GOING THE SAME 'WEIGH'?
SPOUSAL CORRELATIONS IN OBESITY IN THE UK

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1. Introduction

The UK, along with many developed nations, has seen a significant rise in obesity rates over the last few decades. A quick search on Google reveals around 22 million websites related to weight loss tips. However, no matter how many websites claim to know the secrets of successful weight loss, the causes of obesity are still not completely understood. It is likely that the current obesity epidemic cannot be explained solely by genetic factors. Rising obesity rates have been partially attributed to environmental factors as well as technological change and innovations which have led to a more sedentary lifestyle, and a subsequent energy imbalance resulting in weight gain (Philipson and Posner 1999, Peters 2003, Jeffery and Utter 2003, Lin et al. 2004).

Christakis and Fowler (2007) found using data on an adult population in the US that social networks significantly impacted the likelihood of becoming obese. Cohen-Cole and Fletcher (2008) claim using data from an adolescent population in the US that after controlling for contextual factors such as the local environment, social networks no longer significantly impacted the likelihood of becoming obese. It is likely that the stage of life you are at may impact the importance of social networks on the likelihood of being obese. The influence of social networks may also differ by country. This paper attempts to build upon the work of Christakis and Fowler (2007) by including contextual factors in our analysis on an adult population. We also use British data as differences in the built environment and lifestyles between the US and the UK may influence correlations in obesity.

To describe spousal[1] correlations in obesity we adopt the Manski (1993) approach used in Christakis and Fowler (2007), to explain the influence of social networks on the spread of obesity. Firstly, individuals may choose to marry someone with similar characteristics as described in the theory of marriage proposed by Becker (1974). This is analogous to correlated effects in Manski's terminology. Secondly, correlations in obesity between partners may be observed because they share the same environment (contextual factors). For example, spouses face the same local prices and food choices. This is equivalent to exogenous effects in Manski (1993). Finally, the propensity of an individual to behave in a certain way may vary with the behaviour of their spouse (social influence). Similar

[1] Following Clark and Etile (2006) spouse and partner are used interchangeably to refer to heterosexual couples who are legally married or cohabiting. Same sex couples are not included in our analysis due to small sample sizes in our data.

consumption patterns which develop over the marriage or spousal behaviours and attitude about weight may lead to correlations in obesity. This is referred to as to endogenous effects in Manski (1993). These three factors are not necessarily mutually exclusive.

We use waves 14 and 16 (2004 and 2006) of the British Household Panel Survey (BHPS) to analyse the correlation in obesity between spouses. These two waves are the only ones in which information on height and weight is available, enabling calculation of Body Mass Index (BMI). BMI is the standard measure to used assess and grade obesity (World Health Organisation (WHO) 1995, 2000, 2004). It is calculated as weight in kilograms divided by height in metres squared. Individuals are classified as obese if their BMI is 30 kg/m^2 or greater. In this paper we use a number of econometric techniques to firstly, analyse the impact of partner characteristics and environmental factors on own likelihood of being obese, and then examine the correlation in the error term from a pair of partner obesity equations. Differences in gender are also explored.

This paper is organised as follows. Section 2 discusses the relevant literature in the context of the theoretical and empirical framework. Section 3 describes the theoretical framework which informs the empirical analysis. Section 4 outlines the data used in the analysis. Section 5 explains the econometric approach. The results and discussion are presented in Section 6. Finally, Section 7 concludes, and as this paper represents work in progress we also outline further work we will do.

2. Previous Literature

There is an extensive literature examining various areas of spousal correlation, including education (Mare 1991, Pencavel 1998, and Qian 1998), health (Wilson 2002), to lifestyle characteristics such as drinking habits (Leonard and Mudar 2003), and smoking patterns (Clark and Etile 2006). However, the impact of spousal interactions on the likelihood of being obese has not been widely studied. Sahn and Younger (2009) look at intra-household BMI as a measure of well-being and inequality in developing countries. In a similar context to the aims of this paper, Christakis and Fowler (2007) examine how spousal interactions influence own likelihood of becoming obese using US data.

Christakis and Fowler (2007) use a cohort from the Framingham Heart Study (1971-2003), identifying 5124 core respondents aged 21 and over, which they term ‘egos’, and 12,607 individuals connected to the respondent in some way that may influence behaviour, denoted as ‘alters’. They adapt Manski’s (1993) approach to explain social interactions, arguing that correlations in obesity can be determined by: 1) shared individual characteristics (which they refer to as homophily); 2) a shared environment (confounding factors); and 3) social influences (induction). They test these hypotheses by analysing the effects of friendship, family, and marital relationships on obesity. Results for married couples indicate that if one spouse became obese the likelihood of the other spouse becoming obese increased by 37%. In addition, men and women were found to have similar effects on the increased probability of each spouse becoming obese.

Cohen-Cole and Fletcher (2008) use US data from the nationally representative Add Health Data Set for 12 to 18 year olds in 1994/1995. They attempt to address some of the weaknesses of Christakis and Fowler (2007) by trying to distinguish between 'confounding' factors and 'induction' effects through the addition of time invariant environmental factors and time variant school specific factors for both individuals and their 'alters'. They find that confounding factors explain the majority of the correlation in friends' likelihood of being obese; refuting the claims made by Christakis and Fowler (2007).

