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Obesity and the Incidence of Chronic Diseases: a Seemingly Unrelated Probit Approach*

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Abstract

Western societies can reduce avoidable mortality and morbidity by better understanding the relationship between obesity and chronic disease. This paper examines the joint determinants of obesity and of heart disease, diabetes, hypertension, and elevated cholesterol. It analyzes a broadly representative Spanish dataset, the 1999 *Survey on Disabilities, Impairments and Health Status*, using a health production theoretical framework together with a seemingly unrelated probit model approach that controls for unobserved heterogeneity and endogeneity. Its findings provide suggestive evidence of a positive and significant, although specification-dependent, association between obesity and the prevalence of chronic illness.

J.E.L. Classification: I12, I18, I19.

Keywords: Obesity; Health production; Body Mass Index; Chronic illness; Diabetes; Hypertension; High cholesterol; Cardiovascular disease

Resum

Per la via d'una millor comprensió de la relació que s'estableix entre obesitat i malalties cròniques les societats desenvolupades poden reduir, de manera significativa, les taxes mortalitat i morbilitat. Aquest article examina els determinants conjunts de l'obesitat i les malalties del cor, diabetis, hipertensió arterial i colesterol elevat. S'analitzen aquests fenòmens mitjançant l'ús de l'*Enquesta de Deficiències, Discapacitats i Estats de Salut 1999* (INE) i per mitjà de la teoria de la producció de salut conjuntament amb un "sure probit model" controlant per heterogeneïtat no observable i endogeneïtat. Els nostres resultats ofereixen evidència d'una associació positiva i significativa, depenent de l'especificació economètrica, entre obesitat i la prevalència de malalties cròniques.

1. Introduction

The 'obesity epidemic' is worldwide. It has been explained as an alteration of mean human body mass due to the economic, social, and biological transformations that follow from urbanization and globalization processes, primarily in western countries (Wang et al., 2002). The burden of obesity has well-known direct effects on individual mobility and disability and impinges indirectly on morbidity rates. Some studies find robust evidence linking obesity to the increasing prevalence of chronic illnesses (Must et al., 1999). The World Health Organization (WHO) Global Strategy on Diet, Physical Activity and Health states that "obesity and overweight pose a major risk for chronic diseases, including type 2 diabetes, cardiovascular disease, hypertension and stroke, and certain forms of cancer," that reduce quality of life and cause sizeable health-care costs (WHO, 2004). That is to say, obesity, itself an avoidable chronic disease, is a substantial risk factor for other chronic diseases (EOTF, EASOTF, 2002).

In some western countries, the prevalence of obesity has risen dramatically. In the United States, 19.8% of adults were obese in the late 1990's, up from only 12% in 1991 (Nestle and Jacobson, 2000). The disease is spreading through Europe at alarming speed (EOTF, EASOTF, 2002; Rigby and James, 2003) and is progressively becoming a primary health problem in southern European countries. In Spain, for example, one out of every two individuals is overweight and 14.5% of the adult population is obese, according to the Ministry of Health[1]. These data are even more worrying if we bear in mind that Spain ranks second only to the United Kingdom among the European Union countries exhibiting the highest increases in obesity rates over the last

[1] Furthermore, recent estimates by the WHO Monica Project find that, in Catalonia, 16% of men and 25% of women suffer from obesity (Evan et al., 2001).

decade (WHO, 2002).[2] The epidemic may lead to a notable increase in mortality rates for the chronic diseases associated with obesity. In Spain, cardiovascular disease is the leading cause of death (killing 31% of men and 41% of women); digestive system conditions account for 10% of total mortality in men and 5% in women. (Spanish National Institute of Statistics, 2002). Rough estimates by the Spanish Society for the Study of Obesity (SEEDO) point out that as much as 7% of the nation's total health expenditure could be due to obesity (Aranceta, et al., 2000).

Despite overwhelming evidence of a clear-cut association between obesity and of chronic illnesses, the joint effects of obesity together with other explanatory factors on the incidence of chronic diseases are poorly understood[3]. In economic terms, obesity can be thought of as a compound variable carrying the effects of other health care inputs, such as lifestyle, rather than as a single negative input into the health production function[4]. Furthermore, it seems plausible to hypothesize that common unobserved factors such as psychological stress and anxiety simultaneously influence the propensity for obesity and the prevalence of chronic diseases. Thus, any obesity-specific effect on a chronic disease may be specification-dependant, especially when cross-sectional survey data are employed.

