

# Predicting adult obesity from measures in earlier life

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Received 19 September 2012

Revised 30 April 2013

Accepted 11 July 2013

Published Online First

19 September 2013

## ABSTRACT

**Background** As most obese adults were not overweight as children, the prediction of adult obesity from childhood body size alone is limited. We constructed a two-way, multifactor risk assessment framework for predicting adult obesity during childhood using the Foresight Obesity System Map and tested it against longitudinal data from the 1958 National Child Development Study.

**Methods** The framework divided study participants according to two categories of risk: 'conditioning factors' (past/fixed events and conditions) and 'intervention factors' (present and modifiable). At the age of 11 years, conditioning factors were 'low/high birth weight' and 'absence of breastfeeding', and intervention factors were 'low childhood activity level' and 'having at least one obese parent'. From a composite score of all four variables, study participants were assigned to one of the four risk groups: low risk, past 'conditioning' risk only, present 'intervention' risk only and high combined risk. ORs and relative risks for the development of future overweight/obesity at ages 23, 33 and 42 years were calculated for each risk group.

**Results** Those identified in the highest risk category at the age of 11 were around twice as likely to become overweight (body mass index (BMI)  $\geq 25$  kg/m<sup>2</sup>) by the age of 23 years, and obese (BMI  $\geq 30$  kg/m<sup>2</sup>) by ages 33 and 42 years, in comparison to their low-risk peers (total sample, N=11 752). Increased prevalence of future obesity was also observed for high-risk children who were not already overweight at the age of 11 (filtered sample, N=9549).

**Conclusions** This framework identifies a greater proportion of the population that is at risk for future obesity than does childhood weight assessment alone.

## INTRODUCTION

The main strategy for predicting adult obesity is based on body size in childhood; people who are overweight in early life tend to remain so in later years.<sup>1 2</sup> Current public health programmes for reducing the future burden of obesity are thus overwhelmingly targeted towards overweight children. Longitudinal studies for predicting obesity emergence across the life course are few,<sup>3</sup> with most being limited to tracking obesity emergence within childhood rather than across the transition from childhood to adulthood.<sup>4 5</sup> Previous studies have highlighted the low sensitivity of an obesity prediction model based solely on childhood body size,<sup>2 6 7</sup> suggesting that other factors must be taken into account. For most people, obesity emerges in mid-adulthood<sup>8</sup> through a complex web of influences across the life course; obesity prediction models based on childhood body size alone will therefore fail to identify much of the population that ultimately becomes obese.

