below)

☐ NO (stop)

1a) Write down <u>what someone said or did</u> that stopped you? Please write a detailed answer.

1b) <u>WHO</u> said or did this to you?_____

2a) If there is another thing that <u>someone said or did</u> that stopped you, please write it down here in as much detail as you can.

2b) <u>WHO</u> said or did this to you? _____

3a) If there is another thing that <u>someone said or did</u> that stopped you, please write it down here – in as much detail as you can.

3b) <u>WHO</u> said or did this to you? _____

Think about the physical activities that you would have liked to do but didn't do in the last 7 days.

1) <u>Did anything about your school</u> stop you from doing the physical activities that you listed on the green piece of paper?

☐ YES (answer the questions below)

☐ NO (stop)

1. Write down the thing <u>about your school</u> that stopped you? Please write a detailed answer.
2. If there is another thing <u>about your school</u> that stopped you, please write it down here – in as much detail as you can.
3. If there is another thing <u>about your school</u> that stopped you, please write it down here – in as much detail as you can.

Think about the physical activities that you would have liked to do but didn't do in the last 7 days.

1) Did anything about the neighbourhood where you live stop you from doing the physical activities that you listed on the green piece of paper?

☐ YES (answer the questions below)

☐ NO (stop)

1. Write down the thing about your neighbourhood that stopped you? Please write a detailed answer.

2. If there is another thing about your neighbourhood that stopped you, please write it down here – in as much detail as you can.

3. If there is another thing about your neighbourhood that stopped you, please write it down here – in as much detail as you can.

Think about the physical activities that you would have liked to do but didn't do in the last 7 days.

1. Is there anything <u>about you</u> that stopped you from doing the physical activities?

☐ YES (answer the questions below)

☐ NO (stop)

1) Write down <u>1 thing about you</u> that stopped you. Remember to write it down in as much detail as you can.
2) If there is <u>another thing about you</u> that stopped you, write it down here in as much detail as you can.
3) If there is <u>another thing about you</u> that stopped you, please write it down here – in as much detail as you can.

Think about the physical activities that you would have liked to do but didn't do in the last 7 days.

1. Is there <u>anything else</u> that stopped you from doing the physical activities <u>that you didn't write down yet</u>?

☐ YES (answer the questions below)

☐ NO (stop)

1) Write down <u>one thing</u> that stopped you. Remember to write it down in as much detail as you can.
2) If there is <u>another thing</u> that stopped you, write it down here in as much detail as you can.
3) If there is <u>another thing</u> that stopped you, please write it down here – in as much detail as you can.

QUESTIONS ABOUT YOU

1. What is your street address (include, if you know, the postal code)?

_____

_____

_____

2a. Have you lived at this address in the last five years? Yes No

2b. If NO, write down the number of different homes that have you lived at in the last five years? _____

3. People living in Canada come from many different cultural and racial backgrounds. Are your parents? (please circle your answer)

A. Both Caucasian (White)
B. Both Aboriginal Canadians (First Nations member, Métis, or Inuit)
C. Both from an Asian/African/Latin American or Middle Eastern background
D. Mixed cultural/ethnic background

4. What is the highest level of schooling your <u>father</u> completed? (please circle your answer)

A. Less than high school
B. Finished high school
C. Trade school (for example, mechanic, technician, journeyman, librarian)
D. Some university
E. Finished university
F. Don't Know or Doesn't Apply
G. Other

5. What is the highest level of schooling your <u>mother</u> completed? (please circle your answer)

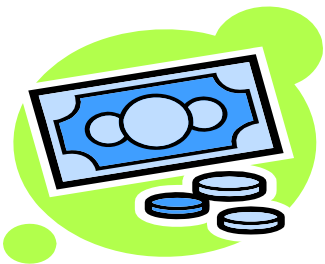
A. Less than high school
B. Finished high school
C. Trade school (for example, mechanic, technician, journeyman, librarian)
D. Some university
E. Finished university
F. Don't Know or Doesn't Apply
G. Other

6. Tell us about your health -- Compared to others your age, how would you rate your health? (please circle your answer)

A. Excellent
B. Very good
C. Good
D. Fair
E. Poor

7. In comparison with other people your own age, would you describe your family's financial situation as? (please circle your answer)

A. Wealthy
B. Well-off
C. Comfortable
D. Adequate
E. Difficult
F. Poor
G. Don't Know



8a. Do you have a job that pays you money? (circle your answer) **Yes** **No**

8b. If yes, <u>about how many hours each week</u> do you work? _____ hours

What is your job? _____

_____

9a. Do you have to do work around the home, like babysitting or chores, that doesn't pay you money? (circle your answer) **Yes** **No**

9b. If yes, <u>about how many hours each week</u> do you work? _____ hours

What do you do? _____

_____

APPENDIX I: Questionnaires and Participant Information Sheets for Study 3

University of Saskatchewan
Bone Mineral Accrual Study
College of Kinesiology

Date: _____ Scheduled Appointment Time _____

Name_____ Birthdate: _____

Health Number (1991): _____

N.M. #:_____ Subject #: ___ SEQ#: 30

Parents Address:_____ City: Saskatoon

Province: SK Postal Code: _____

Telephone #: _____ Is this correct? ❑ Yes ❑ No

Parents New Address: _____

City: _____ Province: _____ Postal Code: _____

Telephone #: _____

Your Current Address: _____ City: Saskatoon

Province: SK Postal Code: _____

Telephone #: (home) _____ (work) _____

Cell Phone #: _____ E-mail address_____

Is this correct? ❑ Yes ❑ No

Your New Current Address: _____

City: _____ Province: _____ Postal Code: _____

Telephone #: (home) _____ (work) _____

Cell Phone #: _____ E-mail address: _____

Martial Status: _____ Single Married: _____ (Name _____)

Name: _____ Date: _____

Subject #: _____

Reported items:

*Father's Height _____ cm

*Mother's Height _____ cm

*Birth Weight _____ g

When you were a baby were you mainly **(please check circle that applies)**:
*Breast-Fed ❍
*Formula-Fed ❍

*If breast Fed, how many months were you exclusively breast fed? _____

*If you do not know these items, who can we contact for this information?

Name: _____ Telephone #: _____

Office use only:

Anthropometry:

Height:	_____cm	Sit Height:	_____cm
	_____cm		_____cm
	_____cm		_____cm
Weight:	_____ kg	Waist Girth:	_____cm
	_____ kg		_____cm
	_____ kg		_____cm
Triceps SF:	_____mm	Biceps SF:	_____mm
	_____mm		_____mm
	_____mm		_____mm
Subscapular SF:	_____mm	Iliac SF:	_____mm
	_____mm		_____mm
	_____mm		_____mm
Supraspinale SF:	_____mm	Medial calf SF:	_____mm
	_____mm		_____mm
	_____mm		_____mm
Elbow Width:	_____cm	Femur Width :	_____cm
	_____cm		_____cm
	_____cm		_____cm
Biceps Girth:	_____cm	Calf Girth :	_____cm
	_____cm		_____cm
	_____cm		_____cm

Office use only:

Please check the following conditions before taking a blood pressure reading:

Participant has:

a) been in a warm environment for 1 hour ❑ Yes

b) had no caffeine or cigarettes for 2 hours ❑ Yes

c) had no alcohol in the last 8 hours ❑ Yes

d) no heavy physical activity in the last 2 hours ❑ Yes

e) bowel/bladder is not full prior to reading ❑ Yes

Diastolic Blood Pressure (DBP) _____ mmHG

Systolic Blood Pressure (SBP) _____ mmHG

Resting Heart Rate (HR) _____ beats per minute

Female Only Questionnaire	_____	Health Questionnaire	_____
Consent Form	_____	Scan Completion	_____
Demographics Form	_____	Copy of Consent Form	_____
Anthropometry	_____	Parking	_____
Blood Pressure	_____	Taxi	_____
Physical Activity Questionnaire	_____	Sears Gift Certificate	_____
24 hr Recall Questionnaire	_____	HPC Fitness	_____
Food Frequency Questionnaire	_____		

Bone Mineral Accrual Study
Females Only Questionnaire

1. How old were you when you started to have menstrual cycles?_____ years old

   Did it occur in:
   - ❑ Spring
   - ❑ Summer
   - ❑ Fall
   - ❑ Winter

2. Are you currently using oral contraceptives?
   - ❑ No
   - ❑ Yes

   If yes, for how long have you used them? _____ Years _____ Months

   What is the brand name of the oral contraceptives that you use? _____

Legally, you cannot be scanned if you are pregnant.

3. Are you pregnant?
   - ❑ No - if No, go to question 4
   - ❑ Yes - if Yes, go to question 5 and complete only questionnaire material
   - ❑ I don't know - go to question 4

4. What is the date of the first day of your last period? _____

5. How many children have you given birth to? _____ If none, go to next page

   List their birthdates:

   Child 1: _____ Did you breastfeed? ❑ No ❑ Yes
   If yes, how many months? _____

   Child 2: _____ Did you breastfeed? ❑ No ❑ Yes
   If yes, how many months? _____

   Child 3: _____ Did you breastfeed? ❑ No ❑ Yes
   If yes, how many months? _____

   Child 4: _____ Did you breastfeed? ❑ No ❑ Yes
   If yes, how many months? _____

Health and Lifestyle Questionnaire
Bone Mineral Accrual Study

General Health and Lifestyle

1. In general, would you say your health is:
   - ❑ Excellent
   - ❑ Very good
   - ❑ Good
   - ❑ Fair
   - ❑ Poor

2. On a scale from 1-5, **compared to other people your age and sex, would you say you are**:

1	2	3	4	5
Much less active				Much more active

3. In the past 12 months how often have you drank some kind of alcohol beverage?
   - ❑ Daily or almost every day
   - ❑ 3 or 4 times a week
   - ❑ Once or twice a week
   - ❑ Once or twice a month
   - ❑ Less than once a month
   - ❑ Never
   - ❑ I don't know.

4. How often do you or did you drink any type of milk (including milk on cereal)? Do not include milk added to coffee or tea.

	Current	Teenage (13-17 y)	Childhood (5-12 y)
Never	( )	( )	( )
Less than once per week	( )	( )	( )
Once per week	( )	( )	( )
Less than once per day but more than once per week	( )	( )	( )
Once per day	( )	( )	( )
More than once per day	( )	( )	( )
More than 3 times per day	( )	( )	( )
Don't know	( )	( )	( )

5. Have you EVER smoked cigarettes regularly; regularly means more than 20 packages in a lifetime or more than 1 cigarette a day for a year?
   - ❑ No
   - ❑ Yes

   If No, go to Question 12.

6. How old were you when you started smoking regularly?
   _____ years old

7. Do you still smoke?
   - ❑ No
   - ❑ Yes

   If YES, go to Question 9

8. If you stopped smoking cigarettes completely, how old were you when you stopped?_____ years old

9. For the entire time that you smoked, on average how many cigarettes did you smoke per day?_____ cigarettes

10. Between the years that you started and last smoked cigarettes, did you ever quit smoking for a year or longer?
    - ❑ No
    - ❑ Yes

    If Yes, go to Question 11; if No, go to Question 12.

11. You may have started and stopped several times. How many years total did you quit?_____ years

12. Are you regularly exposed to someone else's smoke at work (school) or home?
    - ❑ No
    - ❑ Yes

Bone Related History

13. Have you ever had any problems with your bones such as fractures?
    - ❑ No
    - ❑ Yes

    If yes, how many fractures have you had? _____

    Please list the type of fracture and approximate date.

Type	Date
_____	_____
_____	_____
_____	_____

14. Is there a history of osteoporosis in your family?
    - ❑ No
    - ❑ Yes

    If yes, indicate who was affected? _____

15. Is there a history of wrist, hip or spine fractures in your family?
    - ❑ No
    - ❑ Yes

    If yes, indicate who was affected? _____

16. Are you currently taking any medications?
    - ❑ No
    - ❑ Yes

    If yes, what medication(s) are you taking and what are they for?

Medications	For
_____	_____
_____	_____
_____	_____

17. Have you ever been treated for any of the following conditions?

Food allergies	_____ No	_____ Yes
Other allergies	_____ No	_____ Yes
Back pain	_____ No	_____ Yes
Scoliosis	_____ No	_____ Yes
Epilepsy	_____ No	_____ Yes
Osteoporosis	_____ No	_____ Yes
Rheumatoid arthritis	_____ No	_____ Yes
Diabetes	_____ No	_____ Yes
Excess urinary calcium	_____ No	_____ Yes
Asthma	_____ No	_____ Yes
Kidney disease	_____ No	_____ Yes
Chronic liver disease	_____ No	_____ Yes
Gastrointestinal disease	_____ No	_____ Yes
Muscular dystrophy	_____ No	_____ Yes
Osteoarthritis	_____ No	_____ Yes
Anemia	_____ No	_____ Yes
Malabsorption	_____ No	_____ Yes
Excess blood calcium	_____ No	_____ Yes
Hyperparathyroidism *	_____ No	_____ Yes
Hyperthyroidism *	_____ No	_____ Yes
Hypoparathyroidism **	_____ No	_____ Yes
Hypothyroidism **	_____ No	_____ Yes

List other conditions: _____

_____

* hyper = excess

** hypo = deficiency

Bone Mineral Accrual Study
Physical Activity Questionnaire (Adults)

We are trying to find out about your level of physical activity from ***the last 7 days*** (in the last week). This includes activities that make you sweat, make your legs feel tired, or make you breathe hard, such as team sports, running, strenuous occupational activities, and others.

Remember:
3. There are no right and wrong answers — this is not a test.
4. Please answer all the questions as honestly and accurately as you can — this is very important.

---

1. Physical activity in your spare time: Have you done any of the following activities **in the past 7 days** (last week)? If yes, how many times? (Mark only one circle per row.)

