Chapter 2
Trajectories and Transitions in Childhood and Adolescent Obesity

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Introduction

The last few decades have seen a dramatic rise in the prevalence of overweight and obesity in most high-income countries. This rise has been seen across all ages, even in very young children (Wang and Lobstein 2006). Obesity is now recognised as one of the most important public health concerns of our time (Dietz 2004), and places a considerable burden on healthcare systems; the cost of obesity to the UK economy was estimated in 2007 to be £15.38 billion per year, including £4.2 billion in costs to the National Health Service (Public Health England 2014). Although recent evidence suggests that the increase in childhood obesity may be abating in some countries (Han et al. 2010), it is too soon to be certain that the flattening of the epidemic will continue, and there is no evidence of a decline in the currently high levels. There is wide variation in the prevalence of childhood overweight between European countries (Fig. 2.1). This variability does not notably follow patterns of geographic area, country wealth or political system and suggests that there is potential for the lowest levels to be seen across the whole of Europe. Being overweight or obese as a child is associated with both short- and long-term health risks, particularly for cardiovascular health (Owen et al. 2009; Friedemann

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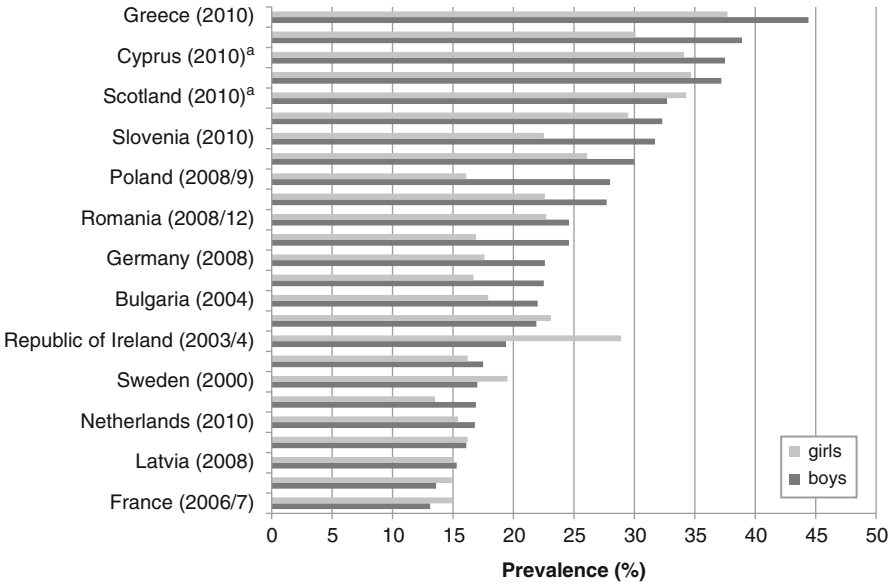

Fig. 2.1 Country-level prevalence of overweight/obesity in children in the European Union Prevalence is shown separately for males (*dark grey*) and females (*light grey*) for countries in the European Union with measured heights and weights, ordered according to prevalence. Data for this figure were obtained from the World Obesity Federation website: http://www.worldobesity.org/site_media/library/resource_images/Child_EU_March_2014_WO.pdf (last accessed 13th March 2014). LD Howe accepts responsibility for accurately converting the data into the above figure [a]Childhood overweight/obesity defined by national reference chart percentiles; for all other countries the International Obesity Task Force (IOTF) criteria were used

et al. 2012) and type two diabetes (Hannon et al. 2005), but also for mental health (Russell-Mayhew et al. 2012) and social outcomes such as educational attainment (von Hinke Kessler Scholder et al. 2012).

A life course perspective can increase our understanding of childhood obesity. There is now strong evidence that pre-natal and early-life factors are involved in the development of childhood obesity (Parsons et al. 1999; Warrington et al. 2013; Howe et al. 2011, 2012; Fairley et al. 2013), and that obesity often begins early in life (Kipping et al. 2008; Reilly and Wilson 2006). Furthermore, despite some interventions with established effectiveness (Picot et al. 2009; Loveman et al. 2011; Ara et al. 2012), adult obesity has proved very difficult to treat (Livhits et al. 2012; Loveman et al. 2011), emphasising the need for early preventative intervention. Utilising longitudinal data from cohort studies enables the study of the dynamic patterns of adiposity development across the life course, and may help to inform the development of intervention strategies. For example, understanding the ages at which obesity tends to develop within children and the degree to which transitions in obesity status occur within children and between childhood and adulthood can provide information about the most appropriate ages for preventative

interventions, and the likely population benefits of policies. Life course studies can also yield information about whether distinct trajectories of obesity development have different impacts on later health, and the ways in which the determinants of obesity alter the patterns of adiposity trajectories.