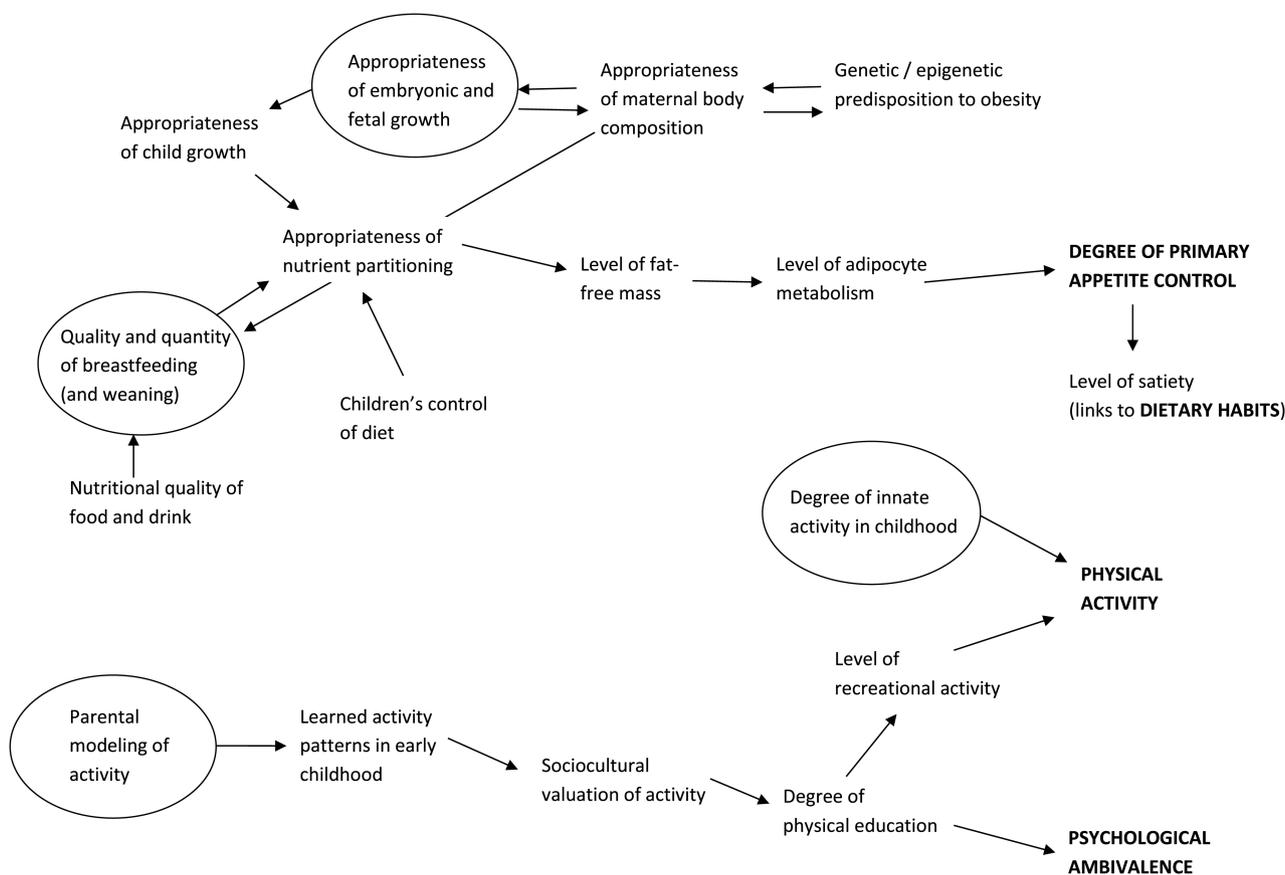
We present a multifactor framework for predicting adult obesity from ecological measures of obesity risk in childhood. This is derived from the *Tackling Obesities: Future Choices* project,<sup>9</sup> carried out by the UK government think-tank Foresight, which has provided the most wide-ranging ecological perspectives on obesity causation to date. One of its key outputs was the Foresight Obesity System Map (FOSM),<sup>10</sup> which is a visual representation of the complex interactions between 108 variables thought by experts to contribute to the causation of obesity at the population level. These variables range from individual-level biology to wider social and environmental factors.

To date, there has been little attempt to operationalise the information contained in the FOSM towards the development of a practical public health tool for obesity prevention.<sup>11</sup> Of the 108 FOSM variables, 17 were identified as being relevant for obesity interventions at early-life stages.<sup>10</sup> Collectively, these are seen to influence three key obesity drivers: biological mechanisms that regulate appetite; amounts of energy expended; and conflicting motivations that inform individual behaviours (figure 1). The variables depicted in the FOSM are conceptual rather than directly measurable. Our aim was to identify potential proxy variables for the early-life FOSM variables from existing longitudinal datasets, and to evaluate the combined predictive power of these for obesity emergence later in life.

## METHODS

The analysis plan involved the selection of early-life FOSM variables to create a multifactor obesity risk framework, identification of measurable proxies for these from an on-going British birth cohort study, and testing of these combined variables against obesity rates within the same database. The Waterloo<sup>12</sup> classification scheme, a practical tool for identifying those most at risk for undernutrition based on past as well as present risk, inspired our approach. We constructed a two-way framework (figure 2) and termed these past/fixed and present/modifiable categories of variables 'conditioning factors' and 'intervention factors', respectively. From the 17 early-life FOSM variables (figure 1), conditioning factors include biological characteristics that cannot be currently manipulated, past life events that are no longer malleable and unpredictable influences of the wider social and economic environment. Intervention factors, in contrast, are potentially modifiable. Some can shift in response to changing home dynamics and educational environments, while others may respond to wider changes in the economy or society. Some variables can change their status from intervention factor to conditioning factor across the life course. Quality and

