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RESEARCH ARTICLE

Lean legacy, heavy heritage: family history of diabetes and its association with young adult body mass index

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Abstract

Substantial intergenerational transmission of diabetes mellitus (DM) risk exists. However, less is known regarding whether parental DM and DM among extended family members relate to adult offspring's body mass index (BMI), and whether any of these associations vary by sex. Using data from the National Longitudinal Study of Youth 1997 cohort (NLSY97), we assess the sex-specific relationship between DM present in first-degree parents and second-degree relatives and BMI among the parents' young adult offspring.

Multivariate regressions reveal a positive relationship between parental DM and young adults' BMI for both daughters and sons, and the magnitude of coefficients is somewhat larger for the same-sex parent. Further, we observe that the link between parental DM and young adults' BMI is strongest when both parents have diagnosed diabetes. In contrast, the relationship between second-degree relatives with DM and the respondent's BMI is weaker and appears to be sex-specific, through same-sex parent and respondent. Logistic regressions show the association is especially strong when assessing how parental DM status relates to young adults' obesity risk. These results generally persist when controlling for parental BMI. The findings of this study point to the need to better distinguish the role of shared family environments (e.g., eating and physical activity patterns) from shared genes in order to understand factors that may influence young adults' BMI. Young adult offspring of parents with diabetes should be targeted for obesity prevention efforts in order to reduce their risks of obesity and perhaps diabetes.

Keywords: Demography; Obesity; Population Health

Introduction

Diabetes is a serious threat to human health that is growing more prevalent. The age-standardized prevalence rate of diabetes grew from 9.8% to 12.4% among U.S. adults from 1988 to 2012 (Menke *et al.*, 2015). Currently, over 34 million individuals in the U.S. are estimated to have diabetes mellitus (DM), and another one-third of adults are at risk of developing type 2 diabetes mellitus (T2DM) in the next several years (Centers for Disease Control and Prevention, 2020). Several studies find a strong intergenerational association between parental and offspring diabetes risk (Aasbjerg *et al.*, 2020; American Diabetes Association, 2000; Hemminki *et al.*, 2011; Meigs *et al.*, 2000; Nguyen *et al.*, 2009). Heritability estimates of T2DM range from 20 to 80% (Ali, 2013), and the relative risk of T2DM is around 2.7 for persons who have first-degree relatives with T2DM compared to those without this family health history (InterAct Consortium *et al.*, 2013).

Controversy exists regarding causality for intergenerational DM transmission (Arslanian *et al.*, 2005). Family history of the disease may be due to both genetic (Sladek *et al.*, 2007; Voight *et al.*, 2010) and shared environmental risk factors (Kaprio *et al.*, 1992). The health effects of family history vary by age, with higher risks for those age 20 and over (Annis *et al.*, 2005) but with mixed findings for minor children (Gilliam *et al.*, 2007). In particular, maternal or paternal diabetes influences the risks of diabetes for both sons and daughters in one study (InterAct Consortium *et al.*, 2013), but only for daughters in another (Balkau *et al.*, 2017). Maternal transmission is more evident than paternal transmission in other studies. Maternal diabetes relates to risks of diabetes for both sons and daughters (Thompson, 2014) or shows more robust associations with offspring (sex not specified) (Chernausek *et al.*, 2016). These results demonstrate that it is important to examine sex-specific models, with the sex of both parents and offspring included. They also suggest that intergenerational transmission by mothers (Chernausek *et al.*, 2016) to daughters may be more common, perhaps reflecting *in utero* exposure influences (Pettitt *et al.*, 2008), the mother's predominant role in influencing children's food intake and activity levels, or even the modelling of mothers' behaviours to daughters (Elfhag & Linné, 2005).