	No	1-2	3-4	5-6	7 times or more
Rock climbing.....	❍	❍	❍	❍	❍
Rowing/canoeing.....	❍	❍	❍	❍	❍
Tennis/squash .....…	❍	❍	❍	❍	❍
Stair climber (or other similar equipment).....	❍	❍	❍	❍	❍
Walking for exercise.....	❍	❍	❍	❍	❍
Heavy yard work	❍	❍	❍	❍	❍
Jogging or running.....	❍	❍	❍	❍	❍
Bicycling .....……	❍	❍	❍	❍	❍
Aerobics (or other exercise class)...	❍	❍	❍	❍	❍
Swimming .....…	❍	❍	❍	❍	❍
Baseball, softball .....…	❍	❍	❍	❍	❍
Dance .....…	❍	❍	❍	❍	❍
Football	❍	❍	❍	❍	❍
Badminton .....……	❍	❍	❍	❍	❍
Soccer.....	❍	❍	❍	❍	❍
Street/floor hockey.....	❍	❍	❍	❍	❍
Volleyball	❍	❍	❍	❍	❍
Basketball .....……	❍	❍	❍	❍	❍
Skating (in-line/ice).....	❍	❍	❍	❍	❍
Cross-country skiing .....…	❍	❍	❍	❍	❍
Ice hockey/ringette .....…	❍	❍	❍	❍	❍
Martial arts.....……	❍	❍	❍	❍	❍
Weight training.....…….	❍	❍	❍	❍	❍
Other:					
_____..…	❍	❍	❍	❍	❍
_____..…	❍	❍	❍	❍	❍

2. **In the last 7 days**, *during the morning*, how often were you very active (for example: playing sports, exercise classes, strenuous occupational activity, strenuous household or child rearing tasks)? (Check one only.)

None..……..... ❍
1 time last week..... ❍
2 or 3 times last week..... ❍
4 or 5 times last week..... ❍
6 or 7 times last week..... ❍

3. **In the last 7 days**, *after lunch and before supper*, how often were you very active (for example: playing sports, exercise classes, strenuous occupational activity, strenuous household or child rearing tasks)? (Check one only.)

None..... ❍
1 time last week ..... ❍
2 or 3 times last week ..... ❍
4 or 5 times last week..... ❍
6 or 7 times last week..... ❍

4. **In the last 7 days**, *during the evening*, how often were you very active (for example: playing sports, exercise classes, strenuous occupational activity, strenuous household or child rearing tasks)? (Check one only.)

None ..... ❍
1 time last week ..... ❍
2 or 3 times last week ..... ❍
4 or 5 last week..... ❍
6 or 7 times last week ..... ❍

5. **On the last weekend**, how often were you very active (for example: playing sports, exercise classes, strenuous occupational activity, strenuous household or child rearing tasks)? (Check one only.)

None .....… ❍
1 time..... ❍
2 — 3 times..... ❍
4 — 5 times..... ❍
6 or more times..... ❍

6. Which *one* of the following describes you best for the **last 7 days**? Read *all five* statements before deciding on the *one* answer that describes you.

   F. All or most of my free time was spent doing things that involve little physical effort ..... ❍

   G. I sometimes (1 — 2 times last week) did physical things in my free time (e.g. played sports, went running, swimming, bike riding, did aerobics) ..... ❍

   H. I often (3 — 4 times last week) did physical things in my free time .....……. ❍

   I. I quite often (5 — 6 times last week) did physical things in my free time ……..... ❍

   J. I very often (7 or more times last week) did physical things in my free time. ……. ❍

7. Mark how often you did physical activity (for example: playing sports, exercise classes, strenuous occupational activity).

	None	Little bit	Medium	Often	Very often
Monday	❍	❍	❍	❍	❍
Tuesday	❍	❍	❍	❍	❍
Wednesday.....	❍	❍	❍	❍	❍
Thursday.....	❍	❍	❍	❍	❍
Friday	❍	❍	❍	❍	❍
Saturday	❍	❍	❍	❍	❍
Sunday	❍	❍	❍	❍	❍

- Were you sick last week, or did anything prevent you from doing your normal physical activities? (Check one.)

  Yes .....…. ❍
  No ..... ❍

  If Yes, what prevented you? _____

Bone Mineral Accrual Study
Leisure Time Exercise Questionnaire

1. Considering a **7-day period** (a week), how many times on the average do you do the following kinds of exercise for **more than 15 minutes** during your **free time** (write in each circle the appropriate number).

TIMES PER WEEK

a) STRENUOUS EXERCISE
(HEARTS BEATS RAPIDLY)

(i.e., running, jogging, hockey, football, soccer, squash, basketball, cross country skiing, roller skating, vigorous swimming, vigorous long distance bicycling

b) MODERATE EXERCISE
(NOT EXHAUSTING)

(i.e. fast walking, baseball, tennis, easy bicycling, volleyball, badminton, easy swimming, alpine skiing, popular and folk dancing)

c) MILD EXERCISE
(MINIMAL EFFORT)

(i.e. yoga, archery, fishing from river bank, bowling, horseshoes, golf, snowmobiling, easy walking)

2. Considering a **7-day period** (a week), during your **leisure-time**, how often do you engage in any regular activity long enough to **work up a sweat** (heart beats rapidly)?

OFTEN	SOMETIMES	NEVER/RARELY
1. [ ]	2. [ ]	3. [ ]

UNIVERSITY OF SASKATCHEWAN
FOOD FREQUENCY QUESTIONNAIRE

Name _____ Today's Date _____
[Subject code:_____]

Please list **nutritional supplements** used:

_____

_____

_____

_____

_____

_____

1. We want to know how often you eat or drink certain foods **each week**.

2. Think about a ***typical week***, not what you ate this week which might be different.

3. **Medium** portion sizes are given to help you determine the usual size of the food or drink.

4. If you eat *much less* than the medium portion size described, then give a fraction. For example, a small glass of milk is "2" the medium, so "2" describes your usual intake. If you drink 2 small glasses of milk every day, this is the same as drinking 7 medium drinks each week.

If you eat *much more* than a medium size portion, then indicate this by giving the number of portions your size is equal to. For example, a very large plate of spaghetti would be 2 or 3 medium portions.

5. Fill out the form similar to this **example**:

- If you drink a Large Chocolate milk (500mL carton) three times a week, then choose 6 medium portions per week because it is 2 times the size of the medium portion, and you have it 3 times (i.e., 3 x 2).

Food Type	Medium Size Portion	How Many Times You Have a *Medium* Portion Each Week
1. **MILK** *How much milk do you drink milk each week?*	glass or drink carton	**7** *[this is what you fill in]* *You may write in daily values e.g.,* **1/day** *(please indicate per day@ if you do)*

Food Type	Medium Size Portion	How Many Times You Have a *Medium* Portion Each Week
1. **MILK** *How much milk do you drink milk each week?* (Treat white or chocolate milk the same)	Glass or Drink carton (8 oz or 250 mL)	
1.a **SOY MILK** ____ regular soy milk ____ Ca fortified soy milk (E.g. *So Good* brand)	Glass or Drink carton (8 oz or 250 mL)	
2. **MILK IN Coffee/Tea** *How often do you have milk (or cream) in coffee or tea?*	1 Tbsp milk(or cream) in one cup of coffee or tea	
3. **MILK ON CEREAL** *How often do you eat cereal with milk each week?*	½ cup milk per bowl of cereal	
Milkshake Purchased at	10 oz	
Milk- Based DESSERT Ice Cream/ice milk, soy dessert, pudding, custard	½ cup (one scoop or pudding cup)	
Yogurt (also mini-go, Yop etc.)	one container (175 mL)	
4. **CHEESE** cheese single slice (in sandwich or as snack)	single slice	
----->hard cheese (such as cheddar)	piece (1 oz)	
----->soft cheese	Serving Size:	
----->cottage cheese	Serving Size:	

5. **BREAD** *How often do you eat bread each week?* (*Remember sandwiches*) Bread, roll, bun	1 slice bread 1 small roll 1/2 bagel	
Food Type	**Medium Size Portion**	**How Many Times You Have a *Medium* Portion Each Week**
6. **BUTTER/MARGARINE** Butter	one pat (1 teaspoon)	
Margarine	one pat (1 teaspoon)	
7. **LUNCH and DINNER ITEMS**	**Medium Size Portion**	**How Many Times You Have a *Medium* Portion Each Week**
____ Tofu	one piece (1 inch cube)	
____ Spaghetti with tomato sauce or noodles and sauce	1 plate (1 cup)	
7. **LUNCH and DINNER ITEMS** (continued)	**Medium Size Portion**	**How Many Times You Have a *Medium* Portion Each Week**
____ Macaroni and cheese	1 plate (1 cup)	
____ Canned Salmon (in sandwich or casserole)	1 serving (1 oz)	
____ Canned sardines	1 serving = 4 small fish	
____ Tuna (in sandwich or casserole)	1 serving (1 oz)	
____ Seafood (shrimp, lobster, salmon steak etc.)	3 oz	
____ Lasagna	1 square	
____ Perogies	Give usual number eaten:	

____ Do you have sour cream?	Circle: Yes or No?	
____ Tacos, burritos with cheese, beans, lettuce etc.	1 regular	
____ Pizza -->take-out -->frozen mini pizza	one slice one round	
____ Baked beans or other beans or lentils	½ cup	
____ Green salad	1 bowl (1 cup)	
____ Potatoes: mashed with milk	one scoop (½ cup)	
____ Eggs: any type	one whole (with yolk)	
____ Cream soups (made with milk)	one bowl (1 cup)	
____ Orange	one medium	
____ Orange Juice - regular ____ Orange Juice with Calcium	one juicepack (1 cup)	
_____ Eggo-type waffle or pancake	one	
____ Homemade or restaurant Pancake, Waffle, French Toast	one 5"across	
____ Broccoli, Spinach, Beet greens or kale	½ cup	
____ Taco chips, Nacho chips	28 g (2 small bag) or small bowl	

Questions or Comments:

PLEASE LIST Every FOOD and DRINK You Ate Yesterday

UNIVERSITY OF SASKATCHEWAN

Name: _____
Age: _____
Date: _____

Time	Food Items	Type & Preparation	Amount	Brand Name or Where Bought
Morning				
Mid-morning				
Noon Meal				
Midday				

Evening Meal				
Before Bed				
EXAMPLE	***CEREAL***	***CORN FLAKES***	***1 cup***	***Kellogs***

Was this intake usual? Circle one: Yes No (*if No, explain why not*_____
_____)
Did you take any vitamins/minerals during this time? Circle one: Yes No (if Yes, list names:
_____
_____

Family Cardiovascular Health Questionnaire

Including living and deceased, were any of your biological that is, blood relatives including grandparents, parents, brothers, sisters ever told by a health professional that they had:

a. Diabetes (excluding during pregnancy)?

Yes	□→→→	Type I	□
No	□	Type II	□
Don't Know	□	Both	□

If Yes, which family member?

Mother	□
Father	□
Mother's Mother	□
Mother's Father	□
Father's Mother	□
Father's Father	□
Brother	□
Sister	□
Other	□

What is the youngest age at which a member of your immediate family was diagnosed with diabetes?

_____age in years

b. High blood pressure, excluding during pregnancy before the age of 50?

Yes	□
No	□
Don't Know	□

If Yes, which family member?

Mother	□
Father	□
Mother's Mother	□
Mother's Father	□
Father's Mother	□
Father's Father	□
Brother	□
Sister	□
Other	□

What is the youngest age at which a member of your immediate family was diagnosed with high blood pressure?

_____age in years

c. Stroke before the age of 50?

Yes ☐
No ☐
Don't Know ☐

If Yes, which family member?

Mother ☐
Father ☐
Mother's Mother ☐
Mother's Father ☐
Father's Mother ☐
Father's Father ☐
Brother ☐
Sister ☐
Other ☐

What is the youngest age at which a member of your immediate family ever had a stroke?

_____age in years

d. Heart attack or angina before the age of 50?

Yes ☐
No ☐
Don't Know ☐

If Yes, which family member?

Mother ☐
Father ☐
Mother's Mother ☐
Mother's Father ☐
Father's Mother ☐
Father's Father ☐
Brother ☐
Sister ☐
Other ☐

What is the youngest age at which a member of your immediate was first diagnosed with heart disease?

_____age in years

Blood Pressure Questionnaire

1. Have you been diagnosed by a health professional as having high blood pressure?

Yes	☐
No	☐
Don't Know	☐

2. In the past month did you take medicine for blood pressure?

Yes	☐
No	☐
Don't Know	☐

3. Have you adopted lifestyle changes to treat hypertension?

Yes	☐
No	☐
Don't Know	☐

4. If yes, when did you adopt these changes?

Start: dd/mm/yy _____ Finish: dd/mm/yy _____

5. Please list the lifestyle changes you made.

_____
_____
_____
_____

Name: _____ Subject ID: _____

Fasting Questionnaire

Q1. When was the last time you ate or drank anything other than plain water? [Do not include diet soda, black coffee or tea with saccharine]

Time:_____ AM/PM Date: (dd/mm/yy) _____/_____/_____

Q2. Have you had any of the following since the above time and date:

a. Coffee or team with cream or sugar? [Include milk or non-dairy creamers]
   Yes □
   No □
   Not Sure □

   If yes, record time and date:

   Time: _____ AM/PM Date: (dd/mm/yy) ____/_____/____

b. Alcohol such as beer, wine or liquor?
   Yes □
   No □
   Not Sure □

   If yes, record time and date:

   Time: _____ AM/PM Date: (dd/mm/yy) ____/_____/____

c. Gum, breath mints, lozenges or cough drops, or other cough or cold remedies?
   Yes □
   No □
   Not Sure □

   If yes, record time and date:

   Time: _____ AM/PM Date: (dd/mm/yy) ____/_____/____

Name: _____ Subject ID: _____

d. Antacids, laxatives, or anti-diarrheals?

Yes □
No □
Not Sure □

If yes, record time and date:

Time: _____ AM/PM Date: (dd/mm/yy) ____/_____/____

e. Dietary supplements such as vitamins and minerals? [include multivitamins and single nutrient supplements]

Yes □
No □
Not Sure □

If yes, record time and date:

Time: _____ AM/PM Date: (dd/mm/yy) ____/_____/____

Thank you!

Family Cardiovascular Health Questionnaire

Including living and deceased, were any of your biological that is, blood relatives including grandparents, parents, brothers, sisters ever told by a health professional that they had:

e. Diabetes (excluding during pregnancy)?

Yes	□→→→	Type I	□
No	□	Type II	□
Don't Know	□	Both	□

If Yes, which family member?

Mother	□
Father	□
Mother's Mother	□
Mother's Father	□
Father's Mother	□
Father's Father	□
Brother	□
Sister	□
Other	□

What is the youngest age at which a member of your immediate family was diagnosed with diabetes?

_____age in years

f. High blood pressure, excluding during pregnancy before the age of 50?

Yes	□
No	□
Don't Know	□

If Yes, which family member?

Mother	□
Father	□
Mother's Mother	□
Mother's Father	□
Father's Mother	□
Father's Father	□
Brother	□
Sister	□
Other	□

What is the youngest age at which a member of your immediate family was diagnosed with high blood pressure?