In this chapter, we review evidence from observational studies (i.e. not including data from follow-up studies of interventions) to meet our aims of (i) describing the evidence on the ages at which obesity develops in contemporary cohorts of children and adolescents, (ii) describing the evidence on the persistence (stability, or tracking) of childhood overweight and obesity, and (iii) discussing the added aetiological insight that can be achieved from longitudinal life course studies of childhood overweight and obesity as compared with cross-sectional studies. We conclude by reflecting on the importance of a life course perspective in studies of childhood obesity, and discussing future directions for life course studies in this area.

When Do Children Become Overweight or Obese?

A vast number of studies have documented the prevalence of childhood obesity. In contrast, relatively little research has focused on the *incidence* of obesity during childhood and adolescence. Susceptibility to becoming overweight or obese is likely to vary across the life course due to changing biological, social and behavioural influences. Understanding when in the life course the highest periods of incidence occur is extremely important, since it influences the likely effectiveness of preventative intervention studies targeted at different age groups. In this section of the chapter we will review the evidence on when overweight and obese children became overweight or obese, and discuss the implications of this body of evidence for the optimal timing of obesity prevention interventions.

One British study that generated an enormous amount of media interest was analysis of the EarlyBird cohort by Gardner et al. (2009). This study examined weight gain (defined as an increase in weight z-score [z-scores are age- and sex-standardised measurements, so an increase in weight z-score means an increase in weight given what would be expected from a child's age and sex]) between birth, five years, and nine years in a cohort of 233 children from the south-west of England. The results showed that weight z-scores (using UK 1990 reference data) increased substantially from birth to five years – mean weight z-scores at birth were 0.02 for males and 0.32 for females (i.e. females were considerably heavier at birth than the average birth weight for females in the 1990 reference dataset, but males were similar at birth to the 1990 reference data), increasing to 0.28 at 0.61 at five years for males and females respectively (i.e. both males and females gained more weight between birth and five years than the average pattern of weight gain in the 1990 reference dataset). From five years to nine years there was a much smaller increase in weight z-scores – 0.28 to 0.39 in males and 0.61 to 0.64 in females. The authors reported strong correlations between weight z-score at

ages five and nine; 0.89 in males and 0.84 in females, both $P < 0.001$. The widely-publicised interpretation of these results was that the preschool period was critical in the development of excess weight gain. The implication of this conclusion is that obesity prevention interventions should focus primarily on preschool children. The authors state that "If excess weight gain were prevented during the preschool age, our data imply that a healthier weight might be maintained thereafter". They also suggest that potential risk factors in school-aged children should be de-emphasised, stating "given the pattern of weight gain reported here, the contribution to childhood obesity by computers, school meals, (lack of) sports clubs, (absence of) school playing fields, and the 'school-run' (all of which are vilified regularly by the media) might be questioned". This analysis of the EarlyBird study, however, included only 233 children with weight as the only measure of adiposity, and measurements at just three ages – birth, five and nine years (Gardner et al. 2009).

In response to the EarlyBird study, Hughes et al. conducted similar analyses in the Avon Longitudinal Study of Parents and Children (ALSPAC) (Hughes et al. 2011b). In this cohort, repeated measurements of weight and height were available from birth to age 15 years for 625 children. The authors conducted similar analyses to Gardner et al. (2009), but made use of z-scores for both body mass index (BMI) and weight, rather than relying only on weight as in the EarlyBird publication. In contrast to the findings of Gardner et al., Hughes et al. reported that mean weight and BMI z-scores increased steadily as the children got older, with the changes being most marked after school entry age (age five years). For example, the mean z-scores for weight in study participants were 0.12 at birth, 0.22 at 1 year, 0.20 at 3 years, 0.18 at 5 years, 0.22 at 7 years, 0.37 at 9 years, 0.54 at 11 years, 0.51 at 13 years, and 0.48 at 15 years. The change in z-scores between birth and five years was not statistically significant, whereas the change in weight z-scores between five and nine years was statistically significant; mean difference in males was 0.26, $P < 0.001$ and in females was 0.10, $P < 0.001$. A similar pattern was seen for BMI z-scores, with no statistical evidence of a difference in the rate of change in z-scores when comparing the periods between one and five years or five and nine years (Hughes et al. 2011b). Thus these results do not corroborate the findings of Gardner et al., and instead suggest that weight status is not set by the time of school entry. Considerable excess weight gain (defined by increases in weight and BMI z-scores) was observed in mid-childhood in this cohort. This casts doubt on the idea that obesity prevention initiatives should be focused on preschool children, and instead implies that policies should target children of all ages.

