Socioeconomic and developmental factors from early life and leisure-time physical activity across adulthood

Ahmed Ismail Elhakeem

Thesis submitted for the Degree of Doctor of Philosophy

December 2016





I, Ahmed Ismail Elhakeem, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Abstract

Leisure-time physical activity (LTPA) benefits heath therefore, it is important to understand which factors might influence LTPA. Studies link early life factors with adult health and behaviours but their associations, particularly developmental factors, with LTPA are unclear. Further, examining if associations found change with age may shed light on underlying mechanisms.

The aim of this thesis was to examine associations of socioeconomic and developmental factors from early life with LTPA across adulthood.

Published studies were systematically reviewed to examine associations between childhood socioeconomic position (SEP) and adult LTPA. Remaining objectives were addressed using data from up to 3545 participants from the MRC National Survey of Health and Development (NSHD). Prospectively collected data from early life used in analyses were birth weight, infant motor milestones, ability at school games, upper and lower limb motor coordination and pubertal development. LTPA was self-reported five times between ages 36 and 68. Associations were examined using standard and mixed-effects binary and multinomial logistic regression. Age by early life factor interactions tested if associations varied by age at assessment of LTPA.

Among 36 published studies identified, lower childhood SEP (most commonly indicated by parental occupation and education) was associated with less LTPA in adulthood (particularly among women and in UK cohorts) but there was considerable heterogeneity between studies. In the NSHD, low birth weight, lower ability at games and slower tapping speed in adolescence were associated with lower likelihood of participation in LTPA across adulthood and these associations did not vary by age. There was some suggestion that early maturing boys and later maturing girls were more likely to participate in LTPA in adulthood but this evidence was weak.

Socioeconomic and developmental factors from early life were associated with LTPA across adulthood. The main implications of these findings are that those with low birth weight, less motor competence and lower SEP may require additional support to take up and maintain LTPA across adulthood.

Acknowledgements

Thank you to my primary supervisor Professor Rebecca Hardy and secondary supervisor Dr Rachel Cooper for their immensely helpful comments, discussion and advice throughout my studies. Thank you also to my third supervisor Dr David Bann and to Professor Diana Kuh for their invaluable input on my work. I am also grateful to staff and students at the MRC Unit for Lifelong Health and Ageing at UCL for the helpful discussion during my PhD studies, and especially to Adam Moore and Karen Mackinnon for helping with my data queries and requests. I also thank the study members of the MRC National Survey of Health and Development for their lifelong contribution to this exceptional scientific resource, and the MRC for funding my research. I am dedicating this thesis to my parents Drs Somaya Khalifa and Ismail Elhakeem.

Contents

Abstract	3
Acknowledgements	4
List of Tables	8
List of figures	10
Abbreviations	10
Chapter 1: Introduction	11
1.1 Terminology and key definitions	11
1.2 The importance of physical activity to health	12
1.3 Methodological challenges of measuring physical activity	
1.4 Age and time trends in physical activity	16
1.5 What influences participation in physical activity?	17
1.6 Towards a life course approach	18
1.6.1 Life course perspectives	18
1.6.2 Life course approach to physical activity	20
1.6.3 Socioeconomic and developmental factors from early life and leisure-time phy activity across adulthood	
1.7 Aim, objectives and hypotheses	21
1.8 Structure of thesis	22
Chapter 2: Data and methods	25
2.1 Introduction to the MRC National Survey of Health and Development (NSHD)	25
2.1.1 Historical context of the NSHD	26
2.1.2 Response rates, loss to follow-up and characteristics of non-responders	26
2.2 Leisure-time physical activity across adulthood	30
2.2.1 Measurement and operationalisation of leisure-time physical activity	30
2.2.2 Patterns of LTPA from ages 36 to 68 years	33
2.2.3 Types of LTPA reported at ages 36 and 60-64	35
2.2.4 Physical activity measured by monitors at age 60-64	39
2.3 Methods and analytical strategy	40
2.3.1 General analytical strategy	40
2.3.2 Using mixed-effects models to examine associations with adulthood LTPA	41
Chapter 3: Childhood socioeconomic position and leisure-time physical activity across adulthood	45
3.1 Background	45
3.2 Methods	46
3.2.1 Eligibility criteria	46
3.2.2 Search strategy	47
3.2.3 Study selection	47
3.2.4 Data extraction	47
3.2.5 Quality assessment	48

3.2.6 Synthesis	48
3.3 Results	48
3.3.1 Characteristics of Included studies	49
3.3.2 Results of included studies	50
3.4 Discussion	54
3.4.1 Summary of results	54
3.4.2 Explanation of findings	55
3.4.3 Sources of heterogeneity	57
3.4.4 Implications of findings	58
3.4.5 Strengths and limitations of the review	58
3.4.5 Conclusions	59
Chapter 4: Birth weight and leisure-time physical activity across adulthood	94
4.1 Background	94
4.1.1 Literature review	94
4.1.2 Chapter aim and hypothesis	100
4.2 Methods	103
4.2.1 Explanatory variable	103
4.2.2 Confounding variables	103
4.2.3 Examining associations with LTPA across adulthood	104
4.3 Results	104
4.3.1 Relation of birth weight to covariates	104
4.3.2 Associations between birth weight and LTPA across adulthood	105
4.4 Discussion	110
4.4.1 Summary of results	110
4.4.2 Comparison with other studies	110
4.4.3 Explanation of findings	111
4.4.4 Methodological considerations	115
4.4.5 Implications of findings	115
4.4.6 Conclusions	116
Chapter 5: Motor performance in early life and leisure-time physical activity acros	
5.1 Background	
5.1.1 Literature review	
5.1.2 Chapter aim and hypotheses	
5.2 Methods	
5.2.1 Explanatory variables	
5.2.2 Confounding variables	
5.2.3 Examining associations with LTPA across adulthood	
5.3 Results	
5.3.1 Relation of motor performance measures to covariates	

5.3.3 Age at reaching motor milestones and LTPA across adulthood	. 132
5.3.4 Ability at games and LTPA across adulthood	. 137
5.3.5 Tapping speed and LTPA across adulthood	140
5.4 Discussion	144
5.4.1 Summary of findings	144
5.4.2 Comparison with other studies	144
5.4.3 Explanation of findings	. 145
5.4.4 Methodological considerations	. 147
5.4.5 Implications of findings	. 148
5.4.6 Conclusions	149
Chapter 6: Age at puberty and leisure-time physical activity across adulthood	. 150
6.1 Background	. 150
6.1.1 Literature review	. 150
6.1.2 Chapter aim and hypotheses	. 155
6.2 Methods	. 157
6.2.1 Explanatory variables	. 157
6.2.2 Confounding variables	. 158
6.2.3 Examining associations with LTPA across adulthood	. 159
6.3 Results	. 159
6.3.1 Age at puberty in relation to selected covariates	. 159
6.3.2 Pubertal status at age 15 in boys and LTPA across adulthood	. 160
6.3.3 Age at menarche and LTPA across adulthood	. 163
6.4 Discussion	. 166
6.4.1 Summary of findings	. 166
6.4.2 Comparison with other studies	. 166
6.4.3 Explanation of findings	. 167
6.4.4 Methodological considerations	. 169
6.4.5 Implications of findings	. 169
6.4.6 Conclusions	. 169
Chapter 7: Discussion	171
7.1 Summary of main findings	171
7.1.1 Do developmental factors help explain early life socioeconomic differences in LTPA across adulthood	173
7.2 Implications of findings	. 176
7.3 Methodological considerations	178
7.4 Recommendations for future research	. 182
Thesis publications	. 185
Appendices	186
References	241

List of Tables

Table 1.1 Factors associated with leisure-time physical activity (LTPA) – adopted
from Bauman et al. 2012 (68
Table 2.1 Attrition from ages 36 to 68 and the number of subjects at each age29
Table 2.2 Characteristics of those with and without any LTPA data between ages 36
and 68 years by early life factor
Table 2.3 Leisure-time physical activity information reported by NSHD
participants32
Table 2.4 Proportions % (N) of inactive, moderately active and regularly active in
LTPA at each age in adulthood overall and by sex
Table 2.5 Proportions in each possible trajectory of active (1) or inactive (0) in LTPA
from ages 36 to 68 years overall and by sex35
Table 3.1 Characteristics of the included studies: arranged by region/country and
from older to younger age at measurement of physical activity60
Table 3.2 Results of studies testing the association between parents' occupational
class and leisure-time physical activity (LTPA) in adults: arranged by region/country
and from older to younger age at measurement of physical activity70
Table 3.3 Results of studies testing the association between parents' education and
leisure-time physical activity (LTPA) in adults: arranged by region/country and from
older to younger age at measurement of physical activity82
Table 3.4 Results of studies testing the association between indices and other
measures of childhood socioeconomic position and leisure-time physical activity
(LTPA) in adults: arranged by region/country and from older to younger age at
measurement of physical activity
Table 4.1 Factors associated with rates of intrauterine growth
Table 4.2 Summary of studies examining the association between birth weight and
physical activity: arranged by age at assessment of activity101
Table 4.3 Distribution of birth weight overall and by sex in those with data on
covariates and at least one measure of LTPA (n=3545)105
Table 4.4 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-time
physical activity (LTPA) between ages 36 and 68 years by birth weight group: mixed-
effects binary logistic regression107
Table 4.5 Relative risk ratios (RRR) and 95% credible intervals (95% Crl) of
moderate and regular leisure-time physical activity (LTPA) between ages 36 and 68
years by birth weight group: mixed-effects multinomial logistic regression109

 Table 5.1 Summary of studies examining associations between motor performance
 in early life and physical activity: arranged by age at assessment of physical Table 5.2 Distribution of motor performance measures overall and by sex in those with data on covariates and at least one measure of LTPA......131
 Table 5.3 p-vales for guadratic terms for age at reaching motor milestones.......132
 Table 5.4 Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisure-time physical activity (LTPA) between ages 36 and 68 years by age at reaching infant Table 5.5 Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% Crl) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by age at reaching infant motor milestones: mixed-effects Table 5.6 Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisure-time physical activity (LTPA) between ages 36 and 68 years by tapping speed at age 15: mixed-effects binary logistic regression......141 Table 5.7 Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% Crl) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by tapping speed at age 15: mixed-effects multinomial logistic regression......143 Table 6.1 Summary of studies examining associations between age at puberty and physical activity in adults: arranged by age at assessment of physical activity.....156 Table 6.3 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-time physical activity (LTPA) between 36 and 68 years by age at menarche: mixed-effects binary logistic regression......164 Table 6.4 Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% Crl) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by age at menarche: mixed-effects multinomial logistic regression......165 Table 7.1 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-time physical activity (LTPA) between ages 36 and 68 years by father's occupational class Table 7.2 Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% Crl) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by father's occupational class age 4: mixed effects multinomial

List of figures

Figure 1.1 Conceptual model for thesis24
Figure 2.1 Flow-chart of participant selection28
Figure 2.2 Proportions (%) taking part in LTPA at each age in adulthood by sex34
Figure 2.3 Proportions (%) taking part once or more than once in each sport, outdoor
activity and exercise in the previous month at age 36 by sex
Figure 2.4 Numbers of men and women taking part in team sports and non-team
sports at least once per month at age 3637
Figure 2.5 Proportions (%) taking part once or more than once each sport, outdoor
activity and exercise in the previous year at age 60-64 by sex
Figure 3.1 PRISMA study flowchart (search up to 15th Dec 2014)49
Figure 3.2 Hypothesised pathways underlying associations found between childhood
socioeconomic position and adult leisure-time physical activity
Figure 4.1 Log-odds of leisure-time physical activity for each birth weight group by
age in men and women108
Figure 4.2 Hypothesised pathways underlying associations found between birth
weight and adult leisure-time physical activity114
Figure 5.1 Development of Independent walking119
Figure 5.2 Log-odds of leisure-time physical activity at each adult age by ability in
school games at age 13 in men and women139
Figure 6.1 Log-odds of LTPA across adulthood by pubertal status at age 15 in
boys163

Abbreviations

LTPA = Leisure-time physical activity MVPA = Moderate-to-vigorous physical activity MRC NSHD = Medical Research Council National Survey of Health and Development PA = physical activity PAEE = Physical activity energy expenditure PRISMA = Preferred reporting items for systematic reviews and meta-analyses SEP = Socioeconomic position OR = Odds ratio RRR = Relative risk ratio

Chapter 1: Introduction

Leisure-time physical activity (LTPA) is an important modifiable health behaviour implicated in the prevention of chronic disease and the promotion of health and mental well-being. Understanding which factors from early life might influence LTPA may help inform the design of effective interventions to promote LTPA but few studies have investigated how early life factors might relate to adult LTPA. These have mostly focused on early life socioeconomic factors and fewer still have investigated developmental factors. The aim of this thesis is to use a life course approach to examine the associations between socioeconomic and developmental factors from early life and participation in LTPA across adulthood, by systematically reviewing published studies and using data from the MRC National Survey of Health and Development (NSHD) – a British cohort of 5362 males and females followed up since birth in March 1946.

This chapter presents a broad overview of the research area investigated in this thesis, including evidence for the importance of physical activity to health. The life course epidemiology approach is then introduced and followed by an overview of previous research investigating factors from early life in relation to adult LTPA. The limitations of previous research are then briefly described along with the overall aims of this thesis.

1.1 Terminology and key definitions

Physical activity (PA) refers to bodily movement originating in skeletal muscles and leading to energy expenditure (1). It is a multidimensional construct consisting of occupation, transport, domestic, and leisure-time domains (2). While the different domains of PA may appear self-explanatory, it is worth defining leisure-time physical activity (LTPA) as that which is done during free time out of interest (3). LTPA in turn comprises several subcomponents including exercise, i.e. planned, repetitive and purposeful PA (1) and sports, i.e. PA that is structured, organised and often competitive. The different subtypes of LTPA can vary widely in their duration and intensity of PA required (1, 3).

To understand the health effects of PA and investigate what influences participation, researchers typically quantify PA. Some of the most commonly used parameters are frequency, intensity and duration. Frequency is one of the simplest and most

commonly used ways of quantifying PA and describes the number of times a particular activity is done in a specific time period (e.g. during the previous week). Intensity refers to the work load associated with PA and is usually measured in multiples of the resting metabolic rate, referred to as metabolic equivalents of tasks (METs) (3). The duration of PA is required for calculating METs and refers to the length of time for which any single session of PA lasts (1, 3). PA energy expenditure is a less commonly used measure of PA as it is more difficult to measure and is calculated as the total energy expended per unit time minus the amount of energy expended at rest (3).

Reductions in total energy expenditure over past decades coupled with decreasing occupational PA and increasing screen time, especially in high income countries, has also promoted research interest in the health consequences of sedentary behaviour; where energy expenditure is minimal and prolonged lying/sitting is the dominant posture (4). Evidence suggests that the adverse consequences of sedentary behaviour are distinct from simply not performing sufficient PA i.e. being physically inactive (4). There may be different pathways leading to the development of sedentary behaviour compared with PA as well as differences in associations with health outcomes (5). Finally, a person's ability to be physically active is often described by their physical fitness which includes the components of cardiorespiratory fitness (ability of the circulatory and respiratory systems to support sustained PA) and endurance or the ability to continuously perform PA for a set period of time (1).

1.2 The importance of physical activity to health

Less than a century ago PA was thought by some to contribute to premature mortality (6) and cardiovascular disease (7). However, attitudes have improved since the Second World War and substantial evidence in disciplines ranging from exercise biology to behavioural sciences suggests that PA is a powerful modifiable factor implicated in the prevention of chronic diseases and the promotion of health and mental well-being across life (8). Reviews and meta-analyses have linked higher levels of moderate to vigorous intensity PA with a lower cardiovascular disease (CVD) risk (9), and found a 33% risk reduction for all-cause mortality (95% CI: 28-37) and a 35% risk reduction for mortality from CVD (95%CI: 30-40) among physically active participants (10). Other systematic reviews indicate that higher PA is associated with a reduced cancer risk including those of renal, bladder and gastro oesophageal origins (11-13).

Now that people are living on average longer than ever before, PA is likely to continue playing an increasingly important role in the maintenance of physical capability and independent living in later life (14), thus reducing demand for social, health and other related services. This is supported by evidence from a review showing an overall association among 22 prospective studies between higher PA and lower risk of bone fractures (15), and a review of randomised trials showing the potential for exercise to lower the risk of falls in older populations (16). Regular PA has also been shown to benefit cognitive functioning and to reduce age-related cognitive decline in older adults (17-19). While research suggests the health effects of higher intensity PA follow a dose-response relationship for level of intensity (20), advancements allowing the measurement of low intensity PA indicate such benefits may accrue even at lower intensities which may be particularly important for older populations (21).

Alongside the evidence showing the health benefits of PA, a substantial body of literature has highlighted the negative impact of not performing sufficient PA. Being physically inactive has been described as a greater risk factor for CVD than either high cholesterol or hypertension (22), having a disease burden that is comparable to smoking (23), and as being the fourth leading cause of premature mortality worldwide (8). Research suggests that sedentary behaviour may also be a risk factor for adverse health outcomes including all-cause and chronic disease mortality, irrespective of PA levels (24). However, research has not been consistent (25) with a recent meta-analysis suggesting that PA may considerably reduce the detrimental effects of sedentary behaviour (26). Nevertheless, national and international PA guidelines were developed based on evidence like those described above to encourage PA as well as to promote less sedentary time. For example, current UK PA guidelines for adults encourage the accumulation of at least 150 minutes of moderate intensity PA (or 75 minutes of vigorous intensity) and 2 sessions of strength training per week in addition to minimising time spent sitting (27).

In addition to evidence showing the benefits of PA overall, it has been shown that associations between PA and health differ depending on which PA domain is considered. For example, a prospective study of almost 40,000 participants found stronger associations between higher LTPA and a lower risk of heart failure than for higher total PA (Hazard ratios of heart failure comparing high v low LTPA and high v low total PA: 0.54 [95%CI: 0.44-0.66] and 0.81 [95%CI: 0.69-0.95]) (28). Conflicting associations between occupational PA and LTPA and health outcomes have also been reported. A meta-analysis of prospective studies found more LTPA to be associated with a reduced CVD risk while higher occupational PA was linked to a slightly higher risk (9). However, it could be that more recent findings on occupational PA are explained by confounding. For example, consistent evidence suggests that lower socioeconomic position is related to higher occupational PA (29), and lower socioeconomic position is also associated with higher CVD risk (30). Elsewhere, higher LTPA was associated with better health-related quality of life but associations in the opposite direction were reported for higher domestic and transport-related PA (31). Similar contrasting associations were reported for mobility limitations at old age in a 28-year follow-up study from Finland (32) and for all-cause mortality in a Danish study (33).

The examples cited above suggest that of the different domains of PA the one that is more strongly and consistently associated with health outcomes is LTPA. However, findings may be more consistent for LTPA because of confounding in other domains described above, e.g. socioeconomic position (SEP) and occupational PA (29). Nevertheless, the benefits of LTPA have been reported for a range of different subtypes including an association between more frequent running and lower allcause mortality (34) and more leisure time walking and lower risk of stroke (35), in addition to the potential of strength training to improve mobility and prevent muscle loss in older populations (36). Different types of LTPA might also have different effects on health (37) for example, high impact PA might promote osteogenesis (38) whereas in contrast, swimming has little direct effect on bones (39). LTPA also makes up the majority of time spent in moderate and vigorous intensity PA (40, 41) and may be easier to maintain than other domains (for example, active commuting may change with changing circumstances like distance to work). In addition, as described below in section 1.4, there is evidence from various studies that the prevalence of LTPA has increased in recent decades (42-46). Therefore this suggests that LTPA is potentially more amenable to intervention across all life stages than activity in other domains such as occupational PA (47) and may thus be most informative from a public health intervention perspective.

1.3 Methodological challenges of measuring physical activity

A wide range of methods have been used to assess PA. Some such as the ingestion of doubly labelled water to measure activity-related energy expenditure, and direct observation of individuals can provide precise estimates of PA, but are not generally feasible for use in large population studies (48). Instead, most of what has been learned about PA over the past half century has relied on self-reports of PA through questionnaires, diaries or interviews. Despite their limitations, such methods are reasonably valid and widely accepted as the cheapest and the most convenient way to measure PA in large population studies (49-51).

The collection of self-reported PA involves study members recalling PA that they participated in over a specified period of time (e.g. the previous week, month or year). This makes them particularly useful at capturing parameters such as the frequency and types of easy to recall structured movements in activities like exercise and sports, especially those of a moderate or high intensity (37-39). However, such methods are associated with errors of self-reporting e.g. selective over reporting of information and the known tendency of some participants to provide what they perceive as socially acceptable responses (37-39). In addition, there are recall errors, the magnitude of which can vary depending on the time period considered (37-40). Another limitation of self-reports is their poor assessment of time spent in different intensities of PA; and in particular time spent in light intensity activities (49-53). The latter may be especially problematic in older populations who are likely to spend a lot of their time in light intensity PA, and who may also be more likely to have recall difficulties (52). The lack of national guidelines for light intensity PA has been cited as a direct result of the inability of self-report methods to measure time spent in light intensity PA (54).

Recent years have seen rapid advances in the development of monitors to measure PA, with decreasing costs and improved sophistication of instruments such as accelerometers and heart rate monitors fuelling their everyday use in PA research (37-42). Such monitors address some of the limitations associated with self-reports (e.g. recall bias) and can yield more precise estimates of light intensity PA and inactivity (37-42). Methods for directly assessing PA are especially useful for identifying whether the intensity or duration (but not type) of PA might explain underlying mechanisms (37-42). However, objective methods for the assessment of PA are not without limitations. These include their general unsuitability for some types of PA such as bathing and swimming (although newer waterproof monitors are emerging), inability to delineate domain specific PA (e.g. LTPA), and if worn on the

hip then arm or leg movements (e.g. cycling) are usually not captured (37-42). More recent developments in the field have included the use of combined body movement and heart rate monitors to predict energy expenditure from PA (37-42). Future advances in the analysis and access to raw acceleration data could lead to improvements in the precision of PA data, and may help to provide better measures of energy expenditure but these will require means of validation (37-42). Contrary to some suggestions, the increasing availability and affordability of objective methods of assessing PA should not be viewed as a replacement to self-reported PA. The fact that self-reported and direct measures of PA tend to produce different estimates of PA levels (with a tendency for overestimation of PA by self-reports (55-57), may be due to each method of assessment detecting features of PA that are not easily captured by the other (37-45). Most of the scientific evidence base surrounding PA has been developed on self-reported data, and despite differences in absolute level, both methods have been found to rank people in a similar fashion on their levels of PA (43-45). Therefore, studies should be encouraged which use both types of measurements to take advantage of their respective strengths and provide more holistic information on PA domains, types, energy expenditure, and time spent in different intensities (58).

1.4 Age and time trends in physical activity

Levels of overall PA in populations have declined in recent decades (44, 46), mainly as a result of more sedentary work, transport and home environments. However, the proportion of the population taking part in LTPA has increased over recent decades across all age groups thus supporting its amenability to change (42-46). Age-related drops in PA levels are generally observed across the life course including from childhood into adolescence (59) as well as from adolescence into adulthood (60) and across adulthood into old age (53). It is possible that these longitudinal changes in PA vary between certain groups e.g. SEP although little research has investigated this. Geographic variations such as higher PA in urban compared with rural areas (61) and in Northern compared with Southern European countries (62) have also been documented.

Despite LTPA levels increasing over time, there is much room for improvement. Recent figures from the WHO suggest that 31% of adults worldwide are inactive (defined as no PA), with estimates rising to 50% in the US and Eastern Mediterranean regions (63). In the UK, findings from the latest Health Survey for England show only 52% of men and 45% of women reported participating at least once in sports or exercise during the previous four weeks (64). Statistics compiled by the British Heart Foundation showed that on average, English men spent 2.1 hours and women 1.2 hours per week in sports and exercise in 2012 (65). In a nationally representative UK survey of 70-93-year-old men and women who provided accelerometer estimates of PA, not more than 15% met current guidelines for moderate to vigorous PA (66). Results from the NSHD (1946 British birth cohort) showed that while 67% of the 60-64 year old participants reported partaking in cardiorespiratory activities; only 11% reported taking part in strength training (weights and conditioning exercises) on a regular basis (67); contrary to national PA guidelines which encourage a mixture of both types (27).

1.5 What influences participation in physical activity?

A wide range of factors including individual and environmental characteristics have been shown to be associated with PA levels (Table 1.1). The non-modifiable factors of age and sex have been consistently linked with overall PA, with male and younger compared with female and older adult age being associated with more PA (68). Being in better health has also been associated with more frequent LTPA (68). Hereditary components to PA have been illustrated by twin and family studies, and the role of key brain structures in motivation, reward, and energy balance have also been explored (69). Research also indicates there may be different drivers for different domains of PA. For example, those already physically active during leisuretime are more likely to initiate and maintain active transport choices such as walking and cycling (70).

Genetic differences in response to exercise, socioeconomic and cultural backgrounds, the availability of social support, and previous LTPA levels have all been identified as correlates of LTPA (68). Interest in environmental influences on PA has implicated neighbourhood characteristics. Aspects of the physical and perceived environment such as access to exercise facilities, aesthetics and perceived safety have been associated with LTPA (71), although the evidence is overly reliant on cross-sectional studies and as such biases like self-selection (where those more active move to active-friendly locations) cannot be easily ruled out. Key life stages, events and transitions (e.g. puberty, parenthood, retirement) are strongly associated with changes in LTPA (72) although their long-term impact on later LTPA is less clear. For example, a study where identical repeated questions were asked about

LTPA at four ages around retirement found that levels initially increase during the transition to retirement and subsequently decrease (73).

A large selection of theories have been used to help understand and promote PA including those emphasising planned behaviour, reasoned action and self-determination (74), and they usually try to exploit known intrapersonal associates of PA such as motives, attitudes and intentions to try and explain participation in PA. The trans-theoretical model (75) attempts to describe PA participation in terms of a transition through stages of behaviour change. Other interventions based on ecological models that emphasise the multilevel influences on behaviour and their interactions (e.g. individual with their living environment) have had some success (76) although generally interventions are limited in their ability to change PA. Reviews of various interventions aiming to change PA in children (77) and adults (78, 79) including those with a specific focus on older adults (80) generally find only small effects which are usually short lived. In addition, little is known regarding long-term effectiveness of PA interventions, i.e. >24 months follow-up studies.

Table 1.1 Factors associated with leisure-time physical activity (LTPA) – adopted from Bauman et al. 2012 (68).

Individual	Interpersonal	Environment and policy
 Psychological factors e.g. stress Biological and genetic factors e.g. age, sex, health, genotype Intrapersonal characteristics e.g. motivation, beliefs, self- efficacy -socioeconomic position Life events and transitions Previous LTPA 	 Social support Cultural norms and practices Behavioural modelling from seeing others active 	 Physical environment e.g. availability and aesthetical quality of parks and green space, Perceived environment e.g. perceived safety of parks and neighbourhoods. Policy e.g. sports funding, urban planning

1.6 Towards a life course approach

1.6.1 Life course perspectives

The difficulties associated with initiating, changing, and maintaining PA have stimulated interest in alternate approaches to promoting participation in LTPA like those attempting to understand the origins and development of PA over the life course (81). In recent decades, a life course approach has been used in epidemiology to counter the polarisation of research into adult risk factors and early life experiences that occurred in epidemiology in the early 1990s by combining the two areas to help understand the long-term influences of social, biological, and behavioural factors from early life in combination with later life factors on future health and disease risk (82).

In addition to identifying the pathways involved, a life course approach attempts to understand whether those factors act independently, cumulatively or interact with each other to contribute to the development of disease risk and health behaviours. To achieve this, life course epidemiologists have developed and tested theoretical models that attempt to explain the pathways between the exposures of interest and subsequent outcomes. A central feature to this model building and testing approach is the correct placement of relevant exposures across a person's life time, which often requires the use of longitudinal studies such as birth cohorts (83). The more commonly tested models of life course epidemiology include the critical/sensitive periods and accumulation of risks models.

Sensitive period models describe how exposures during a specific period of time will have a stronger influence on the development of health-related outcomes than exposures outside that period (71, 72). In critical period models exposures during a limited time window can have adverse or protective effects on later health, but they have no effect outside this period (71, 72). The foetal origins of disease hypothesis (84) is an example of a critical period model which initially suggested that intrauterine exposures can permanently alter development and predisposition to later risk of disease. This theory later expanded to include infant and childhood exposures becoming the developmental origins hypothesis (71-73). The accumulation of risk model was initially proposed to explore how separate exposures act either independently or in clusters to have a long-term influence on later health outcomes. However, it has most often been used to investigate if the same exposure (e.g. socioeconomic circumstances) at different time points leads to an accumulation of risk (71, 72). In addition, trajectories of function and behaviour over time are important, more recent developments in life course epidemiology that can help identify those at risk of developing disease and may shed light on the timing of interventions by helping identify potential sensitive periods (85-90).

1.6.2 Life course approach to physical activity

An important aspect when considering PA from a life course perspective is the tracking or stability of PA over time and the study of factors which may influence the tracking (81). Consistent with the association between previous history of PA and later PA, it appears that PA is fairly stable across life, albeit tracking becoming weaker as the time between measures increases (91). Tracking may also be weaker during some life transitions such as from childhood into adolescence, and inactivity tends to track more strongly than PA across the lifespan (47, 81, 91).

Telama's review of PA tracking (91) offers insight into some of the underlying mechanisms and hypotheses of this phenomenon and describes how childhood and adolescent behaviour may prepare an individual for a life time of PA, and how early experience of PA may also make it easier to maintain and reinitiate PA in adulthood. The self-selection hypothesis suggests that those predisposed to better fitness and motor ability will perform more PA as children and as adults compared to those without similar predispositions (91). The development of behaviour capital – the accumulation of individual attributes such as social competence, decision making, attitudes and values, is likely to be more easily attained in childhood and adolescence which may in turn lead to healthy choices throughout life (92).

Other than studies which explore tracking of PA between childhood and adulthood, few have examined the associations between early life factors and adult PA. A proficiency in school sports, extroverted personality, and a more advantaged childhood SEP have been identified as potential early life determinants of more frequent LTPA in adulthood (93, 94). Childhood health (81, 82) cardiorespiratory fitness (82), and genetic factors (95) have in addition been associated with adult LTPA. However, relatively little is known about other developmental influences on adult PA. The following section briefly introduces the early life factors considered in this thesis, namely childhood SEP, and the relatively understudied developmental factors of birth weight, motor performance and timing of maturity that may have long term influences on adult LTPA. More detailed reviews of the literature on each of these topics are presented in the relevant chapters.

1.6.3 Socioeconomic and developmental factors from early life and leisure-time physical activity across adulthood

Several existing studies have examined associations between childhood SEP and adult LTPA but these have not been systematically reviewed which makes drawing conclusions more difficult (82). Developmental life course hypotheses may have relevance for adult LTPA although such studies are rare. For example, adults with low birth weight may be disadvantaged at sports and it is plausible to implicate motor development in the early life origins of adult LTPA through for example a reflection of genetic factors, or neurological competence and capabilities. The relationship between earlier stages of motor development such as attainment of motor milestones and adult LTPA have been less frequently examined with the literature focusing on their associations with later cognitive, psychiatric and developmental pathologies (96). Further, the transition from childhood to adolescence is accompanied by a drop in LTPA levels (59) and the timing of puberty in both boys and girls may be associated with later PA, although research is limited (97). Further, it is not known whether pubertal timing reflects a temporary drop in LTPA which changes once all peers have gone through this transition, or whether it relates to later LTPA in adulthood. It is also unclear whether any associations would extend to LTPA across adulthood. A more detailed critique of the existing literature on each of these factors is presented in the relevant chapters that follow, i.e. chapters 3, 4, 5 and 6.

1.7 Aim, objectives and hypotheses

This thesis has one main aim with four objectives (Figure 1), the majority of which will be addressed using data from the MRC National Survey of Health and Development (NSHD):

Aim: To investigate the associations between socioeconomic and developmental factors from early life and LTPA across adulthood, specifically to:

- Assess the associations between childhood SEP and adult LTPA through a systematic literature review (Objective 1), testing the hypothesis that more disadvantaged socioeconomic circumstances in childhood would be associated with less LTPA (chapter 3).
- Investigate the relationship between birth weight and LTPA across adulthood (Objective 2), testing the hypothesis that low birth weight would be associated with less LTPA (chapter 4).

- Examine whether infant motor development, ability at games and motor coordination in adolescence relate to LTPA across adulthood (Objective 3), testing the hypothesis that poorer performance would be associated with less LTPA (chapter 5).
- Explore the relationship between age at puberty and LTPA across adulthood (Objective 4), testing the hypothesis that early pubertal timing in girls and late puberty in boys would be associated with less LTPA (chapter 6).

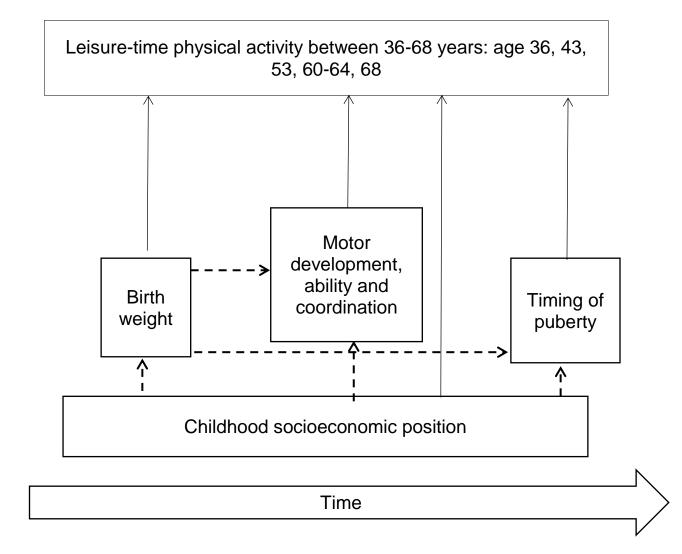
A secondary aim of this thesis is to examine whether associations between each early life factor and adulthood LTPA change with age at assessment of LTPA. Identifying if any associations found change with age at assessment of LTPA may help in understanding of underlying pathways. This could in turn have important implications for the design of future interventions. Therefore, this was investigated when examining associations in each relevant chapter.

1.8 Structure of thesis

This chapter is followed by a description of the data and methods and then the four main chapters before concluding with a discussion chapter. The dataset used in this thesis, the NSHD, is introduced in Chapter 2. This chapter includes a detailed examination of the LTPA data and describes the analytical strategies and methods used to address each objective of the thesis in the subsequent chapters. The conceptual model for this thesis is shown in Figure 1. The arrows indicate the hypothesised interrelationships between the different early life factors considered in this thesis and their relation to adulthood LTPA. Chapters 3, 4, 5 and 6 follow the same structure to examine associations between each set of selected early life factor and adulthood LTPA. Each chapter begins with a literature review followed by rationale for the proposed study and a list of specific hypotheses that are followed by the methods, results and discussion.

Chapter 3 examines how SEP in childhood relates to LTPA in adulthood. Chapter 4 examines associations between birth weight (a marker of prenatal growth) and adulthood LTPA. Chapter 5 examines how motor development, ability at games and motor coordination in adolescence relate to LTPA across adulthood while chapter 6 examines the associations between age at puberty and adulthood LTPA. Chapter 7 summarises the main findings of the thesis and considers the strengths and limitations of the work conducted. This is followed by a discussion of the implications

of the findings which includes analyses carried out in order aid discussion of whether developmental factors help explain early life socioeconomic differences in adult LTPA. This chapter then concludes with recommendations for future research. Figure 1.1 Conceptual model for thesis



Chapter 2: Data and methods

This chapter begins by introducing the MRC National Survey of Health and Development (NSHD), which as detailed in chapter 1 is the dataset used to address the objectives in chapters 3 to 6, and discussing the historical context of this birth cohort. This is followed by a discussion of response rates and missing data and examination of the patterns and types of leisure-time physical activity (LTPA) undertaken in this cohort. The chapter then discusses the analytical strategies employed in subsequent chapters including the development of appropriate LTPA outcomes used to address the research objectives.

2.1 Introduction to the MRC National Survey of Health and Development (NSHD)

The MRC NSHD, also known as the 1946 British birth cohort study, is a nationally representative paternal social class stratified sample of all single births to married parents in England, Wales and Scotland during one week in March 1946 (98). As it was not considered feasible to follow-up all births at that time, a sampling strategy was used to select participants from all eligible births to women with husbands in non-manual and agricultural occupations in addition to 1 in 4 of all births to women with husbands in manual occupations which comprised the majority of the workforce. Initially designed to investigate fertility rates, maternity services and infant health, the cohort of 5,362 males and females were selected from the original maternity survey and have been regularly followed-up throughout their lives (99) with the newest round of data collection at age 68 completed in 2014 and at ages 69-70 just completed.

Follow-up occasions in childhood included data collections at birth and at ages 2, 4, 6, 7, 8, 9, 10, 11, 13 and 15 years. In early years, data on NSHD participants were collected from parents, health care and educational professionals and study participants themselves. In adulthood, information was gathered using postal data collections and home visits by professional interviewers and research nurses (86). Data were also collected during clinic-based assessments carried out at age 60-64 (87). Information on LTPA across adulthood, the outcome of interest for this thesis, was collected at ages 36, 43, 53, 60-64 and 68 years and is described in detail in section 2.2. At age 60-64, PA data were also gathered by activity monitors and these were examined as a secondary outcome at that age. They are described in section 2.2.4.

2.1.1 Historical context of the NSHD

How children were raised in 1940s Britain was influenced by their mothers' socialisation in the pre-war world and the reality of post-war austerity. Working class mothers were most likely to have experienced hardship (100). Food rationing was in existence throughout the war period and remained until 1954 (101). Rations of some items such as fat and bacon were cut even further prior to the NSHD cohorts' birth (88). However, this cohort benefited from the launch of the National Health Service in 1948 (89). During the lifetime of the NSHD, attitudes, advice and the public image of PA have improved and there is evidence that cohort members' views of this public image of PA positively influenced their participation in LTPA (102). During the NSHD's childhood, physical education at school was not particularly concerned with health (103) and it was not until the 1960s and 70s that schools and other organisations began providing out of school activities for children to play and socialise (89). Sports promotion did eventually become more prominent, as evident by the setting up of the advisory Sports Council in 1966 (104) and participation in organised sport was rising from the 1960s onwards (88).

2.1.2 Response rates, loss to follow-up and characteristics of non-responders

Loss to follow-up and missing data can lead to biased estimates of the association between an exposure and an outcome (105). Two approaches are used to deal with missing data in this thesis. First, an attempt is made to understand and describe the reasons for non-response so as to infer the likely direction and magnitude of any resulting bias. This is accomplished in this section by summarising studies of response rates in NSHD and by comparing those with at least one measure of LTPA to those without any LTPA data by each early life factor and selected covariate (Table 2.2). Details of the early life measures and choice of covariates used to address the specific objectives are provided in each chapter and are not described here. Second, mixed-effects models (introduced in section 2.3.2) are used to allow all those with at least one measure of LTPA to be included in analyses under the missing at random assumption thus minimising bias due to missing outcome data (see section 2.3.2).

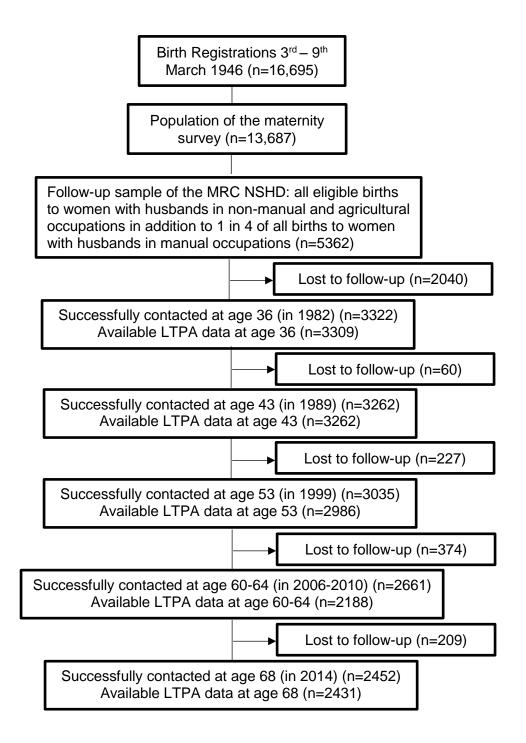
Response rates at each wave were generally high including 86, 87, 83, 84 and 83 per cent successfully contacted at ages 36, 43, 53, 60-64 (98, 106) and 68 years (107) respectively. Attrition rates due to deaths, emigration and living abroad up to

age 53 amounted to 8.7, 8.6, and 2.2 per cent of the original cohort respectively (86). Unavoidable attrition was comparable to that found in similar aged adults from the general population with respect to sex but not with certain socio-demographic characteristics (108). Attrition was higher in those with a lower adult SEP and lower cognitive scores (109). Up to age 60-64 years, NSHD study members were broadly representative of similar aged British born adults living in the UK (94-96). At the last completed round of data collection at age 68 years, 83.4% of 2,943 study participants who were still alive and eligible to participate were successfully contacted. Of the 490 who were not successfully contacted, 11 had died, 453 did not return a questionnaire and 26 questionnaires were returned undelivered.

Figure 2.1 shows a flowchart of participant selection for the thesis. Of those successfully contacted at ages 36 (n=3,322), 43 (n=3,262), 53 (n=3035), 60-64 (n=2,661) and 68 years (n=2,453), 99.6%, 100%, 98.4%, 82.2% and 99.1% respectively provided information on LTPA. In total, 3,766 participants had at least one measure of LTPA (Figure 2.1) and the majority had LTPA data from four of the five different ages in adulthood. A total of 1657 participants had LTPA data from all five ages. In addition, the reasons for attrition from age 36 to 68 and the numbers of participants at each age is also provided in table 2.1. There were some differences in characteristics between those with and without any data on LTPA (Table 2.2). Higher proportions of those with at least one measure of LTPA were female and had fathers in professional, managerial or technical occupations while lower proportions had low birth weight, below-average ability at games and serious childhood illness.

measure of LTPA on attainment of infant milestones, tapping speed, pubertal status or birth order.

Figure 2.1 Flow-chart of participant selection



Age	Not contacted	Successfully contacted	Data on LTPA
36 (1982)	Deaths=323	3322	3309
	Permanent refusal=520		
	Living abroad or Emigrated=584		
	Untraced=553		
43 (1989)	Deaths=365	3262	3262
	Permanent refusal=540		
	Living abroad or Emigrated=618		
	Untraced=276		
53 (1999)	Deaths=469	3035	2986
	Permanent refusal=640		
	Living abroad or Emigrated=580		
	Untraced=330		
60-64	Deaths=808	2661	2188
(2006-	Permanent refusal=594		
2010)	Living abroad or Emigrated=584		
	Untraced=550		
68 (2014)	Deaths=957	2452	2431
	Permanent refusal=620		
	Living abroad or Emigrated=574		
	Untraced=395		

Table 2.1 Attrition from age 36 to 68 and the number of subjects at each age.

All cumulative totals from original cohort in 1946. Numbers extracted from Wadsworth et al. 1992 (108), Wadsworth et al. 2003 (109), Wadsworth et al. 2006 (98), Stafford et al. 2013 (106), Kuh et al. 2016 (107).

Table 2.2 Characteristics of those with and without any LTPA data between ages 36-

68 years by early life factor

>=1 LTPA	No data on LTPA	p-value
neasurement	(n=1596)	
(n=3766)		
49.6	42.5	<0.001
4.7	8.8	<0.001
14.8	19.0	p=0.004
		-
24.5	23.4	p=0.8
16.9	13.7	p=0.5
42.2	43.0	0.8
23.1	20.9	0.008
6.5	17.7	p<0.001
		•
6.6 (SD: 1.51)	6.5 (SD: 1.47)	p=0.06
1.4 (SD: 2.30)	11.3 (SD: 2.34)	p=0.2
3.6 (SD: 2.55)	13.5 (SD: 2.60)	p=0.2
5.6 (SD: 1.77)	5.5 (SD: 1.77)	p=0.1
· · · · ·		•
5.0 (SD: 1.57)	4.9 (SD: 1.52)	p=0.08
x - /		1
	neasurement (n=3766) 49.6 4.7 14.8 24.5 16.9 42.2 23.1 6.5 6.6 (SD: 1.51) 1.4 (SD: 2.30)	neasurement (n=3766)(n=1596)49.642.54.78.814.819.024.523.416.913.742.243.023.120.96.517.76.6 (SD: 1.51)6.5 (SD: 1.47)1.4 (SD: 2.30)11.3 (SD: 2.34)3.6 (SD: 2.55)13.5 (SD: 2.60)6.6 (SD: 1.77)5.5 (SD: 1.77)

Proportions (%) for categorical variables. Mean and standard deviation (SD) for continuous variables. P-values calculated from Chi-squared tests for categorical variables and t-test for continuous variables.

2.2 Leisure-time physical activity across adulthood

2.2.1 Measurement and operationalisation of leisure-time physical activity

At ages 36, 43, 53, 60-64 and 68 years, study participants reported how often they participated in LTPA during nurse interviews or using self-completed questionnaires (Table 2.3, Appendix 7). While questions were also asked about the duration and intensity of bouts in some but not all ages, this thesis focuses on the reported frequency of participation (number of times) in LTPA to avoid added bias and measurement error in classifying participants based on self-reported intensities and/or duration of participation (51, 110). At the age 36 home visit interview, study members were asked to recall the number of times they participated in 27 different sports, exercises and other outdoor leisure-time activities during the previous month based on the Minnesota LTPA questionnaire(93, 111) alongside separate questions on leisure-time walking. At age 43, questions were asked during the interview on participation in sports, vigorous leisure activities or exercise in the previous year including for how many months and how often in those months activities were

performed (112, 113). At ages 53 (interview), 60-64 and 68 years (questionnaires), identical questions were asked about the number of times study members took part in any sports, exercises or vigorous leisure activities during the previous 4 weeks (112, 113).

At each age, study participants were classed as inactive if they reported no participation in LTPA, moderately active if they participated up to four times in LTPA or regularly active if they reported taking part five or more times in LTPA (in the previous month at age 36, per month at age 43, and in the previous 4 weeks at ages 53, 60-64 and 68) (99-101). These three-level categorical LTPA measures were also dichotomised in order to classify participants as either active or inactive during leisure-time at each age (based on whether or not they reported participating at least once per month in any sport, exercise or other vigorous leisure-time activities) (101). This provided comparable data on LTPA across adulthood and allowed comparison of differences between those who take part in LTPA and those that do not across adulthood. In previous research from NSHD, these LTPA variables have been shown to be associated with significant differences in physical capability (112, 113), body composition (114), mental wellbeing (115) and cognition (17, 18). This along with growing evidence of the importance for health of even small amounts of LTPA, particularly among older populations (116-119), suggests that the differences in LTPA operationalised for this thesis are likely to be biologically meaningful (see chapter 7 for further discussion on this point).

These data could also be seen to demonstrate some evidence of validity. For example, those who reported taking part in LTPA at ages 36, 43 and 53 tended to spend greater time in moderate-to-vigorous intensity PA assessed by activity monitors at age 60-64 when compared with others reporting no LTPA (58). Moreover, when self-reported PA measures and data from activity monitors were compared in a subsample of the NSHD they were found to rank study participants similarly by levels of PA at age 60-64 (55).

Table 2.3 Leisure-time physical activity information reported by NSHD participants

Age 36

Number of times participated in the previous month in each of the following sports, outdoor activities and exercises: Badminton; bowls; cricket; exercises like press-ups, situps etc. at home; exercises like press-ups, sit-ups etc. at gym; football; golf; hill/mountain climbing; jogging; rowing; running/athletics; sailing; squash/rackets; swimming; table tennis; tennis; yoga; water skiing; volleyball; scuba diving; basketball; fishing; riding; movement to music; weight training; ballroom dancing; other dancing. Number of times during the previous week cycling outside work. Number of times during the previous month walking for pleasure.

Age 43

Number of times participated in the previous year in any sports, vigorous leisureactivities or exercise

Age 53

Number of times participated in the previous four weeks in any sports, vigorous leisureactivities or exercise.

Age 60-64

Number of times participated in the previous four weeks in any sports, vigorous leisureactivities or exercise.

Number of times in the previous year participated in each of the following activities: swimming – leisurely not laps; swimming – competitive or laps; walking for pleasure – excluding for transport; backpacking, hill walking or mountain climbing; jogging; competitive running; cycling for pleasure – excluding for transport; racing or rough terrain cycling.

Number of times in the previous year participated in each of the following aerobic and gym activities: high impact aerobics, step aerobics; other aerobics; exercises with weights; conditioning exercises e.g. using an exercise bike or rowing machine; floor exercises e.g. stretching, bending, keep fit.

Number of times in the previous year participated in each of the following games and team sports: snooker, billiards, darts; bowling – indoor, lawn or ten pin; tennis or badminton; squash; table tennis; golf; netball, volleyball, basketball; football, rugby or hockey during the season; cricket during the season.

Number of times in the previous year participated in each of the following activities: dancing e.g. ballroom; musical instrument playing, singing; horse-riding; fishing; rowing; sailing, windsurfing, boating; ice-skating; winter-sports e.g. skiing; martial arts, boxing, wrestling.

Age 68

Number of times participated in the previous four weeks in any sports, vigorous leisureactivities or exercise.

2.2.2 Patterns of LTPA from ages 36 to 68 years

The proportions taking part in LTPA generally declined with age across adulthood (Figure 2.2, Table 2.4). Alongside this general decline in LTPA with increasing age is an apparent rise in the proportions that were active at age 53 when compared with age 43 (Figure 2.2, Table 2.4). One possible explanation for this difference is the use of different questions to collect LTPA at ages 43 and 53. This rise in LTPA at age 53 might also be partly due to the effects of survival emerging as participants move into midlife whereby those most active and healthiest are likely to survive longest (120, 121). This could also be partly due to the effects of national interventions; for example, the ACTIVE for LIFE campaign initiated in 1996, 3 years prior to the age 53 assessment was found to have increased the proportions of adults knowledgeable about PA recommendations (122). At ages 36 and 43 years, higher proportions of men than women reported taking part in LTPA but sex-differences were less marked at older ages and there was a slight overall increase in the proportions taking part in LTPA at age 68 (Figure 2.2, Table 2.4). Among those taking part in LTPA, higher proportions of both men and women reported regular rather than moderate participation at each age (Table 2.4). Examining the prevalence of LTPA using the maximum sample sizes available at each age led to similar conclusions. Table 2.5 shows the trajectories of LTPA across adulthood (coded as at least once per month at each age versus none) overall and by sex in those with data at all five ages. Patterns were similar when examined separately in men and women (Table 2.5) and showed that the most prevalent groups in both sexes were those reporting persistent participation in LTPA (n=249) followed by persistent nonparticipation (n=199) at each age (Tables 2.4). Other prevalent groups were those taking part in LTPA at age 36 only (n=137) and the group that continue participating in LTPA up to age 53 but not in their 60s (n=118). Very few study participants took up LTPA at later ages if they were previously inactive at age 36 (Table 2.5).

Table 2.4 Proportions % (N) of inactive, moderately active and regularly active in

LTPA at each age in adulthood overall and by sex.

LTPA (n=1657)				
Overall	Men	Women		
33.1 (548)	29.2 (223)	36.4 (325)		
27.0 (447)	27.4 (209)	26.6 (238)		
40.0 (662)		37.0 (331)		
()	· · ·			
	1 -			
47.7 (791)	43.0 (328)	51.8 (463)		
· · ·		25.1 (224)		
· · ·	• • •	23.2 (207)		
27.2 (401)	· · ·	· · ·		
	p~0.	.001		
43.3 (718)	41.3 (315)	45.1 (403)		
	22.5 (172)	18.6 (166)		
		36.4 (325)		
36.3 (601) 36.2 (276) 36.4 (325) p=0.1				
	•			
62.5 (1036)	63.3 (483)	61.9 (553)		
	· · ·	14.9 (133)		
		23.3 (208)		
- ()	· · ·	· · · ·		
	ľ			
58.4 (967)	57.9 (442)	58.7 (525)		
		13.7 (122)		
		27.6 (247)		
- (-)		0.5		
	Overall 33.1 (548)	OverallMen $33.1 (548)$ $29.2 (223)$ $27.0 (447)$ $27.4 (209)$ $40.0 (662)$ $43.4 (331)$ $p=0$ $47.7 (791)$ $43.0 (328)$ $25.1 (415)$ $25.0 (191)$ $27.2 (451)$ $32.0 (244)$ $p<0$ $43.3 (718)$ $41.3 (315)$ $20.4 (338)$ $22.5 (172)$ $36.3 (601)$ $36.2 (276)$ $p=1$ $62.5 (1036)$ $63.3 (483)$ $14.4 (239)$ $13.9 (106)$ $23.1 (382)$ $22.8 (174)$ $p=1$ $58.4 (967)$ $57.9 (442)$ $13.0 (215)$ $12.2 (93)$ $28.7 (475)$ $29.9 (228)$		

Sample comprises those with all five measures of LTPA. p-value from chi-squared test.

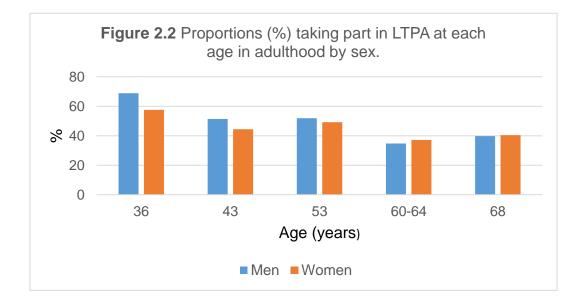


Table 2.5 Proportions in each possible trajectory of active (1) or inactive (0) in LTPA from ages 36 to 68 years overall and by sex.

Trajectories (0=inactive; 1=active) n=1657				ctive)	N (%)		
Age	Age	Age	Age	Age	Overall:	Men: 763	Women: 894
36	43	53	60-64	68	1657 (100)	(46.0)	(54.0)
0	0	0	0	0	199* (12.0)	78 (39.2)	121 (60.8)
0	0	0	0	1	28 (1.7)	14 (50.0)	14 (50.0)
0	0	0	1	0	17 (1.0)	6 (35.3)	11 (64.5)
0	0	0	1	1	12 (0.7)	3 (25.0)	9 (75.0)
0	0	1	0	0	65 (3.9)	31 (47.7)	34 (52.3)
0	0	1	0	1	20 (1.2)	10 (50.0)	10 (50.0)
0	0	1	1	0	16 (1.0)	1 (0.3)	15 (97.7)
0	0	1	1	1	22 (1.3)	8 (36.4)	14 (63.6)
0	1	0	0	0	47 (2.8)	19 (40.4)	28 (59.6)
0	1	0	0	1	11 (0.7)	7 (63.6)	4 (36.4)
0	1	0	1	0	7 (0.4)	1 (14.3)	6 (85.7)
0	1	0	1	1	5 (0.3)	3 (60.0)	2 (40.0)
0	1	1	0	0	34 (2.1)	18 (52.9)	16 (47.1)
0	1	1	0	1	16 (1.0)	6 (37.5)	10 (62.5)
0	1	1	1	0	18 (1.1)	5 (27.8)	13 (72.2)
0	1	1	1	1	31 (1.9)	13 (41.9)	18 (58.1)
1	0	0	0	0	137* (8.3)	68 (49.6)	69 (50.4)
1	0	0	0	1	36 (2.2)	12 (33.3)	24 (66.7)
1	0	0	1	0	18 (1.1)	8 (44.4)	10 (55.6)
1	0	0	1	1	26 (1.6)	10 (38.5)	16 (61.5)
1	0	1	0	0	82 (5.0)	34 (41.5)	48 (58.5)
1	0	1	0	1	37 (2.2)	11 (29.7)	26 (70.3)
1	0	1	1	0	29 (1.8)	12 (41.4)	17 (58.6)
1	0	1	1	1	47 (2.8)	22 (46.8)	25 (53.2)
1	1	0	0	0	88 (5.3)	42 (47.7)	46 (52.3)
1	1	0	0	1	32 (1.9)	17 (53.1)	15 (46.9)
1	1	0	1	0	23 (1.4)	14 (60.9)	9 (39.1)
1	1	0	1	1	32 (1.9)	13 (40.6)	19 (59.4)
1	1	1	0	0	118* (7.1)	64 (54.2)	54 (45.8)
1	1	1	0	1	86* (5.2)	52 (60.5)	34 (39.5)
1	1	1	1	0	69 (4.2)	41 (59.4)	28 (40.6)
1	1	1	1	1	249* (15.0)	120 (48.2)	129 (51.8)

* Prevalent trajectories (>5%).

2.2.3 Types of LTPA reported at ages 36 and 60-64

This section describes the types of LTPA reported by men and women at ages 36 and 60-64 where more detailed PA questions were asked about participation in sports, exercise and other leisure activities (Table 2.3). At both ages and in both men and women, walking (which was not included in the derived LTPA at age 36 variable)

followed by swimming were the most commonly reported types of LTPA (Figures 2.3 and 2.4). Overall, 49% reported walking for pleasure in the last month at age 36 compared to 71% in the last year at age 60-64 and walking was more often reported by women at both ages (Figures 2.3 and 2.4). Around 24% of both men and women reported swimming in the last month at age 36 compared to 33% in the last year at age 60-64. Men were more likely to take part in golf and fishing while women were more likely to report dancing and music activities at both ages. These estimates are consistent with published studies at these ages (67, 93).

At age 36, men were more likely to report football, jogging and squash/rackets (Figure 2.3). Such team activities were not as frequently reported at age 60-64. In addition, over 15% of both men and women reported cycling outside of work in the previous week (Figure 2.3). It was also previously shown that swimming and golf at age 36 were more commonly reported by those interviewed in the summer months (93). Women were more likely to do floor exercises and aerobics at age 60-64 (Figure 2.5). It was previously shown that 62% of active participants reported 2 or more different activities (67). Figure 2.4 shows how many men and women coded as active in LTPA at age 36 were doing team sports and non-team sports and how many were doing both. Together with this figure, examining Spearman's rank correlation coefficients of team sports and non-team sports with overall LTPA suggest that non-team sport activities (r=0.9) form a greater proportion of the activity captured within the derived LTPA measure at age 36 than team sports (r=0.5). In addition, leisure-time walking at age 36 was weakly correlated with the overall LTPA measure at age 36 (r=0.2).

Figure 2.3 Proportions (%) taking part once or more than once in each sport, exercise and other LTPA in the previous month at age 36 by sex.

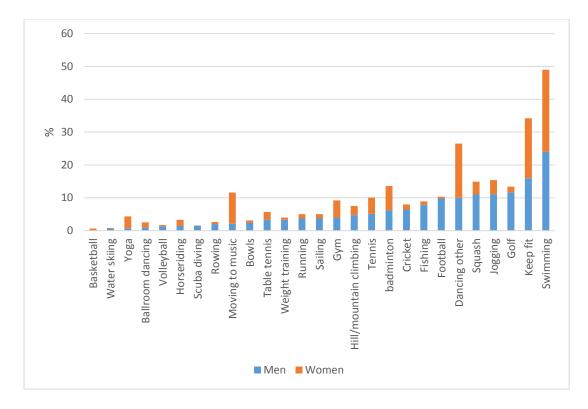
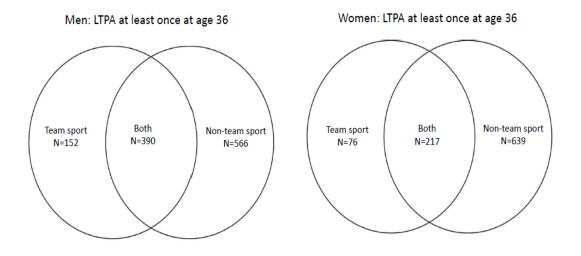


Figure 2.4 Numbers of men and women taking part in team sports and non-team sports at least once per month at age 36.



37

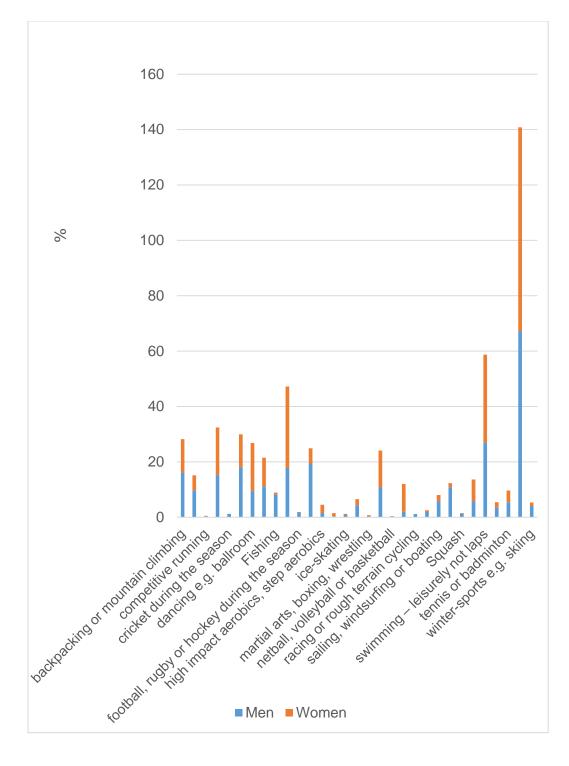


Figure 2.5 Proportions (%) taking part once or more than once in each sport, exercise and other LTPA in the previous year at age 60-64 by sex.

2.2.4 Physical activity measured by monitors at age 60-64

At age 60-64, study participants were invited to wear a movement and heart rate monitor (Actiheart, CamNtech Ltd, Papworth, UK) for five consecutive days including nights attached in a standard position to their chest with two ECG-electrodes at the end of their clinical assessment or during a nurse home visit, and to then return them via the post. Participants were shown how to position the monitor by trained nurses and were provided with written information reminding them to wear the monitors at all times, except when swimming and bathing. These monitors recorded heart rate and movement data in 30 second epochs to derive measures of free living PA. Data were processed by the MRC Epidemiology Unit. Heart rate data were individually calibrated using an 8-minute step test to account for between-individual differences in the relationship between PA intensity and heart rate (123). Group calibration was used where individual calibration was not carried out (those who were seen at their home or who did not undertake a step test (exclusion criteria for the step test were high blood pressure, history of unstable angina, or breathlessness, n = 723) for the translation of heart-rate into activity intensity. All calibration was adjusted for sleeping heart rate, age, sex and β -blocker use (123). Those who participated in this clinic assessment were shown to have lower rates of smoking and obesity and also to have a higher SEP as indicated by higher educational attainment and occupational class than the rest of the cohort (106).

