HEALTH ECONOMICS
Health Econ. **24**: 1403–1421 (2015)
Published online 7 August 2014 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/hec.3094

TOO MUCH OF A GOOD THING? EXPLORING THE IMPACT OF WEALTH ON WEIGHT†

NICOLE AU and DAVID W. JOHNSTON*

Centre for Health Economics, Monash University, Clayton, VIC, Australia

SUMMARY

Obesity, like many health conditions, is more prevalent among the socioeconomically disadvantaged. In our data, very poor women are three times more likely to be obese and five times more likely to be severely obese than rich women. Despite this strong correlation, it remains unclear whether higher wealth causes lower obesity. In this paper, we use nationally representative panel data and exogenous wealth shocks (primarily inheritances and lottery wins) to shed light on this issue. Our estimates show that wealth improvements increase weight for women, but not men. This effect differs by initial wealth and weight—an average-sized wealth shock received by initially poor and obese women is estimated to increase weight by almost 10 lb. Importantly, for some females, the effects appear permanent. We also find that a change in diet is the most likely explanation for the weight gain. Overall, the results suggest that additional wealth may exacerbate rather than alleviate weight problems. Copyright © 2014 John Wiley & Sons, Ltd.

Received 18 September 2013; Revised 06 June 2014; Accepted 09 July 2014

KEY WORDS: obesity; BMI; wealth; instrumental variables; panel data

1. INTRODUCTION

Obesity is one of the greatest public health challenges facing industrialised countries and, like many health conditions, is more common among the socioeconomically disadvantaged (Sobal and Stunkard, 1989; McLaren, 2007). In particular, a strong association between low income and obesity has been widely reported, especially among women.[1] Recent estimates suggest that the risk of obesity is 89% higher for individuals in the poorest wealth quintile relative to the wealthiest quintile in the United States (Hajat *et al*., 2010). Similarly, women living in areas of high disadvantage in Australia are significantly more likely to be overweight or obese than women living in areas of low disadvantage, 63.8% versus 47.7% (Australian Bureau of Statistics 2012). The strong link between obesity and numerous chronic illnesses, including diabetes, cardiovascular diseases and cancer, means that such high obesity rates among the poor can intensify health inequalities and lead to further socioeconomic disadvantage. It also puts increasing pressure on health systems and their financers. Estimates by Cawley and Meyerhoefer (2012) suggest that obesity-related illnesses amount to over $209 billion per year in national medical costs in the United States (in 2008 dollars) or about 21% of national health expenditures.

There are a number of possible reasons for the strong association between poverty and obesity. A lack of income and financial resources can limit an individual's access to fresh and nutritious foods (Dinour *et al*., 2007). Energy dense foods, which can now be produced cheaply through advancements in agricultural

*Correspondence to: Centre for Health Economics, Monash University, Building 75, Clayton, VIC 3800, Australia. E-mail: david.johnston@monash.edu

†Supporting information may be found in the online version of this article.

[1]See Chang and Lauderdale (2005), Dinour *et al*. (2007), Frank and Akresh (2013), García Villar and Quintana-Domeque (2009), Hajat *et al*. (2010), Levine (2011), Sarlio-Lähteenkorva and Lahelma (1999) and Zagorsky (2005).

technology and mass food production (Cutler *et al*., 2003), allow individuals on limited budgets to maintain (or exceed) their energy needs at lower cost (Drewnowski, 2004). It is also suggested that financial insecurity can lead to stress-induced eating and weight gain (Smith *et al*., 2009). Socioeconomically disadvantaged neighbourhoods are less likely to have sufficient infrastructure or facilities for safe active transport and exercise, which may explain why the poor are less physically active than the wealthy (Gordon-Larsen *et al*., 2006). Additionally, commercial diets, gym memberships and personal trainers are unattainable for the poor.

However, the correlation between poverty and obesity may not be causal and instead reflect other characteristics of socioeconomic position, for example, educational attainment, occupation, social environment, health knowledge and health expectations. Similarly, unobserved individual characteristics, such as cognitive ability, risk aversion and rate of time preference (Fuchs, 1982; Cutler and Lleras-Muney, 2010), could drive the association between poverty and weight. Early childhood environments, such as birth weight, breastfeeding duration and maternal smoking during pregnancy, are also potential confounders associated with both socioeconomic position and obesity. The correlation between poverty and obesity could also be driven by reverse causality. There is evidence of obesity leading to lower wages and incomes (see, e.g. Cawley, 2004), and this may be due to employment discrimination (Puhl and Brownell, 2001) or reduced productivity resulting from obesity-related health conditions (Cawley, 2000).

Although empirically challenging, it is important to ascertain whether a lack of financial resources actually causes obesity. Such evidence not only builds a better understanding of the underlying determinants of obesity but also has important policy implications. For example, a finding that wealth reduces obesity, particularly amongst the poor, suggests that poverty-alleviating programmes may have the additional benefit of being effective obesity-reducing policies. Such policies are likely to in turn reduce social health inequalities and lower the medical care burden associated with obesity. On the other hand, if wealth has no effect on or increases obesity, policies should target other factors associated with socioeconomic disadvantage (e.g. neighbourhood infrastructure and health knowledge) to reduce the steep obesity gradient.

Despite the clear importance of understanding the determinants of obesity, there are few established facts regarding the causal effects of wealth on weight, with the small literature on adults containing conflicting evidence. For example, Lindahl (2005) finds one-off wealth shocks reduce the probability of overweight, whereas Kim and Ruhm (2012) find no effects on obesity. Schmeiser (2009) finds permanent increases in income raise obesity among women but not men, Wolfe *et al*. (2012) find permanent increases in income reduce obesity, whereas Cawley *et al*. (2010) finds no income effects on obesity. In a recent study concentrating on adolescents, Akee *et al*. (2013) find the overall effect of permanent increases in income is zero but that adolescents from initially poorer households have larger increases in body mass index (BMI) compared with those from wealthier households, suggesting wealth transfers can have strongly heterogeneous effects.[2]

Aside from large differences in the estimated effects of wealth on weight, it remains unclear whether or not wealth affects weight differently by individual characteristics, such as gender, initial wealth and initial weight. Furthermore, there is a lack of evidence on the dynamics of weight change, and as such, it is unclear whether wealth effects are likely to be temporary or permanent.

In this paper, we investigate the effect of wealth on BMI, weight and obesity using data from a nationally representative, longitudinal study of Australian adults. Causal effects are identified using the occurrence of large increases in wealth during the past year (wealth shock), which we argue are primarily inheritances and lottery wins. Our estimates are primarily derived using fixed-effect (FE) regression models, which remove unobserved time-invariant characteristics (such as cognitive ability and risk aversion) that may jointly affect

[2]A possible reason for the different conclusions is that they have focussed on different populations, such as the elderly (Cawley *et al*., 2010; Kim and Ruhm, 2012), adolescents (Akee *et al*., 2013), low-income households (Schmeiser, 2009), lottery players (Lindahl, 2005) and American Indians (Wolfe *et al*., 2012). Another explanation is that different empirical approaches have been used to identify the causal effects. We focus here on studies that investigate the effects of wealth on weight, but related literatures have investigated the effect of wealth on health (e.g. Meer *et al*., 2003; Gardner and Oswald, 2007; Michaud and Van Soest, 2008; Apouey and Clark, 2013) and health behaviours (e.g. Apouey and Clark, 2013; Van Kippersluis and Galama, 2013).

wealth shocks and weight. To approximate the size of the wealth effect in dollar units, we additionally estimate instrumental-variable fixed-effect (IV-FE) models, using the positive wealth shock as an exogenous source of variation in windfall income.

Our study makes a number of key contributions. First, it provides causal effect estimates for a nationally representative sample of adults from Australia, for which currently no evidence exists. Second, we explore how the wealth effect varies by gender, age, initial wealth, initial BMI and personality. Third, we investigate whether wealth shocks have an immediate or delayed effect on BMI and whether the effects are temporary or permanent. Finally, we examine several potential mechanisms through which wealth may impact on BMI.

Our main finding is that wealth shocks have a small positive effect on weight among females, but not males. An average-sized wealth shock of around \$50,000 increases female weight by 725 g (1.6 lb), BMI by 0.9% and obesity by 2.1 percentage points. Importantly, estimation by subgroups suggests that the effects are strongly heterogeneous, with the largest effects for the initially poor and the initially obese. An average-sized wealth shock received by initially poor and obese women is estimated to increases weight by 4.4 kg (9.7 lb) and BMI by 4.7%. Therefore, despite the very large association between socioeconomic disadvantage and obesity, these results suggest that large increases in wealth are more likely to exacerbate than alleviate weight problems. We further find that a wealth shock increases the expenditure on and frequency of meals eaten outside the home but has no significant effect on physical activity levels, alcohol intake, smoking or labour supply. This suggests that a change in diet is a likely explanation for our results. Importantly, all these results are robust, with the estimated effects from numerous robustness models all quantitatively similar.

