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The impact of obesity on employment

Stephen Morris

Imperial College London, United Kingdom

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Abstract

Using data from two rounds of the Health Survey for England I investigate the impact of obesity on employment. I use three approaches: a univariate probit model; propensity score matching; and IV regression using a recursive bivariate probit model. Conditional on a comprehensive set of covariates, the findings show that obesity has a statistically significant and negative effect on employment in both males and females. In males the endogeneity of obesity does not significantly affect the estimates, and the magnitude of effect is similar across the three methods. In females, failure to account for endogeneity leads to underestimation of the negative impact of obesity on employment.

JEL classification: I10; I12; I18; J21; J23
Keywords: Obesity; Employment; Propensity score matching; Endogeneity; Bivariate probit

1. Introduction

Obesity is a rapidly growing health problem that affects an increasing number of countries worldwide (WHO, 1998).[1] In the United States, over a quarter of all adults are obese (HHSD, 2001), while in England and many other European countries the prevalence of obesity is also rising to epidemic proportions. In 1980 six per cent of males and eight per cent of females in England were obese; by 2003 the prevalence had trebled to 21 per cent and 24 per cent, respectively (Department of Health, 2003). The worldwide growth in obesity is a serious cause for concern because as well as being a debilitating condition in its own right, obesity is an important

E-mail address: s.morris@imperial.ac.uk.

[1] Obesity is normally measured in terms of body mass index (BMI), which is usually computed in adults as an individual's weight in kilogrammes divided by their height in metres squared (NHLBI, 1998). The BMI range generally considered to be healthy is 20 to 25 kg/m^2. Those with a BMI below 20 kg/m^2 are underweight, and those with a BMI in the range 25 to 30 kg/m^2 are overweight. Obesity is usually defined by a BMI over 30 kg/m^2.

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risk factor for a number of major diseases including coronary heart disease, type II diabetes, osteoarthritis, hypertension and stroke (NHLBI, 1998). In the United States obesity is the second leading cause of preventable disease and death next to smoking (HHSD, 2001), while in England seven per cent of all deaths are attributable to obesity (House of Commons Health Committee, 2004).

As well as imposing large morbidity and mortality costs obesity imposes a substantial financial burden. Studies for Australia (Segal et al., 1994), Canada (Katzmarzyk and Janssen, 2004), England (House of Commons Health Committee, 2004), France (Detournay et al., 2000), New Zealand (Swinburn et al., 1997) and the United States (Wolf and Colditz, 1998) report that obesity accounts for between 1% and 8% of national health expenditure. The effect on employers is also considerable; the economic cost of obesity to business in the United States is more than $12 billion per annum (Thompson et al., 1998), and in England there are in excess of 15 million days of medically certified sickness absence from work due to obesity each year (House of Commons Health Committee, 2004). Obesity may have other consequences that affect economic outcomes. For example, obese individuals are more likely to suffer from social stigmatisation and discrimination (NHLBI, 1998), which have been documented in a variety of settings including health care and the labour market (WHO, 1998).

The aim of this paper is to examine the impact of obesity on employment. The analysis is conducted using data from two rounds of the Health Survey for England. I use three approaches: a univariate probit model in which employment measured as a binary variable is regressed against obesity, also measured as a binary variable, plus a comprehensive set of individual and area covariates; propensity score matching using a variety of matching methods; and instrumental variables (IV) regression using a recursive bivariate probit model. I instrument individual obesity using an area level obesity measure: the prevalence of obesity in the area in which the respondent lives. This is positively correlated with individual obesity and is plausibly not itself correlated with the error term in the employment equation.

Several studies have previously analysed the effect of obesity on employment. In the first of two British studies Sargent and Blanchflower (1994) used the 1981 round of the National Child Development Study (NCDS) to examine the impact of obesity on the labour market outcomes of young British adults. They report the impact of obesity (defined as a BMI at the 90th percentile of the sample distribution or greater, and at the 99th percentile or greater) at age 16 years on unemployment at age 23 years, controlling for race, social class, region of residence and ability. Using a logit model the authors report that obesity has an insignificant effect on unemployment. Harper (2000) used a later (1991) round of the NCDS to estimate the impact of obesity, plus general physical appearance, attractiveness and height at age 23 years on unemployment at age 33 years also using a logit model. Obesity was defined by a BMI in the 80–89th percentiles and the 90–100th percentiles of the sample distribution. As with the earlier study the results showed that obesity had an insignificant effect on unemployment for both males and females.

Using a 1994 sample of Finnish adults, Sarlio-Lahteenkorva and Lahelma (1999) used a logit model to analyse the impact of current obesity (measured as a BMI of 30 kg/m^2 or more) on current employment and long term unemployment, defined as being unemployed for two years or more in the previous five year period. Controlling for age, educational attainment, region of residence and limiting longstanding illness they found in females that obesity has a significant and positive impact on long-term unemployment, and an insignificant effect on current employment. For males, obesity had an insignificant effect on both employment measures.

None of these studies investigated the endogeneity of obesity and employment. Both Sargent and Blanchflower and Harper regress current employment against lagged obesity, which may deal with simultaneity, but will not correct for omitted variables that affect both obesity and employment (an example is time preference, which might affect human capital and tastes for work, and hence employment, and also obesity). Cawley (2000a) addresses the endogeneity problem using an IV regression approach. He analyses the impact of BMI on wages and employment in a sample taken from the US National Longitudinal Survey of Youth panel over the period 1981 to 1998. In the employment analysis, probit and IV probit models are estimated, regressing employment against BMI plus covariates. The instruments for BMI are the BMI of a biological child aged six to nine years plus interactions of this with the child's age and gender. The analysis is restricted to females who have borne children. In the probit model the BMI coefficient is not statistically significant. In the IV probit model BMI is found to have a positive effect on employment that is statistically significant at the 10% level.

This paper makes a number of contributions to the literature. First, it investigates the relationship between obesity and employment in England using a number of methods, in order to obtain a better understanding of the mechanisms by which obesity affects employment. Second, it considers males and females of normal working age rather than young adults, who have been the focus of previous British studies. Third, the data are more recent than those in earlier British studies, covering the period 1997–1998. This is useful because as the prevalence of obesity increases over time the impact of obesity on employment may change and older studies will become out of date. Additionally, since the election of the Labour government in May 1997 obesity has become an increasingly prominent policy issue and there have been a number of recent developments to address the health and social effects (Department of Health, 2004). The results presented in this study provide a baseline to assess the impact of these policies on the employment prospects of the obese.