Our paper makes a number of important contributions to the literature. Firstly, we focus primarily on spousal correlations in obesity using a variety of econometric techniques to distinguish between shared individual characteristics, confounding factors, and induction effects. In our analysis we include regional variables as well as labour market variables to build upon the work of Christakis and Fowler (2007). We can therefore test the hypothesis, proposed by Cohen-Cole and Fletcher (2008) that including a richer set of contextual factors in the analysis reduces the significance of social networks on explaining correlations in obesity. Secondly, we use British data to examine spousal correlations. There are large differences in the built environment and lifestyles in the US and UK which may impact the effect of confounding factors on spousal correlations in obesity. For example, for individuals living in rural communities in the Mississippi delta there is on average one supermarket every 190.5 square miles (Blanchard and Lyson 2003). Whereas the average trip for food shopping for a rural resident in the UK is 5.8 miles (Department of Transport 2007). Finally, the majority of the explanatory variables used in the analysis are demand side variables such as individual characteristics and labour market variables. The economic literature which has examined the causes of obesity (for example Lakdawalla and Philipson 2002, Chou et al. 2004, and Rashad et al. 2006) has primarily focused on supply-side factors; therefore it is interesting to analyse how demand side factors, which are determined by individual and household behaviour, impact the likelihood of being obese.

3. Theoretical Framework

This paper adopts the framework described in the seminal work of Manski (1993) to explain correlated outcomes within a group. Following, Christakis and Fowler (2007) we apply this general framework to examine spousal correlations in obesity, which fall into three possible categories: 1) shared individual characteristics (or homophily); 2) contextual or exogenous (confounding) factors; and 3) social influences (induction).

Hypothesis 1: Shared individual characteristics

Spousal correlations in obesity may be the result of spouses sharing similar individual characteristics or positive assortative mating in the marriage market. Becker (1974) developed a theory of marriage based upon the gains of partnership accruing to two rational individuals. Each individual has a set of observable individual characteristics such as weight and smoking status which signal general preferences over other activities and goods such as eating healthy food, exercising, and socialising. These characteristics can then be combined with the characteristics of potential partners to produce household commodities.

It is likely that two types of assortative mating will occur in the marriage market. Firstly, couples may sort according to variables that indirectly affect the likelihood of becoming obese such as education, health, and socioeconomic status. If this is the case, then marital choice has an indirect effect on correlation in spouses' obesity, as Wilson (2002) discussed in relation to spousal correlation in health. Secondly, being obese can also signal preferences for other lifestyle characteristics such as how one chooses to spend leisure time, which may impact the likelihood of being obese. For example, if individuals prefer sedentary leisure activities such as surfing on the internet or watching TV this may increase their likelihood of becoming obese. Contoyannis and Jones (2004) found that healthy and unhealthy lifestyle characteristics tend to cluster in individuals. An individual may then choose a partner who enjoys similar activities to maximise the household production function. It is also possible that being obese may act as an observable signal for unobserved characteristics such as future health and potential life expectancy. Risk aversion to time spent alone in widowhood will result in preferences for partners whose life expectancy will match one's own (Clark and Etile 2006). Finally, if individuals have preferences over appearance, then being obese acts as an observable signal of a person's weight to height ratio[2].

In sum, matching in the marriage market will result in correlated time invariant effects in partners' behaviours. In terms of the empirical analysis, in the current paper we test the direct effects of partner characteristics on spousal correlations in obesity.

Hypothesis 2: Contextual or exogenous factors

Correlations in spousal obesity may be caused by contextual or exogenous effects. Married individuals share the same environment which may lead to correlations in the likelihood of being obese. Access to cycle and walking paths, sports facilities, as well as shops and other amenities within walking distance may impact the likelihood of being obese (Egger and Swinburn 1997). For example, if there

[2] The analysis uses only waves 14 and 16 of the BHPS, therefore have insufficient time periods to distinguish between those couples where one or both spouses were obese at the time of marriage and those couples were one or both spouses became obese over the course of the relationship.

are no opportunities for local physical activity, individuals may be less likely to exercise on a regular basis which could lead to weight gain. This effect on BMI may be endogenous if individuals who do not enjoy physical activity exercise this preference in their choice of home location. It is likely that shared leisure time and taste for leisure activities, which may be influenced by the environment, will develop during marriage, as opposed to before marriage as in Becker (1974).

The number of fast food outlets in the local area may also influence the likelihood of being obese. If cheap unhealthy food is readily available individuals may choose to save time by purchasing food from these outlets rather than consuming healthier time intensive home cooked meals. Jeffery et al. (2006) found that eating at fast-food restaurants was positively associated with BMI; however, proximity to fast-food restaurants was not associated with an increased likelihood of eating at these outlets.

Empirically, if after controlling for individual effects in the model testing variables related to contextual factors, the error terms are correlated in the system of partner equations, this would suggest that contextual factors may explain some of the correlations in spousal obesity.

Hypothesis 3: Social Influences

Spousal correlations in obesity may be influenced by the behaviours of one's spouse. For example, spouses are likely to have meals together and buy joint groceries leading to similar consumption patterns which may influence the likelihood of being obese. Spousal attitudes towards BMI may influence an individual's attitude towards weight maintenance and the 'ideal' weight. Oswald and Powdthavee (2007) theorise about the contagious effects of obesity; if your neighbour becomes obese and you have a concave utility function, you will choose to become fatter as well. This fits within the general literature relating to local norms (see for example Clark (2003) on unemployment and Luttmer (2005) on wellbeing). Social norms influencing behaviour can be used to explain how if one spouse becomes obese, the other partner may change their perception of an 'ideal' weight causing them to also become obese.

As was the case with contextual factors, if after controlling for individual effects, own likelihood of being obese and partner likelihood of being obese are positively correlated in the model testing this hypothesis, this would suggest that social influences may explain some of the correlations in spousal obesity. If time varying partner characteristics, such as labour market variables, dependent children, and physical activity participation, significantly impact own likelihood of being obese this may also suggest that these social influences impact own likelihood of being obese.

4. The Data

We use data from waves 14 and 16 (2004 and 2006) of the British Household Panel Survey (BHPS). Waves 14 and 16 are currently the only two waves which contain information on respondent's height and weight allowing one to calculate BMI. The BHPS is an annual longitudinal study which started in 1991 with approximately 5000 nationally representative private households, where individuals aged 16 or older are surveyed[3]. Additional samples of 1500 households for both Scotland and Wales were added in 1999, and 2000 households in Northern Ireland in 2001. The BHPS questionnaire covers a wide range of topics ranging from employment status, wages, various health measures, and education.