PA intensity was summarised as time spent in different intensities relative to resting metabolic rate (1 standard MET) and as total PA energy expenditure (PAEE) (in kJ/kg/day) (124). A standard definition of 1 MET, which summarised intensity regardless of body composition, was used so as to avoid overestimation n of PA intensity with higher BMI (125). Data were adjusted for wear time and diurnal information bias to allow for variation between individuals in wear time at different times of the day when levels of PA may be expected to vary. Following visual inspection of heart rate and movement traces, participants were excluded if acceleration signals were corrupt or where valid heart rate measurements were not available. These measures have been validated with indirect calorimetry and doubly labeled water in adult samples (125-127). Those with less than 48 hours of wear time were excluded on the assumption that their data may not be representative of their usual everyday activity, thus ensuring that PA data was an accurate reflection of normal PA.

For the purpose of this thesis, time spent in moderate-to-vigorous PA (MVPA) (>3 METs) was considered as secondary outcome to LTPA at age 60-64 since MVPA makes up the majority of time spent in LTPA (40). Thus, the measure of MVPA allows the capture of more strenuous forms of activity above a moderate intensity (128). Associations of each early life factor with PAEE, average PA energy expenditure estimated directly from measured acceleration and calibrated heart rate data, were also presented. PAEE is a measure of the total volume of activity energy expenditure and represents all energy expended above resting, including lighter intensity activities (128). Men (n=834) spent greater time in MVPA: median=0.68 hours/day (interquartile range=0.9) versus median=0.43 hours/day (interquartile range =20.7) versus median=33.5 (interquartile range =16.9) when compared with women (n=888).

2.3 Methods and analytical strategy

2.3.1 General analytical strategy

A systematic analytic process was used to complete the objectives of this thesis and test the hypotheses described in Chapter 1.8.2. Detailed descriptions of the analytic samples and choice of covariates used to address each aim of this PhD are provided in the relevant chapters and therefore, only a general summary of common analyses is provided here. The association between socioeconomic circumstances in childhood and LTPA in adulthood (1st objective) was examined through a systematic literature review that was carried out according to established guidelines and its methods are detailed in the next chapter of this thesis. For the remaining objectives, addressed in chapters 4-6, initial exploratory analyses were carried out to check the distributions of the variables of interest using summary statistics and histograms for continuous variables and frequency tables for categorical ones. Potential early life confounders, factors which are associated with both exposure and outcome but are not on the causal pathway (105), were identified from the literature and by examining inter-relationships of different early life factors as well as their associations with LTPA in NSHD.

The associations between each chosen early life factor (birth weight, attainment of motor milestones, ability at games, tapping speed and age at puberty), and LTPA at each age in adulthood were assessed through a series of binary and multinomial

logistic regression models that were adjusted in a step-wise fashion for hypothesised confounders. These models were restricted to a comparable sample of participants with data on LTPA from each age in adulthood. In addition, at age 36, separate binary logistic regression models were used to examine associations of each early life factor with participation in LTPA involving team sports, other LTPA, and leisure time walking. At age 60-64, linear regression was used to examine associations between each early life factor and MVPA and PAEE assessed by activity monitors in comparable analytical samples comprising those who also had data on LTPA at that age. The estimated ORs of LTPA at age 60-64 are presented alongside results from linear regression analyses. The linear regression estimates were presented as percentage differences in monitored MVPA and PAEE since outcomes are multiplied by 100 and logged (129).

Evidence of effect modification, where effect of an exposure variable changes over different values of another variable (e.g. sex) (105), were examined by including interaction terms in the regression models (to test deviation from additive effects) (130). Continuously measured exposures were tested for deviation from linearity by comparing to models with polynomial terms and those with categorically entered formulations of the exposure (105).

2.3.2 Using mixed-effects models to examine associations with adulthood LTPA

Following separate examination of associations with LTPA at each age in adulthood in relation to early life factors, subsequent analyses explore associations between each early life factor and LTPA across adulthood by making use of these repeated LTPA measurements to include all those with at least one measure of LTPA in the analyses. One advantage of using longitudinal data structures include incorporation of missing LTPA data and thus maximising sample sizes. The use of repeat measurements therefore helps to improve precision of estimates of associations due to the larger sample sizes and reduces risks of type 1 errors (detecting false associations) by limiting the need for multiple testing. Further, by allowing for inclusion of those with incomplete LTPA data these models can also help reduce potential for survival bias which would underestimate associations as the healthiest and most active study participants survive to old age These models can also be used to objectively test either whether associations with early life factors remains constant with age at LTPA measurement or whether associations change with age at LTPA

assessment. These associations were examined using mixed-effects binary and multinomial logistic regression models (131).

Mixed-effects binary logistic regression models (131, 132) were used to calculate the odds ratios (OR) of LTPA (at least once per month) versus no LTPA between ages 36 and 68 years. These models are a generalisation of the linear mixed model for discrete outcomes. Subject-specific random effects are incorporated into the model to allow for correlations among the LTPA responses (residuals). Log-likelihood maximisation was achieved by integrating out the random effects with the use of adaptive Gaussian quadrature as implemented by the STATA command meqrlogit (7 integration points were used). The linear model predictions (predicted log-odds of LTPA) were plotted against age to visualise differences in LTPA across adulthood for different groups of each early life factor. Interaction terms were used to test whether or not the effect of sex and of each early life factor varied by the age at measurement of LTPA.

Mixed-effects multinomial logistic regression models (131, 133) were then used to calculate the relative risk ratios (RRR) of moderate (1-4 times per month) and regular (5 or more times per month) LTPA (versus no LTPA) between ages 36-68 years. These models were estimated using Markov Chain Monte Carlo (MCMC) simulation methods as implemented in the MLwiN software version 2.36 (CMM, University of Bristol) (134-136). MCMC are a family of Bayesian estimation techniques which work by drawing a random sample of values for each parameter from its probability distribution (134, 137-139). The steps followed to fit these models included selecting initial values and specifying prior knowledge and are described in the following paragraph.

To fit these multinomial models using MCMC, parameter estimates from iterative generalised least squares models were specified as initial values. Diffuse (uninformative) prior distributions were selected and used to approximate maximum likelihood estimation. The MCMC algorithm was then run for 500 iterations until each parameter distribution has settled down to its stationary distribution (i.e. the burn-in period when the chains are converging to their posterior distribution). The MCMC algorithm was then run for a further 5000 iterations (the monitoring period) in order to store a monitoring chain for each parameter. Similar burn in and monitoring periods have been used in published studies (140). Point estimates and standard errors are

given by the means and standard deviations of these monitoring chains. (134, 135, 138, 139).

Mixed-effects models can be fit with random intercepts only or with the addition of random coefficients (slopes). Random intercepts models use an exchangeable autocorrelation structure meaning that every pair of LTPA residuals on a study participant has the same correlation; allowing the probability of LTPA to vary between individuals. Alternatively, a more complex autocorrelation structure can be specified by also allowing the coefficients for age at assessment of LTPA to vary across individuals (random slopes for age). This allows each subject to follow a different trajectory of change in LTPA and these models assume that changes in LTPA are driven primarily by a subject's age. For this thesis, the binary mixed-effects models were fit using random intercepts and slopes for age at assessment of LTPA while to aid convergence of the more complex multinomial mixed-effects models, they were fit with an exchangeable autocorrelation structure (i.e. with random individually varying intercepts only). The resultant estimates of both models are interpreted as conditional (subject specific) on these random effects.

In addition, there are other subtle differences in the methods used to estimate the mixed-effects binary models and the more complex multinomial models, and thus subtle differences are to be found in how results are reported in the tables. For example, the measure of variability around the estimated ORs of LTPA between 36-68 years are the classical 95% confidence intervals (95% CI) while the equivalent measure of variability around the estimated RRRs of moderate and regular LTPA obtained within the Bayesian framework of the MCMC estimation are the 95 credible intervals (95% CrI). There are also subtle differences in interpretation; the 95%CI relate to a sequence of similar findings in repeated practice and imply that 95% of intervals contain the true value while the 95CrI is interpreted as 95% probability that the interval contains the true value (131, 134, 135, 137, 139, 141).

In addition, likelihood-ratio tests were carried out after the binary mixed-effects models to estimate p-values which were used to indicate statistical significance of the early life factor of interest. On the other hand, for the multinomial results, the Deviance Information Criterion (DIC) is presented as a measure of model comparison that takes into consideration both the fit of the data and model complexity. Here, better models have smaller DIC values. It is also possible to carry out Wald test after the multinomial analyses to estimate a statistical significance for the early life factor of interest. However, this is not carried out as the DIC statistic along with the measures of variability around the estimates (95%CrI) should suffice for the purpose of this thesis. Further description of the mixed-effects model estimation using MCMC is provided in appendix 6.

Sex by age and early life factor by age interactions were tested using the binary mixed-effects models and where evidence of interaction was found these were incorporated into all binary and multinomial mixed-effects models. There was evidence of a sex by age interaction such that women became slightly more likely than men to report participation in LTPA with increasing age (OR of LTPA for women versus men per year increase in age = 1.02; 95%CI: 1.02 - 1.03, p<0.001) and thus men had more rapid rate of decline in LTPA than women despite initially higher OR's of LTPA at ages 36 and 43. Therefore, where men and women were combined in analyses, a sex by age interaction term was added to all mixed-effects models. Likewise, where an association between an early life factor and LTPA was found to change by age, an early life factor by age at LTPA interaction term was added to the mixed-effects models. All analyses include exact age at 60-64 years and age was centred at 43 years to aid interpretation. In all chapters where primary analyses were carried out, results from the mixed-effects models are presented as main tables with results of associations with LTPA at each age presented in appendices. The following chapters will explore a number of different factors from early life in relation to LTPA in adulthood

Chapter 3: Childhood socioeconomic position and leisure-time physical activity across adulthood

Chapter objective: To examine the association between childhood SEP and LTPA across adulthood.

Many existing studies have examined how socioeconomic factors from early life relate to LTPA in adults but the literature has not been systematically reviewed. This chapter addresses the first objective of this thesis by systematically reviewing all published studies of the association between SEP in childhood and LTPA in adulthood, and examines hypothesised sources of between-study heterogeneity including whether this association varies by age at assessment of LTPA.

3.1 Background

Many health outcomes and behaviours are associated with contemporaneous socioeconomic circumstances such that the lower a person is positioned in the socioeconomic hierarchy the poorer their health outcomes tend to be (142-145). Like many health-related outcomes, evidence from existing reviews indicates that LTPA is associated with contemporaneous socioeconomic circumstances (29, 146, 147). These show that less socioeconomically advantaged youth (147) and adults (29, 146) tend to participate less frequently in LTPA compared with their more advantaged peers. However, inconsistencies in the results as well as disagreement over whether certain indicators of socioeconomic position (SEP) appear to be more strongly related to LTPA than others (29, 146) have been reported. For example, one review found that education was more closely related to contemporaneous LTPA (146) while another found no difference (29).

In addition to more temporally adjacent associations between SEP and health, considerable evidence links childhood socioeconomic circumstances to adult health and behavioural outcomes (148). These associations are typically of substantial magnitude and are not fully explained by the continuity of socioeconomic circumstances from childhood into adulthood (148). It is plausible that adult LTPA mediates some of these associations or that adult LTPA itself exhibits early life socioeconomic origins. However, studies of the association between childhood SEP and adult LTPA have been inconsistent (94) and the literature has not been systematically reviewed thus making it difficult to draw meaningful conclusions.

A systematic review was thus carried out to test the hypothesis that a lower childhood SEP is associated with less participation in LTPA during adulthood. The extent to which associations were explained by the continuity of SEP from childhood into adulthood was examined and sources of between-study heterogeneity (type of childhood SEP indicator, age at assessment of LTPA, country) were explored.

3.2 Methods

This review was carried out following the PRISMA statement guidelines (149) and a pre-defined protocol that was registered with the PROSPERO database (registration number: CRD42014007063) and published in a peer-reviewed journal (150). Study selection, data extraction and quality assessment forms were developed for use at the relevant phases of the review (Appendices 1A to 1E). In addition, the findings of this systematic review were published in a peer-reviewed journal (151).

3.2.1 Eligibility criteria

Included studies were English-language publications based on community-dwelling participants, which tested the association between at least one indicator of childhood SEP (up to 18 years), either recalled or prospectively ascertained, and an LTPA outcome measured during adulthood (25 years and above).

Eligible indicators of childhood SEP were any resource or prestige-based measure of position within a societal structure (152) referring to participants' early life (e.g. parental occupation, parental education, parental income, and childhood housing characteristics). Own education was not be considered an eligible exposure despite its occasional use as an indicator of childhood SEP as it also captures the influence of adult resources (153).

All LTPA outcomes were considered including sport, exercise, and total LTPA (3). The minimum age of 25 years at measurement of PA, which equates the United Nations' definition of adulthood (154), was selected to identify working age samples to help elucidate whether any observed influence of childhood SEP on adult PA exists after accounting for own adult SEP. Reviews, unpublished literature, studies measuring strictly non-LTPA outcomes (e.g. occupational activity only) and studies sampling non-community dwelling participants (e.g. hospital inpatients, care home residents) were excluded.

3.2.2 Search strategy

The following five online databases were searched systematically using free-text synonym keywords to locate all eligible articles available up to 15th December 2014: Embase (from 1974), Medline (from 1946) and PsycINFO (from 1806) via OvidSP interface (Appendix 1A), and CINAHL (from 1937) and SPORTDiscus (from 1985) via EBSCO interface (Appendix 1B). Search terms were tested in preliminary trials to improve the effectiveness of the final search. Proximity and Boolean logic operators and truncation commands were used during the search and modified where necessary for each interface (150). A search of the included papers' reference lists supplemented the electronic database search.

3.2.3 Study selection

Results of the database searches were merged and duplicates excluded and remaining abstracts were screened by myself in addition to another researcher working independently to identify potentially eligible papers in accordance with PRISMA guidelines. Full texts of potentially eligible papers were double screened and ineligible articles were excluded. Disagreements were resolved through discussion between reviewers which I led (Appendix 1C).

3.2.4 Data extraction

The following information was extracted from all included papers by myself in addition to another researcher working independently using an extraction form developed by myself (Appendix 1D): citation details including title and year of publication, study details including design and country/region, exposure and outcome details including type of indicators used and how and when these were ascertained, participant details including gender, age and numbers included in analysis, results i.e. measures of association provided e.g. odds ratios, correlation or regression coefficients, statistical methods used, measures of precision provided e.g. confidence intervals and standard errors, information on adjustment for potential confounding and mediating factors, and lists of potentially eligible papers identified from reference lists. All data were extracted by myself in addition to another researcher working independently and discrepancies were resolved through discussion between all reviewers which I led.

3.2.5 Quality assessment

The quality of each included study was assessed concurrently with data extraction using a version of the Newcastle-Ottawa scale (155) which was modified by myself as recommended by a review of quality assessment tools (156). This led to the development of a concise form that was specifically tailored to appraise study quality for the purposes of this review (Appendix 1E). Study quality was judged based on the following criteria: representativeness, adjustment for covariates, length of follow-up, and methodology used to measure childhood SEP and adult LTPA. Quality scores were calculated as the average ratings of myself in addition to another independently working researcher's scores with a potential range from 0 (lowest quality score) to 9 (highest quality score). Quality rating was not used to exclude studies and was instead integrated into the synthesis of the findings. In this way quality assessment scores helped identify studies whose results may have been influenced by aspects of their methodology and/or design.

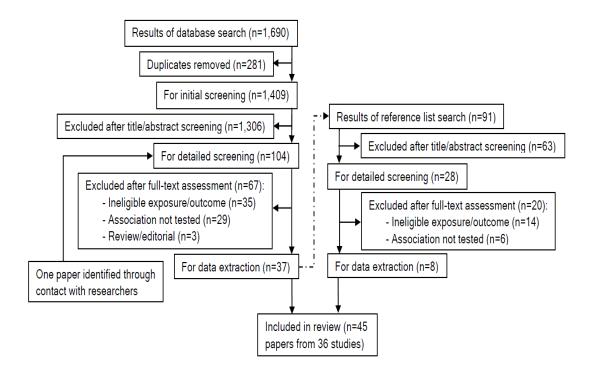
3.2.6 Synthesis

Tables were used to summarise the characteristics and results of included studies. A meta-analysis was initially planned but not attempted as there was considerable variation between studies in the method of reporting results for this to be possible.

3.3 Results

Results of the database searches were merged and duplicates removed leaving 1,409 papers available for title and abstract screening, of which 104 (includes one paper identified through contact with researchers) were eligible for detailed full-text assessment. Sixty-seven articles were subsequently excluded following detailed screening leaving 37 available for data extraction. A further eight eligible papers were identified through reference lists bringing the total number included in this review to 45 papers from thirty-six study samples (Figure 3.1).

Figure 3.1 PRISMA study flowchart (search up to 15th Dec 2014).



3.3.1 Characteristics of Included studies

Characteristics of the included papers are presented in Table 3.1. Most (34/45) were based on European samples including eighteen UK papers reporting on ten different study populations. Nordic countries (eleven papers each from a unique study sample) were the next most common European setting (four from Finland, three from Denmark and two from each of Norway and Sweden), and there were two papers each (from 2 studies) from Belgium and the Netherlands and one paper from Spain. The remaining papers were eight US, one Australian, and two Chinese papers (both reporting findings from the Guangzhou Bio-bank study (GBCS)). Some included papers did not address the review's question as the primary association of interest and treated PA as a confounding or mediating factor but presented relevant associations (157-165).

Study sample sizes varied from 112 to 20,086 and mostly comprised adults whose LTPA was ascertained in midlife. Birth years were from the early 1900s to 1980 and participants were mostly drawn from the general population though four study populations were sampled from occupational settings (160, 161, 166-170). The

majority of included papers (n=34) had a medium quality score (3 to 5) although the range was considerable (0.5 to 7).

Twenty-nine papers (22 studies) relied on participants recalling childhood SEP and in sixteen (14 studies) it was ascertained prospectively. For this review, different measures of childhood SEP were grouped into a) parental occupation, b) parental education and c) indices (combining >1 measure) and other indicators of childhood SEP (e.g. car access). Eight papers (7 studies) (154, 157, 171-175) present results from at least two of the above and four (4 studies) (172, 173, 176, 177) report associations for each group of childhood SEP measures. PA was measured by self-report with the exception of Beunen *et al.* (176) who present both accelerometer and self-reported outcomes. Questions used to collect PA ranged from single-items (177-179) to detailed questionnaires (93). Not all outcomes were LTPA-specific as three papers present outcomes conflating work-related activity and LTPA (157, 174, 180) and some provide no description of what PA domains are included in their outcome (but which are assumed to include LTPA) (158, 163, 181).

3.3.2 Results of included studies

Association between childhood SEP and adult LTPA

Results were presented as prevalence of LTPA by childhood SEP group, correlation between SEP and LTPA or regression coefficients from statistical models. Overall, results tended to either support the hypothesis that a lower childhood SEP is related to less frequent adult LTPA or to find no association. Two studies found evidence of an association between lower childhood SEP and higher adult PA outcomes (174, 182). Results are summarised by three groups of childhood SEP indicators (Tables 3.2-3.4).

Parental occupational class

Thirty papers (22 studies) tested the association between parental occupation during childhood and adult LTPA and twenty-one (16 studies) found evidence that a lower parental occupational class was associated with less frequent LTPA during adulthood (Table 3.2). All UK studies used versions of the Registrar General's Classification (RGSC) to categorise parental occupations into usually four or two groups. Studies

from other countries used similar categorisations to those of the RGSC although several considered farming occupations as separate groups (177, 183-186).

Evidence was available from three British birth cohorts initiated in 1946, 1958 and 1970. A higher prevalence of sports participation in higher paternal occupational groups was reported at age 36 in women from the MRC NSHD; the dataset which was introduced in chapter 2 and used for analyses in subsequent chapters of this thesis (93). Later findings from this cohort (174) showed similar trends for LTPA derived by latent classes in both men and women and trends in the opposite direction for a combined walking during work and pleasure outcome (Table 3.2). Genderadjusted analyses from the next oldest British birth cohort born in 1958, the National Child Development Study (NCDS), showed that a lower parental occupational class was associated with less LTPA at ages 33, 42 and 50 years (173). This association was fully attenuated at age 33 after accounting for other early life factors and following further adjustments (including for own adult SEP), it was only seen at age 50 (173). A second NCDS paper reported a non-significant correlation between parental occupation and exercise at age 50 (159). Father's occupational class measured three times during early life was associated with LTPA at age 34 in the 1970 British cohort study (187).

Manual father's occupation was associated with less LTPA and more inactivity in men (165, 181) and women (171, 175, 188) from the British Regional Heart Study (BRHS) and British Women's Heart and Health Study (BWHHS) respectively. After accounting for age and own occupational class (181, 188), this association was only found in the BWHHS (188). A higher prevalence (167) and higher odds (170) of inactivity were reported in lower parental occupational groups of the Whitehall II study. This association was considerably attenuated and no longer significant following adjustment for adult SEP (167) and a third paper from this cohort reported no difference in levels of inactivity between manual and non-manual parental occupations (160). Findings from the West of Scotland Collaborative Study suggest less exercise in lower parental occupations (166, 169). A weak correlation between higher paternal occupational groups and more LTPA was reported in the Lothian Birth cohort 1936 (LBC1936) and no association was found in analysis adjusted for adult SEP (172). In the Mid-span family study, manual and non-manual groups did not differ by levels of inactivity (189), but the prevalence of sports and exercise was higher in higher father's occupational groups of a Scottish survey (164).

Several Nordic studies reported null findings including Danish (190) and Finnish (185) birth cohorts, a Norwegian study (186), and an analysis of 34 year old Swedes (191). The latter (191) found that women but not men from non-manual paternal backgrounds spent more metabolic equivalent hours/week in LTPA compared with those of manual father's occupations. Higher and lower father's occupational groups were less and more respectively, inactive than the mean level of activity of employed Swedes, but this was not tested at a high significance level (p<0.10) (183). Mostly null findings were reported in the Finnish Health 2000 Survey however, men from lower paternal occupational groups were found to be more inactive than those from higher groups and women with mothers in manual occupations were more likely to be only moderately active compared with daughters of office employee mothers (177).

Dutch adults living near Eindhoven from lower paternal occupational strata were more likely to be inactive and less likely to be frequently active during leisure-time compared with those from families with professional backgrounds (192). After accounting for own occupational class, this association remained for frequent LTPA in women only (192). A Dutch study that only included men from Eindhoven found no difference by parental occupation in the prevalence of activity (162). In Belgian men, father's occupation was associated with leisure-time but not sports or accelerometer indices (176). Age-adjusted findings from an older Spanish sample showed that lower father's occupational groups were more likely to be inactive than higher groups and the association was more evident in women following adjustment for own occupational class (184). Compared with the manual group, non-manual father's occupational groups of a large US sample had a higher prevalence of vigorous exercise (157).

Parental education

Fourteen papers (13 studies) present associations for parental years or level of education and ten (9 studies) found evidence of an association between lower levels of parental education and less frequent LTPA (Table 3.3).

Similar trends to those found for occupation were reported in the NSHD, i.e. less LTPA (93, 174) and more walking (during work and pleasure) (174) in lower parental educational groups. Analysis adjusted for own education showed that those with more highly educated mothers were more active in sports at age 36 (93) but no difference was found when the highest maternal educational group was compared to

the lowest (Table 3.3). Gender-adjusted NCDS analyses comparing those without and with two minimally educated parents showed that the latter were more likely to be physically inactive at ages 33, 42 and 50 years (173). This association was fully attenuated at ages 33 and 40 after other early life factors were included in the analysis and likewise at age 50 following further adjustments including for own adult SEP (173). More parental years in education were weakly correlated with more LTPA in LBC1936 but adjusted analysis did not find an association (172).

Parental education was unrelated to leisure-time physical inactivity in a Danish sample (193), and null-findings were reported by two Finnish studies (177, 194). However, one of the latter (177) found that Finnish women with a primary-level educated parent were more likely to be inactive compared with those with a secondary-level educated parent. LTPA (at age 33 only) was associated with parental education in a Norwegian study but not after adjustment for own education (178). Belgian men's father's education was related to their self-reported sports and leisure-time activity but not accelerometer indices (176). A lower parental education in US adults was associated with less prevalent vigorous exercise (157) and with higher adjusted-odds of low exercise (180). Higher parental education was correlated with higher estimated exercise energy expenditure in a Pennsylvanian sample (163). Three measures of parental education were unrelated to exercise in women physicians born in the US (168) and there was no relationship between parental education and LTPA in an Australian study (195).

Indices and other measures of childhood SEP

Fourteen papers (12 studies) tested associations between indices and other measures of childhood SEP and adult LTPA and seven (6 studies) found an association between a lower childhood SEP and less frequent LTPA in adulthood (Table 3.4).

An index measuring household characteristics and car access during childhood was unrelated to LTPA in LBC1936 [(172) but four measures of housing characteristics and car access were each associated with LTPA in the BWHHS (171, 175). Combining these four indicators and paternal occupation into a summary variable showed that with increasing childhood socioeconomic adversity, women were more likely to be low exercisers (175) and less likely to be more physically active (171). Having more limited household amenities was related to leisure-time physical inactivity at ages 33, 42 and 50 years in NCDS, but not at age 42 when gender was taken into account (173). After adjustment for a range of early life factors, this association was only found at age 50 and associations were considerably attenuated and were no longer observed at any age following the addition of adult covariates, including own SEP, to the analysis (Table 3.4).

Compared with those ranked middle or poor on an index of parental occupation, education, external perceptions of wealth, and housing characteristics, Finnish men who ranked high on the index were less likely to be in the lowest quartile of conditioning activities (196). No difference was found when the prevalence of inactivity was compared in this sample (196). A different Finnish study reports no association between long-term financial problems or regular parental unemployment and LTPA in adults (177). More urban locations of Belgian men's childhood homes were related to higher accelerometer counts but not to any self-reported outcomes (176). An index of parental occupation and education was not associated with Belgian women's sports participation (176).

Increasing disadvantage as indicated by an index of parental education, childhood welfare status and financial level growing up was associated with less participation in vigorous exercise in US adults (179). This association was attenuated but remained statistically significant following adjustment for own adult SEP (179). An index of parental occupation and education was unrelated to activities and hobbies of a Californian sample (197), but in older US adults (198) a higher childhood SEP, indicated by a similar index that included parental income, was associated with more exercise at age 65. The authors tested the role of mediating factors and report that own SEP explained almost half of this association (198). No correlation was found in 112 US participants between a similar index and estimated activity energy expenditure (158). Findings from the GBCS suggest a higher prevalence of inactivity (and a lower prevalence of LTPA) in Chinese participants with more parental possessions during their childhood (182, 199).

3.4 Discussion

3.4.1 Summary of results

This systematic review included 45 papers from 36 study samples and found evidence of less frequent LTPA in adults from less advantaged childhood

socioeconomic backgrounds. Twenty-two studies report results that associate a less advantaged childhood SEP with less frequent adult LTPA; thirteen studies report no association. 61.1% of papers (9/16 studies) that presented results adjusted for own adult SEP reported statistically significant associations between childhood SEP and adult LTPA (Tables 7-9). Studies presenting results before and after adjustment for adult SEP found that accounting for own adult SEP typically partly attenuated associations (Tables 7-9). Compared with the eleven Nordic studies, findings from the ten UK samples tended to be more supportive of the review's hypothesis of less frequent LTPA in adults from lower childhood socioeconomic groups with Nordic studies presenting more null findings (Tables 7-9). Gender-stratified analyses showed more evidence of an association in women compared with men (Tables 7-9). Findings did not differ systematically by type of childhood SEP indicator or age at assessment of LTPA (Tables 7-9).

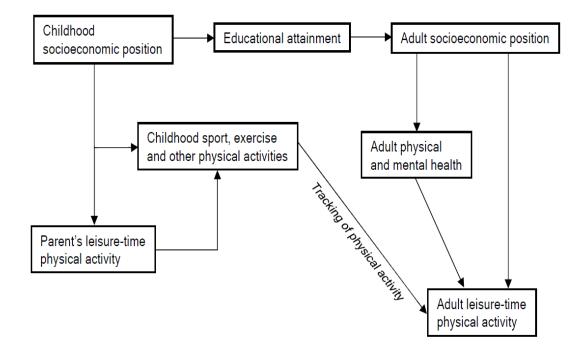
3.4.2 Explanation of findings

Existing reviews link a lower childhood SEP to a wide range of disadvantageous adult outcomes, including physical capability (200), cardiovascular disease (30) and mortality (201). Reviews focusing on different life stages have shown that from childhood through to old age, in cross-sectional analyses, lower socioeconomic groups tend to participate less in LTPA than more advantaged groups (29, 146, 147). In addition to participating less in LTPA during childhood (147), a study of over 2,000 Dutch adults provides evidence that children from lower socioeconomic backgrounds have a lower likelihood of initiating a sport throughout their lives (202).

One possible reason for finding an association between a lower childhood SEP and less frequent adult LTPA is due to the continuity of SEP across life. A lower childhood SEP tends to restrict future SEP (203), partly by predisposing to social pathways operating across life which can limit educational opportunities and ultimately socioeconomic potential, e.g. in occupational class, income and wealth (148). These pathways can influence the availability of, and a person's response to, opportunities for the development of LTPA (148). Furthermore, a recently completed systematic review of 14 studies has shown that those exposed to lower SEP in both childhood and adulthood tend to participate less in LTPA when compared with those who experience upward intergenerational social mobility (204); thus supporting the notion that SEP appears to cumulatively influence LTPA.

Associations between childhood SEP and adult LTPA were reported in several analyses which were adjusted for own adult SEP (Tables 7-9) suggesting that complementary pathways are likely to be involved (Figure 3.2). Participation in sports and exercise in early life tends to be socioeconomically patterned (147) and tracks into adulthood (91), potentially forming an important determinant of adult LTPA. Since adult LTPA also displays a socioeconomic gradient (29, 146), children of lower SEP are likely to have less physically active parents who may in turn unfavourably influence their own children's involvement in LTPA (205). Childhood socioeconomic circumstances may influence the acquisition of sets of interpersonal skills such as decision making, self-efficacy and self-esteem which can help people maintain health behaviour such as LTPA (92). Socioeconomic differences in children's growth and motor development (206) could also contribute to differences in subsequent LTPA.

Figure 3.2 Hypothesised pathways explaining associations found between childhood socioeconomic position and adult leisure-time physical activity.



Twelve studies presenting only null findings do not support the review's hypothesis (Tables 3.2 to 3.4. Participation in sports and exercise is linked to a range of factors other than SEP, including genetics (95), life transitions, culture and policy (68), some of which could play a greater role in determining participation. Evidence for less tracking of LTPA when compared with other health behaviours such as sedentary

behaviour (207) supports this argument although measurement error could explain the lower tracking of LTPA (91). Associations may vary by setting and cohort due to varying influences on LTPA by these factors and are also likely to be influenced by study quality.

3.4.3 Sources of heterogeneity

Inconsistent findings could be due to differences between studies including in design and risk of bias. Despite overall medium study quality, considerable variation between studies in the assessment and formulation of LTPA (Table 3.1) and adjustment for potential confounders (Tables 7-9) can influence associations. Small sample sizes (176, 208) may lead to underpowered studies while multiple tests (176-178) risk detecting false associations. Some of the null findings reported (169, 189) may be due to heterogeneity within childhood SEP groups as a result of using dichotomous indicators. Null findings from the Women Physician Health Study (168) might reflect insufficient variation in childhood socioeconomic background.

Results did not appear to vary by the method of ascertainment of childhood SEP however, using recalled measures of childhood SEP can underestimate associations (209). There was little evidence that the type of childhood SEP indicator used was a source of heterogeneity, suggesting that each indicator sufficiently captures the same underlying construct or that the various aspects of SEP are equally important. This is a similar observation to that of a previous review of European adults (29) but contrary to an earlier and geographically wider review which found education to be more strongly associated with contemporaneous LTPA (146).

Sex differences in the association between childhood SEP and adult LTPA might exist. Like some studies in this review, a previous review found more evidence of an association in women than men between adult SEP and LTPA (146). Absence of a sex difference in how childhood SEP relates to adult's capacity to undertake exercise (200) suggests that the sex differences found in this review are likely explained by social rather than biological pathways.

The tendency for Nordic studies to find less evidence of association compared with UK studies might be due to less variation between socioeconomic groups in Nordic cohorts than in the UK (142). There could in addition be differences in the meaning of occupation between these settings, e.g. in Nordic cohorts, where there was more

prevalent farming occupations (177, 183, 185, 186), SEP could be indicating how urban or rural is the environment, which may be independently related to LTPA (210).

More walking during work and pleasure in lower childhood SEP groups of the NSHD (174) might be explained by the inclusion of work-related PA as part of the outcome, which can be inversely associated with SEP (29). Socioeconomic patterns of LTPA that are different to those usually observed in Western countries have been documented in China (211), which could explain the GBCS findings (182).

3.4.4 Implications of findings

Due to heterogeneity in findings, a better understanding of how childhood SEP relates to adult LTPA is required. Future studies should use prospectively ascertained childhood SEP where this is feasible and examine more detailed measures of LTPA as has been done in NSHD (174). Data from activity monitors could be used in conjunction with questionnaires to derive more holistic LTPA variables that capture parameters such as activity type, energy expenditure and time of day/week that activity is performed. Intra-individual levels of LTPA can fluctuate over time and future research could in addition explore associations with patterns or change in LTPA, as well as different types of LTPA. Strategies for maximising participant retention in long-running studies should be considered so as to minimise bias due to loss to follow-up. To better characterise how associations vary by time and place, age, country, cohort and period differences should be formally tested while accounting for methodological differences. Testing hypothesised pathways (Figure 3.2) can aid our knowledge of how childhood SEP relates to adult LTPA.

Despite the inconsistencies described, childhood socioeconomic circumstances can influence health throughout life (148) and any interventions to improve them are likely to have wide ranging benefits beyond potentially advantaging adult LTPA. As well as improving early life circumstances, intervening to promote adult LTPA could be one means to cut the link between a disadvantaged childhood SEP and poor adult health. Effectively promoting adult LTPA amongst those disadvantaged in childhood may in turn require a better understanding of the mechanisms linking childhood disadvantage to adult LTPA.

3.4.5 Strengths and limitations of the review

Strengths of this review are the systematic process followed to identify and extract data from eligible studies and the searching of multiple databases and reference lists. Double screening, data extraction and quality assessment helped reduce the potential for errors associated with a single reviewer. Limitations include search restrictions to English language and to journal publications, which may introduce publication bias. The fact that presented results were not sufficiently comparable to be combined in a meta-analysis could be considered a limitation and this also meant publication bias could not be formally assessed using funnel plots. However, the inclusion of all papers even where the review's question was not the primary aim, and the findings of no association between childhood SEP and adult LTPA in seventeen papers suggests publication bias is unlikely.

3.4.5 Conclusions

This systematic review found evidence from the NSHD and other studies of an association between less advantaged SEP in childhood and less frequent LTPA in adults (particularly among women and in UK cohorts) but considerable heterogeneity between studies was detected. Future studies should examine more detailed measures of LTPA, investigate underlying pathways and explore country differences. The findings suggest the need to provide additional opportunities and support to enable children from socioeconomically disadvantaged backgrounds to develop and maintain more active leisure pursuits and participate in sports and exercise across life. This systematic review's findings are taken into consideration when addressing the remaining thesis objectives in the following chapters focusing on developmental factors and drawn together in chapter 7.

Table 3.1 Characteristics of the included studies: arranged by region/country and from older to younger age at measurement of physical activity

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Johnson (2011) (172) -UK (1936) -Lothian Birth Cohort 1936 (LBC 1936)	-Scottish birth cohort. -70 years. -1,091 (49.8%).	-PO (main occupation), PE, I&O (number of people per room, shared toilet facilities, whether indoor/outdoor toilet). -Recalled by SM at age 70.	-Level of physical activities such as household chores, keep-fit, heavy exercise and sport. -Physical activity six point score.	3.5
- Lawlor (2004) (188) -UK (1921-40) -British Women's Heart & Health Study (BWHHS)	-Cross-section of women recruited from GP lists in 23 British towns. -60-79 years. -3,444 (100%).	-PO (longest held occupation). -Recalled by SM at age 60-79.	 Hours per week spent on several types of domestic, recreational and sports activities. Physically inactive (< 1 hour/wk. of moderate or vigorous physical activity). 	4
- Hillsdon (2008) (171) -UK (1921-40) -BWHHS	-Cross-section of women recruited from GP lists in 23 British towns. -60-79 years. -4,103 (100%).	-PO (longest held occupation), I&O (house with bathroom; hot water; shared bedroom, car access, and an index of all the above). -Recalled by SM at age 60-79.	-Hours per week spent on several types of domestic, recreational and sports activities. -Moderate to vigorous physical activity hours/wk.	5
- Watt (2009) (175) -UK (1921-40) -BWHHS	-Cross-section of women recruited from GP lists in 23 British towns. -60-79 years. -3,523 (100%).	-PO (longest held occupation), I&O (house with bathroom; hot water; shared bedroom, car access, and an index of all the above). -Recalled by SM at age 60-79.	 Hours per week spent on several types of domestic, recreational and sports activities. Low exercise (< 2 hours/wk. of moderate or vigorous physical activity). 	4.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Ramsay (2009) (165) -UK (1920s-30s) -British Regional Heart Study (BRHS)	-Cross-section of men recruited from GP lists in 24 British towns. -52-74 years. -5,188 (0%).	-PO (longest held occupation). -Recalled by SM at age 52-74.	 Hours per week spent on several types of physical activities including walking, cycling and sports. Physically inactive (none or occasional physical activity). 	2.5
- Wannamethee (1996) (181) -UK (1920s-30s) -BRHS	-Cross-section of men recruited from GP lists in 24 British towns. -40-59 years. -2,188 (0%).	-PO (longest held occupation). -Recalled by SM at age 52-74.	-No description (reference provided). -Physically active.	5
- Stringhini (2013) (170) -UK (1930-53) -Whitehall II (WHII) Study	-Cohort of civil servants employed in London. -40-59 years (phase 3). -6,387 (28.5%).	-PO (main occupation). -Recalled by SM at age 35-55.	 Hours per week spent on moderate and vigorous physical activities. Physically inactive (≤ 1 hour/wk. of moderate and ≤ 1 hour/wk. of vigorous physical activity). 	2
- Heraclides (2008) (160) -UK (1930-53) -WHII Study	-Cohort of civil servants employed in London. -44-69 years (phase 5). -4,598 (26.8%).	-PO (main occupation). -Recalled by SM at age 35-55.	-Hours per week spent on several types of domestic, recreational and sports activities. -Sedentary lifestyle (low quintile of MET score).	3.5
- Brunner (1999) (167) -UK (1930-53) -WHII study	-Cohort of civil servants employed in London. -35-55 years (phase 1). -6,980 (31.6%).	-PO (main occupation). -Recalled by SM at age 35-55.	-Hours per week spent on several types of domestic, recreational and sports activities. -Physically inactive (no moderate or vigorous activities).	3.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Blane (1996) (166) -UK (1908-37) -West of Scotland Collaborative Study	-Cross-section of men employed in 27 Scottish work places. -35-64 years. -5,645 (0%).	-PO (main occupation). -Recalled by SM at age 35-64.	-Hours per week spent on exercise outside work including walking, gardening and golfing. -Exercise hours/wk.	3
- Hart (1998) (169) -UK (1908-37) -West of Scotland Collaborative Study	-Cross-section of men employed in 27 Scottish work places. -35-64 years. -5,567 (0%).	-PO (main occupation). -Recalled by SM at age 35-64.	-Hours per week spent on exercise outside work including walking, gardening and golfing. -Exercise hours/wk.	2.5
- Popham (2010) (164) -UK (1949-68) -2003 Scottish Health Survey	-Cross-section of Scottish residents. -35-54 Years. -2,770 (% unknown).	-PO (when SM was aged 14) -Recalled by SM at age 35-54.	 -Frequency of several types sports and exercises during previous 4 weeks. -Sport and exercise (participated ≥ once in sport/ exercise at moderate/high intensity for ≥ 15 min/day). 	2.5
- Hart (2008) (189) -UK (1937-66) -Mid span family Study	-Cross-section of the 1970s Renfrew/Paisley Study offspring. -30-59 years. -2,338 (55.5%).	-PO. -Reported by parents (SM was aged 6-39).	 Frequency of daily activity and physical activity outside work. No exercise (not very/at all active in daily activities and active for < once/wk. or never outside of work). 	5.5
- Silverwood (2012) (174) -UK (1946) -MRC National Survey of Health and Development (NSHD)	-British birth cohort. -36-53 years. -3,847 (49.6%).	-PO, PE. -Reported by parents (SM was aged 4 and 6).	-Latent classes for a) walking during work and pleasure b) cycling during work and pleasure and c) LTPA. -LTPA (low, gardening & DIY, sports), walking, cycling (low, high).	5.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Kuh & Cooper (1992) (93) -UK (1946) -MRC NSHD	-British birth cohort. -36 years. -2,144 (50.3%).	-PO, PE. -Reported by parents (SM was aged 4 and 6).	 Frequency of several types of sports and recreational activities during previous month. High participation in sport and recreational activities. 	7
- Pinto Pereira (2014) (173) -UK (1958) -National Child Development Study 1958 (NCDS)	-British birth cohort. -33, 42, 50 years. -12,776 had ≥ one measure of LTPA (exact numbers at each age not provided).	 -PO, PE, I&O (index of household amenities: availability of bathroom, indoor lavatory and hot water). -Reported by parents at SM's birth and when aged 7, 11 and 16. 	-Frequency of LTPA such as swimming, going for walks. -Low LTPA (LTPA < once/wk.)	6
- Cheng & Furnham (2013) (159) -UK (1958) -NCDS	-British birth cohort. -50 years. -5,921 (49.4%).	-PO (current or last held occupation). -Reported by parent at SM's birth.	-Frequency of physical exercise. -Exercise score (6-point scale).	3
- Juneau (2014) (187) -UK (1970) -1970 British Cohort Study	-British birth cohort. -34 years. -9,624 (52.2%).	-PO. -Reported by parents at SM's birth and when aged 5 and 10 years.	-Frequency of LTPA during the previous eight weeks. -Estimated LTPA energy expenditure.	5
- Osler (2008) (190) -Denmark (1953) -Metropolit Birth Cohort	-Danish birth cohort of men from Copenhagen. -51 years. -6,292 (0%).	-PO. -Extracted from birth records.	 Frequency of walking, running, cycling and other activities. Sedentary leisure activity (mainly reading, watching TV or having other sedentary activities during leisure). 	6.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Lynch (1997) (196) -Finland (1920s-40s) -Kuopio Ischaemic Heart Disease Risk Factor Study	-Cross-section of men from Eastern Finland. -42-60 years. -2,682 (0%).	-I&O (index of PO, PE, whether family perceived as wealthy, whether family lived on a farm and size of farm). -Recalled by SM at age 42-60.	-Energy expended in LTPA during the previous 12 months, e.g. jogging, swimming, cycling, skiing. -(i) No conditioning activities (ii) Low quartile of conditioning activities.	2.5
- Kvaavik (2011) (178) -Norway (1964-8) -Oslo Youth Study	-Follow-up of Oslo students invited to a health education intervention. -25, 33, 40 years. -240, 329, 407.	-PE. -Reported by parents (SM aged 11-16).	-'How often do you exercise for at least half an hour to the extent that you sweat and/or are short of breath?' -LTPA (twice/wk.).	6
- Jørgensen (2013) (161) -Denmark (≈1971) -Danish Health Care Worker Cohort	-Cohort of Danish women employed as social and health care assistants. -35.4 years (SD=10.5) -1,661 (100%).	-PO (when SM was aged 14). -Recalled by SM at age 35.4.	-Hours per week spent on LTPA. - Low LTPA (<4 hours/wk.).	0.5
- Barnekow-Bergkvist (1998) (191) -Sweden (1958)	-Follow-up of Swedish students. -34 years. -278 (43.5%).	-PO. -Recalled by SM at age 34.	-Hours per week spent on LTPA (includes sports, walking, and cycling) in the previous 12 months. -LTPA MET hours/wk.	3
- Tammelin (2003) (185) -Finland (1966) -North Finland Birth Cohort 1966	-Northern Finland birth cohort. -31 years. -7,794 (53%).	-PO (when SM was aged 14). -Reported when SM aged 14 (unclear if reported by parents/SM).	-Frequency of light and brisk LTPA. -Physically inactive (brisk LTPA < once/wk. and light LTPA <4 times/wk.).	5.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Makinen (2009) (177) -Finland (1970 & older) -Health 2000 Survey	-Regionally stratified cross-section of Finnish adults. -30+ years. -7,112 (55.4%).	-PO, PE, I&O (long-term financial problems in family, regular parental unemployment – both before age 16). -Recalled by SM at age 30+.	-How much do you exercise and strain yourself physically in leisure time?' -Inactive (read, watch TV or do other activities that do not strain me physically); moderately active (walk, cycle or move in other ways for at least 4 hours/wk.).	3.5
-Wichstrøm (2013) (186) -Norway (1973-80) -Young in Norway Study	-Follow-up of students from 67 Norwegian schools. -25-32 years. -2,890-2,923.	-PO. -Reported by SM at age 12-19.	-Hours spent on physical exercise during the previous week. -LTPA hours/wk.	4.5
- Leino (1999) (194) -Finland (1962-71) -Cardiovascular Risk in Young Finns Study	-Follow-up of Finnish children and adolescents. -21-30 years. -432 (53.7%).	-PE. -Reported by SM at age 9-18.	-Frequency and duration of exercise used to form an LTPA index. -Physically inactive ($\leq 25^{th}$ percentile of LTPA index, range = 0–52).	4
- Osler (2001) (193) -Denmark (1961-73) -offspring of Copenhagen City Heart Study (CCHS)	-Follow-up of CCHS offspring aged 6-18 at baseline. -19-31 years. -317 (48.9%).	-PE. -Reported by parents (SM aged 6-18).	-Current level of participation in LTPA and whether active in sports. -Low LTPA (mostly sitting or light activity for ≥4 hours/wk. and not active in sports).	7
- Peck (1994) (183) -Sweden (1900s-60s)	-Cross-section of employed Swedes. -16-74 years. -12,695 (50.4%).	-PO (during SM's childhood). -Recalled by SM at age 16-74.	-Regular LTPA (no description). -No regular LTPA.	1.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Regidor (2004) (184) -Spain (1940 & older)	-Cross-section of an older Spanish population. -60+ years. -3,658 (54.6%).	-PO. -Recalled by SM at age 60+.	-Type of physical activity done in spare time or at any time if retired/unemployed. -Physically inactive (only report sedentary activities e.g. reading, watching TV).	4.5
- Beunen (2004) (176) -Belgium (1956) -Leuven Longitudinal Study of Flemish Boys	-27-year follow-up of Flemish speaking adolescent Belgian boys. -40 years. -166 (0%).	-PO, PE, I&O (degree of urbanisation). -Reported by SM at age 14-18.	-Frequency of sports, activities during leisure-time and accelerometer- measured counts of daily physical activity. -Sport, leisure-time, & counts indices.	5.5
- Scheerder (2006) (208) -Belgium (1961-7) -Leuven Longitudinal Study of Flemish Girls	-20-year follow up of Flemish speaking adolescent Belgian girls. -32-41 years. -257 (100%).	-I&O (index of PO and PE). -Reported by SM at age 12-18.	-Hours per week spent on sports during the previous year. -Level of sports participation (hours/wk./Yr.).	6
- Kamphuis (2013) (162) -Netherlands (1916-51) -GLOBE Study	-Cross-section of men living in or near Eindhoven. -40-75 years. -4,894 (0%)	-PO (when SM was aged 12). -Recalled by SM at age 40-75.	-Hours per week spent on transport, leisure-time and sports related activities. -Physically active (≥3.5 hours/wk. of sports and transport or leisure-time physical activity).	2
-van de Mheen (1998) (192) -Netherlands (1910s- 60s) -Longitudinal Study on Socio-Economic Health Differences	-Cross-section of adults living in or near Eindhoven. -25-74 years. -13,854 (% unknown).	-PO (when SM was aged 11). -Recalled by SM at age 25-74.	-Leisure-time physical exercise (no description). -Frequent LTPA, and no LTPA.	3.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
- Pudrovska (2013) (198) -US (1939-40) -1957 Wisconsin Longitudinal Study	-Long-term follow-up of high school graduates from Wisconsin. -65 years. -5,778 (54.7%).	-I&O (index of PO, PE, family income, father's occupational income and father's occupational education). -Reported when SM was aged 17-18.	-Hours per month spent on light (e.g. walking, gardening, golfing) and vigorous (e.g. aerobics, jogging, swimming) physical activities. -Physical activity index.	6
- Wray (2005) (180) -US (1941 & older). - Health & Retirement Study (HRS); Study of Asset & Health Dynamics (AHEAD)	-Follow-up of middle aged and older US adults. -51-61 years (HRS); 70+ years (AHEAD). -HRS: 6,106 (57%); AHEAD: 3,636 (63%).	-PE. -Recalled by SM at age 51-61 (HRS) and 70+ (AHEAD).	-Whether or not SM is a vigorous exerciser. Includes heavy housework, cycling, aerobics, running, jogging, swimming and physical labour at work. -Low physical activity (not exercising ≥3 times/wk.)	5
- Bowen (2010) (157) -US (1941 & older) -HRS merged with AHEAD and two other cohorts	-Cohort of middle aged and older US adults. -51+ years. -18,465 (60%).	-PO (main occupation), PE. -Recalled by SM at age 51+.	-Whether or not SM is a vigorous exerciser. Includes heavy housework, cycling, aerobics, running, jogging, swimming and physical labour at work. -Vigorous exercisers (≥3 times/wk.).	3
- Carroll (2011) (158) -US (1950s-70s) -Vaccination Immunity Project	-Cross-section of Pennsylvanian adults recruited to a Hepatitis B vaccination project. -40-60 years. -153 (59.8%).	-I&O (index for every 2 years of childhood: whether parents owned home, number of a) bathrooms, b) people living in the home and c) vehicles owned). -Recalled by SM at age 40-60.	-Paffenbarger physical activity questionnaire (no description). -Physical activity kilocalories expended per week.	1
- Frank (2003) (168)	-Cross-section of women	-PE.	-Exercise (no description).	0.5

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^c
-US (1930-50) -Women Physician Health Study	physicians born in the US. -30-70 years. -2,884 (100%).	-Recalled by SM at age 30-70.	-Exercising ≥30 minutes on 3 times per week.	
- Tsenkova (2014) (179) -US (1921-70) -Midlife in the US Study	-Cross-section of US adults who participated in a biomarkers study. -25-74 years. -895 (54.6%)	-I&O (index of PE, childhood welfare status and financial level growing up). -Recalled by SM at age 25-74.	-'How often do you engage in vigorous physical activity long enough to work up a sweat (e.g. running/heavy lifting)?' -Exercise sessions per month.	3
- Kern (2010) (197) -US (1910s) -Terman Life Cycle Study	-Long-term follow-up of Californian children with high IQ. -25-61 years. -1,114 (50%).	-I&O (index of PO and PE). -Reported by parents (SM was aged 11).	-Avocational activities and hobbies including sport, gardening, music, art, writing, photography. -Average physical activity METs.	4.5
- Phillips (2009) (163) -US (1940s-70s) -Adult Health and Behaviour Project	-Cross-section of Pennsylvanian adults without serious illnesses. -30-54 years. -811 (51.4%).	-PE. -Recalled by SM at age 30-54.	-Paffenbarger physical activity questionnaire (no description). -Physical activity kilocalories expended per week.	2.5
- Schooling (2007) (182) -China (1955 & older) -Guangzhou Bio-bank Cohort Study (GBCS)	-Cross-section of Guangzhou community club members. -50+ years. -9,748 (71.9%).	-I&O (number of parental possessions from a watch, sewing machine and bicycle during SMs' childhood). -Recalled by SM at age 50+.	 -IPAQ used (no description). -Inactive, minimally active, and HEPA (vigorous activity ≥ 3 days/wk. at ≥ 1,500 MET minutes/wk, or activity 7 days/wk. at ≥ 3,000 MET minutes/wk.). 	3
- Elwell-Sutton (2011) (199)	-Cross-section of Guangzhou community	-I&O (number of parental possessions from a watch,	-IPAQ used (no description). -Inactive, minimally active, and HEPA	3

-1 st Author (year) -Country ^a (birth year/s) -Study name	-Description -Age at physical activity assessment -Sample size (% female)	-Childhood socioeconomic indicator/s ^b -How these were ascertained ^b	-Physical activity measurement ^c -Outcome/s of interest ^c	QA score ^d
-China (1955 & older) -GBCS	club members. -50+ years. -20,086 (73.2%).	sewing machine and bicycle during SM's childhood). -Recalled by SM at age 50+.	(vigorous activity ≥ 3 days/wk. at ≥ 1,500 MET minutes/wk, or activity 7 days/wk. at ≥ 3,000 MET minutes/wk.).	
- Gall (2010) (195) -Australia (1970s) -Childhood Determinants of Adult Health Study	-20-year follow-up of the Australian Schools Health & Fitness Survey. -26-36 years. -1,973 (52.8%).	-PE. -Recalled by SM at age 26-36.	-Whether or not SM participated in ≥ 3 hours of moderate/vigorous LTPA per week. -LTPA (≥ 3 hours/wk.).	4.5

a UK: United Kingdom; US: United States; Nordic group of countries (Norway, Sweden, Finland and Denmark) considered as one region. b PO: Parental occupation (usually based on father's occupation, more detail can be found in brackets if provided in the paper); PE: Parental education (years and/or level); I&O: Indices and other measures of childhood socioeconomic position (SEP), includes (i) indices combining different indicators of childhood SEP and (ii) single measures which are distinct from parental occupation and education; SM: Study member. c LTPA: Leisure-time Physical Activity; METs: Metabolic equivalents; IPAQ: International Physical activity Questionnaire; HEPA: Health enhancing physical activity: acronym used in the two GBCS papers [61-62]. d QA score: Quality assessment score (average of two assessor's scores possible values are 0 – 9).