The empirical strategies used to estimate such effects should control for some unobserved heterogeneity of health inputs. Given that obesity may correlate with unobservable variables relegated to the error term[5], treatment should be provided to account for potential sources of endogeneity. The joint

[2] And only 34% of the obese pursued a specific treatment to prevent such consequences of obesity as the emergence of chronic illnesses (Martínez *et al.*, 2004).
[3] It is important to model such joint effects, since controlling for them may yield more precise estimates.
[4] On purely empirical grounds it is common to find significant correlation between health inputs.
[5] Unobservable variables may be relegated to the error term when survey data omit relevant factors or when those factors cannot be estimated with 'acceptable precision' based on conventional questionnaires.

determinants of obesity and chronic diseases should be scrutinized for relevant evidence to assist the design of health promotion policies in an 'obesogenic environment'.

The purpose of this paper is to investigate the effect of obesity on those chronic diseases with which it is strongly associated in the medical literature and for which Spanish data are available: type 2 diabetes, cardiovascular disease, hypertension, and high cholesterol. The database employed, Spain's 1999 national *Survey on Disabilities, Impairments and Health Status* (INE, 2001-2004), is representative across regions and age cohorts and includes in its health module information on nearly 70 thousand individuals. In the light of previous studies, we use seemingly unrelated probit models with and without controls for endogeneity to examine the joint influence of an individual's lifestyle and socio-economic characteristics in determining obesity and chronic illnesses. Our findings indicate that the impact of obesity on the incidence of and burden of chronic diseases is significantly determined by the empirical specification. Finally, we test for the effects of several interactions between lifestyles and income and suggest that some 'unhealthy lifestyles' may be economically driven.

The paper is structured as follows. Section 2 examines the association between obesity and chronic diseases and provides a theoretical explanation for this relationship. Section 3 deals with methods and the empirical model. Section 4 reports the results and Section 5 evaluates them.

2. Obesity and the prevalence of chronic illnesses

2.1. The socio-economic determinants of obesity

The socio-economic determinants of obesity are multiple. Empirical evidence of causality is still relatively scarce. Most research to date attempts to explain the

emergence of the so-called 'obesogenic environment' (French et al., 2001). Briefly, the economic effects of industrialization and urbanization and concomitant economic growth have resulted in increasingly sedentary work and leisure activities. This reduction in energy expenditure has been accompanied by a dietary shift to the consumption of increasingly caloric foods containing a higher proportion of fats, saturated fats, and sugars. From an evolutionary perspective, natural selection under conditions of food scarcity leads to the reproduction of the fittest individuals. Given that individual preferences are based on food scarcity, when food is abundant it is to be expected that individuals will gain weight unless they undertake additional activity (Logue, 1998). This imbalance may become structural when excess caloric intake is reinforced by unhealthy menus and sedentary lifestyles.

Some studies use behavioral models of obesity to explain the determinants of the number of calories consumed, suggesting such culprits as changes in relative prices and in the density of fast food restaurants (Chou et al., 2002), reductions in the time cost of preparing meals (Culter et al., 2003), and unemployment and job strenuousness (Ruhm, 2000). Ruhm used time series analyses of US states for 1972 to 1991 to show that obesity increases and physical activity declines during business cycle expansions. Lakadawalla and Philipson (2002) found evidence of a robust negative association between physical activity and obesity.

