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Intergenerational Transmission of Body Mass and Obesity Status in Australia

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Abstract

We estimate the intergenerational transmission of the body-mass index (BMI) and obesity status using the Household, Income and Labour Dynamics in Australia survey. The intergenerational elasticity of BMI between mother and adolescent is 0.242, which is consistent with estimates from other countries. Controlling for adolescent-specific fixed effects reduces the estimated elasticity to 0.043. This suggests that genetics and the permanent environment may explain much of the observed persistence of BMI across generations. Adolescents in Australia whose mothers are with obesity are expected to have an increased probability of being with obesity themselves by 0.094 percentage points where the adolescent obesity prevalence rate is 7.70 percent. This intergenerational persistence in obesity status is much stronger when the mother is with morbid obesity. The degree of intergenerational persistence of BMI and obesity status exhibits a socioeconomic gradient: the transmission is stronger among disadvantaged households relative to better-off households. When coupled with prevailing socioeconomic disparities in obesity rates specifically and health status generally, this dynamic feature of Australian society may further contribute to the inequity in health outcomes in the future.

Keywords: Australia; obesity; BMI; health inequality; HILDA

1. Introduction

Intergenerational mobility has received considerable attention from economists and social scientists. This interest stems from the concern that a lack of intergenerational mobility is an indicator of the lack of opportunity (Solon 1992). To date, much of the literature on intergenerational mobility primarily has focused on the relationship between parental and offspring earnings (for Australia, see, e.g., Leigh 2007 and Murray et al. 2018). The health dimension has not received as much attention 'despite its central importance of health to welfare' (Halliday et al. 2018, p. 26), and 'economists have paid scant attention to the magnitudes or underlying causes of intergenerational linkages in health outcomes' (Thompson 2014, p. 132), although this has improved in recent years. But beyond its centrality to welfare, health is increasingly recognized as a unique dimension of intergenerational mobility in socioeconomic status. As Halliday et al. (2018) demonstrate, individuals in the US experience higher mobility in the health dimension (measured as self-rated health status) than in income.

This paper is concerned with estimating the intergenerational mobility of health in an Australian context. We contribute to studies which examine the intergenerational mobility of health in general (e.g., Bhalotra and Rawlings (2013), Thompson (2014), and Halliday et al. (2018)), but more specifically, to the literature which looks at the intergenerational mobility in weight status.[1] This branch of the academic literature is important since the endowment and experiences of one generation which results in detrimental health outcomes for the next can perpetuate a state of health inequality in a society. As Currie and Moretti (2007, pp. 231–232) state, 'Most people find inequality less pernicious when it is not passed on from generation to generation.'

As a measures of health status, we focus on the body-mass index (BMI) and, in particular, on obesity, which is derived from the BMI.[2] The obesity epidemic is a major public health issue in Australia (AIHW 2017). Overall, the obesity rate in Australia is the fifth highest in the OECD (OECD 2017a). Population-

[1] A few examples of studies in the economics literature which look at the intergenerational transmission of body mass are Dolton and Xiao (2015), Classen and Thompson (2016), Dolton and Xiao (2017).

[2] Adults are classified as with obesity if their BMI, defined as kilograms in weight divided by meters (squared) in height, is greater than or equal to 30; overweight is defined as adults with a BMI that is greater than or equal to 25. These and the other standard weight-status categories associated with the BMI are in the heading of Table 2 in Section 2.

based studies estimate this to be 28 percent of the adult population, with the prevalence increasing if people with overweight are also included: a combined 63 percent (OECD 2017b). The OECD notes that the prevalence of severe obesity among Australian adults has doubled from 5 to 10 percent within the period 1995–2016. Among children, about 25 percent were considered overweight or obese in 2017, and the prevalence is more severe in disadvantaged communities, such as Indigenous children and those living outside major cities (AIHW 2017).

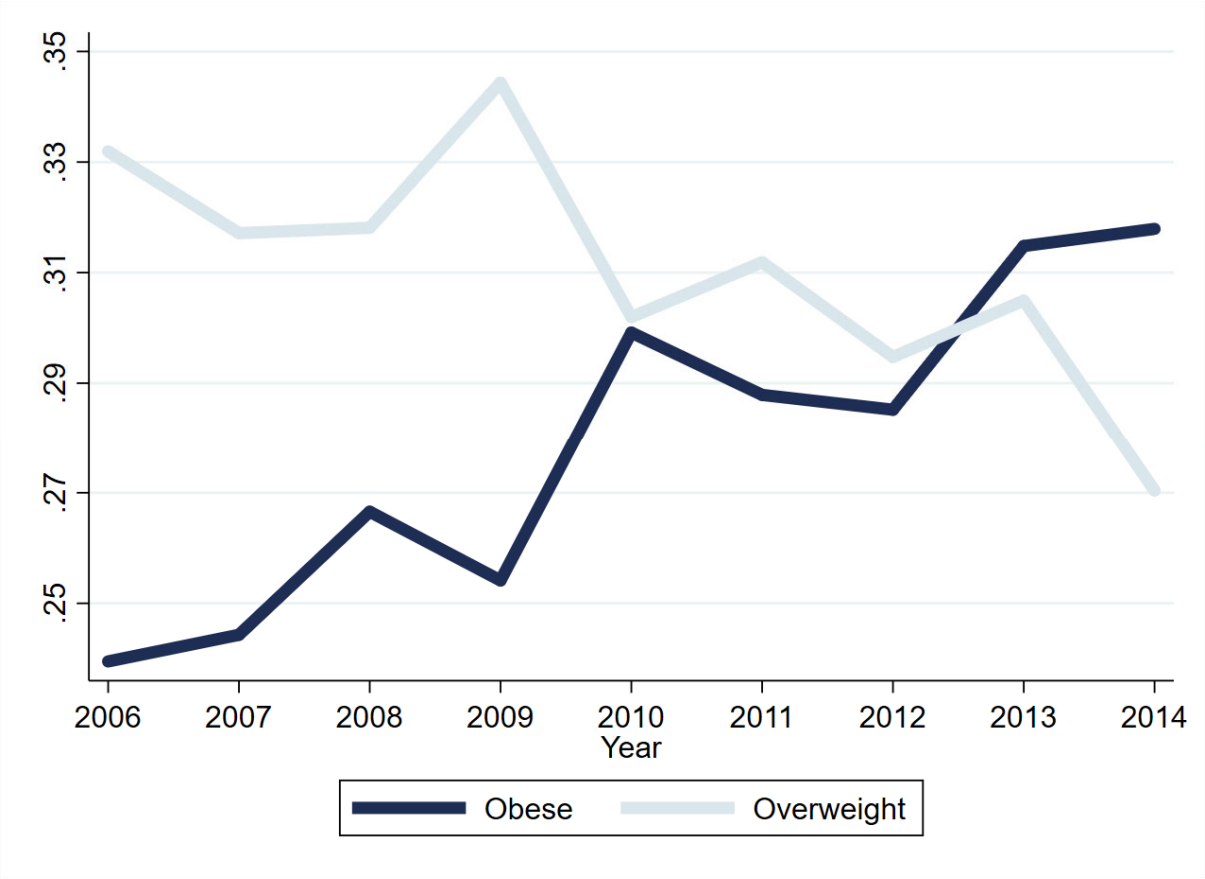
Elevated youth obesity rates have severe ramifications for individuals and for society. Obesity in adolescence is linked with lower future wages (Cawley 2004), higher rates of attempted suicide (Eisenberg et al. 2003), decreased educational performance (Schwimmer et al. 2003), increased risk of early death (WHO 2015), and disability in adulthood (WHO 2015). As the risk of obesity in adulthood is twice as high for children with obesity of any age relative to children without obesity (Serdula et al. 1993), many of the hazards facing adults with obesity are also indirectly faced by adolescents with obesity. Therefore, ailments associated with obesity in adults such as hypertension, certain types of cancer, heart disease, and gall bladder disease are also more likely to affect people who had obesity in adolescence.

