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The utility of childhood and adolescent obesity assessment in relation to adult health

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Abstract

The high prevalence of childhood obesity has raised concerns regarding long-term patterns of adult health and has generated calls for obesity screening of young children. This study examined patterns of obesity and the predictive utility of obesity screening for children of different ages in terms of adult health outcomes. Using the National Longitudinal Survey of Youth, the Population Study of Income Dynamics, and National Health and Nutrition Evaluation Surveys, we estimated the sensitivity, specificity and predictive value of childhood BMI to identify 2, 5, 10, or 15 year-olds who will become obese adults. We constructed models assessing the relationship of childhood BMI to obesity-related diseases through middle age stratified by sex and race/ethnicity. 12% of 18 year-olds were obese. While 50% of these adolescents would not have been identified by screening at age 5, 9% would have been missed at age 15. Approximately 70% of obese children at age 5 became non-obese at age 18. The predictive utility of obesity screening below the age of 10 was low, even when maternal obesity was also included. The elevated risk of diabetes, obesity, and hypertension in middle age predicted by obesity at age 15 was significantly higher than at age 5 (e.g., the RR of diabetes for obese white male 15 year-olds was 4.5; for 5 year-olds, it was 1.6). Early childhood obesity assessment adds limited predictive utility to strategies that also include later childhood assessment. Targeted approaches in later childhood or universal strategies to prevent unhealthy weight gain should be considered.

Keywords

Child; adolescent; adult; obesity; risk assessment; type 2 diabetes mellitus; hypertension; forecasting

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Introduction

The dramatic increase in childhood obesity over the past three decades has generated considerable concern regarding the health of children as well as patterns of obesity and related chronic conditions later in life (1–3). In response, the U.S. Preventive Services Task Force recently recommended that children's body mass index (BMI) should be assessed starting at age 6 (4). The focus on obesity in young children is motivated by the fact that they may suffer from a variety of social and health effects (5, 6), use more health care services (7), and are forming life-long eating and exercise habits (8). These concerns and the prospect of elevated risks for adult obesity and chronic disease have generated a variety of interventions designed to improve childhood nutrition, physical activity, and other health-related behaviors.

Although these interventions may prove useful, the dynamics of obesity from childhood into adulthood that determine their utility remain relatively unexplored. While BMI in adulthood tends to increase more or less consistently in later ages, a number of studies suggest that childhood BMI is more variable, particularly in younger children (1, 9–13). Some obese children increase rapidly in height and cease to be obese, while some non-obese children gain weight rapidly, becoming obese adults (14, 15). This apparent variation in the prevalence and progression of obesity in childhood may be important in assessing the utility of different screening and intervention protocols directed at reducing childhood obesity and its implications for later adult health. While some interventions delivered to children who are obese are effective for a given period of childhood, there is much less evidence demonstrating that these interventions are broadly effective for non-obese children or can alter patterns of obesity into adulthood (16–26). In order to effectively target obesity interventions in childhood, it would be important to correctly identify a significant portion of children likely to become obese adults while not incorrectly labeling others who go on to be normal weight adults (27). The ability of current recommendations for obesity assessment in early childhood to simultaneously achieve both of these goals remains unclear.

This study seeks to assess the developmental patterns of obesity over childhood, the portion of adult obesity and related chronic conditions associated with childhood obesity, and the relative utility of assessing childhood obesity at different ages. A number of longitudinal studies have examined the relationship between childhood obesity and adult obesity and disease risk in specific geographic populations (9–11, 13–15). We extend this prior work by examining the utility of childhood obesity assessment, modeling the relationship between longitudinal patterns of child weight change and growth and adult risks for the U.S. Because no nationally-representative, longitudinal studies of obesity from early childhood through older adulthood exist, this study links longitudinal data from several national studies to create synthetic cohorts capable of providing empirical insight into these issues.

Methods

Overview

We assessed the potential value of child obesity assessment in terms of efficiently detecting individuals at elevated risk of adult obesity and chronic disease risk in the United States. Because no single U.S. nationally-representative, longitudinal dataset exists that characterizes the obesity trajectories from childhood through middle age, we applied the following steps:

- Step 1: Measure the correlation between obesity at earlier ages and later ages in childhood and adolescence using the National Longitudinal Survey of Youth (NLSY) (i.e., predict obesity at age 18 based on BMI status at earlier ages).

Similarly, use the Population Study of Income Dynamics (PSID) to assess this correlation between obesity at age 18 and obesity and chronic disease risk at age 40. We refer to this as the "*predictive utility*" of obesity assessment.

- Step 2: Combine the NLSY and PSID BMI trajectories into a single model of obesity from childhood through middle age (the Stanford Childhood Obesity Prediction and Evaluation (SCOPE) model), using bootstrapping and statistical matching. Impute chronic disease risks based upon the relationship between individual characteristics such as age, sex, race, and BMI in middle age and biological markers of chronic disease as observed in the current adult National Health and Nutrition Evaluation Surveys (NHANES) sample.
- Step 3: Forecast future obesity and chronic disease health risks for current children in the U.S. by applying the SCOPE model to a third nationally-representative dataset, the current child NHANES sample, again using statistical matching to attach SCOPE obesity trajectories to children of a given age in NHANES based on BMI, sex, and race. We refer to this as the "*future obesity and health*" implications of the current childhood BMI patterns.

Definitions

We computed BMI as weight (kilograms) divided by height (meters) squared. We classified childhood BMIs at or above the age- and sex-specific 95^{th} percentiles of the 2000 U.S. Centers for Disease Control and Prevention (CDC) reference standard as obese, while we classified those at or above the 85^{th} and below the 95^{th} percentiles as overweight (28). For adults (i.e., individuals age ≥18), BMIs of 30 and over were classified as obese and between 25 and 30 were classified as overweight. Type 2 diabetes mellitus was defined as fasting plasma glucose ≥126 mg/dl or else a prior diagnosis of type 2 diabetes (29). Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic pressure ≥90 mmHg, or a prior diagnosis of hypertension (30).