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
- Johnson (2011) -UK; Lothian Birth Cohort 1936 -1,091; 70+ Yrs.	Correlation and regression coefficients for a 6-point LTPA score and parental occupation (RGSC 1951: I, II, IIIN, IIIM, IV, V) (per unit change from high to low occupational class in regression model).	r = -0.06 (+, p=0.05)	β = -0.01 (ns)	none education, own occupational class, other childhood SEP, IQ & more
-Lawlor (2004) -UK; British Women's Heart & Health Study (BWHHS) - $3,444^{\circ}$; 60- 79 Yrs.	Prevalence of physical inactivity in six parental occupational groups (RGSC 1980: I, II, IIIN, IIIM, IV, V) and odds of physical inactivity per unit increase from high to low occupational class.	I-IV = -11.4% (1, 3) (+)	OR = 1.17 (49, 50) (+) OR = 1.15 (112, 113) (+)	none age age, own occupational class
- Hillsdon (2008) -UK; BWHHS -4,103 ^ç ; 60- 79 Yrs.	Prevalence of manual parental occupational class (RGSC 1980) in four groups of physical activity hours/wk.	<u>% manual</u> <u>occupations:</u> ≥3-0 (hours/wk.) = - 7.4% {-6.1; -8.6} (+, p<0.001)		none
-Watt (2009)	Percentage difference in low	NM-M = -6.7%		none

Table 3.2 Results of studies testing the association between parents' occupational class and leisure-time physical activity (LTPA) in adults: arranged by region/country and from older to younger age at measurement of physical activity

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
[22] -UK; BWHHS -3,523 [♀] ; 60- 79 Yrs.	exercise between manual (M) and non-manual (NM) parental occupations (RGSC 1980).	{-2.5; -10.9} (+, p<0.01)		
- Ramsay (2009) -UK; British Regional Heart Study (BRHS) -5,188 ³ ; 52- 73 Yrs.	Prevalence of physical inactivity in manual (M) and non-manual (NM) parental occupations (RGSC 1980).	NM-M = -48% (+, p=0.05)		none
- Wannamethe e (1996) -UK; BRHS -5,516 ³ ; 40- 59 Yrs.	Prevalence of physical activity in manual (M) and non-manual (NM) parental occupations (RGSC 1980).	NM-M = 8% (+, p<0.0001) NM-M = 2.4% (ns)		none age, own occupational class
- Stringhini (2013) -UK; Whitehall II (WHII) Study -6,387; 40-59 Yrs.	Odds of physical inactivity in the lowest compared to the highest tertile of parental occupation (RGSC 1980).		OR = 1.37 {1.14; 1.65} (+, p<0.05)	age, sex, ethnicity, CHD, stroke cancer, hypertension, family history of diabetes

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Heraclides (2008) -UK; WHII Study -4,598; 44-69 Yrs.	Prevalence of physical inactivity in manual (M) and non-manual (NM) parental occupations (RGSC 1980).	<u>NM-M:</u> ♂ = 1.9% (ns) ♀ = 1.3% (ns)		none
- Brunner (1999) -UK; WHII Study -6,980; 35-55 Yrs.	Prevalence of physical inactivity in four parental occupational groups (RGSC 1980: I/II, IIIN, IIIM, IV/V).	I-IV (♂) = -4.8% (+, p=0.01) I-IV (♀) = -7.9% (+, p=0.02) I-IV (♂) = -2.6% (ns) I-IV (♀) = -2.9% (ns)		age age, own occupational class
- Blane (1996) -UK; West of Scotland Collaborative Study -5,646 ³ ; 35- 64 Yrs.	Prevalence and regression coefficients for mean exercise hours/wk. by four parental occupational groups (RGSC 1966: I/II, IIIN, IIIM, IV/V).	I/II- IV/V = 0.7 hours/wk. {SE: I/II =0.13; IV/V =0.16}	β = -0.16 {-0.32; 0.01} (ns)	age age
- Hart (1998) -UK; West of Scotland Collaborative Study -5,567 ^{<i>d</i>} ; 35-	Prevalence of exercise hours/wk. in four groups of parental and own occupations (RGSC 1966: 1. stable non-manual 2. moved up 3. moved down 4. Stable manual).	1-4 = 0.5 hours/wk. (+, p=0.002)		age

-1 st Author (year) -Country; study name -Sample size ^a ; age 64 Yrs.	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
- Popham (2010) -UK; 2003 Scottish Health Survey -2,770; 35-54 Yrs.	Prevalence of sport & exercise in four parental occupational groups (RGSC: I/II, IIIN, IIIM, IV/V).	I/II-IV/V = 18.6% {17.7; 19.6} (+)		age, sex
- Hart (2008) -UK; Mid span Family Study -2,338; 30-59 Yrs.	Prevalence of no exercise in manual (M) and non-manual (NM) parental occupations (RGSC 1966) and odds of no exercise per unit increase (1-6) from low to high parental occupational class.	<u>NM-M</u> : ♂ = 3.7% (ns) ♀ = -3.0% (ns)	<u>OR:</u> ♂ = 1.03 (0.91; 1.16) (ns) ♀ = 1.09 (0.98; 1.21) (ns)	none age
-Silverwood (2012) -UK; MRC National Survey of Health and Development (NSHD) ->3,300; 31-	Prevalence of LTPA (low; gardening; sport & leisure), walking and cycling during work & for pleasure (high, low) in four parental occupational groups (RGSC 1970: I/II, IIIN, IIIM, IV/V).	<u>I/II-IV/V: LTPA</u> <u>(sports & leisure):</u> ♂ = 12.2% (+, p<0.001) ♀ = 17.9% (+, p<0.001) <u>I/II-IV/V: Walking</u> <u>(high):</u> ♂ = -17.6% (-,		none

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
53 Yrs.		p<0.001) ♀ = -6.6% (-, p=0.002) <u>I/II-IV/V: Cycling</u> <u>(high):</u> ♂ = -1.0% (ns) ♀ = 2.9% (ns)		
-Kuh & Cooper (1992) -UK; MRC NSHD -2,977; 36 Yrs.	Prevalence of most active in sports & recreational activities in four parental occupational groups (RGSC 1970: I/II, IIIN, IIIM, IV/V).	<u>I/II-IV/V:</u> ♂ = 9.1% (ns) ♀ = 21.4% (+, p<0.001)		none
- Pinto Pereira (2014) -UK; National Child Development Study 1958	Odds of low LTPA per unit increase from high to low parental occupational class (RGSC 1951: I/II, IIIN, IIIM, IV/V).		<u>ORs:</u> age 33 = 1.12 {1.07; 1.16} (+) age 42 = 1.16 {1.11; 1.20} (+) age 50 = 1.23 {1.17; 1.29} (+)	none
(NCDS) -12,776 had ≥ one measure of LTPA; 33, 42, 50 Yrs.			age $33 = 1.06 \{1.01;$ $1.11\} (+)$ age $42 = 1.10 \{1.05;$ $1.15\} (+)$ age $50 = 1.13 \{1.07;$ $1.19\} (+)$	sex

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
			age 33 = 1.01 {0.97; 1.06} (ns) age 42 = 1.05 {1.002; 1.10} (+) age 50 = 1.09 {1.03; 1.15} (+)	sex, parental education, aptitude, household amenities, cognition, lifestyle factors age 16, & more
			age 33 = 1.00 (0.95; 1.05) (ns) age 42 = 1.04 (0.99; 1.09) (ns) age 50 = 1.07 (1.01; 1.13) (+)	as above plus own education, own social class, BMI, mental health, number of children in the household, limiting illness
- Cheng & Furnham (2013) -UK; (NCDS) -5,921; 50 Yrs.	Correlation between an exercise score (1-6) and parental occupation (RGSC 1951: I, II, IIINM, IIIM, IV, V) with higher scores for higher occupational classes.	r = -0.020 (ns)		none
- Juneau (2014) -UK; 1970 British Cohort Study -9,624; 34 Yrs.	Correlation between LTPA (0–224 with 23 unique values) and parental occupation (RGSC: I, II, IIIN, IIIM, IV/V) with higher scores for lower occupational classes.	<u>FO at birth:</u> ♂: r = -0.080 (+, p<0.001) ♀: r = -0.053 (+, p<0.001) <u>FO age 5:</u> ♂: r = -0.048 (+, p<0.001) ♀: r = -0.077 (+,		none

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
	Parameter estimates from structural equation model (zero-inflated Poisson models) for LTPA by parental occupation at birth and ages 5 and 10. (Results presented from an accumulation of risk with additive effects model (best fit), for results for ages 5 and 10 see paper.	p<0.001) <u>FO age 10:</u> ♂: r = -0.086 (+, p<0.001) ♀: r = -0.064 (+, p<0.001)	Parental occupation at birth: logistic portion of model: $\bigcirc = 0.054 \text{ (ns)}$ $\bigcirc = 0.88 \text{ (p<0.05,}$ direction unclear) counts portion of model: $\bigcirc = -0.049 \text{ (p<0.05,}$ direction unclear) $\bigcirc = 0.050 \text{ (p<0.05,}$ direction unclear)	occupational physical activity, transport- related physical activity
- Osler (2008) -Denmark; 1953 Metropolit Birth Cohort -6,292 ³ ; 51 Yrs.	Odds of sedentary leisure activity in low compared to high parental occupational class.		OR = 1.10 {0.97; 1.26} OR = 0.90 {0.78; 1.05}	age age, own education, own occupational class, divorce, cognition

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Jørgensen (2013) -Denmark; Danish Health Care Worker Cohort -1,661 [♀] ; 35.4 Yrs. (mean)	Prevalence of low LTPA in five parental occupational groups (1. higher professional 2. lower professional/non-routine M 3. self- employed 4. skilled blue-collar 5. unskilled blue-collar)	<u>1-5:</u> ♀ = -5.7% (+, p=0.011)		none
-Barnekow- Bergkvist (1998) -Sweden -278; 34 Yrs.	Regression coefficients for LTPA MET hours/wk. comparing non- manual to manual parental occupations.		<u>β:</u> ♂ = reported as ns ♀ = 0.18 (+)	own education, sport club member, two-hand lift, attitudes soccer & handball
- Tammelin (2003) -Finland; 1966 North Finland Birth Cohort -7,794; 31 Yrs.	Odds of physical inactivity in parental occupational groups (1. skilled professional 2. skilled worker 3. unskilled worker 4. farmer) with skilled professional used as reference category.		<u>ORs (4 vs. 1):</u> ♂ = 1.18 {0.94; 1.49} (ns) ♀ = 0.80 {0.63; 1.02} (ns)	after-school sports
- Makinen (2009) -Finland; Health 2000	Odds of inactivity and moderate LTPA relative to high LTPA in father's occupational groups (office employee, manual worker, self-		ORs (farmer vs. office <u>employee):</u> Inactivity (\circlearrowleft) = 1.69 (+) Inactivity (\bigcirc) = 0.97	age

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
Survey -6,262; 30+ Yrs.	employed, farmer) with office employee used a reference category.		(ns) Moderate LTPA (♂) = 1.68 (ns) Moderate LTPA (♀) = 1.08 (ns)	
-3,905; 30+ Yrs.	Odds of inactivity and moderate LTPA relative to high LTPA in mother's occupational groups (office employee, manual worker, self- employed, farmer) with office employee used a reference category.		ORs (farmer vs. office <u>employee):</u> Inactivity (\Im) = 1.49 (ns) Inactivity (\Im) = 0.87 (ns) Moderate LTPA (\Im) = 1.99 (ns) Moderate LTPA (\Im) = 1.40 (+)	age
-Wichstrøm (2013) -Norway ->2,800; 25- 32 Yrs	LTPA in five parental occupational groups (leader, high professional, low professional, manual, farmer/fisherman).	Reported as 'unrelated to LTPA at any time point' (ns)		none
- Peck (1994) -Sweden -13,695; 16- 74 Yrs.	Risk of no regular physical activity compared to the sample average in seven parental occupational groups (self-employed with employees, self- employed w/o employees, higher NM, assistant NM, skilled M,		<u>unskilled manual:</u> ♂ = 1.24 (ns) ♀ = 1.24 (ns) <u>higher non-manual:</u> ♂ = 0.73 (ns) ♀ = 0.73 (ns)	none

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
	unskilled M, farmers).			
- Beunen (2004) -Belgium; Leuven Longitudinal Study of Flemish Boys -166 ³ ; 40 Yrs.	Correlation and regression coefficients for sport, leisure-time and counts indices by parental occupation. Only leisure-time presented in paper.	<u>Leisure-time:</u> r = 0.13 (ns)	<u>Leisure-time:</u> β at 16 Yrs. = 0.17 (+) β at 18 Yrs. = 0.16 (+)	skeletal maturity, sum of skinfolds
- Kamphuis (2013) -Netherlands; GLOBE Study -4,894 ; 40- 75 Yrs.	Prevalence of inactive, little and moderately active in three parental occupational groups (1. professional 2. white collar 3. blue collar).	$\frac{1-3:}{\text{Inactive}} = 1.5\% \text{ (ns)}$ Little active = -0.9% (ns) Moderately active = 2% (ns)		none
-van de Mheen (1998) -Netherlands; Longitudinal Study on	Odds of no LTPA and frequent LTPA by parental occupation (1. higher grade professional 2. lower grade professional/routine NM 3. self- employed 4. high/low skilled M 5. unskilled M) with higher grade		<u>ORs (5 vs. 1):</u> No LTPA = 1.82 (+) Frequent LTPA = 0.59 (+) No LTPA = 1.62 (ns) Frequent LTPA = 0.68	age, sex, religion marriage, urbanisation as above plus own occupational class

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
Socio- Economic Health Differences -13,854; 25- 74 Yrs.	professional used a reference category.		(+ in ♀ only)	
- Regidor (2004) -Spain -3,658;60+ Yrs.	Prevalence of physical inactivity in four parental occupational groups (1. professional, manager, proprietor, clerical worker 2. self-employed farmer 3. skilled/unskilled manual worker 4. paid farm worker).	1-4 (♂) = -9.5% (+, p=0.043) 1-4 (♀) = -7.9% (+, p=0.011)	<u>PRs (4 vs. 1):</u> ♂ = 1.29 {1.07; 1.56} (+, ns: 3 vs. 1) ♀ = 1.17 {1.03; 1.32} (+, ns: 2 vs. 1)	none age
	Odds of physical inactivity in manual (M) compared to non-manual (NM) parental occupations.			age, own occupational class
			(+) ♂ = 1.03 {0.90; 1.17} (ns) ♀ = 1.12 {1.03; 1.23} (+)	age, own occupational class

	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
(2010) -US; Health & Retirement Study, Study of Asset & Health Dynamics, & two other cohorts -18,465; 51+ Yrs.	Prevalence of vigorous exercise in manual (M) and non-manual (NM) parental occupations.	NM-M = 6% (+, p<0.001)		none

a. Both men and women included in analysis unless otherwise stated, N°: analytic sample consists of men only, N*: analytic sample consists of men only, N*: analytic sample consists of women only. b. LTPA: leisure-time physical activity; MET: metabolic equivalent; RGSC: Registrar General's Social Classification (I: professional, II: managerial and technical, IIIN: skilled non-manual, IIIM: skilled manual, IV: partly skilled, V: unskilled); M: manual; NM: non-manual. c. For brevity, prevalence of LTPA shown as crude difference between named childhood SEP groups, along with measure of precision (95% confidence intervals where available unless stated otherwise), SE: standard errors, r: correlation coefficient, OR: odds ratio from logistic regression, PR: prevalence ratio, β : regression coefficient; +: Statistically significant (p<0.05) association between less advantaged childhood SEP and less frequent adult LTPA; -: Statistically significant (p<0.05) association between less advantaged childhood SEP and less frequent adult LTPA; non-significant association (p>0.05) between childhood SEP and adult LTPA. d. BMI: body mass index, CVD: cardiovascular disease, CHD: coronary heart disease.

 Table 3.3 Results of studies testing the association between parents' education and leisure-time physical activity (LTPA) in adults:

arranged by region/country and from older to younger age at measurement of physical activity.

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlation coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Johnson (2011) -UK; Lothian Birth Cohort 1936 -1,091; 70+ Yrs.	Correlation and regression coefficients for 6-point LTPA score and years of parental education.	r = 0.08 (+)	β = 0.03 (ns)	none own education, own occupational class & more
- Silverwood (2012) -UK; MRC National Survey of Health & Development -≥3,100; 31-53 Yrs.	Prevalence of LTPA (low/gardening/sport & leisure), walking and cycling during work & for pleasure (high, low) in four groups of paternal education (1. secondary and greater 2. secondary only or primary and further education or greater 3. primary and further education with no qualifications attained 4. Primary only).	$\frac{1-4:}{}$ Sport & leisure (♂) = 14.5% (+, p<0.001) Sport & leisure (♀) = 20.9% (+, p<0.001) Walking (High) (♂) = - 21.6% (-, p<0.001) Walking (High) (♀) = -8.8% (-, p<0.001)		none
-Kuh & Cooper (1992) UK; MRC NSHD ->2,850; 36 Yrs.	Prevalence of most active in sports & recreational activities in 4 groups of parental education (1. secondary & greater 2. secondary only or primary & further education or greater 3. primary & further education with no qualifications attained 4. Primary only).	$\frac{1-4:}{3}$ (father) = 12% (+, p<0.01) ♀ (father) = 21.3% (+, p<0.001) ♂ (mother) = 2% (+, p<0.001) ♀ (mother) = 19% (+, p<0.001)		none

 -1st Author (year) -Country; study name -Sample size^a; age 	How results presented and interpretation ^b	Correlation coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-2,144; 36 Yrs.	Odds of most active in sport & recreational activities comparing three highest groups of maternal education to the lowest group.		<u>ORs:</u> 1 vs. 4 = 1.24 (0.99; 1.55} (ns) 2 vs. 4 = 1.52 (1.22; 1.91} (+) 3 vs. 4 = 1.24 (1.02; 1.50} (+)	own education, sex, childhood health, personality, and ability at games
-Pinto Pereira (2014) -UK; National Child Development Study 1958 (NCDS) -12,776 had ≥ one measure of LTPA; 33, 42, 50 Yrs.	Odds of low LTPA comparing those with two minimally schooled parents to those without.		$\begin{array}{l} \underline{ORs:}\\ age \ 33 = 1.26 \ \{1.15; \ 1.37\} \\ (+)\\ age \ 42 = 1.28 \ \{1.18; \ 1.38\} \\ (+)\\ age \ 50 = 1.42 \ \{1.29; \ 1.57\} \\ (+)\\ age \ 33 = 1.14 \ \{1.04; \ 1.26\} \\ (+)\\ age \ 42 = 1.13 \ \{1.03; \ 1.24\} \\ (+)\\ age \ 50 = 1.22 \ \{1.10; \ 1.35\} \\ (+) \end{array}$	none sex
			age $33 = 1.05 \{0.95; 1.16\}$ (ns) age $42 = 1.03 \{0.94; 1.13\}$ (ns) age $50 = 1.13 \{1.01; 1.25\}$ (+) age $33 = 1.02 \{0.92; 1.13\}$ (ns) age $42 = 1.00 \{0.91; 1.10\}$	sex, parental education, aptitude household amenities, cognition, lifestyle factors age 16, & more as above plus own education, own social class, BMI, mental

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlation coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
			(ns) age 50 = 1.07 {0.96; 1.19} (ns)	health, number of children in the household, limiting illness
- Kvaavik (2011) -Norway; Oslo Youth Study -240-407 [♂] ; 25, 33, 40 Yrs.	Regression coefficients for LTPA per increase in parental education (college/university/>12 Yrs., high/comprehensive school/12 Yrs., high school/10 Yrs., 1 year of technical college/8–9 Yrs., elementary school/7 Yrs.).		$\frac{\beta \text{ (estimated from figures):}}{\text{age 25 (father)} \approx 0.06 (ns)} \\ \text{age 33 (father)} \approx 0.12 (+) \\ \text{age 40 (father)} \approx 0.01 (ns) \\ \text{age 25 (mother)} \approx 0.05 \\ (ns) \\ \text{age 33 (mother)} \approx 0.12 (+) \\ \text{age 40 (mother)} \approx -0.06 \\ (ns) \\ \frac{\beta \text{ (estimated from figures):}}{\text{age 25 (father)} \approx 0.01 (ns)} \\ \text{age 33 (father)} \approx 0.05 (ns) \\ \text{age 40 (father)} \approx 0.01 (ns) \\ \text{age 33 (mother)} \approx -0.01 \\ (ns) \\ \text{age 33 (mother)} \approx -0.01 \\ (ns) \\ \text{age 40 (mother)} \approx -0.01 \\ (ns) \\ (ns) \\ \text{age 40 (mother)} \approx -0.01 \\ (ns) \\ (n$	sex, whether participated in school health education intervention as above plus own education
- Makinen (2009) -Finland; Health 2000 Survey -6,492; 30+ Yrs.	Odds of inactivity and moderate LTPA relative to high LTPA by parental education (secondary, middle, primary) with secondary education used as reference category.		ORs (primary vs. secondary): Inactivity (\mathcal{C}) = 1.10 (ns) Inactivity (\mathcal{P}) = 1.56 (+) Moderate LTPA (\mathcal{C}) = 1.45 (ns) Moderate LTPA (\mathcal{P}) = 1.37	age

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlation coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c (ns)	Adjustments ^d
			(113)	
- Leino (1999) -Finland; Cardiovascular Risk in Young Finns Study -432; 21-30 Yrs.	Prevalence of physical inactivity in three groups of parental education (1. >12 Yrs. 2. 9-12 Yrs. 3. <9 Yrs.).	1-3 (♂) = -14.7% (ns) 1-3 (♀) = -9.2% (ns)		age
- Osler (2001) -Denmark; offspring of Copenhagen City	Odds of low LTPA comparing the two highest groups of parental education to the		<u>ORs (1 vs. 3):</u> ♂= 1.3 {0.6; 3.0} (ns) ♀= 0.5 {0.2; 1.1} (ns)	none
Heart Study (CCHS). -317; 19-31 Yrs.	lowest group (1. ≥ 9 Yrs. 2. 8- 9 Yrs. 3. <7 Yrs.).		ੇ = 0.7 {0.4; 3.2} (ns) ♀= 0.6 {0.2; 2.4} (ns)	age, own education, own occupational class, smoking status
- Beunen (2004) -Belgium; LLSFB -166 [♂] ; 40 Yrs.	Correlation between sports, leisure-time and counts indices of physical activity and parental education.	r (sport, father) = 0.17 (+) r (sport, mother) = 0.14 (ns) r (leisure-time, father) = 0.14 (ns) r (counts, mother) = 0.15		none
	Regression coefficients for sport, leisure-time and counts indices of physical activity per increase in years of parental education	(ns)	β (sport, father) = 0.19 (+) β (leisure-time, father) = 0.14 (+)	stature (sport index) stature, pulse recovery (leisure-time index)

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlation coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Wray (2005) -US; Health & Retirement Study (HRS); Study of Asset & Health Dynamics (AHEAD) -6,106; 51-61 Yrs.(HRS), 3,636; 70+ Yrs.(AHEAD)	Odds of low physical activity per unit increase (0-17) in years of parental education.		<u>ORs:</u> HRS = 0.964 (+, p≤0.001) AHEAD = 0.878 (+, p≤0.001) HRS = 0.976 (+) AHEAD = 0.910 (+)	age, gender, ethnicity, marriage, interactions as above plus own education, economic resources
- Bowen (2010) -US; HRS,AHEAD & more -18,465; 51+ Yrs.	Prevalence of vigorous exercise in two groups of parental education $(1. > 8)$ years 2. ≤ 8 years).	1-2 (father) = 4% (+, p≤0.001) 1-2 (mother) = 4% (+, p≤0.001)		none
- Phillips (2009) -US; Health & Behaviour Project -811; 30-54 Yrs.	Correlation between exercise kilocalories/wk. and years (1-24) of parental education.	r = 0.084 (+)		none
- Frank (2003) -US; Women Physician Health Study -2,884 [♀] ; 30-70 Yrs.	Prevalence of exercise in six groups of parental education (1. medical school 2. graduate school 3. college graduate 4. some college 5. high school 6. < High school) and three groups of both parent's education) (1. both \geq graduate school 2. mix 3. both \leq graduate school).	1-6 (father) = 2% (ns) 1-6 (mother) = -4% (ns) 1-3 (both) = 5% (ns)		none
-Gall (2010)	Prevalence of LTPA by level	<u>1-3:</u>		none

-1 st Author (year)	How results presented and	Correlation coefficient/	Estimates from statistical	Adjustments ^d
-Country; study name	interpretation ^b	difference in prevalence ^c	modelling ^c	
-Sample size ^a ; age			-	
-Australia; Childhood	of parental education (1. high	∂ = 3% (ns)		
Determinants of Adult	2. medium 3. low).	⊊= 1% (ns)		
Health Study				
-1,973; 26-36 Yrs.				
a. Both men and won	nen included in analysis unless c	otherwise stated, Nೆ: analytic :	sample consists of men only,	N ^º : analytic sample
consists of women or	nly. b. LTPA: leisure-time physica	al activity c. For brevity, preva	lence of LTPA shown as crude	e difference between
named childhood SEI	P groups, along with measure of	precision (95% confidence in	tervals where available unless	s stated otherwise), SE:

standard errors, r: correlation coefficient, OR: odds ratio from logistic regression, PR: prevalence ratio, β : regression coefficient; +: Statistically significant (p<0.05) association between less advantaged childhood SEP and less frequent adult LTPA; -: Statistically significant (p<0.05) association between less advantaged childhood SEP and more frequent adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA. d. BMI: body mass index. **Table 3.4** Results of studies testing the association between indices and other measures of childhood socioeconomic position and leisure-time physical activity (LTPA) in adults: arranged by region/country and from older to younger age at measurement of physical activity.

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Johnson (2011) -UK; Lothian Birth Cohort 1936 -1,091; 70 Yrs.	Correlation and regression coefficients for a 6-point LTPA score and an index of childhood household amenities.	r = 0.00 (ns)	β = 0.02 (ns)	none own education, own occupational class & more
- Hilsdon (2008) -UK; British Women's Heart & Health Study (BWHHS) ->4,100 [♀] ; 60-79 Yrs.	Prevalence of four indicators of childhood household amenities and car access in 4 groups of frequency of physical activity hours/wk.	$\frac{\geq 3-0 \text{ (hours/wk.):}}{\text{shared bedroom} = -7.7\% \{-5.9; -8.7\} (+)}$ no indoor toilet = -8.8% {-7.9;- 9.8} (+) no hot water = -9.6% {-8.6; - 10.4} (+) no car access = -7.9% {-6.8; - 9.1} (+)		none
	Odds of more frequent physical activity per unit increase in childhood SEP (parental occupation, household amenities and car access) with higher scores representing more adversity.		OR = 0.85 {0.81; 0.89} (+) OR = 0.93 {0.89; 0.98} (+) OR = 0.94 {0.90; 0.99} (+)	age age, adult SEP, area deprivation. as above plus smoking, BMI, CVD, respiratory disease

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
- Watt (2009) -UK; BWHHS -3,523 [°] ; 60-79 Yrs.	Difference in prevalence of low exercise between those reporting no and those reporting yes to questions on childhood household amenities and car access.	shared bedroom = 5.4% {1.9; 9.0} (+) no hot water = 6.1% {2.4; 9.8} (+) no indoor toilet = 6.8% {3.1; 10.4} (+) no car access = 7.9% {3.3; 12.4} (+)		none
	Odds of low exercise per unit increase in childhood SEP with higher scores representing more adversity.		OR = 1.12 {1.07; 1.17} (+) OR = 1.06 {1.01; 1.12} (+)	none age, own adult SEP
-Pinto Pereira (2014) -UK; National Child Development Study 1958 (NCDS) -12,776 had ≥ one measure of LTPA; 33, 42, 50 Yrs.	Odds of low LTPA per unit increase (0-18) on index of childhood household amenities (access to bathroom, indoor lavatory and hot water, with higher scores indicating more limited access).		$\frac{\text{Odds ratios:}}{\text{age } 33 = 1.03 \{1.01; \\ 1.04\} (+) \\ \text{age } 42 = 1.03 \{1.01; \\ 1.04\} (+) \\ \text{age } 50 = 1.04 \{1.03; \\ 1.05\} (+)$	none
55, 42, 50 TTS.	inniteu accessj.		age 33 = 1.02 {1.001; 1.03} (+) age 42 = 1.01 {0.999; 1.03} (ns) age 50 = 1.02 {1.01; 1.04} (+)	sex
			age 33 = 1.01 {0.995; 1.03} (ns) age 42 = 1.01 {0.99;	sex, parental education, household amenities, cognition, aptitude,

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
			1.02 $\}$ (ns) age 50 = 1.02 {1.002; 1.03 $\}$ (+) age 33 = 1.01 {0.99; 1.02 $\}$ (ns) age 42 = 1.01 {0.99; 1.02 $\}$ (ns) age 50 = 1.01 {0.999; 1.03 $\}$ (ns)	lifestyle factors at age 16, & more as above plus own education, own social class, BMI, mental health, number of children in the household, limiting illness
- Lynch (1997) -Finland; Kuopio Ischaemic Heart Disease Risk Factor Study -2,682 ³ ; 42-60 Yrs.	Prevalence of conditioning inactivity & low quartile of conditioning activity by an index of parental occupation, parental education & more (1. high 2. middle 3. poor).	No conditioning activity: 1-3 = -0.4% (ns) Low quartile: $1-3^+ = -5.7\%$ (+)		age
- Makinen (2009) -Finland; Health 2000 Survey -6,492; 30+ Yrs.	Odds of inactivity and moderate LTPA relative to high LTPA for those reporting yes to long-term financial problems; regular parental unemployment.		<u>ORs (inactivity):</u> ♂= 1.04 (ns); 1.35 (ns) ♀= 1.18 (ns); 1.45 (ns) <u>ORs (moderate LTPA):</u> ♂= 0.95 (ns); 1.31 (ns) ♀= 1.13 (ns); 1.36 (ns)	age
- Beunen (2004) -Belgium; LLSFB -166ೆ; 40 Yrs.	Correlation and regression coefficients for sport, leisure- time and counts indices per increase in urbanisation score of the childhood home. Only counts results	<u>Counts:</u> r = 0.18 (+)	<u>Counts:</u> β at 14 Yrs. = 0.17 (+) β at 16 Yrs. = 0.15 (+) β at 18 Yrs. = 0.15 (+)	none (correlation) sit reach, pulse recovery, sports participation (regression)

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
	presented in paper.			
- Scheerder (2006) -Belgium; Leuven Longitudinal Study of Flemish Girls (LLSFG) -234 [♀] ; 32-41 Yrs.	Path coefficients for level of sports participation based on an index of parental occupation and parental education (lower class, middle class, upper class).		β from path model = - 0.07 {-0.22; 0.08} (ns)	age, own education, own occupational class, BMI, parent's sport, & more
- Pudrovska (2013) -US; 1957 Wisconsin Longitudinal Study	Path coefficients for exercise per change in index of parental occupation, parental education, family income,		<u>'Total effects'</u> β = 1.117 (+, p<0.001)	none
-5,778; 65 Yrs.	father's occupational income and occupational education.		<u>'Direct effects'</u> ♂ = 0.211 (+, p<0.01) ♀ = 0.091 (+)	marriage, children, alcohol use, smoking status, own SES, health, obesity, depression
			♂ = 0.018 (ns) ♀ = 0.039 (ns)	as above plus high school sports
- Carroll (2011) -US; Vaccination Immunity Project -112; 40-60 Yrs.	Correlation between physical activity kilocalories/wk. and a 6-point index of household amenities and car access (for every 2 years, up to age 18).	r (range) = -0.15 to 0.14 (ns)		none

-1 st Author (year) -Country; study name -Sample size ^a ; age	How results presented and interpretation ^b	Correlations coefficient/ difference in prevalence ^c	Estimates from statistical modelling ^c	Adjustments ^d
-Tsenkova (2014) -US; Midlife in the US Study. -895; 25-74	Regression coefficients for more frequent vigorous exercise (0 – 13.5) per unit increase (increasing disadvantage) on a 6-point index of parental education, childhood welfare status and		β = -0.11 {SE=0.03} (+) β = -0.08 {SE=0.03} (+)	age, sex, race, smoking history. as above plus adult SEP
- Kern (2010) -US; Terman Life Cycle Study -1,114;25-61 Yrs.	financial circumstances. Regression coefficients for overall level and linear change in physical activity per unit increase in standardised index of parental occupation and education.		<u>β (Physical activity</u> <u>level):</u>	none
- Schooling (2007) -China; Guangzhou Bio-bank Cohort Study (GBCS) -9,748; 50+ Yrs.	Prevalence of HEPA ^b , minimally active, and inactive in three groups of (3 items, 1 or 2 items, 0 parental possessions in childhood	<u>HEPA-inactive:</u> ♂ (0 items) = 6.1% (-, p<0.01) ♀ (0 items) = -3.2% (p<0.01, direction unclear)		none
-Elwell-Sutton (2011) -China; GBCS -20,086; 50+ Yrs.	Prevalence of HEPA ^b , minimally active, and inactive by 1-3 items or 0 parental possessions in childhood).	<u>HEPA-inactive:</u> 0 Items = -0.17% (ns) 1-3 items = 0.61% (ns)		none

a. Both men and women included in analysis unless otherwise stated, N³: analytic sample consists of men only, N²: analytic sample consists of women only. b. LTPA: leisure-time physical activity; HEPA: Health enhancing physical activity – acronym used in the two

GBCS papers [61-62]. c. For brevity, prevalence of LTPA shown as crude difference between named childhood SEP groups, along with measure of precision (95% confidence intervals where available unless stated otherwise), SE: standard errors, r: correlation coefficient, OR: odds ratio from logistic regression, PR: prevalence ratio, β : regression coefficient; +: Statistically significant (p<0.05) association between less advantaged childhood SEP and less frequent adult LTPA; -: Statistically significant (p<0.05) association between less advantaged childhood SEP and more frequent adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant association (p>0.05) between childhood SEP and adult LTPA; ns: Statistically non-significant (p>0.05) between chil

Chapter 4: Birth weight and leisure-time physical activity across adulthood

Chapter objective: to examine the association between birth weight (as a marker of exposures in utero) and LTPA across adulthood, and to investigate whether this association varies by age at assessment of LTPA.

In chapter 3, published studies were systematically reviewed in order to examine the association between SEP in childhood and LTPA across adulthood, with the findings indicating that adults with less advantaged childhood SEP were less active in LTPA. This chapter is the first of the primary research studies carried out in this thesis to examine how the less studied developmental factors from early life (highlighted in chapter 1) relate to LTPA across adulthood. The chapter begins with a literature review of studies relevant to the associations between birth weight and later LTPA in order to provide background information, summarise the existing evidence and provide justification for the aims and hypothesis of this chapter outlined at the end of the review. The methods used to address this hypothesis and the main findings are described in subsequent sections of this chapter and complements the more general methods described in chapter 2. This chapter concludes with a discussion of the findings in the context of other relevant studies.

4.1 Background

4.1.1 Literature review

The foetal origins of disease hypothesis was briefly introduced in chapter 1.6.3 as an example of a critical period life course model which helps us to understand why interest has been generated in understanding how size at birth relates to subsequent LTPA. The foetal origins of adult disease hypothesis first proposed by David Barker in the 1990s (84) suggests that certain intrauterine exposures can permanently alter development and predisposition to later risk of disease. While this hypothesis later expanded to include infant and childhood exposures, and may be better positioned within a broader life course perspective (212), the foetal origins hypothesis still provides valuable insight into how exposure to specific stimuli during intrauterine life can lead to foetal adaptations that may shape future health.

Certain stimuli during prenatal life such as undernutrition or excess exposure to glucocorticoids may result in long-term changes to the structure and function of

organs which, along with limiting size at birth, may cause altered homeostatic mechanisms (213, 214). Lower size at birth has been shown to be associated with later cardio-metabolic disease risk in both animal studies and across different human populations which are described in this introduction. Size at birth in relation to gestational age (time from the first day of the mother's last menstrual cycle to birth date) is a marker of foetal growth rate (214) and associations between birth size and adult outcomes tend to be more apparent when gestational age is accounted for (84). This highlights the importance of foetal growth rate as a distinct risk factor from preterm birth (84). However, there may be different as well as shared underlying pathways explaining elevated health risks in people born preterm and those born small for gestational age as described below.

Foetal undernutrition has been proposed by some as the key prenatal stimulus that can lead to disease though the underdevelopment of key organs and increased vulnerability to later environmental influences (84). Different types of evidence support this key role for intrauterine undernutrition in the foetal origins of adult disease (214). These include pseudo-experimental studies like those from the Dutch Hunger winter which provide direct evidence in humans that restricted nutrition during different stages of gestation can lead to increased incidence of coronary heart disease, a more atherogenic lipid profile, impaired blood coagulation and increased stress responsiveness (215-218). In addition, intentional manipulation in animal experiments results in similar phenomena to those observed in humans (219).

Other than restricted nutrition, circumstances where the genetic potential for birth weight is not reached can be the result of reduced gestational age, intra-uterine growth restriction or a combination of both. Research has identified several other factors associated with lower birth weight (220, 221), with the most commonly studied listed in table 4.1. A foetus can also reach a given birth weight via a variety of different growth trajectories with differing resultant body compositions and ultimately different disease risks in adulthood (214).

Table 4.1 Factors associated with rates of intrauterine growth

- Infant sex
- Ethnicity
- Maternal height
- Maternal birth weight
- Pre-pregnancy weight of mother
- Maternal diet
- Gestational weight gain
- Parity of mother
- Maternal smoking, alcohol and drug use
- Paternal weight and height
- Air and food pollutants
- Social adversity and deprivation
- Placental epigenetic modification (mechanisms through which
- some maternal exposures can impact on foetal growth)

From Kramer et al. and Cetin et al. (220, 221)

Prior to discussing why intrauterine growth might be related to later PA it is worth summarising the population studies examining associations between birth weight, the most readily available indicator of intrauterine development and growth, and later health outcomes for which PA is a risk factor. Findings from some of these studies, which tend to either examine association per kg increase in birth weight or to focus on those born with low birth weight and compare them to heavier birth weight groups, are summarised in the following paragraphs.

A meta-analysis of 14 studies reported associations between low birth weight (<2.50 kg) and increased risk of type 2 diabetes (OR for low birth weight when compared with heavier birth weight (\geq 2.50 kg) = 1.32, 95CI: 1.06 to 1.64) (222). Likewise, Wang et al. (223) found those weighing <2.50 kg at birth had a higher risk of CVD compared with those with normal birth weight (OR = 1.19; 95 CI: 1.11–1.27). Elsewhere, a heavier birth weight (per kg increase) was related to lower incident CVD including independently of SEP in a meta-analysis of mostly European studies (224) while in another meta-analysis, a kg increase in birth weight was also associated with lower risk of type 2 diabetes (OR = 0.75; 95CI: 0.70-0.81) (225).

A meta-analysis of 22 published studies showed that a heavier birth weight was associated with reduced rates of death from all-causes, and with lower rates of death from cardiovascular disease (CVD) in mostly middle-aged adults which was not confounded by early life SEP (HR = 0.88; 95% CI: 0.85 - 0.91 per kg increase in birth weight) (226). Findings from other meta-analyses suggest that birth weight may

be more closely associated with blood pressure (227) than with cholesterol levels (228).

Findings from a meta-analysis of fourteen studies (229) and the NSHD (230) showed that heavier birth weights were associated with higher bone mineral content of the spine and hips across adulthood. Other findings from NSHD suggest that a heavier birth weight is associated with a more favourable android/gynoid ratio at age 60--64, i.e. with less fat distribution in the abdomen than in the hips, but not with total fat mass (231). Furthermore, consistent evidence was found in a meta-analysis that a heavier birth weight was associated with higher muscle strength in adults (0.86 kg (95% CI 0.58 to 1.15) increase in grip strength per kg increase in birth weight) (232), which may be explained by reduced muscle fibre development (233) and lower muscle mass in those born with low birth weight (231). These findings suggest that in addition to an altered functioning of organs like the kidneys and blood vessels (as implied from findings in relation to diabetes and CVD), intrauterine undernutrition might also influence skeletal and muscular development, and muscular strength.

Cardiorespiratory fitness, a strong predictor of mortality (234) and recognised correlate of LTPA (68), might mediate the effects of foetal undernutrition on later CVD risk. In a study of almost 200 Dutch children aged 8 years, low birth weight (those below the 10th percentile) was associated with lower performance in a 20metre running test (235). In Irish adolescents, a lower birth weight was weakly associated with lower aerobic fitness independently of PA, pubertal stage and other potential confounders (236). Heavier birth weight was associated with better aerobic fitness and better muscular endurance at age 31 in the North Finland Birth Cohort of 1966 adjusted for sex, gestational age, childhood and adult SEP and adult body size (237). In over 200,000 Swedish men born in 1970s-1980s, both preterm and low birth weight for gestational age were associated with lower exercise maximal load capacity (238). There is also evidence that psychological traits such as temperament and personality in adult humans, which are other recognised correlates of PA (68), may be related to intrauterine experiences (239).

Low birth weight has also been associated with poorer motor and cognitive development (240, 241). For example, in a meta-analysis by Maitra et al. (240), children with low birth weight were found to have increased difficulties in mental, neuro-musculoskeletal and movement related tasks compared with those of normal birth weight and similar difficulties were found for children born pre-term when

compared with full-term births. Thus it is plausible that suboptimal prenatal growth and subsequently poorer motor development in those born with low birth weight could have a negative long-term influence on exercise capacity (47) as a result of lower cardiorespiratory fitness, weaker muscle strength and the presence of chronic disease (242). However while plausible, it remains unclear if LTPA is related to birth weight, and thus whether it may explain some of the associations between birth weight and later health outcomes, as very few studies have investigated this association.

Many animal studies e.g. (243-245) show less voluntary PA in offspring born to undernourished mothers which suggests that exercise-related behaviours may also have prenatal origins. Such prenatal influences may be maintained across life as animal studies have shown that rats from undernourished mothers are less active even at an older adult age when compared with normal offspring (244, 245). Over the past decade, epidemiological studies have also been examining how birth weight might relate to PA in human populations with the majority of that research carried out in children and adolescents. The results of these studies are summarised in Table 4.2 and the following paragraphs.

Recent studies of children and young adolescents where PA outcomes are usually derived from accelerometer outputs have tended not to find associations with birth weight (Table 4.2). For example, pooled analysis from three European cohorts and one South American study with participants aged between 9 and 15 years found no difference in accelerometer derived PA per kg increase in birth weight (246). However, in the South American study, a birth cohort from Pelotas, Brazil, a one kg increase in birth weight was associated with less PA counts/minute but this was no longer statistically significant after adjustment for gestational age (246). Null-findings were also reported between birth weight and daily accelerometer counts at age 11-12 years in a birth cohort from South-West England (247). Likewise, a meta-analysis of nine cohorts aged 2 to 14.5 years that also included the studies described above showed no association between birth weight and PA counts assessed by accelerometer (248). Elsewhere, weak unadjusted associations between low birth weight (<2.50 kg) and lower median self-reported PA at age 10-12 years in a birth cohort Brazil have been reported (249).

In follow-up of Australian adolescents, those in heavier birth weight quartiles spent longer time in self-reported outdoor sporting activities at age 12 (250) and associations were maintained at follow-up examination at age 17-18 years (250). Further, the authors report that when compared with those weighing <2 kg at birth, adolescents weighing >4 kg at birth spent an additional hour per week in LTPA (250). These estimates were adjusted for ethnicity, parental education, home ownership, BMI, exposure to passive smoking and gestational age. As noted in chapter 2, this measure of LTPA (self-reported sporting activity) and data from activity monitors in the studies of younger children described in the previous paragraph are not directly comparable measures of PA (51) which could be one explanation for the different findings. Another more likely explanation is that since birth weight has been associated with PA-related health outcomes later in life, associations between birth weight and LTPA become more apparent in young adulthood as health conditions develop. However, few studies have examined this association in adults especially with follow-up into later adulthood (Table 4.2).

A meta-analysis of thirteen Nordic studies which included adolescents and adults (age range = 14 to 69 years though mostly younger ages) found those weighing <2.76 kg and >4.75 kg at birth were both less likely to participate in LTPA when compared with those weighing 3.26-3.3.75 kg (251). Higher levels of leisure-time physical inactivity were also reported by 23-year old Brazilian women but not men born in Pelotas in 1982 with low birth weight (<2.50 kg) (252). Conversely, in a study from the 1958 British birth cohort, the authors reported that there was no difference in LTPA participation across midlife (ages 33-50) when comparing those of low birth weight (<2.5kg) with those of a higher birth weight but did not present their estimate of association (173). A separate study of 57-70 year old participants from the Helsinki Birth Cohort Study found that bigger size at birth (in terms of weight and length) was associated with higher estimated intensity but not energy expenditure of self-reported LTPA (253).

To summarise, of the existing epidemiological studies that have investigated associations between birth weight and PA, most have examined PA in childhood, adolescence or young adulthood using a variety of different instruments and report inconsistent results with a tendency to find null associations or less LTPA in those born with low birth weight. That is, where associations are found, the evidence suggests that, rather than a linear relationship, it is particularly the low birth weight group who are at risk of less LTPA. Moreover, the influence of birth weight on chronic disease risk is more apparent later in life so it could be that associations with LTPA might also be more apparent in adulthood. Most studies have also relied on a single

measure of PA and thus do not account for inter-individual differences in PA over time. There is therefore a need for studies in adulthood which extend into later life. In addition, studies that are able to examine how associations between birth weight and LTPA might change with age would be useful as assessment of whether any associations found change across adult life may help establish underlying mechanisms which could have important implications for future intervention.

4.1.2 Chapter aim and hypothesis

The aim of this chapter was to examine the association between birth weight and LTPA across adulthood in the NSHD and to explore whether the strength of this association changes with age at assessment of LTPA. The specific hypothesis tested is that those with heavier birth weights would be more likely than those with low birth weight (≤ 2.50 kg) to participate in LTPA across adulthood.

Table 4.2 Summary of studies examining the association between birth weight and physical activity: arranged by age at assessment of physical activity

Reference	Description	Physical activity (PA) outcome	Summary of results	Adjustment for confounding
Salonen et al. (2010) (253)	Sub-study of the Helsinki Birth Cohort Study 1934- 44 Age: 57-70 years.	Self-reported intensity and energy expended in LTPA.	Heavier birth size (weight, BMI and length) associated with higher intensity but not with energy expenditure in LTPA.	age, sex, adult social class, adult BMI.
Pinto Pereira et al. (2014) (173)	1958 British birth cohort Age: 33, 40, 50 years	Low LTPA (< once per week).	No difference between heavier and low birth weight groups (<2.50) - results not presented.	None
Andersen (2009) (251)	Meta-analysis of 13 Nordic studies with participants Age: 14 to 69 years.	Various self-reported LTPA.	Lower odds of LTPA for those weighing <2.76 kg and >4.75 kg compared with those weighing 3.26-3.3.75 kg.	None (adjustment for gestational age strengthened associations; adjustment one at a time for education, BMI and smoking did not influence associations).
Kaseva et al. (2015) (254)	Case-control study (n=104) comparing adults born at term and preterm with birth weight <1.50 kg Age: 25 years (mean)	Accelerometer derived mean PA counts/minute.	No association	age, sex, season, BMI, smoking, parental education.
Kaseva et al. (2012) (255)	Case-control study (n=188) comparing adults born at term and preterm with mean birth weight = 1157g. Age: 21-29 years	Self-reported LTPA	Lower frequency, time, volume and energy expenditure of LTPA for low birth weight preterm compared with controls.	age, sex, BMI, smoking, parental education, extraversion, openness to experience, neuroticism, agreeableness, conscientiousness

Reference	Description	Physical activity (PA) outcome	Summary of results	Adjustment for confounding
Gopinath (2013) (250)	Sydney Childhood Eye Study Age: 12 and 17-18 years.	Self-reported time spent in indoor, outdoor and total PA	heavier birth weight groups associated with increasing time in outdoor and total PA at age 12 (p=0.02) and with increasing outdoor PA at age 17-18 (p=0.04).	age, sex, gestational age, ethnicity, parental education & more.
Øglund (2015) (248)	Meta-analysis of 9 studies from UK, Brazil, India, Netherlands and Jamaica. N=10,667 Age = 2 to 14.5 years (means).	Accelerometer derived PA counts per minute (cpm).	No difference in cpm per kg increase in birth weight overall (-3.1; 95%CI: -10.2 to 4.1). Less cpm per kg increase in birth weight in Pelotas cohort (- 33.4; 95%CI: -61.2 to -5.6)	various from age, sex, gestational age, SEP, BMI.
Ridgway (2011) (246)	Pooled analysis of three European cohorts and one South American study with participants aged between 9 and 15 years.	Accelerometer derived PA counts	No association overall. Less cpm per kg increase in birth weight in Pelotas cohort (included in meta-analysis above).	age, sex, SEP, BMI.
Hallal (2012) (256)	Sub-study of the Pelotas Birth Cohort, Brazil of 457 adolescents. Age: 13	3-5 days of accelerometer PA counts	No association – same study above.	sex, gestational age, family income, maternal schooling, maternal BMI, & more
Hallal (2006) (249)	Pelotas Birth Cohort 1993, Brazil, n=5249). Age: 10-12.	Self-reported sedentary lifestyle: (<300 minutes of physical activity/week and median physical activity score (min/week).	Median physical activity score (p non-parametric K sample test on equality of medians =0.05) <2500: 210 min/week. 2500-3499: 235 min/week >3500: 240 min/week No association with sedentary lifestyle (p trend=0.23 unadjusted; 0.81 adjusted).	Unadjusted for Median physical activity score Sex, maternal education, pre-pregnancy BMI, birth order

4.2 Methods

The analysis carried out in this and remaining chapters (i.e. chapters 5 and 6) uses data from the MRC NSHD which is described in detail in chapter 2.1. LTPA outcomes used in this and subsequent chapters (binary outcomes: inactive (no reported participation) or active in LTPA (participated at least once in any sport, exercise or other vigorous leisure activity in previous month) at each age (i.e. at 36, 43, 53, 60-64 and 68 years) and categorical outcomes: inactive (no reported participation), moderately active (participated between 1 and 4 times per month) or regularly active (participated five or more times per month) at each age) are described in detail in chapter 2, section 2.2.

4.2.1 Explanatory variable

Birth weights were extracted from birth records within 6 weeks of delivery where they were recorded to the nearest quarter of a pound and were subsequently converted to kg. Study participants were grouped into five categories of birth weight (≤ 2.50 kg, 2.51-3.00 kg, 3.01-3.50 kg, 3.51-4.00 kg and 4.01-5.00 kg) as in previous analyses examining this association in younger samples and also similar to NSHD analyses examining other outcomes (257, 258). This categorisation was used to compare each heavier birth weight group to the low birth weight group (≤ 2.50 kg). The ≤ 2.50 kg categorisation used to define low birth weight is similar to that used by other published studies examining associations between birth weight and LTPA (173, 250, 251). No babies weighed over 5.0 kg in the NSHD cohort.

4.2.2 Confounding variables

Birth order and childhood SEP were considered confounders of the association between birth weight and LTPA and included as model adjustments (Appendix B). Birth order was considered as a confounding variable because it was hypothesised that a first born would be more likely to have low birth weight (220, 221) and that higher/later birth order would be associated with more LTPA (247, 249). Childhood SEP was considered as a confounder because it was hypothesised, based on systematic review findings reported in chapter 3, that a lower childhood SEP would be associated with lower participation in LTPA across adulthood (151). It was also hypothesised that low birth weight would be more prevalent among those with a lower childhood SEP (220, 221). Based on mother's report of birth order, study participants were classified as first, second, or third or later born. Father's Registrar General's occupational class at age 4 years was used to indicate SEP in childhood and was grouped into four categories (I&II: professional, managerial or technical, IIINM: skilled non-manual, IIIM: skilled manual and IV&V: partly skilled or unskilled).

4.2.3 Examining associations with LTPA across adulthood

Details of the initial exploratory analyses and investigations of sex interactions and deviation from linearity carried out are described in chapter 2.3.1. Descriptive analyses (chi-squared tests) were initially carried out to examine the distribution of birth weight across the selected covariates. Mixed-effects binary and multinomial logistic regression models were used to examine associations between birth weight and LTPA across adulthood (between ages 36-68 years) in study participants with at least one measure of LTPA. Details of these models including rationale for their use are in chapter 2.3.2. Binary mixed-effects models were used to estimate the ORs of participation in LTPA (versus nonparticipation) by birth weight whereas multinomial mixed-effects models were used to estimate the RRRs of moderate and regular participation in LTPA across adulthood (versus nonparticipation) by birth weight. The associations between birth weight and LTPA at each age in adulthood were also examined with separate binary and multinomial logistic regression models in study participants with complete LTPA data at each age (i.e. all run on the same sample size). All models were adjusted in steps for birth order and father's occupational class. In addition, linear regression was used to examine the difference in MVPA time and PAEE assessed by monitors at age 60-64 by birth weight in the subsample of study participants with these data.

4.3 Results

4.3.1 Relation of birth weight to covariates

The distribution of birth weight overall and by sex in the sample with at least one measure of LTPA and data on the selected covariates is shown in Table 4.3. A total of 163 participants (4.6%) had low birth weight and higher proportions of those with low birth weight were females. Regarding the covariates included in analyses in this chapter, low birth weight was more prevalent among first born participants and those with fathers in partly skilled or unskilled occupational classes (occupational classes IV&V).

	Overall	Men	Women	test of sex-
	N (%)	N (%)	N (%)	difference
<u>Birth weight (kg)</u> ≤ 2.50 2.51-3.00 3.01-3.50 3.51-4.00 > 4.00	163 (4.6) 587 (16.6) 1263 (35.6) 1169 (33.0) 363 (10.2)	66 (3.7) 242 (13.5) 612 (34.2) 634 (35.4) 238 (13.3)	97 (5.5) 345 (19.7) 651 (37.1) 535 (30.5) 125 (7.1)	p<0.001

Table 4.3 Distribution of birth weight overall and by sex in those with data on covariates and at least one measure of LTPA (n=3545)

Sex difference tested by chi-squared test

4.3.2 Associations between birth weight and LTPA across adulthood

Formal tests of deviation from the linear trend were undertaken and showed evidence of non-linear associations between birth weight and LTPA (in all models p<0.04 for polynomial birth weight terms (i.e. birth weight²), with the exception of LTPA at age 68 where both continuous and polynomial terms were not statistically significant). Examining the estimates across groups also suggests this non-linearity. As a result of this, two categorical birth weight variables were used as explanatory variables (5 category ordinal variable and a dichotomous variable). There was no evidence of effect modification by sex in any of the models (in all models p>0.2 using both continuous and categorical birth weight variables by sex interaction terms) therefore, men and women were combined and results adjusted for sex.

There was no evidence of an interaction between birth weight and age when examined in the binary mixed-effect models (p-value for continuous birth weight (kg) by age interaction = 0.5; p-value for categorical (5 groups) birth weight by age=0.2). This suggests that associations did not differ by the age at assessment of LTPA and is consistent with the similar ORs of LTPA at each age in adulthood in study participants with non-missing LTPA data (Appendix 2A). As described in chapter 2, there was a sex by age interaction (the decline in LTPA was greater in men than women) which was added to all mixed-effects models.

Table 4.4 presents the ORs of LTPA across adulthood (between 36-68 years) estimated from mixed-effects binary logistic regression analysis of 3545 men and women (49.4% female) with at least one measure of LTPA and complete data on

birth weight, birth order and father's occupational class. When compared with the low birth weight group, those in all other heavier birth weight groups were more likely to participate in LTPA across adulthood (Table 4.4). Findings were similar when all heavier birth weight groups were combined and compared with the lowest birth weight group adulthood (Table 4.4). There was slight strengthening of this association following adjustment for birth order and slight attenuation after further adjustment for father's occupational class (Table 4.4).

Figure 4.1 presents the linear model predictions (predicted log-odds) of LTPA at each age across adulthood stratified by the mean log-odds of each birth weight group. The plots are further stratified by sex due to the interaction between sex and age which is evident from the higher intercept and steeper decline in the log-odds of LTPA for men compared with women (Figure 4.1). Both these plots show the decline in LTPA for all birth weight groups within each sex and they show that the lowest birth weight group have lower predicted log-odds of LTPA than all other heavier birth weight groups.

Table 4.5 presents the RRRs of moderate and regular participation in LTPA across adulthood (versus no participation) by birth weight estimated using mixed-effects multinomial logistic regression in the same sample (n=3545). When compared with the low birth weight group, those in all other heavier birth weight groups were more likely to be both moderately (1-4 times per month) and regularly (5 or more time per month) active in LTPA across adulthood between 36-68 years (Table 4.5). This association appeared stronger when comparing regular participation with nonparticipation. Adjustment for birth order and childhood SEP led to slight improvements in model fit (Table 4.5).

Results from standard binary and multinomial logistic regression models fit separately to LTPA at each age in those with data on the above covariates in addition to complete data on LTPA (n=1581, 53.6% female) are presented in Appendices 2A and 2B respectively. The findings from these models were consistent with the mixed-effects model estimates described above and suggest that, when compared with the low birth weight group, other study participants in all heavier birth weight groups were more likely to participate in LTPA at each age (Appendix 2A and 2B). These associations were generally slightly strengthened by adjustment for birth order and slightly weakened by further adjustment for father's occupational class.

Examining the ORs of team sports (n=3138), non-team sports LTPA (n=3108) and leisure-time walking (n=3129) at age 36 showed that heavier birth weight groups were more likely than those with low birth weight to participate in all three types of LTPA at that age (Appendix 2C). Lastly, linear regression models examining associations between birth weight and MVPA and PAEE assessed by monitors at age 60-64 in the sample with data on LTPA at that age (n=1583) suggest that heavier birth weight groups, in particular those weighing 2.51 - 3.00 kg and 3.01 to 3.50 kg at birth, tended to spend greater time in MVPA than the low birth weight group however, there was little evidence in support of this association (for all models p>0.3) (Appendix 2C). Birth weight did not appear to be associated with PAEE (for all models p>0.7) (Appendix 2C) whereas birth weight was associated with LTPA at age 60-64 (Appendix 2D).

Table 4.4 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-time physical activity (LTPA) between ages 36 and 68 years by birth weight group: mixed-effects binary logistic regression.

	LTPA at least once per month across adulthood versus no LTPA				
	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)		
<u>Birth weight (kg)</u> ≤ 2.50 2.51-3.00 3.01-3.50 3.51-4.00 > 4.00 test of association	1.00 (reference) 1.76 (1.24 – 2.49) 2.02 (1.45 – 2.79) 1.78 (1.28 – 2.47) 1.67 (1.15 – 2.52) p<0.001	1.00 (reference) 1.77 (1.25 – 2.50) 2.07 (1.50 – 2.87) 1.92 (1.38 – 2.66) 1.93 (1.33 – 2.80) p<0.001	1.00 (reference) 1.71 (1.20 – 2.43) 1.89 (1.35 – 2.63) 1.71 (1.22 – 2.39) 1.74 (1.19 – 2.54) p=0.006		
<u>Birth weight (kg)</u> ≤ 2.50 > 2.50 test of association	1.00 (reference) 1.85 (1.35 to 2.54) p<0.001	1.00 (reference) 1.95 (1.42 to 2.67) p<0.001	1.00 (reference) 1.75 (1.28 to 2.38) p<0.001		

Analytic sample consists of those with at least one measure of LTPA (n=3545). Model 1: adjusted for sex. Model 2: adjusted for sex and birth order. Model 3: as for model 2 plus father's occupational class. Includes sex by age interaction. Tests of association based on likelihood ratio tests comparing models with and without birth weight terms. Figure 4.1 Log-odds of leisure-time physical activity for each birth weight group by age in men and women.

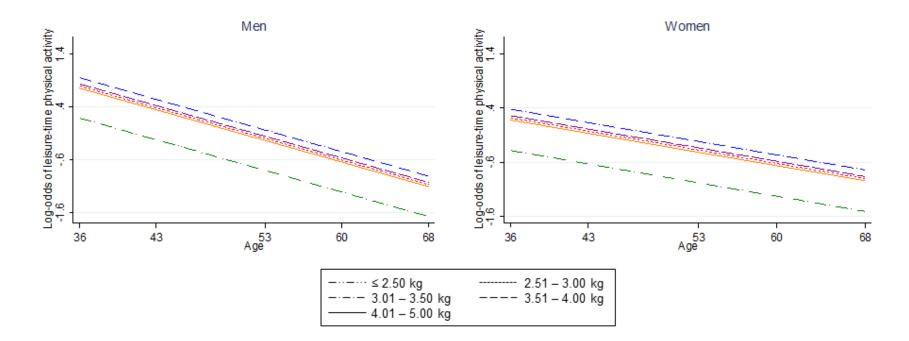


Figure 4.1 legend: Based on the estimated fixed effects (coefficients) $x\beta$ in the model (mean log-odds of leisure-time physical activity at each age for each birth weight group). Log-odds are stratified by sex due to an interaction of sex by age which means that the decline in LTPA is greater in men than women.

LTPA between ages 36-68) of moderate Ll onth) versus no	•	RRR (95% CrI) of regular LTPA (5 or more times per month) versus no LTPA
	Model 1	Model 2	Model 3	Model 1 Model 2 Model 3
Birth weight				
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 1.00 1.00 (reference) (reference)
2.51-3.00	1.59	<u></u> 1.65	<u></u> 1.53	2.18 2.58 2.26
3.01-3.50	(1.17 to 2.08) 1.71	(1.23 to 2.28) 1.84	(1.06 to 1.98) 1.64	(1.50 to 2.97) (1.60 to 3.62) (1.55 to 3.17) 2.36 2.94 2.45
3.51-4.00	(1.32 to 2.19) 1.46	(1.37 to 2.57) 1.65	(1.19 to 2.07) 1.44	(1.77 to 3.06) (1.82 to 4.56) (1.75 to 3.48) 2.21 2.93 2.36
> 4.00	(1.12 to 1.91) 1.43	(1.24 to 2.31) 1.68	(1.01 to 1.82) 1.51	(1.66 to 2.86) (1.90 to 4.08) (1.67 to 3.30) 1.95 2.68 2.26
	(1.05 to 1.94)	(1.22 to 2.44)	(1.06 to 2.00)	(1.37 to 2.66) (1.64 to 4.27) (1.49 to 3.24)
Bayesian DIC	24207.44	24183.82	24126.61	
Birth weight				
≤ 2.50	1.00	1.00	1.00	1.00 1.00 1.00
> 2.50	(reference) 1.52	(reference) 1.71	(reference) 1.45	(reference) (reference) (reference) 2.36 2.65 2.14
	(1.18 to 1.91)	(1.31 to 2.26)	(1.13 to 1.95)	(1.78 to 3.03) (1.86 to 3.74) (1.51 to 3.04)
Bayesian DIC	24206.91	24177.54	24127.55	

Table 4.5 Relative risk ratios (RRR) and 95% credible intervals (95% CrI) of moderate and regular leisure-time physical activity (LTPA) between ages 36 and 68 years by birth weight group: mixed-effects multinomial logistic regression.

Analytic sample consists of those with at least one measure of LTPA (n=3545). Model 1: adjusted for sex. Model 2: adjusted for sex and birth order. Model 3: as for model 2 plus father's occupational class. Includes sex by age interaction. Bayesian DIC statistics indicate fit for whole models.

4.4 Discussion

4.4.1 Summary of results

The main findings of this chapter were that when compared with the low birth weight group, study participants in all other heavier birth weight groups were more likely to participate in LTPA between ages 36 and 68 years. This association between low birth weight and lower likelihood of participation in LTPA was stronger with more frequent participation; heavier birth weight groups were more likely to be moderately active and even more likely to be regularly active than inactive in LTPA across adulthood. Associations were similar when initial sex-adjusted estimates were adjusted for birth order and the strength of this association was only slightly weakened by additional adjustment for childhood SEP. There was no clear evidence that associations between birth weight and LTPA differed by the age at assessment of LTPA indicating that associations with LTPA at earlier adult ages persisted into older adult ages, with all birth weight groups showing similar rates of decline in participation. There was also a weak suggestion that heaver birth weight groups spent greater time in MVPA assessed by monitors than the low birth weight group but no evidence was found that birth weight was related to monitor-based PAEE, both assessed at age 60-64.

4.4.2 Comparison with other studies

Some of the other studies which have previously investigated this association in younger more recently born cohorts support these findings (250-253). These include the meta-analysis described earlier in section 4.1.1 which showed Scandinavian adolescents and adults in lower birth weight groups (range=1.26 to 2.75 kg) were less likely to participate in LTPA than the reference birth weight group (3.26–3.75 kg) (251), and higher levels of leisure-time physical inactivity reported by 23-year old Brazilian women born in 1982 with low birth weight (<2.50 kg) (252). Also supporting these results are findings of less participation in outdoor sporting activity by 12-year old Australian adolescents with low birth weight (<2.00 kg) that persisted over 5-years follow-up (250).

The findings of this chapter showed that associations between low birth weight and lower likelihood of LTPA were consistent across adult life which is similar to animal studies showing offspring from undernourished mothers to be less physically active across life, including at older adult ages, when compared with normal offspring (244, 245). That associations between birth weight and LTPA were apparent at older ages is similar to findings from a Finnish study showing older adults with an average age of 62 years reported higher intensity LTPA if they were bigger at birth in terms of weight and length (253). In additional analyses undertaken in this chapter, heavier birth weight groups tended to also spend more time in monitored MVPA at age 60-64 years when compared with the low birth weight group. This somewhat validates the main findings of an association with LTPA as a majority of LTPA tends to be spent in MVPA (40).

The results presented in this chapter are not consistent with null-associations reported between birth weight and PA levels in children (246, 248). However, as associations between birth weight and chronic disease tend to be more apparent later in life (223, 225) this may also be the case for LTPA. Consistent with this, studies which have examined associations in older adolescents and adults have reported expected associations (250-253). Likewise, it is also thought that associations between preterm birth and LTPA tend to be more apparent in adulthood and adolescence than in childhood (259).

The findings reported here are also in contrast to a study from the next oldest British birth cohort born in 1958 where the authors reported that no differences were found between low (<2.50 kg) and heavier birth weights in levels of LTPA assessed in mid-adulthood (ages 33-50) but no estimate was provided (173). Analyses of 5058 births in the Avon Longitudinal Study of Parents and Children (ALSPAC) is the only other study to examine this association in a British cohort (247). This study found no associations between various birth outcomes (birth weight: per 100g increase, ponderal index: per kg/m³, head circumference and crown-heel length: both per cm increase) and PA counts per minute measured by accelerometers at age 11-12 years (247) but as mentioned above, it may be that associations will become apparent at older ages. Further, it may be that associations are specific to certain domains of PA which are currently more reliably captured by self-report (see section 1.3).

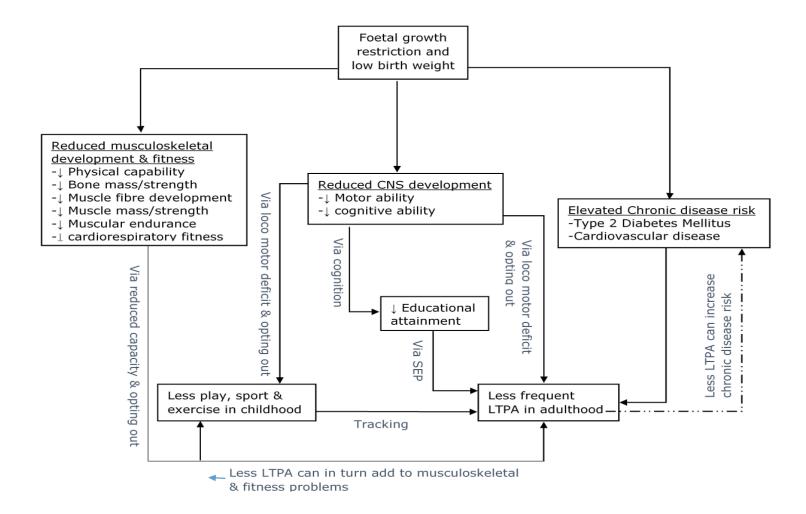
4.4.3 Explanation of findings

The findings of this chapter suggest that prenatal growth may influence participation in LTPA across life (47, 260). The conceptual diagram in Figure 4.2 illustrates how the underlying mechanisms may operate through delayed motor development, an impact on motor skills and coordination required to develop competence at sports, a reduced exercise capacity and subsequent self-selection out of sports and exercise in those born with low birth weight. Motor deficits including difficulties in movement-related tasks and other neurocognitive impairments have been reported in children with low birth weight (240, 241), and may therefore explain the associations found here with adulthood LTPA. In addition, positive attitudes towards LTPA and other health behaviours tend to develop during adolescence (92) and may influence participation in adulthood through the tracking of sports participation (91, 94, 185, 261) (Figure 4.2). Less favourable body composition (229-231, 262) including weaker muscle strength (232) and more prevalent chronic disease, particularly type 2 diabetes and CVD, (84, 214, 222, 223, 225, 257) in those with low birth weight may also contribute to their higher probability of nonparticipation in LTPA across adulthood.

However, the associations found in this chapter between birth weight and LTPA were consistent across adulthood which suggests that health conditions related to birth weight may not play a major role in explaining these findings. This is supported by recently published analyses building on this chapter's findings showing that the association between birth weight and LTPA was only slightly weakened by further adjustment for physical health at age 36 (263); an index of measured weight, blood pressure, lung function (which was associated with birth weight in NSHD (264)) and self-reported health problems, disability, and hospital admissions (265). Furthermore, adult BMI was recently found to explain an association between birth weight and left ventricular structure at age 60-64 in NSHD (266) and body size may also mediate the association between birth weight and LTPA (Figure 4.2). Additional adjustment for BMI at age 36 had little influence on the association reported in this chapter between birth weight and adult LTPA in NSHD (263). This suggests that BMI may only partly mediate the association between birth weight and LTPA. However, as this additional analysis only adjusted for BMI at a single age examining time varying effects of BMI may have explained a greater proportion of this association. Likewise childhood cognitive ability may also partly mediate findings through its relationship with SEP (Figure 4.2) but adjustment for cognitive ability at age 11 only slightly attenuated associations (263).