This is especially relevant since Australian female obesity rates is projected to increase further (Hayes et al. 2017). For the period 2006–2014, we observe an upward trend in the prevalence of obesity for mothers in our dataset even if the prevalence of being overweight is declining in the same period (Figure 1).[3] Without behavioral and policy changes, the trajectory is unlikely to change. This has implications for the sustainability of the public health insurance system as rising obesity rates are associated with increased costs to treat and manage its comorbidities. In Australia, obesity and overweight were estimated to be responsible for 7 percent of the total disease burden, mostly associated with coronary heart disease, diabetes, osteoarthritis, and chronic kidney disease. Over 60 percent of this figure was considered a 'fatal burden' (AIHW 2017). In a PwC report prepared for Obesity Australia (PwC 2015), the projected additional direct and indirect costs associated with obesity up to 2025 if no action is undertaken is AUD 87.7 billion. For the period 2011–2012 alone, the economic impact of obesity for the

[3] The dataset and sample selection are described in more detail in Section 2.

Australian economy was estimated to be AUD 8.6 billion, including productivity costs, health system costs, and carer costs (AIHW 2017).

Figure 1 – Proportion of mothers with obesity and with overweight
Source: HILDA Release 14.



We contribute to the literature by estimating the intergenerational transmission of BMI and obesity status from mothers to their children in Australia. Earlier works in a non-Australian context have identified a significant relationship between maternal and offspring obesity (e.g., Anderson et al. (2003), Classen (2010), Costa-Font (2013), and Whitaker et al. (2010)). Ours is not the first study on this topic in Australia; that distinction belongs to Burke et al. (2001), who look at a sample of 271 Australian schoolchildren. All these children were around 18 years old and were living in Perth, Western Australia. Our estimates come from a representative household survey of the entire country, covering an age range of 15–19 and comprising of 2,683 unique adolescents.

Furthermore, our larger and more representative sample size allows us to characterize the heterogeneity of the intergenerational transmission of BMI and obesity status over the socioeconomic gradient as well

as the degree of obesity of the mother. While BMI and obesity status has been demonstrated to have an SES gradient in other studies (for Australia, see, e.g., O'Dea et al. 2014), we are aware of only three other studies[4] that demonstrate heterogeneity by SES status in the strength of the intergenerational transmission.[5] Such a gradient—if it exists—has implications for further inequities in health outcomes that we observe in Australia. With respect to the degree of obesity, we are able to distinguish between the mother having obesity but not morbid obesity and the mother having morbid obesity.

The majority of the previous studies use ordinary least squares to estimate the link between parental (typically, maternal) and offspring body mass. Such an approach provides an estimate of the intergenerational persistence of body mass, although the resulting estimates, in isolation, do not allow for a discussion of potential pathways for the persistent relationship. Even if there is ample evidence from the international literature (and in the present manuscript) demonstrating intergenerational persistence, distinguishing between permanent and time-varying mediators remain a central but unresolved concern. Lifestyle choices and other health-related decisions, both by parents and their children, will impact on anthropometric health indicators, such as height and weight, but one's genetic endowment will partially determine those outcomes as well as the strength with which lifestyle decisions will impact on those outcomes (Wardle et al. 2008). For example, the impact of eating a donut on one's weight will vary depending on, *inter alia*, one's basal metabolic rate,[6] which itself is determined partly by genes.

Our strategy is to augment the usual OLS estimates of the intergenerational elasticity of BMI by accounting for adolescent-specific fixed effects, which was also the approach taken by Coneus and Spieß (2012) and Dolton and Xiao (2015). The fixed-effects estimator, in this case, allows us to condition on time-invariant heterogeneity associated with each adolescent, which can be construed—with caveats described below—as controlling for the genetic endowment of adolescents as well as the permanent

[4] Costa-Font and Gil (2013) and Classen and Thompson (2016) used income as an indicator of SES. Dolton and Xiao (2015) looked at heterogeneity across family income, parental occupation, and poverty status. In our estimates below, we look at SES status by quartiles of equivalized household income, but also by maternal educational attainment and a neighborhood-based index of deprivation.
[5] Currie and Moretti (2007) demonstrate an SES gradient in the intergenerational transmission of birth weight; Bhalotra and Rawlings (2013) show the gradient for child health as measured by infant survival.
[6] The basal metabolic rate is the rate of energy expenditure over time (e.g., joule per second).

environmental factors that mediate the transmission of this particular health measure (at least, to the extent that these environmental factors do not change over the period we examine).

Whether the transmission of BMI or obesity status is largely predetermined by genetics or the changing environment has important policy implications. Given the costs associated with obesity—both at the individual and the social levels—mentioned previously, there is a strong policy interest in this space.[7] If, say, obesity status is heavily based on genetic features, one may conclude that resources to improve health mobility across generations may be better spent elsewhere since it may be more difficult to reduce intergenerational persistence in that context. Alternatively, and arguably more equitably, one may prescribe interventions that alleviate the condition itself or mitigate the costs associated with it and its comorbidities. After all, as eloquently pointed out by Classen and Thompson (2016, p. 130), using the example of Goldberger (1979), 'even if poor eyesight is entirely due to genetic characteristics, providing access to eyeglasses would likely be an effective policy intervention.' Indeed—at least from an equity perspective—if transmission of obesity status were entirely predetermined by genetics, this makes the argument for a public policy intervention even stronger, as it is surely fairer to compensate people with a disadvantage that they acquired through no fault of their own other than by being born into it.

