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Disentangling the respective roles of the early environment and parental BMI on BMI change across childhood: A counterfactual analysis using the Millennium Cohort Study

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ABSTRACT

This study has two objectives. First, to analyse the respective roles of parental BMI and the wider environment on children's BMI across childhood, using a counterfactual analysis. Second, to determine if the correlations between parents and offspring BMI are partly environmental.

We used data on 4437 girls and 4337 boys born in 2000–2001 in the UK and included in the Millennium Cohort Study. Children's BMI was measured at ages 3 years, 5 years, 7 years, and 11 years. We described the environment using social class and behaviours within the family. At the age of 3, there was no link between the environment and children's BMI. In contrast, there was a clear link between the environment and BMI slopes between 3 and 11 years of age. At the age of 11, we calculated that if all children had the most favourable environment, mean BMI would be reduced by 0.91 kg/m^2 (95% CI: 0.57–1.26) for boys and by 1.65 kg/m^2 (95% CI: 1.28–2.02) for girls. Associations between parents' and offspring BMI remained unchanged after adjustment for environmental variables. Conversely, the link between the environment and children's BMI is partly reduced after adjustment for parental BMI. This confirms that parental BMI is partly a broad proxy of the environment.

We highlighted that if every child's environment was at its most favourable, the mean BMI would be significantly reduced. Thus, the recent rise is likely to be reversible.

1. Introduction

Body mass index (BMI) is determined by multiple factors involving genetic predispositions, energy related behaviours, social processes and factors within the built environment. A number of studies have observed the transmission of BMI between parents and children (Fleten et al., 2012; Lake et al., 1997; Ajslev et al., 2014). Since parents and children share both genes and environments, a number of interacting factors may be involved in this intergenerational transmission. Studies of adoptees supported the hypothesis of a genetic component of BMI, where the adult BMI of adoptees was strongly correlated with both their natural mothers' and fathers' BMI (Stunkard et al., 1986; Sørensen et al., 1998). There is also evidence of a link between some genetic variants and BMI in children and in adults (Speliotes et al., 2010; Frayling et al., 2007; Elks et al., 2014), in particular in genes involved in appetite control (Llewellyn et al., 2014). Nevertheless, genetic change occurs on an evolutionary time frame and therefore cannot explain the recent rise of children's mean BMI in western countries (Eisenmann, 2006). According to results from National Survey of Health and Development (NSHD) and Millennium Cohort Study (MCS), two British birth cohorts including children respectively born in 1946 and 2001, the BMI median of 11-year-old has increased by 1.2 kg/m^2 for boys and 1.7 kg/m^2 for girls between 1957 and 2012 (Johnson et al., 2015). This strongly suggests that environmental factors may be important determinants of childhood BMI (Law et al., 2007). Mechanisms potentially involved may begin during pregnancy, with a number of studies suggesting a direct role of the foetal environment on later BMI (Dabelea et al., 2000; Ravelli et al., 1976; Oken and Gillman, 2003). Numerous researchers also underline correlations between the postnatal environment and subsequent BMI, mainly through dietary factors (Eisenmann, 2006; Slyper, 2004), physical activity and sedentary

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activities (Tremblay et al., 2011) or sleep (Agras et al., 2004). Finally, evidence suggests that childhood BMI is driven by foetal and postnatal environments in interaction with genetic predispositions (Frayling et al., 2007; Llewellyn et al., 2014; Eisenmann, 2006; Law et al., 2007).

The typical environment in western countries has been termed "obesogenic" (Egger and Swinburn, 1997), a term describing the physical, economic, political and sociocultural factors that underlie many unhealthy life-styles (Swinburn et al., 1999; Lake and Townshend, 2006). For example, urban planning tends to favour motor-driven modes of transportation over pedestrian or bicycle modes (Sallis et al., 2012). Healthier diets tend to be more expensive (Drewnowski and Darmon, 2005), out-of-home food portion sizes are increasing (Young and Nestle, 2002) and advertised foods are predominantly high in sugars and fats (Story and French, 2004). This links in with the concept of socio-ecological or bio-ecological model described by many authors working in the field of child development (Bronfenbrenner, 1979), public health (Whitehead and Dahlgren, 1991) and epidemiology (Susser and Susser, 1996), in which environment is conceptualized as a series of concentric circles, moving from individual level characteristics in the middle towards increasingly structural circles including community and societal levels. This obesogenic environment is more likely to impact BMI according to the structuring of social position because people from more deprived social classes are more price-sensitive (Drewnowski and Darmon, 2005), less responsive to health promotion targeting individuals (Peretti-Watel et al., 2013) and live in neighbourhoods with limited access to recreational facilities (Gordon-Larsen et al., 2006).