For the empirical analysis, we use a sample of couples who remain together during the period in question (2004-2006), and for whom information on both partners are available. The sample is restricted to individuals of typical working age (18-65) to focus on spousal correlations in this important demographic group. The sample consists of 1184 individuals, or 592 men and women in each wave who have valid height and weight data. While it is possible that this balanced sample is not representative of all couples, since some will separate during the period of analysis, we do not feel this attrition will pose a serious problem over the short period (2 years) in question.

4.1 Obesity

Obesity is measured using the standard WHO definition (1995, 2000, 2004) and is a binary variable taking the value of one if BMI is equal to or greater than 30 kg/m^2 or zero otherwise. The obesity measure is computed from self-reported height and weight which may be prone to measurement error. A follow up question reveals that a majority of men and women from the BHPS are 'fairly sure' about their weight measurement. Approximately 20% of men and 24% of women respondents in waves 14 and 16 of the BHPS (2004 and 2006) are classified as obese compared to 24% for both genders in data taken from the Health Survey of England (HSE) 2007, where height and weight measures are obtained by a nurse. Given the similarities in proportions of obese individual in the BHPS and HSE samples and the self-declared accuracy of the weight measure, it is likely that measurement error should not significantly impact the results.

4.2 Covariates

In order to examine the impact of shared individual characteristics (hypothesis 1) and social influence (hypothesis 3) on explaining spousal correlations in obesity, the analysis includes a number of demand side variables. Individual characteristics and labour market variables will influence how much time is devoted to weight maintaining activities such as active leisure (exercise) and preparing and consuming healthy food.

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[3] From wave 9 youth data for household members aged 11-15 were included in the survey. Height and weight measures for youth respondents were provided in waves 14 and 16; however in this analysis the youth data is not used.

Firstly, starting with the explanatory variables related to hypothesis 1, education which is usually determined before marriage, acts as an important signal to potential partners in the marriage market. The empirical literature has mostly found positive assortative mating on education (Mare 1991, Pencavel 1998, and Qian 1998). If higher levels of education increase health knowledge, it is possible that those with more education may be more likely to engage in weight maintaining activities, after controlling for individual time preferences.

If an individual chooses a spouse based upon lifestyle characteristics that influence health and BMI, such as preferences for exercise, eating healthy food, and smoking status it is likely that spouses' health will be correlated and may indirectly influence individual BMI (see Wilson 2002). There is substantial evidence from the medical literature (for example, Must et al. 1999, Mokdad et al. 2003, and WHO 2006) that increasing BMI is associated with higher morbidity. Thus, it is likely that those with a higher BMI are more likely to be in poor health. Positive assortative mating over lifestyle characteristics and preferences may also directly include BMI.

Next we will discuss the explanatory variables that are evolving over the span of the marriage and are thus associated with social influences (hypothesis 3). Dependent children will influence how parents allocate their time between market work, non-market work, and leisure. Numerous studies have found that the number of children significantly impact how much time parents devote to exercise (Verhoef and Love 1992, Strenfeld et al. 1999, and Cody and Lee 1999). More children may mean that parents have less time to devote to personal leisure which may impact their likelihood of being obese. On the other hand, it is also possible that parents engage in more active leisure with their children helping them to maintain a healthy BMI.

Variables related to the labour market and the decision to work will affect the position of the budget constraint and the allocation of time, which may influence individual and household BMI. According to neoclassical economic theory, higher levels of non-labour income will increase an individual's reservation wage and decrease the likelihood of participating in the labour market. If this is the case, then higher levels of non-labour income will result in more time devoted to leisure and other activities that help maintain a healthy BMI, conditional on individual preferences.

Employment status will affect how much time is spent participating in active leisure or home production such as cooking meals. Chou et al. (2002) hypothesised that the rise in female labour supply since the 1970s, coupled with the growing availability of restaurants and other alternative sources of cheap food increased the likelihood of being obese; as more women participated in the labour market there is less household time spent on home production and at home meal preparation. On a related note, the amount of hours worked may also influence the likelihood of being obese, if full-time employees have less time to devote to weight maintaining activities than those who work part-time. Nomaguchi and Bianchi (2004) found that part-time employees participate in more physical activity, than their full-time counterparts. However, if individuals choose to work part-time because of poor health, which may or may not be the result of obesity, this could result in part-time employment being associated with an increased likelihood of being obese. If this is true, then hours worked would be endogenously related to the likelihood of being obese, resulting in an upward bias on the estimated coefficient.

Depending on whether the income or the substitution effect dominates, a higher hourly wage rate will either cause individuals to allocate more or less time to market work respectively, impacting the time available for weight maintaining activities. Spouse's wage may also determine how much time each partner chooses to devote to market work which may influence both partners' likelihood of being obese.

In this analysis we control for supply side factors by using a regional variable and the mean BMI of the local authority area. It is assumed that these variables will estimate the effects of contextual factors (hypothesis 2) on spousal correlations in obesity. A complete list of the variables used in this analysis are presented in Appendix A.

4.3 Descriptive Statistics

Descriptive statistics for the sample are shown in Table 1 (at end). The hourly wage is higher for men than women in all weight classifications, with both obese men and women commanding the highest mean hourly wage. More obese men and women are likely to consider themselves in poor health compared to their healthy weight and overweight counterparts. More men in the healthy weight category are smokers and there is a reversed u-shape relationship between smoking and the weight classification groups for women. There does not seem to be a clear relationship between obesity and education for either gender. These descriptive statistics are consistent with our expectations of the effect of individual characteristics on the likelihood of being obese.

Spousal probabilities for having a partner in each BMI category are shown in Table 2. For each BMI category it is more likely to have a spouse with a similar BMI classification.