In addition to the economic determinants, the socio-cultural context is recognized as a key factor in explaining the development of obesity in an individual. Obesity is a household-produced good, so an individual's self-image and social interactions are likely to play a role in determining his or her weight. Indeed, some evidence indicates that an individual's social interactions and body mass production are significantly interdependent (Costa-Font and Gil, 2004). At the same time, eating and physical activity patterns are, to some extent, culturally driven behavior in industrialized nations. Wansink (2004) finds that

the eating environment— environmental factors associated with food intake—is associated with the amount of food consumed. Kan and Tsai (2004) used quantile regression to show that that familiarity with obesity risk factors affects an individual's obesity and that this effect is different for males and females. Another variable connected with health knowledge, schooling, may affect obesity in three ways. It potentially increases the efficiency of health production (Kenkel, 2000; Grossman, 2004). Following health capital theory, education is also likely to influence obesity by contributing to an individual's income. Schooling may also affect obesity by altering time preference (Fuchs, 1982). Indeed, an individual's consumption level has been shown to depend on the rate at which future health benefits are discounted in the individual's consumption decisions; individual fitness is negatively associated with a high rate of time preference (or impatience) as measured using country-based aggregate data (Komlos et al., 2004).

2.2. Obesity and chronic diseases

The medical and epidemiological literature reveals a clear-cut connection between obesity and chronic conditions. Evidence from the US (Sturm and Wells, 2001; Sturm, 2002) suggests that obese individuals self-report chronic conditions such as diabetes, hypertension, asthma, heart disease, and certain forms of cancer 67% more often than normal-weight individuals with no history of smoking or heavy drinking.[6] Using self-report data from individuals and

[6] Other well-known consequences of obesity include, among other conditions: gall-bladder disease, arteriosclerosis, gout, sleep apnea, venous insufficiency, stroke, gastrointestinal effects (e.g., esophagus reflux, gastritis, vesicular lithiasis, fat liver, colitis, cancer of the colon, hemorrhoids), genital urinary sequels (urinary incontinence, kidney stones, menstrual alterations, prostate cancer, benign and malignant lesions in the uterus and in the breast), osteoarticular effects (muscular hypotrophy, arthritis of lower members and vertebral column, hernia), psychological alterations (loss of self-esteem, depression), pulmonary sequels (fatigue, lack of air, pulmonary thrombosis), reproductive problems (disturbed ovulation,

physicians, Paeratakul et al. (2002) determined that the prevalence of diabetes, hypertension, and high-serum cholesterol increases with increasing body weight. WHO's World Health Report 2002 estimates that more than 7% of the disease burden in developed countries is caused by increases in the average body mass index (BMI). Jung (1997) and Sowers (2003) associate a high BMI with cardiovascular disease risk factors, including hypertension, high total and LDL cholesterol, high triglyceride levels, and low HDL cholesterol. Fat, in essence, is a biologically active tissue that produces chemical messengers (e.g., adiponectin, resistin, angiotensin-2, tumor necrosis factor-α, and interleukins) that can affect cardiovascular risk factors (Sowers, 2003).

Wolf and Coldits (1998) find that obesity is responsible for 61% of the costs of type 2 diabetes, 17% of the costs of hypertension, 34% of the costs of endometrial cancer, and 30% of the costs of gall-bladder disease. Obesity, particularly when centrally distributed—often referred to as abdominal obesity—predisposes an individual to diabetes via increased portal delivery of fatty acids to the liver from lipolytically active visceral adipose tissue. This process induces both hepatic insulin resistance and reduced hepatic insulin clearance (Lewis et al., 2002). Interestingly, recent research (Knowler et al., 2002) suggests that lifestyle interventions which caused an average weight loss of less than 7 kg in a 6 month period with some later weight gain led to a 58% reduction of cumulative diabetes incidence in the intervention groups.

Although the precise mechanisms involved in the etiology of obesity-related hypertension are not fully understood, they are thought to involve activation of the sympathetic nervous system, physical pressure on the kidney, cytokine effects, and excessive sodium re-absorption. A systematic review by the Cochrane Collaboration (Mulrow et al., 2000) suggests that a weight loss of 4–8% can be expected to reduce both systolic and diastolic blood pressures by

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infertility, difficulty in sexual relations) and social consequences (isolation, discrimination in social settings and at work and at school).

approximately 3 mm Hg. Although this is clearly a useful reduction at the population level, it may not be sufficient for the individual hypertensive patient even if achieved and maintained. The Framingham Heart Study (Hurbert et al., 1983) reported that in obesity the relative risk of coronary heart disease increased more than twofold after correction for other known risk factors.