Studying the specific role of the environment on BMI during childhood is necessary because of the relative unsuccessfulness of lifestyle interventions in adulthood to reduce adult BMI (Catalano, 2003). Yet, BMIs in childhood and in adulthood are closely correlated (Whitaker et al., 1997). And, BMI in adults is related with mortality and disabilities in most of the high-income countries (GBD 2013 Risk Factors Collaborators et al., 2015). Current evidence is mainly focused on the specific role of parents' social class or neighbourhood deprivation, and suggests an age-related and contextual effect. Studies on school-aged children and adolescents show a link between offspring's BMI and parents' social class (Kinra et al., 2000; Wang, 2001), early socioeconomic adversity (Bae et al., 2014) or deprived neighbourhood environment (Oliver and Hayes, 2008; Burdette and Needham, 2012). However, no such associations were found when children were 3 years of age (Brunt et al., 2008; De Spiegelaere et al., 1998; Semmler et al., 2009). Moreover, this association between deprived environment and children's BMI may be a recent phenomenon (Law et al., 2007) as it was not observed in some of the older British birth cohorts (Power et al., 2003; Duran-Tauleria et al., 1995; Rona and Chinn, 1982). The literature seems to suggest that the link between the environment and BMI may be different according to the different stages of child development, appearing around school-entry age and then increasing during childhood (Semmler et al., 2009).

Both genetic predispositions and environmental factors, in the broadest sense, are likely to affect BMI trajectories over childhood, through prenatal and postnatal processes. We will hypothesise that their respective roles may vary across childhood. This article has two objectives. First, to analyse the respective roles of parental BMI and the wider environment on children's BMI across childhood (see Fig. 1). Second, to determine if the correlations between parent and offspring BMI are partly environmental. Therefore, we will attempt to understand the hypothesized causal relationships between inherited environmental and genetic variables that may be associated with the current observed increases in childhood BMI.

2. Materials and methods

2.1. Participants

The data used here are from the Millennium Cohort Study (MCS), a British birth cohort. At baseline, 18,818 children born in the UK between 2000 and 2002 were included (Connelly and Platt, 2014). Five waves of data collection were conducted at 9 months (baseline) and then at 3, 5, 7 and 11 years of age (wave 5). Our sample selection was based on individuals with data available for children's BMI and mother's BMI before pregnancy, in order to compare results between various waves. We kept one child per family to satisfy the hypothesis of independence between observations. The flowchart is shown in Fig. 2. The final sample size is 8774: 4437 girls and 4337 boys. Mean ages at each wave are respectively 3.1 (0.2), 5.2 (0.2), 7.2 (0.2) and 11.2 (0.3) years of age.

2.2. Measures

2.2.1. Exposure variables

We used children's BMI measured at 3, 5, 7 and 11 years of age. Three variables were created, measuring the BMI slope per year

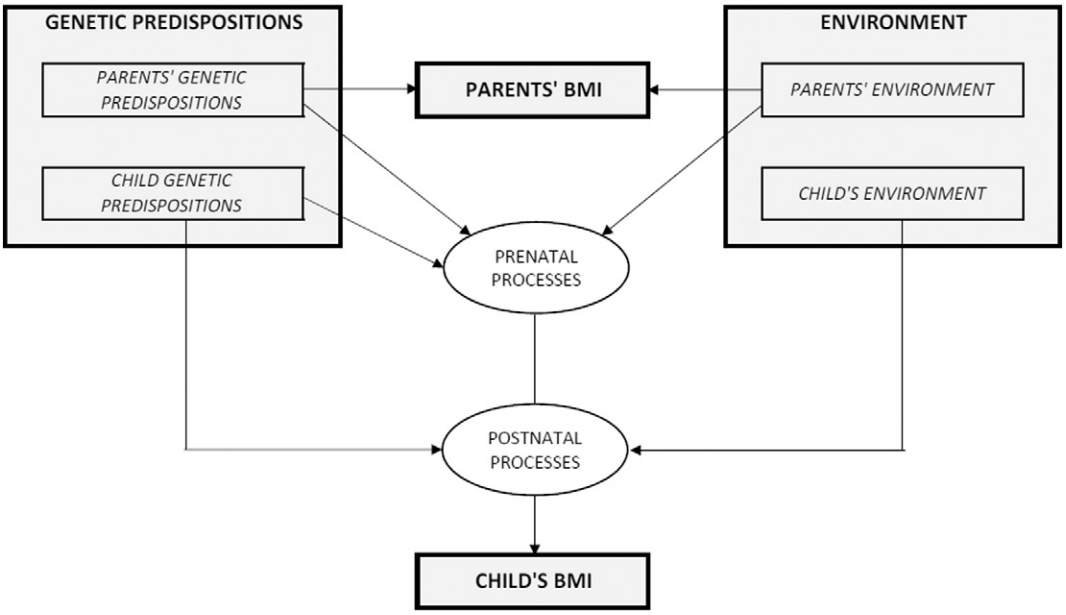

Fig. 1. Conceptual framework.

