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CLS Working Paper 2015/02

March 2015

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Additive association of maternal and paternal body mass index on weight status trajectories from childhood to mid-adulthood in the 1970 British Cohort Study

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ABSTRACT

This study aimed to (i) describe the weight status trajectories from childhood to mid-adulthood and (ii) investigate the influence of maternal and paternal body mass index (BMI) on offspring's trajectories in a nationally representative study in Great Britain. The sample comprised 4174 (43% male) participants from the 1970 British Cohort Study with complete BMI data at ages 10, 26, 30, 34, and 42 years. Individuals' weight status was categorised as overweight/obese or non-overweight/obese at each age, and trajectories of weight status from 10 to 42 years of age were assessed. Sex-stratified multinomial logistic regression models were used to assess associations of maternal and paternal BMI with trajectory group membership, adjusting for potential confounders (e.g., socioeconomic position and puberty). 30% of individuals were never overweight/obese (reference trajectory), 6%, 44% and 8% had childhood, early- and mid-adulthood onset of overweight/obesity (respectively), and 12% other trajectories. In fully adjusted models, higher maternal and paternal BMI significantly increased the risk of childhood (relative risk ratio: 1.2-1.3) and early adulthood onset (1.2) of overweight/obesity in both sexes. Relative risk ratios were generally higher for maternal than paternal BMI in females but similar in males. Early puberty also increased the risk of childhood (1.8-9.2) and early adulthood onset (3.7-4.7) of overweight/obesity. Results highlight the importance of primary prevention, as most individuals remained overweight/obese after onset. Maternal and paternal BMI had additive effects on offspring weight status trajectories across 32 years of the life course, suggesting that prevention/intervention programmes should focus on the whole family.

Keywords: obesity, trajectories, tracking, life course, parental BMI, socioeconomic factors.

INTRODUCTION

The obesity epidemic is one of the greatest public health concerns worldwide (World Health Organisation, 2011; Health and Social Care information Centre, 2014). The increase in the prevalence of obesity has been seen in both adults and children (World Health Organisation, 2011). In England, the prevalence of overweight and obesity in adults has increased from 58% and 49% in 1993 to 67% and 57% in men and women respectively in 2012 (Health and Social Care information Centre, 2014). Obesity in children (2-15 years) has also increased, with peaking rates of 18% and 19% of boys and girls (respectively) in 2004 (Health and Social Care information Centre, 2014). This is worrying since obesity has been shown to track from childhood through to adulthood (Reilly et al., 2003; Venn et al., 2007; Brisbois, Farmer, & McCargar, 2012), and both childhood and adulthood obesity are associated with adverse health outcomes (Park, Falconer, Viner, & Kinra, 2012; Park, Sovio, Viner, Hardy, & Kinra, 2013; Prospective Studies Collaboration, 2009; Reilly et al, 2003).

Parental BMI is one of the most consistent factors associated with offspring obesity (Monasta et al, 2010; Parsons, Power, Logan, & Summerbell, 1999). For example, looking at the association of child to adulthood socioeconomic position (SEP) and obesity in the 1958 British birth cohort, Power, Manor and Matthews (2003) found a significant association of higher maternal and paternal BMI with obesity at 33 years, even when controlling for adulthood (in males) and childhood SEP, and education at 33 years. However, this study (as most in the literature) has only considered obesity at one time-point in adulthood (Parsons et al, 1999), and the influence of parental BMI on different trajectories of weight status (such as the persistence of overweight/obesity from childhood to adulthood) remains largely unknown. Concurrently, there is a lack of studies investigating the association of maternal and paternal BMI with offspring overweight and obesity until later than early-adulthood (Parsons, et al, 1999), much of the existing literature is only observational and suffers from potential confounding effects that were not accounted for, such as SEP and early maturation (Monasta et al, 2010).

Identifying the exact mechanisms through which parental obesity leads to later obesity is difficult, because of the many possible mediators and confounders involved in this

relationship (Monasta et al, 2010; Parsons et al, 1999). Clarifying whether parental BMI has a significant influence in their offspring's lifelong weight status trajectories is important, given the high prevalence of overweight /obesity in adults (World Health Organisation, 2011; Health and Social Care information Centre, 2014), and that it is potentially easier to directly intervene in parents than in children to reduce their overweight (e.g. through change in determinants like diet and physical activity) (Golan, 2006; Monasta et al, 2010). Studying large samples where basic measures of the risk factors have been collected has been suggested as a potential approach to deal with the inherent difficulty in measuring the risk factors associated with overweight/obesity (Parsons et al, 1999). The 1970 British Cohort Study (BCS70) has recently released the data for the 42-year follow-up, presenting a remarkable opportunity to investigate the influence of parental BMI on offspring's weight status trajectories from childhood to mid-adulthood, addressing some of the main limitations of previous studies.

The aims of this study were to (i) describe weight status trajectories from childhood to mid-adulthood in the BCS70 and (ii) investigate the separate influences of maternal and paternal BMI on offspring's weight status trajectories.