Numerous illnesses and chronic conditions are potentially associated with obesity. This paper examines only four: diabetes, high cholesterol, arterial hypertension, and heart disease. We focus on these diseases because each has a significant impact on premature mortality in Spain, each has been the subject of intensive medical research in the past, and because data is available.

2.3. Health and body mass production: a theoretical connection

Understanding the connection between body mass and the prevalence of chronic diseases requires careful attention. The theory of health production offers some theoretical underpinnings to ground our analysis. As usual, let us assume that an individual's behavior results from the maximization of the individual's utility function, $U_i = U(H_i, X_i, B_i)$, which contains as its arguments health, bodily fitness, and a composite variable referring to other goods, subject to both time and budgetary restrictions, following the tradition of Grossman's (1972, 2000) model. That is, individuals allocate time and resources to a variety of goods and activities including food intake and other types of household-produced goods, such as the acquisition of knowledge, to produce health (H_i). If an individual practices sports, eats healthy food, and so forth, he or she may also produce bodily fitness (B_i). The individual also produces other goods which are independent of both health and fitness (X_i). Individual fitness is valued both for its effects on the individual's self-image and for its indirect effect on the production of health. Accordingly, we could express the individual's production of health —and, consequently, the absence of ill health—as follows:

$$H_i = H(B_i, Y_i, Z_i) \quad (1)$$

where health is determined by the individual's fitness, income constraints (Y_i), and other health production determinants (Z_i). Improvements in an individual's fitness are assumed to improve health care production for obvious reasons, subject to the effects of other health production determinants, while the effect of income determines the capacity of individuals to invest in health. One could argue that the model should include time constraints, but this variable was unobserved, so we did not include it in the model explicitly. It could also be argued that time available to produce health depends on individual income, an assumption which might be questioned on the grounds that higher income implies greater opportunity for leisure spending. However, the degree of substitution between time and money is far from perfect, and the 'income effect' resulting from loosened financial restrictions could potentially overcome the substitution effect (Killingsworth, 1983).

From the previous utility maximization, individuals will determine their fitness level, which is likely to depend on the consumption of certain goods including household-produced goods that lead to their desired fitness level (Q_i) and on the individual's income or budgetary restriction (Y_i), as follows:

$$B_i = B(Q_i, Y_i) \quad (2)$$

On the basis of (1) and (2) the empirical analysis of both health production and fitness production depends on identification of the specific effects of each variable. However, as some variables are not known, we have proxied them. We use the incidence of four chronic illnesses which are the main causes of 'avoidable mortality' in Spain and the presence or absence of obesity (or, alternatively, the individual's body mass index) to measure individual fitness[7]. Other variables, such as time preference, enter the model implicitly (Komlos et al., 2004). The value of time is a key determinant of an individual's production

of adult health and avoidance of ill-health for more than one reason. Time is needed to produce health. Also, health care is produced in interaction with education and health knowledge, each of which normally rely heavily on the individual's time preference. Proceeding from this simple theoretical framework, we expect to identify a negative (positive) association between an individual's fitness (obesity) and the prevalence of certain chronic illnesses.

2.4. The empirical model

Obesity and the propensity to chronic illness, the key outcomes under analysis, are defined as dichotomous variables. A latent variable model is employed to examine their relationship. Because decision variables are likely to be connected over time, unobservable variables may affect both the propensity to obesity and the prevalence of a chronic disease. Therefore, our empirical strategy relies on first estimating a seemingly unrelated probit model which does not include obesity as an endogenous dummy[8]. This allows us to determine whether a joint estimation is appropriate but does not assess the incidence of obesity on chronic illness. Then, we test for exogeneity using maximum-likelihood simultaneous estimation of the two probit equations, a method also known as recursive bivariate probit (Maddala, 1983) or as seemingly unrelated probit model with endogenous dummy (Fabbri et al., 2004)[9].