Our estimates show that adolescents in Australia whose mothers are with obesity are expected to have an increased probability of being with obesity themselves by 0.094 percentage points. This is non-trivial considering that the adolescent obesity prevalence rate in our sample is 7.70 percent. This relationship is much stronger when we restrict the sample to mothers with morbid obesity, where the percentage-point difference in the probability of being an adolescent with obesity is estimated to be 0.144. In addition, we estimate that the intergenerational elasticity of the body-mass index between mother and adolescent is 0.242, which is consistent with estimates from other countries (Whitaker et al. 2010; Abrevaya

[7] For example, on 16 May 2018, the Senate of the Parliament of Australia established the Select Committee into the obesity epidemic. Its scope is wide-ranging, but it included reporting on the 'effectiveness of existing policies and programs…to improve diets and prevent childhood obesity' and 'evidence-based measures and interventions to prevent and reverse childhood obesity'. Its final report is available from the following link: https://www.aph.gov.au/Parliamentary_Business/Committees/Senate/Obesity_epidemic_in_Australia/Obesity/Final_Report.

and Tang 2011; Murrin et al. 2012; Dolton and Xiao 2015; Dolton and Xiao 2017). When we account for adolescent-specific fixed effects, the estimated elasticity reduces to 0.043. This suggests, albeit crudely, that genetics and the permanent environment may explain much of the observed persistence of body mass and obesity status across generations. Additionally, we demonstrate that the degree of intergenerational persistence exhibits a socioeconomic gradient: disadvantaged households have more persistent health status relative to better-off households. Finally, we show that the intergenerational elasticity of obesity status is much higher (almost double in percentage-point terms) among mothers with morbid obesity relative to mothers with obesity.

2. Data, sample, and variables

Data are drawn from nine waves (2006–2014) of the Household, Income and Labour Dynamics in Australia (HILDA) survey (Summerfield et al. 2015). Information on respondent BMI was only included in HILDA from Wave 6 onwards, necessarily excluding the first five waves from this study. The data from Waves 6–14 are compiled into a long, unbalanced panel of adolescents and mothers with non-missing BMI and control-variable information. As maternal BMI is likely to be inflated during pregnancy, we impose the following correction: if a mother only reports BMI in the wave in which they indicate pregnancy, they are removed from the sample; however, if the mother reports BMI in multiple waves, her BMI that is reported during pregnancy is replaced with her personal mean BMI. Maternal BMI reported during pregnancy is excluded in the calculation of each personalized mean BMI. This results in the removal of four mothers from the sample, and the replacement of 21 maternal BMI observations.

The total number of unique responding adolescents aged 15–19 in the panel was 4,459. However, after excluding all observations which we were unable to match with the mother, this figure decreases to 3,456. Removing adolescents without a reported BMI in any of the nine waves reduces the sample size to 3,090. We drop observations with no information on maternal BMI, leaving a sample of 2,956 adolescents. Adolescents living with step or foster mothers are also removed. Following this adjustment, the final sample size is 2,683 adolescents.

BMI is derived in HILDA by using the self-reported measures of height and body weight obtained from the self-completion questionnaire (Summerfield et al. 2015). Following the international cutoffs by Cole et al. (2000), binary variables for youth and maternal obesity status are generated. This is relevant for adolescents since they are still developing at this stage, and the standard (i.e., adult-appropriate) BMI cutoffs do not take this into account. Since the BMI is calculated using self-reported height and weight measures, it may be subject to a certain amount of measurement error (Cawley et al. 2015). Given our estimation strategy described in Section 3, there is potential for attenuation bias, especially when we account for fixed effects in the model.

There is a clear, positive relationship between mother and offspring BMI. The correlation between mother's and adolescent's BMI in the whole sample is 0.301. From Table 1, which uses only information from the latest wave in the sample (Year 2014), we note that there are 483 mothers who are with overweight (222) or with obesity (261), which is about 58.5 percent of the sample for that year. About 36 percent of them have children who are also with overweight or with obesity. In contrast, a mother within the recommended BMI range is most likely to have an adolescent who is also within the recommended BMI range (73.5 percent) and is least likely to have an adolescent who is with obesity (5.9 percent).

Table 1 – Mother and adolescent BMI category counts and shares

	Mother BMI Category				
Adolescent BMI Category ↓	Underweight BMI < 18.5 (%)	Normal 18.5 ≤ BMI < 25 (%)	Overweight 25 ≤ BMI < 30 (%)	Obese BMI ≥ 30 (%)	Row Sum (% of Total)
Underweight	1 (7.1)	35 (10.8)	14 (6.3)	14 (5.4)	64 (7.8)
Normal	10 (71.4)	238 (73.5)	152 (68.5)	127 (48.7)	527 (64.2)
Overweight	3 (21.4)	32 (9.9)	45 (20.3)	74 (28.4)	154 (18.8)
Obese	0 (0.0)	19 (5.9)	11 (5.0)	46 (17.6)	76 (9.3)
Column Sum (% of Total)	14 (1.7)	324 (39.5)	222 (27.0)	261 (31.8)	821 (100.0)

Source: HILDA Release 14, Year 2014.

A summary of relevant variables by maternal BMI categories is presented in Table 2. Maternal education is condensed from a detailed variable in HILDA that reports the highest level of educational attain-

ment of an individual. The 'tertiary' category consists of all mothers with a postgraduate degree, graduate diploma, bachelor degree, honors degree, advanced diploma, or certificate III/IV. The other category is mothers who completed Year 12 or below. Shares of four marital-status categories (married, divorced, widowed, or single) are also provided by maternal BMI categories. The employment status of the mothers is also provided, including not being part of the labor force.