2. DATA, DEFINITIONS AND DESCRIPTIVE STATISTICS

2.1. The household, income and labour dynamics in Australia survey

We use data from household, income and labour dynamics in Australia (HILDA) survey, a continuing nationally representative longitudinal survey of Australian households. HILDA began in 2001 with 7682 households and 13,969 individuals, and annual data are currently available until 2011. Each year includes detailed information on income, employment and a range of sociodemographic factors. In 2006, HILDA also began collecting annual self-reported information on height and weight. Most data are collected through face-to-face interviews, where information on more personal data including weight, height, health behaviour and household expenditure are collected through a self-completion questionnaire. We primarily use the latest waves of HILDA that contain BMI, although in some specifications we also use information from earlier waves to create indicators for lagged wealth shocks.

The estimation samples include respondents aged 30 to 70 years in 2006. This age range is chosen as these individuals are most likely to experience a wealth shock and are most at risk of unhealthy weight gain. Additionally, we omit respondents who have missing information on the main outcome variables and covariates, respondents who only appear in one wave (due to the exclusive use of fixed-effects models) and respondents with erratic height values and implausible weight values (height and weight restrictions detailed below).

2.2. Weight measures

Our primary outcome variables are log of BMI, weight in kilogrammes and an indicator for obesity (BMI 30). BMI (kg/m^2) is a measure of weight-for-height used to proxy body fat percentage. Although BMI does not distinguish between fat and fat-free mass and may not correspond to the same level of fatness in different individuals (Burkhauser and Cawley, 2008), it is a simple and widely used population index. We take the natural logarithm of the skewed BMI variable so that its distribution is more symmetric. This transformation also allows us to interpret the coefficient estimates as semielasticities.

We derive BMI using self-reported height and weight. To minimise potential measurement error, we apply several data constraints in order to remove observations with implausible BMI values. First, we omit 653

DOI: 10.1002/hec

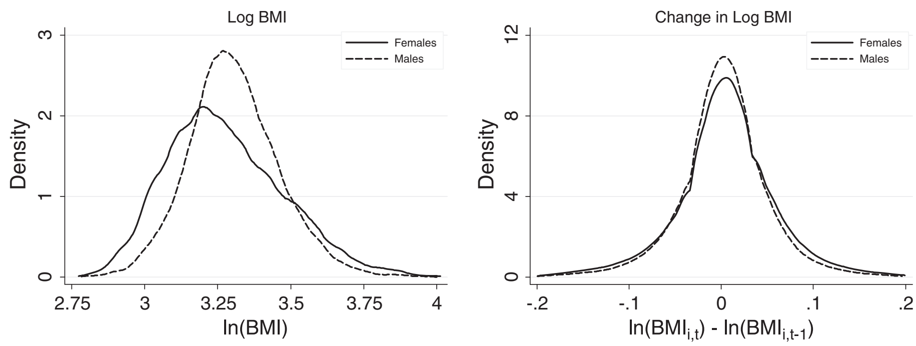

Figure 1. Kernel density estimates of log BMI and change in log BMI by gender

individuals with reported height values that differ by >10 cm across time. Given the 5 year timespan of the data and the adult sample, it is implausible that true height would vary by this extent. Second, we omit 96 individuals with BMI 10 units greater than the average of all other BMI values. In other words, this constraint omits individuals who have implausibly large within-individual year-to-year variation. Third, following Brunello *et al*. (2013), we omit 58 observations with BMI less than 15 or greater than 55. Finally, we omit females who are currently pregnant or who were pregnant in the previous 12 months (855 observations).[3]

The mean BMI for males in our sample is 27.6 kg/m^2, and for females, it is 27.0 kg/m^2. Approximately 25% of males and females in the sample are obese. Figure 1 shows the distribution of log BMI and the annual change in log BMI by gender. Although males are on average heavier, the cross-sectional variation in BMI is clearly larger for females than for males. Females also have greater year-on-year changes in BMI, especially positive changes. Together, these findings show that female weight is less stable than male weight and suggest that female weight may be more reactive to policy or lifestyle changes.

2.3. Wealth measures

Comprehensive wealth surveys were included in HILDA in years 2002, 2006 and 2010. Thus, for these years, we have a measure of household net wealth, which equals household assets (e.g. savings, equity investments, property and collectibles) minus household debt (e.g. property, business and credit card). Mean real household net wealth for our estimation sample equals \$873,448.[4] Estimated nonparametric regressions between log household net wealth and obesity are shown in Figure 2. They show a strong negative relationship for females, with the estimated obesity rate at the 5th and 95th log wealth percentiles (10.1 and 14.9) equalling 32.6 and 16.7, respectively. The relationship is weaker for males with the equivalent obesity rates equalling 32.3 and 19.8.

Limitations of the wealth data are its infrequent collection and its high rate of missing values—for example, for our sample in 2006, around 20% of individuals have missing wealth information. For these reasons, when examining heterogeneity by wealth, we rely on self-reported wealth, which has a high response rate and is available in every wave. HILDA respondents are asked, ‘given your current needs and financial responsibilities, would you say that you and your family are… (1) Prosperous, (2) Very comfortable, (3) Reasonably comfortable, (4) Just getting along, (5) Poor, (6) Very poor?’. The frequencies and mean total real household

[3]Systematic under-reporting of weight and over-reporting of height are also potential problems. As a robustness check, we estimate the regression models using BMI that is adjusted for reporting error using measured and self-reported height and weight from the Australian National Health Survey 2007–2008, following similar methods to Cawley (2004). The estimates using adjusted BMI values are nearly identical to the results using self-reported data, confirming previous findings of a strong correlation between reported and measured BMI (Cawley, 2004; Burton *et al*., 2010).

[4]All wealth and income variables have been deflated to 2010 dollars using the Australian consumer price index.

DOI: 10.1002/hec

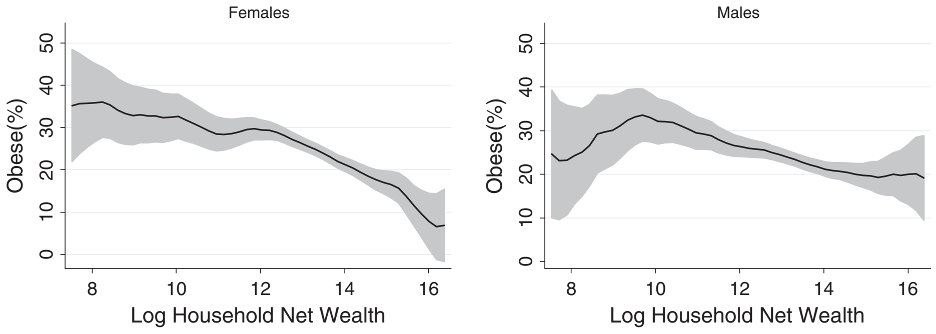

Figure 2. Nonparametric regression estimates of the relationship between obesity and wealth by gender

net wealth (in thousand dollars) for these six responses in our estimation sample are (1) 2.0%, \$3698; (2) 14.5%, \$1655; (3) 52.9%, \$856; (4) 27.18%, \$406; (5) 2.68%, \$193; and (6) 0.7%, \$96.

Figure 3 presents relationships between the self-reported wealth measure and obesity. The relationships are relatively similar to those shown in Figure 2, with a strong negative gradient for females and a weaker negative gradient for males. The statistics presented are particularly noteworthy for females: very poor women are three times more likely to be obese (33% versus 10%) and five times more likely to be severely obese (21% versus 4%) than prosperous women. These obesity gaps demonstrate the importance of understanding the socioeconomic determinants of obesity.

3. EMPIRICAL APPROACH

3.1. Estimating the effect of wealth on weight

The main empirical difficulty with estimating the wealth–weight relationship is the strong possibility of both time-invariant confounding variables (e.g. cognitive ability, risk aversion and time preference) and time-varying confounding variables (e.g. change in employment status). The presence of confounders means that estimates of the effect of wealth on weight from ordinary least squares (OLS) and individual-level fixed-effect (FE) regression models will be biased.