Table 2: Conditional probabilities of spousal BMI categories

	Partner BMI	18.5-25 kg/m^2 (Healthy weight)	25-30 kg/m^2 (Overweight)	> 30 kg/m^2 (Obese)
Own BMI	18.5-25 kg/m^2 (Healthy weight)	0.56	0.35	0.19
	25-30 kg/m^2 (Overweight)	0.34	0.41	0.25
	> 30 kg/m^2 or (Obese)	0.18	0.48	0.34

The table should be read as: Given that you are healthy weight what is the probability that you have a healthy weight, overweight, or obese partner, and the same for the other two BMI categories.

5. Econometric Framework

The first step in this analysis is to establish if and how partner characteristics impact own likelihood of being obese using a random effects probit and a Mundlak (1978) probit model. The significance of time invariant and time varying partner characteristics suggests that partner characteristics and behaviour impact own likelihood of being obese. Next, to distinguish between hypotheses (1), (2), and (3) in explaining spousal correlations in obesity, a pooled bivariate probit model and a Mundlak bivariate model are estimated. The models are described below.

5.1 Random Effects and Mundlak Probit Model

Let $Y_{i,t}^{*}$ be an individual's propensity to be obese in each time period which can be equal to zero for men, *M* and women, *F* respectively. From this the dependent variable, $Y_{i,t}$ is a binary indicator for observed BMI, which is equal to one if BMI is equal to or greater than 30 kg/m^2 (obese), and is zero otherwise. Suppose that $Y_{i,t}$ is a linear function of time varying individual and partner characteristics *X,* time invariant individual and partner characteristics *Z,* time varying individual and partner characteristics which are expected to evolve over the course of the marriages such and the children variable as well as individual and partner labour market variables *L,* regional variables *R,* time varying individual and partner health variables *H,* and a time varying individual and partner physical activity variable *A*:

$$Y_{i,t}^{M} = \beta_M X_{i,t}^{M} + \beta_F X_{i,t}^{F} + \gamma_M Z_{i}^{M} + \gamma_F Z_{i}^{F} + \zeta_M L_{i,t}^{M} + \zeta_F L_{i,t}^{F} + \psi R_{i,t}^{M} + \xi_M H_{i,t}^{M} + \xi_F H_{i,t}^{F} + \kappa_M A_{i,t}^{M} + \kappa_F A_{i,t}^{F} + v_{i,t}^{M} \qquad (1)$$

$$Y_{i,t}^{F} = \beta_F X_{i,t}^{F} + \beta_M X_{i,t}^{M} + \gamma_F Z_{i}^{F} + \gamma_M Z_{i}^{M} + \zeta_F L_{i,t}^{F} + \zeta_M L_{i,t}^{M} + \psi R_{i,t}^{F} + \xi_F H_{i,t}^{F} + \xi_M H_{i,t}^{M} + \kappa_F A_{i,t}^{F} + \kappa_M A_{i,t}^{M} + v_{i,t}^{F}$$

$v_{i,t}^{M} = \alpha_{i}^{M} + u_{i,t}^{M}$ and $v_{i,t}^{F} = \alpha_{i}^{F} + u_{i,t}^{F}$

Where the superscripts *M* and *F* represent men and women respectively, therefore, in the male equation all variables denoted by an *F* superscript are for partner characteristics and the converse is true for the female equations. i indexes individuals in time *t,* and the error term, $v_{i,t}$ is comprised of individual specific unobservable effects, α_i and a random error term, $u_{i,t}$.

If the unobserved individual effects are correlated with the explanatory variables then the random effects specification may give inconsistent results because it violates the condition: $\alpha_i \mid X_i, Z_i, L_i, R_i, H_i, A_i \sim IN(0,\sigma_\alpha^2)$. This is likely to be the case as many of the unobserved individual effects such as skill, motivation, and genetic predisposition towards being obese are likely to be correlated with the explanatory variables. Mundlak (1978) proposed a method to overcome this problem, that accounts for the positive correlation between the explanatory variables X_i, Z_i, L_i, H_i, R_i, and A_i, and the individual effects α_i by modelling this relationship explicitly through taking the group means of the time varying explanatory variables:

$$\overline{X}_i = (1/T)\sum_{t=1}^{T} X_{i,t}, \overline{L}_i = (1/T)\sum_{t=1}^{T} L_{i,t}, \text{ and } \overline{H}_i = (1/T)\sum_{t=1}^{T} H_{i,t} \tag{2}$$

(M and F subscripts are dropped here for ease of exposition)

In the Mundlak probit model the group means in (2) are included as additional explanatory variables in the regressions in equation (1), as a proxy for the time invariant individual effects, α_i. Modelling this dependence allows for unbiased estimation of β,γ,ψ,ζ, and κ, regardless of whether or not X_i, Z_i, L_i, H_i, R_i, and A_i, and α_i are independent (Ebbes et al. 2004).

5.2 Pooled Bivariate and Mundlak Bivariate Probit Models

The pooled bivariate probit model and Mundlak bivariate probit model are estimated to analyse correlations in the residuals of the spousal obesity equations. Four different model specifications are estimated, each with a different set of covariates. Model 1 includes individual characteristics, health, and physical activity variables. Model 2 includes the variables in Model 1 with the addition of labour market variables and the children variable. Model 3 includes the variables in Model 1 with the regional variables. Finally, Model 4 includes all the covariates. These different model specifications are estimated to distinguish between variables relating to the three hypotheses. For example, if after controlling for individual effects in Model 2, ρ is significantly different from zero, this would suggest that hypothesis (3) may explain some of the correlations in spousal obesity. Likewise if ρ is significantly different from zero in the specification of Model 3 which controls for individual effects this would suggest that hypothesis (2) explains some of the correlations in spousal obesity. It is expected that the magnitude of the correlation coefficient will be smaller in Model 4 which includes all of the explanatory variables.