Study populations

We analyzed childhood BMI dynamics using the NLSY Children and Young Adult samples. The samples contain information on the children of women in the original NLSY cohort, providing biennial data on BMI, age, sex, race/ethnicity (31), and sex as well as mothers' ages and BMIs (32–34). The weighted sample represents children born 1970–88 to women aged 21–31 in the U.S. in 1979. To enable assessment of the relationship between childhood BMI and adult obesity, our analysis requires children to have BMI measurements through age 18 (4,884 respondents) (Appendix Table A1).

We analyzed adult BMI dynamics along with the risks of obesity-related diseases through middle age using the PSID and NHANES. The PSID is a nationally-representative study of nearly 9,000 families that contains information on sex, race/ethnicity, BMI, and self-reported diagnoses of diabetes and hypertension. Most measures are available for 1986 and 1999–2009, with self-reported diagnoses available for 1999–2009. We constructed a weighted sample of 18–25 years-olds in 1986 who had follow-up interviews at ages 38–47 (999 respondents) (Appendix Table A2). To capture the relationship between obesity in middle age and physical and biochemical markers of chronic disease, we used the adult NHANES sample (2003–2008), imputing values from NHANES to middle age-adults in the PSID based on their age, sex, race, BMI, and self-reported diabetes and hypertension status, along with other covariates (Appendix B). NHANES contains cross-sectional, nationally-representative data for adults on sex, race/ethnicity, smoking status, anthropometric measures (BMI), systolic and diastolic blood pressure, fasting plasma glucose and glycosylated hemoglobin (HbA1c), and self-reported medication usage and year of diagnosis

for diabetes and hypertension. The weighted sample of adults aged 38–49 in 2003–2008 represents the non-institutionalized U.S. population (2,780 respondents) (Appendix Table A3).

Predictive utility of childhood obesity assessment

We analyzed the test sensitivity and specificity and positive predictive values of childhood obesity status. Test sensitivity is the probability of being overweight or obese at a given age in childhood (e.g., BMI ≥85th percentile at age 10) among those children who were obese at age 18. Test specificity is the probability of being a normal weight child at a given age (e.g., BMI <85th percentile at age 10) among those children who were normal weight at age 18. Lower test sensitivities suggest that a greater proportion of obese 18 year-olds are missed by childhood screening, whereas lower test specificities imply that a greater proportion of children are falsely labeled "at risk for adult obesity". We also used Receiver Operating Characteristic (ROC) curves to compare the trade-offs in sensitivity and specificity that accompany various age- and sex-specific BMI cutoffs based on the CDC's 2000 standardized growth charts. Positive predictive values represented the probability of being obese at age 18 among those children who were above the obesity threshold at a given age.

We similarly assessed the test sensitivity and specificity of using maternal BMI to predict the future adult obesity status of their children (e.g., the proportion of 10 year-olds who became obese at age 18 and whose mothers were obese when the children were 10). Likewise, we assessed the combination of childhood BMI with maternal BMI (i.e., the proportion of obese 18 year-olds who were overweight or obese at age 10 or who were normal weight but had mothers who were overweight or obese).

Future obesity and health

We developed a model to assess the significance of childhood obesity in predicting the risk of obesity at age 18 and the impact of obesity at age 18 on obesity, fasting plasma glucose, HbA1c, systolic and diastolic blood pressure, uncontrolled diabetes, and uncontrolled hypertension for adults in their early 40s. We named the model the Stanford Childhood Obesity Prediction and Evaluation (SCOPE) model. The model applies the developmental patterns of BMI results from the analysis of the NLSY to the age, sex, race/ethnicity, and BMI values representative of non-institutionalized U.S. children aged 2–5 in 2001–2008 based on children in NHANES (2,899 respondents) (Appendix Table A4). Specifically, it alters each child's BMI annually through age 18 based on BMI dynamics from the NLSY. Continued changes in BMI from ages 18 through an individual's early 40s are based on adults in the PSID, and physical and biochemical measures of chronic disease in the early 40s are based on middle-aged adults in NHANES. The SCOPE model is built with the following steps:

- Step 1: We sample with replacement from the age-specific BMI distributions of 2–5 year-olds in the NHANES child sample for subgroups defined in terms of sex and race, creating sex- and race-specific cohort of 10,000s of children.
- Step 2: We sample with replacement BMI trajectories for those age 2 to age 18 from the NLSY children for subgroups defined in terms of sex and race, creating sex- and race-specific cohorts of 10,000s of childhood BMI trajectories.
- Step 3: For each sampled child in NHANES (Step 1), we use statistical matching (described below) to attach an NLSY trajectory (Step 2) within our sex- and race-specific cohorts, creating predicted future BMI trajectories for current U.S. children.

- Step 4: We sample with replacement BMI trajectories for those age 18–25 to age 38–47 from the PSID for subgroups defined in terms of sex and race, creating sex- and race-specific cohorts of 10,000s of adult BMI trajectories.
- Step 5: We estimate cross-sectional quantile regressions relating age, sex, race, smoking status, and diagnosed diabetes and hypertension status to fasting plasma glucose and blood pressure values for middle age adults in the current NHANES sample.
- Step 6: We use the adult NHANES quantile regressions (Step 4) to impute fasting plasma glucose and blood pressure values in middle age (see below) for the sampled PSID adults in middle age (Step 5).
- Step 7: For subgroups defined in terms of sex and race of the NHANES-NLSY future childhood BMI trajectories for current U.S. children (Step 3), we use statistical matching (described below) to attach PSID adult BMI trajectories and their associated adult NHANES imputed middle-age biochemical values (Step 6), creating predicted future BMI trajectories and middle age chronic disease profiles for current U.S. children.