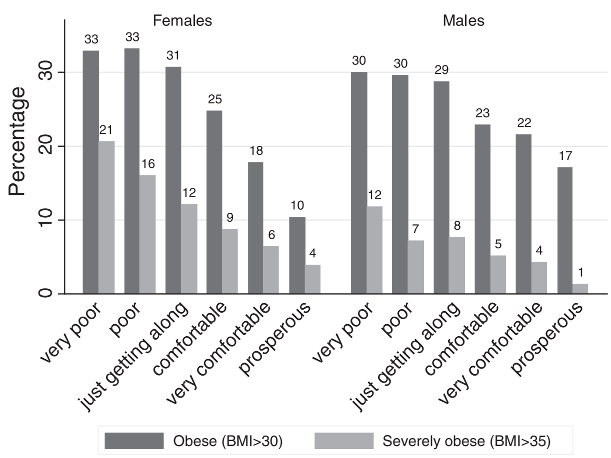

Figure 3. Obesity rates by self-reported wealth and gender

DOI: 10.1002/hec

Our empirical approach is to estimate the effect of a large one-off increase in wealth (a wealth shock), which we believe is uncorrelated with time-varying confounding variables. Specifically, we estimate the following FE regression model:

$$weight_{it} = \alpha_{1i} + \gamma_{1t} + \delta shock_{it} + X_{it}^{'}\beta_1 + \varepsilon_{1it} \tag{1}$$

where $weight_{it}$ is the weight (or equivalently log BMI, obesity) of individual i in year t, α_{1i} is an individual-level fixed-effect that captures all time-invariant individual characteristics, γ_{1t} is a time fixed-effect, $shock_{it}$ is a binary variable that represents the occurrence of a wealth shock during the past year, X_{it} is a vector of characteristics that vary across individuals and time, and ε_{1it} is a random error term. The coefficient δ is the parameter of primary interest and represents the effect of a wealth shock on weight. Equation (1) is analogous to the effect of a lottery win on overweight estimated in Lindahl (2005) and the effect of an inheritance on obesity estimated in Kim and Ruhm (2012), although these studies did not estimate regression models with individual-level fixed-effects.[5]

To quantify the size of the wealth effect in dollar units, we additionally estimate an instrumental-variables fixed-effect (IV-FE) regression model, where income is instrumented by the occurrence of a wealth shock. The first-stage equation of the IV-FE estimation is

$$income_{it} = \alpha_{2i} + \gamma_{2t} + \theta shock_{it} + X_{it}^{'}\beta_2 + \varepsilon_{2it} \tag{2}$$

where $income_{it}$ is annual disposable household income from all sources, including windfall income.[6] The coefficient θ measures the increase in income from a wealth shock. The second stage equation estimates the instrumented effect of income on weight and is given by

$$weight_{it} = \alpha_{3i} + \gamma_{3t} + \lambda\widehat{income}_{it} + X_{it}^{'}\beta_3 + \varepsilon_{3it} \tag{3}$$

where all variables are defined as above. In equation (3), the coefficient λ is the parameter of primary interest and represents the effect of income on weight. The inclusion of the fixed-effects in equations (2) and (3), coupled with our choice of instrumental variable, implies that identification of λ in equation (3) is driven by changes in weight for individuals who have experienced changes in income as a result of a wealth shock. Importantly, the consistency of the IV-FE estimator relies upon the assumption that the wealth shock only affects obesity through its effect on dollars received in the past year and not through other nonpecuniary components of wealth. Given the strictness of this assumption, we predominantly present estimates of the direct impact of receiving a wealth shock (i.e. reduced form equations).

3.2. Description and exogeneity of wealth shocks

In waves 2–11 of HILDA, individuals are asked whether they have experienced during the past year a 'major financial improvement, e.g. won a lottery, received an inheritance'. Individuals are classified as having had a wealth shock if they respond yes to this survey question. Importantly, this variable reflects lottery wins and inheritances, but not other sources of windfall income—the wealth shock variable is not statistically associated with the receipt of income from annuities, pension funds, workers compensation, accident or illness insurance, life insurance, redundancy or severance payouts, gifts from parents or other persons, or company shares, managed funds or property trusts (test statistics reported in Section 4.2).[7] This is important because other

[5]We note that in related literatures on the effect of wealth on health behaviours, some studies have included individual fixed-effects in their specification (e.g. Apouey and Clark, 2013; Van Kippersluis and Galama, 2013).

[6]The sample mean and standard deviation of real annual disposable household income equal \$91,604 and \$79,013, respectively. Household income is used instead of wealth because HILDA measures wealth infrequently (waves 2, 6 and 10) and because it is missing for a high proportion of respondents.

[7]Several previous studies have used lottery wins (Lindahl, 2005; Gardner and Oswald, 2007; Apouey and Clark, 2013; Van Kippersluis and Galama, 2013) and inheritances (Meer *et al*., 2003; Michaud and Van Soest, 2008; Kim and Ruhm, 2012; Van Kippersluis and Galama, 2013) as exogenous sources of variation in wealth.

DOI: 10.1002/hec

sources of windfall income, such as illness insurance and severance payments, may be influenced by time-varying confounding variables.

In our data, the estimated mean change in household disposable income from a wealth shock equals approximately $52,000.

Lindahl (2005) and Kim and Ruhm (2012) demonstrate that lottery wins and inheritances are not randomly distributed across the population and that their occurrence is associated with socioeconomic and demographic factors.[8] Lindahl (2005) overcomes this endogenous selection by restricting the analysis to lottery players. Kim and Ruhm (2012) overcome the nonrandom selection by controlling for a range of characteristics at baseline, including health and BMI, as well as indicators for whether the respondent's mother or father had passed away. Our strategy builds on this approach by controlling for individual-level fixed-effects, which capture all time-invariant individual characteristics (such as cognitive ability, personality, time preference and risk aversion), and by including a range of time-varying covariates (such as education, marital status and death of a relative). To capture additional unobserved time-varying heterogeneity, robustness models also include individual-specific linear time trends ($\pi_i t$) and lagged weight ($weight_{it-1}$) as covariates.

On the basis of this modelling strategy, we present in Section 4.2 a series of robustness and validity tests that suggest our instrument is exogenous. In particular, the tests reveal two important results. First, the probability of a wealth shock is unrelated to past BMI, indicating that anticipation effects are nonexistent and that endogenous changes in lifestyle are unrelated to future shocks. Second, estimation results are robust to all observed contemporaneous events that may be correlated with wealth shocks and weight. Specifically, estimation results are quantitatively similar from models that exclude individuals who have experienced the following life events: a serious injury or illness to themselves, a family member or relative; the death of a family member, relative or friend; retirement from the workforce; being fired or made redundant; changing jobs; and receiving a promotion at work.

4. MAIN RESULTS

The main results are arranged as follows. First, we discuss wealth effect estimates from FE and IV-FE models of BMI, weight and obesity. Second, we present results from a series of robustness models and exogeneity tests. Third, we investigate the possibility of heterogeneity in the effect of wealth. Finally, we present results from dynamic specifications that test for the timing and length of the wealth effect.

4.1. Effects of wealth on BMI and weight

Table I presents estimates from FE and IV-FE models of log BMI, weight (kg) and obesity (BMI > 30) by gender. Only the coefficient estimates for the wealth shock and log annual household income variable are shown, but each model also includes time-varying characteristics: age, age squared, marital status (married, not married but cohabitating, divorced or separated), number of children, highest educational attainment (high school graduate, diploma or trade certificate, university degree) and year dummy variables. Table A1 in the supporting information contains definitions of these covariates, and Table A3 presents the estimated effect of the covariates on log BMI.

Columns (1)–(3) present estimated effects of a positive wealth shock. Under the assumption that the wealth shock variable is exogenous, the estimates are consistent and indicate the extent to which a major inheritance or lottery win will affect an individual's weight. For females, the estimates suggest that a wealth shock in the past

[8]This is also true in our sample. Although age, gender, marital status and number of children are not significantly different between people who report and do not report our wealth shock variable, there is a significant difference in terms of the propensity to have a university degree. The university educated are 0.6 percentage points more likely to report a wealth shock.

Table I. Fixed-effect and instrumental-variable regression models of BMI, weight and obesity

	Fixed-effect models			Instrumental-variable models			
	Log BMI	Weight	Obese	Income	Log BMI	Weight	Obese
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Males							
Wealth shock	−0.002	−0.188	0.007	0.556***	—	—	—
	(0.002)	(0.206)	(0.009)	(0.078)			
Income	—	—	—	—	−0.003	−0.338	0.013
					(0.004)	(0.373)	(0.016)
Females							
Wealth shock	0.009***	0.725***	0.021**	0.513***	—	—	—
	(0.003)	(0.212)	(0.009)	(0.071)			
Income	—	—	—	—	0.018***	1.412***	0.042**
					(0.006)	(0.474)	(0.020)

Figures are coefficient estimates from fixed-effect (FE) and instrumental-variables fixed-effect (IV-FE) regression models. Wealth shock is a binary indicator for a major improvement in financial position during the past 12 months. Income is annual household income in $100,000 units. Regression models also contain the covariates described in Table A1 in the supporting information and year dummy variables. Standard errors clustered at the individual level are shown in parentheses. Male sample size equals 14757 and female sample size equals 15352. The first-stage F-statistic corresponding to models (5)–(7) equals 50.36 for males and 51.51 for females.
**significant at 0.05 level; ***significant 0.01 level.

12 months increases BMI by 0.9%, weight by 725 g (1.6 lb) and obesity by 2.1 percentage points.[9] In contrast, a wealth shock does not have any effect on weight for males. This result is compatible with our earlier descriptive finding that males experience smaller within-individual changes in weight than females (see Figure 1).