The variables used in the pooled bivariate and Mundlak bivariate probit are the same as in the previous models (equations (1) and (2)). However in this case, rather than including partner characteristics as additional explanatory variables, separate male and female obesity equations are estimated simultaneously:

$$Y_{i,t}^{M} = \beta X_{i,t}^{M} + \gamma Z_{i}^{M} + \zeta L_{i}^{M} + \psi R_{i,t}^{M} + \xi H_{i,t}^{M} + \kappa A_{i,t}^{M} + v_{i,t}^{M}$$
$$Y_{i,t}^{F} = \beta X_{i,t}^{F} + \gamma Z_{i}^{F} + \zeta L_{i}^{F} + \psi R_{i,t}^{F} + \xi H_{i,t}^{F} + \kappa A_{i,t}^{F} + v_{i,t}^{F} \qquad (3)$$

$$Cov[v_{i,t}^{M}, v_{i,t}^{F} \mid X_{i,t}^{M}, X_{i,t}^{F}, Z_{i}^{M}, Z_{i}^{F}, L_{i,t}^{M}, L_{i,t}^{F}, H_{i,t}^{M}, H_{i,t}^{F}, A_{i,t}^{M} A_{i,t}^{F}] = \rho$$

The explanatory variables have been defined earlier in equation (1).

In the pooled bivariate probit model it is assumed that the error terms of the male and female equations are not independent; the significance of the covariance (ρ) is a test of correlation of the partner obesity equations.

The Mundlak bivariate probit also includes the group means of the time varying explanatory variables as in equation (2), allowing the explanatory variables to be correlated to the error term. This method allows us to test how much of the correlation in spousal obesity are explained by hypotheses (2) and (3). If after controlling for individual effects, in the models which test hypotheses (2) and (3), ρ is still significant this would suggest that these two hypotheses explain some of the correlations in spousal obesity.

5.3 Endogeneity

It is likely that when modelling the likelihood of being obese in equations (1) and (3) some of the explanatory variables will be endogenously related to obesity due to simultaneous causation. This will lead to an upward bias in the estimated coefficients. For example, wages may influence BMI and BMI may influence wages. Low wages may lead to a higher BMI if individuals can not afford healthy food, gym membership, etc. which help to maintain a healthy weight. Obese individuals may also suffer from statistical discrimination in the job market[4]. The obesity and wages literature, for example, Cawley (2004), Baum and Ford (2004), and Morris (2006) tends to find that the effect of obesity on wages varies by gender and ethnicity. Findings from the medical literature (for example Must et al. 1999, Mokdad et al. 2003, and WHO 2006) show a clear link between obesity and health suggesting that health and BMI may be endogenously related. Individuals may also justify their high BMI by declaring poor health. In work not reported here we have attempted to evaluate the effect of this potential endogeneity of wages and health on our results by estimating equation (1) with lagged wages and health instead of current values to reduce simultaneous causation. Results show that the coefficients on the lagged variables are

[4] If employers assume stereotypes about obese people as a group such as being considered lazy, having poorer health, or being less productive (Rooth 2007) applies to all obese individuals this is a form of statistical discrimination.

not significantly different to those on the current period variables reported in this paper. This suggests hat endogeneity should not significantly bias the results.

6. Results and Discussion

Table 3 (at end)reports the regression results from estimating equation (1) via a random effects probit and a Mundlak probit (with the group means from equation (2)). Marginal effects are shown for the estimated coefficients.

Starting with the area variables, for both genders mean BMI of the local authority district has a positive and significant impact on the likelihood of being obese, with this effect being greater for men. For men in column I, living in the Northwest and Wales, and for women in column III, living in Scotland, has a positive and significant effect on the likelihood of being obese. These results could suggest a few things. It is possible that local norms influence the probability of becoming obese (Oswald and Powdthavee 2007), because this changes the perception of what is acceptable. It may also be that supply side factors specific to the Northwest, Wales, and Scotland such as the concentration of fast food outlets or dietary habits, increase the likelihood of becoming obese; of course these two effects can be mutually reinforcing. Grant et al. (2007) found higher concentrations of obesity among Scottish women compared with English women, whereas obesity prevalence among men in England and Scotland was found to be roughly similar. After controlling for individual effects with the Mundlak probit, mean BMI of the local authority district is still significant for both genders and living in Scotland still has a positive and significant effect on the likelihood of being obese for women, suggesting that the significance of these variables are independent of individual effects.

For women, having a partner with a degree or higher level of education has a negative and significant impact on the likelihood of being obese. Kan and Tsai (2004) found that knowledge about the health risks of obesity significantly impacted BMI, but in our results own education is not significant.

The significance of labour market variables varies by gender. The hourly wage has a positive and significant impact on the likelihood of being obese for men in column I. If there is no significant wage penalty for being obese as was shown in Morris (2006), then it is possible that men may substitute market work for leisure and other weight maintaining activities which could increase their likelihood of becoming obese, suggesting that obesity could be a normal good. For women in columns III and IV being employed full-time has a positive and significant effect on the likelihood of being obese. These results are consistent with Chou et al. (2004) where it was found that the demographic transition beginning in the 1970s which lead to an increase in female labour supply, can partially explain rising obesity rates. As women choose to allocate more time to market work they may not have time to cook time intensive nutritionally rich home cooked meals instead substituting towards calorie dense fast food or restaurant meals. This would also suggest that obesity is a normal good for women.

None of partner labour market variables are significant. If when estimating Model 2, ρ in the system of spousal equations is still significant this may suggest that hypothesis (1) or hypothesis (3), after controlling for individual effects, explain some of the spousal correlations in obesity.