**To cite:** Potter CM, Ulijaszek SJ. *J Epidemiol Community Health* 2013;**67**:1032–1037.

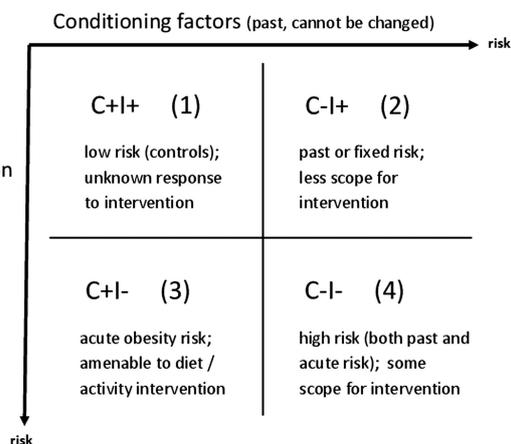


**Figure 1** Relationships between early-life obesity variables within the Foresight Obesity System Map (FOSM).

quantity of breastfeeding, for instance, is subject to intervention during infancy, but becomes fixed at the point of weaning.

For testing the predictive power of this two-way obesity risk framework we accessed longitudinal data from the 1958 National Child Development Study (NCDS),<sup>13</sup> which are available to academic researchers through the UK Data Service. Data were anonymised prior to the authors' access to them, such that individual study participants were not identifiable. The NCDS population comprises all 17 638 people born in England, Scotland and Wales during the week of 3–9 March 1958. The survey has now undergone eight additional sweeps since the

birth year and includes wide-ranging data on health, family, education, employment and social opportunity. In addition to birth weight, heights and weights were recorded during follow-up sweeps when cohort members were 7, 11, 16, 23, 33 and 42 years old; these were self-reported at ages 23 and 42 years and were in line with time-specific measured British samples,<sup>14</sup> and for all other ages they were directly measured. From candidate variables in the NCDS database, we constructed our framework based on data from birth to the 1969 sweep, which included data on family, school and activity that could be used as proxies for FOSM intervention factors when cohort members were 11 years old.



**Figure 2** Obesity risk classifications capturing past and present risk factors.

### Selected variables

FOSM variables are conceptual, and not all are directly measurable. We aimed to select 'upstream' FOSM variables for our framework, which could influence more 'downstream' ones (see figure 1) and which, crucially, were measurable via proxies in the NCDS database. Four early-life FOSM variables were identified on this basis: appropriateness of embryonic and fetal growth, quality and quantity of breastfeeding, degree of innate activity in childhood and parental modelling of activity. These could be approximated by the NCDS variables of birth weight, breastfeeding history, parent and teacher reporting of child's activity at the age of 11 years and parents' body mass index (BMI) status when the child was 11 years old, respectively. At the age of 11 years, the first two variables are 'conditioning factors' (capturing past developmental risk), while the second two are 'intervention factors' (present-day risk) that are potentially modifiable. As our framework stemmed directly from the FOSM—which represents the best cross-disciplinary evidence

base on obesity currently available—covariates that did not map onto FOSM variables were not included in our analysis.

*Birth weight* is easily measurable, routinely recorded and can be indicative of fetal malnutrition or poor growth. There is a robust relationship between small birth weight and increased prevalence of obesity comorbidities in adulthood.<sup>15–16</sup> High birth weight also carries increased risk for these comorbidities, particularly if it is indicative of diabetes within the mother during pregnancy.<sup>17</sup> Within our model, a person was deemed at risk if his/her birth weight was 2.5 kg or less, corresponding to the internationally recognised threshold at which infant mortality and morbidity increases.<sup>18</sup> No internationally standardised cut-off for high birth weight exists, although the USA Centers for Disease Control place it at 4 kg.<sup>19</sup> We used this cut-off for our framework; it captured the vast majority of NCDS participants who were large for gestational age.