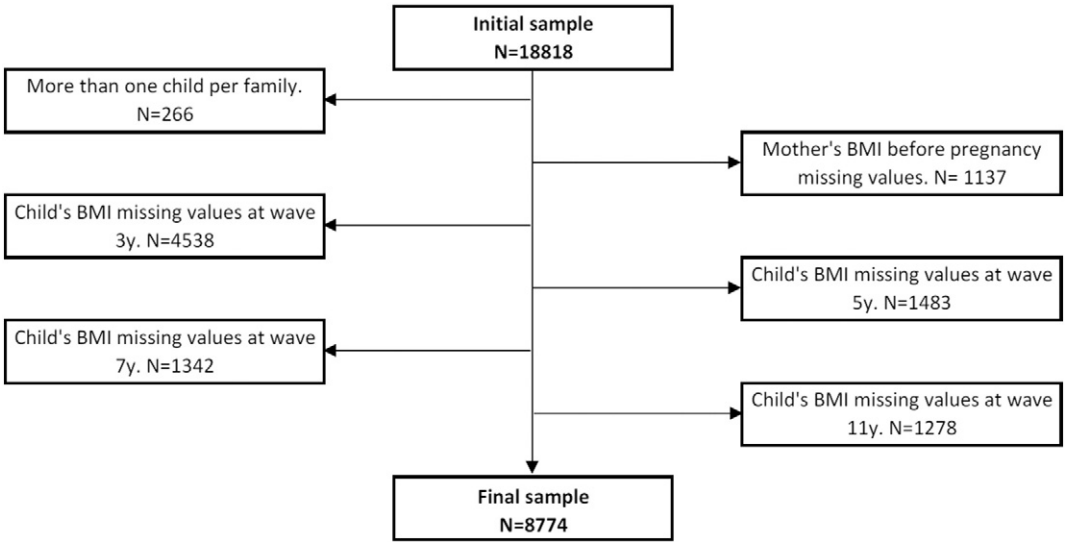

Fig. 2. Flow chart.

between the ages of 3 and 5, 5 and 7, and 7 and 11. Using BMI rather than BMI percentiles is recommended in the case of longitudinal analyses (Cole et al., 2005; Berkey and Colditz, 2007). An analysis of BMI slope per year allowed us to compare coefficients when the timescale between two waves differed.

2.2.2. Parent's BMI

We used continuous self-reported natural mother's BMI before pregnancy and continuous self-reported natural father's BMI. Both were collected at baseline.

2.2.3. Environment

We described environment using parents' social class, maternal and paternal grandparents' social classes and a number of behaviours identified in the literature as being correlated with children's BMI: smoking status during pregnancy (at baseline; no/yes, during the first three months/yes, after the first three months less than 5 cigarettes/yes, after the first three months, 5 cigarettes or more) (Pryor et al., 2011; Iliadou et al., 2010), bedtime (at each wave; Q1 (sooner quartile)/Q2/Q3/Q4/not regular) (Golley et al., 2013; Miller et al., 2015), skipping breakfast (at each wave; everyday/six days a week or less) (Harding et al., 2008), time spent watching TV on weekday (at each wave; 1 h or less/1 to 3 h/more than 3 h) (Swinburn and Shelly, 2008). The social class of the household was defined by the highest social status of the two parents at baseline. We used the National Statistics Socio-economic Classification five-class version, adding a "never worked or absent" category. At age 3 years, we divided bedtime on term-time weekdays into two groups (regular/not regular) and having breakfast was replaced by having regular meals (regular/not regular).

2.2.4. Control variables

We added ethnic group (6 category census classification) (Zilanawala et al., 2015) and the children's age in months at each measurement (BMI model) or at the more recent measurement (BMI slope model) to the models. The objective of this study was not to separate the prenatal effects from the postnatal effects of environment and parental BMI on child's BMI so we did not control for birth weight and gestational age.

2.2.5. Ethics and data

Following ethical approval for the study from an NHS Research Ethnics Committee (MREC), informed consent is obtained from parents, as well as from the children themselves as they grow up (Connelly and Platt, 2014).