Next, we look at health and health related variables. It is possible that if health or health related behaviour are endogenously related to obesity this may lead to an overestimation of the impact of these variables on the likelihood of being obese. The Mundlak probit, by removing individual effects from those explanatory variables where the group mean is taken, removes one form of endogeneity bias caused by a correlation between unobserved individual specific effects and the explanatory variables. Lagged variables which are not shown in this work also suggest no bias from simultaneity. However, these coefficients should still be interpreted with caution. Firstly, looking at the coefficients on the self-assessed health variable, for women in column III, declaring one's self in good and excellent health relative to poor health has a negative and significant impact on the likelihood of being obese. Healthier women may have more energy to devote to weight maintaining activities such as exercise, as could be evident by the negative and significant coefficient on participating in physical activity at least once a week in column III. For men in columns I and II having a partner who is a non-smoker has a negative and significant impact on the likelihood of being obese. As was suggested in Contoyannis and Jones (2004), if healthy lifestyle characteristics cluster in individuals, then men who prefer a healthy lifestyle, which is likely to reduce the likelihood of being obese, may choose a partner with similar preferences; this could also be inferred by the negative association between being obese and having a healthy weight spouse in Table 2. Overall, these results suggest that hypothesis (1) would explain some of the correlations in spousal obesity. For women in columns III having an obese partner has positive and significant impact on the likelihood of being obese. This may suggest social influence effects as the partner obesity variable is no longer significant in the Mundlak probit model which controls for individual effects.

The results reported so far suggest that variables relating to partner characteristics such as smoking status for men and education for women significantly impact the likelihood of being obese. This suggests that either hypothesis (1) or hypothesis (3) could explain some of the spousal correlations in obesity. Mean BMI of the local authority district and some of the regional categories significantly impacted the likelihood of being obese for both genders, suggesting that hypothesis (2) may also explain some of the correlations.

Table 4: Spousal correlations in the errors

	Pooled Bivariate Probit	Mundlak Approach Bivariate Probit
	ρ	ρ
	Column 1	Column 2
Model 1: $X_{i,t}$, $Z_{i,}$, $H_{i,t,}$ and $A_{i,t}$	0.14** (0.05)	0.15* (0.05)
Model 2: $X_{i,t}$, Z_i, $H_{i,t}$, $A_{i,t}$ and $L_{i,t}$,	0.15** (0.05)	0.10* (0.06)
Model 3: $X_{i,t}$, Z_i, $R_{i,t}$ $H_{i,t}$, and $A_{i,t}$	0.11* (0.07)	0.07 (0.05)
Model 4: $X_{i,t}$, Z_i, $L_{i,t}$ $R_{i,t}$ $H_{i,t}$, and $A_{i,t}$	0.07 (0.06)	0.01 (0.06)

*** Indicates significant at 1% level * *Indicates significance at 5% level. *Indicates significance at the 10% level. Standard Errors are in parenthesis.

Table 4 shows the correlation coefficients (ρ) from the system of partner equations via the pooled random effects bivariate probit and the Mundlak bivariate probit (equation (3) and equation (3) with the group means from equation (2)). The coefficients from the explanatory variables are not shown as they do not differ from those in equation (2). The focus of the system of partner equations is not on the individual determinants of the likelihood of being obese but the correlation in the error terms of couples' likelihood of being obese.

The correlation in the error terms from the partner pooled bivariate probits are significant in Models 1, 2, 3, and for the Mundlak probits in Models 1 and 2, suggesting that individual characteristics and social influences may explain some of the observed raw correlations in spousal obesity. After controlling for individual effects, in Model 3, ρ is not significant, suggesting that contextual factors do not explain correlations in spousal obesity. In columns 1 and 2 in Model 4, ρ is also not significant, implying that after controlling for all the covariates, the correlation in spousal obesity is no longer significant. This may be driven by the inclusion of contextual factors in the full model suggesting that shared individual characteristics and social influences may still explain some of the observed spousal correlations in obesity in Table 2.

Our results suggest that shared individual characteristics and social influences explain the majority of the observed raw correlations in spousal obesity. These results are similar to those of Christakis and Fowler (2007) who found using data from an adult population, that social networks were important in explaining the likelihood of becoming obese. Thus, we also refute the claims made by Cohen-Cole and Fletcher (2008), using data from an adolescent population, that shared environmental factors explain the majority of the correlations in obesity in social networks. If social networks influence the likelihood of becoming obese it is likely that they operate at the: 1) household level; 2) community level, and 3) wider society. It is possible that at different life stages different factors will dominate. For example, adolescents because of budget and mobility constraints and a greater desire to be socially accepted may be more influence by the latter two factors as is shown with the results of Cohen-Cole and Fletcher (2008). Whereas for an adult population factors at the household level may play a greater role in influencing the likelihood of being obese which is consistent with results in this paper and Christakis and Fowler (2007).

7. Conclusion

In this paper we test the three hypotheses of shared individual characteristics, contextual factors, and social influence to explain spousal correlations in obesity using BHPS data for 2004 and 2006. Firstly, we test the significance of partner characteristics and contextual variables (regional variables) on the likelihood of being obese. We find that the mean BMI of the local authority is significant for both genders, whereas partner smoking status is significant for males only, and having a spouse with a higher or first degree is significant for women only. These results hold after controlling for individual effects.

Next, we estimate four different model specifications using a pooled bivariate probit and Mundlak approach bivariate probit to try and distinguish how much of the correlations in spousal obesity are explained by the three hypotheses. Results indicate that shared individual characteristics and social influences explain the majority of spousal correlations in obesity. These results refute the claims made by Cohen-Cole and Fletcher (2008) who found that a shared environment explained the majority of correlations in obesity in a social network. However, they are consistent with Christakis and Fowler (2007) who found that social networks significantly impact the likelihood of being obese. It is possible that the impact of social networks on the likelihood of being obese may vary by age and circumstances. Future work should compare demographic group and marital status to test this hypothesis. Effective policy to reduce obesity rates may need to be designed to address specific demographic groups rather than society as a whole.

This paper represents work in progress and in further work we will: (i) allow for correlated individual effects in the male and female obesity equations shown in model (3) to further distinguish between the hypotheses stated in this paper; (ii) model spousal correlations in the continuous measure of BMI; improve our analysis of contextual effects by included indicators of rural/urban areas.