Differences between the analysis of ALSPAC by Hughes et al. and the EarlyBird paper include a much larger sample size, use of both weight and BMI to define adiposity, and analysis of a greater period of childhood – birth to 15 years in ALSPAC, with detailed data across the first five years, in contrast to three measures at birth, five and nine years in the EarlyBird study. Other factors may also contribute to the different findings, including differences in birth years (1992 in ALSPAC, 1995 in EarlyBird), sociodemographic and geographical factors. Further analysis of the ALSPAC data using alternative methodology also suggests that weight status is not set at school entry. Using data on more than 4,000 children, Hughes et al.

(2011a) found a five-year incidence of obesity (defined as BMI above the 95th centile according to the UK 1990 reference, i.e. using a threshold to define obesity as opposed to using z-scores as a continuous variable in the previous paper) of 5 % between ages seven and 11 years. This was higher than the incidence observed between ages 11 and 15 years (1.4 %). In the subset of the cohort for whom data was available before age seven (N = 549), the incidence of obesity was 5.1 % between three and seven years, 6.7 % between seven and 11 years, and 1.6 % between 11 and 15 years (Hughes et al. 2011a). These data do not support the hypothesis that most excess weight gain occurs before age five years; rather they suggest that mid-childhood is a period during which young people are particularly susceptible to develop obesity.

Other studies have also provided evidence for excess weight gain or obesity development in school-aged children. Most of these studies do not have adiposity data from before age five, and so cannot assess changes in weight or BMI z-scores between birth and school entry, but the studies can provide evidence about the degree of weight gain and obesity development during mid- and late-childhood. In one study in the USA, 5,940 children were assessed in Kindergarten (mean age 5.7 years) and then monitored over the next nine years (Datar et al. 2011). At the start of the study, almost 40 % of the children had a BMI above the 75th centile according to Center for Disease Control (CDC) growth charts. This percentage increased by 5.8 % in the first three years of the study, with a mean change in BMI z-scores of 3.3. These first three years from age 5.7 years were the period with the greatest increases in BMI. In this study there was little further increase in BMI after approximately age 13 years. The authors noted that the increases in mean BMI z-score as the children got older were to a large extent driven by increases in obesity (defined as BMI above the 95th centile); obesity prevalence increased by almost 50 % in the first four years – from 11.9 % in Kindergarten to 17.6 % in the third grade, remaining fairly stable after this age. It is worth noting, however, that these figures do demonstrate that the majority of obese children were already obese at entry into Kindergarten.

Further evidence of excess weight gain during childhood comes from analyses of the US National Health and Nutrition Examination Surveys (NHANES) (Flegal and Troiano 2000). In these (cross-sectional) studies, obesity prevalence was found to be higher among 9–11 year olds compared with six to eight year olds, suggesting that some children gain excessive weight and become obese during mid-childhood. These analyses, however, found that there was little difference in the obesity prevalence comparing nine to 11 year olds with older adolescents up to age 17 years (Flegal and Troiano 2000; Ogden et al. 2010).

Support for limited change in obesity prevalence during adolescence (i.e. after approximately age 11 years) is also provided by analysis of the ALSPAC data, which showed only a 1.4 % incidence of obesity between ages 11 and 15 years (Hughes et al. 2011a), and by several other studies. Wardle et al. reported that persistent obesity was generally established before age 11 (Wardle et al. 2006). In analyses of data from 5,863 English school children, the authors reported that obesity prevalence at entry to the study (age 11–12 years) was almost 25 %. During

five years of follow-up, the obesity prevalence did rise – but in the majority of cases this was because of overweight children becoming obese; the proportion of children in the healthy BMI category remained stable. Little evidence was found of new cases of overweight or obesity emerging between ages 11 and 17 years. However, the overall percentages at each age do mask some movement between categories – 7.6 % of children moved from overweight/obese to a normal BMI, and 7.0 % of students moved from normal weight to overweight/obese. A study of Belgian adolescents also supports the notion of limited increase in obesity prevalence after mid-childhood, which showed a stable prevalence between ages 13 and 17 years (Hulens et al. 2001).