Table 2 – Summary statistics by maternal BMI categories

	Underweight	Normal	Overweight	Obese
Mother's education				
Tertiary	85.7	65.4	62.1	57.5
Year 12 and below	14.3	34.5	37.8	42.5
Marital status				
Married	85.7	76.5	75.2	76.2
Divorced	14.3	19.4	20.3	18.8
Widowed	0.0	0.6	0.9	1.1
Single	0.0	3.4	3.6	3.8
Mother's employment				
Employed full-time	21.4	35.2	42.8	34.1
Employed part-time	64.3	45.4	37.4	39.8
Unemployed	0.0	1.9	3.2	3.1
Not in labor force	14.3	17.6	16.7	23.0
SEIFA index				
Lowest decile	14.3	6.8	10.4	9.6
2nd decile	0.0	6.8	9.5	10.3
3rd decile	0.0	9.3	5.9	5.4
4th decile	0.0	8.0	6.8	15.7
5th decile	0.0	8.6	14.9	18.8
6th decile	0.0	10.2	9.5	12.6
7th decile	0.0	13.0	7.7	11.1
8th decile	21.4	13.6	10.4	5.0
9th decile	7.1	11.4	13.5	6.9
Highest decile	57.1	12.3	11.7	4.6
Adolescent birth order				
Eldest	14.3	40.4	33.8	31.0
Had older siblings	85.7	59.6	66.2	69.0
Number of siblings	2.38	2.29	2.14	2.12
Equivalized HH Income	$44,808.59	$49,680.90	$48,994.43	$44,217.69

Source: HILDA Release 14, Year 2014. Figures associated with categorical variables are proportions.

The socioeconomic status of households is ranked according to the Socioeconomic Indexes for Areas (SEIFA) deciles of relative socioeconomic advantage and disadvantage. This measure—developed by the Australian Bureau of Statistics (ABS 2006)—is based on the socioeconomic conditions of a neighborhood and ranks relative advantage and disadvantage at an area level. The prevalence of maternal obesity is higher in more socioeconomically disadvantaged areas, which is consistent with evidence

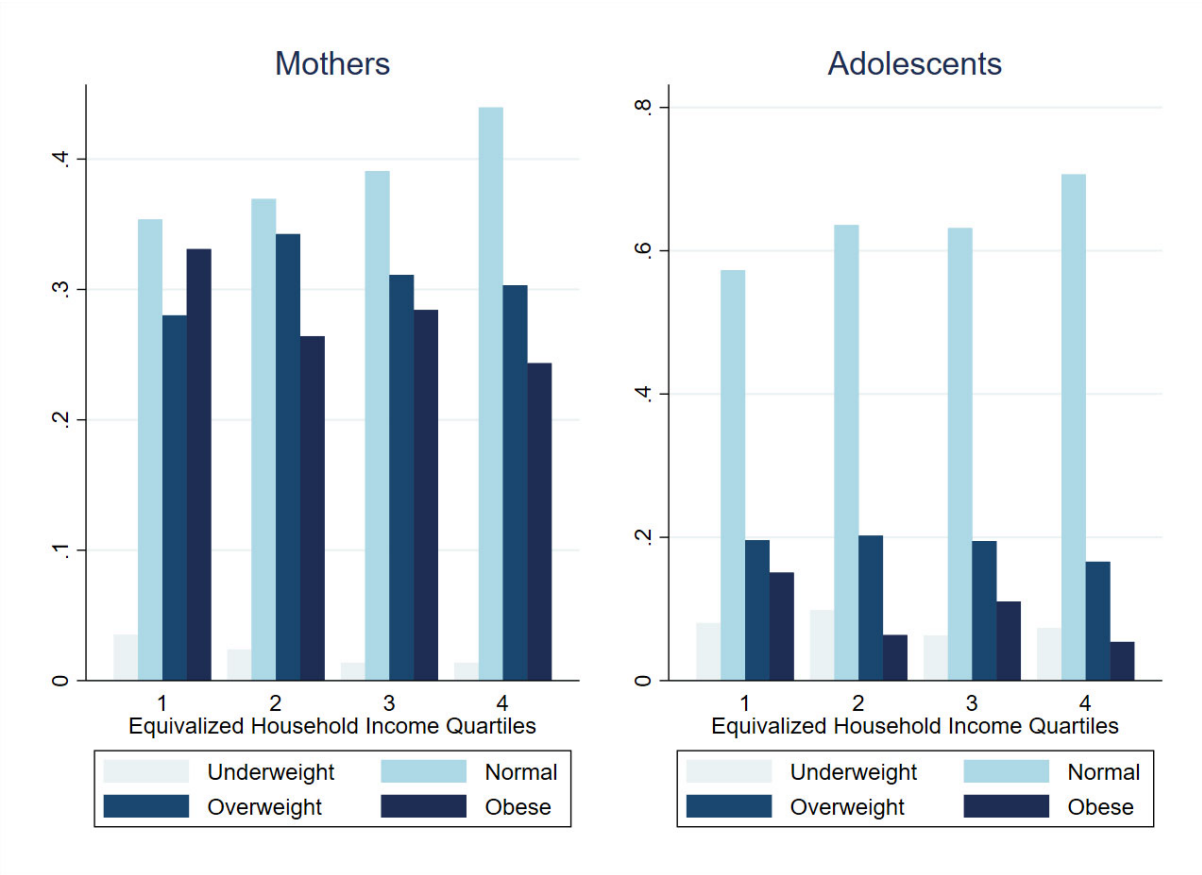
elsewhere in the world (e.g., Walsh and Cullinan (2015) and Wang and Lim (2012)). Mothers with obesity, for example, live predominantly in neighborhoods in the lowest-five deciles of the SEIFA index (59.8 percent).

Total household income does not reflect the fact that larger households require higher levels of income to maintain the same standard of living as smaller households. This makes it difficult to compare the influence of income between households of different sizes and composition (OECD 2013). We use equivalized household income to adjust for heterogeneity in household composition. Although a measure of equivalized income is not provided in HILDA, information is available on the number of household members aged 0–4 years, 5–9 years, and 15+ years, which allows us to calculate the equivalized household income.[8] There is a negative relationship between equivalized household income and maternal BMI category, with the difference in means of the equivalized household income between mothers within the recommended BMI range and mothers with obesity being equal to AUD 5,463.21.

Graphically, we show the prevalence of different weight-status categories by equivalized household income in Figure 2. Adolescent overweight and obesity are associated with lower levels of household income. This relationship is reflected for mothers as well, although the prevalence rates for overweight and obesity are much higher compared to adolescents, who tend to cluster within the recommended BMI range ('normal'): over 60 percent of adolescents in the sample are within the recommended BMI range.

Figure 2 – BMI categories by equivalized household income quartiles

[8] Equivalized household income is calculated by deflating household income by an equivalence factor. To follow Australian convention (ABS 2011), the equivalence factor is calculated according to the 'modified OECD' equivalence scale. This scale allocates points to each household member, with the first adult allocated 1 point, each subsequent person aged 15+ years allotted 0.5 points, and each adolescent under 15 years allocated 0.3 points (OECD 2013). The equivalence factor is the sum of household points. Total household income is divided by the calculated equivalence factor to give equivalized household income (ABS 2011).