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Table 1: *Descriptive Statistics*

VARIABLES		MALES: BMI CATEGORIES			FEMALES: BMI CATEGORIES		
		18-25	**25-30**	**30**	**18-25**	**25-30**	**30**
Age		52.21 [8.02] (656)	52.13 [7.86] (821)	51.76 [7.74] (547)	50.67 [8.31] (840)	51.26 [8.49] (830)	51.08 [8.49] (679)
Region	Greater London	40.78 (73)	34.08 (61)	25.14 (45)	36.55 (72)	35.53 (70)	27.92 (55)
	Southeast	35.76 (265)	39.41 (292)	24.83 (184)	36.36 (284)	34.06 (266)	29.58 (231)
	Southwest	32.21 (106)	43.77 (144)	24.01 (79)	33.91 (117)	36.23 (125)	29.86 (103)
	Midlands	29.72 (159)	43.55 (233)	26.72 (143)	33.21 (181)	35.78 (195)	31.01 (169)
	Northwest	34.31 (93)	38.01 (103)	27.67 (75)	36.68 (106)	35.29 (102)	38.76 (81)
	Yorks, Humb, rest of North	34.74 (156)	39.64 (178)	25.61 (115)	35.24 (160)	35.68 (162)	29.07 (132)
	Wales	31.84 (50)	42.67 (67)	25.48 (40)	36.26 (66)	35.16 (64)	28.57 (52)
	Scotland	31.75 (60)	43.92 (83)	24.34 (46)	33.79 (74)	32.42 (71)	33.79 (74)
Education:							
No qualifications		35.20 (296)	40.55 (341)	24.26 (204)	31.49 (325)	37.31 (385)	31.20 (322)
CSE or O-Level		31.59 (230)	41.76 (304)	26.65 (194)	36.90 (376)	34.35 (350)	28.75 (293)
HND, HNC, teaching or A-level		34.00 (290)	39.31 (351)	24.85 (212)	36.42 (216)	33.73 (200)	29.85 (177)
First or Higher degree		34.33 (149)	38.48 (167)	27.19 (118)	38.42 (146)	33.16 (126)	28.42 (108)
Kids		24.51 (967)	24.98 (1165)	27.26 (730)	27.35 (1064)	25.80 (1062)	25.44 (900)
Non-labour income		5146.13 [6544.88] (966)	4598.96 [6148.11] (1163)	4613.58 [6450.83] (730)	6825.66 [6298.44] (1059)	6904.05 [6473.19] (1058)	6990.73 [6330.99] (894)
Employment	Employed	31.94 (549)	41.54 (714)	26.53 (456)	36.30 (580)	35.48 (567)	28.22 (451)
	Full time	31.51 (477)	41.74 (632)	26.75 (405)	32.71 (278)	37.62 (316)	30.12 (256)
Hourly wage		13.95 [8.79] (433)	14.02 [8.29] (562)	14.82 [9.04] (354)	10.75 [6.31] (520)	10.10 [5.95] (516)	11.49 [21.09] (408)
Health:	Very Poor	31.58 (12)	42.11 (16)	26.32 (10)	25.00 (14)	39.29 (22)	35.71 (20)
	Poor	32.56 (56)	37.21 (64)	30.23 (52)	28.88 (54)	29.41 (55)	41.71 (78)
	Fair	34.17 (218)	37.77 (241)	28.06 (179)	31.60 (206)	34.66 (226)	33.74 (220)
	Good	33.38 (473)	41.35 (586)	25.26 (358)	35.80 (537)	35.93 (539)	28.27 (424)
	Excellent	34.84 (208)	43.22 (258)	21.94 (131)	40.10 (253)	34.87 (220)	25.04 (158)
Non-Smoker		14.79 (967)	9.44 (1165)	10.96 (730)	10.71 (1064)	11.21 (1062)	10.89 (900)
Physical Activity:	Never, almost never	32.94 (167)	39.45 (200)	27.61 (140)	28.88 (173)	34.72 (208)	46.91 (218)
	Once a year	32.43 (36)	42.34 (47)	25.23 (28)	35.77 (44)	39.02 (48)	25.20 (31)
	Several times a year	32.27 (81)	41.83 (105)	25.90 (65)	34.34 (91)	34.34 (91)	31.32 (83)
	At least once a month	33.96 (109)	39.56 (127)	26.48 (85)	40.00 (130)	32.31 (105)	27.69 (90)
	At least once a week	34.35 (574)	41.05 (686)	24.60 (411)	36.52 (626)	35.59 (610)	27.89 (478)

Standard deviations are given in square brackets and parentheses denote the number of individuals in each category. With the exception of hourly wage, age, and non-labour income all values given are percentages. The percentages denote the number of individuals in each BMI category out of the total number of individuals for each question category. For example, for participating in physical activity once a week, the percentage derived for the males in the healthy BMI category is 574/1671, where 1671 is the total number of male responses in all 3 BMI categories who stated that they exercised at least once a week.