The latent class model assumes normality of responses within latent classes but the mixed distribution can accommodate non-normal marginal and joint distributions of response probabilities. The model allows us to correct for some unobserved heterogeneity that might otherwise give rise to the 'omitted

[7] Cross-correlation analysis indicates a very robust and significant correlation above 0.9 between measures of health and the prevalence of all the chronic illnesses considered.
[8] Similar models have been employed before in health-related applications (Atella et al., 2004).

variable bias'. Finally, to provide additional insight into the nature of the joint choices made by individuals, we calculate the marginal effects of covariates on the probabilities of choosing each type of outcome and on the joint probabilities of each combination of alternatives. The marginal effect allows us to simulate changes in socio-economic characteristics which may link obesity with the propensity to suffer specific chronic diseases.

2.4.1. The seemingly unrelated probit

Let us assume that obesity is identified by the latent variable q_{1i}^{*} and that q_{2i}^{*} is the latent variable measuring the presence of a specific chronic disease. (To condense this presentation, a single variable for a single disease is described. Multiple diseases were actually modeled.) Because these variables are not directly observable, we specify the model as follows:

$$q^{*}{}_{1i} = \beta_1 X_{1i} + \mu_{1i} \tag{3}$$

$$q_{1i} = \begin{cases} 1 \text{ if } q_{1i}^{*} > 0 \\ 0 \text{ otherwise} \end{cases}$$

where X_{1i} refers to the observed determinants of obesity, β_{1i} to the associated parameters, and μ_{1i} is a random error term. Analogously, the propensity to suffer a chronic illness is measured by:

$$q_{2i}^{*} = \beta_2 X_{2i} + \mu_{2i} \tag{4}$$

$$q_{2i} = \begin{cases} 1 \text{ if } q_{2i}^{*} > 0 \\ 0 \text{ otherwise} \end{cases}$$

where X_{2i} refers to the observed determinants of chronic diseases, β_{2i} to the associated parameters, and μ_{2i} is a random error term. Yet, because the two variables are potentially explained by common determinants, the error terms of

[9] A Likelihood ratio and Wald test to examine endogeneity is implemented because a two-stage approach using probit models would not be appropriate (Wooldridge, 2002).

the two models are dependent and distributed as a bivariate normal, so that $E(\mu_{1i}) = E(\mu_{2i}) = 0$, $\text{var}(\mu_{1i}) = \text{var}(\mu_{2i}) = 1$, and $\rho = \text{cov}(\mu_{1i}, \mu_{2i})$. If the error terms of both equations are affected by similar components, $\mu_{ji} = \eta_i + \pi_{ji}$, then, although they are likely to be normally distributed, they will not be independent, but will depend on the value of η_i. A Wald test for $\rho = 0$ indicates whether the models should be jointly estimated.

2.4.2. Seemingly unrelated probit with endogeneity

This model follows the tradition of the simultaneous equation models defined in Maddala (1983) as "model 5". It draws upon a reduced form equation for the potentially endogenous dichotomous variable (obesity) and a structural form equation for the prevalence of chronic diseases as follows:

$$q^{*}_{1i} = \beta_1 X_{1i} + u_{1i} \qquad (5)$$

$$q^{*}_{2i} = \delta_1 q_{1i} + \delta_2 Z_{2i} + u_{2i}$$

As before, q^{*}_{1i}, q^{*}_{2i} are latent variables observed as dummy variables as in (3) and (4), X_{1i}, Z_{2i} are exogenous variables, and $\beta_1, \delta_1, \delta_2$ are parameters of the behavioral function. The error terms of the two models are dependent and distributed as a bivariate normal so that $E(u_{1i}) = E(u_{2i}) = 0$, $\text{var}(u_{1i}) = \text{var}(u_{2i}) = 1$, and $\rho = \text{cov}(u_{1i}, u_{2i})$. The Wald test, and/or a Lagrange multiplier test, provides evidence on the correlation between the unobserved explanatory variables of both equations so that if $\rho = 0$ then q_{1i} is exogenous for the second equation (cf. Fabbri et al., 2004).