Source: HILDA Release 14, Year 2014.

3. Empirical specification

We estimate the intergenerational correlation between BMI from the following baseline equation:

$$Y_{it} = \alpha_1 + \beta_1 X_{it} + u_{it}, \tag{3.1}$$

where Y_{it} is the BMI of adolescent i at time t, X_{it} is the BMI of adolescent i's mother at time t, and u_{it} is the error term. The parameter of interest is β_1, which represents the intergenerational correlation coefficient between maternal and offspring BMI. The intergenerational elasticity is calculated similarly:

$$\log(Y_{it}) = \alpha_2 + \beta_2 \log(X_{it}) + e_{it}, \tag{3.2}$$

where β_2 represents the percentage change in adolescent BMI associated with a one-percent change in mother BMI. In both Equations (3.1) and (3.2), the parameters are estimated via OLS. As pointed out by Classen and Thompson (2016), that BMI transmission would follow a log–log functional form is not informed by theory, so although much of the discussion below concentrates on the estimates of β_2, the estimates of β_1 are also reported to demonstrate robustness to a specific functional-form assumption.

We augment the baseline specifications in Equations (3.1) and (3.2) with vectors of control variables as follows. The first vector contains adolescent-specific control variables (sex, age, number of siblings, and whether adolescent i is the eldest adolescent); the second vector controls for mother-specific attributes (highest educational attainment, age, marital status, and employment status); the third vector contains household-level controls (equivalized household income and SEIFA decile); the final vector contains proxy variables for maternal time preference (smoking status) and indicators for maternal and adolescent emotional distress (whether a close friend of the mother or the adolescent died recently).

The OLS estimates of the intergenerational elasticity of BMI include the influence of both time-invariant and time-varying factors, both observed and unobserved. However, the panel structure of HILDA allows for the removal of time-invariant heterogeneity, such as genetics and environmental factors that do not change in the sample period ('permanent'). We account for this by introducing adolescent-specific fixed effects (FE). These fixed effects include unchanging attitudes toward food, individual-specific risky health behavior, and genetics (Currie 2009; Dolton and Xiao 2015; Classen and Thompson 2016).

The pooled OLS models shed some light on the pathways through which the intergenerational transmission of BMI operates by sequentially adding vectors of control variables and comparing estimates of the parameter of interest. In addition to this, we note that the fixed-effects model removes the influence of all time-invariant characteristics of the adolescent, so these estimates capture only the influence that yearly changes have. Since the pooled OLS estimates do not control for either time-varying or time-invariant heterogeneity while the fixed-effects estimates remove the effect of all permanent heterogeneity, the difference between the two sets of estimates is suggestive of the impact of time-invariant heterogeneity.[9] A more obvious approach would be to compare biological parent–offspring pairs to non-

[9] Note, however, that the sources of identifying variation are different between the OLS and FE estimators. While the OLS estimator uses both the between and the within variation for identification of the parameters, the FE estimator leverages only the within variation. The implication for the FE estimator is that only those with variation in BMI contribute to the estimation. We thank a reviewer for reminding us of this crucial point.

biological parent–offspring pairs (as in Classen and Thompson (2016)); however, in our sample, only 93 such pairs are available such that statistical inference would be largely uninformative.[10]

To estimate whether the strength of the intergenerational elasticity of BMI varies by socioeconomic status, the sample is divided into subsamples by different measures of SES, namely equivalized household income, maternal education, and the SEIFA index. To generate SES cutoffs, household income is divided into quartiles, maternal education is divided into two categories consisting of tertiary (university, certificate, and diploma) and Year 12 and below, and SEIFA deciles are used to create indicators for low-, moderate-, and high-SES neighborhoods. A neighborhood is classified as low SES if it falls in the lowest three deciles of the SEIFA index, moderate SES if it lies between deciles 4–7, and high SES if it is in the top three deciles.

We also estimate a nonlinear probability model where the dependent variable is an indicator for adolescent i being with obesity and the principal independent variable is an indicator for whether the mother of adolescent i is with obesity at time t. The model is estimated via probit, and we report average marginal effects. We emphasize, however, that the estimates are not causal impacts, so 'average marginal effects' should be understood to mean, technically, the change in expected probability for an adolescent to be with obesity with respect to the mother switching from being without to being with obesity. Finally, to examine whether the intergenerational transmission of obesity is stronger at the upper part of the distribution of maternal BMI, we estimate a model where the dependent variable is an indicator for adolescent obesity and the main independent variable is an indicator for the mother having morbid obesity (BMI ≥ 40), and we compare it to a model where the indicator used is whether a mother has obesity but not morbid obesity (40 > BMI ≥ 30). In all model estimations, standard errors are robust to heteroskedasticity and clustering at the individual level. The delta method is used to calculate the standard errors of the average marginal effects resulting from the nonlinear probability models.

[10] Indeed, estimates based on this subsample have extremely large standard errors, and we suppress reporting them here. Classen and Thompson (2016), who used 247 adoptees, encountered the same problem. Subsample analyses in their paper were based on gender, race, and income, with the number of observations ranging from 44 observations to 141.

4. Results

4.1 Intergenerational transmission of BMI

Table 3 shows the OLS estimates of the intergenerational correlation coefficient of mother–offspring BMI. Moving through the columns from left to right corresponds to increasing the set of control variables to account for offspring characteristics (Column (2)), mother characteristics (Column (3)), household characteristics (Column (4)), and proxy variables for maternal time preference and emotional distress of the offspring or mother (Column (5)). The estimated intergenerational correlation of BMI is 0.205 for the full model.[11]

Table 3 – Intergenerational correlation of BMI (OLS)
Dependent variable: Adolescent BMI

	(1)	(2)	(3)	(4)	(5)
Mother BMI	0.212***	0.213***	0.205***	0.202***	0.205***
	(0.015)	(0.015)	(0.015)	(0.015)	(0.015)
Offspring controls		Y	Y	Y	Y
Mother controls			Y	Y	Y
Household controls				Y	Y
Proxy variables					Y
Observations	6690	6690	6690	6690	6690

Notes: Standard errors are robust to heteroskedasticity and clustering at the individual level; they are enclosed in parentheses. A constant is estimated but not displayed here. The offspring control variables include sex, age, number of siblings, and whether the adolescent is the eldest child. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 4 shows the OLS estimates of the intergenerational elasticity of mother–offspring BMI. The elasticity of BMI decreases slightly with the inclusion of control variables, although it remains statistically significant throughout. The estimates range from 0.250 to 0.238, and the estimate in the full model (Column (5)) shows that a 10-percent increase in mother BMI is associated with a 2.42-percent increase in adolescent offspring BMI.