3. Data and statistical analyses

All analyses were performed using STATA®V11. A p-value inferior to 0.05 indicates statistically significant findings. Descriptive and bivariate statistics (Tables 1 and 2) were carried out on non-imputed data. To control for possible bias due to missing data, we imputed data for covariates using the multiple Imputation by Chained Equations (ICE) program. The imputation model was performed on the final sample and included all the covariates but excluded exposure variables (children's BMI). For natural father's BMI, 20.8% were unknown at wave 1. We used multiple imputation when father's BMI was known in another wave. If it was not known (14.3%), we attributed the father's BMI mean. A binary variable father's BMI known or imputed/unknown was added to the models.

Our analysis of children's BMI took two forms. First, we analysed children's BMI at ages 3 years and 11 years. Second, we analysed children's BMI slope per year between data collection points: between 3 years and 5 years; 5 years and 7 years; and 7 years and 11 years. All linear regressions were stratified by sex. Each of the three BMI slope per year models was analysed as a single regression model with interactions between wave and explanatory factors. These systematic interactions (see Eq. (1)) avoided the problem of the clustering of observations: the likelihood-ratio test against the hierarchical model was not significant. This modelling technique allowed us to both calculate and compare coefficients for each wave and each explanatory factor.

$$\text{BMI slope}_{i,j} = 1_{\{\text{wave}==i\}} * (\beta_{0,i} + \beta_{1,i} X_{i,j}) + \varepsilon_{i,j} \quad (1)$$

where i is the wave 3, 4 or 5, j the child and X the explanatory factors.

We decided to perform a counterfactual analysis (Rubin, 1974) which would allow us to summarise all the information obtained by

Table 1
Description of parents and children's BMI in kg/m^2.

	Girls (N = 4437)	Boys (N = 4337)
BMI at age 3	16.63	16.92
BMI slope per year — 3 to 5 years	−0.18	−0.27
BMI slope per year — 5 to 7 years	0.17	0.07
BMI slope per year — 7 to 11 years	0.71	0.62
BMI at age 11	19.35	18.92
Mother's BMI	23.82	23.78
Father's BMI	26.05	25.98

Table 2
Descriptive statistics on the subsample for men and women and BMI mean in kg/m^2.

	Girls (N = 4437)			Boys (N = 4337)		
		BMI mean			BMI mean	
	N (%)	At age 3	At age 11	N (%)	At age 3	At age 11
Parents' social class[1]		NS	***		NS	***
I	1992 (44.9%)	16.64	18.93	2035 (46.9%)	16.94	18.61
II	535 (12.1%)	16.55	19.34	507 (11.7%)	16.82	18.82
III	260 (5.9%)	16.58	19.50	232 (5.3%)	16.89	18.95
IV	385 (8.7%)	16.63	19.83	347 (8.0%)	16.94	19.54
V	768 (17.3%)	16.65	20.00	718 (16.6%)	16.89	19.23
Never worked — absent	85 (1.9%)	16.62	20.37	93 (2.1%)	17.04	19.39
Missing	412 (9.3%)			405 (9.3%)		
Paternal grand parents' social class[1]		NS	**		*	NS
I	923 (20.8%)	16.58	18.97	937 (21.6%)	16.99	18.70
II	469 (10.6%)	16.56	18.92	470 (10.8%)	16.84	18.72
III	429 (9.7%)	16.78	19.13	440 (10.1%)	16.88	18.82
IV	288 (6.5%)	16.71	19.48	287 (6.6%)	16.98	18.69
V	1013 (22.8%)	16.60	19.48	949 (21.9%)	16.99	19.11
Never worked — absent	374 (8.4%)	16.64	19.70	353 (8.1%)	16.67	18.79
Missing	941 (21.2%)			901 (20.8%)		
Maternal grand parents' social class[1]		NS	***		**	**
I	1183 (26.7%)	16.66	18.90	1139 (26.3%)	17.03	18.69
II	581 (13.1%)	16.57	19.18	613 (14.1%)	16.86	18.78
III	592 (13.3%)	16.54	19.19	564 (13.0%)	16.76	18.96
IV	356 (8.0%)	16.57	19.56	358 (8.3%)	16.83	18.90
V	1203 (27.1%)	16.70	19.77	1108 (25.5%)	16.98	19.25
Never worked — absent	451 (10.2%)	16.59	19.82	485 (11.2%)	16.80	18.88
Missing	71 (1.6%)			70 (1.6%)		
Smoking status during pregnancy		NS	***		**	***
No smoker	2436 (54.9%)	16.60	19.04	2365 (54.5%)	16.86	18.74
Ex-smoker	654 (14.7%)	16.60	19.20	612 (14.1%)	16.87	18.55
Smoker — only before month 3	877 (19.8%)	16.70	19.74	843 (19.4%)	16.99	19.24
Smoker — after month 3	243 (5.5%)	16.72	20.09	256 (5.9%)	16.98	19.27
Smoker — after month 3, ≥5 cig.	219 (4.9%)	16.73	20.81	256 (5.9%)	17.21	19.96
Missing	8 (0.2%)			5 (0.1%)		
Regular meals — at age 3		NS			***	
No	360 (8.1%)	16.50		341 (7.9%)	16.61	
Yes	4063 (91.6%)	16.64		3980 (91.8%)	16.94	
Missing	14 (0.3%)			16 (0.4%)		
Skipped breakfast — at age 11			***			**
One day a week or more	640 (14.4%)		20.04	418 (9.6%)		19.36
Never	3776 (85.1%)		19.23	3893 (89.8%)		18.86
Missing	21 (0.5%)			26 (0.6%)		
TV/videos per weekday — at age 3		NS			*	
1 h or less	1105 (24.9%)	16.59		977 (22.5%)	16.82	
1 to 3 h	2604 (58.7%)	16.64		2619 (60.4%)	16.96	
More than 3 h	714 (16.1%)	16.64		725 (16.7%)	16.87	
Missing	14 (0.3%)			16 (0.4%)		
TV/videos per weekday — at age 11			***			***
1 h or less	699 (15.8%)		18.62	732 (16.9%)		18.58
1 to 3 h	2895 (65.2%)		19.41	2818 (65.0%)		18.89
More than 3 h	663 (14.9%)		19.83	576 (13.3%)		19.52
Missing	180 (4.1%)			211 (4.9%)		
Regular bedtime — at age 3		NS			NS	
No	315 (7.1%)	16.60		301 (6.9%)	16.98	
Yes	4108 (92.6%)	16.63		4020 (92.7%)	16.91	
Missing	14 (0.3%)			16 (0.4%)		
Bedtime — at age 11			***			***
Q1 (sooner)	639 (14.4%)		18.89	564 (13.0%)		18.52
Q2	1007 (22.7%)		19.12	931 (21.5%)		18.51
Q3	1706 (38.4%)		19.36	1617 (37.3%)		18.84
Q4	901 (20.3%)		19.83	1052 (24.3%)		19.50
Not regular	153 (3.4%)		19.91	131 (3.0%)		19.13
Missing	31 (0.7%)			42 (1.0%)		

