

**Parent's weight status and history of type 2 diabetes
and growth trajectories from childhood to young adulthood**

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ABSTRACT (MAX 150 words)

Type 2 diabetes and obesity are two major health problems in the United States. Evidence suggests that the pathophysiological processes leading to both conditions start in childhood. This study examined the role of parents' metabolic health in the emergence of their children's metabolic health. Specifically, we used data independently collected from two generations through the Panel Study of Income Dynamics (PSID) to delineate growth trajectories from childhood to young adulthood for individuals whose parents did and did not have overweight, obesity or type 2 diabetes. The national PSID cohort has almost two decades of data (from 1999-2015) on weight, height and type 2 diabetes in children and parents. Only children with parents in the normal weight category were in average not overweight or obese at age 24y; children whose parents had obesity or type 2 diabetes were in the overweight category through their childhood, adolescence and young adulthood.

Introduction

Type 2 diabetes and adult obesity are two major health problems in the United States [1]. Evidence suggest that the pathophysiological processes leading to both conditions start already in childhood. Weight status in childhood and adolescence are linked to the risk of obesity in adulthood [2-5] and to the risk and timing of onset of type 2 diabetes [6]. Even among children in the normal weight category, those who became obese as adults had a higher body mass index (BMI) at age 6 years than the children who did not become obese adults [2]. In the U.S., the prevalence of overweight and obesity among American children increased almost 50% between ages 6 and 14 [7]. In contrast, the reversal of obesity in adolescence and young adulthood is difficult, as less than 2% of obese American adolescents were not obese as young adults [8]. These patterns may have major implications for the future burden and prevention of adult obesity and type 2 diabetes in the United States (U.S.) and raise the question of who the children are, who are normal weight in childhood, but likely to progress to overweight or obesity in adolescence or as adults with an increased risk of type 2 diabetes later in life.

Parents' history of obesity and type 2 diabetes is important for the risk of obesity [4] and T2D in their adult children [9] and may also influence their BMI trajectories from childhood to adulthood. In the U.S., among children who were normal weight up to age 17, those who had obese parents had a higher risk of adult obesity than children with non-obese parents [4]. In Taiwan, the odds of children becoming overweight or obese between age 6 and 11 increased with a with 6% for every unit increase in their parents' BMI [10]. In the U.S., children whose parents had type 2 diabetes had higher BMI from early childhood and into adolescence compared to children whose parents did not have type 2 diabetes [11, 12].

However, the generalizability, extent and applicability of these findings remains unclear. Specifically, previous studies were limited to specific populations such as the Pima Indians [12] or sub-national data [4, 11]; they used data from before 1994 [4, 11, 12], and childhood overweight and obesity in the U.S. has doubled since [13], and the same have adult overweight and obesity [14, 15] and type 2 diabetes [16]. Finally, links between parents' weight status and growth trajectories from childhood to young adulthood have to our knowledge not been assessed.

In terms of risk of obesity, children and their parents share genetic risk [17, 18], and to some extent risk factors related to behaviors [19-21]. Parents' weight and type 2 diabetes status may be indicators for BMI development in children – even among those in the normal weight category in childhood. Therefore, the aim of this study is to examine the relationship between parents' weight and type 2 diabetes status and their children's BMI trajectories from childhood to young adulthood and whether these relationships are modified by race and socio-economic status. We use a longitudinal national representative dataset from the United States, the Panel Study of Income Dynamics (PSID), with nine waves of information on measured or self-

reported weight, height, and diabetes in multiple family members between 1999 and 2015, allowing us to link parents' and children's' health over almost 2 decades of life.