Not all the evidence agrees about the stabilisation of obesity prevalence after mid-childhood. For example in an analysis of 2,379 school children in the USA, Kimm et al. showed that the prevalence of both overweight and obesity continued to rise from ages 9–19 years in both black and white females, with very large increases in the late teenage years (Kimm et al. 2002). At age nine 30.6 % of black females and 22.4 % of white females were overweight, rising to 39.3 % and 24.1 % respectively at age 16 and 56.9 % and 41.3 % at age 19.

A number of studies have also examined factors that affect the age of obesity onset, for example finding that maternal obesity (Gordon-Larsen et al. 2007), black ethnicity (Gordon-Larsen et al. 2007), and genetic factors (Hinney et al. 2007) are associated with younger age at becoming overweight/obese.

Thus overall, the evidence suggests rises in the mean level of age-adjusted adiposity and in the prevalence of overweight and obesity from birth across childhood until at least age 11, with some conflicting evidence over whether or not obesity prevalence stabilises after age 11 or rises further across adolescence. This suggests that there is no one age range of childhood and adolescence that should be the main focus of preventative interventions. However, in addition to considering the age distribution of obesity onset, decisions about the timing of interventions should also consider the likely success of intervening at different ages, cost-effectiveness of alternative policies, and the age at which health complications of obesity begin to develop. In relation to cardiovascular disease, childhood adiposity from around age 7–9 is what appears to matter, with weak, inverse or null associations at younger ages (Owen et al. 2009). It is also important to remember that regardless of how obesity prevalence changes across childhood and adolescence, some people will remain in the healthy BMI range across the whole of childhood but become overweight or obese as adults (Whitlock et al. 2005). For example, in a study in which 1,520 people were followed-up over ten years from the age of 14 (Patton et al. 2011), 33 % of participants were overweight (BMI $\geq$25 kg/m^2) at the end of the study (age 24 years). Of these, approximately 40 % had been consistently in the normal BMI range during adolescence, and approximately 80 % had at least one BMI measurement in the normal range at some point during the ten years of follow-up. Approximately half of the obese 24 year-olds had never been classified as obese during adolescence. Various life course events during adulthood have been shown to be associated with adiposity gains in adulthood, including pregnancy (Fraser et al.

2011), suggesting that these may be sensitive periods in adulthood and potentially suitable timings for preventative interventions (Tanentsapf et al. 2011).

Once a Child Becomes Overweight or Obese, How Likely is He/She to Remain So?

Understanding the incidence of overweight and obesity only tells part of the story – the degree of stability or persistence of overweight is also important, i.e. once a child becomes overweight or obese, how likely is he/she to remain so? This concept is referred to as 'tracking' in the epidemiological literature. Information on the degree of tracking of overweight and obesity is valuable for public health planning, since it informs the need for and likely long-term efficacy of preventative interventions in childhood.

A systematic review of the degree of tracking of overweight between childhood or adolescence into adulthood was published in 2008 (Singh et al.). The review included papers listed in MEDLINE, EMBASE and CINAHL up to February 2007. Publications were included if they reported at least one anthropometric measurement during youth ($\leq$18 years) and one anthropometric measurement during adulthood ($\geq$19 years), using body mass index (BMI), skin fold thickness or waist circumference to define body weight status in a general population study (i.e. excluding follow-up of intervention studies or studies in specific clinical populations or those concentrating only on a population that had experienced a certain exposure). Studies had to report an odds ratio (OR) or risk ratio (RR) for overweight youth becoming overweight adults. From the searches, the authors identified 18 studies described in 25 publications. All of these studies found that being overweight or obese during youth was associated with an increased risk of being overweight or obese in adulthood (Singh et al. 2008).

Four studies included in the systematic review stratified their analysis by weight status in childhood and all found that the odds of persistent overweight/obesity were greater with increasing level of overweight. When studies included more than one measure of overweight/obesity during youth, most found that the degree of tracking was greater at older ages – this could represent stronger tracking at older ages or may be a result of the shorter time intervals between the measurement in youth and measurement in adulthood, or of height changing less at older ages.