MANOVA results: NS no significant link between BMI and the environment variable, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.
[1] NS-SEC: I — managerial, administrative and professional occupations, II — intermediate occupations, III — small employers and own account workers, IV — lower supervisory and technical occupations, V — semi-routine and routine occupations.

multiple regressions from the environmental variables. Here, we evaluated the overall impact of the environment, without looking for a causal link between each variable and children's BMI. Counterfactual analyses are rooted in theories of causation. Here we used a counterfactual analysis taking the assumption that genetic predispositions are equally distributed across the environment (Holzapfel et al., 2010, 2011; Lawlor et al., 2008). Based on this assumption, the differences associated with environmental variables we observed can be interpreted as differences due to the environment.

- We defined the "effect" of the environment as the difference between the children's observed versus estimated mean BMI, in the theoretical

Table 3
Pearson's correlation table of parents' and children's BMI for boys.

Boys	BMI — age 3 years	BMI — age 5 years	BMI — age 7 years	BMI — age 11 years	Maternal BMI	Paternal BMI
BMI — age 3 years	1					
BMI — age 5 years	0.57 [0.55–0.59]	1				
BMI — age 7 years	0.56 [0.54–0.58]	0.73 [0.71–0.74]	1			
BMI — age 11 years	0.43 [0.41–0.46]	0.59 [0.57–0.60]	0.81 [0.80–0.82]	1		
Maternal BMI	0.15 [0.12–0.18]	0.19 [0.17–0.22]	0.27 [0.24–0.30]	0.32 [0.29–0.34]	1	
Paternal BMI	0.11 [0.08–0.14]	0.19 [0.16–0.22]	0.21 [0.18–0.24]	0.23 [0.21–0.26]	0.14 [0.11–0.17]	1

situation where every child's environment would be its most "favourable". We defined the most favourable environment as: the most advantaged parental and grandparental social classes, a mother who had not smoked during pregnancy, the earliest quintile of bedtime on term-time weekdays, having breakfast every day, no more than 1 h per day spent watching TV/videos.

- We similarly defined the "effect" of parental BMI as the difference between the child's observed versus estimated mean BMI if no parent had a BMI higher than 25 kg/m^2, using the estimated beta of maternal and paternal BMI, and the mean BMI and the rate of overweight mothers and fathers. As we mainly interpreted the robustness of the links between the environment, parental BMI and child's BMI, the choice of the 25 kg/m^2 cut-off does not have an influence.