_____age in years

g. Stroke before the age of 50?

Yes	☐
No	☐
Don't Know	☐

If Yes, which family member?

Mother	☐
Father	☐
Mother's Mother	☐
Mother's Father	☐
Father's Mother	☐
Father's Father	☐
Brother	☐
Sister	☐
Other	☐

What is the youngest age at which a member of your immediate family ever had a stroke?

_____age in years

h. Heart attack or angina before the age of 50?

Yes	☐
No	☐
Don't Know	☐

If Yes, which family member?

Mother	☐
Father	☐
Mother's Mother	☐
Mother's Father	☐
Father's Mother	☐
Father's Father	☐
Brother	☐
Sister	☐
Other	☐

What is the youngest age at which a member of your immediate was first diagnosed with heart disease?

_____age in years

Blood Pressure Questionnaire

1. Have you been diagnosed by a health professional as having high blood pressure?

Yes	☐
No	☐
Don't Know	☐

2. In the past month did you take medicine for blood pressure?

Yes	☐
No	☐
Don't Know	☐

3. Have you adopted lifestyle changes to treat hypertension?

Yes	☐
No	☐
Don't Know	☐

4. If yes, when did you adopt these changes?

Start: dd/mm/yy _____ Finish: dd/mm/yy _____

5. Please list the lifestyle changes you made.

_____
_____
_____
_____

BONE MINERAL ACCRUAL STUDY

PRE-TESTING GUIDELINES
(Morning appointment)

1. Blood and urine tests will be done at the Royal University Hospital Test Center
2. The Test Center is drop in only (no appointments)
3. Opening Hours: 8.00 am-4.00pm, Monday-Friday (except bank days and every third Friday)
4. Phone Number: 655-2519* Phone in advance to verify that the test center is open
5. Fill out the medication and fasting questionnaire prior to the sampling of blood and urine
6. Take with you the Community Lab Requisition form (purple form) plus all attached forms and hand in at the test center desk
7. If you have never been to the test center go to the main (old) entrance of the Royal University Hospital and follow signs for the mall. The test center is located in the hospital mall, close to the gift shop.

GUIDELIJNES TO FOLLOW

- Please refrain from the following:
  - Eating or drinking anything other than water during the **12 hours** prior to your testing appointment
  - Smoking and using other tobacco and nicotine products during the **2 hours** prior to your clinic appointment
  - Drinking any alcoholic beverages **on the day of** your appointment (from midnight)
  - Exercising **on the day of** your clinic appointment (from midnight)
  - Donating blood **2 days** prior to your clinic appointment

- Medication use:
  - Take your medications as usual on the day of your appointment

APPENDIX J: Published Letters to the Editor-in-Chief (re. Study 1)

INCOHERENCE WITH STUDIES USING ACTIGRAPH MTI AMONG CHILDREN AGE 6–12 YR

Dear Editor-in-Chief:

Activity monitors, and especially the ActiGraph MTI (ActiGraph MTI, LLC, Fort Walton Beach, FL), have come into wide use as a measure of physical activity (PA) among children (6–12 yr). Sherar et al. (6), using this device, addressed the important issue of the influence of physical maturity on the gender inequalities in PA. We commend the efforts by the authors to address these important questions. However, the authors used an inappropriate MET-to-count prediction equation and found a daily moderate to vigorous PA (MVPA) > 100 and about 80 min·d^{-1} across chronological and biological ages, respectively. These findings are as challenging as they are amazing, because concomitantly published data revealed a geometric mean of about 16 min·d^{-1} among children of a similar age group (3). If it should be acknowledged that PA is likely influenced by sociocultural and environmental disparities, and even if the PA among children deals with important interindividual variations, such a discrepancy between studies is surprising. The gap between the two reports is mainly due to an incoherent use of MVPA cutoff points.

Previously, we have reported an important bias (mean error of 113 min·d^{-1} in our sample) and some risks of misclassification when using an inadequate threshold to define PA among children (2). As opposed to earlier data (1,8), more recent studies (5,7) showed that the cut point of 3 METs underestimates the level of moderate intensity of PA behavior among children. This translates into an inflation of the time spent in MVPA, which is neither realistic regarding the growing prevalence of sedentary-related diseases (e.g., obesity and its comorbidities) nor reliable regarding the present PA recommendations for children. If, in a given sample, almost 100% of children meet the PA recommendations, one could ask oneself some questions about the true value of PA in combating sedentary-related diseases, and about the relevance of the recommendations themselves. Moreover, the MET-to-count cut-point used by Sherar et al. (6) may increase the risk of type I error when comparing boys with girls, because it is not discriminative enough.

"Brisk walking" is often considered as a reference of moderate intensity of PA. When compared with adults, the energy cost of this type of activity may be higher among children because of some physiological and biomechanical factors. Thus, the required 3 METs for adults are not applicable to children, and some data suggested a value close to 6 METs as the lower boundary of moderate intensity of activity for children (4,7). Accordingly, it is difficult to have confidence in the conclusion of the authors about the effect of physical maturity (biological age) on the gender inequalities in PA. Because comparisons between studies need to be based on solid background, the data provided by Sherar et al. (6) need to be reconsidered. Finally, we suggest that the correct cutoff point to define MVPA should not be under approximately 3000 counts per minute for children.

Comlavi B. Guinhouya, PhD
Hervé Hubert, PhD
Institute of Engineering in Health of Lille
University of Lille 2
Lille, France

DOI: 10.1249/MSS.0b013e318168da5a

REFERENCES

1. Freedson PS, Sirard J, Debold E, et al. Calibration of the Computer Science and Applications, Inc. (CSA) accelerometer. *Med Sci Sports Exerc*. 1997;29(Suppl.):S45.
2. Guinhouya CB, Hubert H, Soubrier S, Vilhelm C, Lemdani M, Durocher A. Moderate-to-vigorous physical activity among children: discrepancies in accelerometry-based cut-off points. *Obesity*. 2006;14:774–7.
3. Mattocks C, Leary S, Ness A, et al. Intraindividual variation of objectively measured physical activity in children. *Med Sci Sports Exerc*. 2007;39(4):622–9.
4. Pate RR, Trost S, William C. Critique of existing guidelines for PA in young people. In: Biddle S, Sallis JF, Cavill N, editors. *Young and Active?* London (UK): Health Education Authority; 1998. p. 162–72.
5. Puyau MR, Adolph AL, Vohra FA, Butte NF. Validation and calibration of physical activity monitors in children. *Obes Res*. 2002; 10:150–7.
6. Sherar LB, Esliger DW, Baxter-Jones ADG, Tremblay MS. Age and gender differences in youth physical activity: does maturity matter? *Med Sci Sports Exerc*. 2007;39(5):830–5.
7. Treuth MS, Schmidtz K, Cattelier DJ, et al. Defining accelerometer thresholds for activity intensity in adolescent girls. *Med Sci Sports Exerc*. 2004;36(7):1259–66.
8. Trost SG, Pate RR, Freedson PS, Sallis JF, Taylor WC. Using objective physical activity measures with youth: how many days of monitoring are needed? *Med Sci Sports Exerc*. 2000;32(2):426–31.

RESPONSE

Dear Editor-in-Chief:

We thank Drs. Guinhouya and Hubert for their comments and interest in this important area of research. Using similar methods to Trost et al. (4), we investigated whether the observation that girls are less active than boys is confounded by gender differences in physical maturity. We aligned the accelerometer data on chronological and biological age and found that when aligned on biological age the gender differences in physical activity disappeared (3).

Drs. Guinhouya and Hubert express concern about our use of Trost et al.'s (4) moderate to vigorous physical activity (MVPA) cut points because they felt these intensity thresholds were too low. Drs. Guinhouya and Hubert advocate the use of the Actigraph MVPA cut points of ≥ 3000 counts per minute. They suggest that if we were to use a more "discriminative" MVPA cut point that the gender differences in physical activity would persist despite any biological age adjustment. We assume their rationale stems from studies that show the gap in physical activity between genders increases as intensity increases (2,5). However, our data do not support this as we showed that when aligned on biological age, gender differences in vigorous physical activity only (counts per minute ranging from 3311 to 4381) also disappeared; therefore, we believe our conclusions remain justified.

We agree that there needs to be a consensus on accelerometer cut points used to delineate physical activity intensity levels. Unfortunately, many questions remain that preclude definitive cut-point selection for children and/or adults (see accelerometry best practices paper by Ward et al. (6) for a complete discussion). We caution against the selection of cut points that produce physical activity guideline compliance rates with face validity. For example, we have shown that a *prima facie* assessment of physical activity using *average day* accelerometry data can yield markedly different "meeting the guidelines" results compared with more rigorous assessments using *daily*, *bouts-only*, and a combination of both *daily and bouts-only* physical activity (see Figs. 3 and 5 in Esliger and Tremblay 2007 (1)). In this example, the percentage of children meeting the 60-min physical activity guideline ranges from 97% to 3% even though Trost et al. (4) cut points were used.

It is important to remember that accelerometers measure minutes (or indeed seconds) of physical activity that in the past could/would not have been captured with questionnaires. As such, we should not be surprised that children and even some adults may accumulate many minutes of MVPA. Our challenge going forward is to develop evidence-based cut points to determine the amount, intensity, and pattern of accelerometer assessed physical activity that provides health benefits.

Lauren Sherar, MSc
Adam Baxter-Jones, PhD
College of Kinesiology
University of Saskatchewan
Saskatoon, Canada

Dale Esliger, MSc
University of Exeter
School of Sport and Health Sciences
Exeter, United Kingdom

Mark Tremblay, PhD
Research Institute, Children's
Hospital of Eastern Ontario
Ottawa, Canada

DOI: 10.1249/MSS.0b013e318168da6a

REFERENCES

1. Esliger DW, Tremblay MS. Physical activity and inactivity profiling: the next generation. *Appl Physiol Nutr Metab.* 2007;32(2E): S195–207.
2. Fuchs R, Powell KE, Semmer NK, Dywer JH, Lippert P, Hoffmeister H. Patterns of physical activity among German adolescents: the Berlin–Bremen Study. *Prev Med.* 1988;17:746–63.
3. Sherar LB, Esliger DW, Baxter-Jones ADG, Tremblay MS. Age and gender differences in youth physical activity: does physical maturity matter? *Med Sci Sports Exerc.* 2007;39(5):830–5.
4. Trost SG, Pate RR, Sallis JF, et al. Age and gender differences in objectively measured physical activity in youth. *Med Sci Sports Exerc.* 2002;34(2):350–5.
5. Van Mechelen W, Twisk JW, Post GB, Snel J, Kemper HC. Physical activity of young people: the Amsterdam Longitudinal Growth and Health Study. *Med Sci Sports Exerc.* 2000;32(9):1610–6.
6. Ward DS, Evenson KR, Vaughn A, Rodgers B, Troiano RP. Accelerometer use in physical activity: best practices and research recommendations. *Med Sci Sports Exerc.* 2005;37(11 Suppl.): S582–8.

SPECIAL COMMUNICATIONS

APPENDIX K: Published Manuscript from Study 1

Age and Gender Differences in Youth Physical Activity: Does Physical Maturity Matter?

LAUREN B. SHERAR[1], DALE W. ESLIGER[1], ADAM D. G. BAXTER-JONES[1], and MARK S. TREMBLAY[1,2]

[1]*College of Kinesiology, University of Saskatchewan, Saskatoon, Saskatchewan, CANADA; and* [2]*Statistics Canada, Ottawa, Ontario, CANADA*

BASIC SCIENCES

ABSTRACT

SHERAR, L. B., D. W. ESLIGER, A. D. G. BAXTER-JONES, and M. S. TREMBLAY. Age and Gender Differences in Youth Physical Activity: Does Physical Maturity Matter? *Med. Sci. Sports Exerc.*, Vol. 39, No. 5, pp. 830–835, 2007. **Purpose:** To investigate whether observed gender differences in objectively measured physical activity (PA) in children (8–13 yr) are confounded by physical maturity differences. **Methods:** Four hundred and one children (194 boys and 207 girls) volunteered for this study. An Actigraph accelerometer was used to obtain seven consecutive days of minute-by-minute PA data for each participant. Minutes of moderate to vigorous PA per day (MVPA), continuous minutes of MVPA per day (CMVPA), and minutes of vigorous PA per day (VPA) were derived from the accelerometer data. Age at peak height velocity (APHV), an indicator of somatic maturity, was predicted in all individuals. Gender differences in the PA variables were analyzed using a two-way (gender × age) ANOVA. **Results:** Levels of PA decreased with increasing chronological age in both genders ($P < 0.05$). When aligned on chronological age, boys had a higher MVPA at 10–13 yr, a higher CMVPA at 9–12 yr, and a higher VPA at 9–13 yr ($P < 0.05$). When aligned on biological age, PA declined with increasing maturity ($P < 0.05$); however, gender differences between biological age groups disappeared. **Conclusion:** The observed age-related decline in adolescent boys and girls PA is antithetical to public health goals; as such, it is an important area of research. To fully understand gender disparities in PA, consideration must be given to the confounding effects of physical maturity.
Key Words: ACCELEROMETRY, ADOLESCENCE, MEASUREMENT, PEAK HEIGHT VELOCITY

One of the most pervasive findings in epidemiological studies of physical activity (PA) is the decline in PA with age. Although PA has been shown to decline throughout the lifespan (2,3,21), cross-sectional and longitudinal studies have shown that the decline in self-reported PA is greatest during the adolescent years. For example, a Finnish longitudinal study has shown that in boys, the greatest overall decline in PA was between 12 and 18 yr, and in girls, it was between 12 and 15 yr. Additionally, data from the Amsterdam Longitudinal Growth and Health Study (32) document the greatest decline in PA between 13 and 16 yr of age. Lastly, in a cross-sectional U.S. sample, Caspersen and colleagues (4) found that the greatest reduction in self-report PA occurred between 15 and 18 yr of age.

In addition to the consistent documentation of an age-related decline in PA in both boys and girls, it is also well established that boys are more active than girls at all ages (7,9,16,18,19,26,30). For example, an investigation of accelerometer-assessed PA in 375 students (grades 1–12) has shown that boys participated in more minutes of daily moderate to vigorous PA (MVPA) (with the exception of grades 1–3) and more minutes of daily vigorous PA (VPA) (30). Further evidence using a large sample ($N = 2185$) from the European Youth Heart Study has shown that 9- and 15-yr-old boys spent 20 and 36% more time than girls in daily moderate PA (16). In a Canadian study of 1057 normal-weight children, it was again found that boys spent more time in PA than girls. Specifically, boys in grades 3, 7, and 11, respectively, spent 9, 22, and 27% more time in MVPA than girls (27).