METHODS

Sample

The BCS70 has followed just under 17,200 people born in Great Britain (England, Scotland, and Wales) in one week in April 1970 from birth onwards (Centre for Longitudinal Studies, 2014). Nine main data collections have taken place at 0 (1970), 5 (1975), 10 (1980), 16 (1986), 26 (1996), 30 (2000), 34 (2004), 38 (2008), and 42 (2012) years of age. At the most recent sweep, 57% (N = 9842) of the participants were still in the study and they remained broadly representative of the national population of men and women of the same age. The sample for the present study comprised 4174 (1782 male; 2392 female) participants with BMI data at 10, 26, 30, 34, and 42 years of age. The sample was not noticeably different from the 4625 (53%) participants who remained in the study and had BMI data at the most recent sweep (42 years) but did not meet the eligibility criteria for the present study.

Participants' body mass index

Weight and height were measured by community medical officers, health visitors or school nurses at 10 and 16 years of age according to standard protocols, and were self-reported in questionnaires at 26 years and face-to-face interviews at 30, 34, and 42 years of age. The amount of data collected at age 16 was considerably less than at all other ages (e.g., valid BMI: 5723 participants at 16 years versus ≥7303 participants at other ages). This was due to a prolonged industrial action carried out by teachers in 1986, who were responsible for conducting the educational tests; the delayed start of the survey meant that a high number of children had left school before assessments could be undertaken and information for many of these could not be obtained. As such, data from the 16-year follow-up was not used for the current study. Because only height was assessed at 5 years of age and no anthropometry was collected at 38 years of age, the 5- and 38-year follow-ups were also not used in the present study. It was therefore possible to compute BMI (weight (kg)/ height $(m)^2$) at ages 10, 26, 30, 34, and 42 years. Due to the small number of obese participants at each age, weight status at each age was categorised as either "1 = overweight/obese" or "0 = non-overweight/obese". In adulthood, participants' BMI was classified as overweight/obese when ≥25 kg/m^2, and non-overweight/obese if <25 kg/m^2. At 10 years of age, the International Obesity Task Force BMI cut-off points for overweight (Cole, Bellizzi, Flegal, & Dietz, 2000) were used to classify cohort members as overweight/obese or non-overweight/obese.

Parental body mass index and confounding variables

Maternal and paternal weight (kg or stones and pounds) and height (meters or feet and inches) were reported by the mother when cohort member was 10 years of age. Data in stones/pounds and feet/inches were transformed into kg and meters respectively, and parental BMI was subsequently calculated. Birth weight (in grams) and gestational age at birth (in completed weeks) was recorded by the midwife who had undertaken the delivery or the senior midwife. Duration of breastfeeding was reported by the mother as "never breastfed", "<1 month", "≥1 month but <3 months", and "≥3 months", when the

participants were aged five years. A binary breastfeeding variable with the responses "<3 months" or "≤3 months" was computed. Evidence of puberty at age 10 years was assessed and recorded during the medical examination by a Community Medical Officer, a health visitor or the school nurse, by answering "yes"/"no" to the question "Did your examination reveal (…) Any evidence of puberty?". Maternal and paternal occupation was reported by the mother at the 10-year follow-up, and classified according to the 1980 OPCS Classification of Occupations (Office of Population Censuses and Surveys, 1980). A binary socio-economic position (SEP) variable with the responses "high SEP" (if the mother, father, or both belonged to a professional or managerial social class) or "medium-low SEP" (if both parents belonged to skilled, partly-skilled or unskilled social classes) was derived. Maternal education was also reported by the mother at the 10-year follow-up, and a binary variable comprising "A-levels or above" (A-levels are the requirement for university access) and "below A-levels" was computed.

Weight status trajectories

Weight status trajectories between 10 and 42 years of age were defined manually for males and females separately, using a three step procedure. To maximise sample size, the closely spaced 26 and 30 year surveys were collapsed into one time-point; participants who were overweight/ obese at either age or both ages were coded as overweight/obese, and participants who were normal weight at both ages were coded as normal weight. We first tabulated all possible combinations of the binary weight status variable across the four ages to produce 16 trajectories (i.e., 2^4) and examined the frequency of participants belonging to each trajectory (see appendix 1). Second, all trajectories comprising <5% of the sample were collapsed into one trajectory called "other"; those comprising ≥5% of the sample were retained as individual trajectories: "never overweight/obese", "overweight/obesity onset at 42 years", "overweight/obesity onset at age 34 years", "overweight/obesity onset at ages 26 or 30 years", and "overweight/obesity onset at age 10 years". Thirdly, to improve power and because both trajectories represented two timings of early adulthood onset, the following two trajectories were collapsed into one: "overweight/obesity onset at age 34 years" and "overweight/obesity onset at ages 26 or 30 years". The final outcome variable consisted of five trajectories: 1) "never overweight/obese", 2) "overweight/obesity onset in

mid-adulthood", 3 "overweight/obesity onset in early-adulthood", 4) "overweight/obesity onset in childhood", and 5) "other trajectories".

Statistical Analyses

Descriptive statistics were used to describe the sample and the trajectories of weight status. All continuous variables were tested for normality of distribution with the Shapiro-Francia test and found to be non-normally distributed. As such, Mann-Whitney U tests were used to assess differences in continuous and ordinal variables between sexes, and between those included and excluded from analyses. Chi-square tests were used to assess differences in categorical variables between sexes, and between those included and excluded from analyses.