Bodyweight and diabetes risk have a complicated relationship (Ma & Popkin, 2017). Foremost, obesity is a risk factor for the incidence of diabetes (Bray *et al.*, 2016; Li *et al.*, 2020; Narayan *et al.*, 2007; Srinivasan *et al.*, 2003). To the extent that parental DM status precedes the body mass index (BMI) level of their offspring, identifying how these excess BMI risks arise due to parental DM may help target individuals who can alter their BMI risk factors. Specifically, parental diabetes has been linked to greater BMI or obesity risk among their minor children, including from non-Hispanic White mothers (but not fathers) to 10- to 17-year-olds (Chernausek *et al.*, 2016; Weinstock *et al.*, 2015), from Swedish mothers and fathers to 12-year-olds (Tojjar *et al.*, 2020), and from Louisiana parents (mothers and fathers not tested separately) to their children aged 4–11, 12–18, and 19–32 (Srinivasan *et al.*, 2003). Research has also demonstrated relationships between parental DM and obesity or overweight among their adult children (Cederberg *et al.*, 2015) or from maternal DM only to adult BMI or central obesity (Tan *et al.*, 2008). These effects might reflect both genetic and shared social environmental family influences on food intake and physical activity. Fewer studies examine the associations between parental diabetes and offspring weight in young adulthood, a time when food and activity choices may reflect young adults' personal preferences or concerns about health risks rather than family habits.

Rarely have studies of intergenerational transmission of parental DM to offspring BMI assessed the role of second-degree relatives, such as grandparents, aunts, or uncles, or the effects of transmission from one vs. both parents In a French study, diabetic grandparents but not parents were associated with greater risk of being overweight in 4-year-olds (Jouret *et al.*, 2007). In a sample from India, DM in both parents was more strongly associated with overweight or obesity in offspring than when DM was present in only one parent (Praveen *et al.*, 2010). Yet in another study, a clinical trial with nearly 500 parents with offspring aged 10 to 17, the offspring risk of excess BMI was similar whether one or both parents had diabetes (Chernausek *et al.*, 2016). These results suggest studies should expand the network of kin under consideration to include more second-degree relatives, test for the effects of dual-parent diagnoses of DM on offspring BMI, and introduce larger and nationally representative samples.

Our approach is informed by general principles of inheritance. Individuals who are more closely related simply have more genes in common than more distant relatives. Specifically, first-degree relatives share on average 50% of their (nuclear) DNA while second-degree relatives share 25%. We extend this to suggest that shared DNA, and the health risks they may engender, will also lead to shared disease risks. Accordingly, under this strict interpretation, first-degree relatives should have a shared association of disease that is twice that for second-degree relatives if the genes in question are causal and are transmitted with autosomal dominant inheritance. This is a baseline prediction that is naturally complicated by several factors. First, the 'disease' associations between relatives are cross-phenotype (association between parental DM and adult offspring

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BMI). Second, closer relatives generally share more environmental factors than more distant relatives, which would make these associations stronger between first rather than second-degree relatives for reasons that may have no genetic basis. Third, there is the possibility that individuals with a disease or their offspring may act to reduce risks to offspring with preventive action (Hariri *et al.*, 2006), which would reduce rather than promote worse outcomes for these descendants. Nonetheless, our approach will help identify the total (net) effects of family history of DM on respondent BMI via these mechanisms and it will quantify the relative magnitude of the associations between first- and second-degree relatives.

Our analyses contribute to the literature in several ways. First, we investigate whether a family history of T2DM among one or both parents- *and* second-degree relatives is associated with the BMI of young adults. Second, many of the studies we reviewed have involved clinical samples that may have narrower eligibility criteria, such as when children (Chernausek *et al.*, 2016; Weinstock *et al.*, 2015) or parents (Praveen *et al.*, 2010) are sampled because they have diabetes. In contrast, our study uses the National Longitudinal Study of Youth, a large and nationally representative sample. Third, we examine whether the sex of the parent and child, as well as sex-linked parent-child pairs, are important in the relationship between parental DM and offspring BMI. Finally, we focus on BMI in early adulthood, when life changes, such as moving away from parental homes, and increases in autonomy may lead to changes in food consumption and activity levels and thereby alter the transmission of obesity risk.

Methods

Sample

We use the National Longitudinal Study of Youth, 1997 (NLSY97) cohort (Bureau of Labor Statistics U.S. Department of Labor, 2019a) (Cooksey, 2018). The NLSY97 cohort is a nationally representative sample of adolescents aged 12 to 16 as of January 1, 1997, and born between 1980 and 1984. Followed annually until 2010 and biennially thereafter, annual response rates are consistently above 80 per cent (Bureau of Labor Statistics U.S. Department of Labor, 2019b). Approximately nine thousand ($N = 8{,}984$) respondents are in the original cohort. We excluded 2,295 respondents who were missing height and weight information used to construct BMI or whose BMI values were exceptionally low (<15.0) or high (>60.0). Another 971 respondents were excluded because they had not yet completed the 'Health at Age 29' NLSY module, which is the basis for our calculation of family diabetes history. A small number were excluded because they had missing data on diabetes family history ($N = 25$) or education ($N = 31$). Some respondents had multiple instances of the above exclusion criteria. After these exclusions, our study sample has 3,130 males and 3,140 females.