The observation that at a given age, boys are more active than girls may be well established, but at least one group believes the finding to be confounded. A longitudinal study by Thompson et al. (26) has found that during adolescence, the gender differences in self-reported PA disappeared when differences in physical maturity were controlled (26). The main concern regarding confounding is that, on average, girls mature approximately 2 yr before boys (14,24). For example, on average, boys reach peak height velocity, a somatic indicator of physical maturity, at approximately 14 yr of age, whereas girls, on average, reach the same maturity milestone at approximately 12 yr of age. Adolescence is recognized as a period of great physical, psychosocial, cognitive, and emotional change.

Address for correspondence: Lauren B. Sherar, 87 Campus Drive, Physical Activity Complex, Saskatoon, Saskatchewan, S7H 1P1; E-mail: lauren.sherar@usask.ca.
Submitted for publication July 2006.
Accepted for publication January 2007.

0195-9131/07/3905-0830/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/mss.0b013e3180335c3c

Each of these changes (which may be linked to physical maturity) influences adolescent participation in PA (20). Therefore, the consistent observation that boys are more active than girls during adolescence may purely be an artifact of boys maturing later than girls and, as a result, altering their PA behavior later.

Thompson et al. (26) have determined that gender differences in PA disappear when aligned on maturational age, suggesting that physical maturity may be intricately involved in the adolescent decline in PA. This has important public health implications. For example, adolescent PA interventions may need to target biological maturity groups rather than chronological age groups (i.e., grades in school) to effect positive change. However, to the authors' knowledge, the study by Thompson et al. (26) is the only one to date to consider biological maturation in the study of adolescent PA and has yet to be replicated using an objective assessment of PA. Therefore, the purpose of the present study was to investigate whether observed gender differences in objectively measured PA in children (8–13 yr) are confounded by physical maturity differences.

METHODS

Participants. Data from 194 boys and 207 girls, aged 8–13.9 yr, were included in this study. Participants between the ages of 8.0 and 13.9 yr were chosen because they are likely to be predominantly pre-PHV (on average, boys reach PHV at 14 yr of age, and girls reach PHV at 12 yr of age). This age group is important to study because most of the variation in physical maturity occurs during the years approaching PHV (14,24). Participants comprised four groups of children residing in Canada: 1) rural-living children from Saskatchewan (N = 127), 2) urban-living children from Saskatchewan (N = 91), 3) Old Order Mennonite children from southwestern Ontario (N = 119), and 4) Old Order Amish children from southwestern Ontario (N = 64). Information on the recruitment of participants can be found elsewhere (1,28). Each child gave written assent, and parental consent was also obtained. All procedures were approved by the institutional research ethics board.

Chronological age. Decimal age was calculated by subtracting date of birth from the measurement date. Chronological age categories were constructed using 1-yr intervals, for example, individuals between 8.00 and 8.99 yr of age were grouped in the 8-yr-old category. These age categories are consistent with the age-specific accelerometer cut points (30). The decision to use whole-year age categories in truncated format (e.g., 8.00–8.99 = 8) rather than centered on whole-year age midpoint (e.g., 7.50–8.49 = 8) was taken out of necessity. Because the physical activity data were calculated using whole-year age categories in truncated format according to the age-specific cut-points described by Trost et al. (30), we were obligated to present the data in this format. This is important because it ensures that children grouped in the same age category are held to the same count-intensity standard when determining how many minutes of physical activity they accumulate.

Anthropometry. Measurements were taken for stature, sitting height, and body mass. All measurements were performed by a professional fitness and lifestyle consultant certified by the Canadian Society for Exercise Physiology. Leg length was calculated by subtracting sitting height from stature.

Physical maturity (biological age). A common maturity-assessment technique in longitudinal studies is the determination of years from attainment of peak height velocity (PHV) (13). PHV is an indicator of somatic maturity and reflects the age at maximum growth rate in stature during adolescence (age at PHV, APHV). In the present cross-sectional study, each individual's years from APHV were predicted using a gender-specific multiple-regression equation that included height, sitting height, leg length, chronological age, and their interactions (15). The prediction of years from APHV results in a continuous measure of biological age. Biological age categories were constructed using 1-yr intervals, such that the −1 APHV age group included observations between −0.49 and −1.50 yr from (i.e., before) APHV.

Physical activity. Physical activity levels were directly measured for seven consecutive days, using the Actigraph 7164 accelerometer (Actigraph, LLC, Fort Walton Beach, FL). The Actigraph is a uniaxial accelerometer that detects vertical acceleration in the magnitude of 0.05–2.00g with a frequency response of 0.25–2.50 Hz (31). All accelerometers underwent a calibration check on a hydraulic shaker plate at varying accelerations and frequencies before being used in the study (6). Only accelerometers with intra- and interinstrument reliability values below 5% coefficient variation were used. Participants wore the Actigraph over the right hip using a waist-mounted nylon belt. Participants were asked to record when the monitor was put on in the morning and when it was removed in the evening before bed, for the purpose of distinguishing between activity time and sleep time. On completion of the data collection, the data were electronically downloaded, resulting in a file containing minute-by-minute movement counts for each child. After data were scanned for spurious measures, sleep time was determined from the log sheets, and activity counts were added to the data file for unworn daytime periods for which the activity was included on the log sheet (using the compendium of physical activities and MET-to-count conversion values derived via regression equations published by Trost et al. (29)). Files with minimal levels of incomplete data underwent imputation procedures to ensure that 7 d of data were available for analysis. A more complete description of the data-reduction procedures can be found elsewhere (5,28).

TABLE 1. Subject characteristics by gender, chronological age (CA), and biological age (BA).

CA Group (yr)	N	Stature (cm)	Body Mass (kg)	BA Group (yr)	N	Stature (cm)	Body Mass (kg)
Boys							
8	11	129.2 (7.8)	26.8 (4.7)	−5	12	127.1 (4.4)	25.3 (2.2)
9	39	137.8 (7.2)	34.6 (7.2)	−4	23	134.6 (5.6)	31.0 (5.8)
10	42	142.8 (7.2)	37.4 (7.2)	−3	62	141.7 (5.8)*	36.7 (4.2)*
11	43	148.0 (7.2)	44.0 (8.7)	−2	55	149.5 (4.8)*	43.3 (5.9)*
12	43	153.8 (8.0)	44.8 (8.3)*	−1	36	158.1 (5.6)*	50.7 (8.5)*
13	16	157.9 (8.5)	48.3 (8.0)				
Girls							
8	8	127.2 (5.2)	25.3 (3.6)	−3	15	130.3 (4.1)	27.7 (1.8)
9	32	135.8 (6.4)	33.0 (6.6)	−2	39	138.2 (6.7)	33.4 (5.9)
10	56	143.3 (6.3)	38.9 (8.3)	−1	64	145.4 (4.4)	40.6 (6.4)
11	46	149.0 (7.1)	43.2 (9.6)	0	47	151.6 (5.4)	45.6 (8.7)
12	56	155.8 (6.6)	49.6 (9.6)	1	36	160.2 (4.6)	54.3 (7.1)
13	9	159.9 (4.2)	52.5 (6.2)				

Means (SD). Biological age was calculated in years from estimated age at peak height velocity (APHV). * Significantly ($P < 0.05$) greater than girls. Observations that fell outside −5 to −1 for girls and −3 to 1 for boys were excluded because of the small number; thus, $N = 401$ when aligned by CA and $N = 389$ when aligned by BA.

The average number of minutes of moderate to vigorous PA (MVPA; accumulated minutes ≥ 3 METs), continuous minutes of MVPA (CMPVA; accumulated minutes ≥ 3 METs clustered in bouts ≥ 10 min), and minutes of vigorous PA (VPA; accumulated minutes ≥ 6 METs) per day were calculated using age-specific cut points (30).

Statistical analysis. Independent *t*-tests were used to investigate the gender difference in stature and body mass at each chronological and biological age. Gender and age (chronological and biological) differences in PA were tested using a two-way ANOVA. Included in each model were the main effects for age category and gender, and the interaction of gender and age. Physical activity data were log transformed because of the positive skewedness of MVPA, VPA, and CMVPA. The alpha level was set at 0.05. SPSS (version 11.5) was used to analyze the data.

RESULTS

The subjects' physical characteristics, by chronological and biological age category, are shown in Table 1. There were no significant differences between boys and girls in body mass or stature, except for age 12, where girls were significantly ($P < 0.05$) heavier than boys. When the data were aligned on biological age, boys were significantly taller and heavier than girls at all ages before PHV that were common between the groups.

For ease of interpretation, Figure 1 shows the arithmetic means and standard deviations (i.e., nontransformed) for MVPA, CMPVA, and VPA. When aligned on chronological age, PA showed the characteristic decrease with increasing age; however, not all decreases between age categories were significant ($P > 0.05$) (Fig. 1A, C, E). The boys were found to have a higher MVPA at 10–13 yr, a higher CMVPA at 9–12 yr, and a higher VPA at 9–13 yr compared with the girls ($P < 0.05$). For MVPA, the gender difference was the smallest at 8 yr of age (9.7%) and the greatest at 13 yr of age (30.5%). For CMPVA, the gender difference ranged from 20.9% at 9 yr to 52.1% at 11 yr. For VPA, the gender difference ranged from 20.8% at 9 yr to 46.3% at 11 yr.

When aligned on biological age, PA decreases with increasing biological age; however, not all decreases between biological age categories were significant ($P > 0.05$) (Fig. 1B, D, F). There were no significant differences in MVPA, CMVPA, or VPA between girls and boys at each biological age ($P < 0.05$).

Among the boys, the largest chronological age difference (relative to the previous chronological age category) in MVPA, CMVPA, and VPA occurred between 8 and 9 yr of age and was 28.4, 38.8, and 49.5%, respectively. Among the girls, the largest chronological age difference in MVPA, CMVPA, and VPA also occurred between 8 and 9 yr of age and was 23.6, 47.9, and 37.1%, respectively. Among the boys, the largest biological age difference (relative to the previous biological age category) in MVPA, CMVPA, and VPA occurred between −5 and −4 yr from APHV and was 28.4, 41.3, and 55.4%, respectively. Among the girls, the largest biological age difference in MVPA, CMVPA, and VPA occurred between −3 and −2 yr from APHV and was 23.0, 39.8, and 40.8%, respectively.

DISCUSSION

The present finding that accelerometer-assessed PA declines in both genders with increasing chronological age from 8 to 13 yr supports previous research (10,16,30). As expected, when boys and girls were compared on the basis of chronological age, boys accumulated significantly more MVPA, CMVPA, and VPA at most ages between 8 and 13 yr. The fact that boys are more active than girls is a fairly consistent finding; however, the magnitude of the gender differences seems to depend on the type and intensity of the activity (25,29). For example, Trost and colleagues (29) have shown that for VPA, the average gender gap was substantial at 45%, whereas the average gender gap for MVPA was only 11%. Results from the present study also demonstrate that the gender difference is greater for VPA than for MVPA. The greatest decrease in MVPA, VPA, and CMVPA occurred between 8 and 9 yr of age in boys as well as girls.

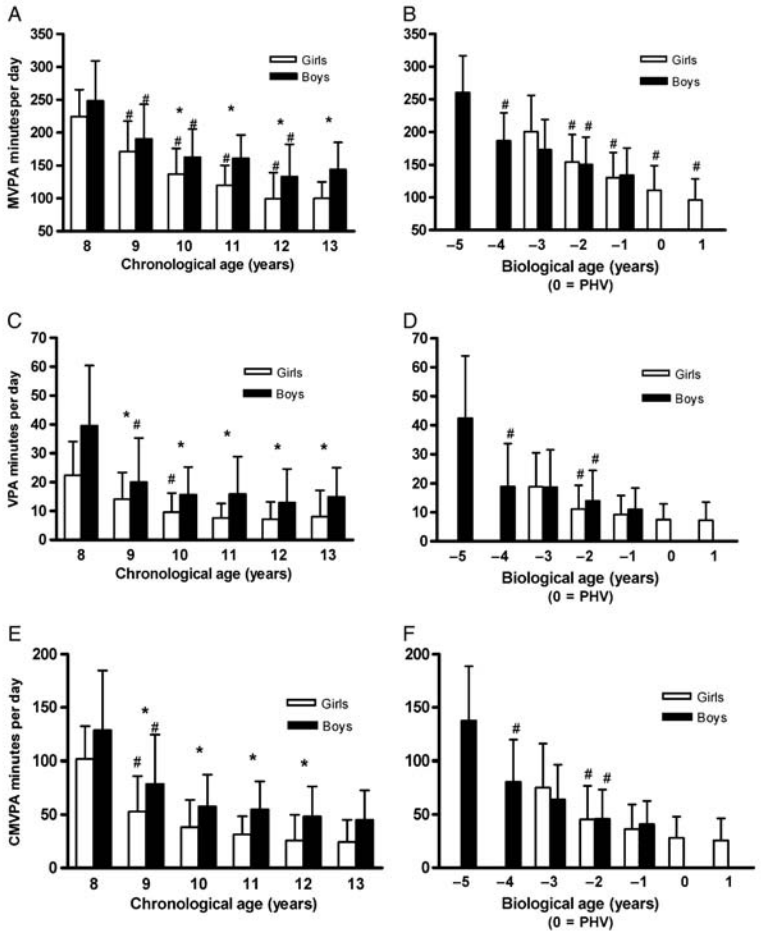


FIGURE 1—Objectively measured physical activity (PA) variables (mean ± SD) of boys and girls by chronological age (*A*, minutes of moderate to vigorous PA (MVPA); *C*, minutes of vigorous PA (VPA); *E*, continuous minutes of MVPA (CMVPA)) and biological age (years from age at peak height velocity (APHV)) (*B*, MVPA; *D*, VPA; *F*, CMVPA). * Significant gender difference within age category ($P < 0.05$); # significant difference from previous age category ($P < 0.05$).

The results from this study also support the findings of Thompson and colleagues (26) demonstrating that the gender differences in PA disappeared when aligned on physical maturity. This finding supports the notion that maturity differences between genders (i.e., on average, girls mature earlier than boys) may be one reason why research consistently shows that girls are less active than boys of the same chronological age. To the authors' knowledge, this is the first study to demonstrate this phenomenon using accelerometer-assessed PA.