Despite the very large association between socioeconomic disadvantage and obesity for females (see Figures 2 and 3), these results suggest that large increases in wealth are likely to worsen rather than reduce weight problems. This finding is not surprising; although additional wealth increases the affordability of healthier goods and services, it does not provide easier access to important socioeconomic elements, such as skills, preferences, peers and environment, that may encourage healthier lifestyles. It is unclear why we find a significant wealth effect for females, but not males. It may relate to gender differences in preferences in spending patterns, such as on food and impulse purchases (Pahl, 1990; Wood, 1998), and the greater susceptibility of women to gain weight in response to emotional states and increases in caloric intake (Chiriboga *et al.*, 2008; Hays and Roberts, 2008).

In the right-hand side panel of Table I (columns (4)–(7)), we present estimates from instrumental-variable fixed-effects (IV-FE) models. This exercise allows us to approximate the size of the wealth effect on obesity in dollar units. The limitation of this approach is that it ignores the potential effect of other inherited assets (e.g. property) that have not been liquidated and therefore relies upon the assumption that wealth shocks only affect obesity through its effect on dollars received in the past year (including from reported windfall income). Column (4) reports the effect of a wealth shock on household annual disposable income or, in other words, the first-stage equation from the IV-FE model.[10] The estimates suggest that a wealth shock increases household income on average by $51,300 for females and $55,600 for males, which is about 55% of average household income (gender difference in first-stage effects is not statistically significant; t-statistic = 0.40). As expected, the estimates are statistically significant with F-statistics equal to 50.36 for males and 51.51 for females. In columns (5)–(7), we present the IV-FE estimates. The female IV-FE estimates suggest a $100,000 increase in wealth increases BMI by 1.8%, weight by 1.4 kg (3 lb) and obesity by 4.2 percentage points.

[9]Throughout the paper, we provide estimates from linear models of obesity. Importantly, all such results are comparable to estimates from (conditional) logit models of obesity. For example, the male and female logit estimates corresponding to column 3 in Table 1 have p-values equal to 0.394 and 0.035. The limitation of conditional logit models is that there is a large reduction in observations due to the omission of individuals without time variation in obesity, and marginal effects cannot be calculated without additional untestable assumptions.
[10]The income measure includes income from all sources, such as salaries and wages, investment income, business income, government welfare and windfall income, and is deflated to 2010 prices using the consumer price index.

As discussed in Section 2, because of data limitations, we are unable to conduct an IV-FE analysis using household wealth (rather than income). However, we have experimented with using self-reported wealth. In particular, we reestimated the IV-FE models in Table I using a binary variable that indicates that the household is 'just getting along', 'poor' or 'very poor' (see Section 2 for a discussion of the self-reported wealth variable). The first-stage estimates suggest that a wealth shock decreases this self-reported 'poverty' indicator by 8.5 percentage points (F-statistic = 33.9). The female second-stage IV-FE estimates suggest that 'poverty' reduces BMI by 10.5% (t-statistic = -2.98), weight by 8.4 kg (t-statistic = -3.07) and the probability of obesity by 22 percentage points (t-statistic = -1.96).

The FE and IV-FE estimates suggest that wealth has a significantly positive impact on female weight. This result is very different to results from 'standard' ordinary least squares (OLS) models, in which the estimated effects of measured and self-reported wealth are significantly negative (see Table A2 in the supporting information). Equivalent estimated effects from FE models are also much lower than the FE estimates in Table I (see Table A2 in the supporting information). One explanation for why OLS and FE estimates of measured and self-reported wealth are lower than the Table I estimates is that BMI has a direct negative effect on employment and income for females. Supporting this explanation are studies by Cawley (2004), Greve (2008) and Johar and Katayama (2011), which find a significant negative BMI effect on female wages (but less so for males). Another plausible explanation is that classical measurement error in wealth attenuates FE estimates.

Our findings are different to that of Lindahl (2005) and Kim and Ruhm (2012) who find that lottery wins have a negative effect on overweight and inheritances have an insignificant effect on obesity, respectively. However, neither study examined the effects separately by gender or included individual-level fixed-effects.[11] Another reason for the differences in results is that the sample populations are different in each study. Lindahl (2005) focusses on lottery players in Sweden, whereas Kim and Ruhm (2012) focus on older Americans born from 1931 to 1941. These populations are likely to differ in many ways from the general adult population in Australia, including in their behavioural responses to wealth shocks.

In order to gain a greater perspective on the importance of our estimated weight effects, we also estimate models for individual's weight satisfaction (results available upon request). In 2007 and 2009, individuals are asked 'how satisfied are you with your current weight?'. For females, it is estimated that a wealth shock reduces the probability of feeling satisfied or very satisfied by 11 percentage points (t-statistic = -3.76). These estimates are large when compared to a mean satisfaction level of 30% and therefore suggest that the relatively modest weight effects in Table I are causing large decreases in weight satisfaction. This is potentially of concern given weight dissatisfaction has important repercussions for self-esteem and mental well-being (Stunkard and Wadden, 1992; Luppino *et al*., 2010).[12]

4.2. Robustness models and exogeneity tests

The estimates presented in Table I rely on the assumption that conditional on the individual-level fixed-effect and the time-varying characteristics, wealth shocks are exogenously determined. This assumption is invalid if time-varying unobservables determine both weight and wealth shocks. For example, suppose some random event altered an individual's level of risk aversion and that this change caused an increase in the likelihood of obesity (e.g. due to changes in lifestyle) and a wealth shock (e.g. due to more gambling). Another potential issue is that some types of wealth shocks, such as inheritances, may be anticipated. Anticipated wealth shocks may lead to changes in lifestyle behaviour and weight before receipt of the additional wealth. It is difficult to directly test for such scenarios; however, if our wealth shock is exogenous, then we would not expect weight in

[11]If we pool genders and estimate a cross-sectional (OLS) model we find that the wealth shock has significantly positive effects that are larger than the reported FE effects. Log BMI is estimated to increase by 1.4% and weight is estimated to increase by 1.5 kg. These positive OLS effects are driven by significantly positive effects for both men and women.

[12]Despite the large observed increases in weight dissatisfaction, it is generally found that wealth shocks (such as lottery wins and inheritances) have overall positive effects on mental well-being (See for example, Gardner and Oswald, 2007; Au and Johnston, 2013).

DOI: 10.1002/hec

period t to be associated with a wealth shock in period $t+1$ (a test in the spirit of Granger (1969)). We test for such an association and find that current weight (measured by BMI and BMI categories) is not significantly associated with wealth shocks in the following year (t-statistics for log BMI, overweight, obesity and severe obesity equal -0.06, 0.21, -0.04 and -0.31, respectively). This suggests that anticipation effects are nonexistent and endogenous changes in lifestyle are unrelated to future wealth shocks.

Our models are also misspecified if the wealth shock affects weight through pathways other than an increase in wealth. The likelihood of nonwealth pathways depends upon the types of wealth shocks individuals are reporting. Given the two explicit examples provided with the survey question ('won lottery' and 'received an inheritance'), we expect that a large proportion of affirmative responses constitute lottery wins and inheritances. However, it is possible individuals reported other types of wealth shocks. In a separate part of the HILDA questionnaire, respondents are asked to report whether, in the past year, they received additional income from a number of listed sources, and therefore, we are able to test the association between these sources and the wealth shock variable. We find that the reporting of a wealth shock is not significantly related to the receipt of the following: annuity or life insurance or pension fund payments (t-statistic $= -0.57$); workers compensation or accident/illness insurance (t-statistic $= 0.78$); redundancy or severance payments (t-statistic $= 1.27$); gifts from parents (t-statistic $= -0.32$); gifts from other persons (t-statistic $= 0.45$); or income or dividends from company shares, managed funds or property trusts (t-statistic $= -0.44$). In contrast, the wealth shock variable predicts a 32 percentage point increase in the reporting of inheritance / bequests (t-statistic $= 14.33$).

It is difficult to imagine how a lottery win could directly affect weight other than through an increase in wealth. However, a potential alternative pathway associated with inheritances is grief: an individual that is grieving the death of a relative or friend may over-eat regardless of the additional wealth.[13] In Table II, we test the robustness of our results to this type of alternative pathway by reestimating our primary models with subsamples who have not experienced the death of a family member, relative or friend (a death event) and by also reestimating our models with indicators for these events as additional covariates. If the results reported in Table I are driven predominantly by grief, rather than from wealth changes, the estimated effects should reduce in magnitude when the regressions are estimated without individuals who are experiencing death-related grief (or when these variables are controlled for in the full sample). In Table II, we also test the robustness of our results to two additional alternative pathways: serious injury or illness to self, family member or relative (health event); and retirement from the workforce, being fired or made redundant, changing jobs or receiving a promotion at work (work event); Table A1 in the supporting information contains definitions of these event variables. The preceding analysis suggests that the wealth shock variable is not associated with income improvements that are correlated with health or work events (e.g. illness insurance and severance payments); however, we present these additional tests to provide further evidence of robustness.