Variables

Dependent variables

We use self-reported data on height at age 25 and weight reported at age 30 or 31 (depending on the timing of the biannual interview) to calculate BMI. After excluding the extreme BMIs described above, we compute categories of BMI: healthy weight ($15 < \text{BMI} < 25$), overweight ($25 \leq \text{BMI} < 30$), and obese ($\text{BMI} \geq 30$ and $\text{BMI} \leq 60$).

Independent variables

Self-reported family history of diabetes is measured at age 29. Diabetes family history questions were worded such that a distinction between type 1 and type 2 could not be made, although type 2 is 20 times more common than type 1 DM (American Diabetes Association, 2018). We can differentiate those who report family histories of DM among parents, siblings, or second-degree

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relatives (i.e., aunts, uncles, grandparents) on the maternal and paternal sides of the family. However, we recognize the potential that respondents have incomplete information concerning their family health history, especially when reporting second-degree relatives.

We measured *first-degree relatives with diabetes* based on a series of questions that ask whether either of the respondent's biological parents or siblings have been told by a doctor that they have DM. Only a small percentage of the respondents reported that their siblings had DM (277 out of 8985 respondents or 3 per cent of the sample) so we did not focus on siblings but instead created separate variables that indicate whether their father or mother had been told that they have DM. As a robustness check, we did estimate alternative models where we include sibling DM status (available from the authors upon request) and the results did not change. Respondents were also asked about their second-degree relatives' DM status. Specifically, DM for second-degree relatives was ascertained using two questions that asked how many of your mother's (father's) brothers, sisters, or parents have been ever told by a doctor that they have diabetes. This information was coded as two dichotomous variables where one identifies whether there were any second-degree relatives with DM on the maternal side and the other identifies any second-degree relatives with DM on the paternal side of the respondent's family. Other variables addressed whether respondents did not know their family history of DM, separately for first-degree and second-degree relatives.

Control variables

In all of our multivariate analyses, we control for the respondent's race/ethnicity (measured in 1997 with categories of non-Hispanic White; Black non-Hispanic; Hispanic, any race; and other), educational attainment (measured at age 25 with categories of less than high school, high school graduate, some college, college graduate, and postgraduate education), marital status (i.e., measured at age 30 or 31 to include married or not married), parental status (measured at age 30 or 31 to include any children versus none), the family of origin's income-to-needs ratio (measured from the responding parent's 1997 report), and their parent's categorical BMI (again measured from responding parent's 1997 report and using categories of healthy weight, overweight, and obese). If the 1997 income-to-needs ratio was missing, the non-missing mean was imputed and a dummy variable was included to capture whether the value had been imputed. If the responding parent was missing on BMI, then we used the data from the non-responding parent if they had valid data on this variable. When parental BMI was less than 15.0 or greater than 60, the variable was set to missing.

Analytical strategy

We present sex-specific descriptive and multivariate results. For the multivariate analyses, we estimate a series of regression models where the dependent variables are as follows: (1) BMI at age 30–31, (2) overweight versus healthy weight, and (3) obese versus healthy weight. The first model is evaluated with an OLS regression and the latter two models with logistic regressions. For all three specifications of the dependent variable, we first estimate the associations between DM history among parents and the respondent's BMI and then estimate a second model where we include measures of DM history of both first- and second-degree relatives.

To understand further the link between parental diabetes and offspring's weight-related outcomes, we additionally test whether the link with young adults' BMI varies by the number of parents who have the diagnosis and the sex of the parent with diabetes. Parental BMI, measured at the start of the panel in 1997, is typically available for the parent who filled out the survey, which is usually the mother. Given that the examination of sex-specific linkages may be altered by the control of one parent's BMI, the supplementary materials provide analyses with and without parental BMI control.

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All analyses were conducted using STATA software version 15 (StataCorp, 2017), and all descriptive and multivariate analyses were weighted using NLSY-generated weights for panel designs (National Longitudinal Surveys, 2023) to adjust for possible selection bias inherent in eliminating respondents who did not provide the height and/or weight information needed to construct BMI. No evidence of problematic multicollinearity was detected in any of the multivariate analyses (Belsley *et al.*, 2005).