While the empirical evidence for a protective effect of *breast-feeding* against obesity later in life remains equivocal,<sup>20–21</sup> some studies are suggestive of such an effect.<sup>22</sup> Maternal recall of breastfeeding initiation has been found to be accurate across multiple studies,<sup>23</sup> including some recalled up to 20 years later.<sup>24</sup> Duration of breastfeeding may also be important for conferring a protective effect against obesity, but its recall might be less accurate after the child is 3 years old.<sup>23</sup> Within the NCDS, breastfeeding was recalled by the mother when the cohort member was 7 years old, with little detail as to duration; responses to the question ‘breastfed partially or wholly?’ were limited to ‘don’t know’, ‘no’, ‘less than 1 month’ or ‘more than 1 month’. As nuanced information on breastfeeding duration was not collected while the cohort member was an infant, we limited our at-risk assessment to those for whom no breastfeeding was reported.

The FOSM variable ‘Degree of innate activity in childhood’ refers to the ‘degree to which physical activity is part of typical childhood behaviour’.<sup>10</sup> This *habitual activity* includes, but is not exclusive to, organised recreational activity and is therefore difficult to capture empirically. From the NCDS data, we identified five questions regarding activity levels at the age of 11 years: the frequency reported by a parent of the child’s use of public parks, recreation grounds, swimming pools and indoor play centres during the past 12 months, as well as a teacher’s assessment of the child’s frequency of participation in sports outside of school hours. Children who did none of these five activities ‘often’ (taken to be the best indication of habitual activity, versus alternative responses of ‘do not know’, ‘never’ or ‘sometimes’) were given an at-risk mark.

‘Parental modeling of activity’ was the most difficult FOSM variable to approximate, as no data on parent activity levels were available. However, *parental BMI* could be calculated for both parents when the cohort member was 11 years old, when parent height and weight were self-reported to the nearest inch and half-stone (7 lb) increment, respectively. Although evidence strongly indicates that children of obese parents are more likely to become obese themselves,<sup>3</sup> the mechanisms for this influence remain unclear.<sup>25</sup> However, evidence that children of obese parents spend more time engaging in sedentary activities<sup>26</sup> suggests a link between parent BMI status and modelling of activity. As body size is never permanently fixed, interpreting parent BMI status as an ‘intervention’ factor enables us to better approximate the complex influence of parents on their children’s health status,<sup>25</sup> versus a more rigid genetics/programming interpretation that would categorise it as a fixed ‘conditioning’ factor. In the NCDS, parental obesity was relatively rare (see table 1); we therefore gave an at-risk mark for parental

modelling of activity to cohort members who had at least one obese parent ( $BMI \geq 30 \text{ kg/m}^2$ ) at the age of 11 years. While parental BMI might inadvertently capture multiple variables within the FOSM, it is also a potentially more robust measure than self-reported activity levels, which correlate poorly with directly measured activity levels.<sup>27</sup>

### Risk classifications and calculations

Participants received a Conditioning Factor score of C+ if they were of normal birth weight and had been breastfed for any amount of time. Any designation of an at-risk factor garnered a classification of C-. Similarly, participants with a positive Intervention Factor score (I+) were reported as doing some frequent activity and did not have an obese parent; otherwise, they were classified as I-. Binary variables (at-risk/no risk) were created for each factor using SPSS (V.16), which were then combined and coded for the four obesity risk classifications depicted in figure 2 (1=low risk, 2=past risk, 3=acute/modifiable risk, 4=high risk). ORs were calculated for each of the three risk categories, with respect to the low-risk category of C+I+, of future overweight (including obesity, adult  $BMI \geq 25 \text{ kg/m}^2$ ) at ages 23, 33 and 42 years. ORs were also calculated for each risk category for future obesity (adult  $BMI \geq 30 \text{ kg/m}^2$ ) at the same ages. The prevalence of obesity and overweight increased markedly in the cohort between ages 23 (in 1981) and 33 (in 1991); we therefore also calculated relative risks for each category at each age, which is the more intuitive risk measure, but which tends to diverge from the standardly reported OR when the outcome of interest becomes common.<sup>28</sup>