Analyses were conducted separately for males and females. This was to test whether parental BMI affects weight status trajectories differently in males and females, and because of the sex inequality in the consequences of overweight/obesity, potential differences in the association of early life factors (such as SEP) with adulthood overweight/obesity (Heraclides, Witte, & Brunner, 2008; Khlat, Jusot & Ville, 2009; Pudrovska, Logan & Richman, 2014), and the significant differences in the prevalence of weight status trajectories. Univariable multinomial logistic regression was used to assess the associations of maternal and paternal BMI (separately) with weight status trajectory membership. Multivariable multinomial logistic regression was then used to test how these associations change after adjusting for the confounding effect of birth factors (step 1) and puberty (step 2), maternal education and SEP (step 3). This was firstly done for maternal and paternal BMI separately, and subsequently with maternal and paternal BMI in the same model. Interactions between maternal and paternal BMI were also tested. Post-hoc sensitivity analyses were undertaken using parental zBMI instead of BMI. Alpha-value was set at 0.05, with Bonferroni correction applied to multiple comparisons. All analyses were conducted in Stata (v.12) statistical software package.

RESULTS (see end for tables)

Details of the 4174 participants included for analyses can be seen below on Table 1. More females (57.3%) than males were included for analyses. Significantly more females showed evidence of puberty at 10 years than males (p<0.001), whereas males had significantly higher birth weight and BMI at 42 years of age than females (p<0.001). All other variables were broadly similar for both sexes.

Trajectories of weight status

Significantly more females than males were never overweight/obese and had mid-adulthood onset of overweight/obesity (p<0.001), whereas the prevalence of males having early adulthood onset of overweight/obesity was roughly double the prevalence of females (62.7% versus 30.6%; p<0.001). Prevalence of childhood onset of overweight/obesity was the same in females and males (see Table 2). Importantly, of those overweight/obese in childhood (229 females; 129 males), the wide majority remained overweight/obese in at least two time points during adulthood – 85% of females and 92% of males (see table in appendix 1).

The influence of parental BMI on trajectories of weight status

In females, both maternal and paternal BMI were significantly and positively associated with increased risk of childhood, early- and mid-adulthood onset of overweight/obesity, and having “other trajectories” versus never being overweight/obese in unadjusted regression models (Table 3). In males, higher maternal BMI was associated with increased risk of having childhood and early adulthood onset of overweight/obesity, and of belonging to “other trajectories” versus never being overweight/obese. Higher paternal BMI was associated with increased risk of having childhood and early adulthood onset of overweight/obesity versus never being overweight/obese in unadjusted regression models (Table 4). These associations remained significant for males and females even when considering both maternal and paternal BMI in the same model (unadjusted for other variables). Interaction between maternal and paternal BMI was not significant (results not shown) and, therefore,

was not included in the subsequent multivariable regression models. Because results of the multivariable regression models were similar when analyses where conducted with maternal and paternal BMI separately and in the same model, only the results for the models including both maternal and paternal BMI are presented below (Tables 3 and 4). Results for the multivariable regression models with maternal BMI only and paternal BMI separately can be seen in the tables presented in Appendices 2-5.

In females after adjustment for birth factors, puberty and SEP factors (Table 3), higher maternal BMI remained significantly associated with higher risk of childhood, early- and mid-adulthood onset of overweight/obesity, and of belonging to "other trajectories" compared with never being overweight/obese (all $p \leq 0.017$). Higher paternal BMI significantly increased the risk of childhood and early adulthood onset of overweight/obesity, and belonging to "other trajectories" compared with never being overweight/obese (all $p<0.001$). High SEP was protective of childhood and early adulthood onset of overweight/obesity ($p<0.04$), whereas early puberty increased the risk of childhood and early adulthood onset of overweight/obesity, and of belonging to "other trajectories" compared to never being overweight/obese (all $p<0.001$). Females with (versus without) evidence of puberty at 10 years of age showed increased risk of childhood and early adulthood onset of overweight/obesity, and belonging to "other trajectories versus never being overweight/obese (all $p<0.001$). No other significant associations were found.

In the fully adjusted model for males (Table 4), both higher maternal and paternal BMI significantly increased the risk of childhood and early adulthood onset of overweight/obesity compared to never being overweight/obese (all $p<0.001$). Early puberty also increased the risk of childhood (9.14; 1.76-47.56) and early adulthood onset of overweight/obesity (4.81; 1.14-20.32) compared with never being overweight/obese (both $p<0.04$). Additionally, having a high (versus normal-low) birth weight significantly increased the risk of early adulthood onset of overweight/obesity (2.67; 1.33-5.34), whereas having a higher gestational age significantly decreased the risk of early adulthood onset of overweight/obesity (0.88; 0.79-0.98) in relation to never being overweight/obese (Table 4). No association was found between trajectories of weight status and breastfeeding and maternal education in both sexes.