We performed these counterfactual analyses via different models. Model 1a estimated the overall effect of the environment on the children's BMI or BMI slope, adjusted for control variables (ethnicity and age). Model 1b estimated the effect of parental BMI on children's BMI or BMI slope, adjusted for control variables. Model 2 included the environment and parental BMI as explanatory variables adjusting for control variables. We calculated the difference between the effect of the environment (or parental BMI) calculated in Model 1a (or Model 1b) versus in Model 2.

Bootstrapping with 1000 samplings was run to obtain confidence intervals. We implemented sensitivity analyses for models at ages 3 years and 11 years, using excluded observations for which we knew mother's BMI and children's BMI at age 3 years (N = 4103) or mother's BMI and children's BMI at age 11 years (N = 2941) (Supplementary material).

4. Results

Bivariate analyses showed similar links for girls and boys between the environment, parental BMI and children's BMI at ages 3 and 11 years (Tables 2–4). At 3 years of age, there was no clear link between the environment and children's BMI. For girls, no variable characterizing the environment was associated with children's BMI at 3 years of age. For boys, smoking during pregnancy was associated with higher BMI at age 3 years however low grand parents' social class and no regular meals were linked with lower BMI at age 3 years. The link between parents' social class and children's BMI highlighted the emergence of a social gradient between 3 and 11 years of age. At age 11 years, all the variables characterizing the environment we used were correlated with children's BMI, and suggested a link between a favourable environment and lower BMI.

We found similar correlations between parental BMI and children's BMI for girls and boys (Tables 3 and 4). For both, the correlations between maternal BMI and children's BMI were stronger than those for paternal BMI and children's BMI ($p < 0.001$). The correlations between parental BMI and children's BMI increased between 3 and 11 years of age ($p < 0.001$). Moreover, strong correlations were found between children's BMI at each age (Tables 3 and 4).

The counterfactual analyses (Tables 5 and 6) confirmed that the effect of the environment appeared clearly after 3 years of age whereas there was no link between the environment and BMI at age 3 years. The link between the environment and BMI slopes was greater between the ages of 7 and 11 compared to both the 3 to 5 year age group and the 5 to 7 year age group respectively, for both girls and boys ($p < 0.001$). Between 7 and 11 years of age, the mean BMI slope per year would be 0.16 kg/m^2 (95% CI: 0.11–0.22) lower for boys and 0.22 kg/m^2 (95% CI: 0.16–0.28) lower for girls if every child had been exposed to the most favourable environment. As a result, at age 11 years, if every child had been exposed to the most favourable environment, the mean BMI would be 0.91 kg/m^2 (95% CI: 0.57–1.25) lower for boys and 1.65 kg/m^2 (95% CI: 1.28–2.02) lower for girls. Introducing parental BMI in Model 1a partly reduced the estimated link between the variables we used to define the environment and children's BMI (Model 1a–Model 2).

The counterfactual analyses showed that in the theoretical situation where no parents had BMI higher than 25 kg/m^2, the mean BMI would be 0.60 kg/m^2 (95% CI: 0.53–0.67) lower for boys and 0.64 kg/m^2 (95% CI: 0.57–0.71) lower for girls. The link between parental BMI and children's BMI (Model 1b) remained unchanged after adjustment for environment variables (Model 1b–Model 2) for both girls and boys and at each age.

5. Discussion

The environment was not related to children's BMI at age 3 years, but became increasingly important in determining childhood BMI over time. The environment was strongly related to children's BMI at age 11. Parental BMI was related to children's BMI from the age of three and remained so throughout childhood. These findings suggest a robust association between parental and child BMI, and an increasing association between environmental factors and child BMI, over time.

Living in a "favourable" environment was significantly correlated with lower BMI for girls and boys after 3 years of age. The influence of the familial, behavioural and social environment affected children's BMI between 3 and 5 years of age and increased again between 7 and

Table 4
Pearson's correlation table of parents' and children's BMI for girls.

Girls	BMI — age 3 years	BMI — age 5 years	BMI — age 7 years	BMI — age 11 years	Maternal BMI	Paternal BMI
BMI — age 3 years	1					
BMI — age 5 years	0.64 [0.62–0.66]	1				
BMI — age 7 years	0.59 [0.57–0.61]	0.79 [0.77–0.80]	1			
BMI — age 11 years	0.45 [0.43–0.48]	0.65 [0.64–0.67]	0.82 [0.81–0.83]	1		
Maternal BMI	0.16 [0.13–0.19]	0.24 [0.21–0.27]	0.26 [0.23–0.29]	0.31 [0.28–0.33]	1	
Paternal BMI	0.11 [0.08–0.14]	0.18 [0.15–0.21]	0.20 [0.17–0.23]	0.26 [0.23–0.28]	0.18 [0.15–0.20]	1

Table 5
Counterfactual analyses for boys of BMI at age 3 years, BMI at age 11 years, BMI gain per year between 3 and 5 years old, 5 and 7 years old, and 7 and 11 years old.