The rest of the paper is structured as follows. Section 2 discusses the model underpinning the analysis and the econometric framework. Section 3 discusses the data and variables used. The estimation results are in Section 4 and Section 5 concludes.

2. Analysis and estimation

2.1. Preliminaries

There are four reasons why obesity and employment may be correlated.

(1) Obesity causes unemployment. This might arise for two reasons. First, obesity can be a debilitating health condition (NHLBI, 1998). Therefore, all else equal, the obese are likely to be less productive than the non-obese, and therefore less likely to be employed. Second, there may be discrimination against the obese. Based on Balsa and McGuire (2003) this might arise for three reasons. First, there may be prejudice by employers, reflecting their distaste for obese workers and the psychological costs incurred when dealing with them (Moon and McLean, 1980). Second, there may be stereotyping by employers, arising from a belief that the obese are less productive (Everett, 1990). Third, discrimination may arise through uncertainty, or a lack of knowledge about the productivity of obese workers (Pagan and Davila, 1997).

(2) Unemployment causes obesity. For example, unemployed individuals, who have lower incomes, are more likely to consume cheaper more fattening food (Cawley, 2004).

(3) There are unobserved variables, such as time preference mentioned above, that are correlated with both obesity and employment.
(4) Obesity may be measured systematically with error due to unobserved factors correlated with employment. This might arise if individuals in lower socioeconomic groups are more likely to under or over report their BMI all else equal.

The aim of the paper is to identify the first effect and to produce unbiased estimates that are not contaminated by the other effects. I use three methods: a univariate probit model; propensity score matching; and, a recursive bivariate probit model. These are described below.

2.2. Univariate probit

Let employment Y be a binary variable taking the value one if the individual is in paid employment and zero otherwise. Suppose that Y is a linear function of obesity B and other variables:

$$Y_i = a_0 + a_1 S_i + a_2 H_i + a_3 F_i + a_4 C_i + a_5 B_i + u_i \tag{1}$$

where u is an error term, i indexes individuals, and S, H, F and C are vectors of variables that affect employment, including human capital variables and other variables that affect tastes for work. S measures education and schooling; H measures health status; F measures home and family variables that affect tastes for work; and C is a vector of additional control variables that affect employment, such as age, sex and ethnicity. (1) can be estimated by a single equation univariate probit model:

$$\begin{aligned} y_i^* &= \beta_0 x_i + dB_i + \mu_{0i} \\ E[\mu_0] &= 0 \\ Var[\mu_0] &= 1 \end{aligned} \tag{2}$$

where y^* is an unobserved latent variable and $x = \{S, H, F, C\}$. Empirically we observe the binary variable y that takes the value one if the individual is in employment ($y_i^* \geq 0$) and zero otherwise ($y_i^* < 0$). B is a binary variable taking the value one if the individual is obese and zero otherwise. μ is an error term, and β and d are coefficients. d is a measure of the impact of obesity on employment. Given that B is a dummy variable the marginal effect (ME) of being obese on the probability of being in paid employment is the sample average of changes in the marginal predicted probability of being in paid employment with discrete changes in B keeping all other variables x at their observed values:

$$\mathrm{ME} = \frac{1}{n}\sum_{i=1}^{n} \left[\Phi\left(\hat{\beta}_0 x_i + \hat{d}B_i | B = 1\right) - \Phi\left(\hat{\beta}_0 x_i + \hat{d}B_i | B = 0\right)\right] \tag{3}$$

where Φ is the standard normal distribution function, $\Phi(\hat{\beta}_0 x + \hat{d}B)$ is the marginal predicted probability of being in paid employment and is computed for each observation using the estimated coefficients, and n is the number of individuals in the sample.

2.3. Propensity score matching

ME in (3) is a measure of the average treatment effect on the treated (ATT), i.e., the mean effect of obesity on employment for those who are obese. The above method, and the bivariate probit model outlined below, impose two assumptions. First, the impact of obesity on employment is assumed to be constant across all individuals. Second, they use observations outside the area of 'common support' (i.e., individuals in the obese or non-obese groups are included in the estimation sample even if there are no similar individuals of the other group in terms of their values of the covariates). An alternative approach to calculating the ATT, which can avoid these restrictions, is based on the propensity score (Rosenbaum and Rubin, 1983). The underlying principle consists of matching treated with untreated (i.e., obese with non-obese) individuals in terms of their observable characteristics x, and then comparing the employment of obese and non-obese individuals that have the same obesity propensity. This can avoid the two restrictions described above because, first, the ATT is obtained by averaging individual level differences in employment between the obese and matched non-obese. Second, the matching process may be restricted to the region of common support, ensuring that comparisons between obese and non-obese individuals occur only between individuals with broadly similar observable characteristics.

An overview of the approach is given by Becker and Ichino (2002), and a recent application examining public versus private education and pupil achievement is given by Vandenberghe and Robin (2004). Following Becker and Ichino (2002)[2] I proceed by first estimating an obesity propensity score $p(x)$ for each individual in the sample using the estimated coefficients from a probit regression of B on x. I then test that individuals with the same propensity score $p(x)$ have the same distribution of observed covariates independently of obesity status by splitting the sample into blocks of $p(x)$ so that the mean value of $p(x)$ among obese and non-obese individuals is the same. Then, within each block the means of each observable characteristic x are tested using a t-test to check they do not differ between the two groups. Next, I compute the ATT by matching obese and non-obese individuals on the basis of their propensity score. I use four matching methods: stratification matching; nearest neighbor matching; kernel matching; and, radius matching. See Becker and Ichino (2002) for further details. The difference in employment between obese and matched non-obese individuals is then computed. The ATT is obtained by averaging these differences across the m matches:

$$\mathrm{ATT} = \frac{1}{m}\sum_{j=1}^{m} \left[y_j^{j\in B=1} - y_j^{j\in B=0} \right] \tag{4}$$

The standard error for the ATT is calculated using a bootstrapping procedure, from the standard deviation of the ATT after 200 bootstrap replications.

The approach described above is run with and without the common support condition. When this condition is applied observations in the non-obese group are discarded if they have values of $p(x)$ less than the minimum or greater than the maximum estimated value of $p(x)$ in the obese group.