Statistical methods

All analyses were undertaken using Stata 11/SE (Stata Corp, College Station, Texas). All analyses adjusted for the sampling designs of the respective surveys (35).

To construct each individual's longitudinal BMI path from childhood through adulthood and their associated markers of chronic disease, we first used each dataset's sampling weights and bootstrapped from the datasets based on these weights to generating synthetic cohorts with 10,000s of observations for each dataset (i.e., NHANES children, NLSY children, PSID adults) (36, 37). We then applied statistical matching approaches to link the datasets together, finally imputing biochemical measures of chronic disease based on quantile regressions.

Statistical matching—Statistical matching using sex, age, race/ethnicity and BMI enables greater consistency for the predicted longitudinal patterns of BMI and risks across data sets (Appendix B) (38). Our matching procedure employed all variables that the datasets had in common and which were measured in the same way. Within subgroups defined by age, sex, and race, statistical matching was based on similar BMI values. For each observation (i) in the first dataset (e.g., NHANES child sample), we computed the probability (p_{ij}) of matching an observation (j) in the second dataset (e.g., NLSY child longitudinal dataset), where p_{ij} is defined based on overlap spline polynomial weights (w_{ij}) and $\varphi()$ is the standard normal cumulative density function (38):

$$w_{ij}=\Phi\left(\frac{BMI_j-BMI_i+b}{a}\right)-\Phi\left(\frac{BMI_j-BMI_i-b}{a}\right)$$

$$p_{ij}=\frac{w_{ij}}{\sum_j w_{ij}}$$

The parameters a and b define the normalized kernel used for statistical matching (i.e., how strongly differences in BMI values reduce the likelihood of being matched). For matching children in the NHANES and NLSY, we set b to 0.1 and a to 0.1 since the range of BMI values for children 2–5 is relatively small. For those age 18 in NLSY and PSID, we set b to 0.2 and a to 0.1 since the range of BMI values widens in adulthood. This implied that for a child with a give BMI, 65% of probability mass of matches fell within 0.1 BMI units, 93% within 0.2 BMI units, and 99% within 0.3 BMI units. For adults, these were 50%, 82%, and

96%, respectively. Because we use bootstrapped datasets with 10,000s of observations, statistical matching between them defines a distribution of future BMI paths for each individual in the first dataset based on individuals who are more similar to them in the second dataset being more likely to contribute to their future BMI paths to the distribution.

Imputing biochemical measures—We imputed fasting plasma glucose, glycosylated hemoglobin, systolic blood pressure, and diastolic blood pressure values for the middle aged adults in the PSID. To do so, using the NHANES adult sample (2003–2008), we estimated quantile regressions describing how the distribution of fasting plasma glucose, glycosylated hemoglobin, systolic blood pressure, and diastolic blood pressure depended on sex, race/ethnicity, age, BMI, smoking status, a previous diabetes diagnosis, the duration of diagnosed diabetes, the use of diabetes medications, diagnosed hypertension, and the use of hypertension medications (Appendix B) (39). For each individual in the bootstrapped PSID dataset and each biochemical value we wished to impute, we drew a uniform random number between 1 and 99 to determine which quantile the individual fell within, we then computed the predicted value based on the quantile drawn and the individual's observed characteristics (i.e., the covariates in the regression). Thus, individuals who were, for example white, non-smoking men age 40 with BMI 30 who had no history of diagnosed diabetes or hypertension could have different values for their fasting plasma glucose consistent with the distribution of these values for similar individuals in NHANES.

Face validation and sensitivity analyses—We compared results of both our analyses within a single dataset (e.g., NLSY) and between multiple datasets (e.g., the SCOPE model) to similar analyses we undertook using The National Longitudinal Study of Adolescent Health (i.e., Add Health), an independent dataset that has information on anthropometry from teen years through individuals in their early 30s. We assessed the test sensitivity and specificity estimates from NLSY which included some self-reported BMI values by restricting the NLSY sample to those with objective measures. We also compared the estimated test characteristics from NLSY to those we estimated in the Add Health study dataset. Finally, we compared the relationship between an individual's BMI as a teenager to BMI in the mid-20s in the NLSY-PSID statistically matched dataset created for the SCOPE model and those observed in the Add Health study dataset. To assess the sensitivity of the SCOPE model's predictions to the statistical matching procedures used, we repeated the main SCOPE analysis by predicting obesity and chronic disease outcomes at age 18 and in middle age using statistical matching that required greater concordance of BMI for individuals between datasets with the same age, sex, and race-specific subgroups (children: a=0.05, b=0.05; adults; a=0.1, b=0.1). Statistical matching with these parameters greatly increased the likelihood of matching a BMI value ±0.2 kg/m^2 but also increased the variance of estimates because it reduced the number of potential individuals to match.

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Results

Childhood and adult obesity dynamics

There is considerable age-dependent variability in the association between childhood and adult obesity in the NLSY data. Approximately 12% of 18 year-olds had BMIs ≥30. The prevalence of children with BMI ≥95^{th} standardized CDC percentile is highest in 2 year-olds (20–24%), dropping by age 5 to 12–13% (Table 1). Overweight and obesity generally increases through teenage years. While 22% (21% for boys and 23% for girls) of 5 year-old

children had a BMI ≥85th percentile, they comprised only 50% (48% for boys and 52% for girls) of individuals who were obese at age 18 (Table 2). Consequently, half of obese 18 year-olds would not have been detected by an obesity assessment at age 5. More than 70% of children with BMI ≥85th percentile at age 5 became non-obese at age 18.