There was a considerable degree of heterogeneity in the estimates of the degree of tracking of overweight/obesity from youth into adulthood. The authors rates each included study as 'high' or 'low' methodological quality based on whether they met a list of pre-defined criteria for the conduct and reporting of observational studies. Amongst studies rated as high-quality, relative risks for overweight children becoming overweight adults ranged from 2 to 10. Relative risks for obese youth becoming obese adults were generally higher. Heterogeneity between studies may have arisen for multiple reasons: the studies were based in different populations with

varying levels of obesity, ages of measurement in both youth and adulthood varied considerably, measurement protocols and definitions of overweight and obesity varied, as did statistical analysis procedures. The studies included in this systematic review are necessarily limited to populations who are now adults; it is therefore possible that the degree of tracking of overweight and obesity from childhood into adult may differ in younger populations who are growing up now, during the obesity epidemic, and in the future. A further consideration is that all studies included in the review were conducted in high-income countries, and so the extent of tracking within low- and middle-income countries is largely unknown.

In addition to the findings about persistence of overweight and obesity from childhood into adulthood described above, studies have also looked at tracking *within* childhood and adolescence. Amongst 2,747 female and 2,488 male participants of ALSPAC, the majority (70.7 % of females, 74.6 % of males) had a BMI within the normal range at both 9–12 and 15 years, whilst 13.4 % of females and 12.2 % of males were overweight/obese at both ages (Lawlor et al. 2010). Amongst female participants, 11.1 % moved from being overweight/obese at age 9–12 to normal BMI at age 15, and 4.8 % moved from normal BMI to overweight/obese; these percentages were 9.1 % and 4.1 % in males. Thus in this population, between 9–12 and 15 years, a greater number of children moved from being overweight/obese to having a normal BMI than developed obesity. Wright et al. carried out further analyses of ALSPAC data, looking at the tracking of body composition between ages seven and 11 using BMI and leg-to-leg bioelectrical impedance (BIA) (Wright et al. 2010). The BIA data were used to generate measures of lean and fat mass independent of height, gender and age, which were then internally standardised, with the 85th and 95th centiles considered as over-fat and very over-fat. In the more than 6,000 children included in their analysis, Wright et al. showed that of the children who were overweight by BMI at age seven, 21 % had reverted to a normal BMI by age 11 and 16 % had become obese. Using the BIA data, of those children who were over-fat at age seven, 12 % progressed to being very over-fat at age 11, 33 % remained over-fat and 55 % dropped to the normal fat category. Of the children who were very over-fat at age seven, 57 % remained so at age 11, 30 % dropped to over-fat and 13 % dropped to the normal fat category. There was therefore a slightly higher degree of tracking when using BMI as the measure of adiposity compared with BIA-assessed fat mass. In a separate analysis of ALSPAC data, Reilly et al. (2011) showed that 34 % of overweight (by BMI) children at age seven become obese by age 13. The OR for progression from overweight at age seven to obese at age 13 was 18.1 (95 % CI 12.8–25.6).

Wardle et al. followed more than 5,000 school children in London between the ages of 11 and 15 years (Wardle et al. 2006). The year to year correlations between BMI measurements were extremely high – 0.94 for a one year interval, reducing to 0.90, 0.86, and 0.82 for two, three, and four year intervals. A similar degree of tracking was observed for measurements of waist circumference, with correlations going from 0.86 for a one year interval between measurements to 0.70 for a four year interval. Using correlation coefficients to assess tracking can, however, be

problematic since large correlations can arise even in the absence of strong tracking if the average slope over time is low.

Several studies have examined factors that may affect the degree of tracking of adiposity. There is little consistency in findings about whether or not tracking differs between males and females (Singh et al. 2008). Some evidence suggests a stronger degree of tracking in overweight children whose parents are obese (Wright et al. 2010). Socioeconomic position (SEP) and ethnicity, both known to be associated with childhood obesity (El-Sayed et al. 2011, 2012; Singh et al. 2010), may also affect tracking. One small Danish study (N = 384) found that low SEP participants were twice as likely to maintain overweight between ages 8–10 years and 14–16 years compared with high SEP participants, and twice as likely to develop overweight between the two time points (Kristensen et al. 2006). Analyses of the ALSPAC cohort found that there were no clear maternal education differences in the proportion of overweight seven year-olds becoming obese by age 15. There was, however, some indication that normal BMI seven year olds were slightly more likely to become overweight by age 15 if they were from lower maternal education categories, and children from higher maternal education categories are more likely to return from overweight/obese at age seven to a normal BMI at age 15 compared with children from lower maternal education groups (Howe 2013). A study looking at racial differences in overweight/obesity tracking between childhood and adulthood in a biracial cohort in the USA found that, despite initial BMI being similar in black and white children (aged 5–14 years), BMI increased more with age in black than white individuals, and overweight black children were more likely to become obese adults (84 % of black girls compared with 65 % of white girls, with similar findings in males) (Freedman et al. 2005).