	Environment		Parents' overweight	
Boys	Model 1a[1]	Model 1a–Model 2[3]	Model 1b[2]	Model 1b–Model 2
BMI (in kg/m^2)				
At age 3 years	−0.03 [−0.15–0.10]	NC	0.12 [0.09–0.15]***	−0.00 [−0.01–0.00]
At age 11 years	0.91 [0.57–1.26]***	0.27 [0.13–0.42]***	0.60 [0.53–0.67]***	−0.01 [−0.02–0.01]
BMI slope (in kg/m^2/year)				
Between 3–5 years of age	0.08 [0.01–0.15]*	0.02 [0.01–0.03]**	0.05 [0.03–0.07]***	−0.00 [−0.00–0.00]
Between 5–7 years of age	0.07 [0.01–0.13]*	0.03 [0.01–0.05]***	0.05 [0.03–0.07]***	−0.00 [−0.00–0.00]
Between 7–11 years of age	0.16 [0.11–0.22]***	0.03 [0.01–0.05]***	0.07 [0.06–0.08]***	−0.00 [−0.00–0.00]

NC: not calculated because Model 1a did not show significant effect.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.
[1] Model 1a: difference between observed BMI or BMI slope and predicted BMI or BMI slope in a favourable environment (controlled for age and ethnicity) (see Section 2).
[2] Model 1b: difference between observed BMI or BMI slope and predicted BMI or BMI slope for children if no parent had BMI > 25 kg/m^2 (controlled for age and ethnicity).
[3] Model 2: difference between observed BMI or BMI slope and predicted BMI or BMI slope for children in a favourable environment (controlled for age, ethnicity and parental BMI)/if no parent had BMI > 25 kg/m^2 (controlled for age, ethnicity and environment).

11 years of age. At age 3 years, no difference according to the environment was found. The environment may have a weak effect on children's BMI before 3 years old, which is consistent with US data showing small variations of BMI at age 3 since the early 70s (Ogden et al., 2004). A favourable environment may also be correlated with a higher BMI at birth, and with lower BMI slope in the period between 0 to 3 years of age, birth weight being inversely correlated with social status and healthy behaviours (McGovern, n.d.). In contrast, at age 11 years, we calculated that if all children had the most favourable environment, mean BMI would be reduced by 0.91 kg/m^2 (95% CI: 0.57–1.26) for boys and by 1.65 kg/m^2 (95% CI: 1.28–2.02) for girls. This suggests that the role of the environment may be greater for girls than for boys, which is consistent with recent historical trends in child BMI changes (Johnson et al., 2015), and with other studies in adults (Wardle et al., 2002). When analysing the link between parents' social class and children's BMI, we observed the emergence of a strong social gradient between 3 and 11 years of age. It is useful to examine our results in the light of longer term and recent historical trends in child BMI changes. We calculated that the BMI mean at age 11 years in our sample would be 18.0 kg/m^2 for boys and 17.7 kg/m^2 for girls if every child had the most favourable environment. These results are close to the observed average at age 11 years in the 1958 British birth cohort: 17.3 kg/m^2 for boys and 17.6 kg/m^2 for girls (Power et al., 1997). Yet, the 1958 British birth cohort corresponds to a generation likely to be born before the rise in children's BMI mean (Johnson et al., 2015). The possible reduction we estimated (0.91 kg/m^2 for boys, 1.65 kg/m^2 for girls) is also close to the evolution of median BMI of 11-year-old between the British 1946 NSHD cohort and 2001 MCS cohort (1.2 kg/m^2 for boys and 1.7 kg/m^2 for girls) (Johnson et al., 2015). This highlights the important role of the environment on children's BMI and suggests that the recent rise of children's BMI is likely to be reversible and due to modifiable environmental factors.

One of the most important results of our study is in the nature of the association between parental BMI, environment and children's BMI. Our results confirm that parental BMI might capture genetic information, but is also a broad proxy of the environment. The links between maternal and paternal BMI and children's BMI are robust to the adjustment for environmental variables, which is consistent with the fact that BMI is partly genetically transmitted (Speliotes et al., 2010; Frayling et al., 2007; Elks et al., 2014). Conversely, the link between the environment and children's BMI is reduced after adjustment for parental BMI, which shows that parental BMI and environment are not independent.

The environmental factors we used mainly described family- and individual-level environment. We fully acknowledge that this is the main weakness of this work given that we subscribe to a socio-ecological model. Some important factors of children's BMI are not included such as nutritional practices, and community-level, neighbourhood (Burdette and Needham, 2012) or structural level factors like the built environment (Duncan et al., 2014) or schools/preschools (Olesen et al., 2013). Therefore, we most likely underestimated the role of the environment, in its broad sense. A number of environmental factors such as diet may partly explain the links between parent's and child's BMI.