Regardless of BMI percentile used as a threshold to identify children "at risk of adult obesity", screening later in childhood has more favorable combinations of sensitivity and specificity than earlier in childhood (Figure 1). At the standard 85th percentile cutoff, childhood BMI, particularly for children under 10, lacks the sensitivity and specificity that enables accurate prediction of obesity status at age 18. Although sensitivity improves for children above age 10 (Table 2, Figure 2, **Panels A and B**), moderate specificity and 12% obesity prevalence in 18 year-olds imply that the positive predictive value for obesity at age 18 does not exceed 40% at any age (Figure 2, **Panels C and D**).

Including maternal obesity in childhood obesity assessment improves the detection of future obese 18 year-olds (Figure 2, **Panels A and B**), especially in children below age 8. Yet, due to high false positive rates (i.e., many non-obese children whose mothers' BMIs are high go on to be non-obese 18 year-olds), the clinical utility of using maternal obesity in conjunction with childhood obesity assessment is weakened (Figure 2, **Panels C and D**). Using the combined criteria of either maternal BMI ≥30 or child BMI ≥85th percentile at ages 5–7, approximately 75% of obese 18 year-olds would have been detected. However, nearly 30% of all 5–7 year-olds who became non-obese 18 year-olds would have been incorrectly labeled as "at risk for adult obesity." The positive predictive value of combined child and mother obesity assessment does not exceed 25% at any age (Figure 2, **Panels C and D**). Using a detection criterion of both the child and mother being above the thresholds improves the positive predictive value amongst those testing positive but reduces the absolute proportion of obese 18 year-olds actually detected by childhood obesity assessment by 40–55% (data not shown).

When stratifying by race/ethnicity, both childhood obesity and maternal obesity generally have higher sensitivities and lower specificities among black and Hispanic children in the NLSY than among white children. For example, being above the CDC's 85th BMI percentile at age 10 has a sensitivity and specificity for detecting obesity at age 18 of 81% and 79% for white children and 88% and 74% for black and Hispanic children (See Appendix Tables C2 and C311 for detailed results). Given the somewhat higher prevalence of obesity at age 18 among blacks and Hispanics (approximately 16%), the clinical utility of childhood obesity assessment for identifying individuals who will go on to become obese adults is comparable for blacks and Hispanics and for whites.

The analysis of adult obesity dynamics using the PSID suggests that being overweight or obese at age 18 increases the risk of being obese in one's early 40s (Odds Ratios for BMI adjusted for smoking status and race/ethnicity: males: 1.3 [1.2–1.5]; females: 1.6 [1.4–1.8]). Furthermore, overweight and obesity status in one's early 40s predicts higher fasting plasma glucose levels and HbA1c levels as well as higher systolic and diastolic blood pressure in the NHANES adult data. For example, the interquartile range of fasting plasma glucose for 45 year-old white male, non-smokers without diabetes whose BMI was 20 was predicted to be 93–102 mg/dl whereas, for a similar men with BMI 35, it was 101–111 mg/dl. The interquartile range for systolic blood pressure for these two groups was predicted to be 108–121 mmHg and 114–128 mmHg, respectively. (Appendix Tables B1, B2, B3, and B4).

Childhood obesity and links to adult obesity and health

Based on the examined datasets, we project that without intervention, by the time U.S. children currently below age 5 reach their early 40s, substantial numbers will be overweight

and obese, diabetic and hypertensive, especially among black individuals. Specifically, 42% of whites and 52% of blacks will have BMIs ≥30. An additional 35% of white and 34% of blacks will have BMIs between 25 and 30. In this age group, uncontrolled diabetes prevalence is projected to be 5% among whites and 10% among blacks. Uncontrolled hypertension prevalence is projected to be 49% for whites and 57% for blacks.

However, while the prevalence of these conditions is substantial, the relationship between childhood and adult obesity patterns is complex. The ability to predict which children will ultimately become obese adults and which children will have indicators of chronic disease that will worsen in adulthood appears limited for children below age 10 (Table 3 **and** Appendix Table C4). For example, in Table 3, 39.8% of white boys above the 85th percentile at age 15 became obese in their mid-40s whereas 1.4% of those below the 85th percentile became obese in their mid-40s (RR=28.4). In comparison, for 5 year-old white males, 21.7% of those above the 85th percentile became obese in their mid-40s whereas 9.8% below the threshold became obese (RR=2.2). Similarly, 12.6% of those above the 85th percentile at age 15 developed uncontrolled diabetes by their mid-40s whereas 2.8% below the threshold developed diabetes (RR=4.5). For those above and below the threshold at age 5, 7.9% and 4.9% developed uncontrolled diabetes by their mid-40s, respectively (RR=1.6).

Most children have their obesity status assessed at multiple points of time during childhood. Children who remain obese throughout childhood are at the highest risk of continued adult obesity and future chronic disease risk. As current policies seek to initiate childhood obesity assessment at earlier ages, we assessed the marginal increase in predictive utility of beginning assessments in early childhood, finding that additional predictive power is low (Table 4). Even considering obesity assessment conducted at only 3 points during childhood (at ages 5, 10, and 15), relatively few current U.S. children who go on to become obese in their early 40s have BMIs that are above the 85th percentile at age 5 but not above the 85th percentile at age 10 or 15 (white males and females: 12% and 15%; black males and females: 13% and 9%). As obesity assessment is currently often performed annually or biennially, the actual proportions of children who would only test positive prior to age 10 and not afterwards (i.e., only be detected by obesity assessment at an age prior to 10) is considerably smaller (data not shown).