As most overweight/obese adults were not overweight as children, our multifactor framework was intended to predict future obesity irrespective of childhood body size. We therefore retested the framework among a filtered sample ( $N=9549$ ) of cohort members who were not already overweight at the age of 11 years, when risk classification was determined. International sex-specific BMI cut-offs for childhood overweight<sup>29</sup> were used to identify the filtered sample. ORs and relative risks for adult overweight (including obesity), as well as for adult obesity, were again calculated for the three risk groups (C-I+, C+I-, and C-I-) with respect to the low-risk group (C+I+) for all ages.

Overweight including obesity ( $BMI \geq 25 \text{ kg/m}^2$ ) was used as an outcome in preference to overweight ( $25 \text{ kg/m}^2 \leq BMI < 30 \text{ kg/m}^2$ ) because the latter does not clearly increase risk for obesity comorbidities<sup>30</sup>; our concern was to chart life-course progression towards obesity ( $BMI \geq 30 \text{ kg/m}^2$ ), which is the more biologically meaningful category. The overweight including obesity category was particularly salient at the age of 23, when rates of obesity were low.

### RESULTS

Among the total NCDS population, the available data allowed us to calculate a composite (CI) risk classification for 11 752 people at the age of 11 years. Among this sample, 47% were deemed low risk, 29% were at risk on the basis of past developmental factors (C-), 13% were at risk on the basis of present modifiable factors (I-) and 11% were at high risk on the basis of past as well as present risk (C-I-). Table 1 shows the risk prevalence among the NCDS sample for the four individual factors, for the Conditioning and Intervention categories separately, and for the combined four-factor (Conditioning plus Intervention) framework. When the combined framework is employed, 53% of the sample emerges as at risk at the age of 11 for future obesity, a figure in line with the 52% of the sample that was overweight (including obesity,  $BMI \geq 25 \text{ kg/m}^2$ )

**Table 1** Risk prevalence within National Child Development Study for single factors, Conditioning and Intervention factors separately and combined CI framework

	Conditioning factor 1: low or high birth weight	Conditioning factor 2: never breastfed	Intervention factor 1: no regular activity—age 11 years	Intervention factor 2: at least one parent obese—age 11 years	Conditioning risk factor(s)	Intervention risk factor(s)	Two-way framework: Conditioning and/or Intervention risk factor(s) combined
No risk	14 435	9703	11 835	11 371	8366	9803	5520
At risk	2906	4436	2739	1565	5714	3133	6232
Total N	17 341	14 139	14 574	12 936	14 080	12 936	11 752
Per cent measured (of N=17 638)	98.3	80.2	82.6	73.3	79.8	73.3	66.6
<b>Percentage of measured at risk</b>	<b>16.8</b>	<b>31.4</b>	<b>18.8</b>	<b>12.1</b>	<b>40.6</b>	<b>24.2</b>	<b>53.0</b>

by the age of 42 (see table 2A). In contrast, only 10% of the sample was overweight on the basis of age-standardised BMI measurements<sup>29</sup> at the age of 11 (results not shown; see also Cheung *et al*<sup>31</sup> who report similar figures), which indicates the poor sensitivity of childhood BMI as a predictor of adult obesity when used in the isolation of other risk factors.

Table 2A shows that across all ages, the prevalence of obesity among the high-risk group (C–I–) was approximately double that of the low-risk group (C+I+), with a slight rise in obesity prevalence among the past-risk group (C–I+) and a further rise among the acute-risk group (C+I–) in comparison to the low-risk group. Across all ages, ORs for future obesity among the high-risk group (C–I–) were a minimum of 2.288 (at the age of 42 years) and were highly significant. ORs for future obesity were also consistently high and significant for the acute-risk group (C+I–) in comparison to the low-risk group. With the exception of age 42 years (mainly attributable to women; results not shown), the past-risk group (C–I+) did not show significantly increased risk for future obesity, indicating that the effects of birth weight and/or breastfeeding on future overweight/obesity were not particularly pronounced within the NCDS sample.