2.4. Bivariate probit

The above approaches assume that obesity conditional on the covariates is independent of employment, i.e., that obesity does not depend on employment. If obesity is endogenous then the

[2] The analysis uses the Stata commands –pscore–, –atts–, –attnd–, –attk– and –attr– written by Becker and Ichino (2002).

conditional independence assumption is violated and the ATT from the above two methods is biased and an unreliable estimate of the causal effect of obesity on employment. To test and control for endogeneity I use an IV regression method based on a recursive bivariate probit model of the form:

$$\begin{aligned}
&y_i^* = \beta_1 x_i + \delta B_i + \mu_{1i} \\
&B_i^* = \beta_2 x_i + \alpha Z_i + \mu_{2i} \\
&E[\mu_1] = E[\mu_2] = 0 \\
&Var[\mu_1] = Var[\mu_2] = 1 \\
&\mathrm{Cov}[\mu_1, \mu_2] = \rho
\end{aligned} \tag{5}$$

where B^* is an unobserved latent variable such that $B_i^* \geq 0$ if $B_i = 1$ and $B_i^* < 0$ if $B_i = 0$. α and δ are coefficients and Z is a vector of IVs that are correlated with B but not μ_1. The coefficient of interest is δ. ρ is the correlation between the error terms in the obesity and employment equations. A Wald test of the significance of ρ is a direct test of the endogeneity of y and B (Wooldridge, 2002, p. 478). If $\rho = 0$ then it is appropriate to use the univariate probit model (2). If ρ is non-zero then obesity and employment are endogenous, the univariate probit results are biased, and the bivariate probit model should be used. (5) is an appropriate model to use when the dependent variable is binary and we have a binary endogenous explanatory variable (Wooldridge, 2002, pp. 477–8).[3]

To control for endogeneity using (5) requires a suitable instrument Z for obesity. This should have two properties: it must be a non-weak predictor of B conditional on x (i.e., $\alpha \neq 0|x$); and it must be uncorrelated with μ_1 (i.e., $\mathrm{Cov}(Z, \mu_1) = 0$). While it is possible to test the first property by examining the significance of Z in the obesity equation in (5) the second requirement cannot be tested directly and must be maintained (Wooldridge, 2002, p. 86). The instrument used for obesity is the prevalence of obesity in the area in which the respondent lives. This is shown to meet both requirements (see below for a detailed justification).

Marginal effects are computed as for the probit model. The difference is that the estimated coefficients used in (3) are $\hat{\beta}_1$ and $\hat{\delta}$ instead of $\hat{\beta}_0$ and $\hat{d}$. Thus the marginal effects in this case take into account the endogeneity of obesity.

Interpreting the IV results, Imbens and Angrist (1994) and Angrist et al. (1996) show that under standard assumptions $\hat{\delta}$ from (5) is the local average treatment effect (LATE). This measures the average treatment effect for those who change treatment status (i.e. become obese) because they comply with the assignment to treatment implied by the instrument.

3. Data and variables

3.1. Data sources

I use pooled data from two rounds (1997 and 1998) of the Health Survey for England (HSE). The HSE is a nationally representative survey of individuals aged two years and over living in England. A new sample is drawn each year and respondents are interviewed

[3] A similar model is used by MacDonald and Shields (2004) in the context of problem drinking and employment and Brown et al. (2005) in the context of diabetes and employment.

on a range of core topics including demographic and socioeconomic indicators, general health and psychosocial indicators, and use of health services. Additionally, there is a follow up visit by a nurse at which various physiological measurements are taken, including height and weight.

The area level data used in the analysis were assembled from three sources. First, I use the Allocation of Resources to English Areas (AREA) dataset for comprehensive data on deprivation, health and accessibility to health care services at the local authority ward level across England. The dataset was constructed for a project that examined the determinants of the use of hospital and community health services and general practice in England (Sutton et al., 2002; Gravelle et al., 2003). Local authority level data on crime rates were obtained from the Neighbourhood Statistics branch of the Office for National Statistics,[4] and data on house prices were obtained from the Land Registry.[5] The area level data were converted to the health authority level. England is divided into 95 health authorities with a mean population of 515,517 residents (range 168,873 to 1,050,626). Mean values of the variables for each health authority were computed based on the proportion of each local authority's/local authority ward's population resident within each health authority, which is available in the AREA dataset. The health authority data were then linked to the individuals in the HSE sample via their recorded health authority of residence.

3.2. Employment and obesity

The employment variable is a binary variable taking the value one if the individual is in paid employment or self-employed and zero if the individual is unemployed or out of the labour force.

The obesity measure is computed for each respondent from the height and weight values obtained during the nurse visit in the HSE. One useful feature of the 1997 and 1998 rounds of the HSE is that height and weight are measured by the nurse and not self reported, reducing the likelihood of systematic measurement error. Obesity is measured using the standard definition and is a binary variable taking the value one if the individual has a BMI over 30 kg/m^2 and zero otherwise. The HSE records respondents' current height and weight. I therefore analyse the impact of current obesity, which was also the focus of previous studies of the impact of obesity on current labour market outcomes (Register and Williams, 1990; Loh, 1993; Pagan and Davila, 1997; Cawley, 2000a,b).[6]

3.3. Covariates

I include a number of other explanatory variables, grouped in four categories. The first category contains education variables, measuring educational attainment (highest educational qualification attained) and years of schooling. The latter is a continuous variable measured as the age at which respondents finished their full time continuous education at school or college minus four years. I also include years of schooling squared.

The second category contains health variables, which are covered comprehensively in the HSE. I include measures of self reported general health, acute ill health, longstanding illness, and

[4] http://www.neighbourhood.statistics.gov.uk/home.asp.

[5] http://www.landreg.gov.uk/propertyprice/interactive/ppr_ualbs.asp.

[6] There is also evidence to support the view that current obesity is a good indicator of obesity at younger ages (e.g., Whitaker et al., 1997).

psychosocial health. Self reported general health is a measure of subjective general health measured in five categories from very good to very bad. Acute ill health is measured by the number of days in the last two weeks the respondent had to cut down on the things they usually do because of illness of injury. In terms of longstanding illnesses respondents are asked whether they have an illness, disability or infirmity that has troubled them over a period of time, and its type by broad disease code. Limiting longstanding illness is categorized by whether any of these illnesses limits respondents' activities in any way. Comorbidities are measured by the number of longstanding illnesses. Psychosocial health is measured by GHQ-12 score, where higher values indicate more severe psychosocial problems.