Method

Data sources and population

We used prospective cohort data from PSID. The PSID began in 1968 with a U.S. nationally representative sample of 4,802 households containing more than 18,000 individuals. Annually (biannually since 1997), information about these individuals and their descendants have been collected. In 1999, PSID began to also collect information about health conditions, including diabetes and weight and height in the head and wife of the households. In 1997 (data only available from 2002 and onwards) the 'Child Development Supplement' (CDS) was initiated and included children under 13 years of age living in a PSID household unit in 2013. In 2005, the 'Transition into Adulthood Supplement' (TAS), following children from the CDS cohort from age 18 and into young adulthood was fielded. Both the CDS and TAS have information on body weight and height.

We used the PSID's the 2015 Individual Data File and 1999-2015 Family files, 2002, 2007, and 2014 CDS waves, 2005, 2007, 2009, 2011, 2013 and 2015 TAS waves, and 2015 Parent Identification file. From the Individual Data File, we drew information on sex, age (month and year of birth), family role (i.e. head, wife, child etc.) and household ID between 1999-2015. From the Family files, information on diabetes and date of diagnosis in the household head, wife, 'wife' or co-habiting woman was extracted, as well as socio-economic characteristics and date of interview. From CDS and TAS information on ethnicity, anthropometric measures and the dates measurements were taken, were extracted. All data were merged into the Individual Data file.

Using the parent's ID from the PSID Parent Identification file, we assigned diabetes and weight and height measures to all individuals.

There were 3,028 individuals with at least two valid values of BMI. Among them, we excluded eight with BMI values >5 standard deviations (SD) lower/higher than their age-sex z-score mean and 537 who were already above age 18 at baseline. Further, we excluded 107 children with no information about diabetes or BMI for either parent. This analytic cohort was 2,460 children/adolescents. Of these, 686 had information on only one parent (kept in the cohort).

Variables

In CDS, body height and weight were measured by trained field workers or reported by respondents based on measures conducted by the family physician. In TAS, body height and weight were self-reported. We created two BMI variables: 1) continuous BMI; and 2) BMI z-score variables. Continuous BMI was calculated body mass index (BMI) as body weight (kg)/body height-squared (m²). Given that height and weight change during growth and development, as does their relation to body fatness, the BMI z-score

variable was created based on sex and age (at the date of measurement) for all ages according to WHO Child Growth Standards using age/month z-score [22]. The WHO Child Growth Reference stops at age 19 years, considering an individual fully developed in terms of height at this age. Therefore, any body weight gain after this point will result in a higher z-score. In our analyses, the z-score for a child/adolescent after age 19 was calculated using the WHO reference for 19-year-old. However, the BMI z-score for children/adolescents older than 19 years will only be displayed graphically to confirm the continuity of BMI trajectories.

For the parents' type 2 diabetes status and BMI, we used the PSID Family File, which has included since 1999 included questions about diabetes, time since diagnosis, asked of the head of household about him/herself and the wife, 'wife' or co-habiting woman. We defined a person as having type 2 diabetes if she/he answered yes to the question 'Has a doctor told you that you have high blood glucose or diabetes' and the diagnosis had occurred after the person turned 30 years. Individuals with a diabetes onset before the age of 30 were coded as not having type 2 diabetes because we expected diabetes with an onset before this age to be type 1 diabetes or another diabetes sub-type. The development of type 1 diabetes and most other subtypes of diabetes is not associated with increasing BMI and we did not expect it to be related to weight in the child/adolescent. We defined a child as having parents with type 2 diabetes if at least one parent reported type 2 diabetes between 1999–2015. Due to the slow onset and progression of type 2 diabetes and the fact that the disease was self-reported every second year, we did not consider time of diagnosis.

We used WHO cut-off for adult BMI [23] to classify the parents as normal weight ($BMI < 25$), overweight ($BMI > 25$) or obese ($BMI > 30$) in the calendar year the child first entered CDS. Next, we created a categorical variable with 3 levels: 1) no parents overweight or obese; 2) at least one parent overweight; or 3) at least one parent obese. For instance, if a child had one overweight and one obese parent, the child would be categorized as having a parent in category 3 (obese category).