Furthermore, better ability at games in adolescence has been shown to be associated with higher LTPA at age 36 in NSHD (93, 173, 267) (examined in relation to LTPA across adulthood in the next chapter) and might be considered a crude indicator of LTPA in youth that potentially mediate the association between birth weight and adulthood LTPA. However, further adjustment for ability in school games only marginally reduced this association (263) thus several pathways in addition to the tracking of PA into adulthood are likely involved in explaining the association between birth weight and adult LTPA (Figure 4.2).

Both physiological as well as social pathways might explain the association between birth weight and adult LTPA. For example, a low birth weight might be associated with less LTPA as a consequence of reduced physical capacity due to low bone and muscle strength, and a reduced aerobic capacity making exercise and other LTPA more challenging and less enjoyable or appealing to those with low birth weights, who may as a result choose to opt out of LTPA (Figure 4.2). Moreover, a reduced exercise capacity is also a reported consequence of reduced gestational age (i.e. preterm birth) (268) and thus associations between low birth weight and less LTPA could be driven by intrauterine growth restriction, a reduced gestational age, or a combination of both. This is discussed in the next section. Figure 4.2 Hypothesised pathways underlying associations found between birth weight and adult leisure-time physical activity.



4.4.4 Methodological considerations

The methodological considerations relevant to all analyses carried out in this thesis are discussed in chapter 7 and only those which are specific to this chapter are discussed here.

One limitation of this study is that, while results were adjusted for hypothesised confounding by birth order and SEP, information on gestational age was not available and therefore it was not possible to distinguish between those born small-for-gestational age and those with low birth weight due to preterm birth. However, there would have been less variation in gestational age in this study population than in more recent born cohorts as preterm births were less likely to survive in the 1940s (269). As a result of reduced survival, there were a limited number of participants classified as having low birth weight in this cohort but despite reduced statistical power, associations were observed with both outcomes. The small number of participants with low birth weight is reflected in the wide confidence intervals around the statistically significant estimates.

In addition, birth weight is the only measure of birth size available in NSHD and is only a proxy marker of the adaptations that a foetus may make to its body's structure and function in response to stress which is experienced in utero. In addition, it was not possible to identify the extent of growth restriction represented by low birth weight as the genetic birth weight potential of each participant was unknown. Moreover, it is unclear if low birth weight participants were more likely to be inactive in LTPA as a result of e.g. chronic disease or if they develop risks associated with intrauterine growth restriction as a result of inactivity. However, by examining how associations change with age at assessment of LTPA, this chapter shows that associations were observed at age 36 and thus likely before disease onset. Other strengths include the collection of measured birth weights from birth records within weeks of delivery and is thus not subject to recall bias and adjustment for important and prospectively ascertained covariates of birth order and childhood SEP.

4.4.5 Implications of findings

Due to both the increasing prevalence of those born small for gestational age (270) and the increased survival rates among those born small for gestational age (271), the findings of this chapter could have important health-related implications for

current as well as future generations of adults of all ages. Moreover, the associations observed are likely to be generalisable to more recently born cohorts since associations have been seen in the same direction in younger cohorts including from Australia (250), Scandinavia (251) and Brazil (252) though the NSHD analyses benefit from follow-up into old age which these other studies do not have. However, in order to allow more meaningful comparison of findings between different UK cohorts, it would be useful to harmonise methods of analyses.

This increased long-term survival of babies born with low birth weight means that there are increasing numbers of adults who were born with low birth weight and thus there may be a growing proportion of the population who are unlikely to be participating in LTPA. Therefore, it is important to recognise that those born with low birth weight may require more support than others if they are to achieve sufficient PA across life to realise its health benefits. Exercise is recognised as important for reducing the adverse cardio metabolic consequences of in utero growth restriction (272) and is also considered safe for the majority of those born preterm (268). Designing appropriate interventions to support LTPA across life may require a better understanding of how other related processes like postnatal growth, motor capability and body composition influence PA in those with low birth weight (Figure 9). Such interventions should encourage a variety of PA types including aerobic exercises such as swimming to improve cardiorespiratory fitness (37) and weight bearing activities such as high impact aerobics classes to promote osteogenesis (38).

Furthermore, there are many other factors which may influence LTPA (Table 1.1) (68) and some of them are potentially more important influences on participation from a population health perspective. Therefore, a discussion of how meaningful these effects are is provided in chapter 7. However, even if it is a relatively small effect, since the prevalence of the exposure (i.e. low birth weight) is increasing its impact will increase.

4.4.6 Conclusions

To summarise, the aim of this chapter was to examine how birth weight as a marker of in utero exposures and intrauterine growth relates to participation in LTPA across adulthood. The findings showed that when compared with low birth weight, all other heavier birth weight groups were more likely to participate in LTPA between 36 and 68 years, including at both moderate and regular levels, and this association did not differ by the age at assessment of LTPA, suggesting that health conditions related to low birth weight may not play a major role in explaining this finding. While taking methodological aspects of this study into consideration, the findings could have important implications for health in current and future generations due to the increasing prevalence and long-term survival of preterm and small-for-gestational age births. Understanding the explanatory roles of different processes like motor coordination, postnatal growth and body composition might help identify appropriate characteristics of interventions for those who are unlikely to be participating in sports and exercise across life. These findings are taken into consideration in the next chapter of this thesis where the associations between motor development and motor coordination in early life and LTPA across adulthood are examined.

Chapter 5: Motor performance in early life and leisure-time physical activity across adulthood

Chapter objective: to examine associations between attainment of motor milestones, ability at games and motor coordination in adolescence and LTPA across adulthood, and to investigate whether associations of earlier developmental factors with adulthood LTPA vary by age.

This chapter examines how indicators of motor development, ability and coordination in early life, which were hypothesised to be on the pathways between birth weight and LTPA, might relate to participation in LTPA across adult life. The chapter begins with a review of the relevant literature on the development and importance of movement skills and provides a rationale for the study. The aims and objectives of the chapter are then presented and followed by a detailed description of the methods used, results and discussion of the findings in the context of the relevant studies.

5.1 Background

5.1.1 Literature review

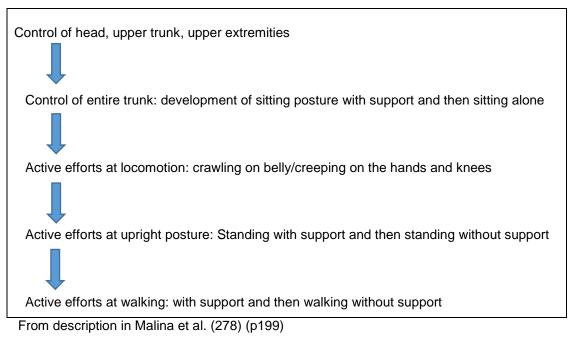
Motor development is the process through which the child acquires movement patterns and skills as a consequence of the growth and development of the brain and nervous system (271). These movements start during the foetal and new-born periods as spontaneous rhythmical arm and leg movements that help to build and strengthen muscles necessary for acquiring later more complex movements (273). The acquisition or achievement of key motor skills such as learning to sit, stand and walk independently occur rapidly during the first two years of life and are described as infant milestones (274).

The rate at which developmental motor milestones are achieved varies between children both within and between different countries but not between different sexes (275, 276). However despite this variation, not learning to sit unsupported by 12 months or walk independently before 24 months in all countries are considered clear indicators that the infant should be referred to developmental specialists (274, 277). Infant motor development is considered closely related to the growth of the cerebellum (which controls the development and maintenance of neuromuscular

coordination, balance and cognition) since its cell contents tend to reach their lifetime maximum levels by 18 months of age (274, 278).

Early fundamental motor milestones are generally acquired sequentially such that later milestones will often depend on the achievement of earlier ones (278) (Figure 5.1). However, environmental circumstances like child rearing practices, maternal mental health and maltreatment can have crucial influences on the expression and order of these developmental stages through their interaction with the underlying biological processes (274, 278). In addition, body composition and muscle strength of the infant as well as their temperament and motivation to move can influence the order and appearance of motor milestones (273). Low birth weight (reflecting either preterm births or being small for gestational age) which was shown in chapter 4 to be associated with nonparticipation in LTPA across adulthood in NSHD is also known to be associated with delayed infant motor development (278).

Figure 5.1 Development of Independent walking



Children begin to actively explore their environment once independent mobility is acquired. Other motor and coordination skills such as running, jumping and turning are then acquired in the 2nd year of life (278). As their physical environment expands, children gain the ability to move down hills, over and under barriers, and over varied terrain (i.e. more complex motor skills). As they become more socially active children use their learned movement skills to dance, play and learn athletic skills (273). Motor

performance (e.g. manual dexterity, hand-eye coordination and balance) tend to decrease from young adulthood to old age (279, 280) however, the acquisition of motor skills is a lifelong process (273).

The fundamental movement skills described above are precursor patterns to more specialised and complex skills in games, sports and recreational activities. It is thought that PA drives motor skill development in early childhood and that higher levels of motor skill competence offer a greater motor repertoire for the older child to engage in sports and games (281). It is also hypothesised that from middle and later childhood motor competence drives PA levels and that this relationship continues to increase in strength over the lifespan (281). Thus, there are likely to be bidirectional associations between motor ability and LTPA such that each asset benefits the other and both may contribute to influencing later LTPA (281-284). Therefore, studies with cross-sectional design are not able to clearly establish whether study participants were more active because of a better motor ability or whether motor ability is improved by greater PA.

Cross-sectional associations between children and adolescents' proficiency in motor skills (e.g. running, jumping, hopping, skipping, sliding, striking, dribbling, balancing) and greater cardiorespiratory fitness are well documented (285). Longitudinal associations have also been reported between better movement and object control skills (e.g. running, jumping, hopping, catching, kicking, throwing) assessed in pre-schoolers and greater fitness, muscular endurance and flexibility in adolescence (286). Systematic reviews have also reported associations, mainly from cross-sectional studies, between better motor skills (e.g. kicking, catching, throwing) and more frequent sports and organised PA in children and adolescents (285, 287).

One indicator of motor integrity often used in clinical settings to detect motor and cognitive impairments is finger and foot-tapping speed (288). Tapping speed is considered a neuropsychological test of motor speed and lateralised coordination that requires rapid information processing and is thought to reflect the maximum frequency of impulses that a motor centre can receive or emit (289). Finger-tapping speed provides a marker of the motor function of upper limbs including sensorimotor brain function, specifically the primary motor cortex, cerebellum, pre-supplementary motor area and premotor cortex (290). Foot-tapping speed provides a measure of lower limb motor function and is performed mostly by the distal muscles related to voluntary, discrete, skilled movements and controlled by the lateral descending

system; the corticospinal and rubrospinal tracts (291, 292). Males tend to tap faster than females (293). Tapping speed was previously examined as part of an NSHD study into the developmental origins of midlife physical function with the findings showed that higher finger and foot-tapping speeds in adolescence were associated with better performance in chair-rising tests and standing balance at age 53 (294).

Related findings from NSHD showed, contrary to the authors' hypothesis, evidence of a nonlinear association between attainment of independent standing and walking and midlife physical performance (standing balance and chair rising) which were not explained by adjustment for current LTPA and other covariates (294). These findings suggested that those with early or late attainment of milestones had poorer physical function than those reaching milestones around the modal age (294). Related findings also from NSHD showed that earlier ages at standing and walking unsupported (reported by the mother at 24 months) were associated with higher grip strength at age 53 but that this association was no longer statistically significant after adjustment for birth weight and other covariates (295). Elsewhere, in the 1966 Finnish birth cohort, earlier ages at standing and walking unsupported (reported by the mother at 12 months with no data collected after this age) were associated with higher muscle strength, endurance and aerobic fitness in 31 year old adults (237).

As muscle strength, fitness and balance are correlates of LTPA (68) a compelling case could be made for long-term associations between ages at motor development and motor competence in early life and LTPA and some longitudinal studies of mostly short-term follow-up duration have been carried out (Table 5.1). Better motor coordination reported by parents at age 6 months and assessed at 7 years was associated with higher accelerometer counts at age 11-13 years in ALSPAC (247, 296). Portuguese children with high levels of motor skills and coordination (e.g. balance, jumping, hopping) at age 6 had higher fitness (297) and self-reported PA (297, 298) at ages 6 and 10 years and showed less decline in PA when compared with those ranked as having average or low motor coordination (298). Better motor skills of Finnish adolescents were associated with more time in self-reported PA six years later (299). Australian adolescents who were more competent at catching, kicking and throwing seven years earlier also spent more time in organised PA and at higher self-reported intensities of PA (300).

The earliest evidence of more long-term associations between childhood motor ability and LTPA comes from NSHD where Kuh and Cooper (93) found study participants with a higher teacher-rated ability at games at age 13 were more likely to participate in LTPA at age 36. Similar associations have recently been reported in the two British birth cohorts initiated after NSHD. In the 1958 British birth cohort, those with hand control/coordination problems at ages 7, 11 and 16 years as rated by teacher were more likely to be inactive in LTPA in mid-adulthood (173) and when compared with those always active in LTPA between 33-50 years, they were more likely to be always inactive and to be decreasingly active between 33-50 years (267). They were also less likely to be increasingly active when compared with those always inactive (267). Likewise, when compared to participants from the 1970 British birth cohort with low motor coordination at age 10 (throwing, balance and walking backwards), those ranked high were more likely to participate at least once/week in LTPA at age 42, but motor coordination was not associated with LTPA at age 16 (Table 5.1) (301).

Other than the above studies which examined motor skills in children and adolescents, very few have examined how attainment of motor milestones in infants relates to later LTPA, which may represent a reflection of genetic factors, or neurological competence and capabilities (248). A study of over 200 Australian toddlers (302) showed later age at walking was associated with lower PA measured by accelerometer at 19 months (Table 5.1) although this is likely a function of how long infants have been walking. Models from a Finnish birth cohort adjusted for several covariates showed that later ages at standing and walking unsupported (reported by mother when aged 12 months) were weakly associated with a lower school physical education grade, less frequent participation in sports and a lower participation in different types of sports at age 14 (303). More recent pooled analysis from longitudinal studies of Finnish twins showed those who learned to stand and walk earlier, as recalled by parents when twins were 12 years old and twins with better motor proficiency in childhood had higher estimated energy expenditure from self-reported LTPA at ages 25 and 34 years (304).

However, almost all studies which examined associations between measures of childhood motor function including attainment of motor milestones and subsequent PA have examined LTPA at only one point and usually in adolescents. It is therefore unclear if these associations extend across adult life to older ages and to other indicators of motor coordination such as tapping speed.

5.1.2 Chapter aim and hypotheses

The aim of this chapter is to examine the associations of age at attainment of infant motor milestones, ability at school games and motor speed and coordination in adolescence with LTPA across adulthood, and to investigate whether associations with adulthood LTPA change with age. The specific hypotheses tested are that later age at reaching infant motor milestones would be associated with lower likelihood of LTPA. That lower ability at games would be associated with lower likelihood while higher ability at games would be associated with higher likelihood of LTPA across adulthood and that better motor speed and coordination in adolescence as indicated by faster finger and foot-tapping speed would be associated with higher likelihood of LTPA across adulthood.

Table 5.1 Summary of studies examining associations between motor performance in early life and physical activity: arranged by age at assessment of physical activity.

Reference	Description	Indicator of motor development/coordination	Physical activity (PA) outcome	Summary of results	Adjustment for confounding
Pinto Pereira et al. 2014; 2015 (173, 267)	1958 British birth cohort (n=12271) Age: 33, 40 and 50 years	number of ages at which hand control or coordination problems were present (rated by teacher at 7, 11 and 16Y)	low LTPA (less than once per week)	hand control or coordination problems associated with low LTPA at each age and with LTPA patterns between 33– 50 years.	various early life and adulthood covariates
Smith et al. 2015 (301)	1970 British birth cohort (n=4879) Age: 16 and 42 years	throwing, balancing and walking backwards assessed at age 10 used to classify children to low, medium, high motor coordination.	participation in LTPA (>1/week) at ages 16 and 42 years. TV viewing age 16 and 42 years.	better motor coordination associated with more LTPA at age 42 and less TV viewing age 16 but not with LTPA at age 16 years. E.g. fully adjusted OR of LTPA at 42 years for high versus low=1.18 (95%CI: 1.02 – 1.36).	sex, BMI, TV viewing & sports age 10,childhood SEP, parents BMI & smoking.
Aaltonen et al. 2015 (304)	longitudinal studies of Finnish twins (n=3300 twin pairs) Age: 24-34 years	Parental report of perceived difference in motor development between twins: turning over from back to stomach, standing, walking, climbing stairs unaided, motor proficiency in childhood and adolescence	METs of LTPA	Earlier age at walking unsupported and better motor proficiency were associated with higher LTPA.	birth weight, birth order.
Jaakkola et al. 2015 (299)	224 Finnish adolescents	composite of flamingo standing (balance), leaping	METs, LPA, MPA and VPA	better score on movement skills were	sex, BMI, baseline PA.

Reference	Description	Indicator of motor development/coordination	Physical activity (PA) outcome	Summary of results	Adjustment for confounding
	Age: 18 years (mean)	(locomotor) and figure-8 (manipulative) tests.	from IPAQ.	associated with higher METs, LPA, MPA and VPA.	
Ridgway et al. 2009b (303)	1966 Finnish birth cohort Age: 14 years	ages at standing and walking unsupported reported at 12 months	school PE grade; frequency of sports; number of different sports performed.	later attainment of milestones was weakly associated with lower PE grade, less frequent sports and lower number of different sports. E.g. PE grade per later month at walking = - 0.06 (95%Cl: -0.08 to -0.04), p<0.001.	sex, gestational age, birth season, childhood SEP, birth weight, BMI at 14 years.
Green et al. 2011 (296)	Avon Longitudinal Study of Parents and Children (ALSPAC) Age: 11-13 years	Motor coordination tested at 7 years: manual dexterity, ball skills and balance.	daily average moderate to vigorous physical activity participation (3600 cpm)	poor targeting skill/object control at age 7–8 years weakly associated with less MVPA in boys but not in girls.	season, age, neonatal factors, childhood SEP, maternal PA & smoking, myopia
Mattocks et al. 2008 (247)	Avon Longitudinal Study of Parents and Children (ALSPAC) Age: 11-12 years	Motor coordination assessed at 6 months by questionnaire	PA counts per minute assessed by accelerometer	Motor coordination at 6 months associated with PA counts in children aged 11-12 years (β =5.77, 95%CI 0.25, 11.29, p= 0.041).	age, sex, childhood SEP
de Souza et al. 2014 (297)	Azorean school children Age: 10 years	composite of balance, jump, hop shifting platforms	Self-reported LTPA METs	better coordination associated with higher LTPA METs	none
Lopes et al. 2011 (298)	Azorean school children Age: 6-10	composite of balance, jump, hop shifting platforms	Self-reported LTPA METs at	lower motor coordination	sex, age

Reference	Description	Indicator of motor development/coordination	Physical activity (PA) outcome	Summary of results	Adjustment for confounding
	years		6, 7, 8, 9 and 10 years	associated with greater decline in LTPA	
Hnatiuk et al. 2013 (302)	206 toddlers from the Melbourne InFANT Program Age: 19 months	Age at walking	accelerometers	Later age at walking associated with lower physical activity: $\beta = -4.30$ minutes/day (95% CI: -6.69, - 1.90).	Unclear as only presented in text
Lubans et al. 2010 (285)	Systematic review in Children, adolescents and young adults Age: various childhood and adolescence	running, jumping, hopping, leaping, galloping, skipping, sliding, striking, dribbling and balance.	self-reports and accelerometers	consistent cross- sectional associations of competence in motor skills and coordination with greater cardiorespiratory fitness, lower BMI and more PA.	various
Holfelder and Schott 2014 (287)	Systematic review in Children, adolescents and young adults Age: various childhood and adolescence	running, jumping, hopping, leaping, galloping, skipping, sliding, striking, dribbling and balance.	self-reports and accelerometers	consistent cross- sectional associations of competence in motor skills and coordination with more PA.	various
Øglund et al 2015 (248)	Systematic review Age: ≤ 18 years.	Early motor development and coordination	accelerometers	weak associations between motor coordination, age at walking and more PA.	various

5.2 Methods

5.2.1 Explanatory variables

Infant motor milestones

Ages at attaining three infant motor milestones (sitting alone, standing alone and walking several steps unsupported) were reported by the mother to the nearest month when the child was 2 years old (in 1948). These resulted in three continuous measures representing the age in months at sitting, standing and walking. As previous findings from NSHD showed that those with early or late attainment of milestones had poorer physical function than those reaching milestones around the modal age (294), each measure was categorised into three groups to compare the early and late developers (approximately equivalent to the 5th and 95th percentiles respectively) with those on time/average developers. For sitting, this resulted in participants grouped as sitting before 6 months, between 6-8 months or after 8 months. For standing, participants were grouped as standing before 9 months, between 9-14 months, or after 14 months. For walking, participants were classified as walking before 11 months, between 11-17 months, or after 17 months. Where evidence of nonlinear associations with LTPA was not found then continuous milestone measures were also examined.

Ability at school games at age 13 years

In 1959, when study participants were aged 13 years old, the school teacher who was most familiar with each study participant completed a school-based questionnaire rating their ability in school games as above average, average or below average compared with their peers (93). Those with above average and below average ability were compared to those with average ability at games. This measure is used as a marker of study participants' overall ability at school-based games including activities requiring competence in motor skills and coordination (e.g. team sports, physical education, athletics), and was previously shown to relate to LTPA at age 36 in NSHD (93).

Tapping speed at age 15 years

Motor speed and lateralised coordination of the upper and lower limbs was assessed by finger and foot-tapping speed tests at a school-based medical examination when study participants were 15 years old (1961). At the medical exam, the school physician recorded the number of times in 15 seconds that study participants could tap the dorsum of their right hand with their left finger and tap the ground with their left foot, with the tests then repeated for the right finger and foot. For the purposes of these analyses, the highest scores of both left and right limbs in finger-tapping and foot-tapping tests were calculated and these were grouped in multiples of 10 for analysis, as done in a previous NSHD study (295).

5.2.2 Confounding variables

Birth weight, birth order, serious childhood illness and father's occupational class were selected as confounders based on existing literature (248, 273, 278, 303-305) and earlier findings from this thesis. Birth weight was selected as a confounder as it was hypothesised that low birth weight would be associated with delayed motor development and poorer motor coordination (278). It was also reported in chapter 4 that birth weight was associated with adulthood LTPA. Birth order was selected as a confounder as it was hypothesised to be associated with all motor indicators and also with LTPA in adulthood. It was also hypothesised that a serious illness in childhood would be associated with later attainment of milestones, lower ability at games and poorer motor coordination (274), and also with less LTPA in adulthood (68). Finally, father's occupational class was chosen as a confounder because it was hypothesised, based on systematic review findings reported in chapter 3, that a lower childhood SEP would be associated with lower participation in LTPA across adulthood (151). It was also hypothesised that childhood SEP would be associated with attainment of motor milestones, ability at games and also with poorer motor coordination (305, 306).

Birth weight was extracted from birth records within 6 weeks of delivery and grouped into five categories with the low birth weight set as reference (see chapter 4). Birth order was reported by the mother and study participants were classified as first, second or third and later born. Information was obtained on serious childhood illness in the first 5 years of life which required hospital stay lasting a minimum of 28 days. This information was used to group participants into whether or not they had any serious illness. Illnesses included poliomyelitis, disorders of the central and peripheral nervous systems and mental disability which could be associated with motor development and function. A range of other illnesses requiring hospital stay including congenital conditions and pneumonia were also included in this group. Father's Registrar General's occupational class at age 4 (in 1950) was used to indicate childhood SEP and was grouped into four categories (see chapter 4).

5.2.3 Examining associations with LTPA across adulthood

Details of initial exploratory analyses and investigations of sex interactions and deviation from linearity which were carried out are described in chapter 2, section 2.3.1. Descriptive analyses (chi-squared tests for categorical variables and mean estimation/t-tests for continuous variables) were initially carried out to examine the distribution of each early life motor performance measure with the selected covariates as well as the interrelationships among the motor performance measures. Mixed-effects binary and multinomial logistic regression models were used to examine associations between each early life motor indicator and LTPA across adulthood (between ages 36-68 years) in study participants with at least one measure of LTPA (see chapter 2.3.2 for detail of these models). Binary mixed-effects models were used to estimate the ORs of participation in LTPA (versus nonparticipation) by each early life motor indicator whereas multinomial mixed-effects models were used to estimate the RRRs of moderate and regular participation in LTPA across adulthood (versus nonparticipation) by each early life motor indicator. The associations between each early life motor indicator and LTPA at each age in adulthood were also examined with separate binary and multinomial logistic regression models in study participants with complete LTPA data at each age.

All models were fit separately for each early life motor indicator and adjusted in steps for (a) birth weight, birth order and serious illness, and (b) father's occupational class. In addition, linear regression was used to examine the difference in MVPA time and PAEE assessed by monitors at age 60-64 and each early life motor indicator in the subsample of study participants with these data.

5.3 Results

5.3.1 Relation of motor performance measures to covariates

The distribution of each early life motor performance indicator overall and by sex in the sample with at least one measure of LTPA and data on the selected covariates is shown in Table 5.2. There was little difference between males and females in the age at attaining infant milestones. Higher proportions of boys than girls were rated as below average in school games at age 13 but boys had faster finger- and foot-tapping speed than girls at age 15 (Table 5.2).

Later attainment of all three milestones of sitting, standing and walking was associated with lower birth weight and serious childhood illness while earlier attainment of all milestones was related to later birth order. For all three milestones, later attainment was associated with higher father's occupational class with higher proportions of those in lower father's occupational classes reaching milestones at an earlier age. Below average ability at school games was more prevalent among those born with low birth weight while above average ability was related to later birth order. Higher proportions of those with childhood illness were below average at school games while lower proportions were above average. Below average ability at games was also more prevalent in lower father's occupational classes. Faster tapping speed was positively associated with birth weight such that heavier birth order such that first born children tended to tap faster than those who were later born while those with childhood illness tapped slower than those without any such illness. Lastly, faster tapping speed was associated with higher father's occupational classe.

	Overall	Males	Females	test of sex- difference
Age at reaching milestones in months (n=3217) Sitting (mean age) Sitting (age groups) ≤ 5m 6-8m	6.6 (0.04) 604 (18.9) 2308 (71.7)	6.6 (1.5) 312 (19.2) 1152 (70.8)	6.5 (1.5) 292 (18.4) 1156 (72.8)	p=0.05 p=0.4
≥ 9m	305 (9.5)	164 (10.1)	141 (8.9)	
Standing (mean age) Standing (age groups)	11.4 (0.04)	11.5 (2.4)	11.3 (2.1)	p=0.1 p=0.07
≤ 8m 9-14m ≥ 15m	175 (5.4) 2779 (86.4) 263 (7.1)	88 (5.4) 1389 (85.3) 151 (9.3)	87 (5.5) 1390 (87.5) 112 (7.1)	p olor
Walking (mean age) Walking ((age groups) ≤ 10m 11-17m	13.6 (0.04) 263 (8.2) 2695 (83.8)	13.7 (2.6) 126 (7.7) 1356 (83.3)	13.6 (2.5) 137 (8.6) 1339 (84.3)	p=0.2 p=0.1
≥ 18m	259 (8.1)	146 (9.0)	113 (7.1)	
<u>Ability at school games at age 13 (n=3108)</u> Above average Average Below average	590 (19.0) 2057 (66.2) 461 (14.8)	312 (19.9) 991 (63.0) 269 (17.1)	278 (18.1) 1066 (69.4) 192 (12.5)	p<0.001
<u>Number of taps in 15</u> seconds at age 15 in multiples of 10 (n=2882) Finger taps Foot taps	5.6 (0.03) 5.0 (0.03)	5.8 (1.8) 5.1 (1.6)	5.5 (1.7) 5.0 (1.5)	P<0.001 p=0.2

Table 5.2 Distribution of motor performance measures overall and by sex in those

 with data on covariates and at least one measure of LTPA.

Numbers and % for categorical variables and mean and standard deviation for continuous variables. Sex difference tests were Chi squared for categorical variables and t-test for continuous variables.

5.3.2 Interrelationships among motor performance measures

Higher proportions of those who were late at sitting, standing and walking unsupported were below average at school games at age 13 years while earlier milestone attainment was associated with above average ability. For example, among those walking at or after 18 months, prevalence of above average ability was 4.0% and below average ability was 12.3% (p<0.001). This association was also seen when examining continuous milestones measures using logistic regression. There was little evidence of association between attainment of milestones and fingerand foot-tapping speeds at age 15 however, above average ability at school games at age 13 was associated with faster tapping speeds while below average ability was related to slower speed.

5.3.3 Age at reaching motor milestones and LTPA across adulthood

Formal tests of deviation from the linear trend showed evidence of non-linear associations between milestones and LTPA which were stronger for age at walking (significant p-values for quadratic milestones) (Table 5.3). Therefore, modelling of ages at sitting and standing as continuous (in addition to categorical) variables was justified whereas age at walking was modelled as a categorical variable. There was no evidence of effect modification by sex in any of the models with age at reaching milestones (in all models p>0.2) so all results are presented for both sexes combined and adjusted for sex. As described in chapters 2 and 4, there was a sex by age interaction which was added to all mixed-effects models.

There was no evidence of an interaction with age suggesting that any associations found between milestones and LTPA did not vary by age at assessment of LTPA (p=0.9 for continuous sitting by age interaction, p=0.8 for categorical sit by age interaction, p=0.3 for continuous standing by age interaction, p=0.6 for categorical standing by age interaction, p=0.9 for categorical walking by age interaction). This is consistent with the similar ORs of LTPA at each age in adulthood (Appendix 3A).

	• •		0	0	
			p-values		
LTPA	36	43	53	60-64	68
Sit	0.2	0.2	0.1	0.6	0.01
Sit ²	0.3	0.3	0.2	0.5	0.02
stand	0.5	0.3	0.06	0.1	0.1
Stand ²	0.6	0.5	0.2	0.2	0.2
walk	0.02	0.001	0.01	0.1	0.1
walk ²	0.03	0.006	0.03	0.2	0.1

 Table 5.3 p-vales for quadratic terms for age at reaching motor milestones.

Models include linear and quadratic terms for age at reaching motor milestones and adjusted for sex, maximum sample size used at each age.

Table 5.4 presents the ORs of LTPA across adulthood estimated from mixed-effects binary logistic regression analysis of 3217 men and women (49.4% female) with at least one measure of LTPA and complete data on motor milestones, birth weight, birth order, childhood illness and father's occupational class. Per month later age at standing was associated with 0-7% higher ORs of LTPA across adulthood (Table 5.4). When compared with the group walking at 11-17 months, those walking before 11 months were less likely to participate in LTPA and similar but less striking differences were found when comparing those standing before 9 months with those standing between 9-14 months (Table 5.4). These sex-adjusted associations were slightly strengthened by additional model adjustments for birth weight, birth order and childhood illness but the strength of these associations was considerably attenuated following further adjustment for father's occupational class. There was no evidence of association between age at sitting and ORs of LTPA across adulthood (Table 5.4).

Table 5.5 presents the RRRs of moderate and regular participation in LTPA (versus no LTPA) across adulthood by age at reaching milestones estimated using mixedeffects multinomial logistic regression in the same sample (n=3217). Like for the binary mixed-effects models reported in Table 5.4, these models showed that those walking ≤ 10 months were slightly less likely to be moderately and regularly active in LTPA across adulthood than those walking at 11-17 months, and these associations were attenuated by adjustment for childhood SEP (Table 5.5). The ORs of any LTPA and RRRs of moderate and regular LTPA (versus none) at each age in adulthood in 1457 men and women (54.0% female) with all five measures of LTPA and data on milestones, birth weight, birth order, childhood illness and father's occupational class are presented in Appendix 3A and Appendix 3B respectively. As for the mixed-effects models, these models also showed little evidence of association between age at sitting and LTPA, and some associations between earlier ages at standing and walking and lower likelihood of LTPA which were fully attenuated by adjustment for childhood SEP (Appendices 3A and 3B).

Examining the ORs of team sports (n=2842), non-team sports LTPA (n=2818) and leisure-time walking (n=2836) at age 36 showed little evidence of associations between the ages at reaching milestones and any of these outcomes (Appendix 3C). Lastly, age at reaching each motor milestone (n=1466) was not clearly associated with time spent in MVPA or with PAEE assessed by activity monitors at age 60-64 years in the sample with data on LTPA at that age (Appendix 3D).

Table 5.4 Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisure-time physical activity (LTPA) between ages 36 and 68 years by age at reaching infant motor milestones: mixed-effects binary logistic regression

	OR (95% CI) of LTPA at least once per month between ages 36 and 68 years versus no LTPA				
	Model 1	Model 2	Model 3		
Age at reaching infant milestones in months Sitting					
≤ 5m (n=604) 6-8m (n=2308) ≥ 9m (n=305) test of association	0.92 (0.77 to 1.09) 1.00 (reference) 1.04 (0.82 to 1.32) p=0.6	0.93 (0.78 to 1.11) 1.00 (reference) 1.10 (0.87 to 1.40) p=0.5	0.98 (0.82 to 1.16) 1.00 (reference) 1.01 (0.80 to 1.28) p=0.95		
per one month increase test of association	1.03 (0.98 to 1.08) p=0.2	1.04 (0.99 to 1.09) p=0.09	1.01 (0.97 to 1.06) p=0.7		
<i>Standing</i> ≤ 8m (n=175) 9-14m (n=2779) ≥ 15m (n=263) test of association	0.78 (0.57 to 1.07) 1.00 (reference) 1.02 (0.79 to 1.31) p=0.3	0.79 (0.58 to 1.08) 1.00 (reference) 1.08 (0.84 to 1.39) p=0.3	0.83 (0.62 to 1.13) 1.00 (reference) 0.94 (0.73 to 1.20) p=0.5		
per one month increase test of association	1.03 (1.00 to 1.06) p=0.05	1.04 (1.01 to 1.07) p=0.02	1.01 (0.98 to 1.05) p=0.3		
<i>Walking</i> ≤ 10m (n=263) 11-17m (n=2695) ≥ 18m (n=259) test of association	0.69 (0.54 to 0.89) 1.00 (reference) 0.98 (0.76 to 1.26) p=0.02	0.72 (0.56 to 0.93) 1.00 (reference) 1.08 (0.84 to 1.39) p=0.03	0.84 (0.66 to 1.08) 1.00 (reference) 0.95 (0.74 to 1.22) p=0.4		

Analytic sample consists of those with at least one measure of LTPA (n=3217). Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Includes sex by age interaction. Tests of association based on likelihood ratio tests comparing models with and without milestones terms. **Table 5.5** Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% CrI) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by age at reaching infant motor milestones: mixed-effects multinomial logistic regression

LTPA between ages 36-68	RRR (95% CrI) of moderate LTPA (1-4 times per month) versus no LTPA				RRR (95% Crl) of regular LTPA 5 or more times per month) versus no LTPA		
	Model 1	Model 2	Model 3		Model 1	Model 2	Model 3
Age at reaching infant milestones in months							
Sitting							
≤ 5m (n=604)	0.96	0.97	1.02	,	0.89	0.90	0.98
6-8m (n=2308)	(0.80 to 1.14) 1.00 (reference)	(0.81 to 1.17) 1.00 (reference)	(0.86 to 1.21) 1.00 (reference)	((0.70 to 1.09) 1.00 (reference)	(0.72 to 1.14) 1.00 (reference)	(0.76 to 1.21) 1.00 (reference)
≥ 9m (n=305)	1.08	1.13	1.06		1.02	1.09	1.02
Bayesian DIC	(0.84 to 1.37) 22073.87	(0.89 to 1.39) 22061.50	(0.84 to 1.32) 22003.29	((0.76 to 1.39) -	(0.82 to 1.41) -	(0.76 to 1.34) -
per later month	1.01	1.03	1.00		1.02	1.06	1.01
Bayesian DIC	(0.97 to 1.05) 22084.35	(1.00 to 1.08) 22050.60	(0.97 to 1.05) 21995.67	((0.96 to 1.06) -	(1.00 to 1.10) -	(0.97 to 1.06)
Standing							
≤ 8m (n=175)	0.83	0.83	0.88		0.80	0.78	0.85
9-14m (n=2779)	(0.59 to 1.18) 1.00 (reference)	(0.61 to 1.11) 1.00 (reference)	(0.64 to 1.17) 1.00 (reference)	((0.53 to 1.18) 1.00 (reference)	(0.53 to 1.14) 1.00 (reference)	(0.57 to 1.22) 1.00 (reference)
≥ 15m (n=263)	(0.84 to 1.42)	(0.88 to 1.41)	0.998 (0.77 to 1.27)	((0.72 to 1.39)	(0.79 to 1.40)	0.90 (0.65 to 1.23)
Bayesian DIC	22092.66	`22078.16´	21990.71	·	-	-	-

per later month	1.04	1.04	1.03	1.01	1.03	1.02
Bayesian DIC	(1.01 to 1.07) 22075.90	(1.01 to 1.07) 22073.14	(1.00 to 1.06) 21992.70	(0.98 to 1.06) -	(0.99 to 1.06) -	(0.98 to 1.04) -
Walking						
≤ 10m (n=263)	0.75	0.79	0.92	0.65	0.68	0.84
	(0.58 to 0.96)	(0.61 to 1.02)	(0.71 to 1.16)	(0.48 to 0.87)	(0.49 to 0.95)	(0.61 to 1.11)
11-17m (n=2695)	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 18m (n=259)	1.07	1.14	1.06	0.91	0.99	0.91
	(0.84 to 1.35)	(0.88 to 1.45)	(0.83 to 1.33)	(0.67 to 1.22)	(0.70 to 1.32)	(0.67 to 1.25)
Bayesian DIC	22070.28	22061.97	21992.32	-	-	-

Analytic sample consists of those with at least one measure of LTPA (n=3217). Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Includes sex by age interaction. Bayesian DIC statistics indicate fit for whole models.

5.3.4 Ability at games and LTPA across adulthood

There was no evidence of effect modification by sex in any of the models with ability at school games (in all models p>0.4, with the exception of a marginal evidence of interaction at age 36 (p=0.05)) so all results are presented for both sexes combined and adjusted for sex. Ability at school games at age 13 was associated with adult LTPA and there was some evidence that this association changed with age (p=0.06 for ability at games by age at LTPA interaction in binary mixed-effects models). Therefore, results from a binary mixed-effects model with an interaction term for ability at games and age at LTPA are summarised in a plot showing the log-odds of LTPA at each age by ability at school games (Figure 5.2). Associations with LTPA at each age are presented in Appendices 3E and 3F and summarised in the text below.

The ORs of any LTPA and RRRs of moderate and regular LTPA (versus none) at each age in adulthood in 1442 men and women (53.4% female) with all five measures of LTPA and data on ability at school games, birth weight, birth order, childhood illness and father's occupational class are presented in Appendix 3E and Appendix 3F respectively. When compared with those rated as average at school games, those with above average ability were more likely to participate in LTPA at each age in adulthood, e.g. sex-adjusted ORs (95%CI) of LTPA at ages 36 and 68 for above average versus average games ability were 1.48 (1.09 to 2.01) and 1.45 (1.11 to 1.91) (Appendix 3E).

When compared with those rated as average at school games, there was a suggestion that those with below average ability were less likely to participate in LTPA at ages 36 and 43 but not at older ages, e.g. sex-adjusted ORs (95%CI) of LTPA at ages 36 and 68 for below average versus average games ability were 0.78 (0.56 to 1.08) and 1.03 (0.75 to 1.43) (Appendix 3E). These associations were unaffected by adjustment for hypothesised confounding by birth weight, birth order, hospitalising childhood illness and father's occupational class (Appendices 3E and 3F). The multinomial analyses showed that, up to age 53, associations between above average ability and participation in LTPA were more apparent when comparing regular participation with none. However, at ages 60-64 and 68 years, games ability was associated with both moderate and regular participation in LTPA (Appendix 3F).

Figure 5.2 shows a plot of the log-odds of LTPA (at least once per month versus no LTPA) at each age in adulthood by teacher-rated ability at school games at age 13

estimated from mixed-effects binary logistic regression analysis of 3108 men and women (49.4% female) with at least one measure of LTPA and complete data on ability in school games, birth weight, birth order, childhood illness and father's occupational class. These plots are presented separately for men and women because of a sex by age interaction which means that the decline in LTPA is greater in men than women. An interaction term of ability at games by age is also included (p=0.06). Consistent with separate models from each age, the plots show that those with above average ability at games had the highest likelihood of LTPA across adulthood and those with below-average ability the lowest. In addition, the plots show that those with average ability at games had more pronounced decline in likelihood of LTPA than both these groups (Figure 5.2).

Examining the ORs of team sports (n=2784), non-team sports LTPA (n=2756) and leisure-time walking (n=2777) at age 36 showed that ability at school games was associated with both team sports and non-team sports LTPA however, games ability was not associated with leisure-time walking (Appendix 3G). Lastly, analyses of the objective measures of PA recorded at age 60-64 in those with data on LTPA at that age (n=1424) showed weak associations in the expected direction between better ability at games and higher levels of MVPA and PAEE. The estimates suggest that, when compared with average ability at games, the group with above average ability tended to spend greater time in MVPA and had higher PAEE with opposite being the case for the group with below average ability (p=0.3 from both final models). Games ability was more strongly associated with LTPA at that age (Appendix 3H)

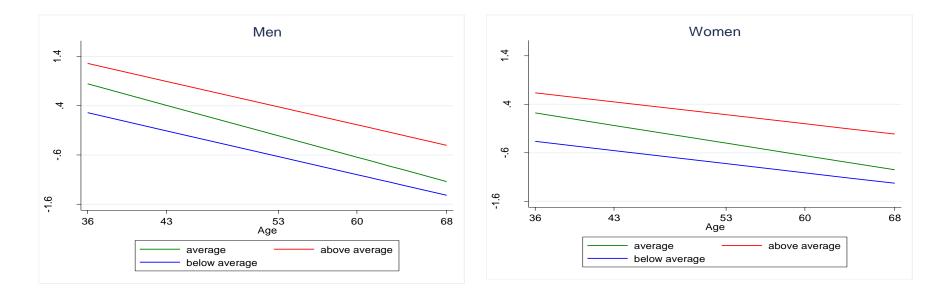


Figure 5.2 Log-odds of leisure-time physical activity at each adult age by ability in school games at age 13 in men and women.

Figure 5.2 legend: Based on the estimated fixed effects (coefficients) $x\beta$ in the model (mean log-odds of leisure-time physical activity at each age for those with below average, average and above average ability at school games at age 13). Log-odds are stratified by sex due to an interaction of sex by age which means that the decline in LTPA is greater in men than women.

5.3.5 Tapping speed and LTPA across adulthood

Formal tests of deviation from the linear trend showed no evidence of non-linear associations between tapping scores and LTPA at any age (in all models p>0.3) therefore, treating the ordinal variables as continuous finger and foot-tapping scores was justified. There was no evidence of effect modification by sex in any of the models (in all models p>0.4, with the exception of foot-tapping and LTPA at age 60-64, p=0.01),) so all results are presented adjusted for sex. Faster finger- and foot-tapping speeds were associated with higher likelihood of participation in LTPA across adulthood including after adjustment for hypothesised confounders. There was no evidence of an interaction between finger- and foot-tapping speed and age at assessment of LTPA (p=0.4 for finger-tapping by age interaction; p=0.5 for foot-tapping by age interaction) suggesting associations persisted across adulthood. This is consistent with the similar ORs of LTPA at each age in adulthood in study participants with non-missing LTPA (Appendix 3I).

Table 5.6 presents the ORs of LTPA across adulthood per 10-unit higher finger and foot-tapping scores estimated using mixed-effects binary logistic regression analysis in 2882 men and women (49.2% female) with at least one measure of LTPA and complete data on motor milestones, birth weight, birth order, childhood illness and father's occupational class. Per 10-unit higher number of both finger and foot taps were associated with higher likelihood (ORs) of participating in LTPA across adulthood between 36-68 years (Table 5.6). These associations were only slightly attenuated by further adjustment for all selected early life covariates (Table 5.6).

Table 5.6 Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisure-time physical activity (LTPA) between ages 36 and 68 years by tapping speed at age 15: mixed-effects binary logistic regression.

	OR (95% CI) of LTPA at least once per month between ages 36 and 68 years versus no LTPA					
	Model 1 Model 2 Model 3					
Number of taps age 15 (per 10-unit increase) Finger-tapping	1.10 (1.05 – 1.14)	1.09 (1.04 – 1.13)	1.07 (1.03 – 1.11)			
test of association	p<0.001	p<0.001	p=0.001			
Foot-tapping test of association	1.11 (1.06 – 1.16) p<0.001	1.10 (1.05 – 1.15) p<0.001	1.09 (1.04 – 1.14) p<0.001			

Analytic sample consists of those with at least one measure of LTPA (n=2882). Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Includes sex by age interaction. Tests of association based on likelihood ratio tests comparing models with and without finger-/foot-tapping speed terms.

Table 5.7 presents the RRRs of moderate and regular LTPA across adulthood per 10-unit higher finger and foot-tapping scores estimated using mixed-effects multinomial logistic regression analysis in the same sample (n=2882). Per 10-unit higher scores on both finger and foot-tapping speed tests were associated with higher likelihood (RRRs) of both moderate and regular LTPA (versus none) across adulthood between 36-68 years (Table 5.7). These associations were only slightly attenuated by further adjustment for all selected early life covariates (Table 5.7).

The ORs of any LTPA and RRRs of moderate and regular LTPA (versus none) at each age in adulthood per 10-unit higher finger and foot-tapping scores in 1347 men and women (53.6% female) with all five measures of LTPA and data on finger and foot-tapping speed, birth weight, birth order, childhood illness and father's occupational class are presented in Appendix 3I and Appendix 3J respectively. There was consistent evidence found to suggest that higher finger and foot-tapping speed at age 15 years were associated with higher likelihood of LTPA at each age in adulthood (Appendices 3I and 3J).

Examining the ORs of team sports (n=2584), non-team sports LTPA (n=2559) and leisure-time walking (n=2577) at age 36 showed that tapping speed was associated with all three outcomes at that age (Appendix 3K). Lastly, analyses of the objective measures of PA recorded at age 60-64 in those with data on LTPA at that age

(n=1326) showed that faster tapping speed was weakly associated with greater time spent in MVPA (in all models p>0.1) but not with PAEE (in all models p>0.5) assessed by monitors at 60-64 years (Appendix 3L).

Table 5.7 Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% Crl) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by tapping speed at age 15: mixed-effects multinomial logistic regression.

LTPA between ages 36-68	``	RRR (95% Crl) of moderate LTPA (1-4 times per month) versus no LTPA		RRR (95% Crl) of regular LTPA (5 or more times per month) versus no LTPA		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Number of taps age 15 (per 10- unit increase) (n=2882)						
Finger-tapping	1.07	1.07	1.05	1.13	1.13	1.10
2 2	(1.02 to 1.11)	(1.03 to 1.12)	(1.01 to 1.08)	(1.08 to 1.17)	(1.08 to 1.19)	(1.06 to 1.14)
Bayesian DIC	`19982.29 <i>´</i>	`19984.83 <i>´</i>	19935.50	· -	-	-
Foot-tapping	1.07	1.07	1.06	1.14	1.12	1.10
	(1.02 to 1.12)	(1.02 to 1.12)	(1.02 to 1.10)	(1.07 to 1.20)	(1.06 to 1.18)	(1.06 to 1.15)
Bayesian DIC	19986.76	19984.04	19938.12	· /	- /	

Analytic sample consists of those with at least one measure of LTPA (n=2882). Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Includes sex by age interaction. Bayesian DIC statistics indicate fit for whole models.

5.4 Discussion

5.4.1 Summary of findings

The aim of this chapter was to test the hypotheses that 1) later infant age at attainment of motor milestones, 2) below-average ability at games, and 3) slower tapping speed in adolescence would be associated with less participation in LTPA across adulthood. Regarding milestones, there was little evidence overall that age at reaching milestones was related to adult LTPA however, associations in the opposite direction to that hypothesised between early standing and walking and lower likelihood of participation in LTPA were found which were attenuated after adjustment for father's occupational class. Regarding ability at school games, those rated as above average at games by their school teacher were consistently more likely to participate in LTPA across adulthood when compared with those with average ability. Conversely, those rated as below average were slightly less likely to participate in LTPA than those with average ability but only at younger adult ages; due to a greater decline in likelihood of LTPA for those with average ability when compared with those with above or below average ability. Regarding tapping speed, faster finger- and foottapping speed were both associated with higher likelihood of participation in LTPA across adulthood. Associations between ability at games and tapping speed with adulthood LTPA were largely unaffected by adjustment for hypothesised early life confounders.

5.4.2 Comparison with other studies

The findings of this chapter showed that associations between ability at games and motor coordination (tapping speed) from early life with LTPA were consistent across a 32-year period in adulthood and demonstrated, for the first time, associations between motor function in adolescence and participation in LTPA across the sixth decade of life which were robust to adjustment for hypothesised confounders. Overall, the findings regarding ability at games and tapping speed are consistent with the few other epidemiological studies that have examined associations between motor performance in early life and adulthood LTPA. These include associations in the 1958 British birth cohort between hand control/coordination problems at ages 7-16 years and lower self-rated sports aptitude at 16 and less LTPA between 33-50 (173). In addition, hand control/coordination problems in the 1958 cohort were also associated with persistent leisure-time inactivity between 33-50 years (267) (Table

5.1). The findings of this chapter are also consistent with those from the 1970 British birth cohort where poorer motor skills at age 10 (throwing, balance and walking backwards) were associated with lower likelihood of LTPA at age 42 (301).

While the findings of this chapter support the hypotheses outlined in 5.1.2 for ability at games and tapping speed and tend to be congruent with previous studies which examined similar associations in other younger populations (Table 5.1), this was not the case for infant milestones. Here, unexpected direction of association between earlier attainment and less LTPA were found which were attenuated after adjustment for childhood SEP. These initial associations contradict those in the only other study to examine how milestones relate to adult LTPA. That study showed those twins who learned to stand and walk earlier reported higher LTPA between 25-34 years than their less advanced twin (304).

Conversely, the finding that later attainment of all three infant motor milestones were associated with lower ability in games at 13 years could be considered consistent with few existing studies in children (Table 5.1) such as associations in a Finnish birth cohort between later ages at standing and walking unsupported with a lower school PE grade, less frequent participation in sports and a lower participation in different types of sports at age 14 (303). Moreover, those with lower ability in games had slower finger and foot-tapping speed which may support studies reporting reciprocal associations of motor ability with PA and fitness (282-284) and others that suggest LTPA improves psychomotor speed and coordination (289). No other studies identified in my comprehensive literature review have examined tapping speed from early life in relation to later LTPA.

5.4.3 Explanation of findings

This chapter shows that better motor skills, speed and coordination in early life as indicated by above average ability at games and faster finger and foot-tapping speed were consistently associated with higher likelihood of participation in LTPA across adulthood but concludes that attainment of infant motor milestones was not associated with adulthood LTPA. These findings suggest that motor function in adolescence may influence participation in LTPA across adulthood including at older adult ages and support the notion that the degree of motor competence is partially responsible for PA (47, 242, 248, 281, 300).

It is unclear why early walkers would be less likely to participate in LTPA than those on average. The maturation deviance hypothesis (307) (see chapter 6) could be an explanation as it states that those who are early or late at development might be worse off than those who develop on time. However, this explanation is not supported by the finding that higher proportions of early sitters, standers and walkers were rated as above average at games at age 13 years. The fact that associations between walking before 11 months and lower likelihood of participation in LTPA were attenuated by adjustment for father's occupational class suggest confounding by childhood SEP thus pointing to a social rather than biological explanation for this finding. This is supported by an examination of the social patterning of milestones where earlier attainment of milestones was more prevalent in lower father's occupational classes, which was in turn shown in chapter 3 to be associated with less LTPA in adulthood.

On the other hand, genetic or neurological factors may explain why those with later attainment of milestones tended to have lower ability in games at 13 since this measure likely reflects motor function including skills and coordination at that age. In addition, lower games ability was related to slower tapping speed and assuming those with better games ability were more active in sports then potential mechanisms for association of ability in games with tapping speed could include effects of PA on oxidative capacity in the brain and trophic effects of PA on the CNS (289).

Those rated as above average at games by their school teacher were consistently more likely to participate in LTPA across adulthood when compared with those with average ability. This suggests that those with better motor skills and good competence in activities such as team sports at school continue to participate in LTPA across life including across adulthood and is consistent with the notion of tracking of PA from childhood to adulthood (91). Conversely, those rated as below average were slightly less likely to participate in LTPA than those with average ability but only at younger adult ages due to a greater decline in LTPA for average ability when compared with both above or below average ability. It is unclear why the average group experiences the greatest decline in LTPA and this requires investigation in other long running studies. However, one explanation may be that those who were above average at school games maintain LTPA participation throughout life and those with below average ability take up participation due to awareness of their need to improve involvement in any kinds of LTPA while those who were ranked average feel no compulsion to maintain LTPA.

146

Lastly, tapping speed in adolescence was consistently associated with LTPA across adulthood. Although the phenomenon underlying tapping is not well understood (288), the pathways behind the associations found between faster tapping speed and LTPA may include a reflection of differences in development of the CNS, cerebellum and other brain structures. That this association did not weaken with age may be congruent with the hypothesis that motor skill competence and PA will strengthen over time from early childhood to adulthood (281) however, this chapter only examined associations across later adulthood rather than during the transition from childhood to adulthood and thus this specific hypothesis could not be examined.

The consequences of poorer motor skills and coordination in early life may explain why some adults participate and other do not. For example, it has been hypothesised that proficiency in motor skills underpins an active lifestyle in that children with better motor ability might perceive greater self-competence in sports which would in turn results in them participating in LTPA (47, 242, 273, 281, 300). Without competence in movement skills such as running and jumping, and object control skills such as kicking and catching, children are less likely to access the range of PA options available to establish an active lifestyle. The limited motor competence may lead to unpleasant experiences in movement activities which may discourage participation in sport in childhood and LTPA across life (242, 273, 281). In addition, these findings were independent of birth weight and thus not explained by the effects of low birth weight on LTPA described in the previous chapter. The differences in findings between milestones and the two adolescent indicators of motor performance may reflect differences between motor development in infancy and adolescence and suggests the importance of adolescence for developing LTPA skills.

5.4.4 Methodological considerations

The methodological considerations relevant to all analyses carried out in this thesis are discussed in chapter 7 and only those which are specific to this chapter are discussed here.

Reporting bias by parental social class for milestones is possible but unlikely since associations in the expected direction between earlier attainment of milestones and better ability at games were found. Tapping speed was directly measured by a school-based physician at medical exam however, the movement involved in singlefinger tapping is complex and affected by visual and auditory stimuli, emotional and physical health, and factors that impact the skeletal and nervous systems and potentially also influence LTPA. These factors may therefore partially explain the associations found in this chapter between faster tapping speed and higher likelihood of LTPA. The use of the highest scores of each hand and foot is an advantage of this analysis as it accounts for dominant hand performance however, studies are needed to further understand the processes underlying upper and lower limb tapping speed. Faster psychomotor speed has been reported in young athletes than in non-athletes (289) and thus studies future should adjust for adult motor or physical capability in order to examine the possibility of reverse causality bias (i.e. the possibility that tapping speed is influenced by PA).

5.4.5 Implications of findings

The findings reported in this chapter suggest that motor skills including lateralised speed and coordination in adolescence are important correlaes of participation in LTPA across adulthood. When considered in the context of other recent longitudinal studies they suggest that ways of improving motor skills and coordination in early life could have great benefits for promoting LTPA across life. Reviews of interventions to improve motor development, skill and coordination tend to report successful improvements in children's motor skills and PA (308-310). This should be reassuring and the findings of this chapter suggest these interventions may have long-term benefits for LTPA in adulthood. Both motor skill development and increasing PA should simultaneously be targeted in PA interventions (281). In addition, the finding of associations between school games ability and adult LTPA highlights that interventions should include those based in school and involving teachers and supported by studies showing that schools with greater opportunities for sports participation can help adolescents maintain participation into adulthood (311).

There is a decline in motor function, including tapping ability (312), as well as PA (53) and physical capability (313) with age therefore future studies should collect repeat measures of motor function so as to investigate the interrelationships of motor ability and LTPA over the life course. This will help to further understand the relative strengths of each direction of association and could shed further light on whether motor ability is the main driver for LTPA or whether LTPA is the main driver of motor ability. Studies should also investigate whether this decline is modified by PA as some studies suggest that while older active participants have slower movement and

reaction time than young active participants they tend to perform better than both old inactive and young inactive participants (289). This chapter's findings suggest that interventions aiming to improve motor function and subsequent participation in LTPA should focus on motor performance of older children and adolescents in order to benefit adult LTPA. However, the scarcity of studies examining the role of infant motor development means that more studies in different cohorts are needed to establish whether attainment of the infant milestones and other indicators of motor skills and coordination in infants and young children are related to subsequent LTPA.

5.4.6 Conclusions

This chapter aimed to examine how attainment of infant motor milestones, ability at school games and tapping speed in adolescence relate to LTPA across adulthood. The findings showed there was little evidence of associations between age at attainment of milestones and adulthood LTPA but those with above average ability at games at age 13 and faster tapping speed at age 15 (better motor coordination) were more likely to participate in LTPA across adulthood including at older ages and independently of selected early life covariates including birth weight and SEP. The findings suggest that interventions which improve motor coordination in early life may be best target during late childhood and adolescence and that they have the potential for promoting LTPA across life including at older adult ages. The following chapter examines how age at puberty relates to LTPA in adulthood.

Chapter 6: Age at puberty and leisure-time physical activity across adulthood

Chapter objective: to examine the associations between age at puberty and LTPA across adulthood, and to investigate whether associations with LTPA vary by age.

This chapter examines how age at puberty, i.e. timing of the process of sexual maturation through which the adolescent ultimately develops into the mature adult state, and which was hypothesised to be on the pathways between birth weight and LTPA, might relate to LTPA in adulthood. A review of relevant studies provides background to the aims and objectives of the study and is followed by a description of methods, results and discussion of main findings.

6.1 Background

6.1.1 Literature review

A number of important events unfold during adolescence including the onset of puberty leading to development of reproductive capability (sexual maturation), a growth spurt in height and rapid changes in body proportions and composition (skeletal and somatic maturation) (314-316). Sexual maturation is a continuous process extending from embryonic sexual differentiation through to puberty and to full sexual maturity (47, 242, 273, 281, 300). During puberty, sexual maturation occurs under the influence of gonadal steroid hormones (predominantly testosterone in males and estradiol in females) and the adrenal androgens, primarily dehydroepiandrosterone sulfate (316).

Adrenarche, the production of adrenal androgens, generally occurs 1 to 2 years before the other hormonal changes of puberty, although visible evidence of puberty is generally not apparent until after thelarche (onset of female breast development in girls or testicular enlargement in boys). Adrenarche causes the appearance of pubic hair, adult-type body odour, and acne, and is a separate process from that of the centrally mediated gonadarche (the process by which pituitary gonadotropins cause the ovaries in girls and the testes in boys to begin to grow and increase production of estradiol, testosterone and other sex steroids (316)). Therefore, early and late signs of puberty reflect different on-going hormonal and physiological characteristics (317).

Completion of the hormonally initiated transition into puberty is signalled by the social and psychological transitions to adulthood.

The first secondary characteristics of sexual maturity which indicate the beginning of puberty are breast development in girls and enlargement of genitalia in boys (314). Menarche, which refers to the 1st menstrual period, is the most commonly reported maturity indicator for adolescent girls and represents a late event in sexual maturation (314, 316). The most commonly used criteria for assessing sexual maturity are stages of pubic hair, breast and genital maturation as first described by Tanner in 1962 (318). In stage 1 (pre-pubertal) the secondary sexual characteristics are absent. Stage 2 (early puberty) is initial overt development of secondary sexual characteristics i.e. initial elevation of breasts in girls, initial enlargement of genitals in boys and initial appearance of pubic hair in both sexes. Stages 3 and 4 (mid-puberty) indicate continued maturation of each characteristic. The final stage indicates the adult or mature state (314, 318). Further, puberty is characterised by the adolescent growth spurt. A child generally grows 10 cm in the first year of life, half that (12-13 cm) in the second year, and then 5–6 cm each year until puberty (319). As puberty approaches, the growth velocity slows (preadolescent dip) before its sudden acceleration during mid-puberty. The longer duration of prepubertal growth in combination with a greater peak height velocity in boys results in the average adult height difference of 13 cm between men and women (320).

The average age at starting puberty has declined over past decades, i.e. younger age at entering puberty over time within populations (321). Average age at starting puberty currently varies from 8-13 years in girls and 9-14 years in boys in different populations (321). There are wide variations between children in the age at entering puberty; girls are generally in advance of boys in the timing of puberty but the tempo (rate of development) overlaps considerably (314, 316). Variations in pubertal timing tend to reflect genetic influences although nutrition, psychological and socioeconomic conditions also play a role (319, 322-324). In addition, some early life factors including higher SEP, the presence of family conflict or parental divorce and growth rate in infancy and childhood within populations have been found to be associated with an earlier age at menarche (325).

Studies interested in the influence of pubertal timing have used a variety of selfreported and clinical assessments to assess maturity status. Measures of maturity during adolescence vary depending on the system examined and include skeletal, sexual and somatic maturity, which are reasonably related systems but the measures are specific to each system (307, 326). Assessment of skeletal age (with xrays/radiographs) is considered the gold-standard for determining biological maturity but is unfeasible for large population-based studies (326). The presence and status of secondary sexual characteristics has been widely used to examine the relative stage of puberty since these are outward indicators of level of sexual maturity. Somatic maturity can be assessed by the availability of frequently collected (e.g. biennially) longitudinal data which allows estimation of age at onset of the growth spurt and age at peak height velocity (327).