The third category contains home and family variables. I consider housing, marriage and family size variables in this group.[7] The HSE collects information on respondents' marital status and housing tenure. I also control for the number of infants living in the household aged zero or one year and the number of children aged 2 to 15 year living in the household.

In the final category I include additional control variables that may affect employment: gender; age; ethnicity; rurality; region of residence; and, HSE year. I estimate separate models for males and females and include age, age squared and aged cubed in all the models.

I also include sixty two area based indicators to control for the impact of local area characteristics on individual employment. They fall into three categories: deprivation measures; health measures; and health care supply measures. Area deprivation is measured mainly using the Index of Deprivation (ID2000) (DTLR, 2000). These are a set of indicators that describe multiple deprivation across geographical areas in England, based on routinely collected administrative data. The ID2000 comprises seven domains, each reflecting a different aspect of deprivation. The domains measure income deprivation, child poverty, employment deprivation, health deprivation and disability, education deprivation, housing deprivation and access deprivation. An overall index of deprivation is constructed by combining the domains. See Appendix A for more details.

I also include deprivation indicators that measure the proportion of the population receiving job seekers' allowance, the percentage of the population aged 17 or over not going to higher education, the proportion of attendance allowance claimants over 60 years, the proportion of income support claimants over 60 years, the proportion and standardised rate of incapacity benefit/severe disability allowance claimants, and the proportion and standardised rate of attendance allowance/severe disability allowance claimants.

Area deprivation is also measured using house prices (measuring separately the mean area price of detached houses, semi-detached houses, terraced houses, and flats), and area crime rates, (separate rates for violent offences, sexual offences, robbery, burglary from a dwelling, theft of a motor vehicle, and theft from a motor vehicle).

The second set of area based indicators measure the health of the local population. In addition to the health domain from ID2000 three measures of area mortality are used (the all-age standardised mortality ratio [SMR], the SMR among individuals aged 0–64 years, and the SMR among individuals aged 0–74 years), plus the number of births in the local area, and the percentage of births that were of low birth weight.

The third category contains measures of health care supply. Twenty nine indictors measure accessibility to health care in terms of waiting times for hospital services, the number of beds at local hospitals, distance to local hospitals, number of staff at local hospitals, and the distance to and supply of GPs in the local area.

[7] The HSE does not contain data on non-labour income.

3.4. Instrument

In the IV models I instrument individual obesity using the prevalence of obesity in the area in which the respondent lives. This variable was constructed by collapsing individual level values of BMI greater than 30 kg/m^2 measured as a binary variable in the HSE sample across all non-pregnant individuals of working age (18,026 observations) by health authority of residence to produce a dataset of obesity prevalence at the health authority level. The mean number of sample observations per health authority is 190 (range 47 to 405). This health authority level dataset was then merged with the individual level HSE data on respondents' health authority of residence to give for each individual in the sample the prevalence of obesity in the health authority in which they live. Area based measures have been used as instruments for individual level variables in other studies (see for example, Currie and Cole, 1993; Card, 1995; Grabowski and Hirth, 2003; Lo Sasso and Buchmueller, 2004; Sloan et al., 2001).

The effect of area obesity on individual obesity is a peer group effect. Conditional on the other covariates, the effect will be statistically significant for two reasons. First, medical evidence shows that individual obesity is determined by the characteristics of the local population, i.e., food intake and physical activity of peers. Using the terminology of Manski (1993) this as an "exogenous" peer effect. The main risk factors for obesity are excessive intake of high fat and high calorie foods and physical inactivity (NHLBI, 1998). The evidence also shows that environmental influences, which affect attitudes and behaviours to food intake and exercise, are a key determinant of obesity (James, 1995). Obesity prevalence in the local area is a measure of environmental influences that affect obesity; it is a summary measure of food intake and physical activity characteristics of the local population. Hence, based on medical evidence there is a positive correlation between individual and area obesity.

The second reason why area level obesity affects individual obesity is via what Manski describes as an "endogenous" peer effect: the effect of area obesity (peer obesity) on individual obesity all else equal. Holding the characteristics of peers (e.g. their food intake and physical activity) constant, individual obesity is affected by the level of obesity among peers, because it reflects the social norm. In support of this view, there is a small but growing body of evidence which shows that individual obesity is related to the empirical distribution of obesity among peers (Burke and Heiland, 2005a,b).

There is therefore evidence to support a behavioural link between individual and area obesity. The evidence suggests the latter will be a significant predictor of the former, even after controlling for other area variables.

The second requirement of the instrument is that it is not correlated with the error term in the employment equation. If the area prevalence of obesity is correlated with individual employment other than through its impact on individual obesity then plausibly this arises only via its correlation with individual and area deprivation and health (e.g., the local employment rate or level of income). Given that I include a large number of covariates, and in particular a very comprehensive set of area deprivation measures, including measures of employment and income deprivation, then obesity prevalence is not a component of the error term in the employment equation and does not give rise to a correlation between individual obesity and the error term. It is difficult to think of another way in which the area prevalence of obesity affects individual employment other than via its impact on individual obesity, health and/or deprivation. Hence, the instrument is not endogenous, and it passes the orthogonality requirement. Note that this requires that the covariates, in particular the area level indicators, are sufficiently comprehensive that they remove any correlation between area obesity and the error term. This approach is not uncommon: regional variables are often used as instruments for endogenous explanatory variables appearing in individual level equations, which is appropriate provided other regional variables that affect the dependent variable are controlled for (Wooldridge, 2002, p. 89).

To control for the distribution of obesity in the local population I also add to the set of covariates the number of HSE respondents used to generate prevalence of obesity in each health authority, and the standard deviation of BMI in each health authority among HSE respondents.

Four further issues arise from the instrument selection. First, we can now define more specifically the LATE: it is the average impact of obesity on employment for those individuals who became obese only from living in an area with a higher prevalence of obesity. In contrast, the univariate probit and propensity score matching methods give the average effect of obesity on employment among the obese.

Second, in addition to being the core dataset for the analysis, there are good reasons for using the HSE as the data source for area obesity prevalence. The HSE is the only general population survey to routinely collect data on obesity in England. It is also the primary source of epidemiological data that underpins government policy on obesity. Hence, it is the most appropriate source for area obesity prevalence data.

Third, it is possible that observations on peers (i.e. within areas) are not independent; it is therefore necessary to adjust the standard errors in the regressions for within-area correlation (Moulton, 1990).