DISCUSSION

This study sought to (i) describe the trajectories of weight status from childhood to mid-adulthood, and (ii) investigate the influence of parental BMI on weight status trajectories of their offspring. In both sexes, approximately 6% had childhood onset of overweight/obesity, and the vast majority of those overweight/obese in childhood remained overweight/obese in at least two of the three adulthood time-points considered in this study. Maternal and paternal BMI showed additive effects on offspring weight status trajectories across 32 years of the life course, independently of several confounding factors identified in the literature (Monasta et al., 2010; Parsons et al., 1999). In both males and females, maternal and paternal BMI were positively associated with increased risk of childhood and early adulthood onset of overweight/obesity (versus never being overweight/obese). In females, maternal and paternal BMI were also positively associated with increased risk of "other trajectories". This means that, higher parental BMI also increases the risk of females being overweight/obesity at ≥1 time point between 10 and 42 years (apart from mid-adulthood onset of overweight/obesity). These results are consistent with the findings of the systematic review by Parsons et al (1999), who reported that offspring of obese parents were consistently reported to be at risk for increased fatness from childhood into adulthood (from longitudinal studies). In the fully adjusted models, these associations were slightly higher for maternal than paternal BMI in females (e.g. relative risk ratio for always overweight/obese: maternal BMI=1.27, paternal BMI=1.21), but similar for males (see Tables 3 and 4). However, when repeating the analyses using parental BMI z-scores (zBMI), maternal zBMI consistently showed stronger associations with childhood and early adulthood onset of overweight/obesity than paternal zBMI in both males (relative risk ratio: 1.71 and 2.14 (respectively) for maternal versus 1.55 and 1.85 for paternal zBMI) and females (1.85 and 2.50 for maternal versus 1.53 and 1.80 for paternal zBMI). This suggests that the same shift in the distribution of paternal BMI will have a higher influence in the offspring's weight status trajectories for maternal than paternal BMI.

The influence of parental BMI on offspring's overweight/obesity trajectories may operate through a variety of factors, including genetic predisposition, shared environment and the inheritance of unhealthy behaviours (Parsons et al, 1999). Although we have controlled for

maternal education and childhood SEP (known mediators) (Monasta et al, 2010), the extent to which the observed influence of parental BMI on offspring's overweight/obesity trajectories is due to genetic, environmental or behavioural factors (or a combination of the three) cannot be assessed in the current study and require investigation in future studies. Nevertheless, because (i) parental BMI was strongly associated with persistent overweight/obesity, (ii) the population rates of overweight are high and (iii) it is potentially easier to intervene in parents than in children to reduce their overweight (Golan, 2006; Monasta et al, 2010), focusing on the family as a whole and targeting overweight parents in obesity interventions may be a successful strategy to reduce both adulthood overweight and offspring's risk of lifetime overweight/obesity at the population level.

Early puberty was also a significant predictor of childhood and early adulthood onset of overweight/obesity for both sexes, as well as belonging to "other trajectories" in females. These results are consistent with previous findings (Parsons et al, 1999; Prentice & Viner, 2013), and improve upon existing literature which in general has lacked adjustment for SEP and parental fatness (Parsons et al, 1999). Because the available BCS70 data did not allow us to investigate whether obesity before 10 years may have led to early puberty and in turn to the subsequent onset of overweight/obesity, the possibility of reverse causality cannot be rejected and requires investigation in future studies. Nevertheless, the fact that early puberty was consistently associated with "early adulthood onset of overweight/obesity" (which by definition controls for overweight in childhood) indicates that this association between early puberty and subsequent adulthood overweight/obesity may be independent of preceding increased fatness, in line with the results of a recent systematic review by Prentice and Viner (2013).

Only three other factors emerged as significant predictors of weight status trajectories in the fully adjusted model, with mixed results between males and females. In males, higher gestational age significantly reduced the risk of having an early adulthood onset of overweight/obesity, in relation to never being overweight/obese. Although significant, the slight difference in the distribution of gestational age between sexes (39.6 weeks for males, 39.8 weeks for females; p=0.02) is unlikely to have influenced the lack of the same association for females. On the other hand, a birth weight ≥4 kg significantly increased the risk of males having early adulthood onset of overweight/obesity (versus never being

overweight/obese), an association previously reported in the literature (Monasta et al, 2010; Parsons et al, 1999). The higher prevalence of high birth weight in males than in females (11% versus 6%; $p<0.001$) and the very large sample size of males in the early adulthood onset of overweight/obesity trajectory may have influenced to some extent the ability to detect an association in males but not in females. Although the general consensus is that there is a consistent positive relationship between birth weight and later obesity (Monasta et al, 2010; Parsons et al, 1999) and plausible mechanisms have been proposed (Parsons et al, 1999), the nature and strength of this association is less clear in studies that account for potential confounders like in the present study. Both similar and reverse sex-differences in this association have been previously reported (Parsons et al, 1999), and more research is needed to clarify whether this association of birth weight with later obesity is independent of known confounders (e.g. gestational diabetes, parental fatness and SEP). The association of high childhood SEP with lower risk for childhood and early adulthood onset of overweight/obesity only in females cannot be explained by differences in prevalence of high childhood SEP between sexes ($p=0.94$). This sex-inequality has been observed in previous studies using adulthood obesity as the outcome even after adjustment for potential adulthood cofounders (Heraclides, Witte, & Brunner, 2008; Khlat et al., 2009; Pudrovska, Logan, & Richman, 2014). One potential explanation is that traditionally men tend to move upward in the social ladder, resulting in a closing of the social gap in health outcomes from childhood SEP; whereas women tend to remain in the social class of origin, magnifying the effect of parental social class on adulthood overweight (Heraclides, Witte, & Brunner, 2008). However, a considerable amount of literature has reported this effect of early life SEP in both sexes (Monasta et al, 2010; Parsons et al, 1999; Power, Manor, & Matthews, 2003). For example, Power, Manor and Matthews (2003) have reported a significant association of early life social class with obesity at age 30 years for both males and females from the British 1958 birth cohort study. Among other factors, differences in sample sizes, timing and type of the childhood SEP variable, the number of potential confounders adjusted for, and using weight status at only one time-point (versus lifelong trajectories) as the outcome could explain the contradictory results. More studies adopting a life course approach like the present study and controlling for the main known confounders/mediators (e.g. childhood weight status, early puberty and parental BMI) are

needed, to clarify whether a sex-inequality exists for the SEP effect on later obesity or it is resultant of the selected data and methodology of different studies.