Results

Table 1 provides descriptive information on the female and male respondent samples used in the analysis. One or more parents have DM for 21% of females and 19% of males. The average BMIs for young adult women and men are 28.17 and 28.18, respectively. We find that 59% of women and 64% of men in our sample were either overweight or obese at age 30–31.

Reports of parental diabetes range from 11% to 13% across respondent reports of mothers' and fathers' diabetes status, which is consistent with the CDC's report of a 12% diabetes prevalence in 2020 (Centers for Disease Control and Prevention, 2020). Percentages of respondents with one or more second-degree relatives with a DM diagnosis range from a low of 24% for males reporting on second-degree relatives on their father's side to a high of 34% for females reporting on second-degree relatives on their mother's side. It is important to note that the two questions for second-degree relatives were worded as follows: 'How many of your mother's/father's brothers, sisters, or parents have ever been told by a doctor that they have diabetes?' These relatively higher percentages of DM among second-degree relatives compared to reports for mothers and fathers are a function of the fact that there are more of these relatives, and thus, the chances that at least one has been diagnosed with DM should be higher.

The racial/ethnic profile of our sample broadly mirrors the racial/ethnic profile of US young adults reported in the 2010 Census, where 72.4 per cent identified as White, 12.6 per cent identified as Black or African American, and 16.3% identified as Hispanic or Latino (Humes *et al.*, 2011).[1] The modal educational attainment of our sample is a high school diploma. This reflects the relatively young age of the NLSY respondents as many of them may not have attained their final educational degree at the time of the interviews used for our analyses. That said, the educational distributions we observe for young women and men are consistent with the frequency distributions listed in the NLSY documentation (Bureau of Labor Statistics U.S. Department of Labor, 2019a).

Tables 2 and 3 present the multivariate results for the OLS regressions for BMI outcomes and the logistic regressions that examine (1) the risk of being overweight versus healthy weight and (2) the risk of being obese versus healthy weight. The BMI estimates reveal a fairly consistent pattern for the association between a history of parental diabetes and BMI in young adulthood. Offspring with a father or a mother who has diabetes have a significantly higher BMI, a pattern that holds for both daughters (Table 2) and sons (Table 3), although the magnitude of the estimated Model 1 coefficients is somewhat higher for the same-sex parent. That is, having a mother with diabetes rather than a father with diabetes is associated with a modestly higher BMI for daughters (coefficients of 1.92 vs. 1.44), while the reverse is true for sons (coefficients of 1.39 vs. 0.96). Associations between parental DM history and offspring BMI retain significance when adjusting for the presence/absence of second-degree relatives with DM (OLS Model 2, of Tables 2 and 3).

Logistic regression estimates presented in Tables 2 and 3 disclose that a parental history of diabetes is associated with an increased risk of obesity relative to a healthy weight for young adult women but not for young men. In contrast, the estimated odds ratios for overweight versus healthy weight associated with parental diabetes are generally insignificant for both the females and males.

[1]These figures are not disaggregated by age. In addition, there is overlap between race and ethnicity percentages in the Census publication. Both features somewhat limit the direct comparability of Census estimates to our data.

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Table 1. Weighted descriptive statistics