Fourth, in terms of interpreting the coefficient on area obesity in the obesity equation there is a "reflection problem", identified by Manski (1993), in that it is difficult to separate the two types of peer effect. In other words, it is difficult to identify whether the impact of area obesity on individual obesity is due to peer characteristics (e.g. food intake and physical activity of peers), or peer outcomes all else equal (e.g. obesity of peers holding food intake and physical activity of peers constant). As noted by Ichino and Maggi (2000) without detailed information on the characteristics of the peer group it is not possible to disaggregate the effects. This is problematic if, for example, we wish to evaluate policies for reducing obesity: if peer effects are endogenous then these policies will generate what Manski calls a "social multiplier"; if they are exogenous they will not generate this effect. In this study identification of the peer group effect is not essential; the important point is that the behavioural link between area obesity and individual obesity conditional on the covariates is justified and can be demonstrated empirically.

3.5. Sample size and sampling issues

The total sample size combining all HSE observations across 1997 and 1998 is 35,200. Individual observations are excluded: because they have missing information on employment and/or obesity; because they are outside the normal working age; or, because they are pregnant. It is not possible to include the first group in the estimation sample. It is possible to include the second and third groups, but not very sensible because the effect of obesity on employment will be confounded. Excluding individuals outside the normal working age (18 to 65 years for males, 18 to 60 years for females) reduces the sample to 18,302. 276 pregnant females are dropped from the sample along with a further 19 observations with missing employment data. 1,040 of the remaining 18,007 observations are excluded because they have invalid obesity measures. This reduces the number of observations in the final estimation sample to 16,967, of whom 8,324 are males and 8,643 are females.

In the 1997 and 1998 rounds of the HSE the samples of children but not adults were deliberately boosted to include greater numbers of children. Since adult respondents were not over or under sampled each observation has a weight of unity in the regressions.

As noted above, and following Moulton (1990) who demonstrates the pitfalls in failing to control for within area dependence when estimating the effects of area level variables on individual level outcomes, I adjust the standard errors in the obesity and employment regression models to control for area (health authority) level clustering.

To maximise the sample size I impute missing values for all the covariates. For continuous variables missing values are imputed using the linear prediction from a regression of the variable on the other covariates. For binary and categorical variables missing values are assigned to the omitted category. To allow for the possibility that items are not missing at random I include dummy variables for all imputed items to indicate item non response. I use this approach in preference to other methods for dealing with missing data, such as hotdecking, because in the sample items may not be missing at random. If the dummy variable is insignificant non-responders' employment is affected in the same way as the responders by the imputed variable and the imputation has increased sample size without biasing results. If the dummy variable is significant then responders and non-responders are affected in different ways by the variable and inclusion of the missing item dummy variable enables estimation of an effect for responders that is not contaminated by the imputation for non responders.

4. Results

The proportion of the sample in each obesity category is in Table 1. Only 34% of males and 43% of females are in the healthy category, while 17% and 19%, respectively, are obese. In males the overweight category has the highest proportion of the sample (46%), while in females the healthy category is the largest (43%). Table 1 also shows the percentage of the sample in each category that is employed. In all categories there are more employed males than females. In males the highest proportion employed is in the overweight category, while in females the highest proportion is in the healthy category. 74% of obese males and 62% of obese females are employed. The sample means and standard deviations of the variables used in the analysis are available from the author on request.

The main results are in Tables 2–4. Selected results from the regression models are in Appendix B. The main univariate probit results are in Table 2. The coefficients on the obesity variable, their statistical significance, and the marginal effects are reported, along with the explanatory power of the models measured by the pseudo-R^2. In males obesity has a statistically significant and negative effect on employment, with a marginal effect of −0.021. In females the direct effect of obesity on employment is small, positive and insignificant.

Table 1
Employment by obesity category

Obesity category	BMI (kg/m^2)	Males (n=8,324)		Females (n=8,643)	
		% Sample	% Employed	% Sample	% Employed
Underweight	<20	3	66	7	55
Healthy	20–25	34	78	43	69
Overweight	25–30	46	81	31	66
Obese	>30	17	74	19	62

Table 2
The impact of obesity on employment: univariate probit

	Males			Females		
	Coef.	z	ME	Coef.	z	ME
Males						
Obese	−0.113	−2.1	−0.021	0.015	0.4	0.004
Observations		8,324		8,643		
Pseudo-R^2		0.363		0.218		

Individual level covariates are included in both models for educational attainment, years of full time education, self reported general health, days of acute sickness, longstanding illness, number of longstanding illnesses, GHQ-12 score, marital status, housing tenure, number infants 0 to 1 years in household, number children 2 to 15 years in household, age, ethnic group, year, month of interview and item non-response. Area level covariates are included for rurality, deprivation, health, supply of health services, respondents used to generate obesity prevalence in health authority, standard deviation of BMI in health authority among HSE respondents, and region.
In both models the standard errors are adjusted for health authority level clustering.

With respect to the propensity score matching results, preliminary analyses indicated that a balanced distribution of observed covariates was achieved independently of obesity status (results not shown). The ATT results are in Table 3. Results are presented for the four matching methods with and without common support. In the case of radius matching results

Table 3
The impact of obesity on employment: propensity score matching

	Without common support				With common support			
	No. obese	No. non-obese	ATT	t	No. obese	No. non-obese	ATT	t
Males								
Stratification matching	1,450	7,358	−0.019	−1.5	1,450	7,292	−0.019	−1.6
Nearest neighbour matching	1,450	1,191	−0.024	−1.2	1,450	1,191	−0.024	−1.1
Kernel matching	1,450	6,874	−0.034	−2.9	1,450	6,810	−0.034	−3.0
Radius matching (radius = 0.1)	1,450	6,874	−0.065	−4.5	1,450	6,810	−0.065	−4.6
Radius matching (radius = 0.01)	1,448	6,869	−0.068	−5.1	1,448	6,809	−0.068	−5.2
Radius matching (radius = 0.001)	1,427	6,672	−0.063	−4.6	1,427	6,665	−0.063	−5.0
Radius matching (radius = 0.0001)	1,260	3,462	−0.038	−2.2	1,260	3,462	−0.038	−2.2
Females								
Stratification matching	1,667	7,532	0.002	0.1	1,667	7,530	0.002	0.1
Nearest neighbour matching	1,668	1,328	−0.004	−0.2	1,668	1,328	−0.004	−0.2
Kernel matching	1,668	6,975	−0.013	−1.1	1,668	6,973	−0.013	−1.0
Radius matching (radius = 0.1)	1,668	6,975	−0.063	−5.7	1,668	6,973	−0.063	−4.9
Radius matching (radius = 0.01)	1,651	6,973	−0.061	−4.7	1,651	6,973	−0.061	−4.8
Radius matching (radius = 0.001)	1,607	6,834	−0.052	−3.6	1,607	6,834	−0.052	−3.8
Radius matching (radius = 0.0001)	1,383	4,322	−0.021	−1.2	1,383	4,322	−0.021	−1.1