Table 3: Individual and Partner Characteristics and Contextual Factors

Dependent Variable: Obese	**MEN**		**WOMEN**	
Explanatory	**RE Probit**	**Mundlak Probit**	**RE Probit**	**Mundlak Probit**
	Column I	**Column II**	**Column III**	**Column IV**
Age	0.29 (0.26)	0.46 (0.98)	-0.31 (0.23)	-0.59 (0.82)
Age squared	0.002 (0.002)	0.004 (0.008)	0.003 (0.002)	0.008 (0.007)
Partner age	-0.05 (0.04)	-0.84 (0.41)	-0.002 (0.003)	0.004 (0.40)
Kids	-0.45* (0.22)	-0.32 (0.49)	0.27 (0.17)	0.09 (0.47)
Education				
CSE or O-Level	-0.45 (0.46)	-0.42 (0.46)	0.31 (0.38)	0.10 (0.41)
A-level	-0.36 (0.44)	-0.62 (0.45)	0.37 (0.43)	0.39 (0.48)
Higher or First Degree	0.24 (0.55)	0.14 (0.56)	0.44 (0.51)	-0.07 (0.58)
Partner Education				
CSE or O-Level	0.50 (0.46)	0.63 (0.48)	-0.37 (0.37)	-0.59 (0.42)
HND. HNC. Teaching or A-level	0.53 (0.51)	0.32 (0.54)	-0.11 (0.36)	-0.21 (0.42)
Higher or First Degree	0.44 (0.62)	-0.18 (0.64)	-0.97* (0.51)	-1.08* (0.58)
Region				
Southeast	0.56 (0.83)	0.22 (0.83)	-0.44 (0.55)	-0.17 (0.61)
Southwest	1.33 (0.87)	1.19 (0.86)	-0.03 (0.06)	0.13 (0.67)
Midlands	1.25 (0.83)	0.69 (0.82)	-0.08 (0.05)	0.01 (0.62)
Northwest	1.70* (0.87)	1.36 (0.87)	-0.68 (0.65)	-0.54 (0.72)
Yorks, Humberside, and rest of North	0.85 (0.85)	0.76 (0.84)	-0.78 (0.60)	-0.76 (0.68)
Wales	1.61* (0.97)	1.43 (0.96)	-0.86 (0.72)	-0.74 (0.83)
Scotland	1.10 (0.93)	0.83 (0.95)	1.29* (0.66)	0.96* (0.74)
Mean BMI of LAD	0.55** (0.09)	0.50** (0.09)	0.31** (0.07)	0.30** (0.09)
Nonlabourincome	0.0001 (0.0001)	0.0001 (0.0001)	0.00001 (0.00003)	0.00001 (0.00003)
Hourly wage	0.05* (0.02)	0.05 (0.06)	0.01 (0.01)	0.001 (0.01)
Partner hourly wage	0.01 (0.01)	0.004 (0.02)	0.04* (0.02)	0.02 (0.06)
Job Status				
Employed	0.03 (0.09)	0.06 (0.16)	-0.16 (0.14)	-0.35 (0.17)
Employed full-time	-0.82 (0.19)	-0.75 (0.19)	0.48* (0.28)	0.52* (0.31)
Partner Job Status				
Employed	0.06 (0.04)	0.13 (0.06)	0.07 (0.06)	0.35 (0.16)
Partner Employed full -time	0.58 (0.34)	0.44 (0.31)	0.54 (0.61)	0.37 (0.10)
Health States				
Poor	2.67 (1.66)	1.37 (1.71)	-1.43 (1.44)	-1.15 (1.43)
Fair	0.83 (0.53)	0.08 (0.59)	-2.10 (1.33)	-0.86 (1.35)
Good	0.61 (0.53)	-0.09 (0.62)	-2.55* (1.34)	-1.00 (1.43)
Excellent	0.42 (0.55)	-0.37 (0.71)	-3.03* (1.35)	-1.34 (1.56)
Partner Health States				
Poor	0.62 (0.48)	0.27 (0.51)	-0.74 (0.50)	-0.62 (0.55)
Fair	0.67 (0.38)	0.19 (0.45)	-0.64 (0.37)	-0.003 (0.38)
Good	-0.33 (0.39)	-0.92 (0.53)	-0.76 (0.36)	-0.01 (0.43)
Excellent	0.26 (0.41)	-0.60 (0.67)	-0.47 (0.38)	0.39 (0.52)
Non-Smoker	0.25 (0.43)	0.39 (0.44)	0.18 (0.38)	0.14 (0.43)
Partner Non- Smoker	-0.72* (0.41)	-0.82* (0.41)	0.32 (0.39)	0.51 (0.43)
Physical Activity				
At least once a week	-0.55 (0.44)	-0.51 (0.44)	-0.59* (0.35)	-0.55 (0.43)
Partner Physical Activity				
At least once a week	0.03 (0.44)	0.13 (0.44)	-0.56 (0.37)	-0.56 (0.43)
Partner Obese	0.001 (0.06)	0.05 (0.09)	0.38* (0.25)	0.22 (0.29)
	n=592	n=592	n=592	n=592

*** Indicates significant at 1% level * *Indicates significance at 5% level. *Indicates significance at the 10% level. Standard Errors are in parenthesis. Marginal effects are shown.

Appendix A: Variable Labels and Definitions

VARIABLE	DESCRIPTION
Dependent Variable	
OBESE	1=30+ kg/m^2, 0=otherwise
Explanatory Variables	
Age	Age in years
Age Squared	Age squared
	Region
Region	1=Greater London
	2=Southeast
	3=Southwest
	4=Midlands
	5=Northwest
	6=Yorks, Humberside, & Rest of North
	7=Wales
	8=Scotland
	9=Northern Ireland
Mean BMI of LAD	Mean BMI of Local Authority District
	Education
Education	0=No qualifications
	1=CSE or O level
	2=HND, HNC, teaching, or A-level
	3=Higher or First Degree
	Children
Kids	Number of dependent children (ranges from 1-7)
Nonlabourincome	Non-labour income: household income minus annual labour income
	Self-Assessed Health
Health States	1=Very Poor
	2=Poor
	3=Fair
	4=Good
	5=Excellent
	Smoking Status
Non-Smoker	0=Yes
	1=No
	Labour Variables
Employed	0=Unemployed, maternity leave, ft student, government training
	1=Employed or self-employed
Employed full-time	0=Part time
	1=Full time
Hourly wage	Hourly wage=Montlypay/Hours x (12/52)
	Physical Activity
Physical Activity	0=Never or almost never
	1=Once a year or less, 2=Several times a year
	3=At least once a month, 4=At least once a week

See www.iser.essex.ac.uk/survey/bhps for more information on BHPS variables.