Table 6
Counterfactual analyses for girls of BMI at age 3 years, BMI at age 11 years, BMI gain per year between 3 and 5 years old, 5 and 7 years old, and 7 and 11 years old.

	Environment		Parents' overweight	
Girls	Model 1a[1]	Model 1a–Model 2[3]	Model 1b[2]	Model 1b–Model 2
BMI (in kg/m^2)				
At age 3 years	0.04 [−0.09–0.16]	NC	0.13 [0.10–0.16]***	−0.00 [−0.01–0.00]
At age 11 years	1.65 [1.28–2.02]***	0.32 [0.18–0.46]***	0.64 [0.57–0.71]***	−0.00 [−0.02–0.01]
BMI slope (in kg/m^2/year)				
Between 3–5 years of age	0.11 [0.05–0.17]***	0.01 [0.00–0.02]**	0.05 [0.03–0.06]***	0.00 [−0.00–0.00]
Between 5–7 years of age	0.14 [0.08–0.20]***	0.02 [0.01–0.03]***	0.05 [0.04–0.06]***	0.00 [−0.00–0.00]
Between 7–11 years of age	0.22 [0.16–0.28]***	0.04 [0.02–0.06]***	0.08 [0.07–0.09]***	−0.00 [−0.00–0.00]

NC: Not calculated because Model 1a did not show significant effect.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.
[1] Model 1a: difference between observed BMI or BMI slope and predicted BMI or BMI slope in a favourable environment (controlled for age and ethnicity) (see methods).
[2] Model 1b: difference between observed BMI or BMI slope and predicted BMI or BMI slope for children if no parent had BMI > 25 kg/m^2 (controlled for age and ethnicity).
[3] Model 2: difference between observed BMI or BMI slope and predicted BMI or BMI slope for children in a favourable environment (controlled for age, ethnicity and parental BMI)/if no parent had BMI > 25 kg/m^2 (controlled for age, ethnicity and environment).

However, the objective of this study is not to determine the specific independent effect of each variable on BMI or BMI slopes, because our observational data do not allow us to determine specific causal links. We suggest that by treating the individual level variables we do have as a set, we attempt to describe the environment in a general way capturing some of more distal environmental factors. For this reason we chose to express the overall information from our models in a counterfactual analysis. Moreover, the proxies selected cannot be extrapolated, given the fact that we studied children born in UK in 2000–2002. The same results may not be observed in countries at different stages of socioeconomic development (Wang, 2001) or with different institutional organisations in particular in preschool care. Attrition and missing data may also introduce a bias in our results. The results may be underestimated because children living in favourable environments are more likely to be included in the study (Supplementary material). We implemented sensitivity analyses using a different sub-sample of the MCS study to test the robustness to a potential selection bias. The results remained unchanged for both Models 1a and 1b (Supplementary material). Only the effect of the environment "explained by parental BMI" was lower than in our sample. Another limitation was due to the lack of information on BMI and behaviours before 3 years of age. This prevented us from interpreting the absence of a link between the environment and the children's BMI at age 3 years. More generally, having more measures of BMI per child would have allowed us to be more precise in the description of the role of environment over childhood and specifically, its role in the adiposity rebound.

Despite these limitations, this study has a number of strengths. It is a longitudinal population-based study collecting data prospectively across the life span, with a large sample size. We had measures of children's BMI at different ages. We implemented a model which did not constrain the link between environment, parental BMI and BMI slope during childhood to be stable between 3 and 11 years of age. It permitted us to show that the role of environment is more important during the period between 7 and 11 years of age compared to earlier age periods [3 to 5 and 5 to 7]. The originality of this work is to use a counterfactual analyses to estimate the overall mean impact of the environment on BMI and on BMI slopes across different periods between 3 and 11 years old for both girls and boys.

Our study highlighted that if every child's environment was at its most favourable, mean BMI would be significantly reduced. Moreover, the environment becomes increasingly important in determining childhood BMI over time. Thus, the recent rise of mean BMI in populations of children is likely to be reversible. However there is a lack of knowledge on how to reverse this trend other than by targeting individual behaviours. The broader environment in which socially stratified populations live is a potential target for interventions. Research on complex real-life public health interventions and lifecourse research such as our study, remain separate areas of public health investigation. Bringing these two areas together where longitudinal data analysed within a lifecourse approach may be used to provide new hypotheses on how public policies may improve public health.

Tranparency Document

The Transparency Document associated with this article can be found, in the online version.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ypmed.2016.05.028.

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