Main control variables were poverty and race, as previous studies have shown that people living in poverty in high-income countries and non-whites are more likely to have type 2 diabetes [24, 25] and be obese. We generated a binary poverty variable based on household income, size of family and number of children under 18 years; poverty was defined according to the poverty threshold from the US Census Bureau [26]. In PSID ethnicity/race was self-reported, and most of the children/adolescents were white (52%) or African-American (42%). Therefore, we created a binary variable with white as the reference group. Descriptive statistics included other characteristics expected, based on the literature, to be associated with patterns of weight and type 2 diabetes in children and their parents: sex and birthweight of the child and parents' age and sex (all at baseline). Child's age was the time scale and not included as a variable in the models.

Statistical Analyses

Baseline was defined as the calendar year a child or adolescent would enter CDS or TA. Time of first entry varied from 2002 to 2014. We examined baseline characteristics of the study population in medians (P25, P75) for continuous variables and percentages for categorical variables. We fitted children's BMI and BMI z-score trajectories using mixed-effects models. Random intercept and slope were included to account for the within-person correlation arising from the longitudinal nature (repeated measurements) of the dataset. The main advantage of this method is that it neither requires that all individuals have the same number of measurements nor that they are captured at the same time points. We used age as the time scale and modeled change with linear, quadratic and cubic terms to account for non-linear changes in BMI. Exposure to parents' T2D (no/yes) and weight status (normal weight/overweight/obese) was investigated in separate models.

To investigate the potential modifying effect of poverty and race, we included interaction terms between each of these and parents' type 2 diabetes status. Interactions for all combinations of age, parental T2D and race or poverty were included. Due to the large number of potential interactions, it was only feasible to keep a linear time term with sufficient power. Therefore, these analyses were restricted to the BMI z-score as outcome and included z-score measurements only up to age 19.

Results

Study population

Children had BMI measurements at between 2 (10.5%) and 7 (10.8%) timepoints, with 5 as the median, covering the age range from 5 to 31 years.

Baseline characteristics are summarized in Table 1. Children were on average 12 years old at year of entry and half were girls; 52% were white race. Overall, 35% of the children came from household with an income below the poverty threshold, 20% had a least one parent with type 2 diabetes and 80% had a least one parent being overweight or obese. Children whose parents did and did not have type 2 diabetes were similar in terms of birthweight and poverty level, but children with a parent with type 2 diabetes were older, had older parents with higher BMI and were more likely to be non-white ($p < 0.05$ for all tests).

TABLE 1 HERE

Children with overweight or obese parents had a higher birthweight than children with normal-weight parents. Children with obese parents were non-white more often than children with a normal weight or overweight parent ($p < 0.05$ for all tests). Both children with normal weight or obese parent were more likely to live in households with an income below the poverty level than children with an overweight parent. Then

percentage of children having a parent with type 2 diabetes increase from 8% among children with only normal weight parents to 16% among children with at least one overweight parent to 32% in children with one obese parent ($p < 0.001$).

BMI trajectories

Parent's type 2 diabetes status

Estimated mean BMI and BMI z-score trajectories by parents' type 2 diabetes status are displayed in Figure 1A and 1B, respectively. Children with at least one parent with type 2 diabetes had on average a 0.74 kg/m² (0.03-1.45) higher BMI than those with parents not reporting type 2 diabetes. This difference increased even further during childhood, reaching 1.70 kg/m² (1.21-2.19) at age 15 and then plateauing. At age 8, the annual increase (slope) was 1.12 kg/m² (1.05-1.20) among children of parents without type 2 diabetes and 1.39 kg/m² (1.23-1.56) among those whose parents had type 2 diabetes (p -value for difference = 0.003).

FIGURE 1 HERE

The corresponding BMI-for-age-and-sex z-score showed that children with no parents with type 2 diabetes followed normal weight growth patterns (BMI z-score < 1) from childhood through adolescence (average BMI z-score of 0.78 at age 8, range 0.70-0.86) (Figure 1B). In contrast, children with at least one parent with type 2 diabetes were in the overweight category (BMI z-score < 1 to < 2) at age 8 and remained in this category throughout the period of observation (average z-score of 1.15 at age 8, range 0.97-1.33), with a significantly higher z-score than children without a parent with type 2 diabetes (p -value = 0.0002) (Figure 1B).