Variable	Definition	Females		Males	
		Mean or proportion	Standard deviation	Mean or proportion	Standard deviation
Respondent's BMI at age 30–31	Self-reported weight in kilograms divided by the square of self-reported height in metres	28.17	7.31	28.18	6.56
Respondent healthy weight	1 = BMI is 15 to 24.9, 0 = otherwise	041	0.49	0.30	0.46
Respondent overweight	1 = BMI is 25.0 to 29.9, 0 = otherwise	0.27	0.44	0.33	0.47
Respondent obese	1 = BMI is greater than 29.9, 0 = otherwise	0.32	0.47	0.31	0.46
Mother has diabetes	1 = respondent says mother has diabetes, 0 = otherwise	0.12	0.33	0.11	0.31
Father has diabetes	1 = respondent says father has diabetes, 0 = otherwise	0.13	0.34	0.12	0.32
Second-degree relatives with diabetes, mother's side	1 = one or more of mother's relatives has diabetes, 0 = otherwise	0.34	0.47	0.29	0.46
Second-degree relatives with diabetes, father's side	1 = one or more of father's relatives has diabetes, 0 = otherwise	0.29	0.45	0.24	0.43
One parent has diabetes	1 = one parent has diabetes, 0 = otherwise	0.21	0.41	0.19	0.39
Both parents have diabetes	1 = both parents have diabetes, 0 = otherwise	0.02	0.15	0.02	0.13
Mother only has diabetes	1 = mother only has diabetes, 0 = otherwise	0.10	0.30	0.09	0.29
Father only has diabetes	1 = father only has diabetes, 0 = otherwise	0.11	0.31	0.10	0.30
Parents don't know	1 = unknown if parents had diabetes, 0 = otherwise	0.01	0.10	0.01	0.11
Second-degree relatives, mother don't know	1 = unknown if any of mother's relatives have diabetes, 0 = otherwise	0.08	0.28	0.10	0.29
Second-degree relatives, father don't know	1 = unknown if any of father's relatives have diabetes, 0 = otherwise	0.19	0.40	0.19	0.39
No mother/father diabetes	1 = neither the respondent's mother nor father has diabetes, 0 = otherwise	0.76	0.42	0.82	0.39
None of the mother's second-degree relatives have diabetes	Reference group	0.58	0.49	0.64	0.48
None of the father's second-degree relatives have diabetes	Reference group	0.52	0.50	0.62	0.49

(*Continued*)

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Table 1. (*Continued*)

Variable	Definition	Females		Males	
		Mean or proportion	Standard deviation	Mean or proportion	Standard deviation
White non-Hispanic	1 = respondent identifies as White, non-Hispanic, 0 = otherwise	0.70	0.46	0.70	0.46
Black non-Hispanic	1 = respondent identifies as Black, non-Hispanic, 0 = otherwise	0.16	0.37	0.16	0.36
Hispanic	1 = respondent identifies as Hispanic, 0 = otherwise	01.3	0.33	0.13	0.11
Other	1 = respondent identifies as Asian, Pacific Islander, American Indian, Mixed Race, or Other, 0 = otherwise	0.01	0.11	0.01	0.11
Less than high school	1 = respondent has less than 12 years of education, 0 = otherwise	0.06	0.23	0.06	0.25
High school graduate	1 = respondent is a high school graduate, 0 = otherwise	0.45	0.50	0.55	0.50
Some college	1 = respondent has attended some college, 0 = otherwise	0.10	0.31	0.09	0.29
College graduate	1 = respondent is a graduate of a four-year college, 0 = otherwise	0.25	0.43	0.22	0.42
Post-bachelor	1 = respondent has some post-college education, 0 = otherwise	0.14	0.35	0.12	0.32
Married	1 = respondent is married at age 30–31 interview, 0 = otherwise	0.48	0.50	0.44	0.50
Has children	1 = respondent has one or more children at age 30–31 interview, 0 = otherwise	0.63	0.48	0.51	0.50
Parent healthy weight	1 = parental BMI in 1997 is 15.0–25.0, 0 = otherwise	0.39	0.49	0.39	0.49
Parent overweight	1 = parental BMI is 25.0–29.9, 0 = otherwise	0.27	0.44	0.24	0.43
Parent obese	1 = parental BMI is greater than 29.9, 0 = otherwise	0.19	0.40	0.21	0.41
Parent weight missing	1 = parental BMI is missing, 0 = otherwise	0.15	0.35	0.16	0.36
Family Income-to-Needs in 1997	Ratio of family income to the federal poverty threshold in 1997	3.06	2.43	3.17	2.82
1997 Income-to-Needs imputed	1 = yes, 0 = no	0.24	0.43	0.24	0.43
N		3,140		3,130	

The relationship between DM among second-degree relatives and young adult BMI, controlling for parental DM is weaker than the relationships observed for parental DM as would be expected given that, compared to the parents, second-degree relatives share fewer genes and likely share fewer lifestyle factors with the young women and men who are the focus in this study. Estimates presented in Table 2 for maternal second-degree DM are weakly associated with higher BMI and risk of obesity among female offspring and the presence of second-degree paternal relatives with diabetes is associated with higher BMI and higher odds of being overweight for

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Table 2. Multivariate parameter estimates of familial history of diabetes on respondent's BMI: Females[a]