2.4.3. A two-stage probit least squares model

Dichotomizing the obesity variable, as in the two previous models, potentially discards information. As an alternative, we estimated the effect of body mass on the incidence of chronic diseases, treating the latter as a potentially endogenous variable by instrumenting the effect of obesity using the two-stage probit least squares estimation method described in Maddala (1983) for simultaneous equations models in which one of the endogenous variables is continuous and the other endogenous variable is dichotomous (Maddala, 1983; Keshk, 2003). Let us present a generic two–equation model where q_{3i}^{*} is a continuous and observed variable measuring the individual's body mass which is simultaneously determined with the incidence of a chronic illness as defined in (3). X_{3i} refers to the instruments of an individual's body mass, γ_i, β_i are the estimated coefficients, and $\varepsilon_{2i}, \varepsilon_{1i}$ are the error terms of each equation as follows:

$$q_{3i}^{*} = \beta_3 X_{3i} + \varepsilon_{2i} \qquad (6)$$

$$q^{*}{}_{2i} = \gamma_i q_{3i}^{*} + \beta_2 X_{2i} + \varepsilon_{1i}$$

Accordingly, both equations are treated as endogenous, corresponding to Maddala's (1983, 244-245) "model 3."

3. Data and variables

3.1. Data and variables

Our empirical work was based on micro-level data retrieved from the health questionnaire of Spain's *Survey on Disabilities, Impairments and State of Health* of 1999 (SDISH-1999) (INE, 2001-2004).[10] This survey is a large, nationally representative survey designed for the purpose of gathering data on the characteristics and situations of persons with disabilities and on general health

[10] SDISH-1999 is organized into four questionnaires, on the household, on disabilities and impairments, on limitations and impairments, and the on health status of the population. (INE, 2001-2004)

conditions. The survey followed a stratified two-phase sampling procedure, in which first-stage units are the census sections and second-stage units are the main family dwellings. Within each sampled household every resident was investigated with the objective of identifying those individuals with disabilities. A random procedure was used to select respondents within each household. The stratification criterion used was based on municipality size. Reliability at the regional and national level was ensured using a sample of approximately 80,000 dwellings distributed across 3,000 census sections.

Once the interviewee is selected, the health questionnaire asks for self-perceived state of health, anthropometric characteristics, consumption of medicines, chronic diseases, history of accidents, criminal offences, consumption of health care and social services, problems of accessibility, eating habits, and life style choices (such as tobacco use, alcohol consumption, or physical exercise).[11] The initial SDISH-1999 health module sample included 69,555 individuals. We excluded 8,049 observations for individuals younger than 16 years old. Of the remaining 61,506, we dropped 500 people whose weight was not reported, 2 whose height was missing, and 6,618 whose household income was missing. We also deleted from the sample 227 observations with missing values for any of the following: eating habits, physical exercise at work and in spare time, tobacco and alcohol consumption, marital status, and level of education. After list-wise deletion, the sample contains 54,159 individuals aged 16 to 99 years old, of whom 24,871 (45.92%) are men and 29,288 (54.08%) are women,[12] and represents close to 90% of the entire adult Spanish population. Definitions of all variables employed in the regressions are detailed in Table 1. Data on the four chronic illnesses examined

[11] For the technical details of the SDISH-1999 methodology, see the survey reports themselves (INE, 2001-2004) or the website of the Spanish National Statistical Institute, http://www.ine.es.

[12] One potential concern is how to account for weight gain in pregnancy. In this sample, pregnant women were asked to report their weight before pregnancy.

was collected exclusively on the basis of *diagnosed illness*, not on the basis of a subject's beliefs.

[Insert Table 1 about here]

To measure obesity, self-reported data on height and weight were employed. This allowed us to define the widely accepted "body mass index" (BMI) indicator for each respondent. This index, defined as weight in kilograms divided by the square of height in meters (kg/m^2), enables us to obtain an estimate of the prevalence of obesity[13]. The World Health Organization (1997) defines a BMI of 25 to 29.9 kg/m^2 as overweight and a BMI of $\geq$30 kg/m^2 as obese.

It has been suggested that self-reported anthropometric variables contain measurement error: heavier persons are more likely to underreport their weight, and this may lead to underestimates of a country's obese population (Chou et al., 2004).[14] In the Spanish context, previous studies indicate that self-reported body mass suffers from systematic underreporting (Quiles-Izquierdo and Vioque, 1996). Aranceta et al., (1998, 2000) studied the prevalence of obesity in Spanish adult population aged 25-60 using cross-sectional nutrition surveys performed on regionally representative random samples[15]. They found that 13.6% of adult Spaniards were obese in 1997 and 14.5% in 2000. The fact that the prevalence of obesity as estimated from our database is lower (12.8%) reflects the expected underestimation in self-reported weight data. Unfortunately, there is no way to estimate a correlation coefficient for our total sample (as did Chou et al., 2004) even though self-reported obesity at the regional level is highly correlated with, and follows the patterns found in, observational studies.