Limitations and strengths

This study had some limitations that deserve consideration. Only 47.4% of 8799 cohort members with valid BMI at 42 years were included for analysis due to participants missing ≥1 independent variables, which could have impacted on the results (e.g., reducing statistical power to detect some associations). However, there was no significant difference in parental BMI and prevalence of high birth weight between those with and without complete data to compute trajectories of weight status; all other variables were significantly different between those with and without complete data to compute trajectories of weight status. Furthermore, differences between those included and excluded from the fully adjusted models were only significant in SEP (males and females), and breastfeeding in females; all other variables were similar between included and excluded participants. There was a slightly higher proportion of participants with high SEP in those included versus excluded from the fully adjusted models (36% versus 26% in males; 36% versus 29% in females). As sample sizes for each independent variable varied widely due to missing data and the observed differences were generally minor in size, it is unlikely that these differences would have had an impact on the results and their generalizability to the wider BCS70 cohort. As data were drawn from a national birth cohort, the findings are highly generalizable to the British population relatively contemporaneous in age to those in the BCS70 cohort. It is unclear how relevant the findings are to more recent age cohorts, as populations born in more recent years have been shown to have higher mean BMI and higher rates of overweight/obesity (Clarke, O'Malley, Johnston, & Schulenberg, 2009; Stamatakis, Primatesta, Chinn, Rona, & Falascheti, 2005). However, we would expect the effects of parental BMI on offspring overweight/obesity to be stronger in younger generations than that observed in the BCS70 cohort.

Weight and height at 10 years were measured but adulthood values were reported by participants, which may have led to biased BMI values and consequent classification of overweight/obesity. However, the recent review by Brisbois et al (2012) have reported

overall good agreement between the results of studies using measured and reported adult BMI. Taking this into account and the strong significant associations found in the present study, it is unlikely that the possible misreporting of weight and height would significantly impact on the results seen for the association of parental BMI with persistent overweight/obesity and early adulthood onset of overweight/obesity.

The fact that trajectories of weight status were manually computed and not drawn from the data using a statistical procedure is both a strength and a limitation of the current study. Using the "manual process" for classification of trajectories of weight status allows us know exactly how each trajectory is characterised, and not rely on regression model specifications to derive weight status trajectories. However, there may have been other potential trajectories that were missed by, for example, joining the trajectories with <5% prevalence in one only category (i.e. "other"). Using growth-mixture modelling (Muthén, 2001), with the five weight status trajectories identified in this study as the starting point, would (i) enable us to confirm whether the results from this study are confirmed when not using a manual procedure to derive the trajectories, (ii) include the 16-year sweep, as the model will be able to deal with the large amount of missing BMI data at this age, and (iii) address the issue of a reduced sample size in the fully adjusted regression models, by imputing the missing variables. We are currently undertaking this work. Other strengths of this study include investigating offspring weight status from childhood through to mid-adulthood in a life course perspective (rather than only one time-point in adulthood), addressing some of the main limitations of many previous studies looking the association of parental BMI with offspring obesity (Parsons et al, 1999; Monasta et al, 2010).

CONCLUSIONS

In conclusion, higher paternal and maternal BMI showed additive associations with offspring weight status trajectories across 32 years of the life course after controlling for potential confounding factors identified in the literature (Parsons et al, 1999; Monasta et al, 2010). In particular, maternal and paternal BMI are significantly associated with higher risk of childhood and early adulthood onset of overweight/obesity, and "other trajectories" (females only) versus never being overweight/obese. Early puberty was also significantly

associated with higher risk of childhood and early adulthood onset of overweight/obesity for both sexes. Other factors such as high SEP (females), gestational age and high birth weight (males) also showed significant associations with childhood and early adulthood onset of overweight/obesity, but these associations were not consistent between sexes. The identified weight status trajectories highlight the importance of primary prevention, as most individuals remained overweight/obese after onset at any age and both childhood and adulthood obesity have been consistently associated with increased risks of type two diabetes, coronary heart disease and all-cause mortality later in life (Park et al, 2012, 2013; Prospective Studies Collaboration, 2009). Early maturing overweight/obese children and those with overweight/obese parents are potential priority targets for interventions, due to their higher risk for persistent overweight/obesity through to mid-adulthood in both males and females. Prevention/intervention programmes should focus on the family as a whole since maternal and paternal BMI showed additive effects on offspring weight status trajectories from childhood to mid-adulthood.

ACKNOWLEDGEMENTS

The authors declare no competing interests. Permission for download and use of the BCS70 data was granted by the UK Data Service.