Acknowledging the potential role of biological maturity in adolescent PA may help explain findings from previously published work. For example, Klasson-Haggebo and Anderssen (10) note that the rate of longitudinal decline in PA was similar for both boys and girls (25.9 and 27.2%, respectively) but that the age at which PA levels began to decline was younger for girls than boys. The authors provide no explanation for this finding; however, we speculate that the earlier decline in PA shown in girls is attributable to their earlier age of maturation because, on average, girls reach every maturity milestone earlier than boys (14). Consistent with previous research (14,22), the average APHV in the present study was 1.65 yr younger in girls compared with boys. The average APHV for boys (13.54 ± 0.81 yr) and girls (11.89 ± 0.58 yr) falls within the range of ages reported in previous literature (14,22).

The age-related decline in PA is well accepted but is not well understood (12). The present study provides evidence

to suggest that, at least in 8.0- to 13.9-yr-olds, the decline in PA is associated with biological maturity. Further supporting the importance of biological maturity, Finnish studies have shown that the beginning of the decline in PA is associated with puberty, with early-maturing girls being slightly less active than late-maturing girls (11). Furthermore, there is much evidence, from both cross sectional and longitudinal studies to suggest that the annual rate of decline in PA is much greater during adolescence than during late childhood and adulthood (4,9,25,32). However, most research exploring the age-related decline has not included children younger than 9 yr. The inclusion of younger children in the present study shows that the greatest decrease in MVPA, VPA, and CMVPA was between 8 and 9 yr (preadolescence) in boys as well as girls. However, longitudinal PA data including young children is required to make a definitive determination of the age at which the marked decline in PA occurs.

The consistent finding of an age-related decline in PA, with boys being more active than girls, provides the primary rationale for many interventions targeting adolescent girls. Three examples are the Trial of Activity in Adolescent Girls (TAAG) (23), Project FAB (8), and Girls on the Move (17). Because controlling for biological maturity seems to nullify gender differences in PA, it may be erroneous or misguided to target adolescent females over males when designing interventions. However, research does support gender-targeted PA interventions, because adolescent boys and girls prefer different activities, participate in PA for dissimilar reasons, and may face different barriers to PA (33). Interventions may also need to target girls at an earlier chronological age than boys, considering that, on average, girls mature 2 yr earlier than boys.

Previous research has explored barriers (and correlates) to adolescent PA in an effort to reduce the decline in PA over time. However, the relationship between barriers to PA and biological maturity has not yet been addressed. Identifying maturity-related barriers to PA during adolescence may provide valuable information that can be used to design interventions to help boys and girls of varying maturation overcome these barriers. In the future, the consideration of biological maturity may also cause us to rethink the implementation of adolescent PA interventions. The majority of interventions are based on chronological age groups (i.e., grades) in schools. Within one grade, there is considerable variation in biological age and in the level of biological maturity attained. For example, some 12-yr-old girls are already sexually mature, others are in the process of maturing, and others will not begin the process for several more years. Yet, all the girls have the same chronological age and are typically in the same grade in school. This wide variation in physical maturity was evident in the present sample of adolescents, with boys' APHV spanning 4.78 yr and girls' APHV spanning 4.84 yr. Therefore, in the design and implementation of school-based PA interventions, there may be a need to consider the variation in physical maturity within gender and grades. That said, one must acknowledge the potential hurdles in conducting a PA intervention based on maturity. For example, adolescents may be more comfortable participating in interventions with their peers in the same grade. Also, considering that most school scheduling is based on chronological age grades, organizing maturity-based interventions that span many grades may be problematic.

The present sample of boys and girls spanned 8.0–13.9 yr of age; therefore, some of the participants (especially the boys) were likely prepubertal. To completely document the confounding effects of biological maturity on age and gender differences in adolescent PA, there is a need for an objective assessment of PA and for an assessment of biological maturity on children older then 13.9 yr of age. The present sample is also cross-sectional; APHV was predicted, and as such, it is likely less accurate than when observed in a longitudinal study.

CONCLUSION

The observed age-related decline in adolescent boys and girls PA is antithetical to public health goals, and as such, it is an important area of research. To fully understand gender disparities in PA, consideration must be given to the confounding effects of biological age.

This research was supported by a grant from the Canadian Population Health Initiative of the Canadian Institute for Health Information, and by the Community and Population Health Research (CPHR) Training Program delivered by the Saskatchewan Population Health and Evaluation Research Unit (SPHERU).

REFERENCES

1. Bassett, D., M. S. Tremblay, D. W. Esliger, J. L. Copeland, J. D. Barnes, and G. E. Huntington. Physical activity levels in children of an Old Order Amish community. *Med Sci Sports Exerc.* 38(5 Suppl.):S81–S82, 2006.
2. Bijnen, F. C., E. J. Feskens, C. J. Caspersen, W. L. Mosterd, and D. Kromhout. Age, period, and cohort effects on physical activity among elderly men during 10 years of follow-up: the Zutphen Elderly Study. *J. Gerontol. A Biol. Sci. Med. Sci.* 53:M235–M241, 1998.
3. Caspersen, C. J., and R. K. Merritt. Physical activity trends among 26 states, 1986–1990. *Med. Sci. Sports Exerc.* 27:713–720, 1995.
4. Caspersen, C. J., M. A. Pereira, and K. M. Curran. Changes in physical activity patterns in the United States, by sex and cross-sectional age. *Med. Sci. Sports Exerc.* 32:1601–1609, 2000.
5. Esliger, D. W., J. L. Copeland, J. D. Barnes, and M. S. Tremblay. Standardizing and optimizing the use of accelerometer data for free-living physical activity monitoring. *J. Phys. Act. Health* 3:366–383, 2005.

6. Esliger, D. W., and M. S. Tremblay. Technical reliability assessment of three accelerometer models in a mechanical setup. *Med. Sci. Sports Exerc.* 38:2173–2181, 2006.
7. Jago, R., C. B. Anderson, T. Baranowski, and K. Watson. Adolescent patterns of physical activity differences by gender, day, and time of day. *Am. J. Prev. Med.* 28:447–452, 2005.
8. Jamner, M. S., D. Spruijt-Metz, S. Bassin, and D. M. Cooper. A controlled evaluation of a school-based intervention to promote physical activity among sedentary adolescent females: project FAB. *J. Adolesc. Health* 34:279–289, 2004.
9. Kimm, S. Y., N. W. Glynn, A. M. Kriska, et al. Longitudinal changes in physical activity in a biracial cohort during adolescence. *Med. Sci. Sports Exerc.* 32:1445–1454, 2000.
10. Klasson-Heggebo, L., and A. Anderssen. Gender and age differences in relation to the recommendations of physical activity among Norwegian children and youth. *Scand. J. Med. Sci. Sports* 13:293–298, 2003.
11. Laakso, R., and R. Telama. (1981) Sport participation of Finnish youth as a function of age and schooling. In: Telama, R. and X. Yang. Decline of physical activity from youth to young adulthood in Finland. *Med. Sci. Sports Exerc.* 32:1617–1622, 2000.
12. Livingstone, M. B., P. J. Robson, J. M. Wallace, and M. C. McKinley. How active are we? Levels of routine physical activity in children and adults. *Proc. Nutr. Soc.* 62:681–701, 2003.
13. Malina, R. M. Adolescent growth and maturation: selected aspects of current research. *Yearb. Phys. Anthropol.* 21:63–94, 1978.
14. Malina, R. M., C. Bouchard, and O. Bar-Or. *Growth, Maturation and Physical Activity*, Champaign, IL: Human Kinetics, pp. 307–333, 2004.
15. Mirwald, R. L., A. D. Baxter-Jones, D. A. Bailey, and G. P. Beunen. An assessment of maturity from anthropometric measurements. *Med. Sci. Sports Exerc.* 34:689–694, 2002.
16. Riddoch, C. J., L. B. Andersen, N. Wedderkopp, et al. Physical activity levels and patterns of 9- and 15-yr-old European children. *Med. Sci. Sports Exerc.* 36:86–92, 2004.
17. Robbins, L. B., K. A. Gretebeck, A. S. Kazanis, and N. J. Pender. Girls on the move program to increase physical activity participation. *Nurs. Res.* 55:206–216, 2006.
18. Sallis, J. F. Epidemiology of physical activity and fitness in children and adolescents. *Crit. Rev. Food Sci. Nutr.* 33:403–408, 1993.
19. Sallis, J. F., J. E. Alcaraz, T. L. McKenzie, and M. F. Hovell. Predictors of change in children's physical activity over 20 months. Variations by gender and level of adiposity. *Am. J. Prev. Med.* 16:222–229, 1999.
20. Sallis, J. F., J. J. Prochaska, and W. C. Taylor. A review of correlates of physical activity of children and adolescents. *Med. Sci. Sports Exerc.* 32:963–975, 2000.
21. Schoenborne, C. A. Health habits of U.S. adults, 1985: the "Alameda 7" revisited. *Public Health Rep.* 101:571–580, 1986.
22. Sherar, L. B., A. D. Baxter-Jones, and R. L. Mirwald. Limitations to the use of secondary sex characteristics for gender comparisons. *Ann. Hum. Biol.* 31:586–593, 2004.
23. Stevens, J., D. M. Murray, and D. J. Catellier. Design of the Trial of Activity in Adolescent Girls (TAAG). *Contemp. Clin. Trials* 26:223–233, 2005.
24. Tanner, J. M. *Foetus Into Man. Physical Growth From Conception to Maturity*. London, UK: Castlemead Publications, pp. 6–23, 1989.
25. Telama, R., and X. Yang. Decline of physical activity from youth to young adulthood in Finland. *Med. Sci. Sports Exerc.* 32: 1617–1622, 2000.
26. Thompson, A. M., A. D. Baxter-Jones, R. L. Mirwald, and D. A. Bailey. Comparison of physical activity in male and female children: does maturation matter? *Med. Sci. Sports Exerc.* 35:1684–1690, 2003.
27. Thompson, A. M., P. D. Campagna, L. A. Rehman, R. J. Murphy, R. L. Rasmussen, and G. W. Ness. Physical activity and body mass index in grade 3, 7, and 11 Nova Scotia students. *Med. Sci. Sports Exerc.* 37:1902–1908, 2005.
28. Tremblay, M. S., J. D. Barnes, J. L. Copeland, and D. W. Esliger. Conquering childhood inactivity: is the answer in the past? *Med. Sci. Sports Exerc.* 37:1187–1194, 2005.
29. Trost, S. G., R. R. Pate, P. S. Freedson, J. F. Sallis, and W. C. Taylor. Using objective physical activity measures with youth: how many days of monitoring are needed? *Med. Sci. Sports Exerc.* 32:426–431, 2000.
30. Trost, S. G., R. R. Pate, J. F. Sallis, et al. Age and gender differences in objectively measured physical activity in youth. *Med. Sci. Sports Exerc.* 34:350–355, 2002.
31. Tyron, W. W., and R. Williams. Fully proportional actigraphy: a new instrument. *Behav. Res. Methods Instrum. Comput.* 28: 392–403, 1996.
32. Van Mechelen, W., J. W. Twisk, G. B. Post, J. Snel, and H. C. Kemper. Physical activity of young people: the Amsterdam Longitudinal Growth and Health Study. *Med. Sci. Sports Exerc.* 32:1610–1616, 2000.
33. Vu, M. B., D. Murrie, V. Gonzalez, and J. B. Jobe. Listening to girls and boys talk about girls' physical activity behaviors. *Health Educ. Behav.* 33:81–96, 2006.

APPENDIX L: Accepted Manuscript from Study 2

Activity and barriers in girls (8-16 yrs) based on grade and maturity status

Lauren B. Sherar[1], Nancy C. Gyurcsik[1], M. Louise Humbert[1], Roland F. Dyck[2], Susan Fowler-Kerry[3], Adam D.G. Baxter-Jones[1],

[1]College of Kinesiology, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

[2]College of Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

[3]College of Nursing, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

Running title: Physical Activity and Biological Maturity

Abstract

Minimal research has examined whether the decline in PA among adolescent girls is associated with chronological age (CA) or biological age (BA). **Purpose:** To describe the PA levels and perceived barriers to PA of adolescent girls grouped by school grade and maturity status (i.e., early or late maturing) within grades. **Methods:** 221 girls (aged 8-16 years; grades 4-10) wore an Actical accelerometer for 7 days and then completed a semi-structured, open ended questionnaire on perceived barriers to PA over the 7 day period. Predicted age at peak height velocity and recalled age at menarche were used to assess maturity among the elementary and high school girls, respectively. Maturity and grade group differences in PA were assessed using a MANCOVA and independent sample t-test, and barriers to PA using chi squared statistics. **Results:** Daily minutes spent in moderate to vigorous PA decreased by 40% between grades 4 to 10. Within grade groupings, no differences in PA were found between early and late maturing girls ($p>0.05$). Grades 4-6 participants cited more interpersonal (i.e., social) barriers. Grades 9-10 participants cited more institutional barriers to PA, primarily revolving around the institution of school. No differences were found in types of barriers reported between early and late maturing girls. **Conclusion:** Since PA and types of perceived barriers to PA were dependent on grade, future research should work to identify the most salient (i.e., frequent and limiting) barriers to PA by CA in youth. Once reliably identified, multi-pronged intervention strategies to help youth cope with their salient barriers must be tested for effectiveness.

Keywords: accelerometer, menarche, peak height velocity, ecological framework

INTRODUCTION

Paragraph Number 1: Regular participation in physical activity (PA) during childhood and adolescence is important for health and well-being. Further, this type of positive health behavior can track into adulthood (11). Unfortunately, one of the most ubiquitous findings in PA research is the decline in PA with increasing chronological age (CA), which is most marked during the adolescent period (22-24,27). Cross-sectional studies reveal that activity levels can decrease by as much as 50% during adolescence (24,27).

Paragraph Number 2: Little research has been directed towards understanding whether the decline in PA among adolescent girls is associated with their CA or biological age (BA). Whereas CA is determined by the date a child is born, a child's BA is how close the child is to reaching the mature adult state. Biological age does not necessary proceed in concert with the calendar thus children of the same age can differ by several years in BA (13). Understanding the influence of BA on the decline in PA would better inform researchers about the most effective timing of interventions. In a similar vein, understanding whether perceived barriers, defined as personal or situational factors that slow or prevent one from engaging in regular PA (3), differ by CA and/or BA would provide valuable information for future intervention work aimed at reducing or negating salient barriers as a way to impact adolescent girls' regular participation in PA.