Children (now young adults) with no parents with type 2 diabetes reached on average the threshold for overweight first at age 20 (adult definition: BMI ≥ 25) (Figure 1A). The difference between groups remained stable over time.

Parents' weight status

Figure 1C and 1D show mean BMI and BMI z-score trajectories for children whose parents did and did not have overweight or obesity. Children with overweight or obese parents had ~ 1 kg/m² and 4 kg/m² higher BMI, respectively, than those with normal weight parents. Children with normal weight and overweight parents had similar slopes from age 8 to early adulthood (0.89 and 1.06 kg/m² per year, respectively), while children with an obese parent had a steeper increase from age 8-14 (1.40 kg/m² per year [1.29-1.51]). Level differences stabilized from early adolescence.

BMI z-scores trajectories differed from starting at age 8 (normal: 0.43 [0.27-0.59], overweight: 0.67 [0.56-0.79], obese: 1.29 [1.17-1.40]) and these differences remained relatively stable over time. Only those

with normal weight parents exhibited a modest decline in the following years before the start of the upward trend.

After age 19, all three groups of children (now young adults) continued to increase in BMI with similar slopes.

Children with normal or overweight parents were in the normal weight range on average (BMI z-score <1) from childhood to end of adolescence (Figure 1D). In contrast, children with at least one obese parent were in the overweight category on average (BMI z-score <1 to <2) at age 8 and remained in this category throughout the period of observation.

After age 19, all three groups of children continued to increase in BMI. However, children with normal weight parents stayed in the normal weight category in early adulthood whereas children with an overweight parent on average crossed the BMI threshold to overweight at age 24.

Race and poverty

Estimated mean BMI and BMI z-score trajectories by race and poverty status are displayed in Figure 2A and 2B and characterized at ages 8, 12 and 19 in Table 2. Non-white children with no parents with type 2 diabetes had the same BMI z-score trajectories as white children whose parents had type 2 diabetes: ~0.4 higher z-score than white children whose parents did not have type 2 diabetes. Non-white children whose parents had type diabetes had an additional modest higher BMI z-score trajectory starting in early adulthood.

Comparing children whose parents did not have type 2 diabetes according to poverty status in childhood and adolescence showed no differences in BMI z-score before age 16 (p-value for poverty:age interaction=0.02). Further, there was no significant difference in BMI z-score trajectories between those whose parents had type 2 diabetes and who did and did not experience poverty (p-value for dm:age interaction=0.40).

Discussion

The results of our study show that only children of normal weight parents are still in the normal weight category at age 26. Whereas children with or without parents with type 2 diabetes, or parents who are either overweight or obese are in average all in the overweight category by age 24. Compared to children without a parent with type 2 diabetes or obesity, children with a parent with type 2 diabetes or obesity exhibit faster increasing BMI trajectories already from 8 years of age and these children are in average in the overweight their entire childhood and adolescence with significant differences in early adulthood BMI. Non-white children without parents with type 2 diabetes had similar BMI z-score to white children with parents with type 2 diabetes. These differences persist in early adulthood, when most children no longer live with their parents.

Our findings of higher and faster increasing BMI trajectories in children with parents with type 2 diabetes are supported by a study by Srinivasan et al. using data from before 1990 and finding higher BMI and higher rate of change in BMI in children with a parent with diabetes compared to children without parents with diabetes [11]. In our study, both children with and without a parent with type 2 diabetes are \geq than 1 BMI unit (kg/m^2) heavier across childhood, adolescence and young adulthood as compared to the participants in the study by Srinivasan et al. where the participants were children in the 1960s-1970s and young adults (age 19-32) between 1988-1991. Further, in our study children without parents with type 2 diabetes almost had double the rate of change in BMI ($1.12 \text{ kg}/\text{m}^2/\text{year}$) as the children in the older study by Srinivasan et al. ($0.59 \text{ kg}/\text{m}^2/\text{year}$).