Individual level covariates are included in all models for educational attainment, years of full time education, self reported general health, days of acute sickness, longstanding illness, number of longstanding illnesses, GHQ-12 score, marital status, housing tenure, number infants 0 to 1 years in household, number children 2 to 15 years in household, age, ethnic group, year, month of interview and item non-response. Area level covariates are included for rurality, deprivation, health, supply of health services, respondents used to generate obesity prevalence in health authority, standard deviation of BMI in health authority among HSE respondents, and region.
The standard error used to compute the t statistic is the standard deviation of the ATT after 200 bootstrap replications.

Table 4
The impact of obesity on employment: bivariate probit

	Males			Females		
	Coef.	z	ME	Coef.	z	ME
Impact of obesity on employment						
Obese	−0.420	−1.6	−0.084	−0.696	−2.9	−0.213
Observations	8,324			8,643		
ρ	0.172			0.400		
Wald test $\rho=0$ [p value]	$\chi^2(1)=1.6$			$\chi^2(1)=7.1$		
	[0.21]			[<0.01]		
	Coef.	z		Coef.	z	
Impact of the instrument on obesity						
Prevalence of obesity	3.643	7.4		3.584	7.6	
Observations	8,324			8,643		
Wald test instrument=0 [p value]	$\chi^2(1)=54.41$			$\chi^2(1)=58.09$		
	[<0.01]			[<0.01]		

Individual level covariates are included in all models for educational attainment, years of full time education, self reported general health, days of acute sickness, longstanding illness, number of longstanding illnesses, GHQ-12 score, marital status, housing tenure, number infants 0 to 1 years in household, number children 2 to 15 years in household, age, ethnic group, year, month of interview and item non-response. Area level covariates are included for rurality, deprivation, health, supply of health services, respondents used to generate obesity prevalence in health authority, standard deviation of BMI in health authority among HSE respondents, and region.
In all models the standard errors are adjusted for health authority level clustering.
The instrument in the IV models is the prevalence of obesity across individuals living in the health authority in which the respondent lives.

are presented for four neighbourhood sizes. The number of obese and matched non-obese is reported in Table 3, along with the ATT and the t statistic derived from the bootstrapping procedure.

In the radius matching models the number of matched obese and non-obese individuals declines as the radius decreases, as expected. The change is substantial when the radius is reduced to 0.0001.

There are few differences between the models run with and without the common support condition. While as expected there are generally fewer non-obese individuals in models run with common support, the point estimate of the ATT is the same as in those models run without it to at least three decimal places.

In males, in all cases except for the nearest neighbour matching models the ATT is significant and negative (with stratification matching the results are borderline significant). It is worth bearing in mind that with nearest neighbour matching some of the matches between obese and non-obese individuals may be poor because for some obese individuals the nearest neighbor may have a very different propensity score but nevertheless contribute to the estimation of the ATT.

Comparing the different matching methods in males, one striking result is the similarity in the magnitude of the ATT, ranging from −0.019 with stratification matching to −0.068 with radius matching (radius=0.01). The ATT is similar, but (with the exception of stratification matching) slightly larger than the ME in the probit model.

In females, except for three of the radius matching models the ATT is generally insignificant. This is consistent with the univariate probit results in females.

The IV regression results are in Table 4. The bottom panel reports the significance of the instrument on obesity. As expected, even after controlling for the full set of covariates, the prevalence of obesity in the local area is a highly significant predictor of individual obesity in both males and females, indicating that the instrument satisfies the non-weakness requirement. The coefficients have a positive sign showing that, as expected, area obesity is positively associated with individual obesity. Appendix C shows the impact of the area prevalence of obesity on individual employment, conditional on the full set of covariates except individual obesity. The prevalence of obesity has a positive and insignificant effect on employment in males and a negative and significant effect in females. The insignificant effect in males suggests that while there is positive correlation between unemployment and individual obesity, and individual obesity and area obesity, there is no correlation between individual unemployment and area obesity. Note that the property of being positively correlated is not necessarily transitive (Langford et al., 2001).

In the top panel of Table 4 the main IV results are reported. In males, obesity has a significant (at the 10% level) and negative effect on employment, with a marginal effect of −0.084. In females the direct effect is also statistically significant and negative: obese females have an employment probability that is 0.213 lower than non-obese females.

ρ is positive. This means that unexplained factors that affect obesity are positively correlated with unexplained factors that affect employment. Using a Wald test I fail to reject the hypothesis that $\rho=0$ in males across the different models. This suggests that, assuming the instrument is valid, the endogeneity of obesity does not significantly affect the univariate probit estimates in males. In females the hypothesis that $\rho=0$ is rejected suggesting that in this group the univariate probit results are biased and underestimate the negative impact of obesity on employment. Comparing the IV results to the propensity score matching results, in males the ME in the IV model is slightly larger but of the same order of magnitude as the ATT in the propensity score matching models. In females the ME is significantly more negative. These results are consistent with the view that ρ is positive, but not significantly so in males.

I reran the regression analyses focusing on the economically active only (i.e., dropping from the sample those who are out of the workforce), and on individuals with 14 or more years of schooling and 11 or fewer years of schooling (the 75th and 25th percentiles of the years of schooling variable in the sample are 14 years and 11 years, respectively). I dropped the education variables when estimating the models for years of schooling groups. I report here the univariate probit results for males and the IV results for females. Limiting the sample to those who are economically active suggests that obesity has a negative but significant (at the 10% level) effect on employment in males (the marginal effect is −0.013), and a negative but insignificant effect in females. The lack of a significant effect in females may be due to the small number of females in the sample who were economically active but unemployed (322 of 5,998 economically active females) or it may be due to the fact that in females obesity has a greater effect on being out of the workforce than being active but unemployed.