For the acute-risk group as well as the high-risk group and for overweight/obesity as well as obesity, the value of the OR falls across the life course, but nonetheless remains meaningful by the age of 42 years. As expected, the relative risk figures for overweight/obesity at ages 33 and 42 years were notably lower than the corresponding OR values, since overweight/obesity prevalence by this point was far above 10%.<sup>28</sup> The relative risk figures for obesity, however, paralleled the magnitude of the corresponding OR values at all ages—indicating a genuine and persistent increased risk for the C–I– group across the life course.

Table 2B shows the ORs and relative risks for each group among a filtered sample (N=9549) of cohort members who were not already overweight/obese at the age of 11 years, when risk classifications were assessed. Among the high-risk group, significant and meaningful results were again obtained, across all adult ages and for overweight/obesity as well as obesity, in comparison to the low-risk group. No significant increased risk was found for those in the past-risk group (C–I+), suggesting that those with early-life risk who ultimately became overweight or obese had usually already done so by the age of 11. The lower obesity prevalence among the filtered, non-overweight sample in comparison to the total sample indicates that some of those at increased risk according to our framework were already overweight by the age of 11 years and continued to remain so at

later ages. However, the consistently observed increased risk among the C–I– group suggests that the framework correctly identified those most likely to become overweight or obese later in life, even among those who exhibited normal weight-for-height at the time of risk assessment.

## DISCUSSION

This analysis yields two key findings. The first is that a focus only on overweight children, which accounts for much of the literature on obesity prediction and drives most obesity prevention strategies, may not account for most of the currently observed prevalence of obesity among adults. Our results are consistent with a previous analysis of the NCDS cohort, which demonstrated that although participants with high BMI in childhood tended to remain in the higher percentiles as adults, the vast majority of cohort members who were obese by the age of 33 years would not have been identified as fat children.<sup>14</sup> Medical and public health programmes that focus exclusively on overweight children will fail to identify the substantial number of at-risk people who could potentially benefit from intervention, but for whom obesity does not emerge until adulthood.

The second concerns the value of an ecological, multifactor obesity risk framework that spans multiple phases of the life course. The increased likelihood of future obesity among the high-risk group within the non-overweight sample suggests that more complex processes were in operation<sup>32</sup> than people merely tracking from childhood to adulthood along high BMI centiles. An alternative focus on obesity risk in connection with the developmental origins of health and disease hypothesis, which focuses exclusively on the fetal and infant phases of the life course,<sup>33</sup> may similarly mask the influence of processes that occur beyond infancy and unnecessarily limit the scope for intervention. Our analysis did not indicate a strong neonatal effect on adult obesity in isolation, but in combination with later ‘intervention’ factors it informed our successful identification of those who were most at risk for future obesity within this large longitudinal sample; if anything, the NCDS data might under-represent the capacity of this multifactor framework for predicting obesity within the British population, as sample attrition has occurred somewhat more rapidly in groups that might be expected to have higher obesity rates.<sup>34</sup> As a first testing of this framework using real-world data, our analysis was necessarily limited by which, and to what extent, conceptual variables from the FOSM could be approximated by NCDS variables. However, in spite of such limitations, this framework

**Table 2.** Sample sizes, ORs, and relative risks for overweight (including obese) and obesity at later ages, by risk category at the age of 11 years