Regardless of the chosen approach to childhood obesity, complementary strategies that focus on obesity in early and middle adulthood appear warranted as well, given that many individuals who are normal weight throughout childhood become obese in adulthood. Of current U.S. children whose BMI is below the 85th percentile at age 15, we project that few will be obese at age 18 (between 1.2% and 3.1% depending on sex and race/ethnicity). However, even for this group, by their early 40s, between 28% and 46% are projected to have BMI ≥30, between 2.6% and 7.3% to have uncontrolled diabetes, and 31% and 69% to have uncontrolled hypertension (Table 3).

Discussion

Our findings suggest that developmental patterns of childhood obesity are dynamic and that effective assessment and intervention strategies are likely to be complex. Most obese adults were normal weight children, and many obese children become non-obese adults, consistent with prior studies. (1, 9–13). Accordingly, the predictive utility of BMI assessment in young childhood is relatively poor, even when maternal obesity status is also considered. However, BMI becomes increasingly stable in pre-teen and teen years, so using childhood BMI as an indicator of future obesity risk and as a target for screening programs may prove more useful in older children.

Calls for BMI-based assessment to target interventions in early childhood may need reconsideration in light of these findings (8, 40). Early childhood obesity strategies would be justified if they could accurately identify substantial numbers of children at-risk for adult obesity and chronic diseases who would not otherwise have been identified later in life or if interventions delivered in early childhood were substantially more effective than those delivered later in life. For example, the emphasis on early versus later childhood and adolescence may depend in part on what ages relevant health behaviors begin to become more firmly entrenched, with counseling and focus on healthy lifestyles on the part of physicians and public health professionals beginning prior to this.

The findings of this study may also suggest that a highly targeted approach to early childhood obesity interventions may prove less useful than more universal strategies, such as the “Let’s Move” campaign advocated by First Lady, Michele Obama (41–43). In either case, interventions that target obesity in adults are necessary complements to childhood obesity policies given the elevated risk present in even normal weight 18 year-olds (44). Weighing the value of childhood obesity assessment relative to more universal strategies depends on the relative costs and benefits of failing to intervene with a child who would otherwise become obese as an adult and intervening with children who would not have become obese even without intervention. The appropriate scope and mix of preventive and therapeutic strategies for childhood obesity will require continued research, particularly as new, more effective interventions are developed both in the United States and other countries facing a high prevalence of childhood obesity (45).

This study has several limitations.

The SCOPE model projects future patterns of BMI change based on past trends derived from particular data sources (the NLSY and PSID). Because of how the sample for the NLSY is constructed (i.e., requiring observation on children through age 18 by a particular calendar year based upon a cohort defined by women who were in a particular age range in 1979), we consider a sample of children that over-represents women who had their first child at a younger age (mean age at first birth: 21.8 years [Interquartile Range: 19–25 years]) compared to mothers in the whole data set (mean age at first birth: 23.3 years [IQR: 19–27 years]). To the extent that earlier maternal age in the sample predicts differential BMI and growth patterns of children, this could, in principle, bias results. Similarly, younger maternal age could affect the observed BMI values of mothers and their predictive value as well. Additionally, though substantially less common and less effective in the past, it is possible that these sources may contain some individuals who received obesity interventions. Changes in a range of determinants of obesity trends would alter model projections. Historical increases in childhood obesity prevalence have slowed, though extreme childhood obesity may be growing (3, 46). Our findings suggest that consistent obesity throughout childhood, which likely implies BMI well-above the obesity threshold, is highly predictive of adult obesity (Table 4). However, unless childhood obesity becomes substantially less dynamic for a large percentage of children, the predictive utility of BMI assessment in early childhood will likely remain relatively low. While adult BMIs and chronic disease burdens are alarmingly high, these trends also appear to be flattening (2).

Another concern is that the weight and height data in the PSID and for older children in the NLSY are self-reported and subject to potential reporting biases (47, 48). However, when we restricted our analyses to only those children whose height and weight had been directly measured, the general patterns of sensitivity and specificity that were found in the full sample remained (Appendix Table C5). We also compared our BMI distributions in the late teens and early 20s along with test characteristics of BMI measurements in earlier teen years to identify individuals who would become overweight or obese in their late teens and early

20s to those measured in the National Longitudinal Study of Adolescent Health (Add Health), a more recent study of individual BMI patterns over time, finding reasonable similarity and no strong evidence of cohort effects (Appendix Tables C6, C7, and C8).

The use of different datasets to create predictive models could introduce inaccuracies in risk assessment because of cohort effects or because complex relationships may not be captured via statistical matching. We assessed the sensitivity of our projections to the particular datasets used and to the particular parameters of statistical matching, finding that they were largely consistent (Appendix Tables C7, C8, and C9). Of some additional reassurance, a recently published article examining the longitudinal disease risks of over 30,000 Israeli males from later teenage years through age 45 finds similar teen BMI-related gradients of middle age BMI, blood pressure, fasting plasma glucose, and, importantly, elevated relative risks of type 2 diabetes to those we report here (49). Though chronic diseases impact adult health into old age, our projections end in individuals' early 40s due to a lack of longitudinal data from the PSID or other studies for the U.S. that link BMI to chronic diseases in old age.

Due to sample size limitations, our analyses could not assess the relationship between childhood and adult obesity for Hispanic and other minority populations. Patterns of obesity and chronic diseases are known to differ by race and ethnicity, by urban and rural location, and by socioeconomic status (31, 50, 51). Therefore, applying the model's projections to unexamined minority or other subgroups may not be appropriate.