More educated males who are obese are significantly less likely to be employed than educated males who are not obese (the marginal effect is −0.046), with a negative and insignificant effect among less well educated males. In females the impact of obesity on employment is negative and insignificant among those with 14 or more years of schooling and negative and significant among those with 11 or fewer years (the marginal effect is −0.306).

5. Concluding remarks

In this paper I investigate the impact of obesity on employment in England using three different methods and a dataset containing a rich set of variables likely to affect employment. Assuming plausibly that the instrument is valid I identify the causal effect of obesity on employment from other factors that might cause the two variables to be correlated.

In the IV models I find that obesity has a statistically significant and negative impact on employment in both males and females. The impact of obesity in the IV models is more negative than in the other models, which shows that failure to account for endogeneity will underestimate the negative impact of obesity on employment. I find that the upward bias obtained without correcting for endogeneity is statistically significant in females but not in males. This suggests that it is appropriate to use the IV results in females but that the other methods may be appropriately used in males.

In males the univariate probit and propensity score matching also reveal a significant and negative impact of obesity on employment, which is slightly lower but of the same order of magnitude as the IV results. This small difference may be due in part to the (insignificant) endogeneity of obesity, but also due to the fact that the univariate probit and propensity score matching provide measures of the ATT while the focus in the IV regression is on the LATE.

In females the overall finding is also of a negative impact of obesity on employment. The IV results show that after controlling for unobserved heterogeneity the impact of obesity on employment is more negative than with the other methods. This indicates that there are omitted variables that are positively correlated with both obesity and employment. For example, it is plausible that the obese have a higher time preference rate and so put less emphasis on their future health, and also that individuals with a higher time preference rate are less likely to invest in their human capital and so are more likely to be employed rather than be out of the labour force in the education sector. The findings are also consistent with evidence that the obese are more likely to understate their true weight, as are those in employment (Boström and Diderichsen, 1997), though measurement error is unlikely to be the cause of the bias since the obesity measure relies on objective nurse visits rather than self reports.

Comparing the IV results for males and females, the findings also suggest that the negative consequences of obesity on employment are greater for females than for males. This result is consistent with the findings from other studies analysing the impact of obesity on labour market outcomes (Baum and Ford, 2004; Cawley, 2004; Sargent and Blanchflower, 1994; Harper, 2000; Sarlio-Lahteenkorva and Lahelma, 1999).

Overall, the findings in this study demonstrate that obesity has a negative impact on employment. This is likely to arise because obesity is a debilitating health condition that has an independent effect on productivity and therefore employment. It might also arise due to discrimination against the obese. Further research is needed to identify the nature and extent of the discrimination effect.

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Appendix A

A.1. The Indices of Deprivation 2000

The Indices of Deprivation 2000 (ID2000) comprises seven domains or dimensions of deprivation measured at the small area (ward) level (DTLR, 2000). The ID2000 includes measures of:

- Income deprivation (measured in the income domain)
- Child poverty (a subset of the income domain, measured in the child poverty domain)
- Employment deprivation (employment domain)
- Health deprivation and disability (health domain)
- Education, skills and training deprivation (education domain)
- Housing deprivation (housing domain)
- Deprived geographical access to services (access domain)

The income domain measures the proportion of the total population who are on a low income and in receipt of means tested benefits. The child poverty domain is based on a subset of these indicators and reflects the proportion of children living in low-income households. The employment domain measures enforced exclusion from work through unemployment, sickness or disability. The health domain identifies areas with higher than expected numbers of people whose quality of life is impaired by poor health and disability or whose life is cut short by premature death. The education domain measures the key educational characteristics of an area that contribute to the overall level of deprivation and disadvantage. The housing domain covers those in unsatisfactory housing, including the homeless. The access domain measures the extent to which people have poor geographical access to certain key services. The overall index of deprivation is constructed by combining the weighted, exponentially transformed, ranks of each domain. The components of each domain are listed below.

A.2. Components of each domain in the ID2000

Income domain (the first four items are also used in the child poverty domain)
Children in Income Support households for 1998
Children in Income Based Job Seekers Allowance households for 1998
Children in Family Credit households for 1999
Children in Disability Working Allowance households for 1999
Adults in Income Support households for 1998
Adults in Income Based Job Seekers Allowance households for 1998
Adults in Family Credit households for 1999
Adults in Disability Working Allowance households for 1999
Non-earning, non-IS pensioner and disabled Council Tax Benefit recipients for 1998
Employment domain
Unemployment claimant counts for 1998
People out of work but in TEC delivered government supported training January 1999
People aged 18–24 on New Deal options June 1999
Incapacity Benefit recipients aged 16–59 for 1998
Severe Disablement Allowance claimants aged 16–59 for 1999

Health domain
Comparative Mortality Figure for men and women at ages under 65 for 1997 and 1998
Proportion receiving Attendance Allowance or Disability Living Allowance in 1998
Proportion receiving Incapacity Benefit or Severe Disablement Allowance for 1998 and 1999
Age and sex standardised ratio of limiting long-term illness for 1991
Proportion of births of low birth weight (<2,500 g) for 1993–97

Education domain
Working age adults with no qualifications for 1995–1998
Children aged 16 and over who are not in full-time education for 1999
Proportions of 17+ population who have not successfully applied for HE for 1997 and 1998
Key stage 2 primary school performance data for 1998
Primary school children with English as an additional language for 1998
Absenteeism at primary level for 1998

Housing domain
Homeless households in temporary accommodation 1997–98
Household overcrowding for 1991
Poor private sector housing for 1996

Access domain
Access to a post office for April 1998
Access to large food shops 1998
Access to a GP surgery for October 1997
Access to a primary school for 1999