Age 23 years				Age 33 years			Age 42 years		
<b>A. Total sample (N=11,752): all children at the age of 11 years</b>									
Risk group†	N (% Overweight‡)	OR§ (95% CI)	Relative risk§ (95% CI)	N (% Overweight)	OR (95% CI)	Relative risk (95% CI)	N (% Overweight)	OR (95% CI)	Relative risk (95% CI)
C+I+ (low)	4285 (14%)	1	1	3915 (40%)	1	1	3930 (49%)	1	1
C-I+ (past)	2616 (16%)	1.132 (0.989 - 1.295)	1.111 (0.991 - 1.245)	2330 (42%)	1.095 (0.986 - 1.215)	1.055 (0.992 - 1.121)	2366 (51%)	1.076 (0.971 - 1.191)	1.037 (0.986 - 1.091)
C+I- (acute)	1186 (20%)	1.482*** (1.255 - 1.749)	1.386*** (1.210 - 1.586)	1055 (47%)	1.309*** (1.142 - 1.501)	1.165*** (1.081 - 1.255)	1061 (56%)	1.325*** (1.156 - 1.518)	1.143*** (1.074 - 1.216)
C-I- (high)	975 (27%)	2.249*** (1.908 - 2.651)	1.906*** (1.681 - 2.160)	852 (56%)	1.907*** (1.642 - 2.215)	1.398*** (1.303 - 1.501)	836 (63%)	1.767*** (1.516 - 2.061)	1.285*** (1.209 - 1.365)
Totals	9062 (17%)			8152 (43%)			8193 (52%)		
Risk group	N (% Obese <sup>¶</sup> )	OR (95% CI)	Relative risk (95% CI)	N (% Obese)	OR (95% CI)	Relative risk (95% CI)	N (% Obese)	OR (95% CI)	Relative risk (95% CI)
C+I+ (low)	4285 (2%)	1	1	3915 (9%)	1	1	3930 (13%)	1	1
C-I+ (past)	2616 (2%)	1.303 (0.918 - 1.851)	1.297 (0.919 - 1.829)	2330 (11%)	1.148 (0.968 - 1.361)	1.132 (0.972 - 1.320)	2366 (15%)	1.191* (1.028 - 1.381)	1.163* (1.024 - 1.321)
C+I- (acute)	1186 (4%)	2.575*** (1.785 - 3.716)	2.509*** (1.759 - 3.578)	1055 (15%)	1.764*** (1.446 - 2.153)	1.647*** (1.387 - 1.956)	1061 (20%)	1.766*** (1.480 - 2.107)	1.610*** (1.393 - 1.860)
C-I- (high)	975 (6%)	3.701*** (2.600 - 5.268)	3.540*** (2.523 - 4.968)	852 (19%)	2.301*** (1.880 - 2.816)	2.052*** (1.733 - 2.430)	836 (25%)	2.288*** (1.906 - 2.747)	1.967*** (1.704 - 2.271)
Totals	9062 (3%)			8152 (11%)			8193 (15%)		
<b>B. Filtered sample (N=9,549): non-overweight children at the age of 11 years</b>									
Risk group†	N (% Overweight‡)	OR§ (95% CI)	Relative risk§ (95% CI)	N (% Overweight)	OR (95% CI)	Relative risk (95% CI)	N (% Overweight)	OR (95% CI)	Relative risk (95% CI)
C+I+ (low)	3658 (12%)	1	1	3360 (37%)	1	1	3353 (47%)	1	1
C-I+ (past)	2147 (12%)	1.046 (0.886 - 1.233)	1.040 (0.899 - 1.203)	1920 (39%)	1.069 (0.952 - 1.200)	1.042 (0.970 - 1.119)	1957 (48%)	1.040 (0.930 - 1.162)	1.021 (0.966 - 1.082)
C+I- (acute)	927 (14%)	1.288* (1.044 - 1.589)	1.247* (1.040 - 1.494)	816 (41%)	1.176* (1.006 - 1.374)	1.104* (1.005 - 1.212)	825 (51%)	1.162 (0.998 - 1.354)	1.080* (1.001 - 1.166)
C-I- (high)	707 (20%)	1.915*** (1.552 - 2.364)	1.733*** (1.458 - 2.060)	627 (51%)	1.757*** (1.480 - 2.086)	1.372*** (1.256 - 1.499)	609 (58%)	1.582*** (1.328 - 1.884)	1.243*** (1.151 - 1.341)
Totals	7439 (13%)			6723 (39%)			6744 (49%)		
Risk group	N (% Obese <sup>¶</sup> )	OR (95% CI)	Relative risk (95% CI)	N (% Obese)	OR (95% CI)	Relative risk (95% CI)	N (% Obese)	OR (95% CI)	Relative risk (95% CI)
C+I+ (low)	3658 (1%)	1	1	3360 (7%)	1	1	3353 (11%)	1	1
C-I+ (past)	2147 (1%)	1.211 (0.700 - 2.097)	1.209 (0.702 - 2.083)	1920 (8%)	1.140 (0.922 - 1.411)	1.129 (0.928 - 1.375)	1957 (12%)	1.099 (0.921 - 1.312)	1.088 (0.930 - 1.273)
C+I- (acute)	927 (2%)	2.186* (1.204 - 3.967)	2.164* (1.203 - 3.893)	816 (9%)	1.392* (1.063 - 1.823)	1.355* (1.060 - 1.733)	825 (14%)	1.364** (1.088 - 1.709)	1.313** (1.079 - 1.597)
C-I- (high)	707 (2%)	2.020* (1.032 - 3.953)	2.003* (1.034 - 3.881)	627 (14%)	2.181*** (1.679 - 2.833)	2.015*** (1.602 - 2.536)	609 (17%)	1.774*** (1.400 - 2.248)	1.639*** (1.344 - 2.000)
Totals	7439 (1%)			6723 (8%)			6744 (12%)		