Adolescence and puberty are characterised by the development of many important self-initiated behaviours like smoking and LTPA (315, 328) which can track into adulthood, contributing to the role of adolescence as a possible sensitive life course period that can influence later health in addition to the biological changes described above (329). Moreover, besides the accepted importance of puberty and adolescence for the development of health behaviours, several studies suggest that deviation from normal timing of puberty (i.e. early or late maturity) may influence health and behaviour including LTPA. Several studies, including systematic reviews, guided by a priori selected hypotheses have examined the relationship between pubertal timing and PA (Table 6.1). The two main hypotheses considered by these studies are the early maturation hypothesis (330) and maturation deviance hypothesis (307). The early maturation hypothesis (330) proposes that early maturing adolescents, due to interruptions to their normal course of behavioural development, are at risk of adopting unhealthy behaviour. The maturation deviance hypothesis suggests that both early and late onset of puberty are associated with psychosocial problems in adolescents (307) and adulthood (331).

Two systematic reviews of cross-sectional studies (and a few of longitudinal studies with short follow-up duration of up to 24 months) tended to agree that overall, girls who matured earlier had lower PA levels while boys with advanced maturity showed greater involvement with PA (97, 332). The reviewed literature generally supported the early maturation hypothesis in girls with studies in boys sometimes favouring the maturation deviance hypothesis. However, these findings may be due to reverse causation where, for example, it may be that girls who are physically inactive mature earlier than others (333). Most studies included only samples of adolescent girls (97, 332) and thus associations in boys have been relatively understudied. There are

several biological and social pathways through which altered age at puberty may operate to influence PA during adolescence.

In male adolescents, advanced maturation tends to be associated with greater height, weight, weight for height, lean mass (i.e. muscle), and better performance in physical tasks (334). Consequently, larger size and muscle strength in early maturing boys could provide an advantage and motivation for sports participation during adolescence (97) making early maturing males better suited for performing most forms of exercise, particularly those requiring strength, speed, and power. In females, advanced maturation is also associated with greater gains in height, weight, and weight-for height but gains in weight are mainly associated with increased fat rather than muscle mass (335). Consequently, later maturing girls may be better suited to most exercise activities, particularly those that involve endurance or weight bearing (334).

Due to their older appearance, those maturing earlier than their chronological-age peers might be treated like adults and socialise with older friends, making them more susceptible to negative peer influences (336). Since PA declines with chronological age during adolescence, this means early maturing adolescents may adopt the lower levels of PA of their new older peer group. They might also be pressured by expectations which are above their own social, emotional and cognitive development (97). It is thought that the early secondary sexual characteristics might be more important than later signs for girls' PA for example, due to self-consciousness and a perceived discomfort associated with breast development (97). Moreover, it is possible that maturity-related variations in PA may be less pronounced in those with a high level of PA during childhood and preadolescence (97).

Some studies have also examined associations between pubertal stage and PA and attempted to understand underlying mechanisms through examining hypothesised mediators. A study of 11-year old girls found that early maturity (based on Tanner breast development stage, maternal report of Pubertal Development Scale, and estradiol blood levels) was associated directly, and indirectly via lower mental wellbeing and non-enjoyment of PA, with lower levels of moderate-vigorous PA at age 13 (337). More mature 11-year old girls (based on the Tanner scale which takes into account pubic hair and breast development) reported more negative perceptions of their physical appearance (based on their body fat and appearance) and less enjoyment of PA (338). In German adolescents aged 11-17 years, pubertal timing

based on public hair development, voice breaking (boys) and menarche (girls) was associated with boys' but not girls' LTPA after controlling for age, body fat and other socio-demographic covariates (339); surprisingly, compared with average maturing boys, early maturers were more likely to be inactive and late maturers less likely (339). Elsewhere, the proportion of skin covered by exercise-induced sweat was found to be higher in girls in more advanced puberty (340).

Evidence for the tracking into adulthood of PA behaviours developed in adolescence (91) suggests it is possible that maturity-related changes in LTPA could be maintained into later life. However, only a few studies, reporting mainly null-findings, have examined the long-term associations of age at puberty with LTPA in adults (Table 6.1). Self-perceived relative pubertal status reported by 12-19 year old Norwegian adolescents was not related to overall level of LTPA measured three times over thirteen years of follow-up (186). In that study however, early pubertal timing was associated with higher LTPA at the first follow-up assessment taken two years later though both males and females were included in the analyses and the authors did not describe their finding thus making detailed interpretation challenging (186). Further, this was based on perceived maturity (Table 6.1) which may not be concordant with independently or clinically assessed maturity. Elsewhere, age at puberty based on axillary hair stage in boys and age at menarche in girls (Table 6.1) was not associated with LTPA measured between 33 and 50 years in NCDS 1958 (173). Conversely, a later age at peak height velocity (later age at puberty) was correlated with more participation in sports by 40-year old Belgian men from a small study with multiple testing of hypotheses (176) and thus higher likelihood of chance finding.

Studies have also investigated the potential long-term health consequences of the age at entering puberty relative to peers. Analysis adjusted for multiple testing from the large UK Biobank cohort with self-reported health problems and recalled pubertal timing found earlier age at puberty to be associated with several health outcomes in men and women including type II diabetes and other metabolic, cardiovascular and psychiatric disorders (341). The findings showed that when compared with the average or on-time group, later age at reaching puberty (menarche in girls, voice breaking in boys) was associated with higher risks of these chronic disease outcomes (341). However, using recalled measures of pubertal development has been shown to be less accurate than when prospective and objective assessments are used in a study from NSHD (342). In men from NSHD, earlier age at reaching

puberty (based on a composite index of the development of genitalia, voice breaking, axillary and pubic hair) was associated with higher blood pressure at age 53 years (343) and it was suggested that less LTPA in men that were early maturing boys could be one explanation for this association (343). Other findings from NSHD showed that earlier age at menarche was associated with higher levels of triglycerides and total cholesterol at age 53 (344).

Due to the scarce number of studies on the long-term association of age at puberty with LTPA, more research is needed to understand if age at puberty might influence LTPA in adulthood in both men and women. This chapter aims to address these critical gaps in evidence.

6.1.2 Chapter aim and hypotheses

This chapter aimed to use prospective assessments of secondary sexual characteristics in boys, age at menarche of girls and repeat measurements of LTPA across adulthood to investigate the associations between age at puberty and LTPA across adulthood including whether these associations vary by age at assessment of LTPA. The hypotheses tested are that early maturing girls would be less likely to participate in LTPA across adulthood than their average-age maturing peers while early maturing boys would be more likely to participate.

Table 6.1 Summary of studies examining associations between age at puberty and physical activity in adults: arranged by age at assessment of physical activity

Reference	Description	Puberty assessment	Physical activity outcome	Summary of results	Covariates included in analysis
Pinto Pereira et al. 2014 (173)	British birth cohort born 1958 Age: 33, 40, 50	Axillary hair stage in boys (absent, sparse, intermediate, or adult). age at menarche in girls (\leq 11, 12, 13, 14, or \geq 15 years)	Leisure-time physical inactivity (< once/week)	No associations were found (no estimate provided)	Unadjusted
Beunen et al. 2004. (176)	27-year follow- up 166 of Flemish speaking adolescent Belgian boys Age: 40	Age at peak height velocity (no description)	Frequency of sports, other leisure-time activities and accelerometer counts of daily physical activity retro cohort	later age at peak height velocity correlated with more participation in sports (r=0.17, p<0.05) but not with leisure-time or counts indices.	None
Wichstrøm et al. 2013 (186)	3,251 Norwegian students. Age: 12 to 19 years at baseline (T1)	When you look at yourself now, do you think that you are more or less physically mature compared to others (of the same sex) of your age?" using seven response options ranging from much later to much earlier	LTPA hours/week reported three times over a 13-year period	LTPA T2 (in 1994) 0.10 (SE: 0.04) (p<0.01) LTPA T3 (in 1999) 0.03 (SE: 0.06) LTPA T4 (in 2006) 0.04 (SE: 0.04) Direction of associations unclear from study report however, suggest that earlier maturity associated with higher LTPA at T2 only	age, sex, BMI, athletic self- concept & others

6.2 Methods

6.2.1 Explanatory variables

Boys' pubertal status at age 15

Boys' pubertal stage was based on development of the secondary sexual characteristics which were assessed as part of a school-based medical exam when study members were aged 15. Boys were examined for the development of genitalia (categorised as infantile, early, advanced), voice breaking (no, starting, completely), pigmented pubic hair (no, sparse, profuse) and the presence of axillary hair (no, yes). For the purpose of analyses carried out in this chapter, a composite four level categorical measure of pubertal stage was created to group boys as (a) prepubescent (infantile genitalia or early adolescent genitalia but no pubic or axillary hair and voice not broken), (b) early puberty (early development of genitalia and some pubic or axillary hair or voice starting to break), (c) advanced puberty (advanced development of genitalia but at least one other indicator not fully mature) and (d) fully mature (advanced development of genitalia, and profuse pubic hair and axillary hair and voice broken). This categorisation is the same as that used in previous NSHD analyses (343).

Age at menarche

Mothers reported their daughters' age at menarche at the age 15 medical exam. All women including those who had not reached menarche by 15 years were then asked to recall this information at age 48 years. This retrospective information was used to replace missing values from those girls who had not reached menarche by age 15 and who recalled a consistent age at menarche (i.e. not reported reaching menarche before 15 years). For the purpose of these analyses, age at menarche was coded as a continuous representing per year later age at menarche and as a categorical variable, which was used to group girls as reaching menarche ≤ 11 years (early maturing girls), 12 years, 13 years and ≥ 14 years (late maturing girls). This categorisation is similar to that used in some of the other studies examining this association (173).

Sensitivity analysis measure: age at peak height velocity (APHV)

In addition to reported age at menarche in girls and clinically assessed pubertal status in boys, an alternative measure of pubertal timing; the age at peak height velocity (APHV), was recently derived for all NSHD participants and is used here as a sensitivity analyses to examine whether similar results are obtained. This measure was developed by Professor Tim Cole (UCL Institute of Child Health) using Super-Imposition by Translation And Rotation (SITAR) method (327, 345). Briefly, SITAR is a type of growth curve model that can be used to analyse height and weight data to derive measures of growth tempo (i.e. pubertal timing). A shape invariant growth model is estimated along with subject specific parameter indicating APHV. This method works best with frequently repeated data and as a result of this height and weight data from NSHD were augmented with height and weight data from the ALSPAC cohort which has more frequent measurements (346, 347). This measure is standardised to a mean of 0 and a standard deviation of 1 and later APHV indicates later puberty (327, 345).

6.2.2 Confounding variables

The associations between boys' pubertal stage at age 15 and age at menarche in girls and adulthood LTPA were initially examined unadjusted, then adjusted for birth weight, birth order and illness and with final models further adjusted for father's occupational class. Birth weight was selected as a confounder as it was hypothesised that low birth weight would be associated with earlier age at puberty. It was also reported in chapter 4 that birth weight was associated with adulthood LTPA in NSHD. Birth order was selected as a confounder as it was hypothesised that earlier birth order would be associated with earlier age at puberty and also with LTPA in adulthood. It was also hypothesised that serious illness in childhood would be associated with age at puberty (325), and also with less LTPA in adulthood. Finally, father's occupational class was chosen as a confounder because it was hypothesised, based on systematic review findings reported in chapter 3, that a lower childhood SEP would be associated with lower participation in LTPA across adulthood (151). It was also hypothesised that childhood SEP would be associated with age at puberty (325).

6.2.3 Examining associations with LTPA across adulthood

Details of initial exploratory analyses are described in chapter 2.3.1. Descriptive analyses (chi-squared tests for categorical variables and t-tests for continuous variables) were initially carried out to examine the distribution of age at puberty with the selected covariates and motor measures. The associations between age at puberty and LTPA at each age in adulthood were examined initially with separate binary and multinomial logistic regression models in study participants with complete LTPA data at each age. In these models, the inactive group of study participants, i.e. those reporting no participation in LTPA, were used as reference group. Binary models were used to estimate the ORs of participation in LTPA at each age (versus nonparticipation) by age at puberty. Multinomial models were used to estimate the RRRs of moderate and regular LTPA at each age (versus nonparticipation) by age at puberty.

Separate mixed-effects binary and multinomial logistic regression models were then used to examine associations between age at puberty and LTPA across adulthood (between ages 36-68 years) in study participants with at least one measure of LTPA. Details of these models including rationale for their use are in chapter 2.3.2. All standard and mixed-effects models with LTPA as outcome were adjusted in steps for (1) birth weight, birth order and serious illness, and (2) father's occupational class. In addition, at age 60-64, linear regression was used to examine the sex-adjusted difference in MVPA time and PAEE by age at puberty in the subsamples with this data.

6.3 Results

6.3.1 Age at puberty in relation to selected covariates

The distribution of boys' pubertal status and girls' age at menarche is shown in Table 6.2. Most girls (65%) reached menarche at ages 12-13 years and most boys (65%) showed signs of early or advanced puberty at age 15 (Table 6.2). The hypothesised associations between birth weight, birth order and age at puberty were more evident in girls and suggest that menarche \leq 11 years is more common in first born girls and less prevalent in the heaviest birth weight group (4.01-5.00 kg). There was some

suggestion that higher proportions of early maturing boys were from higher father's occupational class but less differences were evident in females.

Age at puberty was not associated with infant motor milestones or tapping speed at age 15. There was also little evidence that ability at school games at age 13 was associated with age at puberty (e.g. % above average ability for fully mature boys=22.0 and preadolescent boys=16.5, p for trend =0.6, and in girls, % below average ability for menarche \leq 11 years=15.6 and menarche \geq 14 years =11.8, p for trend =0.2).

 Table 6.2 Distribution of boys' pubertal status and girls' age at menarche.

	Males	Females
Pubertal status in boys at age 15 (n=1499)		
fully mature	370 (24.7)	
advanced puberty	457 (30.5)	
early puberty	512 (34.2)	
prepubescent	160 (10.7)	
Age at menarche in years (n=1409)		
≤11y		238 (16.9)
12y		402 (28.5)
13y		501 (35.6)
≥14y		268 (19.0)

Data shows numbers (%) in those with at least one measure of LTPA and data on covariates

6.3.2 Pubertal status at age 15 in boys and LTPA across adulthood

There was evidence that associations changed with age at assessment of LTPA (p=0.05 for boys' pubertal status by age interaction in binary mixed-effects models). Therefore, results from a binary mixed-effects model with an interaction term for pubertal status and age at LTPA are summarised in a plot showing the log-odds of LTPA at each age by pubertal status (Figure 6.1).

The ORs of any LTPA and RRRs of moderate and regular LTPA (versus none) at each age in adulthood in 636 men with all five measures of LTPA and data on pubertal status, birth weight, birth order, childhood illness and father's occupational class are presented in Appendix 4A and Appendix 4B respectively. When compared with those classed as fully mature at age 15, there was a suggestion that later maturing boys were less likely to participate in LTPA at ages 36 and 43 years but these associations were not as strong at older ages (Appendix 4A). For example, unadjusted ORs (95%CI) of LTPA for early puberty versus fully mature at ages 43 and 68 were 0.69 (0.45 to 1.05, overall p=0.3 across the four groups) and 0.83 (0.55 to 1.25, overall p=0.7). Adjustment for birth weight, birth order, childhood illness and father's occupational class generally had little influence on estimates (Appendix 4A). The findings were similar when examining the RRRs of moderate and regular LTPA (versus none) at each age but there was also a suggestion that later maturing boys were less likely to participate moderately (1-4 times per month) in LTPA (versus none) at age 68 (Appendix 4B). For example, fully-adjusted RRRs (95%CI) of moderate and regular LTPA at age 68 for early puberty versus fully mature were 0.51 (0.27 to 0.97) and 0.98 (0.61 to 1.59) respectively (overall p=0.3 across the four pubertal groups) (Appendix 4B).

Figure 6.1 shows a plot of the log-odds of LTPA (at least once per month versus no LTPA) at each age in adulthood by pubertal status at age 15 estimated from mixedeffects binary logistic regression analysis of 1499 men with at least one measure of LTPA and complete data on pubertal status, birth weight, birth order, childhood illness and father's occupational class. Overall, these plots show small differences between groups in the log-odds of LTPA across adulthood. Those who were fully mature tended to have the highest likelihood of participating in LTPA at ages 36 and 43 but this group showed the greatest decline in log-odds of LTPA with increasing age, reaching similar levels as the prepubescent group by age 53 (Figure 6.1). Conversely, those in early and advanced puberty had less pronounced declines in LTPA and differences between all groups reduced over time (Figure 6.1).

Examining the ORs of team sports (n=801), non-team sports LTPA (n=794) and leisure-time walking (n=800) at age 36 by men's pubertal status at age 15 showed little evidence of association with any of these outcomes though there was a suggestion of a stronger association with non-team sport LTPA than for team sports and leisure-time walking (Appendix 4C). Analyses at age 60-64 in men with data on LTPA and monitored PA at that age (n=686) showed that pubertal status at age 15 was not associated with LTPA or PAEE though there was a suggestion that those who were prepubescent at age 15 spent more time in MVPA when compared with those who were fully mature boys (fully-adjusted difference in MVPA time = 27.7% (95%CI: -3.2 to 59.3, overall p=0.1) (Appendix 4D). Lastly, sensitivity analyses showed that conclusions were similar when using APHV as an indicator of age at puberty. For example, unadjusted ORs of LTPA across adulthood per one standard

deviation increase in APHV in males were 0.998 (95%CI: 0.98 to 1.01, n=1789, p=0.7). Fully adjusted difference in MVPA time assessed by monitors at age 60-64 per one standard deviation increase in APHV in males was 1.1% (95%CI: -0.2 to 2.3, n=795, p=0.09).

Figure 6.1 Log-odds of LTPA across adulthood by pubertal status at age 15 in boys.

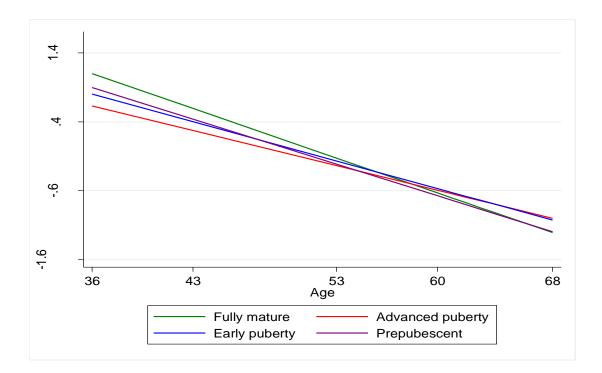


Figure 6.1 legend: Based on the estimated fixed effects (coefficients) $x\beta$ in the model (mean log-odds of leisure-time physical activity at each age by boys' pubertal status at age 15). Estimated from an unadjusted model and includes a pubertal status-by-age at LTPA interaction term (p=0.05).

6.3.3 Age at menarche and LTPA across adulthood

There was no evidence of an interaction between age at menarche and age at assessment of LTPA when examined in the binary mixed-effect models (p-value for categorical age at menarche by age interaction=0.9, p-value for continuous age at menarche by age at LTPA interaction=0.7). This suggests that associations did not differ by the age at assessment of LTPA and is consistent with the similar ORs of LTPA at each age in adulthood in study participants with non-missing LTPA data (Appendix 4E).

Table 6.3 presents the ORs of LTPA (at least once per month versus no LTPA) across adulthood by age at menarche estimated from mixed-effects binary logistic regression analysis of 1409 women with at least one measure of LTPA and complete data on menarche, birth weight, birth order, childhood illness and father's occupational class. When compared with early maturing girls (menarche ≤11 years), there was weak evidence that later maturing girls were slightly more likely to

participate in LTPA across adulthood. Results of the multinomial mixed-effects models in the same sample are in Table 6.4 and also showed weak evidence of associations in the same direction between age at menarche and LTPA across adulthood. Consistent with the mixed-effects models, the results from binary and multinomial models at each age also suggest that age at menarche was not associated with participation LTPA in adulthood (Appendices 4E and 4F).

Examining the ORs of team sports (n=859), non-team sports LTPA (n=855) and leisure-time walking (n=856) at age 36 by age at menarche showed that early maturing girls appeared less likely to participate in team sports at age 36 (p=0.03 for all models) but that age at menarche was not associated with non-team sport LTPA or with leisure-time walking (Appendix 4G). Age at menarche was not associated with time spent in MVPA or PAEE assessed by monitors at 60-64 years (n=686) (Appendix 4H). Lastly, sensitivity analyses showed that findings were similar when using APHV as an indicator of age at menarche, for example, unadjusted ORs of LTPA across adulthood per one standard deviation increase in APHV in females was 1.00 (95%CI: 0.99 to 1.01, n=1747, p=0.8). Fully-adjusted difference in MVPA time assessed by monitors at age 60-64 per one standard deviation increase in APHV in females was 0.0% (95%CI: -1.2 to 1.1, n=817, p=0.9).

Table 6.3 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-time physical activity (LTPA) between 36 and 68 years by age at menarche: mixed-effects binary logistic regression.

	OR (95% CI) of LTPA at least once per month between ages 36 and 68 years versus no LTPA				
	Model 1 Model 2 Model 3				
Age at menarche					
≤11 (n=238)	1.00 (reference)	1.00 (reference)	1.00 (reference)		
12 (n=402)	1.08 (0.80 to 1.47)	1.10 (0.82 to 1.49)	1.14 (0.85 to 1.53)		
13 (n=501)	1.15 (0.86 to 1.54)	1.17 (0.88 to 1.57)	1.21 (0.91 to 1.61)		
≥14 (n=268)	1.15 (0.83 to 1.59)	1.18 (0.85 to 1.63)	1.19 (0.87 to 1.64)		
test of association	p=0.8	p=0.7	p=0.6		

Analytic sample consists of those with at least one measure of LTPA (n=1409). Model 1: unadjusted. Model 2: adjusted for birth weight, birth order and childhood illness. Model 3: as for model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without age at menarche term. **Table 6.4** Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% CrI) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by age at menarche: mixed-effects multinomial logistic regression.

	RRR (95% CrI) of moderate LTPA (1-4 times per month) versus no LTPA			RRR (95% Crl) of regular LTPA (5 or more times per month) versus no LTPA		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Age at menarche (n=1409)						
≤11 (n=238)	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12 (n=402)	1.06	1.11	1.19	1.11	1.15	1.26
· · ·	(0.80 to 1.46)	(0.83 to 1.49)	(0.90 to 1.50)	(0.75 to 1.63)	(0.80 to 1.60)	(0.90 to 1.67)
13 (n=501)	1.10	1.15	1.21	1.19	1.24	1.34
, , , , , , , , , , , , , , , , , , ,	(0.82 to 1.43)	(0.85 to 1.51)	(0.93 to 1.61)	(0.85 to 1.59)	(0.82 to 1.84)	(1.02 to 1.82)
≥14 (n=268)	<u> </u>	1.26	`	<u> </u>	`	1.20
· · · ·	(0.87 to 1.62)	(0.89 to 1.75)	(0.98 to 1.74)	(0.74 to 1.64)	(0.76 to 1.73)	(0.82 to 1.76)
Bayesian DIC	`10099.59 <i>´</i>	`10088.38 ´	`10062.02 ´	-	-	-

Analytic sample consists of those with at least one measure of LTPA (n=1409). Model 1: unadjusted. Model 2: adjusted for birth weight, birth order and childhood illness. Model 3: as for model 2 plus adjustment for father's occupational class. Bayesian DIC statistics indicate fit for whole models.

6.4 Discussion

6.4.1 Summary of findings

The aim of this chapter was to examine how puberty status at age 15 years in boys and timing of menarche in girls relates to LTPA across adulthood. The results showed that pubertal timing was generally not associated with LTPA in adulthood. Overall, pubertal status in boys at age 15 was not associated with adulthood LTPA however, early maturing boys (fully mature at age 15) were somewhat more likely to participate in LTPA at ages 36 and 43 but experienced the greatest decline in participation with increasing age. In addition, early maturing boys also spent less time in MVPA assessed by activity monitors at age 60-64 than late maturing boys. Age at menarche was not associated with adulthood LTPA though there was a suggestion that early maturing girls (menarche ≤11 years) may be less likely to participate in LTPA across adulthood. In addition, menarche was also not associated with MVPA or PAEE assessed by monitors at age 60-64.

6.4.2 Comparison with other studies

Very few studies have examined how age at puberty relates to LTPA in adulthood. At least 3 other studies have examined associations between pubertal timing and LTPA in adult populations (173, 176, 186). Analyses from the next oldest British birth cohort (1958 NCDS) showed no associations between both pubertal stage in boys at age 16 (based on groups of axillary hair development) and menarche age and LTPA at ages 33, 40 and 50 years (173). This could be considered consistent with the overall conclusions of this chapter that pubertal timing was generally not associated with LTPA in adulthood in NSHD although, no estimate was provided in that study and thus direction of association is unclear (173).

The findings of this chapter are also consistent with null associations (adjusted for sex and other covariates) reported between self-rated maturity status reported by 12-19 years Swedish adolescents and subsequent LTPA assessed 3 times over 13 years follow-up (186). Interestingly, Wichstrøm et al. (186) found weak associations between earlier pubertal timing and more LTPA but only at the youngest (p<0.01) and not the two older follow-up ages to early adulthood (Table 6.1). This may be somewhat comparable to the findings in this chapter that association between age at

puberty in boys and LTPA in adulthood appear to weaken with age. However, as both males and females were included in their analyses it is unclear if associations vary by sex (186). Beunen and colleagues (176) found that later APHV (and thus later maturity) correlated with more participation in sports but not with leisure-time or accelerometer counts indices in a small sample of 40-year old Belgian men (Table 6.1). This contrasts with the findings in this chapter which suggest that early maturing boys appeared to be slightly more likely to be active in LTPA than their peers around this age (at ages 36 and 43). Beunen et al.'s (176) findings may be more aligned with associations between later maturity in boys and greater time spent in monitored MVPA at age 60-64 though these were different measures and not directly comparable. The finding in NSHD that early maturing boys were more active in LTPA at earlier adult ages is consistent with findings from adolescent studies which suggest that early maturing boys tend to be more athletic and active in sports than their later maturing peers (97, 332).

The suggestion that early maturing girls were less likely to participate in LTPA across adulthood is similar to the direction of associations reported by systematic reviews of studies in adolescent samples (97, 332) which suggest that girls who matured earlier had lower PA levels than later maturing girls. Tracking of LTPA could explain why these associations would be seen in adulthood.

6.4.3 Explanation of findings

Overall age at puberty was not an important correlate of LTPA across adulthood in either men or women. One explanation for the weak mostly null associations found is that maturity-related variations in the PA of adolescents reported in cross-sectional and mostly short-term follow-up studies (97, 332) may just represent a transitionary effect on PA behaviours which diminishes in importance once all peers have transitioned beyond puberty into adulthood. Consistent with this, it could be speculated that the less active early maturing female and late maturing male adolescent may substitute involvement in competitive sport with recreational forms of exercise such as cycling, jogging, walking, or aerobic exercise classes and as a result differences between maturity groups will not be substantial as they age into adulthood. However, as mentioned, there was more nuance to the findings and thus other explanations are also needed Studies show early maturing boys have greater self-esteem, confidence and popularity when compared with later-maturing boys, and are more active in adolescence (97, 332). Therefore, tracking of PA may explain the weak associations found between earlier maturity and LTPA at younger adult ages. Furthermore, the finding that early maturing boys appear to have the greatest decline in LTPA may be because some men who were very active as adolescents and young adults (e.g. through frequent involvement in team sports) give up participation in midlife, possibly because of health related conditions associated with early puberty. This may be supported by findings from UK Biobank showing that compared to the average group, both earlier and later puberty timing in women and men was associated with higher risks of various health outcomes that include cancers, cardio-metabolic, gastrointestinal, musculoskeletal, and neuro-cognitive categories' (341). The finding that early maturing boys also spent less time in MVPA assessed by monitors at age 60-64 requires further investigation including in other adult cohorts but may be due to residual confounding, for example by adult SEP and work status.

The direction of the weak associations between age at menarche and LTPA reported in this chapter (early maturing girls less likely to participate in LTPA) is consistent with the early maturation hypothesis (330). This hypothesis proposes that early maturing adolescents, due to interruptions to their normal course of behavioural development, are at risk of adopting unhealthy behaviour, and also by the findings from UK Biobank mentioned above (341). Early maturing girls might be embarrassed by the developmental changes in their bodies (97, 337). Early maturity in females is also associated with greater gains in weight post puberty that are predominantly associated with increased fat in adolescents (316). Consequently, early maturing girls have been shown to participate less in sports and exercises (97, 332), and the tracking of lower levels of PA by early maturing adolescent girls may be one explanation for why early maturing girls might participate less in LTPA as adults. However, the findings of this chapter suggest that overall, age at menarche is not associated with women's participation in LTPA across adulthood and thus maturity related differences in PA reported in adolescent girls may diminish once all girls transition to adulthood.

6.4.4 Methodological considerations

The methodological considerations relevant to all analyses carried out in this thesis are discussed in chapter 7 and only those which are specific to this chapter are discussed here.

There were relatively small numbers of participants in the extreme puberty groups which would have reduced statistical power. This meant it was necessary to combine all those girls who reached menarche at or before 11 years into a single group and those reaching menarche at or after 14 years into a single group. Although this categorisation resulted in groups based on pubertal timing which were relatively comparable to those for boys' pubertal status groups, it may be that it is the very extreme groups where differences in LTPA may be observed – and which could not be tested in NSHD. In addition, the fact that conclusions were similar when using the continuous measure of APHV lends further credibility to these findings.

6.4.5 Implications of findings

Due to the scarcity of research, more studies may be needed to examine the associations between age at puberty and LTPA in adulthood. However, the findings of this chapter coupled with those of the few other studies that have investigated this association suggest that this may not be the best avenue for research since they tend to be in agreement that pubertal timing may not be an important factor associated with LTPA in adulthood. These findings should be considered reassuring as they suggest a transitionary effect of pubertal timing on PA which diminishes following the transition to the mature adult state. However, the findings also suggest that late maturing boys may require some support to participate in LTPA at younger adult ages. On the other hand, it is also possible that early maturing boys may require more support at older adult ages to spend greater time in MVPA.

6.4.6 Conclusions

The aim of this chapter was to examine whether age at puberty, based on development of secondary sexual characteristics in boys at age 15 and menarche in girls relates to LTPA across adulthood. Overall, the findings showed there were only weak associations between age at puberty and adulthood LTPA in either sex.

However, early maturing boys appeared more likely to participate in LTPA at younger adult ages and experienced the greatest decline in LTPA with increasing age. There was also a suggestion that early maturing girls were less likely to participate in LTPA across adulthood. Overall, these findings are considered reassuring as they suggest that associations between pubertal timing and PA in adolescents described in previous studies may represent a transitionary effect which loses importance over time and once all peers have transitioned into adulthood. However certain groups may benefit from more support at different ages. The following chapter summarises the main findings of this thesis and their implications, along with overall methodological considerations and recommendations for future research.

Chapter 7: Discussion

This thesis has examined the associations between socioeconomic and developmental factors from early life and LTPA across adulthood using systematic reviews and analyses of empirical data in the NSHD. This chapter begins by summarising the main findings of each chapter. It then considers whether associations between childhood SEP and adult LTPA are influenced by the other developmental factors studied in this thesis before moving on to discussing the overall implications and importance of the findings presented. A discussion of the methodological considerations and recommendations for future work conclude the chapter.

7.1 Summary of main findings

This thesis used a systematic review and data from NSHD and found that socioeconomic and developmental factors from early life were associated with LTPA across adulthood.

Chapter 3 was a systematic review of published studies which had examined associations between SEP in childhood and LTPA in adulthood. Of 36 studies identified, 22 found that adults from less advantaged childhood socioeconomic backgrounds participated less in LTPA. These associations were found to be more prevalent in British compared with Nordic studies and in women compared with men, and did not appear to differ by type of childhood SEP indicator or age at assessment of LTPA. Chapter 3 also showed that adjustment for own adult SEP typically partly attenuated these associations and a subsequent review that developed directly from this chapter's findings showed that cumulative exposure to certain SEP in both childhood and adulthood rather than social mobility per se appears more important for adult LTPA (204).

Chapters 4, 5 and 6 then used data from NSHD to examine how the previously less frequently studied developmental factors of birth weight, motor development, ability and coordination and pubertal timing might relate to LTPA across adulthood, thus addressing the greater need for empirical data analyses of these associations.

Chapter 4 found that when compared with the low birth weight group (i.e. <2500g), study participants in all other heavier birth weight groups were more likely to

participate in LTPA between ages 36 and 68 years, including after adjustment for a range of covariates selected a priori.

Chapter 5 found that those with above average ability in school games and faster finger- and foot-tapping speed in adolescence had higher likelihood of participation in LTPA across adulthood between ages 36 and 68 years, including after adjustment for hypothesised covariates. This chapter showed that there was little difference in adulthood LTPA between those with below average and average ability in school games, particularly at older ages. Chapter 5 also found little evidence that age at reaching motor milestones was related to adult LTPA but found unexpected associations between walking independently before 11 months and lower likelihood of participation in LTPA across adulthood which were fully attenuated by adjustment for childhood SEP.

Chapter 6 found that age at puberty in boys and girls was generally not associated with LTPA across adulthood. This chapter's findings suggest that early maturing boys were slightly more likely to participate in LTPA at younger adult ages but had the greatest decline in participation across adulthood and that differences between all groups reduced with increasing age. Conversely this chapter's findings suggest that girls reaching menarche ≤11 years may be slightly less likely to participate in LTPA across adulthood.

In each chapter a novel contribution is made to our understanding of how different factors from early life may relate to LTPA across adulthood. Few previous studies have repeated LTPA assessments spanning 32 years of adulthood and none have examined whether associations vary by age at assessment of LTPA, which is important because it may help identify underlying mechanisms. What remains unclear is what influence developmental factors have on the association of childhood SEP with adult LTPA, which may be important because, as hypothesised in the conceptual framework of this thesis (Figure 1.1), these factors are likely to be interrelated, and developmental factors have been shown to be socioeconomically graded (221, 305, 348). Therefore, prior to discussing the implications of the findings of this thesis, the following subsection briefly examines the influence of adjustment for these developmental factors on the association of childhood SEP with adulthood LTPA.

7.1.1 Do developmental factors help explain early life socioeconomic differences in LTPA across adulthood

This thesis has shown that SEP, birth weight, ability at games and motor coordination in early life were associated with subsequent LTPA in adulthood. However, few studies have accounted for developmental factors when examining the associations between childhood SEP and adult LTPA because few have the relevant data to test this. This is despite the fact that these developmental factors may be important because they have been shown to be associated with health outcomes in adults (86, 230, 231, 294, 295). One of the only studies which has recently examined this was carried out in the next oldest British birth cohort, NCDS 1958 (173, 267). In their analyses, Pinto Pereira and colleagues showed that father's occupational class measured at birth was associated with LTPA in mid-adulthood even after adjustment for several early life factors including hand coordination problems and sports ability in addition to adult factors like own occupational class (173, 267).

Using the NSHD cohort, this section reports findings from analyses examining the associations between childhood SEP and adult LTPA which were initially adjusted for sex and subsequently mutually adjusted for birth weight, ability at games and tapping speed which were shown in chapters 4 and 5 to be associated with LTPA across adulthood. In addition, these second models also included adjustment for the prespecified covariates of birth order and serious childhood illness. As the age at reaching infant motor milestones and age at puberty were not clearly associated with LTPA overall they were not included as covariates. The analyses were also carried out for monitored MVPA and PAEE at age 60-64 as outcomes.

When compared with those with fathers in highest non-manual occupational groups I&II, those with fathers in manual occupational groups (IIIM, IV & V) were less likely to participate in LTPA across adulthood (Table 7.1) including at both moderate and regular levels of participation (Table 7.2). Adjustment for developmental factors of birth weight, ability at games and motor coordination (tapping speed) in adolescence had no influence on these associations (Table 7.1, Table 7.2). There was no interaction between SEP and age suggesting that this association did not vary by age (p<0.3 for father's occupational groups by age interaction) which is in agreement with estimates from models from each age (Appendices 5A and 5B). This is also consistent with the systematic review findings reported in chapter 3 which showed that age at assessment of LTPA was not a source of between-study heterogeneity.

Lastly, when compared with those in group I&II, those in manual father's occupational groups spent less time in MVPA assessed by monitors at age 60-64 even after adjustment for developmental factors, and there was weaker evidence relating childhood SEP to monitored PAEE at 60-64 (Appendix 5C).

Therefore, the developmental factors examined in this thesis seem to have very little influence on the association of childhood SEP with adult LTPA. However, the findings from chapters 4 and 5 suggest that there may be groups of individuals with low birth weight and/or poorer motor skills and coordination in early life who may benefit from additional support to participate in LTPA and maintain participation across life including into old age. Consequently, interventions targeting both sets of early life factors, i.e. socioeconomic circumstances, birth weight and motor performance are likely to be important for promoting LTPA across adulthood. The following section discusses the implications of the findings of this thesis.

Table 7.1 Odds ratios (OR) and 95% confidence intervals (95% CI) of leisure-timephysical activity (LTPA) between ages 36 and 68 years by father's occupational classage 4: mixed effects binary logistic regression.

	OR (95% CI) of LTPA at least once per month between ages 36 and 68 years versus no LTPA	
	Model 1	Model 2
Father's occupational class age 4		
professional/managerial/technical (n=559)	1.00 (reference)	1.00 (reference)
skilled non-manual (n=481)	1.01 (0.81 to 1.27)	1.02 (0.82 to 1.28)
skilled manual (n=794)	0.50 (0.41 to 0.61)	0.51 (0.42 to 0.63)
partly skilled or unskilled (n=735)	0.38 (0.31 to 0.47)	0.42 (0.34 to 0.51)
overall test of association	p<0.001	p<0.001

Analytic sample consists of those with at least one measure of LTPA (n=2722). Model 1: adjusted for sex. Model 2: as for model 1 plus adjustment for birth weight, birth order, childhood illness, ability at games and finger-tapping speed. Includes sex by age interaction. Tests of association based on likelihood ratio tests comparing models with and without father's occupational class term. **Table 7.2** Relative risk ratios (RRR) and 95% Bayesian credible intervals (95% CrI) of moderate and regular participation in leisure-time physical activity (LTPA) between ages 36 and 68 years by father's occupational class age 4: mixed effects multinomial logistic regression.

LTPA between ages 36-68	RRR (95% Crl) of moderate LTPA (1-4 times per month) versus no LTPA		RRR (95% Crl) of regular LTPA (5 or more times per month) versus no LTPA	
	Model 1	Model 2	Model 1	Model 2
<u>Father's occupational class age 4</u> professional/managerial/technical (n=559) skilled non-manual (n=481) skilled manual (n=794) partly skilled or unskilled (n=735) Bayesian DIC	1.00 (reference) 1.07 (0.89 to 1.34) 0.53 (0.43 to 0.63) 0.45 (0.37 to 0.54) 18930.56	1.00 (reference) 1.07 (0.92 to 1.26) 0.63 (0.54 to 0.73) 0.59 (0.50 to 0.68) 17946.57	1.00 (reference) 0.99 (0.77 to 1.28) 0.45 (0.35 to 0.56) 0.31 (0.24 to 0.39) -	1.00 (reference) 1.03 (0.74 to 1.33) 0.49 (0.37 to 0.61) 0.35 (0.25 to 0.45)

Analytic sample consists of those with at least one measure of LTPA (n=2722). Model 1: adjusted for sex. Model 2: as for model 1 plus adjustment for birth weight, birth order, childhood illness, ability at games and finger-tapping speed. Includes sex by age interaction. Bayesian DIC statistics indicate fit for whole models.

7.2 Implications of findings

As discussed in Chapter 1, regular LTPA provides many health benefits that include reduced rates of early death from chronic disease. In contrast, physical inactivity is a major contributor to morbidity and premature mortality (13, 20, 28, 116). As such, the identification of factors associated with participation in LTPA is important and may help inform the design of public health interventions which aim to promote participation in LTPA. Relatively little is known about how factors from early life may influence later PA and this thesis makes an important contribution by furthering our understanding of the associations between socioeconomic and developmental factors and LTPA across adulthood.

The findings of the systematic review in chapter 3 suggest that interventions to improve socioeconomic circumstances in early life may benefit adult LTPA. Moreover, a subsequent systematic review of thirteen published studies which complements chapter 3's findings showed that cumulative exposure to lower SEP in both childhood and adulthood was associated with lower LTPA among adults from different countries. Thus a potential outcome of policies and interventions which aim to minimise exposure to socioeconomic adversity at any point in life may be increased LTPA among adults. However, there is yet little evidence of the effectiveness of such policies and initiatives (349) although some successful interventions have been reported (349, 350). For example, an evaluation of The District Approach, an area-based intervention which aims to ease problems of employment, education, housing and the physical environment, safety, and social integration in 40 of the most deprived districts in the Netherlands showed it led to increased leisure walking (350).

Chapters 4's findings suggest that it is important to recognise that those born with low birth weight may require more support than others if they are to achieve sufficient LTPA across life to realise its health benefits. The increased prevalence and longterm survival of those with low birth weight in the last several decades means that there are now increasing numbers of adults who were born with low birth weight and thus there may be a growing proportion of the population who are unlikely to be participating in LTPA. Thus, the findings of this chapter could have important healthrelated implications for current as well as future generations. As discussed in chapter 4, exercise is recognised as important for reducing the adverse cardio metabolic consequences of in utero growth restriction (260) and is also considered safe for the majority of those born preterm (256). Designing appropriate interventions to support LTPA across life may require a better understanding of how other related processes like postnatal growth, motor capability and body composition influence PA in those with low birth weight.

Findings from chapter 5 suggest that motor skills including speed and coordination in adolescence, but not age at reaching infant motor milestones, are important factors associated with participation in LTPA across adulthood. As discussed in chapter 5, this suggests that interventions targeting motor performance of older children and adolescents may have long-term benefits for LTPA in adulthood. Both motor skill development and increasing PA should simultaneously be targeted in PA interventions (281). Schools with greater opportunities for sports participation have been shown to help adolescents maintain participation into adulthood (311). Schools should adopt or reform PA policies to promote PA among students (351) and these should include providing support to teachers to effectively promote PA (352). On the other hand, it may be that improving motor skills in early life is more relevant for developing superior athletic performance rather than population level LTPA participation (353). Finally, findings from chapter 6 suggest that age at puberty may not be such an important factor. As discussed, these findings should be considered reassuring as they suggest that associations reported between pubertal timing and PA in adolescence (97, 332) may diminish in adulthood.

Taken together, the overarching implications of this thesis are that those people with low birth weight, a less advantaged SEP and poorer motor performance in early life may require more support than others in order to participate in LTPA across adult life so as to accrue its health benefits. Therefore, interventions in early life could have benefits for lifelong LTPA. Further, as described in chapter 2, very few study participants took up LTPA at later ages if they were previously inactive at age 36 (Table 2.5), which is consistent with findings from NCDS 1958 (267). These findings of the tracking of LTPA emphasise that interventions targeted at adult populations should aim to promote earlier uptake and continued participation in LTPA. It is therefore also important that policymakers and professionals understand the challenges associated with behaviour change at the individual level as well as the structural aspects behind people's participation in LTPA (81).

As shown in the previous section of this chapter (7.1.1), of the early life factors examined in this thesis, childhood SEP appears to be the most consistent correlate

of adulthood LTPA. Therefore, where there are limited resources, these should be funnelled towards interventions targeting early life socioeconomic circumstances rather than addressing developmental factors like birth weight and motor skills. In addition, targeting socioeconomic factors might also help with other developmental factors. For example, incentivising sports club membership for young children and their families could potentially lead to population level improvements in motor skills which might in turn promote lifelong participation in LTPA.

Furthermore, the findings of this thesis should be taken in context of the other factors which influence LTPA (Table 1.1) some of which could potentially be more important influences on participation at different life stages. For example, participation in LTPA is a volitional behaviour and thus its long-lasting character and cognitive, emotional, and action components need to be considered (81). In addition, age, sex, health status, self-efficacy, and motivation have been identified as individual level correlates and the built environment such as urban planning, transportation systems, and parks and trail that enables or disables participation in LTPA have also been identified as distal-level correlates within the ecological model (68) (Table 1.1). Moreover, factors that might influence LTPA are likely to vary between children and adults. For example, physical competence, parental support and support from significant others, e.g., peers are important for children's LTPA (354). In adulthood, when PA is seen as sociocultural by nature, the need for repeated social reinforcement especially in life transitions such as a change in employment and family structure, is emphasised (355).

In addition, in modern developed societies such as the UK, people who choose to incorporate LTPA in their daily routines must schedule and plan their LTPA by replacing other competing behaviours (e.g. sedentary behaviour). Therefore, making a choice of engaging or not engaging in LTPA can be situation or condition dependent. For example, though an individual wants to regularly engage in LTPA, if they do not have time due to work or other responsibilities, one might not be able to engage in LTPA regardless of say their birth weight or motor skills in the early years. Therefore, considering these early life factors within the context of the wider environment and infrastructure that reinforces LTPA is likely to become increasingly popular for improving population levels of LTPA.

7.3 Methodological considerations

The specific methodological considerations of the systematic review carried out in chapter 3 and the NSHD analyses conducted in Chapters 4–6 are discussed within each chapter. To avoid repetition, only the shared methodological considerations of chapters 4-6 will be described below.

As was illustrated in figure 2.1, there was some loss to follow-up in NSHD, as expected in long-running studies. As described in table 2.2, this led to only slight differences in characteristics between those included and those with missing data. A survival selection bias is also possible and may have biased results towards the null. For example, if those healthiest and most physically active from the low birth weight group survive to an older age this would underestimate true associations.

However, as described in chapter 2, the mixed-effects models maximise sample size and improve precision of estimates of association as all individuals with at least one measure of LTPA are included in the analyses. One important assumption of these mixed-effects models is that the LTPA data is missing at random (131) Data are said to be missing at random if there is no systematic difference between the observed and missing values after accounting for differences in observed data i.e. the probability that an LTPA value is missing may depend on observed values in the data but not additionally on the missing value itself (356, 357). This assumption is difficult to check in practice however, complete case analyses where just those with all LTPA measures were included showed similar associations to the mixed-effects models (see Appendices 2A to 5B). These models also allowed an investigation of whether associations with LTPA change with age thus shedding valuable light on underlying mechanisms. In addition, another important strength of the analyses carried out in this thesis included the prospective cohort design which reduces recall bias in collection of early life exposures and hypothesised covariates.

The associations observed in chapters 4 and 5 between birth weight, adolescent motor performance and adult LTPA may be generalisable to more recently born cohorts since associations between birth weight and motor performance in early life and adult LTPA have been seen in the same direction in younger cohorts (Tables 4.2 and 5.1). The finding of these associations in more contemporary cohorts with different confounding structures to those in NSHD supports the generalisability of the findings of this thesis (358). However, it remains to be seen whether associations will track into older age in these cohorts in the same way as in NSHD. This is because in older cohorts like NSHD, the contextual circumstances surrounding early life

exposures may not be directly relevant to those experienced by contemporary cohorts (100, 358), for example, the unique experience of food rationing during the NSHD cohort's early years (101).

The LTPA outcomes used in this thesis were self-reported and thus could be subject to recall bias and misclassification error. However, as discussed in chapter 1 (section 1.3), self-reports allow collection of contextual circumstances surrounding PA making them suitable for capturing activity types and domains like LTPA (50, 51). Moreover, as discussed in chapter 2, when self-reports of LTPA at age 60-64 were compared with data from activity monitors, both methods ranked participants by levels of PA similarly (55). If there was differential reporting of LTPA by different groups of the early life exposures of interest this could bias the findings. For example in relation to the systematic review findings from chapter 3, obesity tends to be more prevalent in lower SEP groups (359-361) and obese individuals have previously been found to be more likely to overestimate their levels of PA and energy expenditure (362) therefore, differential reporting of LTPA by different groups of the other early life exposures examined (i.e., birth weight, motor milestones, games ability, tapping speed and age at puberty) may also be possible.

Furthermore, different questions were used at different ages to derive LTPA across adulthood. These were based on identical questions at ages 53, 60-64 and 68 but a different set of questions were asked at ages 36 and 43 (Table 2.3). This may inevitably lead to some misclassification of LTPA across adulthood. In addition, the LTPA outcomes derived were discrete rather than continuous measures such that dichotomous and categorical outcomes were derived. Although this meant that comparable data were available to ascertain LTPA across adulthood, it may be that some information is lost through the use of these discrete outcomes at the expense of comparability over time. However, the approach used here potentially avoids added bias and measurement error in classifying participants based on self-reported intensities and/or duration of participation (51). Moreover, given that current UK PA guidelines for adults encourage at least 150 minutes of moderate intensity PA (or 75 minutes of vigorous intensity) and 2 sessions of strength training per week (27) it could be argued that those participating in LTPA \geq 5 times per month cannot be considered as being regularly active, and thus a higher cut-off point may have been more appropriate (though this would have led to a small group of participants and thus lower statistical power).

The analyses carried out in this thesis were limited by ages at which LTPA was collected from NSHD participants, i.e. at ages 36, 43, 53, 60-64 and 68 years. While these data provide information on LTPA over a long period in adulthood (32 years), information on LTPA was not available before 36 years, after 68 years, or between the different measurement ages. Having more repeated assessments would have allowed for more detailed analyses. For example, the fact that there were no data on LTPA at younger adult ages (before 36 years) including data covering the transition from adolescence to adulthood means that it was unclear how age at puberty may have related to LTPA during the study participants' teenage years and their 20s. The associations between age at puberty of both boys and girls and later LTPA may have been stronger at these younger ages although, the findings of chapter 5 suggest that even if associations were present at these younger ages, pubertal timing may not be particularly important for LTPA later in life. In addition, it is evident that PA in childhood tracks to adolescence as well as adulthood and the stability of PA is moderate to high over the life course from youth to adulthood (91). Therefore, examining motor skills in adolescence and LTPA in adulthood without adding PA in adolescence to the equation as a mediator or at least as a confounder is a limitation.

In additional analyses carried out in this thesis examining team sports and non-team sports LTPA and leisure-time walking at age 36, birth weight and tapping speed were found to be associated with participation of all types of LTPA however, ability at school games was more strongly related to team sports, pubertal status more closely related to non-team sports in men, and early menarche only related to team sports. As described in the implications (section 7.2), this highlighted further implications regarding the associations examined between early life factors and overall LTPA. Investigating associations for each type of team and non-team sports requiring different competencies (e.g. swimming, aerobics, football) could have provided further insight and led to additional implications however, the small numbers of participants reporting each different activity would have made inference from such analyses challenging.

Additional analyses were carried out in this thesis using other PA outcome measures of MVPA and PAEE assessed by monitors at age 60-64 in a comparable sample to those with data on LTPA at age 60-64 however, results using the two different sets of measures (self-reported LTPA and monitored MVPA and PAEE) were not always consistent. For example, while associations in the same direction were observed for self-reported LTPA and monitored PA in relation to most early life factors (birth weight, ability at games, tapping speed and father's occupational class), there was little evidence against the null hypothesis of no association when examining monitored MVPA and even less so for PAEE. The differences in results between LTPA and monitored PA, which as discussed in chapter 1 do not provide directly comparable measures of PA (51), do provide useful insight into the associations of early life factors with later PA.

These monitored PA data cover daily PA and are not able to provide contexts related to the PA performed i.e. type and duration. As such these measures incorporate all incidental activities, some or all of which might be considered non-voluntary and may include daily activities which were not hypothesised to be related to early life factors. For example, participants may have spent some of their time in MVPA doing housework LTPA is volitional in that individuals choose to take part in these activities such as sports and exercise. Further, the overall lack of association with these monitored PA outcomes suggests that higher levels of energy expenditure do not explain the associations between the early life factors examined and adult LTPA. In addition, the guestions asked about LTPA referred to much longer periods of time (to the last 4 weeks/month and per month) which can capture more information about PA than the 3–5 days over which monitored PA was assessed. Also worth considering is that since LTPA was assessed by self-report, an alternative but less likely explanation (given the explanation above) is that reporting bias explains the associations observed. Furthermore, there is also likely to be some selection bias such that healthier participants are more likely to have been eligible to be invited to wear and agree to wear the activity monitors (106) which might lead to an underestimation of association with early life factors.

Lastly, while examining whether associations change with age at LTPA helps identify if processes related to ageing might contribute to findings, a better explanation of findings could have been gained through formal analysis of underlying pathways (363), including those that operate across life (see section 7.4 Recommendations for future research).

7.4 Recommendations for future research

This thesis is one of only a few studies to have investigated the relationship between factors from early life and later LTPA therefore, additional research is needed to further understand the mechanisms behind the associations between socioeconomic and developmental factors and LTPA across adulthood. This section provides a list of recommendations for future research to build on the work carried out in this thesis.

As discussed in the previous section, the unique circumstances surrounding the early life of the NSHD cohort means it will be important to replicate findings from NSHD in other cohorts including those that have been born more recently. Cross-cohort research should be pursued where possible since replication of findings in cohorts with different confounding structures would add credence to observed associations (358). Therefore, it would be useful to combine PA data from different cohorts and harmonise methods of analyses and this should include formal testing of cohort differences whilst also accounting for methodological differences, as was previously done for measures of physical capability from 8 UK cohorts (313). Moreover, integrating findings from cross-cohort and cross-setting comparisons and different statistical approaches with different sources of bias could help lead to better inference regarding causality in the observed associations (364, 365).

It is worth noting that the lack of standard instruments for the assessment of PA as exemplified by the studies included in the systematic review in chapter 3 means that combining results from different cohorts is likely to be more challenging for PA. Future studies should therefore be aware of the role different measures may have in their analyses. In particular, studies should consider the role of precision, context and possible social patterning of specific PA types. Further, studies with larger sample sizes than NSHD might also be better placed to examine associations by types of LTPA performed, e.g. whether associations vary between light LTPA such as leisurely walking and more demanding LTPA such as high impact aerobics.

Since the analyses carried out in this thesis were only adjusted for hypothesised early life confounders, future studies examining associations between early life factors and later LTPA should also consider adult as well as early life factors in order to more fully understand the pathways operating between exposures in early life and later LTPA (82, 90, 366). These studies could also make use of advances in mediation analysis to fully unravel the underlying pathways (363). Further, as was carried out in this thesis, any future studies which possess repeat measures of LTPA could also investigate whether associations with LTPA change with age in order to better identify whether processes related to ageing explain their findings.

Studies with more detailed assessments collected in early life could also address some of the limitations of the analyses carried out in this thesis and help to better understand how developmental factors relate to later LTPA. For example, in the Cork BASELINE Birth Cohort Study, data are available on body size during gestation, postnatal motor developments and on PA data at age 5 years (357) however, few long running studies with such data are yet available. As younger detailed cohorts mature, their data may help improve our understanding of how factors like infant motor development and age at puberty might relate to later LTPA. Studies with detailed measures of motor performance can also help us to elucidate the mechanisms linking faster tapping speed in adolescence with greater adult LTPA, and whether this reflects the co-evolution of motor performance and LTPA across life.

Genetic epidemiological studies investigating causal relationships could also be used to increase understanding of how developmental factors might influence LTPA. For example, studies with genetic markers for low birth weight have replicated observational associations between low birth weight and adult chronic disease risk (367-369) and these genetic markers could also be used as instrumental variables relating birth weight to later LTPA (370). Understanding how associations between SEP and LTPA vary between men and women, by place and the role of mediating factors like education could also provide insights into underlying pathways. For example, a study found father's education to be more important than own education in explaining differences in self-rated health in Eastern when compared with Western Europe (371).

Furthermore, rapid advancements in the development of PA monitors and in the analysis of their outputs means that in the near future it may be possible to identify context surrounding PA and obtain objective measures of LTPA that are less susceptible to reporting bias (372-374). These could offer an exciting additional avenue to investigate the importance of early life factors to adults' participation in LTPA.

Thesis publications

Elhakeem A, Cooper R, Bann D, Kuh D, Hardy R. Birth weight, school sports ability and adulthood leisure-time physical activity. *Med Sci Sports Exerc* 2017;49(1):64-70.

Elhakeem A, Hardy R, Bann D, Caleyachetty R, Cosco TD, Hayhoe RPG, Muthuri SG, Wilson R, Cooper R. Intergenerational social mobility and leisure-time physical activity in adulthood: a systematic review. *J Epidemiol Community Health* 2016; doi:10.1136/jech-2016-208052.

Elhakeem A, Cooper R, Bann D, Hardy R. Childhood socioeconomic position and adult leisure-time physical activity: a systematic review. *Int J Behav Nutr Phys Act* 2015;12:92.

Elhakeem A, Cooper R, Bann D, Hardy R. Childhood socioeconomic position and adult leisure-time physical activity: a systematic review protocol. *Syst Rev* 2014;3:141.

Elhakeem A, Hardy R, Bann D, Kuh D, Cooper R. Motor performance in early life and leisure-time physical activity up to age 68 years. *Submitted*.

Elhakeem A, Cooper R, Bann D, Kuh D, Hardy R. Is age at puberty associated with leisure-time physical activity across adulthood? Findings from a British birth cohort. *Submitted*.

Appendices

Appendix 1A Joint search (15/11/13) using OvidSP for systematic review of the association between childhood SEP and adult LTPA: Medline (from 1946), Embase (from 1974), PsycInfo (from 1806).

	Search Terms	Results
1.	(physical* activ*)	149602
2.	(leisure adj3 time)	13294
3.	(sport*)	117207
4.	(exercise)	422666
5.	(walk*)	170868
6.	(recreational)	29163
7.	(father* adj3 (occupation* or education*))	3813
8.	(mother* adj3 (occupation* or education*))	8246
9.	(parent* adj3 (occupation* or education*))	24841
10.	(father* adj3 (income or manual))	556
11.	(mother* adj3 (income or manual))	3045
12.	(parent* adj3 (income or manual))	3882
13.	(father* adj3 (social class or social status))	393
14.	(mother* adj3 (social class or social status))	378
15.	(parent* adj3 (social class or social status))	1143
16.	(child* adj3 (social class or social status))	1438
17.	(early-life adj3 (social class or social status))	23
18.	(adolescen* adj3 (social class or social status))	267
19.	(father* adj3 (socioeconomic or socio-economic))	254
20.	(mother* adj3 (socioeconomic or socio-economic))	1194
21.	(parent* adj3 (socioeconomic or socio-economic))	3593
22.	(child* adj3 (socioeconomic or socio-economic))	6336
23.	(adolescen* adj3 (socioeconomic or socio-economic))	1057
24.	(early adj3 (socioeconomic or socio-economic))	773
25.	(early-life adj3 (socioeconomic or socio-economic))	254
26.	(child* adj3 (deprivation or poverty))	3740
27.	(early-life adj3 (deprivation or poverty))	152
28.	(adolescen* adj3 (deprivation or poverty))	394
29.	(child* adj3 overcrowding)	63
30.	(adult*)	2082336
31.	(midlife or mid-life)	11851
32.	(old*)	2597207
33.	(later-life)	18689
34.	1 OR 2 OR 3 OR 4 OR 5 OR 6	792966
35.	7 OR 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14 OR 15	56533
	OR 16 OR 17 OR 18 OR 19 OR 20 OR 21 OR 22 OR 23	
	OR 24 OR 25 OR 26 OR 27 OR 28 OR 29	
36.	30 OR 31 OR 32 OR 33	4339427
37.	34 AND 35 AND 36	1197
38.	Limit 37 to humans	1163
39.	Remove duplicates from 38	620

Appendix 1B Joint search (15/11/13) using EBSCO for systematic review on the association between childhood SEP and adult LTPA: CINAHL (from 1937), SPORTDiscus (from 1985).

	earch Terms	Results
	physical* activ*)	76,786
2. (le	eisure N3 time)	4,348
	sport*)	846,131
4. (e	exercise)	270,574
5. (v	valk*)	51,286
	ecreational)	15,262
7. (f	ather* N3 (occupation* or education*))	586
	nother* N3 (occupation* or education*))	2,065
9. (p	parent* N3 (occupation* or education*))	9,061
10. (f	ather* N3 (income or manual))	94
11. (n	nother* N3 (income or manual))	623
12. (p	parent* N3 (income or manual))	650
13. (f	ather* N3 (social class or social status))	49
14. (n	nother* N3 (social class or social status))	55
15. (p	parent* N3 (social class or social status))	152
16. (c	child* N3 (social class or social status))	275
17. (e	early-life N3 (social class or social status))	4
18. (a	adolescen* N3 (social class or social status))	81
19. (f	ather* N3 (socioeconomic or socio-economic))	40
20. (n	nother* N3 (socioeconomic or socio-economic))	180
21. (p	parent* N3 (socioeconomic or socio-economic))	465
22. (c	child* N3 (socioeconomic or socio-economic))	1,037
23. (a	adolescen* N3 (socioeconomic or socio-economic))	308
24. (e	early N3 (socioeconomic or socio-economic))	137
25. (e	early-life N3 (socioeconomic or socio-economic))	48
	child* N3 (deprivation or poverty))	951
27. (e	early-life N3 (deprivation or poverty))	28
28. (a	adolescen* N3 (deprivation or poverty))	227
	child* N3 overcrowding)	12
	adult*)	1,162,887
	nidlife or mid-life)	4,812
	bld*)	465,808
	ater-life)	4,484
	OR 2 OR 3 OR 4 OR 5 OR 6	1,086,633
	OR 8 OR 9 OR 10 OR 11 OR 12 OR 13 OR 14 OR 15 OR	15,315
	6 OR 17 OR 18 OR 19 OR 20 OR 21 OR 22 OR 23 OR 24	
	R 25 OR 26 OR 27 OR 28 OR 29	
	0 OR 31 OR 32 OR 33	1,418,632
	4 AND 35 AND 36	554
38. D	uplicates removed from 37	525

Appendix 1C Study selection form

A. Reference details		
A1. Reference Manager ID number (Ref ID)		
A2. 1 st Author		
A3. Title of paper		
A4. Journal, volume, year of publication		
A8. Assessor's name, date of assessment		
B. Eligibility		
B1. Study included in systematic review?	Yes	No
C. Reason(s) for exclusion (if excluded)		
C1. Outcome not in adults (≥ 25 yrs.)	Yes	No
C2. Ineligible exposure	Yes	No
C3. Ineligible outcome	Yes	No
C4. Review article	Yes	No
C5. Duplicate (Insert Ref ID of other study)	Yes	No
	Ref ID:	
C6. Other		

Appendix 1D Data extraction form

A1. Ref ID, 1 st author, title	,			
publication year,				
A2. Assessor's name and date of	Ĕ			
assessment				
B. Study details				
B1. Name of study/cohort				01
B2. Design	Prospective	Retrospective		Other
B2A. If other:	cohort	cohort	t control	
B2A. If other: B3. Country, setting				
C. Childhood socioeconomic positio	24			
C1. Parental occupation	Yes			No
C1. Parental occupation C2. Parental education	Yes			No
C3. Other measures (list):	105			NO
C4. How ascertained	Prospectively		P	etrospectively
C5. Age recorded	Trospectively	1	K	cuospectively
C6. Age referred to				
D. Physical activity (PA) outcomes				
D1. Parameters measured	Frequency	Туре	Duration	Intensity
D2. Type of leisure-time PA	Sport/	Gardening/	Total	Other
(LTPA) measured	exercise	DIY	LTPA	Juici
D2A. If other please describe	SACIOISC		LIIII	I I
D3. How ascertained	Self-reported		Obie	ective methods
D4. Age ascertained	ported	1	56/6	
D5. Variable details	Binary	Ordinal	Continuous	Other
D5A. If other:				
E. Available participant numbers				
E1. Baseline	Yes	No		If yes, number
E2. Excluded	Yes	No		If yes, number
E3. Refused	Yes	No		If yes, number
E4. Lost to follow-up	Yes	No		If yes, number
E5. Other losses	Yes	No		If yes, number
E6. Included in analysis	Yes	No		If yes, number
E7. All accounted for	Yes	No		
F. Analysis				
F1. How results analysed	Descriptive/	Logistic	Linear	Other
	Trend	regression	regression	
F1A. If other:				
F2. Included in analysis	Men and	Men		Women
	women	only		only
F3. Only significant results	S Yes		·	No
presented				
G. Summary of results				
G1. Prevalence/Mean difference	Yes			No
G2. Odds/Risk ratios	Yes			No
G3. Regression coefficients	Yes			No
G4. Confidence intervals (CIs)/ P-	- Yes			No
value/standard errors (SE)				
G5. Other	Yes			No
G5A. If other				
H. References for screening				
H1. Reference numbers				
I. Effect estimates	— 0.00 :	1	0.501 67 77	
Association Number	Type of effect estimate a		95% CI; SE;	Confounders
tested analysed	category comparison/valu	e of estimate	p-value	included in
1.	unit change		+	analysis

Appendix 1E Quality assessment form based on amended version of the Newcastle-Ottawa Scale.