Appendix B

B.1. Selected regression results of the impact of obesity on employment

	Males				Females			
	Univariate probit		Bivariate probit		Univariate probit		Bivariate probit	
	Coef.	z	Coef.	z	Coef.	z	Coef.	z
Obese	−0.113	−2.1	−0.420	−1.6	0.015	0.4	−0.696	−2.9
Education variables								
Educational attainment [a]								
Higher Education less than a degree	−0.123	−1.7	−0.112	−1.6	−0.151	−2.2	−0.119	−1.7
A level or equivalent	−0.267	−3.6	−0.261	−3.6	−0.396	−6.0	−0.376	−5.7
GCSE or equivalent	−0.173	−2.4	−0.166	−2.4	−0.361	−5.5	−0.331	−5.0
CSE or equivalent	−0.107	−1.3	−0.091	−1.1	−0.453	−4.6	−0.395	−4.1
Other qualification	−0.270	−1.7	−0.258	−1.6	−0.453	−5.2	−0.411	−4.5
No qualification	−0.335	−4.1	−0.320	−4.1	−0.724	−9.5	−0.670	−8.9
Years of full-time education	0.114	1.9	0.111	2.0	0.068	1.3	0.051	0.9
Years of full-time education squared/100	−0.714	−2.8	−0.709	−2.9	−0.548	−2.3	−0.489	−2.0
Selected health variables								
Self reported general health [b]								
Good	−0.083	−1.8	−0.067	−1.4	0.009	0.2	0.036	0.9
Fair	−0.386	−7.3	−0.354	−6.0	−0.191	−3.4	−0.126	−2.2
Bad	−1.279	−10.8	−1.237	−10.0	−0.825	−7.8	−0.695	−5.5
Very bad	−1.918	−6.6	−1.866	−6.4	−0.912	−4.9	−0.776	−3.9
Limiting longstanding illness	−0.452	−6.9	−0.450	−6.8	−0.304	−5.1	−0.310	−5.1

(continued on next page)

Appendix B (*continued*)

	Males				Females			
	Univariate probit		Bivariate probit		Univariate probit		Bivariate probit	
	Coef.	z	Coef.	z	Coef.	z	Coef.	z
Home and family variables								
Marital status [c]								
Married	0.327	5.5	0.345	5.8	−0.042	−0.8	−0.024	−0.5
Separated	0.142	1.0	0.152	1.1	−0.101	−1.0	−0.102	−1.0
Divorced	−0.066	−0.8	−0.064	−0.8	0.030	0.4	0.025	0.4
Widowed	0.163	0.9	0.176	1.0	−0.146	−1.2	−0.118	−0.9
Housing tenure [d]								
Buying with a mortgage	0.446	9.1	0.439	9.0	0.366	7.0	0.359	7.1
Part rent part mortgage	−0.286	−0.8	−0.277	−0.7	0.029	0.1	0.066	0.3
Rent	−0.359	−5.9	−0.357	−5.8	−0.239	−4.0	−0.205	−3.2
Living rent free	0.121	0.7	0.116	0.7	0.190	1.0	0.261	1.4
No. infants 0 to 1 years in household [e]								
1	0.065	0.6	0.072	0.6	−0.921	−16.8	−0.876	−14.4
2	−0.449	−1.6	−0.473	−1.7	−1.571	−6.2	−1.483	−5.5
No. children 2 to 15 years in household [h]								
1	−0.001	0.0	−0.004	−0.1	−0.408	−7.9	−0.390	−7.5
2	−0.079	−1.1	−0.080	−1.1	−0.789	−15.3	−0.767	−13.7
3	−0.186	−1.5	−0.190	−1.5	−0.955	−10.7	−0.922	−10.0
4	−0.391	−1.8	−0.371	−1.8	−1.486	−9.2	−1.427	−9.1
5	−0.884	−1.9	−0.890	−1.8	−1.661	−4.4	−1.652	−4.4
6	−0.652	−1.6	−0.685	−1.8	−0.904	−1.7	−0.938	−1.8
Selected additional control variables								
Age/100	23.541	4.2	23.668	4.2	28.848	4.8	28.580	5.0
Age/100 squared	−31.495	−2.3	−31.505	−2.3	−48.055	−3.1	−47.487	−3.2
Age/100 cubed	1.525	0.2	1.402	0.1	16.951	1.3	16.919	1.4
Ethnic group [f]								
Black Caribbean	−0.469	−3.2	−0.486	−3.3	−0.100	−0.6	−0.021	−0.1
Black African	−0.380	−1.9	−0.353	−1.7	−0.109	−0.9	−0.036	−0.3
Black other	−0.608	−1.6	−0.611	−1.6	−0.722	−2.4	−0.688	−2.3
Indian	−0.491	−4.0	−0.519	−4.3	−0.240	−2.2	−0.258	−2.3
Pakistani	−0.298	−2.2	−0.318	−2.4	−0.956	−5.6	−0.959	−5.8
Bangladeshi	−0.742	−2.0	−0.754	−2.0	−0.520	−1.9	−0.567	−1.9
Chinese	−0.411	−2.9	−0.413	−2.9	−0.234	−1.6	−0.249	−1.8
Other non−white ethnic group	−0.469	−3.2	−0.486	−3.3	−0.100	−0.6	−0.021	−0.1
Rurality [g]								
Suburban	0.056	0.8	0.048	0.7	0.136	2.8	0.125	2.5
Rural	0.165	2.4	0.152	2.2	0.119	2.1	0.107	2.0
Observations	8,324		8,324		8,643		8,643	
Pseudo-R^2	0.363				0.218			
ρ			0.172				0.400	
Wald test $\rho=0$ [p value]			$\chi^2(1)=1.6$ [0.21]				$\chi^2(1)=7.1$ [<0.01]	

Individual level covariates are also included for days of acute sickness, types of longstanding illness, number of longstanding illnesses, GHQ-12 score, year, month of interview and item non-response. Area level covariates are also included for rurality, deprivation, health, supply of health services, respondents used to generate obesity prevalence in health authority, standard deviation of BMI in health authority among HSE respondents, and region.
In all models the standard errors are adjusted for health authority level clustering.
[a] The baseline category is Degree.
[b] The baseline category is Very good.
[c] The baseline category is Single.
[d] The baseline category is Own outright.
[e] The baseline category is 0.
[f] The baseline category is 0.
[g] The baseline category is White.
[h] The baseline category is Urban.

Appendix C

C.1. Impact of the instrument on employment

	Males		Females	
	Coef.	z	Coef.	z
Prevalence of obesity	0.649	0.8	−2.068	−3.2
Observations	8,324		8,643	
Wald test instrument=0 [p value]	$\chi^2(1)=0.6$ [0.43]		$\chi^2(1)=10.2$ [<0.01]	

Individual level covariates are included for educational attainment, years of full time education, self reported general health, days of acute sickness, longstanding illness, number of longstanding illnesses, GHQ-12 score, marital status, housing tenure, number infants 0 to 1 years in household, number children 2 to 15 years in household, age, ethnic group, year, month of interview and item non-response. Area level covariates are included for rurality, deprivation, health, supply of health services, respondents used to generate obesity prevalence in health authority, standard deviation of BMI in health authority among HSE respondents, and region.
In both models the standard errors are adjusted for health authority level clustering.

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