	OLS regression of respondent BMI (t-ratios in parentheses)		Logit odds ratios for overweight Vs. normal weight (95% CI in parentheses)		Logit odds ratios for obese vs. normal weight (95% CI in parentheses)	
Independent variables	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Mother has diabetes	1.92**	**1.69****	0.97	0.93	1.60**	1.49**
	(3.87)	**(3.36)**	(0.70–1.35)	(0.66–1.30)	(1.19–2.15)	(1.10–2.03)
Father has diabetes	1.44**	1.27**	1.22	1.14	1.71**	1.64**
	(3.60)	(3.12)	(0.90–1.67)	(0.83–1.56)	(1.27–2.29)	(1.21–2.22)
Second-degree relatives with diabetes, mother's side[b]	–	0.58† (1.87)	–	1.11 (0.88–1.39)	–	1.21† (0.97–1.52)
Second-degree relatives with diabetes, father's side[c]	–	**0.65**** **(2.10)**	–	1.25† (0.99–1.58)	–	**1.15 (0.91–1.46)**
N	3,140	3,140	2,034	2,034	2.297	2,297
Adjusted R^2/Pseudo R^2	0.12	0.13	0.02	0.03	0.12	0.12
Model fit	$F_{(17,\ 3,122)} = 24.88$**	$F_{(21,\ 3,118)} = 20.96$**	$\chi^2_{(17)} = 58.94$**	$\chi^2_{(21)} = 64.71$**	$\chi^2_{(17)} = 268.65$**	$\chi^2_{(21)} = 272.96$**

**$p < 0.05$, †$p < 0.10$.

Note. All of the multivariate estimation controls for the respondent's race/ethnicity, educational attainment, marital status, parental status, absence of knowledge about diabetes family history, household income-to-needs ratio in 1997, presence of minor children in the household at the time BMI is measured, and their parent's categorical BMI. The parameter estimates for these independent variables are available from the authors upon request.

[a]Coefficient estimates that are significantly different between females and males have been bolded.

[b]The omitted category in this dummy variable sequence are those respondents who indicated that there were no second-degree relatives on their mother's side of the family who had a diabetes diagnosis.

[c]The omitted category in this dummy variable sequence are those respondents who indicated that there were no second-degree relatives on their father's side of the family who had a diabetes diagnosis.

Table 3. Multivariate parameter estimates of familial history of diabetes on respondent's BMI: Males[a]

	OLS regression of respondent BMI (t-ratios in parentheses)		Logit odds ratios for overweight vs. normal weight (95% CI in parentheses)		Logit odds ratios for obese vs. normal weight (95% CI in parentheses)	
Independent variables	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Mother has diabetes	0.96**	**0.85****	0.81	0.78	1.39†	1.31
	(2.30)	**(2.04)**	(0.56–1.17)	(0.53–1.13)	(0.97–1.99)	(0.90–1.90)
Father has diabetes	1.39**	0.97**	1.07	1.03	1.53**	1.33
	(3.52)	(2.49)	(0.77–1.49)	(0.74–1.44)	(1.09–2.13)	(0.93–1.89)
Second-degree relatives with diabetes, mother's side[b]	–	0.33 (1.26)	–	1.15 (0.91–1.47)	–	1.29† (0.99–1.68)
Second-degree relatives with diabetes, father's side[c]	–	**1.38** (4.77)****	–	1.10 (0.84–1.43)	–	**1.52**** **(1.14–2.02)**
N	3,130	3,130	2,134	2,134	1,913	1,913
Adjusted R^2/Pseudo R^2	0.09	0.10	0.03	0.03	0.10	0.11
Model fit	$F_{(17,\ 3,112)} = 17.81$**	$F_{(21,\ 3,108)} = 16.29$**	$\chi^2_{(17)} = 67.95$**	$\chi^2_{(21)} = 71.90$**	$\chi^2_{(17)} = 191.75$**	$\chi^2_{(19)} = 208.54$**

**p<.05, †p<.10.

Note. All of the multivariate estimation controls for the respondent's race/ethnicity, educational attainment, marital status, parental status, absence of knowledge about diabetes family history, household income-to-needs ratio in 1997, presence of minor children in the household at the time BMI is measured, and their parent's categorical BMI. The parameter estimates for these independent variables are available from the authors upon request.