Note: A study can be awarded a maximum of two stars for each numbered item – (except number 5). A. Selection 1) Representativeness of the exposed cohort a) Truly representative of the source population. *** *** b) Somewhat representative of the source population. ***** c) Selected group of users e.g. nurses, volunteers. d) No description of the derivation of the cohort. 2) Ascertainment of childhood socioeconomic position (SEP) a) Prospectively from parents/participants when aged ≤18 years. ★★ b) Retrospectively collected with attempts to reduce recall bias (e.g. lifegrid and structured interview techniques). *c) Retrospectively collected without attempts to reduce recall bias. d) No description. **B.** Comparability 3) <u>Comparability of cohorts on the basis of the design/analysis</u>
a) Study controls for adult SEP. **★** b) Study controls for any additional relevant factors (e.g. age, sex). * c) Only unadjusted model presented. C. Outcome 4) <u>Assessment of physical activity</u>
a) Objective methods (heart-rate monitoring/accelerometer). **★ ★** b) Self-reported using validated questionnaire/diary/interview. * c) Self-report. d) No description. 5) Adequacy of cohort follow-up a) Complete follow up - all subjects accounted for. **★** b) Subjects lost to follow up unlikely to introduce bias (≥75% follow-up or description provided of those lost). *c) <75% follow-up and no description of those lost. d) No statement.

Appendix 2A Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by birth weight group: standard binary logistic regression (n=1581)

	LTPA at least once per month versus no LTPA Model 1 Model 2 Model 3				
OR (95% CI)	OR (95% CI)	OR (95% CI)			
	01((35% 01)	01((35 % 01)			
1 00 (reference)	1 00 (reference)	1.00 (reference)			
		1.67 (0.97 to 2.87)			
		1.93 (1.16 to 3.23)			
	. ,	,			
		1.55 (0.93 to 2.61)			
		1.72 (0.95 to 3.11)			
p=0.08	p=0.07	p=0.1			
1.00 (reference)	1.00 (reference)	1.00 (reference)			
1.81 (1.12 to 2.94)	1.87 (1.15 to 3.04)	1.73 (1.06 to 2.84)			
p=0.02	p=0.01	p=0.03			
1.00 (reference)	1.00 (reference)	1.00 (reference)			
, , , , , , , , , , , , , , , , , , ,		1.29 (.75 to 2.25)			
		1.93 (1.15 to 3.24)			
	. ,	1.74 (1.03 to 2.94)			
		1.93 (1.06 to 3.50)			
		p=0.02			
P 0.0-	P 0.000	P 0.0-			
1 00 (reference)	1 00 (reference)	1.00 (reference)			
		1.72 (1.04 to 2.85)			
		p=0.03			
ρ=0.05	p=0.02	μ=0.05			
(1,0,0)	1.00 (reference)	1.00 (reference)			
		1.00 (reference)			
		1.42 (0.83 to 2.43)			
	. ,	1.80 (1.08 to 2.99)			
		1.64 (0.98 to 2.74)			
		1.56 (0.87 to 2.80)			
p=0.08	p=0.06	p=0.2			
1.00 (reference)	1.00 (reference)	1.00 (reference)			
1.66 (1.02 to 2.69)	1.79 (1.10 to 2.92)	1.65 (1.01 to 2.69)			
p=0.04	p=0.02	p=0.05			
1 00 (reference)	1 00 (reference)	1.00 (reference)			
	, , , , , , , , , , , , , , , , , , ,	2.10 (1.13 to 3.89)			
		2.10 (1.15 to 3.78) 2.10 (1.16 to 3.78)			
		2.16 (1.19 to 3.91)			
		1.89 (0.97 to 3.67)			
	1.81 (1.12 to 2.94) p=0.02 1.00 (reference) 1.30 (0.75 to 2.23) 1.93 (1.16 to 3.21) 1.73 (1.04 to 2.89) 1.67 (0.94 to 2.97) p=0.02 1.00 (reference) 1.71 (1.04 to 2.80) p=0.03 1.00 (reference) 1.43 (0.84 to 2.43) 1.84 (1.12 to 3.03) 1.68 (1.01 to 2.78) 1.41 (0.80 to 2.49) p=0.08 1.00 (reference) 1.66 (1.02 to 2.69)	1.70 (0.997 to 2.92) $1.73 (1.01 to 2.96)$ $2.03 (1.23 to 3.35)$ $2.07 (1.25 to 3.43)$ $1.67 (1.01 to 2.77)$ $1.73 (1.04 to 2.88)$ $1.72 (0.97 to 3.07)$ $1.86 (1.03 to 3.34)$ $p=0.08$ $p=0.07$ $1.00 (reference)$ $1.00 (reference)$ $1.81 (1.12 to 2.94)$ $1.87 (1.15 to 3.04)$ $p=0.02$ $1.35 (.78 to 2.32)$ $1.30 (0.75 to 2.23)$ $1.35 (.78 to 2.32)$ $1.93 (1.16 to 3.21)$ $2.05 (1.23 to 3.43)$ $1.73 (1.04 to 2.89)$ $1.92 (1.15 to 3.23)$ $1.67 (0.94 to 2.97)$ $2.06 (1.15 to 3.71)$ $p=0.02$ $p=0.006$ $1.00 (reference)$ $1.00 (reference)$ $1.71 (1.04 to 2.80)$ $p=0.02$ $p=0.03$ $p=0.02$ $1.00 (reference)$ $1.48 (0.87 to 2.52)$ $1.84 (1.12 to 3.03)$ $1.94 (1.18 to 3.21)$ $1.68 (1.01 to 2.78)$ $1.84 (1.11 to 3.06)$ $1.41 (0.80 to 2.49)$ $1.70 (0.95 to 3.03)$ $p=0.08$ $p=0.06$ $1.00 (reference)$ $1.79 (1.10 to 2.92)$ $p=0.04$ $p=0.02$ $1.00 (reference)$ $1.20 (reference)$ $1.66 (1.02 to 2.69)$ $1.79 (1.10 to 2.92)$ $p=0.04$ $p=0.02$ $1.00 (reference)$ $1.20 (reference)$ $1.22 (1.25 to 3.94)$ $2.22 (1.24 to 3.99)$ $2.14 (1.19 to 3.84)$ $2.22 (1.24 to 3.99)$ $2.21 (1.23 to 3.98)$ $2.35 (1.30 to 4.25)$			

test of association	p=0.07	p=0.05	p=0.1
≤ 2.50 > 2.50 test of association	1.00 (reference) 2.12 (1.20 to 3.75) p=0.009	1.00 (reference) 2.23 (1.26 to 3.96) p=0.006	1.00 (reference) 2.10 (1.18 to 3.73) p=0.01
<i>LTPA 68 years</i> ≤ 2.5 2.51-3.0 3.01-3.5 3.51-4.0 > 4.0 test of association	1.00 (reference) 1.60 (0.91 to 2.81) 1.54 (0.91 to 2.62) 1.70 (0.999 to 2.91) 1.41 (0.78 to 2.56) p=0.3	1.00 (reference) 1.61 (0.92 to 2.82) 1.56 (0.92 to 2.65) 1.74 (1.01 to 2.97) 1.46 (0.80 to 2.67) p=0.3	1.00 (reference) 1.55 (0.88 to 2.74) 1.45 (0.85 to 2.48) 1.56 (0.91 to 2.69) 1.35 (0.73 to 2.50) p=0.5
≤ 2.50 > 2.50 test of association	1.00 (reference) 1.59 (0.95 to 2.66) p=0.08	1.00 (reference) 1.61 (0.96 to 2.71) p=0.07	1.00 (reference) 1.50 (0.89 to 2.52) p=0.1

Analytic sample consists of those with leisure-time physical activity data from all five ages. $\leq 2.50 \text{ kg} (n=70)$, 2.51-3.00 kg (n=259), 3.01-3.50 kg (n=585), 3.51-4.00 kg (n=508), 4.01-5.00 kg (n=159). Model 1: adjusted for sex. Model 2: adjusted for sex and birth order Model 3: model 2 plus adjustment for father's occupational class. Models at age 60-64 are also adjusted for exact age in years. Tests of association based on likelihood ratio tests comparing models with and without the early life exposure.

	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA		s per RRR (95% CI) of regular LTPA (5 or mo per month) versus no LTPA			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Birth weight (kg)						
LTPA 36 years						
≤ 2.50	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
2.51-3.00	1.36	1.37	1.34	2.14	2.17	2.07
	(0.73 to 2.54)	(0.73 to 2.57)	(0.71 to 2.53)	(1.10 to 4.16)	(1.11 to 4.22)	(1.06 to 4.05)
3.01-3.50	1.46	<u> </u>	`	`	2.88	2.64
3.51-4.00	(0.81 to 2.63)	(0.82 to 2.66)	(0.77 to 2.55)	(1.49 to 5.26)	(1.53 to 5.41)	(1.40 to 4.99)
	1.01	1.02	0.93	2.56	2.68	2.36
> 4.00	(0.55 to 1.83)	(0.56 to 1.86)	(0.51 to 1.72)	(1.36 to 4.81)	(1.42 to 5.06)	(1.24 to 4.48)
	1.24	1.28	1.21	2.43	2.68	2.44
	(0.63 to 2.47)	(0.64 to 2.56)	(0.60 to 2.45)	(1.20 to 4.91)	(1.31 to 5.46)	(1.19 to 5.00)
≤ 2.50	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
> 2.50	1.26	1.28	1.22	2.56	2.65	2.42
	(0.72 to 2.22)	(0.73 to 2.26)	(0.68 to 2.16)	(1.39 to 4.71)	(1.44 to 4.90)	(1.30 to 4.49)
LTPA 43 years						
≤ 2.50 [°]	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
2.51-3.00	1.65	1.72	1.67	0.99	1.02	0.97
	(0.82 to 3.34)	(0.85 to 3.48)	(0.82 to 3.40)	(0.50 to 1.94)	(0.52 to 2.01)	(049 to 1.92)
3.01-3.50	2.01	2.15	2.05	1.84	1.96	1.81

Appendix 2B Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by birth weight group: standard multinomial logistic regression (n=1581).

3.51-4.00 > 4.00	(1.03 to 3.93) 1.89 (0.96 to 3.71) 1.62 (0.76 to 3.45)	(1.10 to 4.22) 2.11 (1.07 to 4.17) 2.03 (0.94 to 4.37)	(1.04 to 4.05) 1.94 (0.97 to 3.85) 1.94 (0.89 to 4.21)	(0.99 to 3.43) 1.59 (0.85 to 2.98) 1.74 (0.87 to 3.49)	(1.05 to 3.65) 1.76 (0.94 to 3.31) 2.13 (1.05 to 4.32)	(0.97 to 3.40) 1.57 (0.83 to 2.97) 1.98 (0.97 to 4.03)
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
> 2.50	(0.97 to 3.56)	2.03 (1.06 to 3.92)	(0.99 to 3.72)	(0.86 to 2.89)	(0.93 to 3.12)	1.56 (0.85 to 2.88)
LTPA 53 years						
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	` 1.37 (0.66 to 2.83)	1.42 (0.69 to 2.94)	1.36 (0.65 to 2.84)	1.45 (0.78 to 2.58)	` 1.49 (0.81 to 2.76)	1.43 (0.77 to 2.66)
3.01-3.50	1.59 (0.80 to 3.16)	1.70 (0.85 to 3.38)	1.57 (0.78 to 3.15)	1.98 (1.11 to 3.54)	2.08 (1.17 to 3.71)	1.92 (1.07 to 3.46)
3.51-4.00	1.60 (0.80 to 3.18)	(0.90 to 3.58)	1.60 (0.80 to 3.23)	(0.96 to 3.08)	1.86 (1.03 to 3.34)	1.64 (0.91 to 2.98)
> 4.00	1.21 (0.55 to 2.63)	(0.69 to 3.35) (0.69 to 3.35)	(0.63 to 3.20) (0.63 to 3.10)	(0.82 to 3.03)	1.85 (0.95 to 3.58)	(0.87 to 2.30) 1.70 (0.87 to 3.32)
	(0.00 to 2.00)	(0.00 10 0.00)	(0.00 10 0.10)	(0.02 10 0.00)	(0.00 10 0.00)	(0.07 10 0.02)
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.51 (0.78 to 2.92)	1.65 (0.85 to 3.22)	1.52 (0.78 to 2.98)	1.75 (0.99 to 3.06)	1.86 (1.06 to 3.27)	1.71 (0.97 to 3.02)
LTPA 60-64 years						
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	1.95 (0.87 to 4.41)	2.03 (0.90 to 4.60)	2.01 (0.88 to 3.58)	(1.00 to 5.02)	2.26 (1.01 to 5.07)	2.17 (0.97 to 4.88)
3.01-3.50	1.76 (0.81 to 3.83)	1.89 (0.86 to 4.14)	1.86 (0.84 to 4.09)	2.48 (1.15 to 3.35)	2.53 (1.17 to 5.46)	2.34 (1.08 to 5.07)

3.51-4.00	1.43	1.61	1.55	2.98	3.07	2.75
> 4.00	(0.65 to 3.17)	(0.72 to 3.58)	(0.69 to 3.46)	(1.38 to 6.44)	(1.42 to 6.66)	(1.26 to 3.99)
	1.13	1.39	1.38	2.41	2.55	2.36
	(0.46 to 2.80)	(0.55 to 3.50)	(0.54 to 3.47)	(1.04 to 5.54)	(1.10 to 5.92)	(1.01 to 4.50)
≤ 2.50	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
> 2.50	(1.62 (0.76 to 3.47)	(1.80 (0.84 to 3.87)	1.76 (0.82 to 3.81)	(1.22 to 5.51)	(1.24 to 5.63)	2.44
LTPA 68 years						
≤ 2.50	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
2.51-3.00	0.90	0.91	0.88	2.28	2.28	2.20
	(0.42 to 1.92)	(0.42 to 1.94)	(0.41 to 1.89)	(1.12 to 4.63)	(1.12 to 4.64)	(1.07 to 4.50)
3.01-3.50	0.82 (0.40 to 1.66)	0.84 (0.41 to 1.70)	0.79 (0.39 to 1.62)	(1.12 to 1.00) 2.21 (1.12 to 4.37)	2.23 (1.13 to 4.40)	2.06 (1.04 to 4.09)
3.51-4.00	(0.40 to 1.00)	(0.41 to 1.70)	(0.53 to 1.02)	(1.12 to 4.37)	(1.13 to 4.40)	(1.04 to 4.03)
	1.16	1.21	1.11	2.22	2.24	2.00
	(0.58 to 2.35)	(0.60 to 2.46)	(0.54 to 2.28)	(1.12 to 4.40)	(1.13 to 4.46)	(0.99 to 4.00)
> 4.00	(0.36 to 2.33) 1.02 (0.46 to 2.29)	(0.00 to 2.40) 1.11 (0.49 to 2.51)	1.05 (0.46 to 2.40)	(1.12 to 4.40) 1.76 (0.83 to 3.74)	(1.13 to 4.40) 1.80 (0.84 to 3.84)	(0.33 to 4.66) 1.66 (0.77 to 3.57)
≤ 2.50	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
> 2.50	0.96	0.99	0.93	2.18	2.20	2.03
	(0.49 to 1.89)	(0.50 to 1.95)	(0.47 to 1.84)	(1.12 to 4.24)	(1.13 to 4.29)	(1.04 to 3.98)

Analytic sample consists of those with leisure-time physical activity data from all five ages. $\leq 2.50 \text{ kg}$ (n=70), 2.51-3.00 kg (n=259), 3.01-3.50 kg (n=585), 3.51-4.00 kg (n=508), 4.01-5.00 kg (n=159). Model 1: adjusted for sex. Model 2: adjusted for sex and birth order Model 3: model 2 plus adjustment for father's occupational class. Models at age 60-64 are also adjusted for exact age in years. P-values for tests of association: Age 36 a. categorical birth weight: Model 1=0.01, Model 2=0.007, Model 3=0.02. b. binary birth weight: Model 1=0.006, Model 2=0.004, Model 3=0.01. Age 43 a. categorical birth weight: Model 1=0.03, Model 2=0.01, Model 3=0.03. b. binary birth weight: Model 1=0.09, Model 2=0.04, Model 3=0.09. Age 53 a. categorical birth weight: Model 1=0.3, Model 2=0.2, Model 3=0.4. b. binary birth weight: Model 1=0.1, Model 2=0.06, Model 3=0.1. Age 60-64 a. categorical birth weight: Model 1=0.03, Model 2=0.03, Model 2=0.05, Model 3=0.1. b. binary birth weight: Model 1=0.02, Model 2=0.01, Model 2=0.05, Model 3=0.1. b. binary birth weight: Model 1=0.02, Model 2=0.01, Model 2=0.05, Model 3=0.1. b. binary birth weight: Model 1=0.02, Model 2=0.01, Model 2=0.05, Model 3=0.1. b. binary birth weight: Model 1=0.02, Model 2=0.01, Model 2=0.01, Model 1=0.03, Model 2=0.05, Model 3=0.1. b. binary birth weight: Model 1=0.02, Model 2=0.01, Model 3=0.02. Age 68 a. categorical birth weight: Model 1=0.1, Model 1=0.1, Model 1=0.02, Model 2=0.01, Model 3=0.02. Age 68 a. categorical birth weight: Model 1=0.1, Model 1=0.1, Model 1=0.02, Model 2=0.01, Model 3=0.02. Age 68 a. categorical birth weight: Model 1=0.1, Model 1=0.1, Model 1=0.03, Model 2=0.01, Model 2=0.01, Model 3=0.02. Age 68 a. categorical birth weight: Model 1=0.1, Model 1=0.1, Model 1=0.02, Model 2=0.01, Model 3=0.02. Age 68 a. categorical birth weight: Model 1=0.1, Model 1=0.1

2=0.1, Model 3=0.2. b. binary birth weight: Model 1=0.04, Model 2=0.04, Model 3=0.07. Tests of association based on likelihood ratio tests comparing models with and without birth weight.

Appendix 2C OR of LTPA types and leisure-time walking at age 36 by birth weight: standard logistic regression.

	Model 1	Madal O	
	iniedol i	Model 2	Model 3
OR of team sports versus none at			
age 36 (n=3138)	4.00 ((1.00 (4.00 ((
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	1.58 (0.94 to 2.64)	1.58 (0.95 to 2.64)	1.53 (0.91 to 2.56)
3.01-3.50	1.84 (1.13 to 3.00)	1.85 (1.14 to 3.02)	1.77 (1.08 to 2.90)
3.51-4.00	1.89 (1.16 3.08)	1.93 (1.18 to 3.15)	1.81 (1.10 to 2.97)
> 4.00	1.61 (0.94 to 2.74)	1.69 (0.99 to 2.89)	1.60 (0.93 to 2.74)
test of association	p=0.06	p=0.05	p=0.1
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.78 (1.11 to 2.87)	1.81 (1.12 to 2.92)	1.72 (1.06 to 2.77)
test of association	p=0.02	p=0.02	p=0.03
OR of non-team sports versus			
none at age 36 (n=3108)			
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	1.50 (1.02 to 2.18)	1.50 (1.03 to 2.20)	1.45 (0.99 to 2.12)
3.01-3.50	1.55 (1.08 to 2.21)	1.57 (1.10 to 2.24)	1.49 (1.04 to 2.14)
3.51-4.00	1.41 (0.99 to 2.03)	1.47 (1.03 to 2.11)	1.37 (0.95 to 1.97)
> 4.00	1.44 (0.96 to 2.16)	1.56 (1.03 to 2.34)	1.46 (0.97 to 2.21)
test of association	p=0.2	p=0.2	p=0.3
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.48 (1.05 to 2.09)	1.52 (1.08 to 2.15)	1.44 (1.02 to 2.04)
test of association	p=0.03	p=0.02	p=0.04
OR of leisure-time walking versus			
none at age 36 (n=3129)			
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	1.22 (0.83 to 1.78)	1.22 (0.83 to 1.80)	1.18 (0.80 to 1.74)
3.01-3.50	1.56 (1.08 to 2.25)	1.59 (1.10 to 2.29)	1.51 (1.05 to 2.19)
3.51-4.00	1.52 (1.05 to 2.19)	1.58 (1.09 to 2.29)	1.48 (1.02 to 2.15)
> 4.00	1.08 (0.72 to 1.64)	1.18 (0.78 to 1.79)	1.12 (0.73 to 1.69)
test of association	p=0.005	p=0.006	p=0.01
≤ 2.50	1.00 (reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.42 (1.00 to 2.02)	1.46 (1.03 to 2.09)	1.39 (0.97 to 1.98)
test of association Model 1: adjusted for sex. Mo	p=0.001	p=0.04	p=0.07

Model 1: adjusted for sex. Model 2: adjusted for sex and birth order Model 3: model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without birth weight term. **Appendix 2D** OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by birth weight: standard linear regression – comparable sample.

Birth weight (kg) n=1583			
	Model 1	Model 2	Model 3
OR of LTPA at least once per			
month versus none at age 60-64			
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	2.07 (1.13 to 3.78)	2.12 (1.16 to 3.89)	2.11 (1.14 to 3.89)
3.01-3.50	1.89 (1.06 to 3.37)	1.97 (1.10 to 3.51)	1.92 (1.07 to 3.43)
3.51-4.00	1.78 (1.00 to 3.18)	1.93 (1.08 to 3.46)	1.83 (1.02 to 3.30)
> 4.00	1.49 (0.79 to 2.83)	1.70 (0.89 to 3.26)	1.66 (0.86 to 3.19)
test of association	p=0.1	p=0.1	p=0.2
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.84 (1.05 to 3.22)	1.96 (1.12 to 3.45)	1.12 (0.91 to 1.37)
test of association	p=0.03	p=0.02	p=0.3
% difference in MVPA age 60-64			
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	18.5 (-12.1 to 49.1)	19.0 (-11.6 to 49.6)	18.2 (-12.4 to 48.8)
3.01-3.50	16.2 (12.6 to 44.9)	16.9 (-11.9 to 45.7)	15.4 (-13.4 to 44.2)
3.51-4.00	7.4 (-21.5 to 36.3)	9.1 (-20.0 to 38.1)	6.9 (-22.1 to 36.0)
> 4.00	9.7 (-22.6 to 42.0)	12.6 (-20.1 to 45.2)	11.1 (-21.6 to 43.8)
test of association	p=0.5	p=0.6	p=0.5
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
> 2.50	12.9 (-14.9 to 40.7)	14.3 (-13.6 to 42.2)	12.8 (-15.1 to 40.7)
test of association	p=0.4	p=0.3	p=0.4
% difference in PAEE age 60-64			
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
2.51-3.00	0.2 (-10.9 to 11.3)	0.3 (-10.8 to 11.5)	-0.02 (-11.1 to 11.1)
3.01-3.50	2.7 (-7.8 to 13.2)	2.9 (-7.5 to 13.4)	2.3 (-8.2 to 12.8)
3.51-4.00	0.02 (-10.5 to 10.5)	0.6 (-10.0 to 11.1)	-0.2 (-10.8 to 10.4)
> 4.00	1.9 (-9.8 to 13.6)	2.9 (-9.0 to 14.7)	2.2 (-9.7 to 14.1)
test of association	p=0.8	p=0.8	p=0.9
≤ 2.50	1.00(reference)	1.00 (reference)	1.00 (reference)
> 2.50	1.3 (-8.8 to 11.4)	1.7 (-8.5 to 11.8)	1.0 (-9.1 to 11.2)
test of association	p=0.8	p=0.7	p=0.8
Analytic sample consists of	-	-	

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64. Model 1: adjusted for sex. Model 2: adjusted for sex and birth order Model 3: model 2 plus adjustment for father's occupational class. Models at age 60-64 are also adjusted for exact age in years. Tests of association based on likelihood ratio tests comparing models with and without birth weight term.

		east once per month vers	
	Model 1 OR (95% CI)	Model 2 OR (95% CI)	Model 3 OR (95% CI)
<u>Motor milestones</u> months			
LTPA age 36			
Sitting			
≤ 5m	0.76 (0.57 to 1.00)	0.77 (0.58 to 1.01)	0.79 (0.60 to 1.06)
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.83 (0.57 to 1.21)	0.85 (0.58 to 1.25)	0.80 (0.54 to 1.17
est of association	p=0.1	p=0.2	p=0.2
per later month	1.03 (0.96 to 1.11)	1.04 (0.96 to 1.12)	1.01 (0.94 to 1.10
est of association	p=0.4	p=0.4	p=0.7
Standing			
≦ 8m	0.79 (0.46 to 1.33)	0.80 (0.47 to 1.37)	0.84 (0.49 to 1.44
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	0.88 (0.60 to 1.29)	0.90 (0.61 to 1.32)	0.80 (0.54 to 1.19
est of association	p=0.6	p=0.6	p=0.5
per later month	0.99 (0.94 to 1.04)	0.99 (0.94 to 1.04)	0.97 (0.93 to 1.03
est of association	p=0.7	p=0.8	p=0.3
Valking			
≤ 10m	0.69 (0.46 to 1.03)	0.70 (0.46 to 1.05)	0.76 (0.50 to 1.14
11-17m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 18m	0.84 (0.57 to 1.25)	0.88 (0.59 to 1.31)	0.79 (0.53 to 1.20
est of association	p=0.2	p=0.2	p=0.3
LTPA age 43			
Sitting			
≤ 5m	1.10 (0.84 to 1.43)	1.16 (0.88 to 1.52)	1.22 (0.92 to 1.61
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	1.09 (0.76 to 1.56)	1.13 (0.78 to 1.63)	1.04 (0.72 to 1.51
est of association	p=0.8	p=0.5	p=0.4
per later month	1.02 (0.95 to 1.10)	1.03 (0.95 to 1.10)	0.995 (0.92 to 1.07
est of association	p=0.5	p=0.5	p=0.9
standing			
≤ 8m	0.92 (0.55 to 1.53)	0.97 (0.57 to 1.62)	1.01 (0.59 to 1.71
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	0.85 (0.59 to 1.23)	0.87 (0.60 to 1.26)	0.75 (0.51 to 1.10
est of association	p=0.7	p=0.8	p=0.3
per later month	1.00 (0.96 to 1.05)	1.00 (0.96 to 1.05)	0.98 (0.93 to 1.03
test of association	p=0.9	p=0.8	p=0.5

Appendix 3A Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by attainment of motor milestones: standard binary logistic regression (n=1452)

<u>Walking</u> ≤ 10m 11-17m ≥ 18m test of association	0.71 (0.48 to 1.06) 1.00 (reference) 0.88 (0.61 to 1.29) p=0.2	0.74 (0.49 to 1.11) 1.00 (reference) 0.93 (0.63 to 1.38) p=0.3	0.84 (0.55 to 1.26) 1.00 (reference) 0.83 (0.56 to 1.23) p=0.5
LTPA age 53			
<u>Siting</u> ≤ 5m 6-8m ≥ 9m test of association	0.81 (0.62 to 1.06) 1.00 (reference) 1.11 (0.77 to 1.61) p=0.2	0.84 (0.64 to 1.10) 1.00 (reference) 1.16 (0.80 to 1.67) p=0.3	0.87 (0.66 to 1.15) 1.00 (reference) 1.08 (0.74 to 1.57) p=0.5
per later month test of association	1.07 (0.99 to 1.15) p=0.09	1.07 (0.99 to 1.15) p=0.08	1.04 (0.97 to 1.12) p=0.3
<u>Standing</u> ≤ 8m 9-14m ≥ 15m test of association	0.91 (0.55 to 1.52) 1.00 (reference) 1.52 (1.04 to 2.23) p=0.08	0.95 (0.57 to 1.59) 1.00 (reference) 1.59 (1.08 to 2.34) p=0.05	1.00 (0.59 to 1.69) 1.00 (reference) 1.43 (0.97 to 2.12) p=0.2
per later month test of association	1.06 (1.01 to 1.12) p=0.01	1.07 (1.02 to 1.12) p=0.009	1.05 (0.998 to 1.10) p=0.06
<u>Walking</u> ≤ 10m 11-17m ≥ 18m test of association	0.91 (0.61 to 1.36) 1.00 (reference) 1.12 (0.76 to 1.64) p=0.8	0.95 (0.64 to 1.42) 1.00 (reference) 1.18 (0.80 to 1.75) p=0.7	1.05 (0.70 to 1.57) 1.00 (reference) 1.07 (0.72 to 1.59) p=0.9
LTPA age 60-64			
<u>Sitting</u> ≤ 5m 6-8m ≥ 9m test of association	1.10 (0.83 to 1.44) 1.00 (reference) 0.81 (0.55 to 1.18) p=0.4	1.12 (0.85 to 1.47) 1.00 (reference) 0.81 (0.55 to 1.20) p=0.4	1.16 (0.88 to 1.54) 1.00 (reference) 0.76 (0.51 to 1.12) p=0.2
per later month test of association	0.98 (0.91 to 1.06) p=0.6	0.98 (0.91 to 1.06) p=0.6	0.96 (0.89 to 1.03) p=0.3
<u>Standing</u> ≤ 8m 9-14m ≥ 15m test of association	0.84 (0.49 to 1.45) 1.00 (reference) 0.93 (0.64 to 1.35) p=0.8	0.84 (0.49 to 1.45) 1.00 (reference) 0.98 (0.67 to 1.44) p=0.8	0.87 (0.50 to 1.50) 1.00 (reference) 0.88 (0.59 to 1.29) p=0.7
per later month test of association	0.99 (0.95 to 1.04) p=0.8	1.00 (0.95 to 1.05) p=0.98	0.98 (0.93 to 1.03) p=0.5
<u>Walking</u> ≤ 10m 11-17m ≥ 18m	1.04 (0.69 to 1.56) 1.00 (reference) 0.92 (0.62 to 1.36)	1.05 (0.70 to 1.59) 1.00 (reference) 1.01 (0.68 to 1.51)	1.18 (0.78 to 1.80) 1.00 (reference) 0.92 (0.62 to 1.38)

test of association	p=0.9	p=0.97	p=0.7
LTPA age 68			
<u>Sitting</u> ≤ 5m 6-8m ≥ 9m test of association	0.96 (0.73 to 1.25) 1.00 (reference) 1.00 (0.70 to 1.44) p=0.9	0.96 (0.74 to 1.27) 1.00 (reference) 1.01 (0.70 to 1.45) p=0.97	1.01 (0.76 to 1.33) 1.00 (reference) 0.93 (0.64 to 1.35) p=0.93
per later month test of association	1.03 (0.96 to 1.10) p=0.5	1.03 (0.96 to 1.11) p=0.4	1.00 (0.93 to 1.08) p=0.97
<u>Standing</u> ≤ 8m 9-14m ≥ 15m test of association	0.75 (0.44 to 1.28) 1.00 (reference) 0.80 (0.55 to 1.15) p=0.3	0.75 (0.44 to 1.28) 1.00 (reference) 0.81 (0.55 to 1.17) p=0.3	0.78 (0.45 to 1.34) 1.00 (reference) 0.71 (0.48 to 1.04) p=0.1
per later month test of association	1.01 (0.96 to 1.05) p=0.8	1.01 (0.96 to 1.06) p=0.8	0.99 (0.94 to 1.04) p=0.6
<u>Walking</u> ≤ 10m 11-17m ≥ 18m test of association	0.90 (0.60 to 1.35) 1.00 (reference) 0.97 (0.67 to 1.42) p=0.9	0.90 (0.60 to 1.36) 1.00 (reference) 1.01 (0.68 to 1.48) p=0.9	1.02 (0.67 to 1.54) 1.00 (reference) 0.91 (0.61 to 1.34) p=0.9

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Sitting: $\leq 5m$ (n=272), 6-8m (n=1051), $\geq 9m$ (n=136). Standing: $\leq 8m$ (n=62), 9-14m (1265), $\geq 15m$ (n=132). Walking: $\leq 10m$ (n=107), 11-17m (n=1231), $\geq 18m$ (n=121). Tests of association based on likelihood ratio tests comparing models with and without motor milestone terms.

	RRR (95% CI) of	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA			of regular LTPA (nonth) versus no	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
LTPA age 36						
Sitting						
≤ 5m	0.81	0.82	0.84	0.74	0.75	0.78
	(0.57 to 1.14)	(0.58 to 1.15)	(0.59 to 1.19)	(0.54 to 1.01)	(0.54 to 1.02)	(0.57 to 1.07)
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.89	0.91	0.85	0.79	0.82	0.77
	(0.56 to 1.40)	(0.57 to 1.44)	(0.53 to 1.35)	(0.52 to 1.21)	(0.54 to 1.25)	(0.50 to 1.18)
per later month	1.02	1.01	0.99	1.04	1.05	1.02
	(0.93 to 1.12)	(0.92 to 1.11)	(0.90 to 1.09)	(0.95 to 1.13)	(0.96 to 1.14)	(0.94 to 1.11)
Standing						
≤ 8m	0.91	0.95	0.99	0.71	0.72	0.76
	(0.48 to 1.72)	(0.50 to 1.80)	(0.52 to 1.89)	(0.39 to 1.30)	(0.39 to 1.32)	(0.41 to 1.39)
9-14m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 15m	1.20	1.19	1.10	0.70	0.73	0.64
	(0.78 to 1.87)	(0.76 to 1.85)	(0.70 to 1.72)	(0.45 to 1.09)	(0.47 to 1.14)	(0.41 to 1.02)
per later month	1.01	1.01	0.996	0.98	0.98	0.96
	(0.96 to 1.08)	(0.95 to 1.07)	(0.94 to 1.06)	(0.92 to 1.03)	(0.93 to 1.04)	(0.91 to 1.02)

Appendix 3B Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by attainment of motor milestones: standard multinomial logistic regression (n=1452).

Walking	0.00	1.00	4.40	0.53	0.50	0.50
≤ 10m	0.98	1.02	1.10 (0.69 to 1.79)	0.52	0.52	0.56
11-17m	(0.61 to 1.58) 1.00	(0.63 to 1.65) 1.00	(0.68 to 1.78) 1.00	(0.32 to 0.84) 1.00	(0.32 to 0.85) 1.00	(0.35 to 0.92) 1.00
1 1-17111	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 18m	1.57	1.58	1.47	0.47	0.49	0.44
- 10	(1.01 to 2.42)	(1.02 to 2.46)	(0.94 to 2.30)	(0.28 to 0.77)	(0.30 to 0.82)	(0.27 to 0.74)
LTPA age 43						
Sitting						
<u>≤ 5m</u>	1.18	1.26	1.31	1.03	1.07	1.13
	(0.85 to 1.64)	(0.90 to 1.75)	(0.94 to 1.84)	(0.74 to 1.42)	(0.77 to 1.49)	(0.81 to 1.57)
6-8m	1.00	1.00	`	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 9m	1.45	1.50	1.37	0.77	0.79	0.74
	(0.96 to 2.19)	(0.99 to 2.28)	(0.90 to 2.10)	(0.48 to 1.23)	(0.49 to 1.28)	(0.46 to 1.19)
per later month	1.05	1.05	1.02	0.998	0.9996	0.97
	(0.97 to 1.15)	(0.96 to 1.15)	(0.93 to 1.11)	(0.91 to 1.09)	(0.91 to 1.09)	(0.89 to 1.06)
Standing						
≤ 8m	1.06	1.13	1.18	0.77	0.81	0.85
	(0.58 to 1.95)	(0.61 to 2.08)	(0.63 to 2.19)	(0.40 to 1.49)	(0.41 to 1.58)	(0.43 to 1.67)
9-14m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 15m	0.85	0.88	0.76	0.87	0.89	0.78
	(0.54 to 1.34)	(0.56 to 1.40)	(0.48 to 1.21)	(0.57 to 1.35)	(0.57 to 1.38)	(0.49 to 1.22
per later month	1.03	1.03	1.00	0.98	0.98	0.96
	(0.97 to 1.09)	(0.97 to 1.09)	(0.94 to 1.06)	(0.93 to 1.04)	(0.93 to 1.04)	(0.91 to 1.02

≤ 10m	0.73	0.76	0.89	0.68	0.71	0.78
	(0.45 to 1.21)	(0.46 to 1.27)	(0.53 to 1.49)	(0.41 to 1.13)	(0.43 to 1.17)	(0.47 to 1.30)
11-17m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 18m	0.94	0.99	0.88	0.84	0.88	0.79
	(0.59 to 1.48)	(0.62 to 1.58)	(0.55 to 1.41)	(0.53 to 1.32)	(0.55 to 1.40)	(0.49 to 1.28)
LTPA age 53						
Sitting						
≤ 5m	0.95	0.996	1.03	0.74	0.76	0.80
	(0.66 to 1.35)	(0.70 to 1.42)	(0.72 to 1.48)	(0.55 to 1.00)	(0.56 to 1.04)	(0.58 to 1.09)
6-8m	1.00	1.00	1.00	1.00	1.00	1.00
_	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 9m	1.47	1.51	1.45	0.93	0.97	0.89
	(0.94 to 2.31)	(0.96 to 2.38)	(0.91 to 2.29)	(0.61 to 1.41)	(0.63 to 1.48)	(0.58 to 1.37)
per later month	1.06	1.06	1.04	1.07	1.07	1.04
	(0.97 o 1.17)	(0.96 to 1.16)	(0.94 to 1.15)	(0.98 to 1.16)	(0.99 to 1.16)	(0.96 to 1.13)
<u>Standing</u>						
≤ 8m	0.93	0.99	1.04	0.90	0.93	0.98
	(0.47 to 1.86)	(0.49 to 1.98)	(0.51 to 2.10)	(0.50 to 1.60)	(0.52 to 1.67)	(0.54 to 1.76)
9-14m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 15m	1.70	1.76	1.67	1.45	1.53	1.34
	(1.06 to 2.72)	(1.09 to 2.83)	(1.03 to 2.71)	(0.95 to 2.20)	(0.99 to 2.34)	(0.87 to 2.07)
per later month	1.07	1.07	1.06	1.06	1.07	1.05
	(1.00 to 1.14)	(1.00 to 1.14)	(0.99 to 1.13)	(1.01 to 1.12)	(1.01 to 1.13)	(0.99 to 1.11)
Walking						
≤ 10m	1.16	1.22	1.27	0.78	0.81	0.92
	(0.70 to 1.92)	(0.74 to 2.03)	(0.76 to 2.13)	(0.49 to 1.24)	(0.51 to 1.28)	(0.57 to 1.47)

11-17m	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 18m	(1.15 (0.70 to 1.90)	(1.20 (0.72 to 2.00)	1.13 (0.67 to 1.90)	(101010100) 1.15 (0.75 to 1.75)	(1.23 (0.80 to 1.89)	(Tereference) 1.10 (0.71 to 1.70
LTPA age 60-64						
Sitting						
≤ 5m	1.19 (0.82 to 1.73)	1.24 (0.85 to 1.80)	1.27 (0.87 to 1.86)	1.03 (0.74 to 1.43)	1.04 (0.75 to 1.45)	1.10 (0.78 to 1.53
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.70 (0.39 to 1.24)	0.70 (0.39 to 1.24)	0.65 (0.36 to 1.17)	0.86 (0.55 to 1.35)	0.87 (0.56 to 1.37)	0.81 (0.52 to 1.27
per later month	0.95 (0.86 to 1.06)	0.95 (0.85 to 1.05)	0.92 (0.83 to 1.03)	0.996 (0.91 to 1.09)	1.00 (0.92 to 1.09)	0.98 (0.89 to 1.07
Standing						
≤ 8m	0.71 (0.31 to 1.62)	0.74 (0.32 to 1.68)	0.75 (0.33 to 1.71)	0.93 (0.50 to 1.73)	0.91 (0.49 to 1.70)	0.94 (0.50 to 1.77
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	1.07 (0.65 to 1.78)	1.12 (0.67 to 1.87)	1.04 (0.62 to 1.75)	0.84 (0.53 to 1.33)	0.89 (0.56 to 1.43)	0.78 (0.49 to 1.25
per later month	1.00 (0.94 to 1.07)	1.01 (0.94 to 1.08)	0.99 (0.93 to 1.07)	0.98 (0.93 to 1.04)	0.99 (0.94 to 1.05)	0.97 (0.91 to 1.03
Walking						
≤ 10m	1.24 (0.73 to 2.13)	1.31 (0.76 to 2.27)	1.45 (0.84 to 2.52)	0.91 (0.55 to 1.51)	0.90 (0.54 to 1.49)	1.02 (0.61 to 1.71
11-17m	1.00 (reference)	1.00 (reference)	1.00 (reference)	(0.00 (0 1.01) 1.00 (reference)	(reference)	(reference)
≥ 18m	1.00	1.15	1.06	0.87	0.94	0.84

	(0.58 to 1.71)	(0.66 to 1.20)	(0.61 to 1.85)	(0.54 to 1.39)	(0.58 to 1.51)	(0.52 to 1.36)
LTPA age 68						
Sitting						
≤ 5m	1.05	1.08	1.12	0.92	0.93	0.97
	(0.70 to 1.58)	(0.71 to 1.63)	(0.74 to 1.70)	(0.68 to 1.26)	(0.68 to 1.27)	(0.71 to 1.33)
6-8m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 9m	0.89	0.87	0.80	<u> </u>	`	`
	(0.50 to 1.59)	(0.48 to 1.56)	(0.44 to 1.44)	(0.72 to 1.59)	(0.73 to 1.62)	(0.67 to 1.51)
per later month	1.02	1.01	0.98	1.03	1.04	1.01
	(0.91 to 1.13)	(0.90 to 1.13)	(0.88 to 1.10)	(0.95 to 1.12)	(0.96 to 1.12)	(0.93 to 1.10)
Standing						
<u>≤ 8m</u>	0.78	0.77	0.78	0.75	0.76	0.79
	(0.34 to 1.77)	(0.34 to 1.76)	(0.34 to 1.80)	(0.41 to 1.38)	(0.41 to 1.39)	(0.43 to 1.47)
9-14m	`	`	`	`	`	`
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 15m	`0.71 ´	` 0.73 ́	`0.63 ´	0.82	` 0.83 ́	` 0.73 ́
	(0.39 to 1.31)	(0.39 to 1.34)	(0.34 to 1.17)	(0.54 to 1.24)	(0.55 to 1.26)	(0.48 to 1.12)
per later month	0.99	0.99	0.97	1.01	1.01	0.99
•	(0.92 to 1.07)	(0.92 to 1.07)	(0.90 to 1.04)	(0.96 to 1.07)	(0.96 to 1.07)	(0.94 to 1.05)
Walking						
<u>≤ 10m</u>	1.04	1.03	1.21	0.85	0.86	0.95
	(0.58 to 1.88)	(0.57 to 1.86)	(0.66 to 2.20)	(0.53 to 1.35)	(0.54 to 1.37)	(0.59 to 1.53)
11-17m	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
≥ 18m	0.65	0.66	0.58	1.07	1.12	1.01
	(0.33 to 1.28)	(0.33 to 1.31)	(0.29 to 1.16)	(0.71 to 1.63)	(0.73 to 1.71)	(0.66 to 1.55)

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Sitting:

 $\leq 5m$ (n=272), 6-8m (n=1051), $\geq 9m$ (n=136). Standing: $\leq 8m$ (n=62), 9-14m (1265), $\geq 15m$ (n=132). Walking: $\leq 10m$ (n=107), 11-17m (n=1231), ≥ 18m (n=121). P-values for tests of association: Age 36 a. categorical sitting variable: Model 1=0.4, Model 2=0.4, Model 3=0.5. b. continuous sitting variable: Model 1=0.7, Model 2=0.6, Model 3=0.8. categorical standing variable: Model 1=0.1, Model 2=0.2, Model 3=0.1. b. continuous standing variable: Model 1=0.4, Model 2=0.6, Model 3=0.4. categorical walking variable: Model 1<0.001, Model 2<0.001, Model 3<0.001. Age 43 a. categorical sitting variable: Model 1=0.1, Model 2=0.1, Model 3=0.09. b. continuous sitting variable: Model 1=0.5, Model 2=0.5, Model 3=0.7. categorical standing variable: Model 1=0.8, Model 2=0.9, Model 3=0.8. b. continuous standing variable: Model 1=0.4, Model 2=0.4, Model 3=0.4. categorical walking variable: Model 1=0.5, Model 2=0.6, Model 3=0.7. Age 53 a. categorical sitting variable: Model 1=0.1, Model 2=0.1, Model 3=0.2. b. continuous sitting variable: Model 1=0.2, Model 2=0.2, Model 3=0.6. categorical standing variable: Model 1=0.2, Model 2=0.2, Model 3=0.3. b. continuous standing variable: Model 1=0.03, Model 2=0.03, Model 3=0.1, categorical walking variable: Model 1=0.6, Model 2=0.5, Model 3=0.8, Age 60-64 a, categorical sitting variable: Model 1=0.6, Model 2=0.5, Model 3=0.3. b. continuous sitting variable: Model 1=0.6, Model 2=0.6, Model 3=0.4. categorical standing variable: Model 1=0.8, Model 2=0.9, Model 3=0.6. b. continuous standing variable: Model 1=0.03, Model 2=0.03, Model 3=0.1. categorical walking variable: Model 1=0.9, Model 2=0.8, Model 3=0.7. Age 68 a. categorical sitting variable: Model 1=0.9, Model 2=0.9, Model 3=0.9. b. continuous sitting variable: Model 1=0.7, Model 2=0.7, Model 3=0.9. categorical standing variable: Model 1=0.6, Model 2=0.2, Model 3=0.3. b. continuous standing variable: Model 1=0.9, Model 2=0.9, Model 3=0.8. categorical walking variable: Model 1=0.6, Model 2=0.7, Model 3=0.5. Tests of association based on likelihood ratio tests comparing models with and without the term for motor milestones.

Appendix 3C OR of LTPA types and leisure-time walking at age 36 by attainment of motor milestones: standard logistic regression.

Motor milestones (months)	Model 1	Model 2	Model 3
OR of team sports versus none at			
age 36			
Sitting			
≤ 5m	0.80 (0.64 to 1.01)	0.80 (0.64 to 1.01)	0.82 (0.65 to 1.03
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.88 (0.65 to 1.18)	0.90 (0.67 to 1.21)	0.87 (0.64 to 1.17
test of association	p=0.1	p=0.1	p=0.2
Per later month	1.02 (0.97 to 1.08)	1.03 (0.97 to 1.09)	1.02 (0.96 to 1.08
test of association	p=0.5	p=0.3	p=0.5
OR of non-team sports versus			
none at age 36 (n=2818)			
Sitting			
≤ 5m	0.97 (0.80 to 1.17)	0.98 (0.81 to 1.19)	1.00 (0.83 to 1.22
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.96 (0.74 to 1.23)	0.98 (0.76 to 1.27)	0.95 (0.73 to 1.23
test of association	p=0.9	p>0.9	p=0.9
Per later month	1.00 (0.95 to 1.05)	1.00 (0.95 to 1.06)	0.99 (0.94 to 1.04
test of association	p>0.9	p=0.9	p=0.7
OR of leisure-time walking versus			
none at age 36			
Sitting			
≤ 5m	1.00 (0.82 to 1.23)	1.02 (0.83 to 1.25)	1.05 (0.85 to 1.29
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	1.21 (0.92 to 1.59)	1.26 (0.95 to 1.67)	1.22 (0.92 to 1.61
test of association	p=0.4	p=0.3	p=0.4
Per later month	1.03 (0.97 to 1.08)	1.04 (0.98 to 1.09)	1.02 (0.97 to 1.08
test of association	p=0.3	p=0.2	p=0.4
OR of team sports versus none at			
age 36			
<u>Standing</u>			
≤ 8m	1.02 (0.70 to 1.50)	1.03 (0.70 to 1.50)	1.07 (0.73 to 1.57
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	0.84 (0.61 to 1.15)	0.86 (0.62 to 1.20)	0.82 (0.59 to 1.13
test of association	p=0.5	p=0.7	p=0.4
Per later month	1.00 (0.96 to 1.03)	1.01 (0.96 to 1.04)	0.99 (0.95 to 1.03
test of association	p=0.8	p>0.9	p=0.6
OR of non-team sports versus			
none at age 36			
Standing			
≤ 8m	0.97 (0.70 to 1.35)	0.99 (0.71 to 1.38)	1.02 (0.73 to 1.43
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	0.92 (0.70 to 1.21)	0.95 (0.72 to 1.25)	0.89 (0.67 to 1.17

test of association	p=0.8	p=0.9	p=0.7
Per later month test of association	0.99 (0.96 to 1.03) p=0.8	1.00 (0.96 to 1.03) p=0.8	0.98 (0.95 to 1.02) p=0.4
OR of leisure-time walking versus none at age 36 <u>Standing</u> ≤ 8m	1 02 (0 72 to 1 46)	1.05 (0.74 to 1.50)	1.09 (0.76 to 1.56)
9-14m ≥ 15m test of association	1.03 (0.72 to 1.46) 1.00 (reference) 1.33 (0.98 to 1.80) p=0.2	1.00 (reference) 1.40 (1.03 to 1.91) p=0.09	1.09 (0.76 to 1.36) 1.00 (reference) 1.31 (0.96 to 1.79) p=0.2
Per later month test of association	1.03 (1.00 to 1.07) p=0.08	1.04 (1.00 to 1.08) p=0.05	1.03 (0.99 to 1.06) p=0.2
<i>OR of team sports versus none at age 36</i> Walking			
≤ 10m 11-17m	0.76 (0.54 to 1.05) 1.00 (reference)	0.77 (0.56 to 1.07) 1.00 (reference)	0.83 (0.59 to 1.15) 1.00 (reference)
≥ 18m test of association	0.78 (0.56 to 1.08) p=0.09	0.82 (0.59 to 1.15) p=0.2	0.77 (0.55 to 1.08) p=0.2
OR of non-team sports versus none at age 36 Walking			
≤ 10m	0.82 (0.62 to 1.07)	0.84 (0.64 to 1.10)	0.89 (0.68 to 1.17)
11-17m ≥ 18m	1.00 (reference) 0.97 (0.73 to 1.28)	1.00 (reference) 1.01 (0.76 to 1.34)	1.00 (reference) 0.95 (0.71 to 1.27)
test of association	p=0.3	p=0.4	p=0.7
<i>OR of leisure-time walking versus none at age 36</i> Walking			
≤ 10m	0.88 (0.67 to 1.16)	0.91 (0.69 to 1.20)	0.98 (0.73 1.30)
11-17m ≥ 18m	1.00 (reference) 1.08 (0.81 to 1.46)	1.00 (reference) 1.17 (0.86 to 1.58)	1.00 (reference) 1.10 (0.81 1.49)
test of association	p=0.6	p=0.4	p=0.8

Team sports: n=2842. Non-team sports: n=2818. Leisure-time walking: n=2836. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without motor milestone terms. **Appendix 3D** OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by attainment of motor milestones: standard linear regression – comparable sample.

Motor milestones (months) n=1445	Model 1	Model 2	Model 3
OR of LTPA at least once per month versus none at age 60-64 Sitting			
≤ 5m	1.12 (0.85 to 1.48)	1.15 (0.87 to 1.52)	1.18 (0.89 to 1.57)
6-8m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 9m	0.74 (0.49 to 1.10)	0.78 (0.52 to 1.17)	0.73 (0.48 to 1.10)
test of association	p=0.2	p=0.2	p=0.1
Per later month	0.98 (0.91 to 1.06)	0.98 (0.91 to 1.06)	0.96 (.89 to 1.04)
test of association	p=0.6	p=0.7	p=0.4
% difference in MVPA age 60-64 Sitting			
 ≤ 5m 6-8m ≥ 9m test of association 	-16.1 (-31.1 to -1.0)	-15.7 (-30.9 to -0.6)	-14.9 (-30.0 to 0.2)
	1.00 (reference)	1.00 (reference)	1.00 (reference)
	-3.2 (-24.0 to 17.7)	-1.3 (-22.2 to 19.6)	-3.2 (-24.0 to 17.7)
	p=0.1	p=0.1	p=0.2
Per later month test of association	3.9 (-0.2 to 8.1)	4.2 (0.01 to 8.4)	3.6 (-0.6 to 7.8)
	p=0.06	p=0.05	p=0.09
% difference in PAEE age 60-64 Sitting ≤ 5m 6-8m ≥ 9m test of association	-4.9 (-10.4 to 0.7) 1.00 (reference) -0.8 (-8.4 to 6.9) p=0.2	-4.7 (-10.3 to 0.9) 1.00 (reference) -0.2 (-7.9 to 7.4) p=0.2	-4.4 (-10.0 to 1.1) 1.00 (reference) -0.7 (-8.4 to 7.0) p=0.3
Per later month test of association	1.2 (-0.3 to 2.7)	1.3 (-0.2 to 2.8)	1.1 (-0.4 to 2.7)
	p=0.1	p=0.1	p=0.1
OR of LTPA at least once per month versus none at age 60-64 Standing			
≤ 8m	0.85 (0.51 to 1.43)	0.85 (0.51 to 1.43)	0.82 (0.49 to 1.39)
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m	1.01 (0.68 to 1.49)	1.06 (0.71 to 1.57)	0.65 (0.64 to 1.43)
test of association	P=0.8	P=0.8	P=0.8
Per later month test of association	1.01 (0.96 to 1.06)	1.01 (0.96 to 1.07)	1.00 (0.95 to 1.05)
	p=0.8	p=0.6	p=0.9
% difference in MVPA age 60-64 <u>Standing</u> ≤ 8m 9-14m ≥ 15m test of association	23.3 (-3.9 to 50.5) 1.00 (reference) 20.4 (-0.6 to 41.3) P=0.05	23.6 (-3.6 to 50.9) 1.00 (reference) 22.0 (0.9 to 43.0) P=0.04	22.5 (-4.7 to 50.0) 1.00 (reference) 18.8 (-2.6 to 39.7) P=0.07

Per later month test of association	1.8 (-0.9 to 4.4) p=0.2	2.0 (-0.6 to 4.7) p=0.1	1.5 (-1.2 to 4.2) p=0.3
% difference in PAEE age 60-64			
Standing			
≤ 8m	6.2 (-3.9 to 16.2)	6.4 (-3.6 to 16.5)	6.3 (-3.8 to 16.3)
9-14m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 15m test of association	5.7 (-2.013.4) P=0.2	6.2 (-1.5 to 14.0) P=0.1	5.3 (-2.5 to 13.1) p-0.2
	1 =0.2	1 =0.1	p=0.2
Per later month			
test of association	0.3 (-0.6 to 1.3)	0.4 (-0.6 to 1.4)	0.3 (-0.7 to 12.6)
	p=0.5	p=0.4	`p=0.6
OR of LTPA at least once per month	-	-	-
versus none at age 60-64			
Walking			
≤ 10m	0.92 (0.61 to 1.38)	0.92 (0.61 to 1.39)	1.03 (0.68 to 1.56)
11-17m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 18m	1.02 (0.68 to 1.53)	1.14 (0.76 to 1.71)	1.06 (0.70 to 1.60)
test of association	P=0.9	P=0.7	>0.9
% difference in MVPA age 60-64			
Walking			
≤ 10m	-6.8 (-28.7 to 15.1)	-6.1 (-28.1 to 15.9)	-2.4 (-24.5 to 19.6)
11-17m	1.00 (reference)	1.00 (reference)	1.00 (reference)
≥ 18m	16.0 (-5.8 to 37.8)	19.5 (-2.5 to 41.5)	17.1 (-4.8 to 39.1)
test of association	P=0.3	P=0.2	`P=0.3
% difference in PAEE age 60-64			
Walking			
≤ 10m	-1.4 (-9.4 to 6.6)	-1.1 (-9.2 to 7.0)	.0.2 (-8.4 to 7.9)
11-17m ≥ 18m	1.00 (reference)	1.00 (reference)	1.00 (reference)
test of association	3.6 (-4.4 to 11.6) P=0.6	4.6 (-3.5 to 12.7) P=0.5	4.0 (-4.1 to 12.1) P=0.6
Analytic sample consists of the			

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without motor milestone terms. **Appendix 3E** Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by teacher-rated ability at games age 13 years: standard binary logistic regression (n=1442).

	LTPA at lea	ast once per month vers	us no LTPA
	Model 1	Model 2	Model 3
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Ability at games age 13			
years			
LTPA age 36			
Above average	1.48 (1.09 to 2.01)	1.46 (1.08 to 1.99)	1.48 (1.09 to 2.01)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.78 (0.56 to 1.08)	0.79 (0.57 to 1.10)	0.77 (0.55 to 1.08)
test of association	p=0.005	p=0.008	p=0.006
LTPA age 43			
Above average	1.45 (1.10 to 1.90)	1.43 (1.08 to 1.89)	1.46 (1.10 to 1.94
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.77 (0.56 to 1.06)	0.75 (0.54 to 1.05)	0.75 (0.54 to 1.04
test of association	p=0.003	p=0.003	p=0003
LTPA age 53			
Above average	1.38 (1.05 to 1.83)	1.37 (1.03 to 1.81)	1.38 (1.04 to 1.84
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.97 (0.70 to 1.33)	0.97 (0.70 to 1.35)	0.95 (0.68 to 1.32
est of association	p=0.06	p=0.07	p=0.06
LTPA age 60-64			
Above average	2.08 (1.58 to 2.73)	2.08 (0.80 to 0.96)	2.15 (1.62 to 2.84
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	1.06 (0.76 to 1.48)	1.06 (0.75 to 1.49)	1.06 (0.76 to 1.50
test of association	p<0.001	p<0.001	p<0.001
LTPA age 68			
Above average	1.45 (1.11 to 1.91)	1.44 (1.10 to 1.89)	1.47 (1.11 to 1.94)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	1.03 (0.75 to 1.43)	1.02 (0.74 to 1.42)	1.02 (0.73 to 1.42)
test of association	p=0.03	p=0.03	p=0.02

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Above average (n=269), Average (n=991), below average (n=182). Tests of association based on likelihood ratio tests comparing models with and without term for ability at games.

	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA			RRR (95% CI) of regular LTPA (5 or more times per month) versus no LTPA		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Ability at games age 13 years						
LTPA age 36						
Above average	1.11	1.09	1.10	1.75	1.72	1.74
	(0.76 to 1.62)	(0.75 to 1.60)	(0.75 to 1.62)	(1.27 to 2.42)	(1.24 to 2.39)	(1.25 to 2.42)
Average	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
Below average	0.93	0.95	0.95	0.68	0.68	0.65
	(0.63 to 1.38)	(0.64 to 1.41)	(0.63 to 1.41)	(0.46 to 0.99)	(0.46 to 0.99)	(0.44 to 0.97)
LTPA age 43						
Above average	0.98	0.97	1.00	1.95	1.95	1.97
	(0.69 to 1.40)	(0.68 to 1.39)	(0.69 to 1.43)	(1.43 to 2.67)	(1.42 to 2.68)	(1.43 to 2.71)
Average	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
Below average	0.72	0.72	0.73	0.82	0.79	0.77
	(0.48 to 1.08)	(0.48 to 1.08)	(0.49 to 1.11)	(0.56 to 1.22)	(0.53 to 1.18)	(051 to 1.15)
LTPA age 53						
Above average	1.32	1.33	1.32	1.42	1.39	1.43
	(0.92 to 1.89)	(0.92 to 1.91)	(0.91 to 1.91)	(1.05 to 1.93)	(1.02 to 1.90)	(1.04 to 1.95)
Average	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
Below average	0.97	0.96	0.92	0.96	0.98	0.97
	(0.64 to 1.49)	(0.63 to 1.48)	(0.59 to 1.41)	(0.67 to 1.38)	(0.68 to 1.42)	(0.67 to 1.41)

Appendix 3F Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by ability at school games: standard multinomial logistic regression (n=1442).