Addressing obesity in U.S. children and adults is a public health priority. However, the findings of this study underscore the dynamic relationship between early childhood growth patterns and the prevalence of adult obesity and related health conditions. This complex relationship suggests that any targeted screening strategies in childhood may also need to be coupled with more universal risk reduction approaches designed to effectively reduce risk among all children in the United States. Such comprehensive strategies would respond to the dynamic character of weight gain during childhood and help to ensure that continued improvements in both preventive and therapeutic interventions are effectively implemented over the life-course.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

BP	Blood Pressure
BMI	Body Mass Index
FPG	Fasting Plasma Glucose
HbA1c	Glycosylated Hemoglobin

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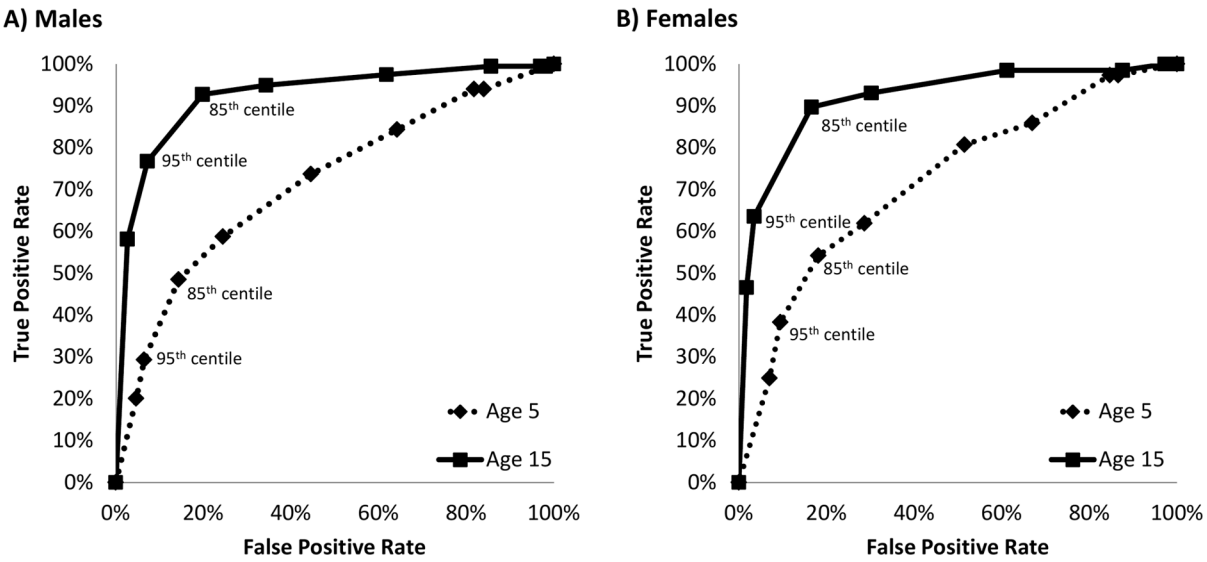

Figure 1. Receiver Operating Characteristic curves for detecting obesity at age 18 at various age- and sex-specific BMI percentile thresholds

Panel A (males) and Panel B (females) show Receiver Operating Characteristic (ROC) curves comparing the true positive rate (sensitivity) and false positive rate (1 - specificity) for various cutoffs based on CDC standardized sex-specific BMI percentiles at ages 5 and 15 years. Thresholds used to generate the ROC curves include the 3rd, 5th, 25th, 50th, 75th, 85th, 95th, and 97th percentiles.