Table 4 – Intergenerational elasticity of BMI (OLS)
Dependent variable: Adolescent log(BMI)

	(1)	(2)	(3)	(4)	(5)
Mother log(BMI)	0.250***	0.250***	0.242***	0.238***	0.242***

[11] Although the discussion below focuses on the subsequent intergenerational elasticity estimates, we report intergenerational correlations here for ease of comparison with other similar studies which also report the estimated correlations as benchmark results. In addition, as mentioned in Section 3, this also demonstrates that the results are robust to a specific function-form assumption.

	(0.016)	(0.016)	(0.016)	(0.017)	(0.017)
Offspring controls		Y	Y	Y	Y
Mother controls			Y	Y	Y
Household controls				Y	Y
Proxy variables					Y
Observations	6690	6690	6690	6690	6690

Notes: Standard errors are robust to heteroskedasticity and clustering at the individual level; they are enclosed in parentheses. A constant is estimated but not displayed here. The offspring control variables include sex, age, number of siblings, and whether the adolescent is the eldest child. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

To account for adolescent-specific, time-invariant heterogeneity, we estimate a fixed-effects model. The results are presented in Table 5. Relative to the results in Table 4, the estimated intergenerational elasticity of BMI is lower with the inclusion of fixed effects although they remain statistically significant at the 10-percent level. For example, comparing the estimates in Column (5), the intergenerational elasticity drops from 0.242 percent to 0.043 percent. This is likely due to the fact that the within variation of BMI is about a third of its overall variation observed in the sample.

This suggests that although genetic and permanent environmental factors (represented by the adolescent fixed effects) account for a large proportion of the intergenerational transmission of BMI in Australia, intergenerational transmission also occurs due to factors that change over time. Time-varying factors account for around 18 percent (0.043 / 0.242) of the intergenerational elasticity. However, as noted earlier (see Footnote 9), it is important to recognize that the sources of identifying variation between the pooled OLS and the FE models are different.[12] In addition, our sample of adolescents aged 15–19 would have experienced time-invariant and time-varying household decisions already in earlier periods in their lifecycle, so this is a cautious interpretation of the difference in estimates between the pooled OLS model with and without fixed effects.

Table 5 – Intergenerational elasticity of BMI (Adolescent FE)
Dependent variable: Adolescent log(BMI)

[12] Nonetheless, if we restrict the sample for the pooled OLS to only those with variations in adolescent and maternal BMI, the estimated results (not reported here) are similar to the results using the original estimation sample. Those without variation in the BMI variables comprise 1,093 observations, which includes adolescents who appear only once in the panel dataset. Strictly speaking, however, dropping those observations from the pooled OLS estimation sample could introduce selection bias since the FE estimator does not technically drop them; those observations simply do not contribute to parameter identification in the FE model.

	(1)	(2)	(3)	(4)	(5)
Mother log(BMI)	0.102***	0.040*	0.039*	0.044*	0.043*
	(0.023)	(0.022)	(0.022)	(0.022)	(0.022)
Offspring controls		Y	Y	Y	Y
Mother controls			Y	Y	Y
Household controls				Y	Y
Proxy variables					Y
Observations	6690	6690	6690	6690	6690

Notes: Standard errors are robust to heteroskedasticity and clustering at the individual level; they are enclosed in parentheses. A constant is estimated but not displayed here. The offspring control variables include age and number of siblings. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Estimates of intergenerational BMI elasticity by socioeconomic status are presented in Table 6. The OLS estimates consistently show a statistically significant estimate that is close to the estimates using the whole estimation sample. The estimates decline in magnitude as SES improves except when using maternal educational attainment as an indicator of SES. However, after controlling for adolescent time-invariant heterogeneity, only the lowest SES subgroups continue to have statistically significant elasticity estimates. In particular, the elasticity remains significant only for households below the 25th quartile based on equivalized household income, households in the lowest three deciles of the SEIFA index, and adolescents whose mothers' highest educational attainment is Year 12 or below.[13] The statistically significant elasticity estimates of the FE models using subsamples are all more than twice the size of the FE model using the entire sample. These results suggest that, although time-invariant characteristics influence both maternal and adolescent BMI regardless of socioeconomic status, it is primarily within low SES households that time-varying environmental influences matter more, and this is what is driving the significant estimate produced by using the overall sample with fixed effects.

Table 6 – Intergenerational elasticity of BMI by SES subgroup

Dependent variable: Adolescent log(BMI)

SES Subsample	Observations	OLS	Adolescent FE	Adolescent Obesity

[13] The lack of statistical significance in the FE models could also be due to the reduced number of observations when doing subsample analyses and the lack of within variation. As mentioned earlier, the within variation of BMI is roughly a third of the overall variation. In addition, attenuation bias may be exacerbated in FE models because of the measurement error inherent in self-reported height and weight measures. We thank a reviewer for reminding us of these points. Nonetheless, it is worthwhile pointing out that despite the lower number of observations and less identifying variation, the statistically significant estimates persist for the very disadvantaged subsamples.

				Prevalence
Equivalized HH income				
< 25th quartile	1674	0.266*** (0.028)	0.089* (0.052)	0.105
25–50th quartile	1673	0.266*** (0.030)	0.060 (0.047)	0.076
50–75th quartile	1671	0.194*** (0.028)	–0.016 (0.055)	0.069
> 75th quartile	1672	0.238*** (0.030)	0.014 (0.044)	0.057
SEIFA				
Low SES	1663	0.300*** (0.019)	0.098** (0.049)	0.117
Moderate SES	2794	0.226*** (0.026)	0.038 (0.032)	0.078
High SES	2233	0.212*** (0.029)	0.013 (0.035)	0.045
Maternal education				
Year 12 and below	2787	0.232*** (0.026)	0.095*** (0.032)	0.084
Tertiary	3903	0.255*** (0.021)	–0.015 (0.030)	0.072

Notes: Standard errors are robust to heteroskedasticity and clustering at the individual level; they are enclosed in parentheses. The full set of control variables as well as a constant are included. The offspring control variables include sex, age, number of siblings, and whether the adolescent is the eldest child. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently. Variables that do not change over time within each cross-sectional unit are omitted from the FE models.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