We have not found other studies investigating the impact of parental weight status on BMI trajectories from childhood to adulthood. However, a study by Fan et al. found that among 6-12 children in Taiwan, the odds ratio of having a BMI trajectory outside the normal BMI range increased with increasing parental BMI [10]. Further, Whitaker et al. showed that having an obese parent in childhood increased the odd ratio of the children becoming an obese adult with around 3 times depending on the child age when parent was obese and parent's sex [4].

Although it was only children with ≥ 1 obese parent, who were in the overweight category from childhood to adulthood, it is worth noting that children with ≥ 1 overweight parent – although they remained in the normal weight category in childhood and adolescence – ended up in the overweight category at age 24. Thus, only children with normal weight parents remained in the normal weight category throughout the period of measures.

In terms of race, children of non-white race without parents with type 2 diabetes had similar BMI trajectories as white children without parents with type 2 diabetes and non-white children with parents with type 2 diabetes had even higher BMI trajectories. Poverty did not influence BMI trajectories before age 16 in children whose parents did not have type 2 diabetes. For children whose parents had type 2 diabetes, there was no difference in BMI z-score trajectories between those who did and did not experience poverty. A secular trend study from the U.S. found that low SES was not associated with childhood obesity up to age 9 [27]. In the present study we only saw this pattern for children without parents with type 2 diabetes. A systematic review of cross-sectional studies investigating the association between childhood obesity and socio-economic status was inconsistent in the findings [28]. However, when the review limited SES to parents' education more consistently show an inverse association with childhood obesity. In the present study, we used poverty as an economic indicator. The lack of relationship between poverty and BMI trajectories might also have been influenced by sex and race as a study have shown that overweight decreased with increasing SEP in white girls but remained high in African American girls across SEP groups [27, 29]

This study has several important strengths. It is among the first to investigate BMI trajectories in from childhood, through adolescent and into adulthood as an outcome of parents' weight and type 2 diabetes status. Further, the data were collected within recent time and are nationally representative.

There were limitations to this study that should be noted with the main limitation of the present study being the missing information about BMI and type 2 diabetes in *both* parents in 686 of the study participants. Due to the setup up of the PSID data is collected as household level and if a parent did not live with the child or in another PSID household, there were no data available. The parent and household data included were baseline values and changes over time in poverty or parents' weight status were not included in the analyses. Further, baseline is not the same calendar year for the included children. However, we are not aware of any major period or cohort effect between 2002-2014, which could have influenced the analyses. Lastly, the children have an uneven number of measurements and the measurements are not obtained at the same age in all the children.

Our study takes advantage of 16 years of recent data following parents and their children and provides insight into the importance of parents' weight and type 2 diabetes for children's growth and obesity risks. Previous studies have found that having parents in the obese category or with type 2 diabetes increased the child's risk of both conditions in adulthood. Our study provides updated confirmation of these findings and expand the evidence by highlighting that also children who are normal weight in childhood and adolescent in average ends up as overweight adults if they have at least one overweight parent. Our study also showed that children without parents with type 2 diabetes are both heavier and have faster increasing BMI from childhood to young adulthood than children and young adults also without parents type 2 diabetes, but who grew up in the 1960s-1980s. Our findings of children with parents with obesity or type 2 diabetes being overweight throughout the entire study period are noteworthy as evidence suggest that the duration of childhood obesity may be one of high importance for the risk of developing type 2 diabetes. In conclusion, our findings suggest that BMI trajectories from childhood to young adulthood are shaped by the parents' health. Thus, decreasing or preventing children's weight gain before adulthood through efforts directly focusing on children or adolescents without the inclusion of their parents may be ineffective.

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