[a]Coefficients that are significantly different between females and males have been bolded.

[b]The omitted category in this dummy variable sequence are those respondents who indicated that there were no second-degree relatives on their mother's side of the family who had a diabetes diagnosis.

[c]The omitted category in this dummy variable sequence are those respondents who indicated that there were no second-degree relatives on their father's side of the family who had a diabetes diagnosis.

Table 4. Multivariate OLS parameter estimates of familial history of parental diabetes on female and male respondent's BMI (t-ratios in parentheses)[a]

	Parental sex-blind estimates		Parental sex-specific estimates	
Independent variables	Females	Males	Females	Males
One parent has diabetes	1.54** (4.23)	1.37** (4.44)	–	–
Both parents have diabetes	3.89** (3.92)	1.39 (1.11)	–	–
Mom only has diabetes	–	–	1.79** (3.25)	1.14** (2.67)
Dad only has diabetes	–	–	1.32** (3.07)	1.55** (3.76)
Both parents have diabetes	–	–	3.90** (3.93)	1.38 (1.10)
N	3,140	3,130	3,140	3,130
Adjusted R^2/Pseudo R^2	0.12	0.09	0.12	0.09
Model fit	$F_{(17,\ 3,122)} = 24.88$**	$F_{(17,\ 3,112)} = 18.05$**	$F_{(18,\ 3,121)} = 23.50$**	$F_{(18,\ 3,111)} = 17.07$**

**p<.05, †p<.10.
[a]All of the multivariate estimation controls for the respondent's race/ethnicity, educational attainment, marital status, parental status, absence of knowledge about diabetes family history, household income-to-needs ratio in 1997, presence of minor children in the household at the time BMI is measured, and their parent's categorical BMI. The parameter estimates for these independent variables are available from the authors upon request.

young adult women. In contrast, paternal second-degree DM is significantly associated with higher BMI and risk of obesity among young adult men. There is also weak evidence of an association between one or more maternal second-degree relatives with diabetes and the odds of being obese for young adult men. These patterns suggest the association between DM history of second-degree relatives and BMI may be somewhat sex-specific. Supplemental analyses confirmed these significantly different patterns by testing for interactions with sex (i.e., maternal/paternal sex by offspring sex). The full interaction analysis results are available from authors on request.

To examine the role of parental diabetes further, we next tested to see if the link with young adults' BMI varies by the number of parents who have DM and, separately, by the sex of the parent with DM. These estimates are presented in Table 4. The first two columns of coefficients do not include information about the sex of the parent who has diabetes if only one parent has DM. The last two columns of coefficients distinguish between whether it is the mother or father with DM.

Both sets of estimates reveal that having any parent with DM is significant for offspring BMI. However, for young women, having two parents diagnosed with diabetes is associated with larger values of offspring BMI relative to having only one or no parent who has DM. In contrast, there is no added effect of both parents having DM in the case of young men. The magnitude of the effect is more than doubled for young women when comparing two parents to just mom or dad having DM (coefficients of 3.89 for both parents vs 1.54 for one parent). In the case of parental sex-specific estimates, we observe that the magnitude of the effect is moderately larger for young women if their mother rather than their father has diabetes (1.79 vs. 1.32). Likewise, for the young men, the magnitude of the relationship is moderately larger if their father rather than their mother has diabetes (1.55 vs. 1.14). As expected, the relationship between both parents having diabetes and the young adult's BMI is essentially estimated to be the same across the two specifications. We do estimate separate analyses that first control for parental BMI and then estimate without the parental BMI to see if the patterns of association hold. These analyses suggest that the associations are generally robust across this sensitivity test, with the exception of the male logits that estimate

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the odds of being obese versus normal weight. Comparisons there reveal that initially statistically significant coefficients associated with first- and second-degree relatives having diabetes and young men's risk of obesity often become insignificant when controls for parental BMI are included. These analyses are presented in supplemental tables.