The results presented in Table II show that the results in Table I are robust. For example, when we include covariates representing health, death and work events for the full female sample, the wealth effect on log BMI equals 0.009, which is identical to the estimate without controlling for these events. When we omit any females with a health, death or work event, the wealth effect equals 0.016, which is slightly larger than the estimates for the full sample.

Our third robustness check involves using a model that captures a greater proportion of the time-varying unobserved heterogeneity in weight. All the models presented in Tables I and II contain individual-level fixed-effects and a number of time-varying observable characteristics. However, we imperfectly measure observed determinants of weight gain (e.g. educational attainment), and we completely omit others (e.g. health knowledge), implying the existence of time-varying unobserved heterogeneity. Capturing time-varying unobserved heterogeneity is difficult, but given the panel nature of our data, we can augment equation (1) with individual-level linear time trends ($\pi_i t$), which capture heterogeneity that evolves linearly:

[13]It is also possible that the death of a biological relative provides individuals with information about their remaining longevity and consequently causes individuals to change their (health related) lifestyles. Presumably however, this pathway would induce positive lifestyle changes, which would generate a negative bias and make our estimated effects too small.

DOI: 10.1002/hec

Table II. Estimated effects of a wealth shock using robustness samples and model specifications

	Males		Females	
	Sample size	Log BMI effect	Sample size	Log BMI effect
(1) Full sample	14,757	−0.002 (0.002)	15,352	0.009*** (0.003)
(2) Omit any with a death event	11,754	−0.001 (0.002)	12,014	0.011*** (0.004)
(3) Omit any with a health event	11,799	−0.002 (0.003)	11,559	0.007** (0.003)
(4) Omit any with a work event	11,965	0.002 (0.003)	12,821	0.009*** (0.003)
(5) Omit any with a health, death or work event	7940	0.001 (0.004)	7959	0.016*** (0.006)
(6) Full sample with health, death and work covariates	14,757	−0.002 (0.002)	15,352	0.009*** (0.003)

Figures are coefficient estimates from fixed-effect (FE) regression models. Regression models also contain the covariates described in Table A1 in the supporting information and year dummy variables. Standard errors clustered at the individual level are shown in parentheses. Death event includes the death of a family member, relative or friend. Health event includes a serious injury or illness to self, family member or relative. Work event includes retirement from the workforce, being fired or made redundant, changing jobs, or receiving a promotion at work. Model (6) includes nine additional covariates capturing health, death and work events (see Table A1 in the supporting information).
**significant at 0.05 level; ***significant at 0.01 level.

$$weight_{it} = \alpha_{4i} + \pi_i t + \gamma_{4t} + \delta shock_{it} + X_{it}\beta_4 + \varepsilon_{4it} \tag{4}$$

The results from estimating FE and IV-FE models with individual-level trend terms again confirm the robustness of our main estimates. The estimated FE wealth shock effect and the estimated IV-FE income effect for females equals 0.008 and 0.017, respectively, which are very similar to those presented in Table I (0.009 and 0.018). Full estimation results of equation (4) are available upon request.

Finally, we estimate a FE model that includes a lag of weight as an explanatory variable. This model constitutes an alternative approach for capturing time-varying unobserved heterogeneity in weight. The dynamic FE model is specified as

$$weight_{it} = \alpha_{5i} + \gamma_{5t} + \rho weight_{it-1} + \delta_5 shock_{it} + X_{it}\beta_5 + \varepsilon_{5it} \tag{5}$$

and is estimated using a system GMM specification that contains the weight equation in levels and differences (Blundell and Bond, 1998). In this method, lagged first differences are used as instruments for the equation in levels and lagged levels are used as instruments for the equation in first differences. The key assumptions underlying this method are no autocorrelation in the idiosyncratic error ε_{5it}, and no correlation between the fixed effects and the first difference of the first observation of weight. The estimated FE wealth shock effect from equation (5) on log BMI equals 0.010 (p-value = 0.020), and the estimated lagged log BMI effect equals 0.438 (p-value < 0.001). Full estimation results of equation (5) are available upon request.

4.3. Heterogeneous wealth effects

The relationship between wealth and weight will differ depending upon how wealth is used. For example, additional wealth may be used on unhealthy vices such as drinking or on healthy food that is fresh and nutritious, but also more expensive. Moreover, such preferences may systematically differ by individual characteristics. In this section, we reestimate FE regression models by age, locus of control, initial wealth and initial BMI to test whether wealth has larger effects on weight for certain individuals.

First, we examine differences in wealth effects by age and locus of control. Metabolism tends to decrease as people age (St-Onge and Gallagher, 2010), implying that an equivalent increase in calorie intake may have different weight effects for people of different age groups. Cobb-Clark *et al.* (2012) find that individuals with

DOI: 10.1002/hec

Table III. Estimated effects of a wealth shock for females by initial wealth and BMI

	Sample	Log BMI		Weight	
Initial self-reported wealth					
(1) Poor	4979	0.012**	(0.005)	1.068***	(0.410)
(2) Comfortable	7756	0.010**	(0.004)	0.768**	(0.310)
(3) Prosperous	2597	0.003	(0.005)	0.137	(0.372)
Initial BMI					
(4) BMI ≤ 22.5	3648	0.014***	(0.005)	0.911***	(0.316)
(5) 22.5 < BMI ≤ 25	3420	0.006	(0.005)	0.412	(0.359)
(6) 25 < BMI ≤ 30	4622	0.003	(0.005)	0.303	(0.393)
(7) BMI > 30	3662	0.013**	(0.006)	1.262**	(0.580)
Interaction					
(8) Poor & BMI > 30	1387	0.047***	(0.012)	4.352***	(1.085)

Figures are coefficient estimates from fixed-effect (FE) regression models. Regression models also contain the covariates described in Table A1 in the supporting information and year dummy variables. Standard errors clustered at the individual level are shown in parentheses. Subsamples constructed using wealth and BMI levels in 2006, and models estimated using BMI data from 2007 to 2011. Total sample size for analyses by self-reported wealth varies slightly from Table II due to missing information on initial self-reported wealth.
**significant at 0.05 level; ***significant at 0.01 level.

an internal locus of control are more likely to eat well and exercise regularly, suggesting different preferences for spending windfall income. We reestimate the regression models presented in Table I for the following: (i) younger (30–49 years) and older (50–70 years) individuals; and (ii) individuals with external and internal locus of control.[14] The results suggest that the effect of wealth on weight is not heterogeneous with respect to age or locus of control (full results available upon request).

Second, we estimate whether wealth shocks differ by initial wealth. Preferences for how to spend windfall income are likely to be different for individuals from poorer households than those from wealthier households. This may be due to differences in returns on investment in health-promoting goods and services (Grossman, 1972), differences in the marginal utility experienced from 'bads', such as high-calorie food and alcohol, and differences in individual traits, such as cognitive ability, risk aversion and rate of time preference. Individuals are classified poor (very poor, poor, just getting along), comfortable (reasonably comfortable) or prosperous (very comfortable, prosperous), based on self-reported wealth in 2006, and the regression models are estimated using data from years 2007–2011.

The results in Table III show that wealth shocks have a significantly positive effect on weight for the initially poor (log BMI effect equals 0.012) and initially comfortable (log BMI effect equals 0.010), and no effect for the initially prosperous.[15] The strong positive effect for females from initially poor households is supportive of the positive income effect on BMI found by Akee *et al*. (2013) among adolescents from low-income American Indian households and by Schmeiser (2009) among low-income women who are eligible for the earned income tax credit. Both of these studies examined the effect of a permanent (rather than one-off) increase in household income but nevertheless have findings in accordance with ours.

Third, we examine how the wealth effects differ depending upon the part of the unconditional BMI distribution the individual is from (prior to the wealth shock). Obese individuals are likely to have a greater propensity to gain weight (for genetic or behavioural reasons), and therefore, our *a priori* expectation is that wealth effects will be larger for obese women. Individuals are classified as low normal weight (BMI < 22.5), high normal weight (22.5 ≤ BMI < 25), overweight (25 ≤ BMI <30) and obese (BMI ≥30) according to their 2006 BMI values, and the regression models are estimated using data from years 2007–2011. The results

[14]In 2003, 2004 and 2007, the HILDA survey included seven questions measuring locus of control (LOC). For example, 'I have little control over the things that happen to me' and 'there is little I can do to change many of the important things in my life'. We create a LOC index that is increasing in external control tendencies by summing the responses, which vary from (1) strongly disagree to (7) strongly agree (some questions reverse coded). Individuals are classified as 'external' if they have an above median index value.

[15]Estimated effects for males are not significantly different from zero for any initial wealth group or, for any of the age, locus of control and BMI subgroups.