In addition to tracking of adiposity itself, studies have also revealed persistence of obesity-related behaviours across childhood and adolescence. In a study of 296 children in Victoria, Australia, parents reported their child's television viewing and consumption of frequency of fruit, vegetable, and energy-dense sweet and savoury snacks in 2002/3 and again in 2006 and 2008 (Pearson et al. 2011). Standardised stability coefficients (interpreted as correlation coefficients, i.e. ranging from 0 (no relationship) to 1 (perfect relationship)) between the three time points ranged from 0.65 to 0.73 for television viewing, 0.52 to 0.86 for vegetable consumption, 0.73 to 0.89 for fruit consumption, 0.41 to 0.65 for consumption of energy-dense sweet snacks, and 0.40 to 0.67 for consumption of energy-dense savoury snacks. No consistent differences in tracking were seen between males and females or between children who were five to six or 10 to 12 at baseline.

Jones et al. (2013) conducted a systematic review of tracking of physical activity and sedentary behaviour within early childhood (between birth and age six years) or between early childhood and mid-childhood (6–12 years). Eleven studies were included in their review. The reported tracking coefficients for physical activity were moderate in the majority (60 %) of studies, with just 4 % of studies reporting high tracking; the considerable degree of measurement error in physical activity data may, however, contribute to the apparently moderate tracking. There was a greater degree of tracking for sedentary behaviour, with 33 % of studies reporting high

tracking, and a further 50 % reporting moderate tracking. A further review demonstrated a moderate degree of tracking of sedentary behaviours within young people (Biddle et al. 2010). The authors identified 21 studies, with tracking coefficients from 0.08 to 0.73 for TV viewing, from 0.18 to 0.52 for electronic game/computer use, from 0.16 to 0.65 for total screen time, and from −0.15 to 0.48 for total sedentary time. Tracking coefficients tended to be higher with shorter intervals between measurements. Telema (2009) carried out a systematic review of the stability of physical activity from childhood into adulthood, reporting statistically significant evidence of tracking of physical activity levels across the life course, but with only a moderate degree of tracking between most periods of life and lower tracking in transition periods such as between adolescence and adulthood. The review also indicated that tracking tended to be lower in females compared with males.

Thus in conclusion, there is evidence for a moderate to strong degree of tracking across childhood and adolescence and between childhood and adulthood for both adiposity itself and adiposity-related behaviours such as diet, physical activity and sedentary behaviours. There is, however, some degree of movement both into and out of obesity at all stages of the life course, highlighting the need for preventative interventions across the life course.

Aetiological Insight from Studying Childhood Adiposity Trajectories

Longitudinal analyses of trajectories of adiposity across childhood and adolescence can be extremely useful for advancing our understanding of the aetiology of obesity and its long term health complications. One important scientific question that can only be addressed with life course studies is the extent to which one's obesity history matters for health – i.e. is final attained level of adiposity the key determinant of health, or do different life course adiposity patterns that converge on the same final size have varying health consequences? In the case of childhood obesity, a related question is the extent to which the long-term health sequelae of increased adiposity during childhood are due to permanent and irreversible damage to organs and tissues as a result of being overweight as a child, or the extent to which these associations are driven by tracking of adiposity levels such that overweight children tend to become overweight adults, with damage to tissues and organs only taking place in adulthood. These two scenarios have very different public health implications. In the first instance, if being overweight or obese as a child results in permanent damage to bodily systems, the public health impetus might be on preventing the development of obesity. However, if the second theory is correct and associations between childhood obesity and health in later life are driven solely by the tracking of adiposity, interventions that treat obesity in adulthood could potentially be as effective for preventing disease as those that prevent the development of obesity. Of course other factors need to be considered in this equation, including the cost and effectiveness of both types of intervention.