4.2 Intergenerational transmission of obesity status

This section presents results from probability models where the dependent variable is an indicator for whether the adolescent is with obesity and the principal independent variable of interest is an indicator for having a mother with obesity. Probit models are estimated, but coefficient estimates are suppressed. Instead, average marginal effects are presented, although these 'effects' should be understood to mean a change in expected probability without implying causation. Consistent with earlier presentations in Tables 3–5, we sequentially increase the set of control variables to demonstrate the robustness of the estimated parameters, moving from Columns (1) through to (5).[14]

Table 7 presents average marginal effects of maternal obesity status on the probability of being an adolescent with obesity. As with the pooled OLS and fixed-effects estimates for BMI transmission (Tables

[14] A fixed-effects logit model and a linear probability model with fixed effects are also estimated as analogs of the fixed-effects models for the intergenerational transmission of BMI. Average marginal effects from the logit model with fixed effects and the coefficient estimates from the linear probability model with fixed effects are very similar. However, unlike BMI, there is hardly any variation over time in obesity status for individuals in the sample, so the estimated marginal effects are associated with very large standard errors. Therefore, we refrain from reporting and interpreting them here.

3–5), the average marginal effect that maternal obesity has on adolescent obesity diminishes in magnitude with the introduction of control variables. In the most extensive model in Table 7 (Column (5)), having a mother with obesity increases the likelihood of being an adolescent with obesity by 0.094 percentage points, which should be interpreted relative to the mean adolescent obesity prevalence rate of 7.70 percent in the estimation sample.

Table 7 – Intergenerational transmission of obesity status (probit): Average marginal effects

Dependent variable: Indicator for adolescent obesity

	(1)	(2)	(3)	(4)	(5)
Mother with obesity	0.105***	0.105***	0.096***	0.092***	0.094***
	(0.013)	(0.013)	(0.013)	(0.012)	(0.012)
Offspring controls		Y	Y	Y	Y
Mother controls			Y	Y	Y
Household controls				Y	Y
Proxy variables					Y
Observations	6690	6690	6690	6690	6690

Notes: Standard errors are calculated using the delta method. A constant is estimated but not displayed here. The offspring control variables include age and number of siblings. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.

* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 8 reports the average marginal effects of maternal obesity on the likelihood of adolescent obesity by various SES subsamples. Similar to the intergenerational elasticity of BMI, there is evidence of a relationship between low socioeconomic status and the intergenerational persistence of obesity. The transmission is substantially stronger at the lowest quartile of equivalized household income, at the lowest three deciles of the SEIFA index, and for mothers whose highest educational attainment is Year 12 or below. For example, in low SES neighborhoods, having a mother with obesity is associated with an increase in expected probability of being obese for the adolescent by 0.133 percentage points, which is more than the obesity prevalence rate of 11.70 percent for that subsample.

Table 8 – Intergenerational transmission of obesity status by SES subgroup

Dependent variable: Indicator for adolescent obesity

SES Subsample	Observations	AME of Mother with Obesity	Adolescent Obesity Prevalence
Equivalized HH income			
< 25th quartile	1674	0.132*** (0.023)	0.105
25–50th quartile	1673	0.086***	0.076

		(0.020)	
50–75th quartile	1719	0.072*** (0.018)	0.069
> 75th quartile	1698	0.076*** (0.020)	0.057
SEIFA			
Low SES	1663	0.133*** (0.025)	0.117
Moderate SES	2794	0.104*** (0.018)	0.078
High SES	2206	0.047*** (0.018)	0.045
Maternal education			
Year 12 and below	2787	0.096*** (0.020)	0.084
Tertiary	3903	0.092*** (0.015)	0.072

Notes: Standard errors are calculated using the delta method. The full set of control variables as well as a constant are included. The offspring control variables include age and number of siblings. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

Table 9 shows the average marginal effects of having a mother with obesity and having a mother with morbid obesity on the probability of being an adolescent with obesity. Separate probability models are estimated for two subsamples: mothers whose BMI is within the range [30, 40) and mothers whose BMI is 40 or above. The former is classified as with obesity and the latter as with morbid obesity. As a percentage-point change in the expected probabilities of being an adolescent with obesity, the estimated average marginal effects indicate that having a mother with morbid obesity has an impact that is more than double than that associated with having a mother with obesity but not morbid obesity.

Table 9 – Intergenerational transmission of obesity status by obesity and morbid obesity

Dependent variable: Indicator for adolescent obesity

BMI Category	Average marginal effect
Mother with 40 > BMI ≥ 30	0.067*** (0.011)
Mother with BMI ≥ 40	0.144*** (0.034)
Observations	6690

Notes: Standard errors are calculated using the delta method. The full set of control variables as well as a constant are included. The offspring control variables include age and number of siblings. The mother control variables include her highest educational attainment, age, marital status, and employment status. The household control variables include the equivalized household income and the SEIFA decile. The proxy control variables include the smoking status of the mother and indicators for whether the mother or the offspring had a close friend die recently.
* $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

5. Discussion and conclusion

Our main contributions are in showing the following: intergenerational elasticity estimates of the body-mass index between mother and her adolescent offspring in Australia is consistent with the international

literature. However, when we account for adolescent-specific fixed effects, this elasticity is substantially reduced. This implies that genetics and the permanent environment may account for much of the intergenerational transmission of BMI. Adolescents whose mothers are with obesity are more likely to be with obesity themselves, and this persistence in obesity status is stronger among mothers with morbid obesity relative to mothers with obesity but not morbid obesity. Finally, we show that there is a socio-economic gradient in the transmission of BMI and obesity status, where disadvantaged households demonstrate a stronger persistence. These estimates are based on the Household, Income and Labour Dynamics in Australia survey covering the period 2006–2014. In the following, we discuss these results relative to the existing literature in turn.