DOI: 10.1002/hec

presented in Table III indicate that the effects are particularly large for those who were obese in year 2006. For this subgroup, a wealth shock is estimated to increase BMI by 1.3% and weight by about 1.3 kg (2.8 lb). Given the risk of mortality and morbidity increases as the degree of obesity increases (Bray, 2004), these estimates suggest that wealth shocks for obese individuals may have serious health consequences. The effects are also significantly positive for females with initial BMI < 22.5. This effect may be driven by women who were thin because of poverty (i.e. budget constrained), in which case the weight gain may represent a health improvement, or alternatively by women who felt they could 'afford' to celebrate their windfall income gain in unhealthy ways (e.g. through excessive eating and drinking).[16]

Finally, we estimated the effect of a wealth shock for females who in 2006 were both poor and obese. Poor and obese individuals registered the largest positive effects individually and also constitute groups that are most policy relevant with respect to obesity and food insecurity policies. The estimated effect of a wealth shock for this small subgroup is estimated to increase BMI by 4.7% and weight by about 4.4 kg (9.7 lb). Thus, the observed positive weight gains shown in Tables I–III seem to be largely driven by the effects on a particularly vulnerable and at-risk group of women.[17]

4.4. Dynamic wealth effects

A limitation of the FE estimates presented in Tables I–III is that the contemporaneous wealth shock variable does not allow flexibility in the way wealth can influence weight over time. In this subsection, we present results from a dynamic specification that tests whether the positive effects are immediate or delayed, and temporary or permanent. Essentially, we estimate FE regression models that include a number of lagged wealth shock variables:

$$weight_{it} = \alpha_{4i} + \gamma_{4t} + \lambda_0 W_{it} + \lambda_1 W_{it-1} + \lambda_2 W_{it-2} + \lambda_3 W_{it-3+} + X'_{it}\beta_4 + \varepsilon_{4it} \tag{6}$$

where the four W_{it} terms are dummy variables indicating that a wealth shock occurred in the past 12 months (W_{it}), 13–24 months (W_{it-1}), 25–36 months (W_{it-2}) and 37+ months (W_{it-3+}). These variables allow for the effects to change across time.[18]

The choice of wealth shock dummy variables and the inclusion of the individual-level fixed-effect α_{4i} imply that we are comparing weight after the wealth shock with the weight of the same individual in the years before the shock occurred, which we interpret as an individual's baseline weight. For example, suppose for the log BMI outcome variable, $\lambda_0 = \lambda_1 = \lambda_2 = \lambda_3 = 0.05$. This result would imply that BMI increased by 5% relative to baseline levels immediately following a wealth shock and that this increase was permanent, which would constitute a particularly negative health effect. Conversely if $\lambda_0 = 0$, $\lambda_1 = 0.05$ and $\lambda_2 = \lambda_3 = 0$, this result would imply the effect was delayed, with BMI increasing by 5% 13–24 months following a wealth shock and, temporarily, with BMI reverting back to baseline levels in subsequent years.

Table IV contains estimates of $\lambda_0, \lambda_1, \lambda_2$ and λ_3 for all females, females who were initially poor or comfortable and females who were initially obese. These two subgroups were chosen because the results in Table III suggest the effects are particularly large for these women. The results for all females in columns (1) and (2) indicate that BMI increases by 1.2% and weight increases by 912 g (2.01 lb) in 1–12 months after a wealth shock. In following years, the effects are all positive, but roughly one third to one half the size and are not

[16]In columns 1 and 2 of Table A4 in the supporting information, we additionally present estimated income effects from IV-FE models for poor/comfortable females and obese females. These IV-FE models correspond to those presented in Table 1 and approximate the wealth effects shown in Table 3 in terms of dollar units. For example, the results suggest a $100,000 increase in wealth increases the weight of obese females by 1.7 kg.

[17]The differences in estimates across groups in Table 3 are not driven by differences in the value of the wealth shock. In contrast, the measured income component of the wealth shock is significantly smaller for the initially poor: estimated disposable income effect of a wealth shock equals about $29,800 for the poor group, relative to $65,600 for the comfortable group. Correspondingly, the IV-FE log BMI estimates for poor and comfortable groups equal 3.9% and 1.5%, respectively.

[18]The correlations between the four wealth shock dummy variables are small. The estimated correlations range from 0.029 to 0.129.

DOI: 10.1002/hec

Table IV. Dynamic wealth shock effects for females

	All		Poor/Comfortable		BMI > 30	
	Log BMI	Weight	Log BMI	Weight	Log BMI	Weight
Income shock	(1)	(2)	(3)	(4)	(5)	(6)
0–12 months	0.012***	0.912***	0.014***	1.094***	0.019***	1.613***
	(0.003)	(0.245)	(0.004)	(0.289)	(0.007)	(0.605)
13–24 months	0.004	0.380	0.006	0.516*	0.018**	1.580**
	(0.003)	(0.253)	(0.004)	(0.304)	(0.007)	(0.653)
25–36 months	0.004	0.277	0.004	0.207	0.001	0.013
	(0.003)	(0.246)	(0.004)	(0.308)	(0.009)	(0.755)
37+ months	0.006	0.488	0.005	0.467	0.022**	2.061**
	(0.004)	(0.309)	(0.005)	(0.380)	(0.010)	(0.938)
Sample size	13395	13395	11064	11064	3203	3203

Figures are coefficient estimates from fixed-effect (FE) regression models. Regression models also contain the covariates described in Table A1 in the supporting information and year dummy variables. Standard errors clustered at the individual level are shown in parentheses. Subsamples constructed using wealth and BMI levels in 2006, and models estimated using BMI data from 2007 to 2011. Sample sizes in columns (3) and (4) differ slightly from the total sample size in Table III due to missing data on the lagged wealth shock variable.
*Significant at 0.10 level; **significant at 0.05 level; ***significant at 0.01 level.

statistically significant. Therefore, on average, it appears the positive wealth effects are immediate and mostly temporary.[19] Perhaps individuals 'celebrate' their good fortune before resuming their normal lifestyles. The results for the two subgroups suggest that the effects are somewhat longer lasting. For the poor/comfortable group, weight remains significantly higher 13–24 months after a wealth shock (significant at 10% level). For the obese group, the weight gains appear permanent. Even after 37+ months, log BMI is 2.2% and weight is 2.1 kg higher than before the wealth shock.

5. EFFECTS OF WEALTH ON HEALTH-RELATED LIFESTYLE DECISIONS

Weight is fundamentally an outcome of decisions individuals make about the amount of energy to consume and the amount of energy to expend. Wealth could potentially increase weight among females through a number of different lifestyle decisions that affect energy consumption and energy expenditure. In this section, we use HILDA data on lifestyle factors (meals eaten out, physical activity, alcohol and smoking) to help explain why increases in wealth cause increases in weight for females.

Foods eaten outside home (including restaurant meals and fast food) are typically more energy dense than foods prepared at home (Prentice and Jebb, 2003), and their consumption has been associated with weight gain and obesity (Cutler *et al*., 2003; Chou *et al*., 2004). The first two columns of Table V show wealth shock estimates from FE regression models of the probability of positive weekly expenditure on meals eaten out (column (1)) and the log of weekly expenditure on meals eaten out (column (2)). This information is self-reported and at the household level. It includes restaurant meals, take away, and bought lunches and snacks but excludes alcohol. The results show no effect for males (row A), significant positive effects for all females (row B) and initially poor/comfortable females (row C) on both the probability of positive expenditures and amount spent, and a significant positive effect for initially obese females on amount spent (row D). The estimated effect for obese females on amount spent on meals eaten out was particularly large, with a wealth shock increasing expenditure by 12.6%.[20] This finding is supported by Van Kippersluis and Galama (2013)

[19]The results for males are consistent with those in Table 1. At each lag length, the estimated wealth shock effects on log BMI are near zero: estimates of $\lambda_0, \lambda_1, \lambda_2$ and λ_3 equal −0.00034, −0.00003, 0.00553 and 0.00126, respectively.
[20]IV-FE estimates in TablE0020A4 in the supporting information suggest a \$100,000 increase in wealth increases the expenditure of obese females by 15.1%.