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Table 1. Description of study sample

	Missing data [N (%[1])]		Total (N = 4174)	Females (N = 2392)	Males (N = 1782)	*p* (sex difference)
BMI at 10 years (kg/m^2)	0 (0.0)	Mean (IQR)	16.7 (15.6-18.1)	16.7 (15.5-18.3)	16.7 (15.7-18.1)	0.605
BMI at 42 years (kg/m^2)	0 (0.0)	Mean (IQR)	26.6 (23.6-30.1)	25.2 (22.6-29.4)	27.9 (25.5-30.7)	<0.001
Gestational age (weeks)	864 (20.7)	Mean (IQR)	40 (39-41)	40 (39-41)	40 (39-41)	0.016
Birth weight (kg)	241 (5.8)	Mean (IQR)	3.3 (3.0-3.6)	3.3 (3.0-3.6)	3.4 (3.1-3.7)	<0.001
High birth weight		N (valid %[2])	3617 (92.0)	134 (5.9)	182 (10.9)	
Medium-low birth weight		N (valid %[2])	316 (8.0)	2130 (94.1)	1487 (89.1)	
Breastfeeding duration	569 (13.6)					0.466
<3 months		N (valid %[2])	3127 (86.7)	1796 (86.4)	1331 (87.2)	
≥3 months		N (valid %[2])	478 (13.3)	283 (13.6)	195 (12.8)	
Evidence of puberty at 10 years	18 (0.4)					<0.001
No		N (valid %[2])	3432 (82.6)	1735 (72.8)	1697 (95.7	
Yes		N (valid %[2])	724 (17.4)	647 (27.2)	77 (4.3)	
Maternal BMI (kg/m^2)	116 (2.8)	Mean (IQR)	22.6 (21.0-25.0)	22.5 (21.0-24.9)	22.7 (21.1-25.0)	0.352
Paternal BMI (kg/m^2)	274 (6.6)	Mean (IQR)	24.1 (22.5-26.0)	24.1 (22.5-26.0)	24.1 (22.5-26.3)	0.437
Maternal education at 10 years	251 (6.0)					0.271
Less than A-level		N (valid %[2])	3184 (81.2)	1825 (80.6)	1359 (82.0)	
A-level or above		N (valid %[2])	739 (18.8)	440 (19.4)	299 (18.0)	
Parental SEP at 10 years	114 (2.7)					0.453
Medium-low		N (valid %[2])	2711 (66.8)	1542 (66.3)	1169 (67.4)	
High		N (valid %[2])	1349 (33.2)	784 (33.7)	565 (32.6)	

Legend: BMI - Body mass index; IQR - Inter-quartile range; SEP - Socio-economic position.

[1] percent of total (4174) participants with trajectories of weight status.

[2] percent of total (4174) participants with valid data for each variable.

Table 2. Description of trajectories of weight status included for regression analyses [n (%)].

	Total (n=4174)	**Females** (n=2392)	**Males** (n=1782)
Never OW/OB	1230 (29.5%)	962 (40.2%)	268 (15.0%)*
Mid-adulthood OW/OB onset	334 (8.0%)	231 (9.6%)	103 (5.8%)*
Early adulthood OW/OB onset	1850 (44.3%)	733 (30.6%)	1117 (62.7%)*
Childhood OW/OB onset	258 (6.2%)	148 (6.2%)	110 (6.2%)
Other	502 (12.0%)	318 (13.3%)	184 (10.3%)

Legend: OW/OB - Overweight or obese; *p<0.001 for difference between sexes

Table 3 – Associations between parental BMI and trajectories of weight status in females.

Risk Factor	included n (%)	missing n (%)	Never OW/OB *reference*	OW/OB onset at mid-adulthood RRR (95% CI)	*p*	OW/OB onset at early adulthood RRR (95% CI)	*p*	Always OW/OB RRR (95% CI)	*p*	Other RRR (95% CI)	*p*
Unadjusted individual models											
Maternal BMI	2339 (97.8)	53 (2.2)		1.10 (1.05-1.15)	<0.001	1.21 (1.17-1.25)	<0.001	1.31 (1.25-1.37)	<0.001	1.17 (1.13-1.22)	<0.001
Paternal BMI	2250 (94.1)	142 (5.9)		1.09 (1.03-1.15)	0.002	1.17 (1.12-1.21)	<0.001	1.29 (1.21-1.36)	<0.001	1.15 (1.10-1.21)	<0.001
Mutually adjusted	2230 (93.2)	162 (6.8)									
Maternal BMI				1.10 (10.4-1.15)	<0.001	1.20 (1.16-1.24)	<0.001	1.29 (1.23-1.35)	<0.001	1.16 (1.12-1.21)	<0.001
Paternal BMI				1.08 (1.02-1.14)	0.006	1.14 (1.10-1.18)	<0.001	1.23 (1.16-1.31)	<0.001	1.13 (1.07-1.18)	<0.001
Adjusted for birth factors	1618 (67.6)	774 (32.4)									
Maternal BMI				1.08 (1.02-1.15)	0.007	1.19 (1.14-1.24)	<0.001	1.29 (1.22-1.36)	<0.001	1.18 (1.12-1.24)	<0.001
Paternal BMI				1.04 (0.98-1.12)	0.21	1.16 (1.11-1.21)	<0.001	1.23 (1.15-1.32)	<0.001	1.13 (1.07-1.19)	<0.001
Gestational age				0.93 (0.83-1.03)	0.16	1.00 (0.92-1.08)	0.95	0.95 (0.83-1.08)	0.43	0.95 (0.87-1.05)	0.31
Birth weight											
Birth weight ≤4 kg			reference	-		-		-		-	
Birth weight >4 kg				1,50 (0.79-2.85)	0.21	0.83 (0.50-1.40)	0.49	1.19 (0.54-2.66)	0.66	0.72 (0.36-1.46)	0.40
Breast feeding											
≥3 months			reference	-		-		-		-	