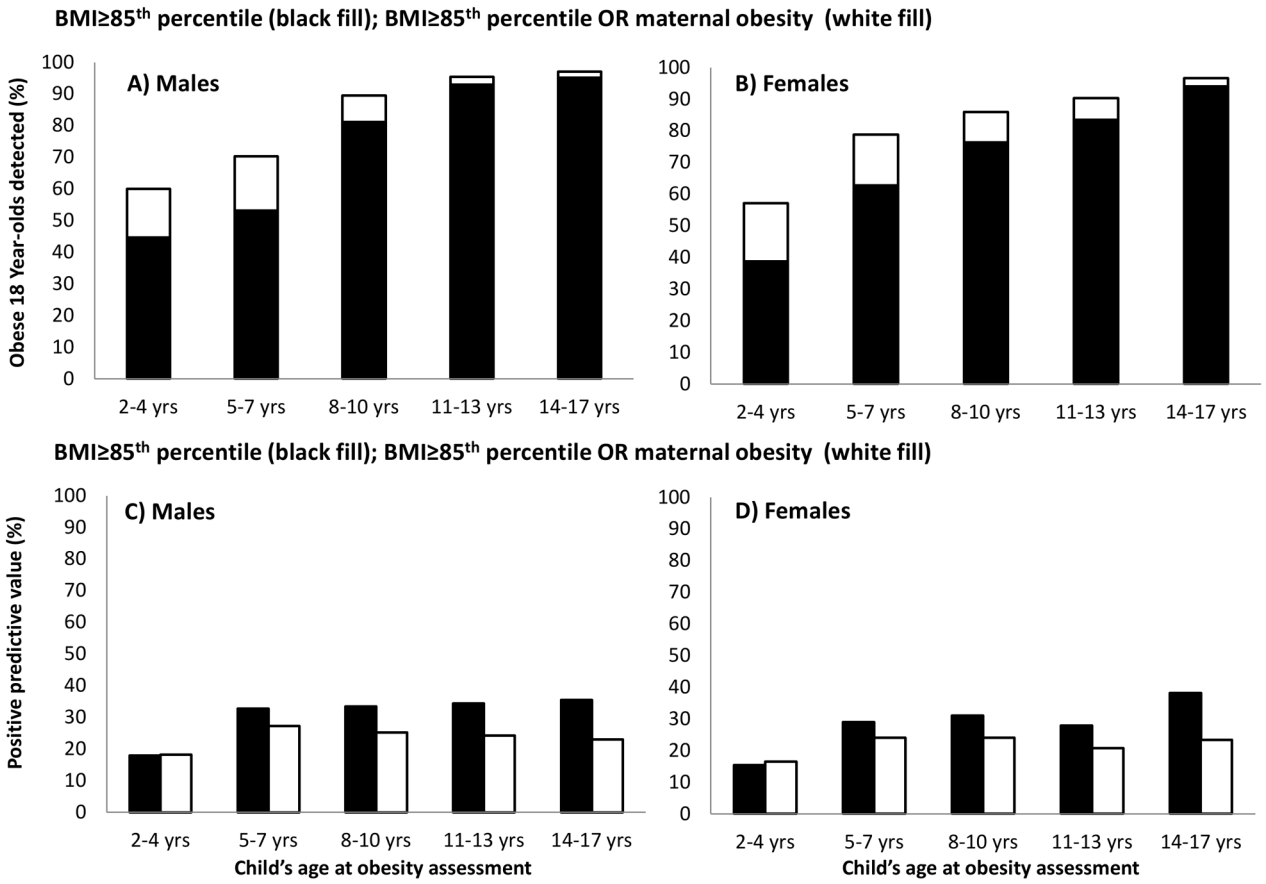

Figure 2. The ability of child and maternal obesity assessment to discriminate future obese 18 year-olds from those who will not become obese

Panels A and B show the proportion of obese 18 year-old males and females, respectively, whose BMIs were above the 85th percentile (black filled bars) at ages 2–17 years and whose BMIs were above the 85th percentile or had mothers who were overweight or obese (white filled bars). Panels C and D show the proportion of males and females, respectively, testing positive on childhood obesity screening at ages 2–17 who become obese 18 year-olds (black filled bars: BMI above the 85th percentile; white filled bars: BMI above the 85th percentile or had a mother who was overweight or obese). (Note that for comparability, all panels in Figure 2 include only the subsample of children who also had maternal BMI measurements).

Table 1

Prevalence of overweight and obesity by age and sex for children and young adults in the NLSY

	Boys			Girls		
Age	**N**	**Overweight (85–95th CDC percentile)**	**Obese (≥95 CDC percentile)**	**N**	**Overweight (85–95th CDC percentile)**	**Obese (≥95 CDC percentile)**
2	663	10%	24%	675	9%	20%
5	838	9%	12%	766	10%	13%
10	1,105	15%	15%	1,128	18%	14%
15	1,066	14%	17%	964	15%	11%

Table 2

Test characteristics for child obesity assessment in the NLSY for identifying obese 18 year-olds*

		Boys							Girls						
					Identifying those with BMI≥30 at 18							Identifying those with BMI≥30 at 18			
Test	**Child Age**	**N**	**Test + %**	**95% CI**	**Sens**	**95% CI**	**Spec**	**95% CI**	**N**	**Test + %**	**95% CI**	**Sens**	**95% CI**	**Spec**	**95% CI**
Child BMI>85th centile	2	663	34	[31–38]	51	[40–62]	68	[64–72]	675	31	[28–35]	44	[33–55]	70	[66–74]
	5	838	21	[19–24]	48	[40–57]	83	[81–86]	766	23	[20–26]	52	[41–62]	81	[77–83]
	10	1,105	30	[27–33]	88	[80–93]	77	[74–79]	1,128	32	[29–34]	84	[76–89]	75	[73–78]
	15	1,066	31	[29–34]	91	[85–95]	78	[75–81]	964	26	[23–29]	92	[85–96]	82	[79–84]
Child BMI>95th centile	2	663	24	[20–27]	37	[27–49]	78	[75–81]	675	20	[18–24]	31	[22–42]	81	[78–84]
	5	838	12	[10–14]	33	[26–42]	92	[90–94]	766	13	[11–16]	38	[28–48]	90	[87–92]
	10	1,105	15	[13–17]	64	[55–72]	90	[88–92]	1,128	14	[12–16]	57	[48–65]	92	[90–93]
	15	1,066	17	[14–19]	74	[66–80]	93	[91–94]	964	11	[10–14]	67	[57–75]	95	[94–96]
Mother BMI≥25	2	695	40	[36–43]	68	[57–77]	64	[60–68]	691	40	[36–43]	69	[58–78]	64	[60–68]
	5	828	45	[42–49]	69	[61–77]	59	[55–62]	756	44	[41–48]	65	[54–75]	58	[54–62]
	10	1,105	49	[46–52]	70	[61–78]	53	[50–56]	1,105	51	[48–54]	77	[69–83]	52	[49–55]
	15	1,010	61	[58–64]	76	[68–82]	42	[38–45]	921	58	[55–61]	76	[67–84]	44	[41–47]
Mother BMI≥30	2	695	15	[12–18]	31	[21–42]	87	[84–90]	691	14	[11–17]	38	[28–49]	90	[87–92]
	5	828	18	[15–21]	36	[28–45]	85	[82–88]	756	19	[16–22]	45	[35–56]	84	[81–87]
	10	1,105	21	[19–24]	43	[34–52]	81	[79–84]	1,105	24	[22–27]	52	[43–60]	80	[77–82]
	15	1,010	30	[27–33]	56	[47–64]	74	[71–77]	921	33	[30–36]	59	[49–69]	70	[67–73]

* CI = binomial 95% confidence interval calculated using the Agresti-Coull method; See Appendix Table C1 for similar test positivity and sensitivity and specificity results for identifying overweight (BMI≥25) 18 year-olds as well as related results stratified by race/ethnicity (Appendix Tables C2 and C3). Test + = Test positive; Sens = sensitivity; Spec = specificity.