	(0.57 to 1.62)	(0.54 to 1.54)	(0.55 to 1.57)	(0.72 to 1.49)	(0.72 to 1.51)	(0.71 to 1.50)
Below average	(reference) 0.96	(reference) 0.91	(reference) 0.93	(reference) 1.04	(reference) 1.05	(reference) 1.03
Average	1.00	1.00	1.00	1.00	1.00	1.00
_	(0.99 to 2.23)	(1.01 to 2.28)	(1.04 to 2.36)	(1.07 to 1.95)	(1.05 to 1.92)	(1.06 to 1.96)
<i>LTPA age 68</i> Above average	1.48	1.52	1.57	1.45	1.42	1.44
Below average	0.84 (0.51 to 1.41)	0.84 (0.50 to 1.41)	0.86 (0.51 to 1.45)	1.20 (0.81 to 2.87)	1.20 (0.82 to 1.77)	1.20 (0.81 to 1.78)
Average	(1.42 to 2.97) 1.00 (reference)	(1.47 to 3.11) 1.00 (reference)	(1.51 to 3.20) 1.00 (reference)	(1.52 to 2.87) 1.00 (reference)	(1.49 to 2.82) 1.00 (reference)	(1.53 to 2.93) 1.00 (reference)
<i>LTPA age 60-64</i> Above average	2.06	2.14	2.19	2.09	2.05	2.12

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Above average (n=269), Average (n=991), below average (n=182). P-values for tests of association: Age 36 Model 1 <0.001, Model 2 <0.001, Model 3 <0.001. Age 43 Model 1 <0.001, Model 2 <0.001, Model 3 <0.001. Age 53 Model 1=0.2, Model 2=0.3, Model 3=0.2. Age 60-64 Model 1 p<0.001, Model 2p<0.001, Model 3p<0.001. Age 68 Model 1=0.1, Model 2=0.1, Model 3=0.09. Tests of association based on likelihood ratio tests comparing models with and without term for ability at games.

Appendix 3G OR of LTPA types and leisure-time walking at age 36 by games ability:

standard logistic regression.

Games ability			
	Model 1	Model 2	Model 3
OR of team sports versus none at			
age 36 (n=2784)			
Above average	1.92 (1.56 to 2.37)	1.92 (1.56 to 2.37)	1.94 (1.56 to 2.40)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.67 (0.51 to 0.89)	0.68 (0.51 to 0.89)	0.68 (0.51 to 0.90)
test of association	P<0.001	P<0.001	P<0.001
OR of non-team sports versus none at age 36 (n=2756)			
Above average	1.33 (1.08 to 1.62)	1.32 (1.09 to 1.62)	1.33 (1.08 to 1.63)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.64 (0.51 to 0.79)	0.65 (0.52 to 0.81)	0.66 (0.53 to 0.82)
test of association	P<0.001	P<0.001	P<0.001
OR of leisure-time walking versus			
none at age 36 (n=2777)			
Above average	1.10 (0.90 to 1.36)	1.09 (0.89 to 1.34)	1.10 (0.89 to 1.35)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	1.09 (0.87 to 1.37)	1.13 (0.90 to 1.42)	1.14 (0.91 to 1.44)
test of association	p=0.6	p=0.5	p=0.4
Model 1: adjusted for sex. Mo	del 2: adjusted for s	ex, birth weight, birt	h order and

Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without ability at games.

Appendix 3H OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by games ability: standard linear regression – comparable sample.

Games ability n=1424			
	Model 1	Model 2	Model 3
OR of LTPA at least once per			
month versus none at age 60-64			
Above average	1.95 (1.48 to 2.56)	1.96 (1.49 to 2.59)	2.03 (1.54 to 2.69)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	0.98 (0.70 to 1.37)	0.98 (0.70 to 1.38)	0.98 (0.69 to 1.38)
test of association	P<0.001	P<0.001	P<0.001
% difference in MVPA age 60-64			
Above average	9.0 (-6.5 to 24.4)	9.1 (-6.4 to 24.6)	9.8 (-5.7 to 25.3)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	-6.2 (-24.4 to 12.0)	-4.8 (-23.1 to 13.5)	-5.2 (-23.5 to 13.1)
test of association	p=0.3	p=0.4	p=0.3
% difference in PAEE age 60-64			
Above average	1.3 (-4.3 to 6.9)	1.3 (-4.3 to 6.9)	1.4 (-4.1 to 7.0)
Average	1.00 (reference)	1.00 (reference)	1.00 (reference)
Below average	-4.3 (-10.9 to 2.3)	-4.0 (-10.6 to 2.6)	-4.2 (-10.8 to 2.4)
test of association	p=0.3	p=0.4	p=0.3

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without ability at games.

Appendix 3I Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood per 10-unit higher finger and foottapping scores at age 15 years: standard binary logistic regression (n=1347).

	LTPA at least once per month versus no LTPA			
	Model 1	Model 2	Model 3	
	OR (95% CI)	OR (95% CI)	OR (95% CI)	
<u>Tapping speed (per 10-unit</u>				
<u>higher number of taps)</u>				
LTPA age 36				
finger-tapping	1.09 (1.02 to 1.16)	1.09 (1.02 to 1.16)	1.08 (1.01 to 1.15)	
test of association	p=0.008	p=0.02	p=0.03	
foot-tapping	1.10 (1.02 to 1.18)	1.10 (1.02 to 1.18)	1.09 (1.01 to 1.18)	
test of association	p=0.01	p=0.02	p=0.02	
LTPA age 43				

finger-tapping test of association foot-tapping test of association	1.06 (0.999 to 1.13) p=0.06 1.08 (1.01 to 1.16) p=0.02	1.05 (0.99 to 1.19) p=0.09 1.07 (1.01 to 1.15) p=0.04	1.04 (0.98 to 1.10) p=0.2 1.07 (0.998 to 1.15) p=0.06
LTPA age 53 finger-tapping test of association foot-tapping test of association	1.06 (1.001 to 1.13) p=0.05 1.08 (1.01 to 1.16) p=0.03	1.06 (0.996 to 1.12) p=0.07 1.08 (1.005 to 1.15) p=0.04	1.05 (0.99 to 1.11) p=0.1 1.07 (0.996 to 1.14) p=0.07
LTPA age 60-64 finger-tapping test of association foot-tapping test of association	1.10 (1.04 to 1.17) p=0.002 1.14 (1.06 to 1.22) p<0.001	1.10 (1.03 to 1.17) p=0.003 2.14 (1.07 to 1.23) p<0.001	1.09 (1.02 to 1.16) p=0.007 1.14 (1.06 to 1.22) p<0.001
LTPA age 68 finger-tapping test of association foot-tapping test of association	1.11 (1.05 to 1.18) p=0.001 1.12 (1.04 to 1.20) p=0.001	1.11 (1.04 to 1.18) p=0.001 1.12 (1.04 to 1.20) p=0.001	p=0.003

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without tapping speed.

	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA		RRR (95% CI) of regular LTPA (5 or more times per month) versus no LTPA			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Tapping speed (per 10-unit higher number of taps)						
LTPA age 36						
finger-tapping	1.07	1.07	1.06	1.11	1.10	1.09
foot-tapping	(0.99 to 1.15) 1.06	(0.99 to 1.15) 1.06	(0.98 to 1.15) 1.06	(1.03 to 1.19) 1.12	(1.03 to 1.18) 1.12	(1.02 to 1.18) 1.11
Toot-tapping	(0.97 to 1.16)	(0.97 to 1.16)	(0.97 to 1.16)	(1.03 to 1.21)	(1.03 to 1.21)	(1.02 to 1.20)
LTPA age 43						
finger-tapping	1.05	1.04	1.03	1.07	1.06	1.06
foot-tapping	(0.97 to 1.13) 1.05	(0.97 to 1.12) 1.05	(0.96 to 1.11) 1.04	(0.99 to 1.15) 1.11	(0.99 to 114) 1.11	(0.98 to 1.14) 1.11
Tool-tapping	(0.97 to 1.14)	(0.96 to 1.14)	(0.95 to 1.13)	(1.03 to 1.21)	(1.02 to 1.20)	(1.01 to 1.20)
LTPA age 53						
finger-tapping	1.04	1.03	1.02	1.08	1.08	1.06
fact topping	(0.96 to 1.12)	(0.95 to 1.12) 1.05	(0.94 to 1.11) 1.04	(1.01 to 1.15) 1.09	(1.00 to 1.15) 1.09	(0.99 to 1.14)
foot-tapping	1.05 (0.96 to 1.15)	(0.96 to 1.15)	(0.95 to 1.14)	(1.01 to 1.18)	(1.01 to 1.18)	1.08 (1.00 to 1.17)
LTPA age 60-64						
finger-tapping	1.15	1.14	1.14	1.08	1.08	1.06
	(1.05 to 1.25)	(1.04 to 1.25)	(1.04 to 1.25)	(1.00 to 1.16)	(1.00 to 1.16)	(0.99 to 1.15)
foot-tapping	1.15	1.15	1.16	1.14	1.14	1.13

Appendix 3J Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by tapping speed: standard multinomial logistic regression (n=1347).

	(1.05 to 1.27)	(1.05 to 1.27)	(1.05 to 1.28)	(1.05 to 1.24)	(1.05 to 1.24)	(1.04 to 1.23)
LTPA age 68						
finger-tapping	1.09	1.09	1.09	1.12	1.12	1.11
	(0.99 to 1.20)	(0.99 to 1.20)	(0.99 to 1.19)	(1.04 to 1.20)	(1.04 to 1.20)	(1.03 to 1.19)
foot-tapping	1.11	1.11	1.11	1.12	1.12	1.12
	(1.00 to 1.23)	(1.00 to 1.23)	(0.99 to 1.23)	(1.04 to 1.21)	(1.04 to 1.21)	(1.03 to 1.21)

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. P-values for tests of association: Age 36 a. finger-tapping Model 1=0.02, Model 2=0.03, Model 3=0.05. b. foot-tapping Model 1=0.02, Model 2=0.02, Model 3=0.04. Age 43 a. finger-tapping Model 1=0.1, Model 2=0.2, Model 3=0.3. b. foot-tapping Model 1=0.03, Model 2=0.08, Model 3 <0.001. Age 53 a. finger-tapping Model 1=0.09, Model 2=0.1, Model 3=0.2. b. foot-tapping Model 1=0.06, Model 2=0.08, Model 3=0.1. Age 60-64 a. finger-tapping Model 1=0.003, Model 2=0.004, Model 3=0.009. b. foot-tapping Model 1 <0.001, Model 2 <0.001, Model 3=0.001. Age 68 a. finger-tapping Model 1=0.003, Model 2=0.004, Model 3=0.09. b. foot-tapping Model 1 <0.001, Model 2 <0.001, Model 3=0.001. Age 68 a. finger-tapping Model 1=0.003, Model 2=0.004, Model 3=0.09. b. foot-tapping Model 1 <0.001, Model 2 <0.001, Model 3=0.001. Age 68 a. finger-tapping Model 1=0.003, Model 2=0.004, Model 3=0.09. b. foot-tapping Model 1 <0.001, Model 2 <0.001, Model 3=0.001. Age 68 a. finger-tapping Model 1=0.003, Model 2=0.004, Model 3=0.09. b. foot-tapping Model 1 <0.001, Model 2 <0.006, Model 3=0.001. Tests of association based on likelihood ratio tests comparing models with and without finger-tapping/foot-tapping speed terms.

Appendix 3K OR of LTPA types and leisure-time walking at age 36 by tapping

speed: standard logistic regression.

Tapping speed			
	Model 1	Model 2	Model 3
OR of team sports versus none at			
age 36 (n=2584)			
finger-tapping	1.08 (1.03 to 1.14)	1.08 (1.022 to 1.13)	1.07 (1.01 to 1.12)
test of association	p=0.003	p=0.005	p=0.01
foot-tapping	1.09 (1.03 to 1.15)	1.09 (1.03 to 1.15)	1.08 (1.02 to 1.15)
test of association	p=0.003	p=0.004	p=0.007
OB of non toom onorto vorovo			
OR of non-team sports versus $(n-2550)$			
none at age 36 (n=2559) finger-tapping	1.07 (1.02 to 1.12)	1.06 (1.02 to 1.11)	1.06 (1.01 to 1.11)
test of association	p=0.004	p=0.007	p=0.02
foot-tapping	1.07 (1.02 to 1.13)	1.07 (1.02 to 1.12)	1.06 (1.01 to 1.12)
test of association	p=0.005	p=0.009	p=0.02
	μ=0.005	p=0.009	p=0.02
OR of leisure-time walking versus			
none at age 36 (n=2577)			
finger-tapping	1.06 (1.01 to 1.11)	1.06 (1.01 to 1.11)	1.05 (1.00 to 1.10)
test of association	p=0.01	p=0.02	p=0.04
foot-tapping	1.05 (1.00 to 1.11)	1.05 (1.00 to 1.11)	1.04 (0.99 to 1.10)
test of association	p=0.05	p=0.08	p=0.1
Model 1: adjusted for sex. Me	odel 2: adjusted for s	sex, birth weight, birth	order and

Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without tapping speed.

Appendix 3L OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by tapping speed: standard linear regression – comparable sample.

Tapping speed n=1326

	Model 1	Model 2	Model 3
OR of LTPA at least once per			
month versus none at age 60-64			
finger-tapping	1.10 (1.03 to 1.17)	1.10 (1.03 to 1.17)	1.09 (1.02 to 1.16)
test of association	P=0.003	P=0.005	P=0.01
foot-tapping	1.14 (1.06 1.22)	1.13 (1.06 to 1.22)	1.13 (1.05 to 1.21)
test of association	P<0.001	P<0.001	0.001
% difference in MVPA age 60-64			
finger-tapping	2.4 (-1.0 to 5.9)	2.2 (-1.3 to 5.7)	1.8 (-1.7 to 5.3)
test of association	P=0.2	P=0.2	P=0.3
foot-tapping	2.8 (-1.1 to 6.6)	2.6 (-1.3 to 6.4)	2.2 (-1.7 to 6.0)
test of association	p=0.2	p=0.2	p=0.3
% difference in PAEE age 60-64			
finger-tapping	0.4 (-0.9 to 1.7)	0.3 (-0.91.6)	0.2 (-1.1 to 1.5)
test of association	P=0.5	P=0.6	P=0.7
foot-tapping	0.5 (-0.9 1.9)	0.4 (-1.0 to 1.8)	0.3 (-1.1 to 1.7)
test of association	p=0.5	p=0.6	p=0.7

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64. Model 1: adjusted for sex. Model 2: adjusted for sex, birth weight, birth order and serious childhood illness. Model 3: model 2 plus adjustments for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without tapping speed.

	LTPA at leas	t once per month v	ersus no LTPA
	Model 1	Model 2	Model 3
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Pubertal status - boys			
LTPA age 36			
fully mature	1.00	1.00	1.00
	(reference)	(reference)	(reference)
advanced puberty	0.65	0.66	0.65
	(0.41 to 1.04)	(0.41 to 1.06)	(0.41 to 1.05)
early puberty	0.77	0.77	0.77
n ron uh co cont	(0.48 to 1.23)	(0.48 to 1.24)	(0.48 to 1.24)
prepubescent	0.79	0.79 (0.41 to 1.51)	0.80
test of association	(0.41 to 1.51) p=0.3	p=0.4	(0.42 to 1.55) p=0.4
	ρ=0.5	p=0.4	p=0.4
LTPA age 43			
fully mature	1.00	1.00	1.00
	(reference)	(reference)	(reference)
advanced puberty	0.69	0.70	0.69
	(0.45 to 1.05)	(0.46 to 1.08)	(0.44 to 1.07)
early puberty	0.69	0.71	0.71
	(0.45 to 1.05)	(0.46 to 1.09)	(0.46 to 1.09)
prepubescent	0.72	0.74	0.77
	(0.40 to 1.29)	(0.41 to 1.33)	(0.42 to 1.41)
test of association	p=0.3	p=0.4	p=0.3
LTPA age 53			
fully mature	1.00	1.00	1.00
-	(reference)	(reference)	(reference)
advanced puberty	0.89	0.91	0.91
	(0.58 to 1.36)	(0.59 to 1.39)	(0.59 to 1.41)
early puberty	0.97	0.997	0.98
	(0.64 to 1.47)	(0.65 to 1.52)	(0.64 to 1.50)
prepubescent	0.78	0.79	0.82
test of secondistion	(0.44 to 1.39)	(0.44 to 1.41)	(0.46 to 1.49)
test of association	p=0.8	p=0.8	p=0.9
LTPA age 60-64			
fully mature	1.00	1.00	1.00
	(reference)	(reference)	(reference)
advanced puberty	1.05	1.07	1.07
	(0.68 to 1.62)	(0.70 to 1.66)	(0.69 to 1.66)
early puberty	0.88	0.90	0.89
	(0.57 to 1.35)	(0.58 to 1.38)	(0.57 to 1.37)
prepubescent	0.92	0.93	0.96
tast of association	(0.50 to 1.69)	(0.51 to 1.71)	(0.52 to 1.79)
test of association	p=0.8	p=0.8	p=0.8
LTPA age 68			

Appendix 4A Odds ratios (OR) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by boys' pubertal status: standard binary logistic regression (n=636).

fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)
advanced puberty	0.78	1.04	0.76
	(0.51 to 1.19)	(0.73 to 1.48)	(0.49 to 1.16)
early puberty	0.83	1.03	0.80
	(0.55 to 1.25)	(0.73 to 1.46)	(0.52 to 1.21)
prepubescent	0.84	<u>`</u> 1.01	0.84
	(0.47 to 1.50)	(0.62 to 1.66)	(0.46 to 1.52)
test of association	р=0.7	р=0.6	р=0.6

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Fully mature N=156, advanced puberty N=195, early puberty N=219, prepubescent N=66. Tests of association based on likelihood ratio tests comparing models with and without term for pubertal status.

	RRR (95% CI) o	f moderate LTPA (1- versus no LTPA	4 times per month)	· · · · · · · · · · · · · · · · · · ·	of regular LTPA (nonth) versus no l	
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
<u>Pubertal status</u> Age 36						
fully mature	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
advanced puberty	0.71	0.72	0.69	0.65	0.67	0.67
	(0.40 to 1.24)	(0.41 to 1.26)	(0.39 to 1.22)	(0.39 to 1.10)	(0.40 to 1.13)	(0.40 to 1.13)
early puberty	0.67	0.68	0.68	0.85	0.86	0.86
	(0.38 to 1.17)	(0.39 to 1.20)	(0.38 to 1.20)	(0.52 to 1.41)	(0.52 to 1.43)	(0.51 to 1.43)
prepubescent	0.72	0.73	0.74	0.84	0.85	0.87
	(0.33 to 1.58)	(0.33 to 1.59)	(0.33 to 1.63)	(0.42 to 1.70)	(0.42 to 1.72)	(0.43 to 1.76)
Age 43						
fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
advanced puberty	0.69	0.71	0.71	0.68	0.69	0.67
	(0.40 to 1.16)	(0.42 to 1.22)	(0.41 to 1.22)	(0.41 to 1.11)	(0.42 to 1.14)	(0.40 to 1.12
early puberty	0.66	0.69	0.68	0.73	0.74	0.74
	(0.39 to 1.11)	(0.41 to 1.17)	(0.40 to 1.16)	(0.45 to 1.18)	(0.45 to 1.21)	(0.45 to 1.22
prepubescent	0.91	0.96	1.01	0.58	0.58	0.61
	(0.46 to 1.82)	0.48 to 1.93)	0.50 to 2.06)	(0.29 to 1.19)	(0.28 to 1.20)	(0.29 to 1.26
<u>Age 53</u>						
fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)
advanced puberty	0.98	0.98	0.99	0.82	0.84	0.85
	(0.57 to 1.69)	(0.56 to 1.70)	(0.57 to 1.72)	(0.51 to 1.33)	(0.51 to 1.36)	(0.52 to 1.39
early puberty	<u></u> 1.04	<u></u> 1.05	<u> </u>	0.94	0.96	0.94

Appendix 4B Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by men's pubertal stage at the age of 15: standard multinomial logistic regression (n=636).

	(0.61 to 1.78)	(0.61 to 1.80)	(0.61 to 1.83)	(0.59 to 1.50)	(0.59 to 1.54)	(0.58 to 1.51)
prepubescent	0.71	0.70	0.74	0.83	0.83	0.88
	(0.32 to 1.55)	(0.32 to 1.54)	(0.33 to 1.64)	(0.44 to 1.59)	(0.43 to 1.60)	(0.46 to 1.71)
Age 60-64						
fully mature	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
advanced puberty	1.00	1.04	0.99	1.10	1.13	1.15
	(0.54 to 1.85)	(0.56 to 1.93)	(0.53 to 1.85)	(0.66 to 1.84)	(0.67 to 1.90)	(0.68 to 1.94)
early puberty	0.88	0.90	0.90	0.90	0.92	0.90
	(0.48 to 1.61)	(0.49 to 1.66)	(0.49 to 1.68)	(0.54 to 1.50)	(0.55 to 1.54)	(0.54 to 1.52)
prepubescent	1.25	1.31	1.27	0.73	0.73	0.79
	(0.57 to 2.77)	(0.59 to 2.91)	(0.57 to 2.85)	(0.34 to 1.58)	(0.34 to 1.59)	(0.36 to 1.73)
Age 68						
fully mature	1.00	1.00 (reference)	1.00	1.00	1.00	1.00
	(reference)		(reference)	(reference)	(reference)	(reference)
advanced puberty	0.48	0.45	0.43	0.95	0.94	0.94
	(0.25 to 0.92)	(0.23 to 0.88)	(0.22 to 0.84)	(0.59 to 1.54)	(0.58 to 1.53)	(0.58 to 1.53)
early puberty	0.56	0.53	0.51	1.02	0.99	0.98
	(0.30 to 1.05)	(0.28 to 0.99)	(0.27 to 0.97)	(0.63 to 1.63)	(0.61 to 1.59)	(0.61 to 1.59)
prepubescent	0.65	0.62	` 0.60 ́	0.97	0.96	`
	(0.27 to 1.57)	(0.26 to 1.51)	(0.25 to 1.47)	(0.50 to 1.88)	(0.50 to 1.86)	(0.51 to 1.95)

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Fully mature N=156, advanced puberty N=195, early puberty N=219, prepubescent N=66. P-values for tests of association: Age 36 Model 1=0.6, Model 2=0.7, Model 3=0.7. Age 43 Model 1=0.4, Model 2=0.5, Model 3=0.5. Age 53 Model 1=0.9, Model 2=0.9, Model 3>0.9. Age 60-64 Model 1=0.9, Model 2=0.8, Model 3=0.9. Age 68 Model 1=0.4, Model 2=0.4, Model 3=0.3. Tests of association based on likelihood ratio tests comparing models with and without pubertal status term.

Appendix 4C OR of LTPA types and leisure-time walking at age 36 by pubertal

status at age 15 in boys: standard logistic regression.

	Model 1	Model 2	Model 3
OR of team sports versus none at			
age 36 (n=801)			
Fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)
Advance puberty	0.92 (0.62 to 1.35)	0.94 (0.63 to 1.39)	0.93 (0.63 to 1.38
Early puberty	0.77 (0.53 to 1.13)	0.79 (0.54 to 1.16)	0.80 (0.54 to 1.18
prepubescent	0.83 (0.49 to 1.42)	0.86 (0.50 to 1.48)	0.89 (0.51 to 1.53
test of association	p=0.6	p=0.6	p=0.7
OR of non-team sports versus			
none at age 36 (n=794)			
Fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)
Advance puberty	0.65 (0.44 to 0.95)	0.65 (0.44 to 0.96)	0.65 (0.44 to 0.96
Early puberty	0.82 (0.56 to 1.19)	0.83 (0.57 to 1.22)	0.84 (0.57 to 1.23
prepubescent	0.72 (0.42 to 1.21)	0.73 (0.43 to 1.24)	0.75 (0.44 to 1.28
test of association	p=0.2	p=0.2	p=0.2
OR of leisure-time walking versus			
none at age 36 (n=800)			
Fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)
Advance puberty	0.74 (0.49 to 1.10)	0.76 (0.50 to 1.14)	0.76 (0.51 to 1.14
Early puberty	0.86 (0.58 to 1.27)	0.88 (0.59 to 1.32)	0.89 (0.59 to 1.33
prepubescent	0.79 (0.46 to 1.36)	0.82 (0.47 to 1.42)	0.84 (0.48 to 1.45
test of association	p=0.5	p=0.6	`p=0.6

for model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without term for pubertal status.

Appendix 4D OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by pubertal status at age 15 in boys: standard linear regression - comparable sample.

Dubortal status n-666							
Pubertal status n=666	NA dal 4	Madalo	Ma dal O				
	Model 1	Model 2	Model 3				
OR of LTPA at least once per							
month versus none at age 60-64							
fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)				
advanced puberty	1.01 (0.66 1.55)	1.03 (0.67 to 1.59)	1.08 (0.70 to 1.66)				
early puberty	0.97 (0.64 to 1.47)	1.01 (0.66 to 1.54)	1.05 (0.69 to 1.62)				
prepubescent	0.76 (0.42 to 1.38)	0.79 (0.43 to 1.44)	0.83 (0.45 to 1.52)				
test of association	P=0.8	p=0.8	p=0.8				
% difference in MVPA age 60-64							
fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)				
advanced puberty	-7.6 (-30.7 to 15.5)	-6.9 (-30.1 to 16.2)	-6.0 (-29.2 to 17.2)				
early puberty	10.3 (-12.5 to 33.0)	12.6 (-10.3 to 35.4)	13.0 (-10.0 to 35.9)				
prepubescent	25.5 (-5.9 to 57.0)	26.7 (-4.7 to 58.2)	27.7 (-3.8 to 59.2)				
test of association	p=0.1	p=0.1	p=0.1				
% difference in PAEE age 60-64							
•	1.00 (reference)	1.00 (reference)	1.00 (reference)				
fully mature	1.00 (reference)	1.00 (reference)	1.00 (reference)				
advanced puberty	-2.0 (-10.8 to 6.7)	-0.2 (-10.4 to 7.2)	-1.5 (-10.3 to 7.4)				
early puberty	2.7 (-6.0 to 11.3)	3.6 (-5.1 to 12.4)	3.5 (-5.2 to 12.2)				
prepubescent	4.7 (-7.3 to 16.6)	5.3 (-6.7 to 17.3)	5.3 (-6.7 to 17.4)				
test of association	p=0.6	p=0.5	p=0.5				
Analytic sample consists of	Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64.						

Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without term for pubertal status.

	I TPA at least	once per month ve	
	Model 1	Model 2	Model 3
	OR (95% CI)	OR (95% CI)	OR (95% CI)
Menarche (years)			
<i>LTPA age 36</i> ≤11	1.00	1.00	1.00
211	(reference)	(reference)	(reference)
12	0.81	0.82	0.90
	(0.51 to 1.30)	(0.51 to 1.32)	(0.56 to 1.46)
13	0.98	0.99	1.06
N14	(0.61 to 1.52)	(0.62 to 1.56)	(0.66 to 1.69)
≥14	0.98 (0.60 to 1.68)	1.04 (0.61 to 1.78)	1.10 (0.64 to 1.87)
test of association	p=0.7	p=0.7	p=0.8
	μ σ	P 011	P 010
LTPA age 43			
≤11	1.00	1.00	1.00
12	(reference) 1.17	(reference) 1.17	(reference) 1.28
12	(0.74 to 1.84)	(0.74 to 1.86)	(0.80 to 2.05)
13	1.05	1.09	1.14
	(0.67 to 1.62)	(0.70 to 1.70)	(0.73 to 1.79)
≥14	1.22	1.28	1.33
	(0.74 to 2.00)	(0.77 to 2.12)	(0.80 to 2.23)
test of association	p=0.8	p=0.8	p=0.7
LTPA age 53			
≤11	1.00	1.00	1.00
	(reference)	(reference)	(reference)
12	1.03	1.06	1.19
13	(0.65 to 1.62) 0.85	(0.67 to 1.68) 0.91	(0.74 to 1.90) 0.98
10	(0.55 to 1.32)	(0.58 to 1.42)	(0.62 to 1.54)
≥14	1.19	1.25	1.34
	(0.72 to 1.96)	(0.75 to 2.09)	(0.80 to 2.25)
test of association	p=0.5	p=0.5	p=0.5
1 TPA ago 60 64			
<i>LTPA age 60-64</i> ≤11	1.00	1.00	1.00
	(reference)	(reference)	(reference)
12	1.20	1.24	1.39
	(0.74 to 1.93)	(0.77 to 2.02)	(0.85 to 2.27)
13	1.33	1.42	1.52
≥14	(0.84 to 2.10) 1.07	(0.89 to 2.28) 1.14	(0.94 to 2.46) 1.20
<u>-</u> 17	(0.63 to 1.80)	(0.67 to 1.95)	(0.69 to 2.06)
test of association	p=0.6	p=0.5	p=0.3
	·		
LTPA age 68	4.00	4.00	4.00
≤11	1.00 (reference)	1.00 (reference)	1.00 (reference)
12	(reference) 0.92	(reference) 0.93	(reference) 1.05
16	0.02	0.35	1.00

Appendix 4E Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by age at menarche: standard binary logistic regression models (n=726).

	(0.58 to 1.46)	(0.59 to 1.48)	(0.65 to 1.68)
13	0.93	0.93	0.995
	(0.60 to 1.44)	(0.59 to 1.45)	(0.63 to 1.57)
≥14	0.99	1.00	1.07
	(0.60 to 1.64)	(0.60 to 1.67)	(0.64 to 1.79)
test of association	p=0.9	p=0.9	p=0.8

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Fully mature N=156, advanced puberty N=195, early puberty N=219, prepubescent N=66. \leq 11 years (n=117), 12 years (n=211), 13 years (n=264), \geq 14 years (n=134). Tests of association based on likelihood ratio tests comparing models with and without age at menarche.

Appendix 4F Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by age at menarche: standard multinomial logistic regression (n=726).

	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA		RRR (95% CI) of regular LTPA (5 or more times per month) versus no LTPA			
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Menarche (years)						
LTPA age 36						
≤11	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12	0.71	0.72	0.76	0.90	0.90	1.02
	(0.40 to 1.26)	(0.40 to 1.28)	(0.42 to 1.37)	(0.53 to 1.52)	(0.53 to 1.54)	(0.59 to 1.76)
13	0.87	0.87	0.92	1.07	1.11	1.21
	(0.50 to 1.52)	(0.50 to 1.53)	(0.52 to 1.62)	(0.64 to 1.79)	(0.66 to 1.86)	(0.71 to 2.05)
≥14	0.95	0.98	1.03	1.00	1.03	1.09
	(0.51 to 1.79)	(0.52 to 1.86)	(0.54 to 1.95)	(0.56 to 1.80)	(0.57 to 1.86)	(0.59 to 1.99)
LTPA age 43						
≤11 [°]	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12	0.998	1.02	1.09	1.37	1.35	1.53
	(0.57 to 1.74)	(0.58 to 1.79)	(0.62 to 1.93)	(0.77 to 2.44)	(0.76 to 2.42)	(0.84 to 2.76)
13	1.06	1.12	1.14	1.03	1.05	1.13
	(0.62 to 1.80)	(0.65 to 1.91)	(0.66 to 1.97)	(0.58 to1.82)	(0.59 to 1.88)	(0.63 to 2.03)
≥14	1.21	1.30	1.33	1.22	1.24	1.31
	(0.66 to 2.21)	(0.71 to 2.40)	(0.72to 2.47)	(0.64 to 2.33)	(0.65 to 2.40)	(0.67 to 2.54)
LTPA age 53						
≤11	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12	0.71	0.73	0.80	1.26	1.30	1.46
	(0.39 to 1.30)	(0.40 to 1.35)	(0.43 to 1.49)	(0.76 to 2.11)	(0.77 to 2.19)	(0.86 to 2.48)

13	0.67	0.71	0.77	1.01	1.07	1.15
	(0.38 to 1.20)	(0.39 to 1.26)	(0.42 to 1.39)	(0.61 to 1.66)	(0.65 to 1.78)	(0.69 to 1.93)
≥14	0.96	1.03	`	1.30	1.34	<u> </u>
	(0.50 to 1.85)	(0.53 to 2.00)	(0.55 to 2.11)	(0.74 to 2.30)	(0.75 to 2.40)	(0.79 to 2.56)
LTPA age 60-64						
≤11	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12	1.43	1.55	1.76	1.06	1.08	`
	(0.74 to 2.78)	(0.79 to 3.04)	(0.88 to 3.49)	(0.60 to 1.86)	(0.61to 1.91)	(0.67 to 2.13)
13	1.16	1.32	1.43	1.39	1.45	1.54
	(0.59 to 2.25)	(0.67 to 2.61)	(0.72 to 2.85)	(0.81 to 2.37)	(0.84 to 2.50)	(0.89 to 2.67)
≥14	<u></u> 1.27	1.48	1.58	0.92	0.96	0.99
	(0.62 to 2.63)	(0.70 to 3.11)	(0.74 to 3.37)	(0.49 to 1.73)	(0.51 to 1.82)	(0.52 to 1.89)
LTPA age 68						
≤11	1.00	1.00	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)	(reference)	(reference)
12	`0.65 ´	`0.65 ´	0.73	1.09	`	` 1.26 ́
	(0.33 to 1.28)	(0.33 to 1.29)	(0.37 to 1.46)	(0.65 to 1.84)	(0.65 to 1.87)	(0.73 to 2.15)
13	0.84	0.82	` 0.88 ́	0.96	0.97	`
	(0.45 to 1.56)	(0.44 to 1.55)	(0.36 to 1.66)	(0.57 to 1.59)	(0.58 to 1.62)	(0.62 to 1.77)
≥14	0.87	0.86	` 0.90 ́	`	`	`
	(0.43 to 1.78)	(0.42 to 1.79)	(0.43 to 1.88)	(0.57 to 1.81)	(0.58 to 1.88)	(0.61 to 2.02)

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Fully mature N=156, advanced puberty N=195, early puberty N=219, prepubescent N=66. \leq 11 years (n=117), 12 years (n=211), 13 years (n=264), \geq 14 years (n=134). P-values for tests of association: Age 36 Model 1 p=0.9, Model 2 p=0.9, Model 3 p=0.9. Age 43 Model 1 p=0.8, Model 2 p=0.8, Model 3 p=0.7. Age 53 Model 1 p=0.5, Model 2 p=0.5, Model 3 p=0.5. Age 60-64 Model 1 p=0.5, Model 2 p=0.3. Age 68 Model 1 p=0.9, Model 2 p=0.9, Model 2 p=0.9, Model 2 p=0.5, Model 3 p=0.9. Tests of association based on likelihood ratio tests comparing models with and without age at menarche term.

Appendix 4G OR of LTPA types and leisure-time walking at age 36 by girls' age at menarche: standard logistic regression.

Menarche (years)	Model 1	Model 2	Model 3
OR of team sports versus none at		Model 2	inicaci c
age 36 (n=859)			
≤11	1.00 (reference)	1.00 (reference)	1.00 (reference)
12	0.90 (0.51 to 1.58)	0.92 (0.53 to 1.62)	1.00 (0.56 to 1.78
13	1.53 (0.91 to 2.59)	1.58 (0.93 to 2.67)	1.69 (0.99 to 2.88
≥14	1.59 (0.89 to 2.82)	1.63 (0.91 to 2.92)	1.70 (.95 to 3.07)
test of association	p=0.03	p=0.03	p=0.03
OR of non-team sports versus			
none at age 36 (n=855)			
≤11	1.00 (reference)	1.00 (reference)	1.00 (reference)
12	0.79 (0.52 to 1.20)	0.82 (0.54 to 1.25)	0.86 (0.56 to 1.32
13	0.87 (0.58 to 1.30)	0.88 (0.58 to 1.33)	0.93 (0.61 to 1.41
≥14	0.88 (0.56 to 1.39)	0.89 (0.56 to 1.42)	0.93 (0.58 to 1.49
test of association	p=0.7	p=0.8	p=0.9
OR of leisure-time walking versus			
none at age 36 (n=856)			
≤11	1.00 (reference)	1.00 (reference)	1.00 (reference)
12	0.83 (0.52 to 1.33)	0.89 (0.55 to 1.43)	0.95 (0.59 to 1.53
13	1.02 (0.64 to 1.62)	1.11 (0.69 to 1.78)	1.16 (0.72 to 1.86
≥14	0.65 (0.39 to 1.07)	0.71 (0.43 to 1.18)	0.72 (0.43 to 1.20
test of association	p=0.2	p=0.2	p=0.2

Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without term for age at menarche.

Appendix 4H OR of LTPA and % difference in monitored PAEE and MVPA (95% confidence intervals) at age 60-64 by girls' age at menarche: standard linear regression – comparable sample.

	Model 1	Model 2	Model 3
OR of LTPA at least once per			
month versus none at age 60-64			
≤11 ³	1.00 (reference)	1.00 (reference)	1.00 (reference)
12	1.18 (0.73 to 1.89)	1.28 (0.79 to 2.07)	1.37 (0.84 to 2.24)
13	1.18 (0.74 to 1.87)	1.31 (0.82 to 2.10)	1.41 (0.87 to 2.27
≥14	0.97 (0.57 to 1.65)	1.10 (0.64 to 1.88)	1.13 (0.66 to 1.95
est of association	p=0.7	p=0.6	`p=0.5
% difference in MVPA age 60-64			
≦11 °	1.00 (reference)	1.00 (reference)	1.00 (reference)
2	.14.0 (-13.2 to 41.1)	17.0 (-10.3 to 44.4)	19.2 (-8.2 to 46.5
3	8.3 (-18.2 to 34.8)	10.7 (-16.0 to 37.4)	12.8 (-14.0 to 39.5
≥14	9.0 (-21.0 to 39.0)	11.8 (-18.6 to 42.3)	12.3 (-18.1 to 42.6
est of association	p=0.8	p=0.7	p=0.6
% difference in PAEE age 60-64			
s11	1.00 (reference)	1.00 (reference)	1.00 (reference)
2	2.8 (-6.5 to 12.0)	3.5 (-5.9 to 12.9)	4.3 (-5.1 to 13.6)
3	2.5 (-6.5 to 11.6)	3.3 (-5.9 to 12.4)	4.0 (-5.1 to 13.2)
14	4.9 (-5.5 to 15.1)	5.5 (-4.9 to 15.9)	5.6 (-4.7 to 16.0)
est of association	p=0.8	`p=0.7 ′́	`p=0.7

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64 Model 1: unadjusted. Model 2: adjusted for birth weight, and birth order. Model 3: as for model 2 plus adjustment for father's occupational class. Tests of association based on likelihood ratio tests comparing models with and without term for age at menarche. **Appendix 5A** Odds ratios (ORs) and 95% confidence intervals (95% CIs) of leisuretime physical activity at each age in adulthood by father's occupational class at age 4: standard binary logistic regression (n=1282).

LTPA at least once per month versus no LTPA

	·		
	Model 1	Model 2	
Father's occupational class age 4			
LTPA age 36 professional/managerial/technical skilled non-manual skilled manual partly skilled or unskilled overall test of association	1.00 (reference) 1.13 (0.78 to 1.63) 0.67 (0.49 to 0.93) 0.53 (0.38 to 0.74) p<0.001	1.00 (reference) 1.16 (0.80 to 1.69) 0.69 (0.50 to 0.96) 0.55 (0.40 to 0.77) p<0.001	
LTPA age 43 professional/managerial/technical skilled non-manual skilled manual partly skilled or unskilled overall test of association	1.00 (reference) 1.12 (0.81 to 1.57) 0.57 (0.43 to 0.78) 0.57 (0.42 to 0.78) p<0.001	1.00 (reference) 1.14 (0.81 to 1.60) 0.61 (0.45 to 0.82) 0.61 (0.44 to 0.84) p<0.001	
LTPA age 53 professional/managerial/technical skilled non-manual skilled manual partly skilled or unskilled overall test of association	1.00 (reference) 0.80 (0.57 to 1.13) 0.57 (0.42 to 0.77) 0.42 (0.31 to 0.58) p<0.001	1.00 (reference) 0.81 (0.57 to 1.14) 0.60 (0.44 to 0.82) 0.45 (0.32 to 0.62) p<0.001	
LTPA age 60-64 professional/managerial/technical skilled non-manual skilled manual partly skilled or unskilled overall test of association	1.00 (reference) 1.03 (0.74 to 1.43) 0.54 (0.39 to 0.73) 0.59 (0.43 to 0.81) p<0.001	1.00 (reference) 1.07 (0.76 to 1.50) 0.54 (0.39 to 0.74) 0.61 (0.44 to 0.85) p<0.001	
LTPA age 68 professional/managerial/technical skilled non-manual skilled manual partly skilled or unskilled overall test of association	1.00 (reference) 1.14 (0.82 to 1.58) 0.60 (0.45 to 0.81) 0.57 (0.41 to 0.78) p<0.001	1.00 (reference) 1.18 (0.85 to 1.65) 0.62 (0.45 to 0.84) 0.58 (0.42 to 0.80) p<0.001	

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex and age. Model 2: as for model 1 plus adjustment for birth weight, birth order, childhood illness, ability at games and finger-tapping speed. Professional/managerial/technical (n=322), skilled non-manual (n=259), skilled manual (n=378), partly skilled or unskilled (n=323). Tests of association based on likelihood ratio tests comparing models with and without term for father's occupational class.

Appendix 5B Relative risk ratios (RRR) and 95% confidence intervals (95% CI) of moderate and regular leisure-time physical activity at each age in adulthood by father's occupational class at age 4: standard multinomial logistic regression (n=1282).

LTPA between ages 36-68	RRR (95% CI) of moderate LTPA (1-4 times per month) versus no LTPA		RRR (95% CI) of regular LTPA (5 or more times per month) versus no LTPA		
	Model 1	Model 2	Model 1	Model 2	
Father's occupational class age 4					
LTPA age 36					
professional/managerial/technical	1.00 (reference)	1.00 (reference)	1.00 (reference)	1.00 (reference)	
skilled non-manual	(reference) 1.50	(reference) 1.53	(reference)	(reference) 0.94	
Skilled non-manual			0.91 (0.61 to 1.27)		
skilled manual	(0.97 to 2.32) 0.79	(0.99 to 2.38) 0.80	(0.61 to 1.37) 0.61	(0.62 to 1.42) 0.63	
Skileu Manual	(0.53to 1.18)	(0.54 to 1.20)	(0.43 to 0.86)	(0.44 to 0.91)	
partly skilled or unskilled	0.62	0.63	0.48	0.50	
party skilled of unskilled	(0.41 to 0.94)	(0.41 to 0.95)	(0.33 to 0.68)	(0.34 to 0.73)	
LTPA age 43					
professional/managerial/technical	1.00	1.00	1.00	1.00	
	(reference)	(reference)	(reference)	(reference)	
skilled non-manual	1.22	1.20	1.06	`	
	(0.82 to 1.81)	(0.81 to 1.80)	(0.71 to 1.57)	(0.72 to 1.63)	
skilled manual	0.45	0.48	0.67	0.71	
	(0.31 to 0.67)	(0.32 to 0.71)	(0.47 to 0.96)	(0.49 to 1.02)	
partly skilled or unskilled	0.69	0.73	0.48	0.51	
	(0.47 to 1.00)	(0.50 to 1.08)	(0.32to 0.71)	(0.34 to 0.80)	
LTPA age 53					

professional/managerial/technical	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)
skilled non-manual	0.77	0.75	0.84	0.85
	(0.49 to 1.19)	(0.48 to 1.17)	(0.58 to 1.22)	(0.58 to 1.24)
skilled manual	0.71	0.74	0.49	0.52
portly skilled or upskilled	(0.48 to 1.04)	(0.50 to 1.10)	(0.35 to 0.70)	(0.36 to 0.73)
partly skilled or unskilled	0.33	0.35	0.48	0.51
	(0.21 to 0.51)	(0.22 to 0.54)	(0.34 to 0.68)	(0.36 to 0.73)
LTPA age 60-64				
professional/managerial/technical	1.00	1.00	1.00	1.00
	(reference)	(reference)	(reference)	(reference)
skilled non-manual	1.43	1.49	0.86	0.90
	(0.90 to 2.26)	(0.93 to 2.38)	(0.59 to 1.26)	(0.61 to 1.33)
skilled manual	0.65	0.66	0.48	0.48
	(0.41 to 1.02)	(0.41 to 1.05)	(0.34 to 0.69)	(0.33 to 0.70)
partly skilled or unskilled	0.77	0.83	0.52	0.54
	(0.48 to 1.22)	(0.52 to 1.34)	(0.36 to 0.76)	(0.37 to 0.79)
LTPA age 68				
professional/managerial/technical	1.00	1.00	1.00	1.00
1	(reference)	(reference)	(reference)	(reference)
skilled non-manual	`	`	1.07	`
	(0.85 to 2.19)	(0.86 to 2.26)	(0.74 to 1.54)	(0.77 to 1.62)
skilled manual	`	0.55	0.61	`
	(0.34 to 0.89)	(0.33 to 0.90)	(0.44 to 0.85)	(0.45 to 0.89)
partly skilled or unskilled	`	0.74	0.50	0.52
	(0.46 to 1.19)	(0.46 to 1.19)	(0.35 to 0.72)	(0.36 to 0.75)

Analytic sample consists of those with leisure-time physical activity data from all five ages. Model 1: adjusted for sex and age. Model 2: as for model 1 plus adjustment for birth weight, birth order, childhood illness, ability at games and finger-tapping speed. Professional/managerial/technical (n=322), skilled non-manual (n=259), skilled manual (n=378), partly skilled or unskilled: standard linear regression (n=323). P-values for tests of association: Age 36 Model 1 p<0.001, Model 2 p<0.001, Model 3 p<0.001. Age 43 Model 1 p<0.001, Model 2 p<0.001, Model 3 p<0.001. Age 60-64 Model 1 p<0.001, Model 2 p<0.001, Model 3 p<0.001. Age 68 Model 1 p<0.001, Model 2 p<0.001. Tests of association based on likelihood ratio tests comparing models with and without father's occupational class. **Appendix 5C** % difference in monitored MVPA and PAEE and OR of LTPA (95% confidence intervals) at age 60-64 by girls' age at menarche: standard linear regression – comparable sample.

Father's occupational class (n=1268	Model 1	Model 2
OR of LTPA at least once per		
month versus none at age 60-64		
professional/managerial/technical	1.00 (reference)	1.00 (reference)
skilled non-manual	1.20 (0.86 to 1.68)	1.21 (0.86 to 1.71)
skilled manual	0.57 (0.41 to 0.76)	0.56 (0.41 to 0.77)
partly skilled or unskilled	0.58 (0.42 to 0.80)	0.60 (0.43 to 0.84)
test of association	P<0.001	P<0.001
% difference in MVPA age 60-64		
professional/managerial/technical	1.00 (reference)	1.00 (reference)
skilled non-manual	-12.6 (-31.7 to 6.6)	-12.5 (-31.8 to 6.7)
skilled manual	-25.7 (-43.0 to -8.4)	-25.1 (-42.5 to -7.6)
partly skilled or unskilled	-18.9 (-36.8 to -1.0)	-17.5 (-35.5 to 0.6)
est of association	p=0.03	p=0.04
% difference in PAEE age 60-64		
professional/managerial/technical	1.00 (reference)	1.00 (reference)
skilled non-manual	-5.9 (-12.9 to 1.0)	-6.1 (-13.1 to 0.8)
skilled manual	-6.6 (-12.8 to -0.3)	-6.3 (-12.6 to 0.0)
partly skilled or unskilled	-6.6 (-13.1 to 0.2)	-6.3 (-12.8 to 0.3)
test of association	p=0.1	p=0.2

Analytic sample consists of those with data on MVPA, PAEE and LTPA at age 60-64. Model 1: adjusted for sex and age. Model 2: as for model 1 plus adjustment for birth weight, birth order, childhood illness, ability at games and finger-tapping speed. Tests of association based on likelihood ratio tests comparing models with and without term for father's occupational class. Multinomial logistic regression is an extension of binary logistic regression and its estimates are interpreted as the relative risk ratio (RRR) of moderate LTPA (1-4 times/month) versus none and RRR of regular LTPA (≥5 times/month) versus none by each early life factor. As for the ORs of LTPA across adulthood estimated with the binary mixed-effects models, to include all those with at least one measure of LTPA in the analyses, the RRRs of moderate and regular (5 or more times per month) LTPA with those reporting no LTPA as reference group were estimated using mixed-effects multinomial logistic regression models.

Appendix 6 Mixed-effects multinomial logistic regression via MCMC simulation

These models were estimated with Markov Chain Monte Carlo (MCMC) simulation, which is a Bayesian estimation techniques that can be used to estimate complex mixed-effects models. As with Bayesian modelling, additional steps (over and above the classical statistical approach) are required for model estimation (model specification, prior knowledge, initial values). Models were initially specified using iterative generalised least squares and the parameter estimates from these models were specified as initial values. Uninformative prior distributions (prior knowledge) were then used to approximate maximum likelihood estimation. MCMC methods make many simulated random draws from the joint posterior distribution of all the parameters, and use these random draws to form a summary of the underlying distributions. The MCMC algorithm (Gibbs sampling and Metropolis Hasting sampling) is then run until each parameter distribution has settled down to its stationary distribution (the burnin period when the chains are converging to their posterior distribution), followed by a further period (the monitoring period) to store a monitoring chain for each parameter. Point estimates and standard errors are given by the means and standard deviations of these monitoring chains. Further details can be found in chapter 2.3.2.

238

Appendix 7 Exact questions asked and used to derive the LTPA measures at each adult age.

Age 36 – 1982

5. *In your spare time* have you taken part in any of these sports or outdoor activities in the last 4 weeks?

Badminton; bowls; cricket; exercises like press-ups, sit-ups etc. at home; exercises like press-ups, sit-ups etc. at gym; football; golf; hill/mountain climbing; jogging; rowing; running/athletics; sailing; squash/rackets; swimming; table tennis; tennis; yoga; water skiing; volleyball; scuba diving; basketball; fishing; riding; movement to music; weight training; ballroom dancing; other dancing. [Tick next to each activity]

State number of times for each activity in the last month

State total time spent to the nearest hour in the last month

Age 43 – 1989

84. Do you regularly take part in any sports or vigorous leisure activities or do any exercises (things like badminton, swimming, yoga, press-ups, dancing, football, mountain climbing or jogging)? If yes, list activities in the space below

How many months in the year do you do this?

1-3 months a year, 3-6 months a year, 6-11 months a year, all year

How often do you do this?

Less than once a month, less than once a week, once a week, more than once a week

On average how long do you spend doing this?

Does it usually make you sweaty and/or out of breath?

Yes | No

Age 53 – 1999

91a. In the last 4 weeks, have you taken part in any sports or vigorous leisure activities or done any exercise in your spare time, not including getting to and from work? *If asked: include things like badminton, swimming, yoga, press-ups, dancing, mountain climbing or jogging and brisk walks for 30 minutes or more.*

Yes | No

91b. On how many occasions in the last 4 weeks did you do these activities? *Enter a numeric value between 1 and 100.*

91c. On how many of these occasions did your exercise make you sweaty and or out of breath? *Enter a numeric value between 1 and 100*.

Age 60-64 – 2006 – 2010

5a. In the last 4 weeks, in your spare time, have you taken part in any sports or vigorous leisure activities or done any exercises, things like badminton, swimming, yoga, conditioning exercise, floor-based exercises, dancing, hill-walking or jogging?

No | Yes

5b. On how many occasions in the last month did you do these activities?

5c. On how many of these occasions were you sweaty and/or out of breath?

Age 68 – 2014

56a. In the *last 4 weeks*, in your spare time, have you taken part in any sports or vigorous leisure activities or done any exercises, things like badminton, swimming, yoga, conditioning exercise, floor-based exercises, dancing, hill-walking or jogging?

No | Yes

56b. On how many occasions in the last month did you do these activities?

56c. On how many of these occasions were you sweaty and/or out of breath?

References

1. Caspersen CJ, Powell KE, Christenson GM. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. Public Health Rep. 1985;100(2):126-31.

2. Pratt M, Macera CA, Sallis JF, O'Donnell M, Frank LD. Economic interventions to promote physical activity: application of the SLOTH model. Am J Prev Med. 2004;27(3 Suppl):136-45.

3. Howley ET. Type of activity: resistance, aerobic and leisure versus occupational physical activity. Med Sci Sports Exerc. 2001;33.

4. Owen N, Sparling PB, Healy GN, Dunstan DW, Matthews CE. Sedentary behavior: emerging evidence for a new health risk. Mayo Clinic proceedings. 2010;85(12):1138-41.

5. Katzmarzyk PT. Physical activity, sedentary behavior, and health: paradigm paralysis or paradigm shift? Diabetes. 2010;59(11):2717-25.

6. Pearl R. Studies in human biology. Baltimore: Williams and Wilkins; 1924.

7. F H. Arteriosclerosis. Archives of Pathology. 1945;39:187-216.

8. Organisation WH. Global recommendations on physical activity for health. 2014.

9. Li J, Loerbroks A, Angerer P. Physical activity and risk of cardiovascular disease: what does the new epidemiological evidence show? Curr Opin Cardiol. 2013;28(5):575-83.

10. Nocon M, Hiemann T, Muller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of physical activity with all-cause and cardiovascular mortality: a systematic review and meta-analysis. Eur J Cardiovasc Prev Rehabil. 2008;15(3):239-46.

11. Keimling M, Behrens G, Schmid D, Jochem C, Leitzmann MF. The association between physical activity and bladder cancer: systematic review and meta-analysis. Br J Cancer. 2014;110(7):1862-70.

12. Behrens G, Leitzmann MF. The association between physical activity and renal cancer: systematic review and meta-analysis. Br J Cancer. 2013;108(4):798-811.

13. Behrens G, Jochem C, Keimling M, Ricci C, Schmid D, Leitzmann MF. The association between physical activity and gastroesophageal cancer: systematic review and meta-analysis. Eur J Epidemiol. 2014;29(3):151-70.

14. Manini TM, Pahor M. Physical activity and maintaining physical function in older adults. Br J Sports Med. 2009;43(1):28-31.

15. Qu X, Zhang X, Zhai Z, Li H, Liu X, Li H, et al. Association between physical activity and risk of fracture. J Bone Miner Res. 2014;29(1):202-11.

16. Gardner MM, Robertson MC, Campbell AJ. Exercise in preventing falls and fall related injuries in older people: a review of randomised controlled trials. Br J Sports Med. 2000;34(1):7-17.

17. Cadar D, Pikhart H, Mishra G, Stephen A, Kuh D, Richards M. The role of lifestyle behaviors on 20-year cognitive decline. J Aging Research. 2012;2012:304014.

18. Richards M, Hardy R, Wadsworth ME. Does active leisure protect cognition? Evidence from a national birth cohort. Soc Sci Med. 2003;56(4):785-92.

19. Singh-Manoux A, Hillsdon M, Brunner E, Marmot M. Effects of physical activity on cognitive functioning in middle age: evidence from the Whitehall II prospective cohort study. Am J Public Health. 2005;95(12):2252-8.

20. Lahti J, Holstila A, Lahelma E, Rahkonen O. Leisure-time physical activity and allcause mortality. PLoS One. 2014;9(7):e101548.

21. Gando Y, Yamamoto K, Murakami H, Ohmori Y, Kawakami R, Sanada K, et al. Longer time spent in light physical activity is associated with reduced arterial stiffness in older adults. Hypertension. 2010;56(3):540-6.

22. National Heart Foundation of Australia. Physical Activity Policy Paper. 2001.

23. Lee IM, Shiroma EJ, Lobelo F, Puska P, Blair SN, Katzmarzyk PT. Lancet Physical Activity Series Working Group. Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. Lancet. 2012;380.

24. Matthews CE, George SM, Moore SC, Bowles HR, Blair A, Park Y, et al. Amount of time spent in sedentary behaviors and cause-specific mortality in US adults. Am J Clin Nutr. 2012;95(2):437-45.

25. Pulsford RM, Stamatakis E, Britton AR, Brunner EJ, Hillsdon M. Associations of sitting behaviours with all-cause mortality over a 16-year follow-up: the Whitehall II study. Int J Epidemiol. 2015;44(6):1909-16.

26. Ekelund U S-JJ, Brown WJ, Fagerland MW, Neville Owen N, Powell KE, MD, Bauman A, I-Min Lee for the Lancet Physical Activity Series 2 Executive Committe the Lancet Sedentary Behaviour Working Group. Does physical activity attenuate, or even eliminate, the detrimental association of sitting time with mortality? A harmonised meta-analysis of data from more than 1 million men and women. Lancet. 2016.

 Department of Health. Physical activity guideline for adults (18-64). 2011.
 Andersen K, Mariosa D, Adami HO, Held C, Ingelsson E, Lagerros YT, et al. Doseresponse relationship of total and leisure time physical activity to risk of heart failure: a prospective cohort study. Circ Heart Fail. 2014;7(5):701-8.

Beenackers MA, Kamphuis CB, Giskes K, Brug J, Kunst AE, Burdorf A.
 Socioeconomic inequalities in occupational, leisure-time, and transport related physical activity among European adults: a systematic review. Int J Behav Nutr Phys Act. 2012;9.
 Galobardes B, Davey Smith G, Lynch JW. Systematic review of the influence of

childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. Ann Epidemiol. 2006;16.

31. Jurakic D, Pedisic Z, Greblo Z. Physical activity in different domains and healthrelated quality of life: a population-based study. Quality of life research. 2010;19(9):1303-9.

32. Hinrichs T, von Bonsdorff MB, Tormakangas T, von Bonsdorff ME, Kulmala J, Seitsamo J, et al. Inverse effects of midlife occupational and leisure time physical activity on mobility limitation in old age--a 28-year prospective follow-up study. J Am Geriatr Soc. 2014;62(5):812-20.

33. Holtermann A, Marott JL, Gyntelberg F, Sogaard K, Suadicani P, Mortensen OS, et al. Occupational and leisure time physical activity: risk of all-cause mortality and myocardial infarction in the Copenhagen City Heart Study. A prospective cohort study. BMJ open. 2012;2(1):e000556.

 Lee DC, Pate RR, Lavie CJ, Sui X, Church TS, Blair SN. Leisure-time running reduces all-cause and cardiovascular mortality risk. J Am Coll Cardiol. 2014;64(5):472-81.
 Jefferis BJ, Whincup PH, Papacosta O, Wannamethee SG. Protective effect of time spent walking on risk of stroke in older men. Stroke. 2014;45(1):194-9.

36. Mayer F, Scharhag-Rosenberger F, Carlsohn A, Cassel M, Muller S, Scharhag J. The intensity and effects of strength training in the elderly. Deutsches Arzteblatt international. 2011;108(21):359-64.

37. Oja P, Titze S, Kokko S, Kujala UM, Heinonen A, Kelly P, et al. Health benefits of different sport disciplines for adults: systematic review of observational and intervention studies with meta-analysis. Br J Sports Med. 2015;49(7):434-40.

38. Tobias JH, Gould V, Brunton L, Deere K, Rittweger J, Lipperts M, et al. Physical Activity and Bone: May the Force be with You. Front Endocrinol (Lausanne). 2014;5:20.

39. Gomez-Bruton A, Montero-Marin J, Gonzalez-Aguero A, Garcia-Campayo J, Moreno LA, Casajus JA, et al. The Effect of Swimming During Childhood and Adolescence on Bone Mineral Density: A Systematic Review and Meta-Analysis. Sports Med. 2016;46(3):365-79.

40. Ainsworth BE, Haskell WL, Herrmann SD, Meckes N, Bassett DR, Jr., Tudor-Locke C, et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. Med Sci Sports Exerc. 2011;43(8):1575-81.

41. Bélanger M, Townsend N, Foster C. Age-related differences in physical activity profiles of English adults. Prev Med. 2011;52.

42. Borodulin K, Harald K, Jousilahti P, Laatikainen T, Mannisto S, Vartiainen E. Time trends in physical activity from 1982 to 2012 in Finland. Scand J Med Sci Sports. 2016;26(1):93-100.

43. Gu JK, Charles LE, Ma CC, Andrew ME, Fekedulegn D, Hartley TA, et al. Prevalence and trends of leisure-time physical activity by occupation and industry in U.S. workers: the National Health Interview Survey 2004–2014. Ann Epidemiol. 2016;26(10):685-92.

44. Hallal PC, Andersen LB, Bull FC, Guthold R, Haskell W, Ekelund U. Global physical activity levels: surveillance progress, pitfalls, and prospects. Lancet. 2012;380(9838):247-57.

45. Kalman M, Inchley J, Sigmundova D, Iannotti RJ, Tynjala JA, Hamrik Z, et al. Secular trends in moderate-to-vigorous physical activity in 32 countries from 2002 to 2010: a cross-national perspective. Eur J Public Health. 2015;25 Suppl 2:37-40.

46. Knuth AG, Hallal PC. Temporal trends in physical activity: a systematic review. J Phys Act Health. 2009;6(5):548-59.

47. Ekelund U. Lifetime lifestyles II: physical activity, the life course, and ageing. In: Kuh D, Cooper R, Hardy R, Richards M, Ben-Shlomo Y, editors. A life course approach to healthy ageing. New York: Oxford University Press; 2014.

48. Westerterp KR. Physical activity and physical activity induced energy expenditure in humans: measurement, determinants, and effects. Frontiers in physiology. 2013;4:90.

49. Haskell WL. Physical activity by self-report: a brief history and future issues. J Phys Act Health. 2012;9 Suppl 1:S5-10.

50. Kelly P, Fitzsimons C, Baker G. Should we reframe how we think about physical activity and sedentary behaviour measurement? Validity and reliability reconsidered. Int J Behav Nutr Phys Act. 2016;13:32.

51. Troiano RP, McClain JJ, Brychta RJ, Chen KY. Evolution of accelerometer methods for physical activity research. Br J Sports Med. 2014;48(13):1019-23.

52. Schrack JA, Cooper R, Koster A, Shiroma EJ, Murabito JM, Rejeski WJ, et al. Assessing Daily Physical Activity in Older Adults: Unraveling the Complexity of Monitors, Measures, and Methods. J Gerontol A Biol Sci Med Sci. 2016.

53. Schrack JA, Zipunnikov V, Goldsmith J, Bai J, Simonsick EM, Crainiceanu C, et al. Assessing the "physical cliff": detailed quantification of age-related differences in daily patterns of physical activity. J Gerontol A Biol Sci Med Sci.2014;69(8):973-9.