<3 months				1.05 (0.64-1.72)	0.84	1.21 (0.85-1.73)	0.29	1.37 (0.69-2.74)	0.37	1.00 (0.65-1.55)	1.00
Adjusted for birth factors and puberty	1614 (67.5)	778 (32.5)									
Maternal BMI				1.08 (1.02-1.14)	0.009	1.18 (1.14-1.23)	<0.001	1.28 (1.22-1.35)	<0.001	1.17 (1.12-1.23)	<0.001
Paternal BMI				1.04 (0.97-1.11)	0.23	1.15 (1.10-1.20)	<0.001	1.21 (1.13-1.30)	<0.001	1.11 (1.05-1.18)	<0.001
Gestational age				0.93 (0.83-1.03	0.15	0.99 (0.92-1.07)	0.88	0.93 (0.81-1.07)	0.32	0.95 (0.86-1.04)	0.25
Birth weight											
Birth weight ≤4 kg			reference	-		-		-		-	
Birth weight >4 kg				1.51 (0.79-2.86)	0.21	0.84 (0.50-1.41)	0.50	1.23 (0.55-2.76)	0.61	0.74 (0.36-1.50)	0.40
Breast feeding											
≥3 months			reference	-		-		-		-	
<3 months				1.06 (0.65-1.73)	0.83	1.26 (0.88-1.80)	0.21	1.55 (0.76-3.14)	0.23	1.08 (0.69-1.68)	0.75
Evidence of puberty at 10 years											
No			reference	-		-		-		-	
Yes				1.30 (0.85-1.99)	0.22	1.73 (1.30-2.31)	<0.001	3.52 (2.21-5.61)	<0.001	2.25 (1.59-3.20)	<0.001
Adjusted for birth factors, puberty and socioeconomic position	1567 (65.5)	825 (34.5)									
Maternal BMI				1.08 (1.01-1.14)	0.017	1.17 (1.13-1.22)	<0.001	1.27 (1.20-1.34)	<0.001	1.17 (1.11-1.23)	<0.001
Paternal BMI				1.03 (0.97-1.11)	0.34	1.15 (1.10-1.20)	<0.001	1.21 (1.13-1.31)	<0.001	1.11 (1.05-1.18)	<0.001
Gestational age				0.93 (0.84-1.03)	0.18	0.99 (0.92-1.07)	0.84	0.92 (0.80-1.06)	0.26	0.96 (0.87-1.06)	0.42

Birth weight									
Birth weight ≤4 kg	reference	-		-		-		-	
Birth weight >4 kg		1.62 (0.83-3.16)	0.16	0.98 (0.57-1.67)	0.93	1.66 (0.73-3.77)	0.23	0.86 (0.42-1.76)	0.67
Breast feeding									
≥3 months	reference	-		-		-		-	
<3 months		1.02 (0.61-1.71)	0.93	1.18 (0.81-1.72)	0.38	1.33 (0.64-2.75)	0.44	0.99 (0.62-1.57)	0.96
Evidence of puberty at 10 years									
No	reference	-		-		-		-	
Yes		1.31 (0.85-2.01)	0.23	1.75 (1.31-2.34)	<0.001	3.68 (2.29-5.92)	<0.001	2.30 (1.62-3.28)	<0.001
Socioeconomic position									
Medium-low	reference	-		-		-		-	
High		0.85 (0.57-1.24)	0.40	0.75 (0.57-0.98)	0.036	0.50 (0.28-0.88)	0.016	0.90 (0.64-1.28)	0.57
Maternal Education									
Less than A-level	reference	-		-		-		-	
A-level or above		0.94 (0.59-1.53)	0.85	0.96 (0.68-1.34)	0.8	0.82 (0.40-1.67)	0.59	0.71 (0.45-1.13)	0.15

Legend: BMI – Body mass index; CI - Confidence interval; OW/OB - Overweight or obese; RRR - Relative risk ratio.

Table 4 - Associations between parental BMI and trajectories of weight status in males.