Paragraph Number 3: **Biological age:** The major growth process occurring during adolescence is puberty. Puberty is a period of rapid hormonal, physiological, and physical changes (13). One cause of the decline in PA among adolescent girls may be related to the experience of pubertal development (i.e., BA or maturity). Two previous studies found that with increasing BA there was a decrease in self-report (23) and objectively

measured (22) PA among adolescent girls. However, when considering the impact of pubertal development on PA, the *timing* of pubertal or biological maturity may be of more importance. Timing of biological maturity refers to an individual's biological maturity status when compared to peers of the same CA (13). In this manner a girl can be referred to as early, average, or late maturing for her CA. Because an early maturing girl is entering puberty at a younger CA than her peers, she may be more sensitive to the physical changes occurring during puberty. In fact, previous research has shown that early biological maturation is associated with poorer body image and increased eating disorders, distress, anxiety and depression (9). As a result of these aforementioned outcomes associated with the timing of biological maturity, new and distinct perceived barriers to PA may be experienced.

Paragraph Number 4: **Perceived barriers to physical activity:** Perceived barriers to PA are defined as factors that make PA difficult or completely inhibit it (3). Barriers can be personal or situational in nature (3). Using an ecological approach (14), personal barriers are typically defined as intrapersonal barriers, which are factors within an individual that prevent activity (e.g., lack of motivation) (5,10). Situational barriers can be further delineated into the categories of interpersonal, institutional, community, or public policy. Interpersonal barriers revolve around informal and formal social support networks (e.g., no friend to be active with). Institutional barriers occur within social institutions (e.g., school-based barrier of not having sufficient playground equipment). Community barriers occur between organizations and institutions within defined boundaries (e.g., lack of community-based activities). Public policy barriers revolve around policies and laws impacting activity (e.g., a law prohibiting road hockey). The importance of identifying

barriers within the aforementioned categories is that distinct intervention strategies may be required for barrier alleviation in the different categories (5,10,14).

Paragraph Number 5: In general, barriers have been identified as a consistent inverse correlate of PA across a diverse range of populations, including adolescents (1). However, recent research has found that although some barriers, such as a lack of motivation, are common across populations, other barriers are specific to the population under study (i.e., population-specific barriers) (4,5,10). For example, among a study population of female and male students in grade 7 through the first year of university, barriers specific to participants in specific grades were found, such as social invitations during one's planned physical activity time (i.e., an interpersonal barrier) among first-year university students (10). The institutional barrier of a heavy school workload was specific to grades 11-12 and first-year university students but not to students in grades 7-10. Further, of note, as grade in school increased, the number of reported barriers also increased.

Paragraph Number 6: To date, all prior research examining barriers to PA in youth and adolescents has focused on CA or grade. However, no research has considered the role that puberty may have on perceived barriers to PA. As indicated previously, girls of the same age vary in their degree and timing of biological maturity (13) and thus may experience different barriers to PA. For example, early maturing girls may be more likely to experience the barrier of concern over how one looks compared to less biologically mature girls (i.e. late maturers).

Paragraph Number 7: Thus, the first purpose of the present study was to describe the PA levels of adolescent girls (i.e., aged 8-16 years) by school grade and maturity status (i.e.,

early or late maturing) within grades. The second purpose was to to identify perceived barriers to PA of adolescent girls by grade and maturity status within grades. We hypothesized that: (a) PA would decrease with increasing grade and (b) within grade groups, early maturing girls would participate in less PA than late maturing girls. Given the expected decline in PA with CA and BA, we also hypothesized that: (a) the average number of barriers to PA would increase as grade in school increased and (b) within grade groups, early maturing girls would experience a greater number of barriers to PA that were specific to the timing of biological maturity, such as embarrassment over pubertal development, when compared to late maturing girls.

METHODS

***Paragraph Number 8*: Participants:** Anthropometric and barriers to PA data were available on 221 girls in grades 4 (n=21; 9-10 yrs), 5 (n=22; 10-11 yrs), 6 (n=19; 11-12 yrs), 7 (n=24; 12-13 yrs), 8 (n=22; 13-14 yrs), 9 (n=55; 14-15 yrs) and 10 (n=58; 15-16 yrs), at 5 different schools (3 elementary and 2 high schools). All schools were publicly funded and located in Saskatoon, Saskatchewan, Canada. Each child gave written assent and parental written informed consent was also obtained. All procedures were approved by the Institutional Research Ethics Board.

***Paragraph Number 9:* Anthropometry:** Measurements were taken for stature, sitting height and body mass. Skinfold thickness at five sites of the body (subscapular, triceps, biceps, iliac crest and medial calf) were used to assess body fat. All measurements were performed by a Professional Fitness and Lifestyle Consultant certified by the Canadian Society for Exercise Physiology. Leg length was calculated by subtracting sitting height

from stature. Girls' stature and body mass was used to calculate their BMI ($kg/(m^2)$).

Paragraph Number 10: **Physical Maturity (Biological age):** A common maturity assessment technique in longitudinal studies is the determination of years from attainment of peak height velocity (PHV), (13). Peak height velocity is an indicator of somatic maturity and reflects the age at maximum growth rate in stature during adolescence (age at PHV, APHV). In the present cross-sectional study, BA was attained by predicting years from reaching PHV. This was acheived using a gender specific multiple regression equation that included measures of stature, sitting height, leg length, CA, and their interactions to predict how many years a girl is from APHV (16) (Equation 1). Subtracting years from APHV from CA yields a predicted APHV.

Equation 1:

Years from reaching PHV = -9.376 + (0.0001882 * Leg Length and Sitting Height interaction) + (0.0022 * CA and leg length interaction) + (0.005841 * CA and Sitting Height interaction) + (0.002658 * Weight by Height ratio)

Paragraph Number 11: The prediction of APHV is most accurate around the attainment of PHV (approximately 12 years of age in girls (13)). In the present sample, high school girls' ages ranged from 14.1-16.7 (15.25 ± 0.61) years; therefore, the regression equation was deemed inaccurate. High school girls' biological maturity was thus estimated using a BA scale related to age of attainment of menarche. The girls were asked if they had experienced menses and if so, what month and year it had occurred. Age at menarche was calculated from date of menarche (month and year) and date of birth. A total of 4 girls

(5%) had not attained menarche. For the maturity status assessment elementary school girls were grouped based on APHV quartiles and the high school girls on age at menarche quartiles. The top quartile was classified as 'early maturers' and the bottom quartile as 'late maturers'. Consequently, these two groups indicate timing of biological maturity relative to the sample and are not intended to indicate either precocious or delayed puberty.

Paragraph Number 12: **Physical Activity:** Physical activity was directly measured on 182 girls (82% of the sample) for seven consecutive days using calibrated Actical accelerometers (Mini Mitter Co., Inc., Bend, OR). Actical is an omnidirectional accelerometer which is sensitive to movement in all directions and is a valid and reliable measure of PA in children and adolescents (18). A 7 day protocol was chosen because it provides reliable estimates of usual PA behavior in children and adolescents and accounts for potentially important differences in weekend versus weekday activity behavior as well as differences in activity patterns within a given day (26). The monitors were attached with an adjustable, elasticized nylon belt. The device was positioned on the right hip, above the iliac crest. When worn on the hip the Actical is most sensitive to vertical movements of the torso and thus is designed for measurement of whole body PA. The Actical is sensitive to movements in the 0.5- to 3-Hz range which allows for detection of sedentary as well as high-energy movements (18); however, it will filter high-frequency movements. The filtered acceleration signal is digitized and integrated over a user-specified epoch interval. At the end of each epoch, the summed value or ''activity count'' is stored in memory. For the current study, 15 second epochs were used.

Paragraph Number 13: Participants were asked to record when the monitor was put on in the

morning and removed in the evening before bed for the purpose of distinguishing between activity time and sleep time. Upon completion of the data collection, the data were electronically downloaded resulting in a file containing 15 second movement counts for each girl. After data were scanned for spurious measures, sleep time was determined from the log sheets. Files with minimal levels of missing data underwent imputation procedures to ensure seven days of data were available for analysis, for more information on procedures see (8). In brief, all files were retained for analysis if it included at least five full days of data, with at least one of those be a weekend day. A non-monitored weekday was replaced by the mean data of the other four weekdays. The monitored weekend day was used to represent the non-monitored weekend day.

Paragraph Number 14: Physical activity data were presented as average accelerometer counts per day and average minutes spent in moderate to vigorous PA (MVPA) per day. Accelerometer counts per day were used to evaluate the raw data without influence of cut-points. The child/adolescent specific intensity cut-point of ≥ 1500 counts per minutes (18) were used to calculate minutes spent in MVPA.

Paragraph Number 15: **Barriers to physical activity:** Barriers to PA were assessed through a semi-structured open-ended survey. The survey was previously piloted for readability and understandability in a sample of grade 4-8 students (N=78) from a school not recruited for participation in the study. Minor modifications in wording were made to the survey for use in the actual study based on the pilot testing. The survey was not pilot tested in the high school students since the format, including the wording, was based on a similar survey used in previously published research with high school students (10).

Paragraph Number 16: After the participants wore the accelerometers for one week (i.e., 7

days), the girls returned the accelerometers and data on barriers to PA were collected on all participants (n=221) through the semi-structured, open-ended survey. An investigator distributed surveys to the students during class and read the instructions on how to complete the questions. The grades 4-8 participants completed the survey during regularly scheduled class day and the grades 9-10 participants during a required physical education class. Participants were first asked "*Were there any physical activities that you would have liked to do but didn't over the last 7 days*?" If answered affirmatively, girls were then asked in an open fashion to list up to five barriers that prevented participation over the last 7 days. This open-ended barrier measure was consistent with those used in other research (10) . Confidentiality of responses was assured.

Paragraph Number 17: **Data analyses:** Independent t-tests and one-way ANOVA were used to investigate the difference in descriptive statistics between grade (i.e. grades 4-6, grades 7 and 8, and grades 9 and 10) and maturity status groups (i.e. early and late), respectively. Differences in PA variables across the same grade and maturity groupings were obtained using a MANCOVA. Body fat (sum of five skinfolds) and CA (in the maturity group comparison only) were added as covariates. It was necessary to control for body fat in the analyses because higher body fat or overweight status is linked with both the independent (i.e. BA) and dependent (i.e. PA) variables (6,25). Physical activity data were log transformed due to the positive skewness of accelerometer counts and MVPA.

Paragraph Number 18: In line with previously published research (5,10), two steps were followed to classify the barriers to PA listed by the participants. First, three researchers independently coded each barrier into one of four categories: intrapersonal, interpersonal, institutional or community. Using the ecological model (14), the following definitions

were provided for use in the classification: (a) Intrapersonal barriers are characteristics of the individual that may prevent PA, (b) Interpersonal barriers are formal and informal social networks and support systems that may prevent PA, (c) Institutional barriers occur within social institutions with organizational characteristics that may prevent PA, and (d) Community barriers occur between organizations, institutions, and informal networks within defined boundaries that may prevent PA. The ecological group 'public policy' was not included in the present study as the majority of the girls were deemed too young to recognize barriers associated with public policy. This is supported by previous research (10), where no public policy barriers were reported by adolescent girls. After classification, the three researchers discussed all barriers that were not similarly characterized (initially the researchers reached agreement on the categorization of 88% of all barriers), until agreement was reached.

Paragraph Number 19: To obtain descriptive statistics on the number of barrier categories and specific types of barriers within each category, percentages of reported barriers were calculated within the three grade groupings: (a) grades 4-6, n=62 (b) grades 7-8, n=46 (c) grades 9-10, n=113. Collapsing the grade groupings increased sample size and was used to investigate the whole adolescent period, while also considering the transition from elementary to high school. An ANOVA and independent t-tests were used to determine if the average number of barriers identified by girls differed among grade and maturity groupings, respectively. A chi squared statistic was used to investigate if the number of responses in each barrier category differed by grade or maturity grouping. The alpha level was set at 0.05. SPSS (version 11.5) was used to analyze the data.

RESULTS

Paragraph Number 20: **Physical activity by grade groupings:** Heights and body mass were within reference standard ranges for all chronological ages (28). Girls in grades 7-8 and 9-10 were significantly taller and heavier, and had a greater BMI and sum of five skinfolds than girls in lower grade groupings (Table 1). A MANCOVA showed that accelerometer counts ($F[2,179] = 27.8$, $p < .05$) and MVPA ($F[2,179] = 32.6$, $p < .05$) significantly decreased with an increase in grade grouping. Bonferroni post-hoc tests revealed a significant difference in accelerometer counts and MVPA among all grade groupings (Table 1). Between grade 4 and grade 10, average counts decreased by 44.03% and MVPA decreased by 40.41%.

Paragraph Number 21: **Physical activity by biological maturity groupings:** The early maturing elementary school girls were taller and heavier ($p<.05$) when compared to late maturing girls (Table 2). In contrast there was no significant difference in the height or weight of early and late maturing high school girls. This represents the 'catch-up growth' of late matures. In terms of PA variables, a MANCOVA (controlling for body fat and CA) revealed no significant differences in accelerometer counts or MVPA between early and late maturing elementary school girls. Like wise, there were no significant differences in accelerometer counts or MVPA between early and late maturing high school girls.

Paragraph Number 22: **Barriers to physical activity by grade groupings:** Out of 221 girls, 160 (72.4%) listed a PA they would have liked to do but did not do over the previous seven days. In total, 374 barriers were listed by these 160 participants, with an average of 2.34 ± 1.37 barriers being reported per participant. When examined by grade group, the

average number of barriers identified by girls in grades 4-6 was 2.19 $\pm$ 1.10, grades 7-8 was 2.40 $\pm$ 1.35 and grade 9-10 was 2.38 $\pm$ 1.44. A one way ANOVA examined the grade effect. There were no differences in the frequency of barriers listed among the grade groups (p>.05).

Paragraph Number 23: **Barrier categories by grade groupings:** Figure 1 displays the overall percentage of barriers that were reported in each specific barrier category (intrapersonal, interpersonal, institutional and community) by grade groupings. A 4 (barrier category) x 3 (grade grouping) chi square test was significant (χ^2 (6; N=77) =20.41, p<.05), indicating that overall the number of responses in specific barrier categories was related to grade groupings. Follow-up tests using adjusted residuals showed that grades 4-6 participants cited significantly more interpersonal barriers (adjusted residuals=3.2, p<.05) and grades 9-10 participants cited significantly more institutional barriers (adjusted residuals = 2.7, p<.05).