54. Lee IM, Shiroma EJ. Using accelerometers to measure physical activity in large-scale epidemiological studies: issues and challenges. Br J Sports Med. 2014;48(3):197-201.

55. Espana-Romero V, Golubic R, Martin KR, Hardy R, Ekelund U, Kuh D, et al. Comparison of the EPIC Physical Activity Questionnaire with combined heart rate and movement sensing in a nationally representative sample of older British adults. PLoS One. 2014;9(2):e87085.

56. Prince SA, Adamo KB, Hamel ME, Hardt J, Connor Gorber S, Tremblay M. A comparison of direct versus self-report measures for assessing physical activity in adults: a systematic review. Int J Behav Nutr Phys Act. 2008;5:56.

57. Scholes S, Coombs N, Pedisic Z, Mindell JS, Bauman A, Rowlands AV, et al. Ageand sex-specific criterion validity of the health survey for England Physical Activity and Sedentary Behavior Assessment Questionnaire as compared with accelerometry. Am J Epidemiol. 2014;179(12):1493-502. 58. Golubic R, Martin KR, Ekelund U, Hardy R, Kuh D, Wareham N. Levels of physical activity among a nationally representative sample of people in early old age: results of objective and self-reported assessments. Int J Behav Nutr Phys Act. 2014;11.

59. Dumith SC, Gigante DP, Domingues MR, Kohl HW, 3rd. Physical activity change during adolescence: a systematic review and a pooled analysis. Int J Epidemiol. 2011;40(3):685-98.

60. Gordon-Larsen P NM, Popkin BM. Longitudinal physical activity and sedentary behavior trends: adolescence to adulthood. Am J Prev Med. 2004;27(4):277-83.

61. Martin SL, Kirkner GJ, Mayo K, Matthews CE, Durstine JL, Hebert JR. Urban, rural, and regional variations in physical activity. J Rural Health. 2005;21(3):239-44.

62. Martinez-Gonzalez MA, Varo JJ, Santos JL, De Irala J, Gibney M, Kearney J, et al. Prevalence of physical activity during leisure time in the European Union. Med Sci Sports Exerc. 2001;33(7):1142-6.

63. Organisation WH. Prevalence of insufficient physical activity. 2008.

64. Centre HaSCI. Statistics on Obesity, Physical Activity and Diet: England 2014. 2014.

65. Townsend N WK, Williams J, Bhatnagar P, Rayner M Physical Activity Statistics 2015. In: Foundation BH, editor. London 2015.

66. Jefferis BJ, Sartini C, Lee IM, Choi M, Amuzu A, Gutierrez C, et al. Adherence to physical activity guidelines in older adults, using objectively measured physical activity in a population-based study. BMC Public Health. 2014;14:382.

67. Martin KR, Cooper R, Harris TB, Brage S, Hardy R, Kuh D. Patterns of leisure-time physical activity participation in a British birth cohort at early old age. PLoS One. 2014;9(6):e98901.

68. Bauman AE, Reis RS, Sallis JF, Wells JC, Loos RJ, Martin BW. Lancet Physical Activity Series Working Group: correlates of physical activity: why are some people physically active and others not? Lancet. 2012;380.

69. Eisenmann JC, Wickel EE. The biological basis of physical activity in children: revisited. Pediatr Exerc Sci. 2009;21(3):257-72.

70. Merom D, Miller YD, van der Ploeg HP, Bauman A. Predictors of initiating and maintaining active commuting to work using transport and public health perspectives in Australia. Prev Med. 2008;47(3):342-6.

71. Humpel N, Owen N, Leslie E. Environmental factors associated with adults' participation in physical activity: a review. Am J Prev Med. 2002;22(3):188-99.

72. Engberg E, Alen M, Kukkonen-Harjula K, Peltonen JE, Tikkanen HO, Pekkarinen H. Life events and change in leisure time physical activity: a systematic review. Sports Med. 2012;42(5):433-47.

73. Stenholm S, Pulakka A, Kawachi I, Oksanen T, Halonen JI, Aalto V, et al. Changes in physical activity during transition to retirement: a cohort study. Int J Behav Nutr Phys Act. 2016;13:51.

74. King AC, Stokols D, Talen E, Brassington GS, Killingsworth R. Theoretical approaches to the promotion of physical activity: forging a transdisciplinary paradigm. Am J Prev Med. 2002;23(2 Suppl):15-25.

75. Prochaska JO DC. Stages of change in the modification of problem behaviors. In: Hersen M ER, Miller PM, editor. Progress in Behavior Modification. Illinois: Sycamore Press; 1992. p. 184–214.

76. Cochrane T, Davey RC. Increasing uptake of physical activity: a social ecological approach. J R Soc Promot Health. 2008;128(1):31-40.

77. Metcalf B, Henley W, Wilkin T. Effectiveness of intervention on physical activity of children: systematic review and meta-analysis of controlled trials with objectively measured outcomes (EarlyBird 54). BMJ. 2012;345:e5888.

78. Conn VS, Hafdahl AR, Mehr DR. Interventions to increase physical activity among healthy adults: meta-analysis of outcomes. Am J Public Health. 2011;101.

79. Pavey TG, Taylor AH, Fox KR, Hillsdon M, Anokye N, Campbell JL, et al. Effect of exercise referral schemes in primary care on physical activity and improving health outcomes: systematic review and meta-analysis. BMJ. 2011;343:d6462.

80. van der Bij AK, Laurant MG, Wensing M. Effectiveness of physical activity interventions for older adults: a review. Am J Prev Med. 2002;22(2):120-33.

81. Hirvensalo M LT. Life-course perspective for physical activity and sports participation. Eur Rev Aging Phys Act 2011;8:13-22.

82. Kuh D B-SY. A life course approach to chronic disease epidemiology. 2nd edition ed. New York: Oxford University Press; 2004.

83. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. J Epidemiol Community Health. 2003;57(10):778-83.

84. Barker DJ. The developmental origins of adult disease. J Am Coll Nutr. 2004;23(6 Suppl):588s-95s.

85. Allinson JP, Hardy R. The Presence of Chronic Mucus Hypersecretion across Adult Life in Relation to Chronic Obstructive Pulmonary Disease Development. Am J Respir Crit Care Med 2016;193(6):662-72.

86. Ben-Shlomo Y CR, Kuh D. The last two decades of life course epidemiology, and its relevance for research on ageing. Int J Epidemiol. 2016;45(4):973-88.

87. Cooper R, Muniz-Terrera G, Kuh D. Associations of behavioural risk factors and health status with changes in physical capability over 10 years of follow-up: the MRC National Survey of Health and Development. BMJ open. 2016;6(4):e009962.

88. Ferrucci L, Cooper R, Shardell M, Simonsick EM, Schrack JA, Kuh D. Age-Related Change in Mobility: Perspectives From Life Course Epidemiology and Geroscience. J Gerontol A Biol Sci Med Sci. 2016.

89. Hardy R, Lawlor DA, Kuh D. A life course approach to cardiovascular aging. Future Cardiol. 2015;11(1):101-13.

90. Kuh DC, R. Hardy, R. Richards, M. Ben-Shlomo, Y. A life course approach to healthy ageing. New York: Oxford University Press; 2014.

91. Telama R. Tracking of physical activity from childhood to adulthood: a review. Obes Facts. 2009;2.

92. Schooling M, Kuh D. A life course perspective on women's health behaviours. In: Kuh D, Hardy R, editors. A life course approach to women's health. Oxford: Oxford University Press; 2003.

93. Kuh DJ, Cooper C. Physical activity at 36 years: patterns and childhood predictors in a longitudinal study. J Epidemiol Community Health. 1992;46.

94. Tammelin T. A review of longitudinal studies on youth predictors of adulthood physical activity. Int J Adolesc Med Health. 2005;17.

95. Aaltonen S, Ortega-Alonso A, Kujala UM, Kaprio J. Genetic and environmental influences on longitudinal changes in leisure-time physical activity from adolescence to young adulthood. Twin Res Hum Genet. 2013;16.

96. Murray GK, Jones PB, Kuh D, Richards M. Infant developmental milestones and subsequent cognitive function. Ann Neurol. 2007;62(2):128-36.

97. Sherar LB, Cumming SP, Eisenmann JC, Baxter-Jones AD, Malina RM. Adolescent biological maturity and physical activity: biology meets behavior. Pediatr Exerc Sci. 2010;22(3):332-49.

98. Wadsworth M, Kuh D, Richards M, Hardy R. Cohort Profile: The 1946 National Birth Cohort (MRC National Survey of Health and Development). Int J Epidemiol. 2006;35(1):49-54.

99. Kuh D, Pierce M, Adams J, Deanfield J, Ekelund U, Friberg P, et al. Cohort profile: updating the cohort profile for the MRC National Survey of Health and Development: a new clinic-based data collection for ageing research. Int J Epidemiol. 2011;40(1):e1-9.

100. Blane D. Historical note: early years of the 1946 British birth cohort study. Longitudinal and Life Course Studies. 2010;3:305-9.

101. Wadsworth M, J. B. A Companion to Life Course Studies: The Social and Historical Context of the British Birth Cohort Studies: Routledge Advances in Sociology; 2011.

102. Schooling C. Health behaviour in a social and temporal context. London: University College London; 2001.

103. Randall MW WW. Objective of the physical education lesson: G Bell and Sons; 1955.

104. Holt R MT. Sport in Britain 1945-2000: Wiley-Blackwell; 2000. 228 p.

105. Rothman. Epidemiology: An Introduction 2nd edition ed. New York: Oxford University Press; 2012.

106. Stafford M, Black S, Shah I, Hardy R, Pierce M, Richards M, et al. Using a birth cohort to study ageing: representativeness and response rates in the National Survey of Health and Development. Eur J Ageing. 2013;10(2):145-57.

107. Kuh D, Wong A, Shah I, Moore A, Popham M, Curran P, et al. The MRC National Survey of Health and Development reaches age 70: maintaining participation at older ages in a birth cohort study. Eur J Epidemiol. 2016;31(11):1135-47.

108. Wadsworth M, Mann SL, Rodgers B, Kuh DJ, Hilder WS, Yusuf EJ. Loss and representativeness in a 43 year follow up of a national birth cohort. J Epidemiol Community Health. 1992;46(3):300-4.

109. Wadsworth M, Butterworth SL, Hardy RJ, Kuh DJ, Richards M, Langenberg C, et al. The life course prospective design: an example of benefits and problems associated with study longevity. Soc Sci Med. 2003;57(11):2193-205.

110. Arem H, Keadle SK, Matthews CE. Invited commentary: meta-physical activity and the search for the truth. Am J Epidemiol. 2015;181(9):656-8.

111. Taylor HL, Jacobs DR, Jr., Schucker B, Knudsen J, Leon AS, Debacker G. A questionnaire for the assessment of leisure time physical activities. J Chronic Dis. 1978;31(12):741-55.

112. Cooper R, Mishra G, Kuh D. Physical activity across adulthood and physical performance in midlife: findings from a British birth cohort. Am J Prev Med. 2011;41(4):376-84.

113. Dodds R, Kuh D, Aihie Sayer A, Cooper R. Physical activity levels across adult life and grip strength in early old age: updating findings from a British birth cohort. Age Ageing. 2013;42(6):794-8.

114. Bann D, Kuh D, Wills AK, Adams J, Brage S, Cooper R. Physical activity across adulthood in relation to fat and lean body mass in early old age: findings from the Medical Research Council National Survey of Health and Development, 1946-2010. Am J Epidemiol. 2014;179(10):1197-207.

115. Black SV, Cooper R, Martin KR, Brage S, Kuh D, Stafford M. Physical Activity and Mental Well-being in a Cohort Aged 60-64 Years. Am J Prev Med. 2015;49(2):172-80.

116. Arem H, Moore SC, Patel A, Hartge P, Berrington de Gonzalez A, Visvanathan K, et al. Leisure time physical activity and mortality: a detailed pooled analysis of the dose-response relationship. JAMA Intern Med. 2015;175(6):959-67.

117. Hamer M, Sabia S, Batty GD, Shipley MJ, Tabak AG, Singh-Manoux A, et al. Physical activity and inflammatory markers over 10 years: follow-up in men and women from the Whitehall II cohort study. Circulation. 2012;126(8):928-33. 118. Hupin D, Roche F, Gremeaux V, Chatard JC, Oriol M, Gaspoz JM, et al. Even a lowdose of moderate-to-vigorous physical activity reduces mortality by 22% in adults aged >/=60 years: a systematic review and meta-analysis. Br J Sports Med. 2015;49(19):1262-7.

119. Warburton DE, Nicol CW, Bredin SS. Health benefits of physical activity: the evidence. CMAJ. 2006;174.

120. Howe CJ, Cole SR, Lau B, Napravnik S, Eron JJ, Jr. Selection Bias Due to Loss to Follow Up in Cohort Studies. Epidemiol. 2016;27(1):91-7.

121. Picciotto S, Hertz-Picciotto I. Commentary: healthy worker survivor bias: a stillevolving concept. Epidemiol. 2015;26(2):213-5.

122. Hillsdon M, Cavill N, Nanchahal K, Diamond A, White IR. National level promotion of physical activity: results from England's ACTIVE for LIFE campaign. J Epidemiol Community Health. 2001;55(10):755-61.

123. Brage S, Ekelund U, Brage N, Hennings MA, Froberg K, Franks PW, et al. Hierarchy of individual calibration levels for heart rate and accelerometry to measure physical activity. J Appl Physiol (1985). 2007;103(2):682-92.

124. Brage S, Brage N, Franks PW, Ekelund U, Wong MY, Andersen LB, et al. Branched equation modeling of simultaneous accelerometry and heart rate monitoring improves estimate of directly measured physical activity energy expenditure. J Appl Physiol (1985). 2004;96(1):343-51.

125. Brage S, Brage N, Franks PW, Ekelund U, Wareham NJ. Reliability and validity of the combined heart rate and movement sensor Actiheart. Eur J Clin Nutr. 2005;59(4):561-70.

126. Assah FK, Ekelund U, Brage S, Wright A, Mbanya JC, Wareham NJ. Accuracy and validity of a combined heart rate and motion sensor for the measurement of free-living physical activity energy expenditure in adults in Cameroon. Int J Epidemiol. 2011;40(1):112-20.

127. Thompson D, Batterham AM, Bock S, Robson C, Stokes K. Assessment of low-tomoderate intensity physical activity thermogenesis in young adults using synchronized heart rate and accelerometry with branched-equation modeling. J Nutr. 2006;136(4):1037-42.

128. Cooper AJ, Simmons RK, Kuh D, Brage S, Cooper R. Physical activity, sedentary time and physical capability in early old age: British birth cohort study. PLoS One. 2015;10(5):e0126465.

129. Cole TJ. Sympercents: symmetric percentage differences on the 100 log(e) scale simplify the presentation of log transformed data. Statistics in medicine. 2000;19(22):3109-25.

130. kaufman J. Interaction reaction. Epidemiol. 2009;20(2):159-60.

131. Liu X. Methods and Applications of Longitudinal Data Analysis: Academic Press: Elsevier; 2015.

132. Szmaragd C CP, Steele F. Subject specific and population average models for binary longitudinal data: a tutorial. Longitudinal and Life Course Studies 2013;4(2):147-65.

133. Hedeker D. A mixed-effects multinomial logistic regression model. Stat Med. 2003;22(9):1433-46.

134. Browne WJ. MCMC Estimation in MLwiN v2.36. In: Centre for Multilevel Modelling University of Bristol. editor. 2016.

135. Leckie G, Charlton C. runmlwin - A Program to Run the MLwiN Multilevel Modelling Software from within Stata. Journal of Statistical Software. 2013;52(11):1-40.

136. Rasbash J, Steele, F, Browne, W.J, Goldstein H. A user guide to MLwiN Version 2.36. In: Centre for Multilevel Modelling University of Bristol. editor. 2016.

137. Greenland S. Bayesian perspectives for epidemiological research: I. Foundations and basic methods. Int J Epidemiol. 2006;35(3):765-75.

138. Gelman A, Rubin DB. Markov chain Monte Carlo methods in biostatistics. Stat Methods Med Res. 1996;5(4):339-55.

139. Hamra G, MacLehose R, Richardson D. Markov chain Monte Carlo: an introduction for epidemiologists. Int J Epidemiol. 2013;42(2):627-34.

140. Ferreira-Gonzalez I, Marsal JR, Ribera A, Permanyer-Miralda G, Garcia-Del Blanco B, Marti G, et al. Background, incidence, and predictors of antiplatelet therapy discontinuation during the first year after drug-eluting stent implantation. Circulation. 2010;122(10):1017-25.

141. Greenland S. Bayesian perspectives for epidemiological research. II. Regression analysis. Int J Epidemiol. 2007;36(1):195-202.

142. Mackenbach JP, Stirbu I, Roskam AJ, Schaap MM, Menvielle G, Leinsalu M.
Socioeconomic inequalities in health in 22 European countries. N Engl J Med. 2008;358.
143. Marmot MG. Fair society, healthy lives: strategic review of health inequalities in England post-2010. 2010.

144. Pampel FC KP, Denney JT. Socioeconomic Disparities in Health Behaviors. Annu Rev Sociol. 2010;36:349–70.

145. Winkleby MA JD, Frank E, Fortmann SP. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. Am J Public Health. 1992;82(6): 816–20.

146. Gidlow C, Johnston LH, Crone D, Ellis N, James D. A systematic review of the relationship between socio-economic position and physical activity. Health Educ J. 2006;65.
147. Stalsberg R, Pedersen AV. Effects of socioeconomic status on the physical activity in

adolescents: a systematic review of the evidence. Scand J Med Sci Spor. 2010;20.
148. Kuh D, Power C, Blane D, Bartley M. Socioeconomic pathways between childhood and adult health. In: Kuh D, Ben-Shlomo Y, editors. A life course approach to chronic disease epidemiology. New York: Oxford University Press; 2004.

149. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ. 2009;339.

150. Elhakeem A, Cooper R, Bann D, Hardy R. Childhood socioeconomic position and adult leisure-time physical activity: a systematic review protocol. Syst Rev. 2014;3.

151. Elhakeem A, Cooper R, Bann D, Hardy R. Childhood socioeconomic position and adult leisure-time physical activity: a systematic review. Int J Behav Nutr Phys Act. 2015;12(1):1-27.

 Krieger N, Williams DR, Moss NE. Measuring social class in US public health research: concepts, methodologies, and guidelines. Annu Rev Public Health. 1997;18.
 Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of

socioeconomic position (part 1). J Epidemiol Community Health. 2006;60. 154. Affairs UNDoEaS. Definition of youth.

http://www.un.org/esa/socdev/documents/youth/fact-sheets/youth-definition.pdf. Accessed 08 Jun 2015.

Ottawa Hospital Research Institute. The Newcastle-Ottawa Quality Assessment
 Scale. <u>http://www.ohri.ca/programs/clinical_epidemiology/nosgen.pdf</u>. Accessed 10 Jan 2014.
 Sanderson S, Tatt ID, Higgins JP. Tools for assessing quality and susceptibility to

bias in observational studies in epidemiology: a systematic review and annotated bibliography. Int J Epidemiol. 2007;36.

157. Bowen ME. Coronary heart disease from a life-course approach: findings from the health and retirement study, 1998–2004. J Aging Health. 2010;22.

 Carroll JE, Cohen S, Marsland AL. Early childhood socioeconomic status is associated with circulating interleukin-6 among mid-life adults. Brain Behav Immun. 2011;25.
 Cheng H, Furnham A. Personality traits, education, physical exercise, and childhood

neurological function as independent predictors of adult obesity. PLoS One. 2013;8.

160. Heraclides A, Witte D, Brunner EJ. The association between father's social class and adult obesity is not explained by educational attainment and an unhealthy lifestyle in adulthood. Eur J Epidemiol. 2008;23.

161. Jørgensen MB, Nabe-Nielsen K, Clausen T, Holtermann A. Independent effect of physical workload and childhood socioeconomic status on low back pain among health care workers in Denmark. Spine (Phila Pa 1976). 2013;38.

162. Kamphuis CB, Turrell G, Giskes K, Mackenbach JP, Lenthe FJ. Life course socioeconomic conditions, adulthood risk factors and cardiovascular mortality among men and women: a 17-year follow up of the GLOBE study. Int J Cardiol. 2013;168.

163. Phillips JE, Marsland AL, Flory JD, Muldoon MF, Cohen S, Manuck SB. Parental education is related to C-reactive protein among female middle aged community volunteers. Brain Behav Immun. 2009;23.

164. Popham F. Intergenerational social class stability and mobility are associated with large absolute differences in adult participation in sport and exercise. Br J Sports Med. 2010;44.

165. Ramsay SE, Whincup PH, Morris RW, Lennon LT, Wannamethee SG. Are childhood socio-economic circumstances related to coronary heart disease risk? Findings from a population-based study of older men. Int J Epidemiol. 2007;36.

166. Blane D, Hart CL, Davey Smith G, Gillis CR, Hole DJ, Hawthorne VM. Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. BMJ. 1996;313.

167. Brunner E, Shipley MJ, Blane D, Davey Smith G, Marmot MG. When does cardiovascular risk start? Past and present socioeconomic circumstances and risk factors in adulthood. J Epidemiol Community Health. 1999;53.

168. Frank E, Elon L, Hogue C. Transgenerational persistence of education as a health risk: findings from the women physicians' health study. J Women's Health. 2003;12.

169. Hart CL, Davey Smith G, Blane D. Social mobility and 21 year mortality in a cohort of Scottish men. Soc Sci Med. 1998;47.

170. Stringhini S, Batty GD, Bovet P, Shipley MJ, Marmot MG, Kumari M. Association of lifecourse socioeconomic status with chronic inflammation and type 2 diabetes risk: the Whitehall II prospective cohort study. PLoS Med. 2013;10.

171. Hillsdon M, Lawlor DA, Ebrahim S, Morris JN. Physical activity in older women: associations with area deprivation and with socioeconomic position over the life course: observations in the British Women's Heart and Health Study. J Epidemiol Community Health. 2008;62.

172. Johnson W, Corley J, Starr JM, Deary IJ. Psychological and physical health at age 70 in the Lothian Birth Cohort 1936: links with early life IQ, SES, and current cognitive function and neighborhood environment. Health Psychol. 2011;30.

173. Pinto Pereira SM, Li L, Power C. Early-life predictors of leisure-time physical inactivity in midadulthood: findings from a prospective British birth cohort. Am J Epidemiol. 2014;180.

174. Silverwood RJ, Pierce M, Nitsch D, Mishra GD, Kuh D. Is intergenerational social mobility related to the type and amount of physical activity in mid-adulthood? Results from the 1946 British birth cohort study. Ann Epidemiol. 2012;22.

175. Watt HC, Carson C, Lawlor DA, Patel R, Ebrahim S. Influence of life course socioeconomic position on older women's health behaviors: findings from the British Women's Heart and Health Study. Am J Public Health. 2009;99.

176. Beunen GP, Lefevre J, Philippaerts RM, Delvaux K, Thomis M, Claessens AL. Adolescent correlates of adult physical activity: a 26-year follow-up. Med Sci Sports Exerc. 2004;36. 177. Makinen T, Kestila L, Borodulin K, Martelin T, Rahkonen O, Prattala R. Effects of childhood socio-economic conditions on educational differences in leisure-time physical activity. Eur J Public Health. 2010;20.

178. Kvaavik E, Glymour M, Klepp KI, Tell GS, Batty GD. Parental education as a predictor of offspring behavioural and physiological cardiovascular disease risk factors. Eur J Public Health. 2012;22.

179. Tsenkova V, Pudrovska T, Karlamangla A. Childhood socioeconomic disadvantage and prediabetes and diabetes in later life: a study of biopsychosocial pathways. Psychosom Med. 2014;76.

180. Wray LA, Alwin DF, McCammon RJ. Social status and risky health behaviors: results from the health and retirement study. J Gerontol B Psychol Sci Soc Sci. 2005;60.

181. Wannamethee SG, Whincup PH, Shaper G, Walker M. Influence of fathers' social class on cardiovascular disease in middle-aged men. Lancet. 1996;348.

182. Schooling CM, Jiang CQ, Lam TH, Zhang WS, Cheng KK, Leung GM. Life-course origins of social inequalities in metabolic risk in the population of a developing country. Am J Epidemiol. 2008;167.

183. Peck MN. The importance of childhood socio-economic group for adult health. Soc Sci Med. 1994;39.

184. Regidor E, Banegas JR, Gutiérrez-Fisac JL, Domínguez V, Rodríguez-Artalejo F. Socioeconomic position in childhood and cardiovascular risk factors in older Spanish people. Int J Epidemiol. 2004;33.

185. Tammelin T, Näyhä S, Laitinen J, Rintamäki H, Järvelin MR. Physical activity and social status in adolescence as predictors of physical inactivity in adulthood. Prev Med. 2003;37.

186. Wichstorm L, Kvalem IL, Soest T. Predictors of growth and decline in leisure-time physical activity from adolescence to adulthood. Health Psychol. 2013;32.

187. Juneau CE, Sullivan A, Dodgeon B, Côté S, Ploubidis GB, Potvin L. Social class across the life course and physical activity at age 34 years in the 1970 British birth cohort. Ann Epidemiol. 2014;24.

188. Lawlor DA, Davey Smith G, Ebrahim S. Association between childhood socioeconomic status and coronary heart disease risk among postmenopausal women.
Findings from the British Women's, Heart and Health Study. Am J Public Health. 2004;94.
189. Hart C, McConnachie A, Upton M, Watt G. Risk factors in the Midspan family study by social class in childhood and adulthood. Int J Epidemiol. 2008;37.

 Osler M, Godtfredsen NS, Prescott E. Childhood social circumstances and health behaviour in midlife: the Metropolit 1953 Danish male birth cohort. Int J Epidemiol. 2008;37.
 Barnekow-Bergkvist M, Hedberg G, Janlert U, Jansson E. Prediction of physical fitness and physical activity level in adulthood by physical performance and physical activity in

fitness and physical activity level in adulthood by physical performance and physical activity in adolescence–an 18-year follow-up study. Scand J Med Sci Sports. 1998;8.

192. Mheen H, Stronks K, Looman CWN, Mackenbach JP. Does childhood socioeconomic status influence adult health through behavioural factors? Int J Epidemiol. 1998;27.

193. Osler M, Clausen JO, Ibsen KK, Jensen GB. Social influences and low leisure-time physical activity in young Danish adults. Eur J Public Health. 2001;11.

194. Leino M, Raitakari OT, Porkka KV, Taimela S, Viikari JS. Associations of education with cardiovascular risk factors in young adults: the Cardiovascular Risk in Young Finns Study. Int J Epidemiol. 1999;28.

195. Gall SL, Abbott-Chapman J, Patton GC, Dwyer T, Venn A. Intergenerational educational mobility is associated with cardiovascular disease risk behaviours in a cohort of young Australian adults: the Childhood Determinants of Adult Health (CDAH) Study. BMC Public Health. 2010;10.

196. Lynch JW, Kaplan GA, Salonen JT. Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. Soc Sci Med. 1997;44.

197. Kern ML, Reynolds CA, Friedman HS. Predictors of physical activity patterns across adulthood: a growth curve analysis. Pers Soc Psychol Bull. 2010;36.

198. Pudrovska T, Anishkin A. Early-life socioeconomic status and physical activity in later life: evidence from structural equation models. J Aging Health. 2013;25.

199. Elwell-Sutton TM, Jiang CQ, Zhang WS, Cheng KK, Lam TH, Leung GM. Socioeconomic influences at different life stages on health in Guangzhou, China. Soc Sci Med. 2011;72.

200. Birnie K, Cooper R, Martin RM, Kuh D, Aihie Sayer A, Alvarado BE. Childhood socioeconomic position and objectively measured physical capability levels in adulthood: a systematic review and meta-analysis. PLoS One. 2011;6.

201. Galobardes B, Lynch JW, Davey SG. Is the association between childhood socioeconomic circumstances and cause-specific mortality established? Update of a systematic review. J Epidemiol Community Health. 2008;62.

202. Kraaykamp G, Oldenkamp M, Breedveld K. Starting a sport in the Netherlands: a lifecourse analysis of the effects of individual, parental and partner characteristics. Int Rev Sociol Sport. 2013;48.

203. Graham H. Socio-economic inequalities across generations: occupation and education. Unequal lives: health and socioeconomic inequalities. Berkshire: Open University Press; 2007.

204. Elhakeem A, Hardy R, Bann D, Caleyachetty R, Cosco T, Hayhoe R, et al. Intergenerational social mobility and leisure-time physical activity in adulthood: a systematic review. J Epidemiol Community Health. 2016.

205. Yao CA, Rhodes RE. Parental correlates in child and adolescent physical activity: a meta-analysis. Int J Behav Nutr Phys Act. 2015;12.

206. Malina RM, Bouchard C, Bar-Or O. Other factors affecting growth, maturation, performance, and activity. In: Malina RM, Bouchard C, Bar-Or O, editors. Growth, maturation, and physical activity. United States: Human Kinetics; 2004.

207. Jones RA, Hinkley T, Okely AD, Salmon J. Tracking physical activity and sedentary behavior in childhood: a systematic review. Am J Prev Med. 2013;44.

208. Scheerder J, Thomis M, Vanreusel B, Lefevre J, Renson R, Vanden Eynde B. Sports participation among females from adolescence to adulthood: a longitudinal study. Int Rev Sociol Sport. 2006;41.

209. Batty GD, Lawlor DA, Macintyre S, Clark H, Leon DA. Accuracy of adults' recall of childhood social class: findings from the Aberdeen children of the 1950s study. J Epidemiol Community Health. 2005;59.

210. Wilcox S, Castro C, King AC, Housemann R, Brownson RC. Determinants of leisure time physical activity in rural compared with urban older and ethnically diverse women in the United States. J Epidemiol Community Health. 2000;54.

211. Kim S, Symons M, Popkin BM. Contrasting socioeconomic profiles related to healthier lifestyles in China and the United States. Am J Epidemiol. 2004;159.

212. Skogen JC, Overland S. The fetal origins of adult disease: a narrative review of the epidemiological literature. JRSM Short Rep. 2012;3(8):59.

213. Hales CN, Barker DJ. The thrifty phenotype hypothesis. British medical bulletin. 2001;60:5-20.

214. Harding JE. The nutritional basis of the fetal origins of adult disease. Int J Epidemiol. 2001;30(1):15-23.

215. Lumey LH, Ravelli AC, Wiessing LG, Koppe JG, Treffers PE, Stein ZA. The Dutch famine birth cohort study: design, validation of exposure, and selected characteristics of subjects after 43 years follow-up. Paediatr Perinat Epidemiol. 1993;7(4):354-67.

Painter RC, Roseboom TJ, Bleker OP. Prenatal exposure to the Dutch famine and disease in later life: an overview. Reproductive toxicology (Elmsford, NY). 2005;20(3):345-52.
Ravelli AC, van der Meulen JH, Michels RP, Osmond C, Barker DJ, Hales CN, et al.

Glucose tolerance in adults after prenatal exposure to famine. Lancet. 1998;351(9097):173-7.218. Roseboom T, de Rooij S, Painter R. The Dutch famine and its long-term

consequences for adult health. Early human development. 2006;82(8):485-91.

219. Vehaskari VM, Aviles DH, Manning J. Prenatal programming of adult hypertension in the rat. Kidney Int. 2001;59(1):238-45.

220. Cetin I, Mando C, Calabrese S. Maternal predictors of intrauterine growth restriction. Curr Opin Clin Nutr Metab Care. 2013;16(3):310-9.

221. Kramer MS. Determinants of low birth weight: methodological assessment and metaanalysis. Bull World Health Organ. 1987;65(5):663-737.

222. Harder T, Rodekamp E, Schellong K, Dudenhausen JW, Plagemann A. Birth weight and subsequent risk of type 2 diabetes: a meta-analysis. Am J Epidemiol. 2007;165(8):849-57.

223. Wang SF, Shu L, Sheng J, Mu M, Wang S, Tao XY, et al. Birth weight and risk of coronary heart disease in adults: a meta-analysis of prospective cohort studies. J Dev Orig Health Dis. 2014;5(6):408-19.

224. Huxley R, Owen CG, Whincup PH, Cook DG, Rich-Edwards J, Smith GD, et al. Is birth weight a risk factor for ischemic heart disease in later life? Am J Clin Nutr. 2007;85(5):1244-50.

225. Whincup PH, Kaye SJ, Owen CG, Huxley R, Cook DG, Anazawa S, et al. Birth weight and risk of type 2 diabetes: a systematic review. JAMA. 2008;300(24):2886-97.

226. Risnes KR, Vatten LJ, Baker JL, Jameson K, Sovio U, Kajantie E, et al. Birthweight and mortality in adulthood: a systematic review and meta-analysis. Int J Epidemiol. 2011;40(3):647-61.

227. Huxley R, Neil A, Collins R. Unravelling the fetal origins hypothesis: is there really an inverse association between birthweight and subsequent blood pressure? Lancet. 2002;360(9334):659-65.

228. Huxley R, Owen CG, Whincup PH, Cook DG, Colman S, Collins R. Birth weight and subsequent cholesterol levels: exploration of the "fetal origins" hypothesis. JAMA. 2004;292(22):2755-64.

229. Baird J, Kurshid MA, Kim M, Harvey N, Dennison E, Cooper C. Does birthweight predict bone mass in adulthood? A systematic review and meta-analysis. Osteoporosis Int. 2011;22(5):1323-34.

230. Kuh D, Wills AK, Shah I, Prentice A, Hardy R, Adams JE, et al. Growth from birth to adulthood and bone phenotype in early old age: a British birth cohort study. J Bone Miner Res. 2014;29(1):123-33.

231. Bann D, Wills A, Cooper R, Hardy R, Aihie Sayer A, Adams J, et al. Birth weight and growth from infancy to late adolescence in relation to fat and lean mass in early old age: findings from the MRC National Survey of Health and Development. Int J Obes (Lond). 2014;38(1):69-75.

232. Dodds R, Denison HJ, Ntani G, Cooper R, Cooper C, Sayer AA, et al. Birth weight and muscle strength: a systematic review and meta-analysis. J Nutr Health Aging. 2012;16(7):609-15.

233. Patel HP, Jameson KA, Syddall HE, Martin HJ, Stewart CE, Cooper C, et al. Developmental influences, muscle morphology, and sarcopenia in community-dwelling older men. J Gerontol A Biol Sci Med Sci.2012;67(1):82-7.

234. Lee DC, Artero EG, Sui X, Blair SN. Mortality trends in the general population: the importance of cardiorespiratory fitness. J Psychopharmacol (Oxford, England). 2010;24(4 Suppl):27-35.

235. van Deutekom AW CM, Vrijkotte TG, Gemke RJ. The association of birth weight and infant growth with physical fitness at 8-9 years of age--the ABCD study. Int J Obes (Lond). 2015;39(4):593-600.

236. Boreham CA, Murray L, Dedman D, Davey Smith G, Savage JM, Strain JJ. Birthweight and aerobic fitness in adolescents: the Northern Ireland Young Hearts Project. Public health. 2001;115(6):373-9.

237. Ridgway CL, Ong KK, Tammelin T, Sharp SJ, Ekelund U, Jarvelin MR. Birth size, infant weight gain, and motor development influence adult physical performance. Med Sci Sports Exerc. 2009;41(6):1212-21.

238. Svedenkrans J, Henckel E, Kowalski J, Norman M, Bohlin K. Long-term impact of preterm birth on exercise capacity in healthy young men: a national population-based cohort study. PLoS One. 2013;8(12):e80869.

239. Raikkonen K, Pesonen AK. Early life origins of psychological development and mental health. Scand J Psychol. 2009;50(6):583-91.

240. Maitra K, Park HY, Eggenberger J, Matthiessen A, Knight E, Ng B. Difficulty in mental, neuromusculoskeletal, and movement-related school functions associated with low birthweight or preterm birth: a meta-analysis. Am J Occup Ther. 2014;68(2):140-8.

241. Miller SL, Huppi PS, Mallard C. The consequences of fetal growth restriction on brain structure and neurodevelopmental outcome. J Physiol. 2016;594(4):807-23.

242. Øglund GP ST, Ekelund U. Early life determinants of physical activity and sedentary time: Current knowledge and future research. Norsk Epidemiologi. 2014;24(1-2):177-86.

243. Baker MS, Li G, Kohorst JJ, Waterland RA. Fetal growth restriction promotes physical inactivity and obesity in female mice. Int J Obes (Lond). 2015;39(1):98-104.

244. Vickers MH, Breier BH, McCarthy D, Gluckman PD. Sedentary behavior during postnatal life is determined by the prenatal environment and exacerbated by postnatal hypercaloric nutrition. Am J Physiol Regul Integr Comp Physiol. 2003;285(1):R271-3.

245. Zhu S, Eclarinal J, Baker MS, Li G, Waterland RA. Developmental programming of energy balance regulation: is physical activity more 'programmable' than food intake? Proc Nutr Soc. 2016;75(1):73-7.

246. Ridgway CL, Brage S, Sharp SJ, Corder K, Westgate KL, van Sluijs EM, et al. Does birth weight influence physical activity in youth? A combined analysis of four studies using objectively measured physical activity. PLoS One. 2011;6(1):e16125.

247. Mattocks C NA, Deere K, Tilling K, Leary S, Blair S N, Riddoch C. Early life determinants of physical activity in 11 to 12 year olds: cohort study. BMJ. 2008;336(7634):26-9.

248. Oglund GP, Hildebrand M, Ekelund U. Are Birth Weight, Early Growth, and Motor Development Determinants of Physical Activity in Children and Youth? A Systematic Review and Meta-Analysis. Pediatr Exerc Sci. 2015;27(4):441-53.

Hallal PC WJ, Reichert FF, Anselmi L, Victora CG. Early determinants of physical activity in adolescence: prospective birth cohort study. BMJ. 2006;332(7548):1002-7.
Gopinath B, Hardy LL, Baur LA, Burlutsky G, Mitchell P. Birth weight and time spent in outdoor physical activity during adolescence. Med Sci Sports Exerc. 2013;45(3):475-80.

251. Andersen LG, Angquist L, Gamborg M, Byberg L, Bengtsson C, Canoy D, et al. Birth weight in relation to leisure time physical activity in adolescence and adulthood: meta-analysis of results from 13 nordic cohorts. PLoS One. 2009;4(12):e8192.

252. Azevedo MR, Horta BL, Gigante DP, Victora CG, Barros FC. [Factors associated to leisure-time sedentary lifestyle in adults of 1982 birth cohort, Pelotas, Southern Brazil]. Revista de saude publica. 2008;42 Suppl 2:70-7.

253. Salonen MK, Kajantie E, Osmond C, Forsen T, Yliharsila H, Paile-Hyvarinen M, et al. Prenatal and childhood growth and leisure time physical activity in adult life. Eur J Public Health. 2011;21(6):719-24.

254. Kaseva N, Martikainen S, Tammelin T, Hovi P, Jarvenpaa AL, Andersson S, et al. Objectively measured physical activity in young adults born preterm at very low birth weight. J Pediatr. 2015;166(2):474-6.

255. Kaseva N, Wehkalampi K, Strang-Karlsson S, Salonen M, Pesonen AK, Raikkonen K, et al. Lower conditioning leisure-time physical activity in young adults born preterm at very low birth weight. PLoS One. 2012;7(2):e32430.

256. Hallal PC, Dumith SC, Ekelund U, Reichert FF, Menezes AM, Victora CG, et al. Infancy and childhood growth and physical activity in adolescence: prospective birth cohort study from Brazil. Int J Behav Nutr Phys Act. 2012;9:82.

257. Hardy R, Kuh D, Langenberg C, Wadsworth ME. Birthweight, childhood social class, and change in adult blood pressure in the 1946 British birth cohort. Lancet. 2003;362(9391):1178-83.

258. Richards M, Hardy R, Kuh D, Wadsworth ME. Birth weight and cognitive function in the British 1946 birth cohort: longitudinal population based study. BMJ. 2001;322(7280):199-203.

259. Dahan-Oliel N, Mazer B, Majnemer A. Preterm birth and leisure participation: a synthesis of the literature. Res Dev Disabil. 2012;33(4):1211-20.

260. Brutsaert TD, Parra EJ. Nature versus nurture in determining athletic ability. Med Sports Sci. 2009;54:11-27.

261. Smith L, Gardner B, Aggio D, Hamer M. Association between participation in outdoor play and sport at 10 years old with physical activity in adulthood. Prev Med. 2015;74:31-5.
262. Kuh D, Hardy R, Chaturvedi N, Wadsworth ME. Birth weight, childhood growth and abdominal obesity in adult life. Int J Obes Relat Metab Disord. 2002;26(1):40-7.

263. Elhakeem A, Cooper R, Bann D, Kuh D, Hardy R. Birth weight, school sports ability and adulthood leisure-time physical activity. Med Sci Sports Exerc. 2016.

264. Cai Y, Shaheen SO, Hardy R, Kuh D, Hansell AL. Birth weight, early childhood growth and lung function in middle to early old age: 1946 British birth cohort. Thorax. 2015.
265. Kuh DJ, Wadsworth ME. Physical health status at 36 years in a British national birth cohort. Soc Sci Med. 1993;37(7):905-16.

266. Hardy R, Ghosh AK, Deanfield J, Kuh D, Hughes AD. Birthweight, childhood growth and left ventricular structure at age 60-64 years in a British birth cohort study. Int J Epidemiol. 2016.

267. Pinto Pereira SM, Li L, Power C. Early Life Factors and Adult Leisure Time Physical Inactivity Stability and Change. Med Sci Sports Exerc. 2015;47(9):1841-8.

268. Hebestreit H, Bar-Or O. Exercise and the child born prematurely. Sports Med. 2001;31(8):591-9.

Tucker J, McGuire W. Epidemiology of preterm birth. BMJ. 2004;329(7467):675-8.
Morisaki N, Esplin MS, Varner MW, Henry E, Oken E. Declines in birth weight and fetal growth independent of gestational length. Obstet Gynecol. 2013;121(1):51-8.

271. Wennerstrom EC, Simonsen J, Melbye M. Long-Term Survival of Individuals Born Small and Large for Gestational Age. PLoS One. 2015;10(9):e0138594.

272. Gatford KL, Kaur G, Falcao-Tebas F, Wadley GD, Wlodek ME, Laker RC, et al. Exercise as an intervention to improve metabolic outcomes after intrauterine growth restriction. Am J Physiol Endocrinol Metab. 2014;306(9):E999-1012.

273. Marin L, Weise, I, Adolph, KE. Locomotor development. In: L B, editor. Parenthood in America: An encyclopedia. Denver: ABC-CLIO; 2000. p. 354-8.

274. Bellman M BO, Sege R. Developmental assessment of children. BMJ. 2013;346(e8687).

275. Group WMGRS. Assessment of sex differences and heterogeneity in motor milestone attainment among populations in the WHO Multicentre Growth Reference Study. Acta Paediatr Suppl. 2006;450:66-75.

276. Group WMGRS. WHO Motor Development Study: windows of achievement for six gross motor development milestones. Acta Paediatr Suppl. 2006;450:86-95.

277. Dosman CF AD, Goulden KJ, . Evidence-based milestone ages as a framework for developmental surveillance. Paediatr Child Health. 2012;17(10):561–8.

278. Malina RM BC, Bar-Or O. Motor development. In: Malina RM BC, Bar-Or O, editor. Growth, maturation, and physical activity. 2nd edition ed. United States: Human Kinetics; 2004. p. 195-213.

279. Branta C, Haubenstricker J, Seefeldt V. Age changes in motor skills during childhood and adolescence. Exerc Sport Sci Rev. 1984;12:467-520.

280. Leversen JS, Haga M, Sigmundsson H. From children to adults: motor performance across the life-span. PLoS One. 2012;7(6):e38830.

281. Stodden D, Goodway J, Langendorfer S, Roberton M, Rudisill M, Garcia C, et al. A developmental perspective on the role of motor skill competence in physical activity: an emergent relationship. Quest. 2008;60(2):290-306.

282. Barnett LM, Morgan PJ, Van Beurden E, Ball K, Lubans DR. A reverse pathway? Actual and perceived skill proficiency and physical activity. Med Sci Sports Exerc. 2011;43(5):898-904.

283. Ulrich BD, Reeve TG. Studies in motor behavior: 75 years of research in motor development, learning, and control. Res Q Exerc Sport. 2005;76(2 Suppl):S62-70.

284. Vandorpe B, Vandendriessche J, Vaeyens R, Pion J, Matthys S, Lefevre J, et al. Relationship between sports participation and the level of motor coordination in childhood: a longitudinal approach. J Sci Med Sport. 2012;15(3):220-5.

285. Lubans DR, Morgan PJ, Cliff DP, Barnett LM, Okely AD. Fundamental movement skills in children and adolescents: review of associated health benefits. Sports Med. 2010;40(12):1019-35.

286. Vlahov E, Baghurst TM, Mwavita M. Preschool motor development predicting high school health-related physical fitness: a prospective study. Perceptual and motor skills. 2014;119(1):279-91.

287. Holfelder B SN. Relationship of fundamental movement skills and physical activity in children and adolescents: a systematic review. Psychology of Sport and Exercise 2014;15(4):382-91.

288. Austin D, McNames J, Klein K, Jimison H, Pavel M. A statistical characterization of the finger tapping test: modeling, estimation, and applications. IEEE J Biomed Health Inform. 2015;19(2):501-7.

289. Spirduso WW. Physical fitness, aging, and psychomotor speed: a review. J Gerontol. 1980;35(6):850-65.

290. Da Silva FN, Irani F, Richard J, Brensinger CM, Bilker WB, Gur RE, et al. More than just tapping: index finger-tapping measures procedural learning in schizophrenia. Schizophr Res. 2012;137(1-3):234-40.

291. Kalaycioglu C, Kara C, Atbasoglu C, Nalcaci E. Aspects of foot preference: differential relationships of skilled and unskilled foot movements with motor asymmetry. Laterality. 2008;13(2):124-42.

292. Numasawa T, Ono A, Wada K, Yamasaki Y, Yokoyama T, Aburakawa S, et al. Simple foot tapping test as a quantitative objective assessment of cervical myelopathy. Spine (Phila Pa 1976). 2012;37(2):108-13.

293. Hubel KA, Reed B, Yund EW, Herron TJ, Woods DL. Computerized measures of finger tapping: effects of hand dominance, age, and sex. Percept Mot Skills. 2013;116(3):929-52.

294. Kuh D, Hardy R, Butterworth S, Okell L, Richards M, Wadsworth M, et al. Developmental origins of midlife physical performance: evidence from a British birth cohort. Am J Epidemiol. 2006;164(2):110-21.

295. Kuh D, Hardy R, Butterworth S, Okell L, Wadsworth M, Cooper C, et al. Developmental origins of midlife grip strength: findings from a birth cohort study. J Gerontol A Biol Sci Med Sci. 2006;61(7):702-6.

296. Green D, Lingam R, Mattocks C, Riddoch C, Ness A, Emond A. The risk of reduced physical activity in children with probable Developmental Coordination Disorder: a prospective longitudinal study. Res Dev Disabil. 2011;32(4):1332-42.

297. de Souza MC, de Chaves RN, Lopes VP, Malina RM, Garganta R, Seabra A, et al. Motor coordination, activity, and fitness at 6 years of age relative to activity and fitness at 10 years of age. J Phys Act Health. 2014;11(6):1239-47.

298. Lopes VP, Rodrigues LP, Maia JA, Malina RM. Motor coordination as predictor of physical activity in childhood. Scand J Med Sci Sports. 2011;21(5):663-9.

299. Jaakkola T, Yli-Piipari S, Huotari P, Watt A, Liukkonen J. Fundamental movement skills and physical fitness as predictors of physical activity: A 6-year follow-up study. Scand J Med Sci Sports. 2015;26(1):74-81.

Barnett LM, van Beurden E, Morgan PJ, Brooks LO, Beard JR. Childhood motor skill proficiency as a predictor of adolescent physical activity. J Adolesc Health. 2009;44(3):252-9.
Smith L, Fisher A, Hamer M. Prospective association between objective measures of childhood motor coordination and sedentary behaviour in adolescence and adulthood. Int J Behav Nutr Phys Act. 2015;12(75).

302. Hnatiuk J, Salmon J, Campbell KJ, Ridgers ND, Hesketh KD. Early childhood predictors of toddlers' physical activity: longitudinal findings from the Melbourne InFANT Program. Int J Behav Nutr Phys Act. 2013;10:123.

303. Ridgway CL, Ong KK, Tammelin TH, Sharp S, Ekelund U, Jarvelin MR. Infant motor development predicts sports participation at age 14 years: northern Finland birth cohort of 1966. PLoS One. 2009;4(8):e6837.

304. Aaltonen S, Latvala A, Rose RJ, Pulkkinen L, Kujala UM, Kaprio J, et al. Motor Development and Physical Activity: A Longitudinal Discordant Twin-Pair Study. Med Sci Sports Exerc. 2015;47(10):2111-8.

305. Morley D, Till K, Ogilvie P, Turner G. Influences of gender and socioeconomic status on the motor proficiency of children in the UK. Hum Mov Sci. 2015;44:150-6.

306. Kolobe TH. Childrearing practices and developmental expectations for Mexican-American mothers and the developmental status of their infants. Phys Ther. 2004;84(5):439-53.

307. Alasker F. Timing of puberty and reactions to pubertal changes. In: M R, editor.
Psychosocial disurbances in young people. Cambridge: Cambridge University Press; 1995. p. 37-82.

308. Lai SK, Costigan SA, Morgan PJ, Lubans DR, Stodden DF, Salmon J, et al. Do school-based interventions focusing on physical activity, fitness, or fundamental movement

skill competency produce a sustained impact in these outcomes in children and adolescents? A systematic review of follow-up studies. Sports Med. 2014;44(1):67-79.

309. Morgan PJ, Barnett LM, Cliff DP, Okely AD, Scott HA, Cohen KE, et al. Fundamental movement skill interventions in youth: a systematic review and meta-analysis. Pediatrics. 2013;132(5):e1361-83.

310. Riethmuller AM, Jones R, Okely AD. Efficacy of interventions to improve motor development in young children: a systematic review. Pediatrics. 2009;124(4):e782-92.

311. Fuller D, Sabiston C, Karp I, Barnett T, O'Loughlin J. School sports opportunities influence physical activity in secondary school and beyond. J School Health. 2011;81(8):449-54.

312. Aoki T, Fukuoka Y. Finger tapping ability in healthy elderly and young adults. Med Sci Sports Exerc. 2010;42(3):449-55.

313. Cooper R, Hardy R, Aihie Sayer A, Ben-Shlomo Y, Birnie K, Cooper C, et al. Age and gender differences in physical capability levels from mid-life onwards: the harmonisation and meta-analysis of data from eight UK cohort studies. PLoS One. 2011;6(11):e27899.

314. Malina RM BC, Bar-Or O. Timing and sequence of changes during adolescence. In: Malina RM BC, Bar-Or O, editor. Growth, maturation, and physical activity. United States: Human Kinetics; 2004. p. 307-36.

315. Patton GC, Viner R. Pubertal transitions in health. Lancet. 2007;369(9567):1130-9.

316. Rogol AD, Roemmich JN, Clark PA. Growth at puberty. Journal Adolesc Health. 2002;31(6 Suppl):192-200.

317. Sherar LB, Baxter-Jones AD, Mirwald RL. Limitations to the use of secondary sex characteristics for gender comparisons. Ann Hum Biol. 2004;31(5):586-93.

318. Tanner J. Growth at adolescence. 2nd Ed ed. Oxford: Blackwell; 1962.

319. Rogol AD, Clark PA, Roemmich JN. Growth and pubertal development in children and adolescents: effects of diet and physical activity. Am J Clin Nutr. 2000;72(2 Suppl):521s-8s.

320. Rogol AD. Sex steroids, growth hormone, leptin and the pubertal growth spurt. Endocrine development. 2010;17:77-85.

321. Sorensen K, Mouritsen A, Aksglaede L, Hagen CP, Mogensen SS, Juul A. Recent secular trends in pubertal timing: implications for evaluation and diagnosis of precocious puberty. Horm Res Paediatr. 2012;77(3):137-45.

322. Fisher MM, Eugster EA. What is in our environment that effects puberty? Reproductive toxicology (Elmsford, NY). 2014;44:7-14.

323. Gajdos ZK, Henderson KD, Hirschhorn JN, Palmert MR. Genetic determinants of pubertal timing in the general population. Mol Cell Endocrinol. 2010;324(1-2):21-9.

324. Pierce M HR. Commentary: The decreasing age of puberty—as much a psychosocial as biological problem? Int J Epidemiol. 2012;41(1):300–2.

325. Mishra GD, Cooper R, Tom SE, Kuh D. Early life circumstances and their impact on menarche and menopause. Women's Health (London, England). 2009;5(2):175-90.

326. Malina RM BC, Bar-Or O. Biological maturation: concepts and assessment. In: Malina RM BC, Bar-Or O, editor. Growth, maturation, and physical activity. United States: Human Kinetics; 2004. p. 277-306.

327. Cole TJ, Pan H, Butler GE. A mixed effects model to estimate timing and intensity of pubertal growth from height and secondary sexual characteristics. Ann Hum Biol. 2014;41(1):76-83.

328. Schooling CM. Life course epidemiology: recognising the importance of puberty. J Epidemiol Community Health. 2015;69(8):820.

329. Viner RM, Ross D, Hardy R, Kuh D, Power C, Johnson A, et al. Life course epidemiology: recognising the importance of adolescence. J Epidemiol Community Health. 2015;69(8):719-20.

330. Petersen A, Taylor B, Aldelson J. The biological approach to adolescence: Biological change and psychosocial adaptation. Handbook of adolescent psychology. New York: Wiley; 1980. p. 117–55.

331. Gaysina D, Richards M, Kuh D, Hardy R. Pubertal maturation and affective symptoms in adolescence and adulthood: Evidence from a prospective birth cohort. Dev Psychopathol. 2015;27(4 Pt 1):1331-40.

332. Bacil ED, Mazzardo Junior O, Rech CR, Legnani RF, de Campos W. [Physical activity and biological maturation: a systematic review]. Revista paulista de pediatria. 2015;33(1):114-21.

333. Bertelloni S, Ruggeri S, Baroncelli GI. Effects of sports training in adolescence on growth, puberty and bone health. Gynecol Endocrinol. 2006;22(11):605-12.

334. Cumming SP, Standage M, Gillison FB, Dompier TP, Malina RM. Biological maturity status, body size, and exercise behaviour in British youth: a pilot study. J Sports Sci. 2009;27(7):677-86.

335. Loomba-Albrecht LA, Styne DM. Effect of puberty on body composition. Curr Opin Endocrinol Diabetes Obes. 2009;16(1):10-5.

336. Mrug S, Elliott MN, Davies S, Tortolero SR, Cuccaro P, Schuster MA. Early puberty, negative peer influence, and problem behaviors in adolescent girls. Pediatrics. 2014;133(1):7-14.

337. Davison KK, Werder JL, Trost SG, Baker BL, 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(12):2391-404.

338. Labbrozzi D, Robazza C, Bertollo M, Bucci I, Bortoli L. Pubertal development, physical self-perception, and motivation toward physical activity in girls. J Adolesc. 2013;36(4):759-65.

339. Finne E, Bucksch J, Lampert T, Kolip P. Age, puberty, body dissatisfaction, and physical activity decline in adolescents. Results of the German Health Interview and Examination Survey (KiGGS). Int J Behav Nutr Phys Act. 2011;8:119.

340. Wilk B, Pender N, Volterman K, Bar-Or O, Timmons BW. Influence of pubertal stage on local sweating patterns of girls exercising in the heat. Pediatr Exerc Sci. 2013;25(2):212-20.

341. Day FR, Elks CE, Murray A, Ong KK, Perry JR. Puberty timing associated with diabetes, cardiovascular disease and also diverse health outcomes in men and women: the UK Biobank study. Sci Rep. 2015;5:11208.

342. Cooper R, Blell M, Hardy R, Black S, Pollard TM, Wadsworth ME, et al. Validity of age at menarche self-reported in adulthood. J Epidemiol Community Health. 2006;60(11):993-7.

343. Hardy R, Kuh D, Whincup PH, Wadsworth ME. Age at puberty and adult blood pressure and body size in a British birth cohort study. J Hypertension. 2006;24(1):59-66.
344. Pierce MB, Kuh D, Hardy R. Role of lifetime body mass index in the association between age at puberty and adult lipids: findings from men and women in a British birth cohort. Ann Epidemiol. 2010;20(9):676-82.

345. Cole TJ, Donaldson MD, Ben-Shlomo Y. SITAR--a useful instrument for growth curve analysis. Int J Epidemiol. 2010;39(6):1558-66.

346. Cole TJ, Kuh D, Johnson W, Ward KA, Howe LD, Adams JE, et al. Using Super-Imposition by Translation And Rotation (SITAR) to relate pubertal growth to bone health in later life: the Medical Research Council (MRC) National Survey of Health and Development. Int J Epidemiol. 2016.

347. Kuh D, Muthuri SG, Moore A, Cole TJ, Adams JE, Cooper C, et al. Pubertal timing and bone phenotype in early old age: findings from a British birth cohort study. Int J Epidemiol. 2016.

348. Barnett LM, Lai SK, Veldman SL, Hardy LL, Cliff DP, Morgan PJ, et al. Correlates of Gross Motor Competence in Children and Adolescents: A Systematic Review and Meta-Analysis. Sports Med. 2016.

349. Olstad DL TM, Minaker LM, Taber DR, Raine KD, Nykiforuk CIJ, Ball K. Can policy ameliorate socioeconomic inequities in obesity and obesity-related behaviours? A systematic review of the impact of universal policies on adults and children. Obes Rev. 2016;17(12):1198–217.

350. Kramer D, Droomers M, Jongeneel-Grimen B, Wingen M, Stronks K, Kunst AE. The impact of area-based initiatives on physical activity trends in deprived areas; a quasi-experimental evaluation of the Dutch District Approach. Int J Behav Nutr Phys Act. 2014;11(1):36.

351. Robertson-Wilson JE, Dargavel MD, Bryden PJ, Giles-Corti B. Physical activity policies and legislation in schools: a systematic review. Am J Prev Med. 2012;43(6):643-9.
352. Arnold J, Bruce-Low S, Henderson S, Davies J. Mapping and evaluation of physical

activity interventions for school-aged children. Public health. 2016;136:75-9.
353. Hrysomallis C. Balance ability and athletic performance. Sports Med. 2011;41(3):22132.

354. Sallis JF, Prochaska JJ, Taylor WC. A review of correlates of physical activity of children and adolescents. Med Sci Sports Exerc. 2000;32(5):963-75.

355. Allender S, Hutchinson L, Foster C. Life-change events and participation in physical activity: a systematic review. Health Promot Int. 2008;23(2):160-72.

356. Cummings P. Missing data and multiple imputation. JAMA Pediatrics. 2013;167(7):656-61.

357. Sterne JA, White IR, Carlin JB, Spratt M, Royston P, Kenward MG, et al. Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. BMJ. 2009;338:b2393.

358. Richmond RC, Al-Amin A, Smith GD, Relton CL. Approaches for drawing causal inferences from epidemiological birth cohorts: a review. Early Hum Dev. 2014;90(11):769-80.
359. Heraclides A, Brunner E. Social mobility and social accumulation across the life course in relation to adult overweight and obesity: the Whitehall II study. J Epidemiol Community Health. 2010;64(8):714-9.

360. Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. Int J Obes Relat Metab Disord. 1999;23 Suppl 8:S1-107.

361. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. BMC Public Health. 2005;5:7.

362. Lichtman SW, Pisarska K, Berman ER, Pestone M, Dowling H, Offenbacher E, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med. 1992;327(27):1893-8.

363. Richiardi L, Bellocco R, Zugna D. Mediation analysis in epidemiology: methods, interpretation and bias. Int J Epidemiol. 2013;42(5):1511-9.

364. Lawlor DA, Tilling K, Davey Smith G. Triangulation in aetiological epidemiology. Int J Epidemiol. 2016;45(6):1866-86.

365. Lipsitch M, Tchetgen Tchetgen E, Cohen T. Negative controls: a tool for detecting confounding and bias in observational studies. Epidemiol. 2010;21(3):383-8.

366. Power C, Hertzman C. Social and biological pathways linking early life and adult disease. Br Med Bull. 1997;53(1):210-21.

367. Horikoshi M, Yaghootkar H, Mook-Kanamori DO, Sovio U, Taal HR, Hennig BJ, et al. New loci associated with birth weight identify genetic links between intrauterine growth and adult height and metabolism. Nature Genetics. 2013;45(1):76-82.

368. Wang T, Huang T, Li Y, Zheng Y, Manson JE, Hu FB, et al. Low birthweight and risk of type 2 diabetes: a Mendelian randomisation study. Diabetologia. 2016;59(9):1920-7.

369. Horikoshi M, Beaumont RN, Day FR, Warrington NM, Kooijman MN, Fernandez-Tajes J, et al. Genome-wide associations for birth weight and correlations with adult disease. Nature. 2016;538(7624):248-52.

370. Lawlor D, Richmond R, Warrington N, McMahon G, Davey Smith G, Bowden J, et al. Using Mendelian randomization to determine causal effects of maternal pregnancy (intrauterine) exposures on offspring outcomes: Sources of bias and methods for assessing them. Wellcome Open Research. 2017;2:11.

371. Monden CW, de Graaf ND. The importance of father's and own education for self-assessed health across Europe: an East-West divide? Social Health Illn. 2013;35(7):977-92.
372. Bonomi AG, Goris AH, Yin B, Westerterp KR. Detection of type, duration, and intensity of physical activity using an accelerometer. Med Sci Sports Exerc. 2009;41(9):1770-7.

373. Chen KY, Janz KF, Zhu W, Brychta RJ. Redefining the roles of sensors in objective physical activity monitoring. Med Sci Sports Exerc. 2012;44(1 Suppl 1):S13-23.

374. De Vries SI, Garre FG, Engbers LH, Hildebrandt VH, Van Buuren S. Evaluation of neural networks to identify types of activity using accelerometers. Med Sci Sports Exerc. 2011;43(1):101-7.