Table V. Estimated effects of a wealth shock on health-related lifestyle decisions

	Positive spend on outside meals	Log spend on outside meals	Moderate weekly physical activity	Number of standard drinks per month	Number of cigarettes per week
	(1)	(2)	(3)	(4)	(5)
(A) Males	0.005	0.060	−0.026	0.543	−0.340
	(0.019)	(0.043)	(0.020)	(1.310)	(1.417)
(B) Females	0.039**	0.064*	−0.019	0.651	−0.784
	(0.019)	(0.035)	(0.019)	(0.635)	(0.898)
(C) Females and Poor/Comfortable	0.059***	0.072*	−0.018	0.914	−0.733
	(0.021)	(0.038)	(0.022)	(0.721)	(1.117)
(D) Females and BMI > 30	0.020	0.126**	−0.054	−0.620	−3.299
	(0.041)	(0.057)	(0.045)	(1.357)	(2.369)
Sample mean	0.824	3.697	0.715	17.021	13.556
Sample standard deviation	0.381	0.785	0.451	31.176	40.628

Figures are coefficient estimates from fixed-effect (FE) regression models. Regression models also contain the covariates described in Table A1 in the supporting information and year dummy variables. Dependent variables in models (1) and (3) are binary variables. The sample mean and standard deviation figures are calculated using females only. Standard errors clustered at the individual level are shown in parentheses.
*Significant at 0.10 level; **significant at 0.05 level; ***significant at 0.01 level.

who, using the British Household Panel Survey and the US Health Retirement Study, also find evidence to suggest that wealth shocks (measured by lottery wins and inheritance) increase expenditures on meals eaten out, although they do not estimate the effect by gender. The finding is also supported by Kuhn *et al*. (2011) who find that winning the Dutch postcode lottery increases expenditure on food away from home.

A limitation of the expenditure data is that it does not allow us to determine the quantity or type of meals consumed by the individual, and consequently, we do not know whether higher expenditure corresponds to higher energy consumption. For example, additional expenditure could be driven by the purchase of food from more expensive restaurants rather than more frequent eating-out. Given this limitation, we supplement the expenditure analysis using additional data in HILDA from 2007 and 2009 on eating-out. We find that a wealth shock increases the probability of eating-out for dinner (at least once per week) by 9 percentage points for females, (t-statistic = 2.66), suggesting that wealth increases the quantity of meals eaten out, in addition to total expenditure.

Physical activity increases energy expenditure and has been shown to play a contributing role in the prevention of weight gain (Van Baak, 1999; Fogelholm and Kukkonen-Harjula, 2000). Similarly, alcohol intake (Wannamethee *et al*., 2004) and smoking, which tends to increase metabolism and reduce appetite (Chiolero *et al*., 2008), have been shown to be independent lifestyle factors that impact on weight. Columns (3)–(5) present estimates of the impact of wealth on the probability of engaging in physical activity (of moderate or vigorous intensity) for 30 min at least once per week, the number of standard drinks usually consumed per month and the number of cigarettes usually smoked per week. For each of these three lifestyle factors, we find that the wealth effect is statistically insignificant and small relative to the sample mean.[21] These findings, coupled with the eating-out results, suggest that gluttony rather than sloth is driving the positive wealth effect among women, although the physical activity measure used here is a crude proxy for energy expenditure and the conclusions could differ if more precise measures were available.

Finally, we investigate a more indirect pathway between increased wealth and weight gain than those presented above, namely labour supply. It is quite possible that a large increase in wealth will reduce female

[21]Previous studies on the impact of wealth on lifestyle provide mixed results. For example, Apouey and Clark (2013) find that wealth is positively associated with smoking and social drinking, whereas Kim and Ruhm (2012) find that wealth increases alcohol consumption but does not affect smoking or physical activity. Using the same datasets as the above studies, Van Kippersluis and Galama (2013) find that although inheritances in the US increase the likelihood of smoking, lottery wins in the UK do not.

DOI: 10.1002/hec

employment, especially for females with weak attachment. Moreover, studies have shown that employment in physically demanding occupations can have large impacts upon weight.[22] Therefore, significant employment effects may point towards lifestyle changes that our proxy measures of energy consumption and expenditure are unable to detect. Replicating the analysis in Table V for employment, work hours (including zeros) and work hours if employed (omitting zeros) shows that labour supply changes are not an important explanation for our positive wealth effects. We find wealth shock effects for employment and work hours equal to −0.016 (*t*-statistic = −1.22), 0.306 (*t*-statistic = 0.56) and 0.950 (*t*-statistic = 1.47), respectively.

6. CONCLUSION

Numerous studies have shown a strong negative relationship between wealth and obesity, but it is unclear whether this relationship is causal. Financial resources may lessen obesity because they allow the purchase of fresh and nutritious foods and provide greater access to active lifestyles. On the other hand, wealth and obesity may be jointly driven by third factors, such as educational attainment, employment, health knowledge and attitudes to risk and time preference. Although empirically challenging, it is important to ascertain whether financial disadvantage causes obesity. In this paper, we shed light on this significant issue by estimating the effect of a large wealth shock on weight using data from a nationally representative, longitudinal study of Australian adults. In addition, we explore how the wealth effect varies by population subgroups; whether wealth effects are immediate or delayed, and temporary or permanent; and the potential mechanisms through which additional wealth may impact weight.

The paper makes a number of contributions. First, we find that despite the very large association between socioeconomic disadvantage and obesity, additional wealth has a significant, but modest, positive effect on weight for females. An average-sized wealth shock of around $50,000 increases female weight by 725 g (1.6 lb), BMI by 0.9% and obesity by 2.1 percentage points. This result implies that additional wealth may exacerbate rather than alleviate weight problems, and as such, research and policy must focus on other associated factors, such as educational attainment (e.g. Brunello *et al.*, 2013), to better understand and reduce the socioeconomic inequalities of obesity.

It is difficult to compare our findings to previous studies, due to differences in identification strategies and study populations. For example, studies that similarly use lottery wins or inheritances as exogenous shocks to wealth find either a negative (Lindahl, 2005) or insignificant (Kim and Ruhm, 2012) wealth effect on obesity, but the study populations are very different to ours. Schmeiser (2009) is one study that similarly finds a positive wealth effect on BMI but is restricted to low-income individuals and uses a permanent increase in household income through earned income tax credits (instead of a wealth shock) as the exogenous variation in income. We acknowledge that one limitation of our study is that the source of the wealth shock is not made explicit in the HILDA survey. However, we show that it is highly correlated with inheritances, while uncorrelated with all other sources of windfall income. We also show that the estimates are consistent across numerous model specifications.

A further contribution of our paper is that we find substantial heterogeneity across subgroups, with the most disadvantaged and at-risk experiencing the largest increases in weight: an average-sized wealth shock received by initially poor and obese women is estimated to increase weight in 1 year by almost 10 lb and BMI by almost 5%. This indicates that a one-off wealth transfer is likely to exacerbate socioeconomic disparities in obesity. As higher levels of BMI are associated with increasingly greater risk of chronic disease, this can have serious health implications. For example, each 2kg/m^2 higher BMI (or each 5% increase in BMI for an obese woman

[22]Lakdawalla and Philipson (2007) find that job-related exercise has causal effects on weight for male workers but not female workers. They conjecture that the insignificant female effects may be driven by poor measurement of job characteristics for females and by occupational selection of female workers by weight status.

DOI: 10.1002/hec

with BMI > 35) is shown to be associated with roughly a 27% higher risk of type 2 diabetes, 12% higher risk of stroke and 11% higher risk of ischemic heart disease (Asia Pacific Cohort Studies Collaboration, 2004).

We further show that increases in weight immediately follow increases in wealth. For most women, the weight gains are temporary, but for the initially obese, they appear to be permanent. The permanency of weight gain in an already at-risk group is concerning from a both a health and economic perspective. Lastly, we find that a wealth shock increases the expenditure on and frequency of meals eaten outside the home, a finding that is supported by previous studies using lottery and inheritance data from the UK, US and the Netherlands (Kuhn *et al*., 2011; Van Kippersluis and Galama, 2013). In contrast to the significant effect on meals eaten out, we find a wealth shock has no significant effect on physical activity levels, alcohol intake, smoking or labour supply, suggesting a change in diet is a likely explanation for our results.

ACKNOWLEDGEMENTS

This paper uses unit record data from the Household, Income and Labour Dynamics in Australia (HILDA) Survey. The HILDA project was initiated and is funded by the Australian Government Department of Families, Housing, Community Services and Indigenous Affairs (FaHCSIA) and is managed by the Melbourne Institute of Applied Economic and Social Research (MIAESR). The findings and view reported in this article, however, are those of the authors, and should not be attributed to FaHCSIA or the MIAESR.

REFERENCES

Akee R, Simeonova E, Copeland W, Angold A, Costello JE. 2013. Young adult obesity and household income: effects of unconditional cash transfers. *American Economic Journal: Applied Economics* **5**(2): 1–28.

Apouey B, Clark A. 2013. Winning big but feeling no better? The effect of lottery prizes on physical and mental health. *CEP Discussion Paper No 1228*.

Asia Pacific Cohort Studies Collaboration. 2004. Body mass index and cardiovascular disease in the Asia-Pacific Region: an overview of 33 cohorts involving 310 000 participants. *International Journal of Epidemiology* **33**(4): 751–758.

Australian Bureau of Statistics. 2012. Australian health survey: updated results, 2011–2012. ABS cat. no. 4364.0.55.003. Canberra: Australian Bureau of Statistics.

Au N, Johnston DW. 2013. An econometric analysis of self-assessed health: what does it mean and what is it hiding? in *HEDG Working Paper 13/31*: University of York.