Our most extensive model returns an intergenerational elasticity estimate of 0.242 with a standard error of 0.017. Murrin et al. (2012) estimated 0.20 for Ireland, Whitaker et al. (2010) estimated 0.27 for England, Dolton and Xiao (2015) estimated 0.141 for China, and Abrevaya and Tang (2011) estimated a range between 0.22 and 0.25 for the US. More recent work by Dolton and Xiao (2017) showed a range of estimates between 0.117 in Mexico to 0.215 in China, with elasticity estimates for Indonesia, the UK (two different cohorts), the US, and Spain falling in between. Since model specifications are not exactly the same and the periods examined are different (the US sample of Dolton and Xiao (2015), for example, cover the period 1988–1994 while we examine the period 2006–2014), direct comparisons among the various estimates are not free of complications, but it is worth noting that despite the heterogeneity in the estimation sample and the model construction, the estimates are somewhat close to each other. In addition, the only other study using Australian data (Burke et al. 2001) also showed a positive relationship between mothers' and their children's BMI.[15]

The principal question in the literature on the intergenerational transmission of BMI and obesity status is the extent to which genetic transmission from parents to offspring determines the strength of persistence. Classen and Thompson (2016) addresses this question directly by comparing non-biological and

[15] Our intergenerational correlation estimates (e.g., 0.205 with an associated standard error of 0.015 for the most extensive model) are also in line with estimates from elsewhere. Classen (2010) estimated a correlation of 0.35 between mother and child at the same stage of the lifecycle using the National Longitudinal Survey of Youth 1979 in the US. Using the British Household Panel Survey, Brown and Roberts (2013) estimated a correlation of 0.25 for mothers and their children aged 11 to 15 years.

biological parent–offspring pairs. Due to limitations of the dataset, we are unable to do that here in any meaningful way. Instead, our approach derives from Coneus and Spieß (2012) and Dolton and Xiao (2015): we compare pooled OLS estimates to estimates from a model which accounts for adolescent-specific fixed effects. Although imperfect, we believe such a comparison of estimates is still informative with respect to the question of whether genetic features and the permanent environment matter more than time-varying characteristics of the members of the household.

There are, however, caveats that must be made explicit. First, the identifying variation is different between the models. For the model with fixed effects, only those with changes in BMI ('within' variation) contribute to identifying the parameters of interest; the pooled OLS estimates use the 'between' variation as well. Second, our adolescents in the sample are from ages 15 to 19 and, presumably, would have been a product of both time-invariant and time-varying factors in the household from earlier stages of their lives. We are also unable to distinguish between genetics and the permanent environment since adolescent-specific fixed effects would account for both. Keeping these issues in mind, we advance that the estimates are suggestive of the mediating effects of time-invariant and time-varying factors, although not definitive.

When we account for adolescent-specific fixed effects, the estimated elasticity of 0.242 reduces to 0.043. Our interpretation is that this is suggestive of the dominant impact of genetics and the permanent environment in explaining the persistence of BMI across generations. The elasticity estimate in Dolton and Xiao (2015) using data from China decreased from 0.141 to 0.131, which is less dramatic than the reduction that we demonstrate here. However, using weight as the outcome variable, Coneus and Spieß (2012) were unable to find any statistically significant intergenerational transmission between mother and child in the German Socio-Economic Panel when they switch to an FE model. Brown and Roberts (2013) noted that observable characteristics explained only 11.2 percent of the intergenerational correlation in BMI. Broadly speaking, our interpretation is more in line with Classen and Thomp-

son (2016) and the much earlier study of Coate (1983), who showed that the intergenerational transmission BMI and obesity status is primarily a genetic phenomenon.[16] However, we are unable to distinguish this genetic component from permanent environmental factors which could also mediate the intergenerational transmission of BMI. Without better data and an appropriate estimation strategy, it would be extremely speculative to comment on this finer distinction, but we note that this is an avenue for future research.

Our estimate of the intergenerational transmission of obesity status is 0.094 percentage points where the adolescent obesity prevalence rate is 7.70 percent. Costa-Font and Gil (2013) estimate 0.258 percentage points, although they use parental obesity status (an indicator for whether either the father or the mother is obese). Classen (2010) report a marginal effect of 31.7 percent. Similar to the limitations to comparing elasticity estimates in different studies, however, we also note that the sample and period examined here are different, but it would seem that the transmission of obesity status is not as strong in our Australian sample as in studies elsewhere. Additionally, we show that the change in the expected probability of being an adolescent with obesity is much higher if the mother has morbid obesity relative to a mother with obesity but not morbid obesity (0.144 vs. 0.067, respectively). This heterogeneity with respect to the degree of obesity is found elsewhere (e.g., Classen and Hokayem (2005) and Classen (2010)).

Finally, we show a clear pattern of BMI elasticity and obesity-status transmission by various indicators of socioeconomic status. We find that the intergenerational persistence of obesity is most severe among low SES households. This is consistent with Costa-Font and Gil (2013). Classen and Thompson (2016) suggested a similar gradient, although their subsamples had very few observations which precluded meaningful statistical inference. However, Anderson et al. (2003), Whitaker et al. (2010), and Dolton and Xiao (2015) are unable to demonstrate a socioeconomic gradient in the degree of transmission of BMI and obesity status.

[16] In contrast, for other diseases like asthma, chronic headaches, diabetes, hay fever, and chicken pox, as well as an anthropometric measure (namely, height), Thompson (2014) concluded that the intergenerational transmission is largely genetically predetermined.

This divergence in conclusions indicates to us that there is space here to conduct more research with better designs to credibly identify the parameters of interest which would be much more informative for policymakers seeking to target interventions that would affect the group experiencing the most difficulty in breaking the intergenerational transmission of poor health. Existing disparities in health outcomes would be difficult to eliminate if those in disadvantaged circumstances are also the ones experiencing the strongest persistence, which is what our study demonstrates.

To conclude, we comment on the uniqueness of health as a dimension of socioeconomic mobility. Halliday et al. (2018) show that self-rated health status is less persistent than income in the US, and thus, they argue that health and income move independently across generations. The most recent estimate of intergenerational household income elasticity for Australia by Murray et al. (2018) using the same dataset we use here puts it at 0.282 with a standard error of 0.049. Our point estimate of the intergenerational transmission of BMI of 0.242 with a standard error of 0.017 is similar to the uncorrected intergenerational income elasticity of Murray et al. (2018).[17] Considering BMI as a measure of health, this would suggest that intergenerational mobility is about the same in terms of health and income in Australia, although the results are not directly comparable because of the different health outcome variable used in Halliday et al. (2018). This represents an opening for future research to examine other dimensions and measures of the intergenerational transmission of socioeconomic and demographic well-being in Australia and elsewhere.

[17] Murray et al. (2018) estimate a bias-corrected (i.e., adjusting for measurement error in parental income) intergenerational elasticity using household income of 0.409. Our estimate of the intergenerational elasticity of BMI is certainly less than that. However, we refrain from comparing the BMI-based intergenerational elasticity to the income-based, bias-corrected intergenerational elasticity since it is not clear how one might adjust measurement error in self-reported BMI whereas a benchmark correction (at least in the context of intergenerational income mobility) is available for the attenuation bias associated with measurement error in income. Details of this correction are available in Murray et al. (2018).

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