The evidence for these alternative hypotheses is somewhat mixed. There is now a very large body of evidence demonstrating that increased adiposity is cross-sectionally and prospectively associated with adverse cardiometabolic health even in very young children, using measures such as circulating levels of insulin, glucose or lipids, or even measurements of the structure and function of the heart (de Jonge et al. 2011; Falaschetti et al. 2010). However, two recent studies provide some encouraging evidence that obese children who normalise their weight status can also improve their cardiometabolic health. In analyses of 5,235 participants of the ALSPAC cohort, prospective associations were demonstrated between BMI, waist circumference and DXA-assessed total body fat mass at age 9–12 years and cardiovascular risk factors (systolic and diastolic blood pressure, concentrations of fasting glucose, insulin, triglycerides, low density lipoprotein cholesterol and high density lipoprotein cholesterol) assessed at age 15–16 years (Lawlor et al. 2010). Females in the cohort who were overweight or obese at ages 9–12 but whose BMI was in the normal range at ages 15–16 had similar odds of adverse levels of cardiometabolic risk factors to those females who were normal BMI at both ages. This pattern was less evident in males; males who normalised their BMI status between the two time points had higher odds of high systolic blood pressure, high concentrations of triglycerides and insulin, and low concentrations of high density lipoprotein cholesterol compared with those who were normal BMI at both ages, but their odds of having adverse levels of all cardiometabolic risk factors were lower than in those who remained overweight/obese at both ages. The continued elevated cardiometabolic risk in these males may be due to greater adiposity; mean BMI was higher in those who were overweight or obese at the first time point and had a BMI in the normal range at the second time point compared with those whose BMI was in the normal range at both ages. A similar research question was addressed in a study of 6,328 participants from four studies with a mean age of measurement in childhood of 11.4 years (SD = 4.0) and follow-up in adulthood on average 23.1 years later (SD = 3.3 years) (Juonala et al. 2011). The authors showed that participants who had been overweight or obese during childhood but whose BMI was in the normal range during adulthood have a similar risk of type 2 diabetes, hypertension, adverse levels of LDL-cholesterol, HDL-cholesterol, triglycerides, and an adverse carotid artery intima-media thickness as compared with participants whose BMI was in the normal range during both childhood and adulthood. Both of these studies therefore provide encouraging evidence that childhood obesity may not in itself cause permanent and irreversible damage to cardiometabolic systems, and that interventions that are successful in treating and reversing childhood obesity may be expected to improve the long-term cardiometabolic health of those people who are successfully treated.

The study by Juonala et al. (2011) also showed a similar level of cardiometabolic risk in participants who were overweight or obese in both childhood and adulthood as compared with participants who had a normal BMI during childhood but were overweight or obese as adults, suggesting that it may be the final attained adiposity level that influences cardiovascular health, and that the trajectory a person has followed to reach that level may be less important. Other studies, however, do

not all support this hypothesis. For instance there is evidence that the duration of obesity is associated with mortality independent of BMI level. Abdullah et al. (2011) conducted analysis of 5,036 participants from the Framingham cohort study. Using data from 48 years of follow-up, they showed that the adjusted hazard ratio for mortality increased with the number of years of obesity; compared with those who were never obese, being obese for 1–4.9, 5–14.9, 15–24.9, and ≥25 years was associated with adjusted hazard ratios for all-cause mortality of 1.51 (95 % confidence interval (CI) 1.27–1.79), 1.94 (95 % CI 1.71–2.20), 2.25 (95 % CI 1.89–2.67) and 2.52 (95 % CI 2.08–3.06), respectively. Similar patterns were found for cause-specific mortality from cardiovascular disease and cancer. The relationships were robust to adjustment for current BMI, and were reduced but not completely eliminated by adjustment for potential intermediate factors such as incident cardiovascular disease and diabetes or biomedical risk factors.