[13] Unfortunately, BMI does not take into consideration body composition (adiposity vs. lean weight) or body fat distribution. This means it may fail to predict obesity among very muscular individuals and the elderly.

[14] Interestingly enough, Chou et al. (2004) find that the correlation between corrected and uncorrected measures of obesity is 0.86 for obesity and 0.99 for BMI.

Obesity can be broadly conceptualized as an imbalance between energy or calories consumed and expended in a given period. In this energy equation, calories may be expended at work (depending on the nature of one's occupation), doing chores at home, or in active play. With this in mind, our empirical specification uses information on physical exertion on the job and on leisure physical activities (sports). We expect to find a negative association between obesity and level of physical exercise. However, it is worth noting a trend in developed societies towards a reduction in the number of hours allocated to leisure activities or household chores as a consequence of an increase in hours worked and in labor force participation rates. The long-term growth in average weight experienced in the recent decades in the USA is due, in part, to a decline in physical activity at home and on the job (Lakdawalla and Philipson, 2002). When the economy strengthens, BMI and obesity increase, physical activity is reduced, and diets become less healthy (Ruhm, 2000). New location patterns favor higher obesity rates by reducing the relative "price" of travel by car and consequently raising the price of other travel options (Vandegrift and Yoked, 2004).[16]

In SDISH-1999, the physical effort involved in the subject's main job or occupation is a categorical variable with four response categories: sitting down for most of the day; standing for most of the day with little movement or exertion; walking, carrying loads, or moving about frequently; and hard work that requires considerable physical exertion (the omitted category). Leisure-time physical activity is classified into four similar categories: sedentary activities (e.g., reading, watching TV, going to the cinema, etc.); some physical exercise or playing a sport occasionally (walking, gardening, leisure workouts); physical

[15] Weight and height were measured on each individual by trained observers following standardized procedures and measuring instruments. The samples were pooled together and weighted according to the distribution of Spanish adult population aged 25-60 years.

[16] However, it should be noted that measuring this feature implies computing a 'generalized cost' of a given trip including time devoted to travel.

activity several times a month (tennis, running, swimming, cycling, etc.); and playing a sport several times a week (the omitted category).

The other variable in the energy balance equation is caloric intake. There is ample evidence of a positive relationship between obesity and a diet consisting of the consumption of energy-dense foods high in saturated fats and sugars. Economic growth, the globalization of food markets, the availability of fast food, and technological change (the introduction of convenience foods and foods requiring minimal preparation time) are the main forces driving societies to less healthy diets (Philipson, 1991). Unfortunately, our data set did not distinguish the specific types of food normally consumed (e.g., fresh fruit, cereals, meat, eggs, fish), which would have helped us to assess the composition of an individual's diet. Instead, we used data on eating habits during the six months preceding the interview. Digestion has a so-called "thermic effect" such that the more often an individual eats, the more calories are burned (de Jonge and Bray, 1997; Ma et al., 2003)[17] Thus, one expects to find a negative association between obesity and the frequency of food consumption. In light of this, we include a variable for "eating habits" which takes the value of 1 if the subject is reported to eat small amounts of food many times during the day and is otherwise zero.[18] (See Table 1.)