Risk Factor	included n (%)	missing n (%)	Never OW/OB *reference*	OW/OB onset at mid-adulthood RRR (95% CI)	*p*	OW/OB onset at early adulthood RRR (95% CI)	*p*	Always OW/OB RRR (95% CI)	*p*	Other RRR (95% CI)	*p*
Unadjusted individual models											
Maternal BMI				1.05 (0.97-1.13)	0.26	1.16 (1.10-1.22)	<0.001	1.23 (1.16-1.31)	<0.001	1.09 (1.03-1.16)	0.006
Paternal BMI				1.05 (0.96-1.15)	0.32	1.17 (1.10-1.23)	<0.001	1.27 (1.17-1.37)	<0.001	1.07 (0.99-1.15)	0.08
	1628	154									
Mutually adjusted	(91.4)	(8.6)									
Maternal BMI				1.02 (0.94-1.11)	0.56	1.13 (1.07-1.18)	<0.001	1.19 (1.12-1.27)	<0.001	1.07 (1.01-1.15)	0.029
Paternal BMI				1.04 (0.94-1.14)	0.44	1.14 (1.08-1.21)	<0.001	1.23 (1.14-1.34)	<0.001	1.06 (0.98-1.15)	0.14
	1178	604									
Birth factors	(66.1)	(33.9)									
Maternal BMI				1.02 (0.93-1.13)	0.64	1.15 (1.08-1.22)	<0.001	1.21 (1.11-1.31)	<0.001	1.10 (1.02-1.18)	0.019
Paternal BMI				1.03 (0.83-1.17)	0.60	1.16 (1.09-1.25)	<0.001	1.25 (1.13-1.39)	<0.001	1.05 (0.96-1.16)	0.26
Gestational age				0.99 (0.92-1.15)	0.88	0.87 (0.79-0.97)	0.011	0.88 (0.74-1.04)	0.14	1.12 (0.97-1.30)	0.13
Birth weight											
Birth weight ≤4 kg			reference								
Birth weight >4 kg				2.01 (0.75-5.38)	0.17	2.76 (1.39-5.51)	0.004	0.80 (0.21-3.07)	0.74	1.72 (0.72-4.10)	0.22
Breast feeding											
≥3 months			reference								

<3 months				0.65 (0.31-1.39)	0.27	0.88 (0.52-1.47)	0.62	0.96 (0.38-2.40)	0.92	0.73 (0.37-1.43)	0.36
Adjusted for birth factors and puberty	1173 (65.8)	609 (34.2)									
Maternal BMI				1.03 (0.93-1.13)	0.61	1.15 1.08-1.22)	<0.001	1.22 (1.12-1.32)	<0.001	1.09 (1.01-1.18)	0.034
Paternal BMI				1.03 (0.92-1.15)	0.66	1.16 (1.08-1.25)	<0.001	1.25 (1.13-1.38)	<0.001	1.05 (0.96-1.15)	0.30
Gestational age				0.99 (0.83-1.17)	0.87	0.87 (0.79-0.97	0.012	0.88 (0.74-1.04)	0.14	1.13 (0.97-1.31)	0.11
Birth weight											
Birth weight ≤4 kg			reference	-		-		-		-	
Birth weight >4 kg				1.99 (0.74-5.35)	0.17	2.66 (1.33-5.30)	0.006	0.77 (0.20-2.95)	0.70	1.69 (0.71-4.04)	0.24
Breast feeding											
≥3 months			reference	-		-		-		-	
<3 months				0.65 (0.31-1.39)	0.27	0.87 (0.52-1.46)	0.61	0.92 (0.36-2.31)	0.85	0.78 (0.39-1.53)	0.46
Evidence of puberty at 10 years											
No			reference	-		-		-		-	
Yes				2.33 (0.32-16.88)	0.40	4.81 (1.14-20.32)	0.033	9.14 (1.76-47.56)	0.009	2.87 (0.52-16.02)	0.23
Adjusted for birth factors, puberty and socioeconomic position	1140 (64.0)	642 (36.0)									
Maternal BMI				1.01 (0.92-1.12)	0.8	1.15 (1.08-1.22)	<0.001	1.22 (1.12-1.33)	<0.001	1.08 (0.99-1.17)	0.07
Paternal BMI				1.02 (0.91-1.15)	0.71	1.16 (1.08-1.24)	<0.001	1.22 (1.10-1.36)	<0.001	1.04 (0.94-1.14)	0.47
Gestational age				0.98 (0.82-1.17)	0.84	0.88 (0.79-0.98)	0.016	0.89 (0.75-1.06)	0.20	1.16 (0.99-1.35)	0.06

Birth weight									
Birth weight ≤4 kg	reference	-		-		-		-	
Birth weight >4 kg		2.12 (0.79-5.71)	0.14	2.67 (1.33-5.34)	0.006	0.78 (0.20-3.01)	0.72	1.75 (0.73-4.19)	0.21
Breast feeding									
≥3 months	reference	-		-		-		-	
<3 months		0.66 (0.29-1.49)	0.32	0.80 (0.46-1.38)	0.42	0.82 (0.32-2.13)	0.69	0.66 (0.32-1.33)	0.24
Evidence of puberty at 10 years									
No	reference	-		-		-		-	
Yes		2.56 (0.35-18.64)	0.36	4.71 (1.11-19.95)	0.035	9.21 (1.77-48.02)	0.008	2.92 (0.52-16.36)	0.22
Socioeconomic position									
Medium-low	reference								
High		1.07 (0.58-1.95)	0.83	0.88 (0.60-1.28)	0.51	0.97 (0.50-1.87)	0.93	0.82 (0.49-1.38)	0.46
Maternal Education									
Less than A-level	reference	-		-		-		-	
A-level or above		0.63 (0.29-1.35)	0.23	0.78 (0.50-1.23)	0.29	0.86 (0.00-6.31)	0.72	0.75 (0.40-1.43)	0.39

Legend: BMI – Body mass index; CI - Confidence interval; OW/OB - Overweight or obese; RRR - Relative risk ratio.