A large body of research has focused on the topic of whether infancy is a sensitive period in the development of obesity and cardiovascular research. Several studies have concluded that rapid growth in infancy is importantly associated with subsequent obesity and adverse cardiovascular health (Ong et al. 2000, 2009; Ong and Loos 2006). However, these studies have generally only examined changes in weight (or weight adjusted for height) during infancy, and have not considered the associations of growth in later periods of childhood with the same outcomes. Without these comparisons (i.e. determining whether weight change in infancy is more strongly associated with later outcomes than is weight change in later childhood) it is difficult to conclude that infancy is a sensitive period. Studies on this topic have used a variety of statistical methods, often less than ideal (Tilling et al. 2011c; Tu et al. 2013) – for instance, relying on repeated z-scores, an approach which does not model the clustering of measurements within individuals and generally can only include people with complete, non-missing, data at all ages. In ALSPAC and Probit, two large studies with lots of repeated measurements of growth, we have used multilevel models to define growth trajectories across childhood (Howe et al. 2013c; Tilling et al. 2011b); an approach which is appropriate for repeated measures data and which can incorporate missing data under a missing at random assumption. In these analyses, we have not found strong support for infancy as a sensitive period in relation to a wide range of cardiometabolic risk factors, including blood pressure (central and peripheral), glucose, lipids, insulin, and non-alcoholic fatty liver disease (Howe et al. 2010; Tilling et al. 2011a; Anderson et al. 2014).

Longitudinal studies also permit in-depth examination of the ways in which the determinants of obesity act throughout the life course, potentially providing deeper aetiological insight than would be possible in cross-sectional studies. For example, genome-wide association studies have identified genetic variants that are associated with obesity and greater BMI in adulthood. Since BMI changes in childhood can result from changes in both height and weight, examining the effect of the genetic variants on growth trajectories in childhood can provide improved understanding of the potential mechanisms through which the genes are acting (Paternoster et al. 2011; Warrington et al. 2013; Cousminer et al. 2013). Risk scores of obesity-related genetic variants have been shown to be associated with changes in height, weight

and BMI across infancy and childhood, with the magnitude of associations changing across the life course (Warrington et al. 2013; Elks et al. 2010, 2012; Hardy et al. 2010). Similar analyses have also been conducted for trajectories of height and blood pressure across childhood and adolescence (Howe et al. 2013a; Paternoster et al. 2011). Longitudinal studies have also provided insight into inter-generational and cross-cohort differences in patterns of growth (Li et al. 2008; Li et al. 2009), and the influence of social determinants on the development of obesity risk (Kakinami et al. 2014; Ding and Gebel 2012; Walsemann et al. 2012); for example analysis of the ALSPAC cohort demonstrated that maternal education differences in offspring BMI begin at an earlier age for females than for males and widen across childhood (Howe et al. 2011), and a study of the Born in Bradford cohort showed that despite lighter birth weights, Pakistani infants gain weight and length quicker than white infants, indicating that the greater risk of obesity in the British Pakistani population may have its origins in very early life (Fairley et al. 2013).

Concluding Remarks

In this chapter, we have shown that life course studies of childhood obesity can offer insight into whether the incidence of obesity differs between periods of childhood and adolescence, and the degree to which childhood overweight and obesity persists across the life course, and we have discussed the utility of such information in assessing the potential consequences of targeting obesity prevention interventions at specific age groups. Our review of the literature concluded that incidence of overweight and obesity was high across all ages, and that despite some movement between categories, children who become overweight or obese are likely to remain overweight or obese. Together, this body of evidence suggests that obesity prevention policies should target all ages. We have also shown that longitudinal studies can provide deeper understanding of aetiological questions than is possible in cross-sectional studies – for instance such studies can evaluate whether the life course adiposity trajectory influences health independently of the final attained adiposity level, or can provide insight into the timing and mechanisms through which determinants of obesity exert their influence. As with all life course studies, research on the life course epidemiology of childhood obesity faces methodological challenges (Davey Smith et al. 2009; Davey Smith et al. 2007; Ness et al. 2011; Howe et al. 2013b). Cohort effects are likely to be considerable – today's children are growing up in an environment that is far more obesogenic than the environment experienced by today's adults. Continued study of new cohorts is therefore crucial (Cooper et al. 2012), as is utilising innovative methodological tools such as Mendelian Randomization (Davey Smith et al. 2009; Lawlor et al. 2008; Palmer et al. 2012), cross-cohort comparisons (Brion et al. 2011) and family-based designs (Brion 2013; Howe et al. 2012; Lawlor and Mishra 2009) that improve the strength of causal inference that can be reached from observational data. Given the growing burden of non-communicable diseases in low- and middle-income settings (Miranda

et al. 2008), life course studies in these settings are also important, since the vast majority of literature in this area to date comes from high-income countries, and the findings do not necessarily generalise.

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