†Assessed from four variables (see Table 1) at the age of 11 years

‡Overweight including obesity, body mass index (BMI) >25 kg/m<sup>2</sup>

§Significance levels: (\*)=p<0.05, (\*\*)=p<0.01, (\*\*\*)=p<0.001

¶Obesity, BMI >30 kg/m<sup>2</sup>

shows initial promise for predicting who is most likely to become obese later in life, irrespective of childhood body size.

### What is already known on this subject

- ▶ Most obese adults were not overweight as children. Current obesity predictions are based on childhood body size and do not account for multiple risks across the life course.

### What this study adds

- ▶ We propose a multifactor framework for predicting obesity that accounts for past (fixed) as well as present (modifiable) risk, based on the complex causal pathways represented in the UK Foresight Obesity System Map.
- ▶ Testing this framework against longitudinal British birth cohort data (N=11,752) shows it to predict future adult obesity among 11-year-olds considered at risk by this model, whether or not they were already overweight in childhood.
- ▶ The framework could be developed towards a public health tool for obesity prevention, by identifying those most at risk for future obesity including many who would not have been identified based on childhood body size alone.

**Acknowledgements** The authors would like to thank Foresight for permitting the use of the Foresight Obesity System Map for this purpose. The School of Anthropology and Museum Ethnography at the University of Oxford provided on-going support and resources (including salary support) to carry out the research. We are indebted to the many researchers and participants who have taken part in the collection of primary National Child Development Study data. This study is a secondary analysis of NCDS data made available to academic researchers by the Centre for Longitudinal Studies (Institute of Education, London, UK) and UK Data Service (formerly Economic and Social Data Service, Universities of Essex and Manchester); on-going sweeps of the NCDS are funded by the UK Economic and Social Research Council. Other British birth cohort researchers, particularly those carrying out the 1946 National Survey of Health and Development, provided insight on working with British cohort data in general; we especially thank Diana Kuh, Rebecca Hardy and Leah Li for their expertise. We heartily thank colleagues within Oxford's Unit for Biocultural Variation and Obesity who provided feedback on the overall framework concept and/or comments on the study results, including John Komlos, Klim McPherson, Charlie Foster and Harry Rutter.

**Contributors** SJU derived the original model and developed the preliminary analysis. CMP carried out secondary analysis and wrote the first draft of the paper. Both authors have been involved in redrafting the paper.

**Competing interests** None.

**Provenance and peer review** Not commissioned; externally peer reviewed.

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