Paragraph Number 24: **Specific type of barriers by grade groupings:** Tables 3 through 6 contain example descriptors of specific barrier types within the categories of intrapersonal, interpersonal, institutional or community. Some of the most apparent examples among these barriers are outlined below.

Paragraph Number 25: As Table 3 shows, the two specific intrapersonal barrier types of having an illness/injury (e.g. “I wanted to go to my hockey practice but I got sick”) and a lack of time (e.g. “[I] didn't have enough time to go running”) comprised 69% of all responses by grades 4-6, 52% of all responses by grade 7-8 participants, and 34% of all responses by grade 9-10 participants. Interestingly the intrapersonal barrier of the lack of motivation/lazy (e.g. “I did not feel very motivated this week”) began to emerge,

comprising 16% and 29% of all responses in grades 7-8 and grades 9-10, respectively. Also, the barrier of paid work (e.g. “I guess working evenings and weekends makes a lot less time for physical activity”) was a prominent barrier (27% of all responses) among participants in grades 9-10.

Paragraph Number 26: As seen in Table 4, the two specific types of interpersonal barriers, friends did not want to go with me (e.g. “I wanted to go swimming but my friends bailed”) and a person in authority said no to activity (e.g. “My mom said no because she didn't want to drive me”) comprised the majority of all responses by grades 4-6, grades 7-8 and grades 9-10 participants (85%, 71% and 89%, respectively). The intrapersonal barrier of coaches/teachers altering scheduling of activity (e.g. “I wanted to play soccer but my soccer practice was cancelled”) was cited among all grades but was more prominent among the elementary school participants (grade 4-8).

***Paragraph Number 27*:** In terms of institutional barrier category, school physical education or extra-curricular activity scheduling (e.g. “I wanted to do physical activity at lunch but had drivers ed.”) and the timing of other organized activities conflicted (e.g. “I had to baby-sit at church instead of swimming”) encompassed 80% of all responses by grades 4-6, 50% of all responses by grades 7-8 and 34% of all responses by grades 9-10 (Table 5). Too much homework (e.g. “When I have too much homework I can't go skating”) emerged in grade 9-10 as the most prominent manifestation of this type of barrier (55% of all responses).

Paragraph Number 28: In terms of community barriers, some consistency existed across grades with weather is too cold/snowing (e.g. “Too much snow, so I couldn't go for my usual dog walk”) comprising 83% of all grade 4-6, 68% of all grade 7-8 and 75% of all

grade 9-10 responses (see Table 6). Although cited less frequently, the community barrier 'the facility is too far away' (e.g. "We didn't have a [swimming] pool close by) was reported consistently across grades (grades 4-6, 17%, grades 7-8, 17% and grades 9-10, 18%).

Paragraph Number 29: **Barriers to physical activity by biological maturity groupings:** When examined by maturity grouping, the average number of barriers identified by early maturers was 2.43 $\pm$ 1.17 and by late maturers was 2.47 $\pm$ 1.44. An independent sample t-test revealed no significant difference between the means.

Paragraph Number 30: **Barrier categories by biological maturity groupings:** Figures 2 and 3 display the overall percentage of barriers that were reported in each specific barrier category (intrapersonal, interpersonal, institutional and community) by maturity groupings for elementary and high school girls, respectively. The group was split by school (i.e. elementary and high school) because previous analyses showed that older girls experienced different barriers to PA than younger girls. A 4 (barrier category) x 2 (maturity grouping) chi square test was not significant. Thus, the overall frequency of responses in specific barrier categories was not related to maturity groupings for either elementary or high school students. Further examination of the specific types of barriers reported in each category did not show, as expected, the reporting of any maturity-related barriers.

DISCUSSION

Paragraph Number 31: **Physical activity by grade and maturity groupings:** The first objective of the study was to examine the levels of PA across grades in school and

maturity groups. Our finding, that accelerometer assessed MVPA declines with an increase in grade, supports previous research (22-24,27).

***Paragraph Number 32*:** Until recently the specific relationship between levels of PA and biological maturity status had not been addressed. In the last year (i.e., January to December, 2007) five studies have been published which compare PA levels among maturity groups, with mixed results. Niven and colleagues (17) found no difference in self-reported PA among early, average and late maturing 11-year old girls. In contradiction, a longitudinal study of 2247 adolescent girls found that early maturing girls participated in more minutes of self-reported VPA than late maturing girls (29). Baker and colleagues (2) showed that early maturing girls (assessed via a combination of estradiol, breast development and mother's report on pubertal development using the pubertal development scale at age 11) participated in less accelerometer measured PA at age 13 when compared to late maturing girls. Likewise, a study by Riddoch and colleagues (19) showed that parental reported pubic hair and breast development was inversely related to PA among 2933 eleven year old girls.

Paragraph Number 33: The present study found no significant differences in participation in MVPA between early and late maturing girls, which contradict findings from the two other studies (2,19) that used an accelerometer. The contradictory finding in the present study could be related to the indicator(s) used to assess maturity. When conducting pediatric research it is important to remember that maturity indicators (e.g. pubic hair stage, genital stages, APHV, age at menarche, skeletal age etc.) are not equivalent. Predicted APHV and recalled age at menarche were the maturity indicators used in the present study. It is possible that development of secondary sexual characteristics, the

maturity indicator featured in the aforementioned studies, is more closely related to the girls' disengagement from PA than timing of peak velocity in height and onset of menstruation. Secondary sex characteristics are the first overt sign of puberty, whereas PHV and menarche are later occurring pubertal events (21). The appearance of secondary sex characteristics, in particular breast development, may cause a girl to feel self conscious, or even uncomfortable about participating in PA. Although, in the present study, the elementary school girls with earlier predicted APHV and the high school girls with younger recalled age at menarche are likely more sexually mature, the direct relationship between development of secondary sex characteristics and participation in PA could not be assessed. Wickel and Eisenmann (30) also used predicted APHV to create maturity groups and found no relationship between maturity status and pedometer determined PA in 14-15 year old girls.

Paragraph Number 34: **Barriers to physical activity by CA and BA:** The hypothesis that the average number of barriers to PA would increase as grade increased was not supported. There were however, fluctuations in the types (i.e., diversity) of the ecologically-based barrier categories that were reported across grade groupings. Interpersonal barriers were the primary type of barrier category reported by elementary students in grades 7-8. Among the high school students, mainly intrapersonal and institutional barriers were reported. The latter finding is consistent with findings from a previous study that used an ecological framework to examine the pattern of barriers among students from grade 7 through first year of university (10).

Paragraph Number 35: Fluctuations in the specific types of barriers within each ecological category across the grade groupings were also found. The finding that elementary school students did not report the institutional barrier of too much homework as a barrier, in contrast to the high reporting of this barrier among high school students, may be a reflection of the increased academic demands that occur during high school. Similar findings have been reported in other research (1,10). The intrapersonal barrier of a lack of motivation/lazy was frequently reported by participants in grades 9-10, which again parallels finding from previous research (10). The mounting consistency of the reporting of these types of barriers by high school students suggests that an effective intervention strategy may be one which helps high school students learn and become confident in their self-regulatory skills and abilities to cope with such barriers (12,31). For example, self-regulatory skills that may help students cope with homework and a lack of motivation barriers may include: a) setting goals in regards to amount of time spent doing regular PA and homework, b) self-monitoring of these behaviors and thus whether goals are being achieved, and c) the re-establishment of weekly goals on a continuous basis.

Paragraph Number 36: Also of interest was the reporting of the interpersonal barrier of a person in authority not permitting activity. Over half of the participants in grades 4-6, 7-8, and 9-10 reported this particular barrier. This finding suggests that opportunities to participate in PA may not be completely under the direct control of youth and adolescents of these particular chronological ages. As such, intervention strategies to make authority figures, such as parents and teachers, aware of their responsibility in creating PA opportunities for youth and adolescents may be effective.

Paragraph Number 37: The present study is the first to consider a girl's maturity status (i.e. early or late maturing) in concert with perceived barriers to PA. The hypothesis that early maturing girls would experience more barriers to PA was not supported. No significant differences were evident in the frequency of reported barriers or in the specific types of barriers listed between early and late maturing girls. This finding was surprising considering that previous research has found that early pubertal maturation is associated with negative psychological well-being among girls (9). Furthermore, a longitudinal study specifically designed to explore the relationships among maturity status, psychological well-being and PA in 178 adolescent girls found that early maturity at age 11 predicted lower psychological well-being at age 13 including depression (e.g. I hate myself, I have difficulty sleeping), global self-worth (e.g. I am often disappointed with myself), and weight related maturity fears (e.g. I don't like changes in my body because they make me feel fat) (7). As such, population-specific barriers to PA that revolve around maturity should also be expected to be reported.

Paragraph Number 38: One plausible explanation for the lack of support for our hypothesis surrounding PA, barriers to PA and maturity status may revolve around the measure of BA used in our study versus perceptions of BA that may have existed by the study participants. That is, previous research (15) identifying the psychological traits and health behaviours of early and late maturing girls and boys have advocated the use of a subjective, self-report measure of BA. In such a measure, adolescents rate their pubertal development in relation to their peers. A subjective, self-report measure of physical maturity may be more closely related to the identification of specific maturity-related barriers to PA than an objective measure of physical maturity (as used in this study). An

objective measure of maturity may have no personal meaning to the adolescent; an early maturing girl who does not perceive herself as early maturing will likely not experience/report maturity-related barriers to PA as a girl who does perceive herself as early maturing. Future research would benefit from examining the discourse between a subjective, self-report measure and an objective measure of maturity and their association with PA and barriers to PA.

Paragraph Number 39: **Considerations:** When interpreting these results it is necessary to consider the limitations of identifying early and late maturing girls through within-sample comparisons (i.e. maturity quartiles (Q); Q1=early maturity, Q4=late maturity). Many studies (2,7,17,19,30) exploring the characteristics of early and late maturing adolescents use within sample comparison to classify individuals into maturity groups. This is likely because, in general, adolescent girls are average maturing, leaving only a minority presenting extreme maturity. For example, 97% of the present elementary school sample's APHV fell within the range of 11.0-12.0 years (mean = 11.80, SD= 0.48 years). It may be that within a grade at school it is only the few girls presenting a degree of physical maturity which is extremely different from their peers who are at heightened risk for disengagement from PA. Future research examining the PA and barriers to PA of extreme early and late maturing girls is warranted.

Paragraph Number 40: This study used an ecological approach in the study of barriers to PA. The advantage of an ecological approach is that a range of personal and situation barriers are captured, which may require different intervention strategies (14). For example, to alleviate the impact of salient intrapersonal barriers, which may be of particular relevance to high school students, one strategy might be to target an improvement in the

management of extra curricular time (e.g. paid work and homework). An additional advantage of the present study is the use of objective monitoring of PA. The limitations of self report measures of PA, in particular biases to do with accurate recalling of PA participation, are well recognized (20).

Paragraph Number 41: Limitations to this descriptive study must also be acknowledged. First, due to the cross sectional study design, information on the stability and variability of barrier categories and specific barrier types was not obtained. Second, one must not assume that because certain barriers were reported by a greater number of participants, that such barriers are the most salient. Rather, to capture saliency, both the frequency with which a specific barrier occurs as well as the extent to which a specific barrier limits PA must be assessed (see (4) for a review of this issue). Third, the results pertained to a select group of youth and thus may not be generalizable. Finally, biological maturity was assessed through predicting APHV among the elementary school girls and recalled age at menarche among the high school girls. Predicting APHV is likely less accurate than when observed in a longitudinal study and recalling age at menarche is subject to error.

CONCLUSION

Paragraph Number 42: Adolescent girls from grades 4-10 displayed a decrease in MVPA with increasing grade. However, when grouped by maturity status (early and late maturers) no differences were found in PA levels between maturity groups. Girls reported a multitude of barriers to PA within all grades. Types of barriers were dependent on grade but not maturity status. We therefore suggest that future research should aim to identify salient (i.e., frequent and limiting) personal and situational barriers to PA by CA in youth and

adolescents. Once reliably identified, multi-pronged intervention strategies to help youth and adolescents cope with their salient barriers can be tested for effectiveness.

ACKNOWLEDGEMENTS

The authors wish to thank Dale Esliger for his extensive advice on accelerometer data reduction and the Saskatoon Public School board for their participation in the research. The authors would also like to express appreciation for the helpful advice supplied by Dr. Nazeem Muhajarine. Sherar holds a Community and Population Health Research (CPHR) scholarship. The research was funded by a Saskatchewan Health and Research Foundation (SHRF) grant, a CPHR grant and a North American Society for Paediatric Exercise Medicine (NASPEM) grant. Results of the present study do not constitute endorsement by ACSM.

Reference List

1. Allison KR, Dwyer JJ, and Makin S. Perceived barriers to physical activity among high school students. *Prev. Med*. 1999;28:608-615.

2. Baker BL, Birch LL, Trost SG, and Davison KK. Advanced pubertal status at age 11 and lower physical activity in adolescent girls. *J. Pediatr*. 2007;151:488-493.

3. Bandura A. *Self-efficacy: The Exercise of Control*. New York, NY: WH Freeman; 1997. 282 p.

4. Brawley LR, Martin KA, Gyurcsik NC. Problems in assessing perceived barriers to exercise: confusing obstacles with attributions and excuses. In: Duda JL, editors. *Advances in Sport and Exercise Psychology Measurement*. Morgantown, WV: Fitness Information Technology, Inc; 1998. p. 337–50.

5. Brittain DR, Baillargeon T, McElroy M, Aaron DJ and Gyurcsik NC. Barriers to moderate physical activity in adult lesbians. *Women Health*. 2006;43:75-92.

6. Davison KK, Susman EJ, and Birch LL. Percent body fat at age 5 predicts earlier pubertal development among girls at age 9. *Pediatrics*. 2003;111:815-821.

7. Davison KK, Werder JL, Trost SG, Baker BL, and Birch LL. Why are early maturing girls less active? Links between pubertal development, psychological well-being, and physical activity among girls at ages 11 and 13. *Soc. Sci. Med*. 2007;64:2391-2404.

8. Esliger DW, Copeland JL, Barnes JD, and Tremblay MS. Standardizing and optimizing the use of acceloremeter data for free-living physical activity monitoring. *J Phys. Act. Health*. 2005;3:366-383.

9. Graber JA, Brooks-Gunn J, and Warren MP. The vulnerable transition: puberty and the development of eating pathology and negative mood. *Womens Health Issues*. 1999;9:107-114.