Table 3

Adult outcomes predicted for US children aged 2–5 stratified by race/ethnicity, sex, and childhood BMI assessment (below/above CDC 85th percentile)*

		White males				White females			
		Age 5		Age 15		Age 5		Age 15	
Age outcome measured	**Measure**	**Below**	**Above**	**Below**	**Above**	**Below**	**Above**	**Below**	**Above**
18	Mean BMI	23.6	26.2	22.0	29.5	22.3	25.0	21.5	28.8
18	BMI ≥30 (%)	9.8	21.7	1.4	39.8	4.8	14.7	1.2	33.2
Early 40s	BMI ≥30 (%)	33.4	44.6	27.5	56.6	45.6	65.7	41.2	89.4
Early 40s	BMI ≥25 (%)	84.2	91.6	80.7	98.4	66.5	81.5	63.6	97.2
Early 40s	Diabetes (%)	4.9	7.9	2.8	12.6	4.1	8.7	2.6	16.4
Early 40s	Mean FPG (mg/dl)	106	109	104	113	101	106	100	113
Early 40s	Mean HbA1c (%)	5.5	5.6	5.4	5.7	5.4	5.6	5.4	5.9
Early 40s	Mean Diabetes duration (yrs)	0.3	0.5	0.1	0.8	0.2	0.5	0.1	1.0
Early 40s	Hypertension (BP) (%)	57.6	59.5	56.8	61.2	39.8	44.1	38.9	49.3
Early 40s	Mean Systolic BP (mmHg)	122	122	122	123	116	118	116	119
Early 40s	Mean Diastolic BP (mmHg)	91	92	91	92	88	89	88	89

		Black males				Black females			
		Age 5		Age 15		Age 5		Age 15	
Age outcome measured	**Measure**	**Below**	**Above**	**Below**	**Above**	**Below**	**Above**	**Below**	**Above**
18	Mean BMI	24.3	27.0	22.3	29.8	24.4	27.7	22.4	30.2
18	BMI ≥30 (%)	12.1	26.5	1.5	41.7	15.0	32.7	3.1	47.7
Early 40s	BMI ≥30 (%)	42.5	51.8	37.0	59.2	54.4	68.2	46.0	78.3
Early 40s	BMI ≥25 (%)	83.3	89.5	79.9	94.0	83.4	90.6	78.7	96.6
Early 40s	Diabetes (%)	6.6	9.2	4.9	11.5	10.5	15.4	7.3	19.2
Early 40s	Mean FPG (mg/dl)	109	111	107	113	109	114	106	118
Early 40s	Mean HbA1c (%)	5.6	5.7	5.6	5.8	5.8	5.9	5.7	6.1
Early 40s	Mean Diabetes duration (yrs)	0.1	0.2	0.0	0.3	0.6	1.1	0.2	1.6
Early 40s	Hypertension (BP) (%)	63.1	64.3	61.4	66.9	49.9	54.3	47.1	57.7
Early 40s	Mean Systolic BP (mmHg)	125	126	124	126	120	122	119	124
Early 40s	Mean Diastolic BP (mmHg)	93	93	92	93	89	91	89	91

* Similar results are shown using the CDC's age- and sex-specific 95 percentile cutoff (Appendix Table C4). FPG = fasting plasma glucose; HbA1c = glycosylated hemoglobin; BP = blood pressure. Middle age outcomes other than BMI were based on biological markers of chronic disease (FPG, HbA1c, Systolic BP, and Diastolic BP) and their associated cut-offs which were imputed from the current adult NHANES sample based on PSID measures including age, sex, race, BMI, smoking status, self-reported diabetes, self-reported hypertension, and duration of these self-reported conditions. Though more individuals have hypertension than have diabetes, the two outcomes are correlated, with diabetic individuals having a relative risk of hypertension of 1.1–1.3 compared to non-diabetic individuals depending on sex and race.

Table 4

The relationship of age-specific patterns of childhood obesity and adult obesity risks, stratified by sex and race/ethnicity

Childhood obesity patterns (≥85th CDC percentile)			Black				White			
			Females		Males		Females		Males	
at age 5	at age 10	at age 15	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)
No	No	No	41.6	42.2	42.9	35.6	61.6	36.1	53.7	23.8
Yes	No	No	7.6	47.7	9.9	38.9	8.8	46.2	8.7	30.5
No	Yes	No	8.2	56.6	7.4	41.0	8.4	61.1	6.9	45.0
Yes	Yes	No	4.6	59.6	3.5	40.6	4.0	66.9	2.7	47.8
No	No	Yes	7.9	73.6	6.5	47.2	4.1	86.3	6.5	57.4
Yes	No	Yes	2.9	73.8	2.9	53.6	1.2	84.2	2.0	56.2
No	Yes	Yes	13.8	78.9	15.5	60.2	6.5	90.3	12.6	55.5
Yes	Yes	Yes	13.4	81.6	11.4	65.9	5.5	91.8	7.0	57.8

Childhood obesity patterns (≥95th CDC percentile)			Black				White			
			Females		Males		Females		Males	
at age 5	at age 10	at age 15	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)	% with obesity pattern	Of these, obese in early 40s (%)
No	No	No	61.8	48.9	61.9	37.6	81.6	43.4	72.2	29.6
Yes	No	No	7.8	58.3	8.3	41.1	6.0	57.7	6.7	37.9
No	Yes	No	8.6	68.0	8.3	46.0	5.3	77.9	4.5	55.0
Yes	Yes	No	3.6	70.8	2.3	49.9	1.9	82.7	1.1	57.2
No	No	Yes	4.9	80.5	4.4	57.6	1.7	93.9	5.5	57.9
Yes	No	Yes	1.4	80.7	1.2	67.0	0.4	97.4	1.1	56.3
No	Yes	Yes	6.8	84.7	9.2	72.5	2.0	95.1	6.6	54.4
Yes	Yes	Yes	5.1	85.1	4.4	77	1.2	95.0	2.4	55.7