Discussion

Our results show a strong and consistent relationship between parental DM and young adult obesity. These relationships between parental DM and offspring BMI have been noted in past research for younger offspring ages (Chernausek *et al*., 2016; Tojjar *et al*., 2020; Weinstock *et al*., 2015). This relationship has also been found in past research on older offspring, including those averaging 33 years old (Nguyen *et al*., 2009), 38 years old (Tan *et al*., 2008), 40 years old (Aldhous *et al*., 2015), 57 years old (Cederberg *et al*., 2015), and 60 years old (Alharithy *et al*., 2018). Although we argued that young adulthood might be a time when offspring could adopt healthier habits to prevent higher BMI, the evidence is inconsistent with the idea that young adults use this turning point in their lives to attain greater health. Perhaps young adults with parental DM could benefit from counselling that highlights the genetic risks of DM and how healthier BMIs can delay or prevent DM, as some programs aim to achieve (Koehly *et al*., 2015; Waxler *et al*., 2012).

We also estimated whether the presence of DM among second-degree relatives would enhance models that already control for parental DM. The findings from this study point to the lesser role of DM among second-degree relatives in influencing offspring BMI, once parental DM is controlled. Nevertheless, DM among secondary relatives retains significance in approximately half the models for BMI and obesity. These effects suggest a role for genetic transmission, given that aunts and uncles are less likely than parents to share environments with respondents. In general, any effect from second-degree relatives is taken to indicate greater probability of genetic transmission (Nielsen *et al*., 2015).

In addition, there is some support for the idea of sex-specific patterns of results. In the case of the BMI equations, the magnitude of coefficients is larger for mother-daughter pairs and father-son pairs than their opposite-sex counterparts. In addition, paternal second-degree relatives with DM are associated with both an elevated BMI and elevated risk of obesity for male respondents. Past research has found some indications that maternal DM may be more predictive of offspring BMI or DM than paternal DM (Alcolado & Alcolado, 1991; Groop *et al*., 1996). In addition, some studies found maternal transmission is more predictive of daughter's than son's BMI or DM (Tan *et al*., 2008). Tan et al. suggested that among other reasons, there may be genomic imprinting, whereby maternally and paternally derived genes may alter one's susceptibility to metabolic problems. Or, intrauterine environmental exposures might transmit risks maternally. Finally, mothers may often determine food intake among offspring and perhaps model both food and activity patterns for offspring, especially for young girls (Karter *et al*., 1999). The trends in our results support these possibilities.

One advantage of our study is the employment of a large representative sample. Our finding that 59% of women and 64% of men in our sample were either overweight or obese at age 30–31 may seem high, but these results are consistent with data from the Behavioral Risk Factor Surveillance System (BRFSS). Exact age-specific national comparisons to these numbers are difficult to identify given that all of the individuals in our NLSY sample were age 30–31 when they reported their weight. BRFSS data, which uses similar self-reported height and weight questions, reveal that nationally between 2010 and 2014 (when NLSY97 respondents reached the age of 30), 57.3% of females and 69.5% of males aged 30–31 were overweight or obese (Centers for Disease Control & Prevention, 2021). As reported earlier, both racial/ethnic and educational levels in our sample mirrored national statistics. Based on these figures, we conclude that our sample is more broadly representative of the population than many of the smaller or clinical samples utilized in past research.

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While our findings have strong external validity, they also have several contextual limitations. First, these analyses rely on self-reported height and weight measurements which could be subject to recall error but this is less likely to be true in the case of the discrete weight categorizations (e.g., obese). Second, we are not able to control for a family of origin behavioural patterns for diet and exercise, which might condition the associations we observe in these data.

These analyses suggest several areas that merit more research. First, diabetes is underdiagnosed in general and we might be observing the effects of known DM only. There could be 'second-order underreporting of familial DM by the respondent since even the parents do not know and yet the true but unobserved diabetes could still affect the young adult's BMI. Second, future research should aim to incorporate measures of DM medication into the analysis, especially for population-based samples such as the NLSY. It is an open question of whether there might be a significant difference in the influence of parental DM if the parent is actively managing DM with medication and behavioural health strategies. Third, future research could assess how parental transmission of DM might be affected by parental marital status. Knowledge of family history and subsequent risk assessment might be importantly affected by whether the respondent's parents are still married and the degree to which the respondent has contact with each parent. Finally, future research should aim to better understand the nature of connections between parental DM and offspring BMI, as our analysis lends evidence for a significant association with risk of obesity but not risk of overweight. Such results underscore the importance of targeting young adults for healthy weight programmes and interventions, in an effort to prevent or delay the onset of obesity and DM. In summary, results from this study represent an important step forward in better understanding the relative influence of shared genes versus family environment as they relate to measures of health such as body weight.

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