10. Gyurcsik NC, Spink KS, Bray SR, Chad K, and Kwan M. An ecologically based examination of barriers to physical activity in students from grade seven through first-year university. *J. Adolesc. Health*. 2006;38:704-711.

11. Hallal PC, Victora CG, Azevedo MR, and Wells JC. Adolescent physical activity and health: a systematic review. *Sports Med*. 2006;36:1019-1030.

12. Maddux JE, and Gosselin JT. Self-efficacy. In: Leary MR, Tangney JP, editors. *Handbook of self and identify*. New York: Guilford; 2003, pp.37-68.

13. Malina RM, Bouchard C, and Bar-Or O. Timing and sequence of changes during adolescence. In. Malina RM, Bouchard C, Oded Bar-Or, editors.*Growth, maturation and physical activity*. 2nd ed. Illionois: Human Kinetics;2004. p. 307-33.

14. McLeroy KR, Bibeau D, Steckler A, and Glanz K. An ecological perspective on health promotion programs. *Health Educ. Q*. 1988;15:351-377.

15. Michaud PA, Suris JC, and Deppen A. Gender-related psychological and behavioural correlates of pubertal timing in a national sample of Swiss adolescents. *Mol. Cell Endocrinol*. 2006;254-255:172-8.

16. Mirwald RL, Baxter-Jones AD, Bailey DA, and Beunen GP. An assessment of maturity from anthropometric measurements. *Med. Sci. Sports Exerc*. 2002;34:689-694.

17. Niven AG, Fawkner SG, Knowles A, and Stephenson C. Maturational differences in physical self-perceptions and the relationship with physical activity in early adolescent girls. *Pediat. Exerc Sci.* 2007;19:472-480.

18. Puyau MR, Adolph AL, Vohra FA, Zakeri I, and Butte NF. Prediction of activity energy expenditure using accelerometers in children. *Med. Sci. Sports Exerc*. 2004;36:1625-1631.

19. Riddoch CJ, Bo Anderson L, Wedderkopp N, Harro M, Klasson-Heggebo L, Sardinha LB, Cooper AR, and Ekelund U. Physical activity levels and patterns of 9- and 15-yr-old European children, *Med. Sci. Sports Exerc*. 2004;36:86-92.

20. Shephard RJ. Limits to the measurement of habitual physical activity by questionnaires. *Br. J. Sports Med*. 2003;37:197-206.

21. Sherar LB, Baxter-Jones A.D and Mirwald RL. Limitations to the use of secondary sex characteristics for gender comparisons. *Ann. Hum. Biol*. 2004; 31:586-593.

22. Sherar LB, Esliger DW, Baxter-Jones AD, and Tremblay MS. Age and gender differences in youth physical activity: does physical maturity matter? *Med. Sci. Sports Exerc*. 2007;39:830-835.

23. Thompson AM, Baxter-Jones AD, Mirwald RL, and Bailey DA. Comparison of physical activity in male and female children: does maturation matter? *Med. Sci. Sports Exerc*. 2003;35:1684-1690.

24. Troiano RP, Berrigan D, Dodd KW, Masse LC, Tilert T, and McDowell M. Physical activity in the United States measured by accelerometer. *Med. Sci. Sports Exerc*. 2008;40:181-188.

25. Trost SG, Kerr LM, Ward DS, and Pate RR. Physical activity and determinants of physical activity in obese and non-obese children. *Int. J. Obes. Relat. Metab. Disord*. 2001;25:822-829.

26. Trost SG, Pate RR, Freedson PS, Sallis JF Taylor WC. Using objective physical activity measures with youth: how many days of monitoring are needed? *Med. Sci. Sports Exerc.* 2000;32:426-431.

27. Trost SG, Pate RR, Sallis JF, Freedson PS, Taylor WC, Dowda M, and Sirard J. Age and gender differences in objectively measured physical activity in youth. *Med. Sci. Sports Exerc*. 2002;34:350-355.

28. U.S. Department of Health and Human Services. 2000 *CDC Growth charts for the United States: Methods and development* (Rep. No. 246). Washington, DC: Department of Health and Human Services; 2002. 28 p.

29. van Jaarsveld CH, Fidler JA, Simon E. and Wardle J. Persistent impact of pubertal timing on trends in smoking, food choice, activity, and stress in adolescence. *Psychosom. Med*. 2007;69:798-806.

30. Wickel EE. and Eisenmann JC. Maturity-related differences in physical activity among 13-14- year old adolescents. *Pediatr. Exerc. Sci*., 2007;19:384-392.

31. Woodgate J, Brawley LR, and Weston ZJ. Maintenance of cardiac rehabilitation exercise adherence: effects of task and self-regulatory self-efficacy. *J App. Soc. Psych*. 2005;35:183-222.

Figure Legends

Figure 1: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by grade groupings. Note. Percentages for each grade group may not equal 100 due to rounding error.

Figure 2: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the elementary school girls. Note. Percentages for each grade group may not equal 100 due to rounding error.

Figure 3: Percentage of barriers reported that were intrapersonal, interpersonal, institutional and community barriers by maturity groupings for the high school girls. Note. Percentages for each grade group may not equal 100 due to rounding error.

Table 1: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for girls in different grade groups

	Grade Grouping		
Measure	Grades 4-6 (n=58)	Grades 7-8 (n=43)	Grades 9-10 (n=81)
Age (yrs)	10.68 (0.91)*	13.12 (0.64)*	15.28 (0.63)*
Weight (kg)	39.36 (9.71)*	51.72 (9.95)*	62.49 (13.17)*
Height (cm)	145.13 (9.0)*	159.56 (7.30)*	164.86 (6.17)*
BMI (kg/m^2)	18.47 (3.16)	20.27 (3.53)	22.94 (4.41)*
Sum 5 SF (mm)	53.46 (24.02)*	55.43 (20.22)*	77.48 (30.89)*
Accelerometer Counts/day	758297.8 (306646.4)*	568840.4 (302541.3)*	424430.4 (191799.40)*
MVPA (average min/d)	151.96 (57.75)*	113.6 (57.11)*	84.48 (34.67)*

*= significantly ($P<.05$) different from the previous grade grouping

Table 2: Mean (SD) anthropometric and physical activity variables (accelerometer counts, MVPA) for early and late maturing elementary and high school girls

School	Measure	Maturity Status	
		Early Maturers (n=25)	Late Maturers (n=25)
Elementary (Grades 4-8)	Age (yrs)	11.46 (1.38)	12.22 (1.32)
	Weight (kg)	49.23 (10.67)*	38.73 (9.93)
	Height (cm)	157.95 (10.21)*	145.7 (10.17)
	BMI (kg/m^2)	19.65 (3.63)	17.94 (2.55)
	Sum 5 SF	55.41 (24.6)	46.92 (16.05)
	Accelerometer counts/day	803623 (318242)	685021 (357441)
	MVPA (average min/d)	158.86 (58.08)	138.55 (68.38)
		Early Maturers (n=19)	Late maturers (n=19)
High (Grades 9-10)	Age (yrs)	15.34 (0.63)	15.44 (0.59)
	Weight (kg)	66.71 (12.62)	60.38 (14.24)
	Height (cm)	166.88 (5.09)	166.08 (4.74)
	BMI (kg/m^2)	23.92 (4.2)	21.82 (4.67)
	Sum 5 SF	82.05 (26.77)	66.14 (29.76)
	Accelerometer counts/min	385946 (183137)	455391 (257523)
	MVPA (average min/d)	74.97 (30.68)	89.23 (47.19)

* = significant ($P < .05$) difference between early and late maturing girls

Table 3: Types of intrapersonal barriers listed by the participants

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Illness/Injury	46% (n=10)	24% (n=6)	25% (n=19)
Lack of time	23% (n=5)	28% (n=7)	9% (n=7)
Lack of motivation/Lazy	5% (n=1)	16% (n=4)	29% (n=22)
No Money to buy equipment or access facilities	14% (n=3)	16% (n=4)	15% (n=11)
Was doing something else	14% (n=3)	12% (n=4)	14% (n=3)
Paid Work	-	-	27% (n=2)
Didn't want to/feel like it	-	-	7% (n=5)
[a]**Other**	-	4% (n=1)	-
Total Reported Frequency	22	25	75

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Table 4: The interpersonal barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Friends did not want to go with me	18% (n=7)	17% (n=6)	33% (n=18)
A person in authority (e.g. parent or older sibling) said no to activity	67% (n=26)	54% (n=19)	56% (n=30)
Coaches/teachers altered the scheduling of the activity	13% (n=5)	11 (n=4)	4% (n=2)
I could not get a ride	-	9 (n=3)	7% (n=4)
[a]**Other**	3% (n=1)	9 (n=3)	-
Total reported frequency	39	35	54

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Table 5: Institutional barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
School phys ed or extra-curricular activity scheduling	60% (n=6)	20% (n=2)	20% (n=10)
The timing of other organized activities conflicted	20% (n=2)	30% (n=3)	14% (n=7)
Too much homework to do activity	-	20% (n=2)	55% (n=27)
[a]**Other**	20% (n=2)	30% (n=3)	10% (n=5)
Total	10	10	49

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping.

[a]Other barriers were those cited five or less times

Table 6: Community barriers to physical activity

Type of barrier	Grades 4-6	Grades 7-8	Grades 9-10
Weather is too cold/Snowing	83% (n=5)	68% (n=8)	75% (n=21)
The community facility is too far away	17% (n=1)	17% (n=2)	18% (n=5)
[a]**Other**	-	17% (n=2)	7% (n=2)
Total	6	12	28

Note. The numbers in parentheses represent the number of times the barrier was cited in the sample by grade grouping. [a]Other barriers were those cited five or less times

Figure 1

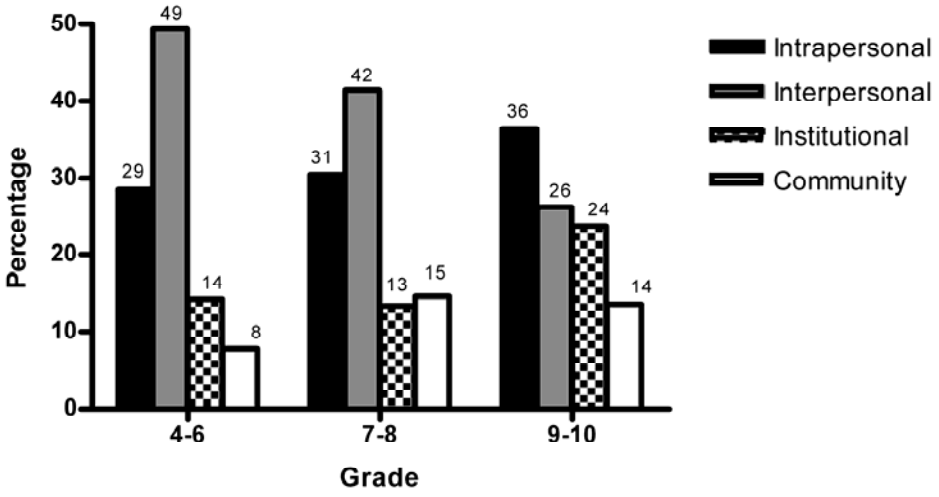

Percentage
50
40
30
20
10
0
49
42
36
29
31
26
24
14
13
15
14
8
4-6
7-8
9-10
Grade
Intrapersonal
Interpersonal
Institutional
Community

Figure 2

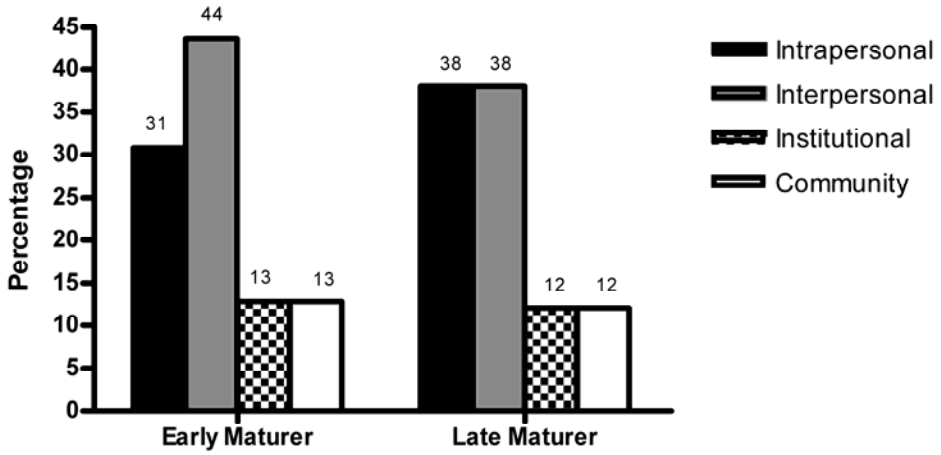
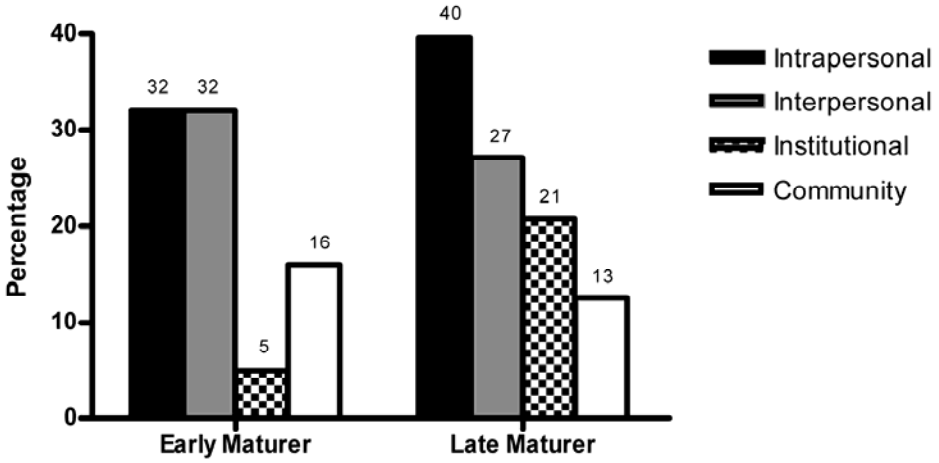

45
40
35
30
25
20
15
10
5
0
Percentage
44
31
13
13
38
38
12
12
Early Maturer
Late Maturer
Intrapersonal
Interpersonal
Institutional
Community

Figure 3


40
30
20
10
0
Percentage
32
32
5
16
40
27
21
13
Early Maturer
Late Maturer
Intrapersonal
Interpersonal
Institutional
Community