Blundell R, Bond S. 1998. Initial conditions and moment restrictions in dynamic panel data models. *Journal of Econometrics* **87**(1): 115–143.

Bray GA. 2004. Medical consequences of obesity. *Journal of Clinical Endocrinology & Metabolism* **89**(6): 2583–2589.

Brunello G, Fabbri D, Fort M. 2013. The causal effect of education on body mass: evidence from Europe. *Journal of Labor Economics* **31**(1): 195–223.

Burkhauser RV, Cawley J. 2008. Beyond BMI: the value of more accurate measures of fatness and obesity in social science research. *Journal of Health Economics* **27**: 519–529.

Burton NW, Brown W, Dobson A. 2010. Accuracy of body mass index estimated from self-reported height and weight in mid-aged Australian women. *Australian and New Zealand Journal of Public Health* **34**(6): 620–623.

Cawley J. 2000. An instrumental variables approach to measuring the effect of body weight on employment disability. *Health Services Research* **35**(5): 1159–1179.

Cawley J. 2004. The impact of obesity on wages. *Journal of Human Resources* **39**(2): 451–474.

Cawley J, Meyerhoefer C. 2012. The medical care costs of obesity: an instrumental variables approach. *Journal of Health Economics* **31**(1): 219–230.

Cawley J, Moran J, Simon K. 2010. The impact of income on the weight of elderly Americans. *Health Economics* **19**(8): 979–993.

Chang VW, Lauderdale DS. 2005. Income disparities in body mass index and obesity in the United States, 1971–2002. *Archives of Internal Medicine* **165**(18): 2122.

Chiolero A, Faeh D, Paccaud F, Cornuz J. 2008. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *The American Journal of Clinical Nutrition* **87**(4): 801–809.

Chiriboga DE, Ma Y, Li W, Olendzki BC, Pagoto SL, Merriam PA, Matthews CE, Hebert JR, Ockene IS. 2008. Gender differences in predictors of body weight and body weight change in healthy adults. *Obesity* **16**(1): 137–145.
Chou SY, Grossman M, Saffer H. 2004. An economic analysis of adult obesity: results from the behavioral risk factor surveillance system. *Journal of Health Economics* **23**(3): 565–587.
Cobb-Clark D, Kassenboehmer S, Schurer S. 2012. Healthy habits: the connection between diet, exercise, and locus of control. in *IZA Discussion Paper No. 6789.* IZA Bonn, Germany.
Cutler DM, Lleras-Muney A. 2010. Understanding differences in health behaviors by education. *Journal of Health Economics* **29**(1): 1–28.
Cutler DM, Glaeser EL, Shapiro JM. 2003. Why have Americans become more obese? *The Journal of Economic Perspectives* **17**(3): 93–118.
Dinour LM, Bergen D, Yeh M-C. 2007. The food insecurity–obesity paradox: a review of the literature and the role food stamps may play. *Journal of the American Dietetic Association* **107**(11): 1952–1961.
Drewnowski A. 2004. Obesity and the food environment: dietary energy density and diet costs. *American Journal of Preventive Medicine* **27**(3, Supplement 1): 154–162.
Fogelholm M, Kukkonen-Harjula K. 2000. Does physical activity prevent weight gain—a systematic review. *Obesity Reviews* **1**(2): 95–111.
Frank R, Akresh I. 2013. Social patterning in body mass index (BMI) among contemporary immigrant groups: the emergence of a gradient. *Demography* **50**(3): 993–1012.
Fuchs VR. 1982. Time preference and health: an exploratory study. In Economic Aspects of Health, Fuchs VR (ed.), University of Chicago Press: Chicago; 93–120.
García Villar J, Quintana-Domeque C. 2009. Income and body mass index in Europe. *Economics & Human Biology* **7**(1): 73–83.
Gardner J, Oswald AJ. 2007. Money and mental wellbeing: a longitudinal study of medium-sized lottery wins. *Journal of Health Economics* **26**(1): 49–60.
Gordon-Larsen P, Nelson MC, Page P, Popkin BM. 2006. Inequality in the built environment underlies key health disparities in physical activity and obesity. *Pediatrics* **117**(2): 417–424.
Granger CW. 1969. Investigating causal relations by econometric models and cross-spectral methods. *Econometrica: Journal of the Econometric Society* **37**(3): 424–438.
Greve J. 2008. Obesity and labor market outcomes in Denmark. *Economics & Human Biology* **6**(3): 350–362.
Grossman M. 1972. On the concept of health capital and demand for health. *Journal of Political Economy* **80**(2): 223–225.
Hajat A, Kaufman JS, Rose KM, Siddiqi A, Thomas JC. 2010. Do the wealthy have a health advantage? Cardiovascular disease risk factors and wealth. *Social Science & Medicine* **71**(11): 1935–1942.
Hays NP, Roberts SB. 2008. Aspects of eating behaviors ‘disinhibition’ and ‘restraint’ are related to weight gain and BMI in women. *Obesity* **16**(1): 52–58.
Johar M, Katayama H. 2011. Quantile regression analysis of body mass and wages. *Health Economics* **21**(5): 597–611.
Kim B, Ruhm CJ. 2012. Inheritances, health and death. *Health Economics* **21**(2): 127–144.
Kuhn P, Kooreman P, Soetevent A, Kapteyn A. 2011. The effects of lottery prizes on winners and their neighbors: evidence from the Dutch postcode lottery. *The American Economic Review* **101**(5): 2226–2247.
Lakdawalla D, Philipson T. 2007. Labor supply and weight. *Journal of Human Resources* **42**(1): 85–116.
Levine JA. 2011. Poverty and obesity in the U.S. *Diabetes* **60**(11): 2667–2668.
Lindahl M. 2005. Estimating the effect of income on health and mortality using lottery prizes as an exogenous source of variation in income. *Journal of Human Resources* **40**(1): 144–168.
Luppino FS, de Wit LM, Bouvy PF, Stijnen T, Cuijpers P, Penninx BWJH, Zitman FG. 2010. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Archives of General Psychiatry* **67**(3): 220.
McLaren L. 2007. Socioeconomic status and obesity. *Epidemiologic Reviews* **29**(1): 29–48.
Meer J, Miller DL, Rosen HS. 2003. Exploring the health-wealth nexus. *Journal of Health Economics* **22**(5): 713–730.
Michaud PC, Van Soest A. 2008. Health and wealth of elderly couples: causality tests using dynamic panel data models. *Journal of Health Economics* **27**(5): 1312–1325.
Pahl J. 1990. Household spending, personal spending and the control of money in marriage. *Sociology* **24**(1): 119–138.
Prentice AM, Jebb SA. 2003. Fast foods, energy density and obesity: a possible mechanistic link. *Obesity Reviews* **4**(4): 187–194.
Puhl R, Brownell KD. 2001. Bias, discrimination, and obesity. *Obesity* **9**(12): 788–805.
Sarlio-Lähteenkorva S, Lahelma E. 1999. The association of body mass index with social and economic disadvantage in women and men. *International Journal of Epidemiology* **28**(3): 445–449.
Schmeiser MD. 2009. Expanding wallets and waistlines: the impact of family income on the BMI of women and men eligible for the earned income tax credit. *Health Economics* **18**(11): 1277–1294.

Smith TG, Stoddard C, Barnes MG. 2009. Why the poor get fat: weight gain and economic insecurity. *Forum for Health Economics & Policy* **12**: 1–29.
Sobal J, Stunkard AJ. 1989. Socioeconomic status and obesity: a review of the literature. *Psychological Bulletin* **105**(2): 260–275.
St-Onge MP, Gallagher D. 2010. Body composition changes with aging: the cause or the result of alterations in metabolic rate and macronutrient oxidation? *Nutrition* **26**(2): 152–155.
Stunkard AJ, Wadden TA. 1992. Psychological aspects of severe obesity. *The American Journal of Clinical Nutrition* **55**(2): 524S–532S.
Van Baak MA. 1999. Physical activity and energy balance. *Public Health Nutrition* **2**: 335–340.
Van Kippersluis H, Galama TJ. 2013. Why the rich drink more and smoke less: the impact of wealth on health behaviors. Tinbergen Institute, Working paper TI 2013-035:V.
Wannamethee SG, Field AE, Colditz GA, Rimm EB. 2004. Alcohol intake and 8-year weight gain in women: a prospective study. *Obesity* **12**(9): 1386–1396.
Wolfe B, Jakubowski J, Haveman R, Courey M. 2012. The income and health effects of tribal casino gaming on American Indians. *Demography* **49**(2): 499–524.
Wood M. 1998. Socio-economic status, delay of gratification, and impulse buying. *Journal of Economic Psychology* **19**(3): 295–320.
Zagorsky JL. 2005. Health and wealth. The late-20th century obesity epidemic in the U.S. *Economics and Human Biology* **3**(2): 296–313.

DOI: 10.1002/hec