Other key determinants of obesity and certain chronic diseases—heart disease and hypertension, in particular—have to do with an individual's smoking and alcohol drinking decisions. Smokers, it has been shown, have higher metabolic rates than non-smokers and consume fewer calories than non-smokers. Kahn et al. (1997) presented evidence of an inverse relationship between continuing smoking and BMI; Pinkowish (1999) reported that

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[17] Eating frequently is associated with lower insulin concentrations than eating less frequently; insulin tends to increase the deposit of fat.

individuals who quit smoking typically gain weight. Tobacco consumption (cigarette, cigar, and/or pipe smoking) was defined as a dichotomous variable that equals 1 if an individual smoked on a daily basis ("Daily smoking") and is zero if the individual did not smoke or smoked just occasionally. The empirical evidence showing that increased alcohol consumption leads to weight gain is somewhat mixed (cf. Prentice, 1995 and Kahn et al., 1997). Nevertheless, drinking is taken into account in the regressions so long as the alcohol consumed a has high caloric content. The questionnaire asked each interviewee aged 16 and over about the consumption of alcoholic drinks (wine, beer, whisky, etc.) with meals. Again, one binary category was computed ("Daily alcohol consumption") with a value of 1 if a subject drank on a daily basis and zero otherwise (see Table 1).

Finally, our empirical specification monitored socio-demographic categories such as age, age squared, gender, income, income squared, education, and marital status. It should be mentioned that the income variable collected by SDISH-1999, total monthly net income earned by the household, was measured as a categorical variable with 9 response intervals. An interval regression model was calculated to find a continuous household income measurement. The explanatory variables were: age, age squared, male (the omitted category is female), 4 educational categories (the omitted group is illiterate and unschooled), 18 regional variables (the omitted category is Andalusia), private health insurance[19] (the omitted category is public health coverage), and 5 labor activity categories: employed, unemployed, pensioner, student, and inactive[20] (the omitted category). Table A1 in the Appendix gives the results. Once

[18] This variable was explicitly excluded from the diabetes equation. Some treatments for diabetes may lead individuals to increase the frequency of meals in order to regulate their glucose level. Accordingly, the variable was potentially endogenous.

[19] Private insurance includes several forms of insurance all of which provide some sort of entitlement to health care based on the payment of regular fees.

[20] The inactive category includes housekeeping (but not as paid employment), unpaid social work, disabled for work, and other situations.

household income was worked out, we divided it by an equivalence factor (the number of household members powered to 0.5) to adjust for differences in household size. The natural logarithm of net equivalent income and its squared value are used in the equations. The level of education attained by the individual is used as another measure of economic status. This multi-response question (up to 9 responses) was adequately redefined into four dichotomous categories: university education or equivalent, secondary school, primary school, and unschooled and illiterate (omitted category).[21]

3.2. Preliminary evidence

The means and standard deviations of the variables used in the empirical analysis are given in Table 2. Preliminary data analysis of the prevalence of chronic diseases in our sample ranges found 14.89% with hypertension, 10.24% with high cholesterol, 7.26% with heart disease, and 6.06% with diabetes.[22] 12.81% of adults are obese and the mean body mass index, BMI, is around 25.33 kg/m^2 (Std. Dev.= 4.11).

[Insert Table 2 about here]

Certainly, the dataset confirms the accumulated evidence that the prevalence of these diseases is higher for certain population groups. Effectively, the data reveal that cardiovascular diseases are almost non-existent at younger ages but highly prevalent for those over 60 (15.42%). Although no statistical differences were found by gender, consideration of the respondent's age shows that heart disease is more prevalent in men (17.01%) than in women (14.34%). It

[21] Secondary school includes the medium-grade vocational training cycle, the second stage of secondary General Education, and the higher-grade vocational training cycle. Primary school includes primary education or the equivalent and the first stage of secondary General Education.

[22] As a consequence of deleting some missing observations from the dataset, these chronic disease rates are slightly higher, and the obesity rate is slightly lower, than in the original

is also worth noting that the incidence of heart disease was much lower than average (2.20% vs. 7.26%) when the subject's job was reported to involve considerable physical exertion. Fewer men suffer hypertension (11.36%) than women (17.88%) and the prevalence of this chronic illness is also much higher for the older population, affecting approximately 30% of those over 60.

A similar picture emerges for the two other chronic illnesses. High cholesterol is more prevalent in women (11.45%) than in men (8.81%) and is also concentrated in the older segment of the population (17.18%). Interestingly, the prevalence of high cholesterol is low among high consumers of tobacco and those who do physical activities. As for diabetes, the evidence is that it increases with age, sedentary lifestyle, and excess weight. The prevalence of diabetes in individuals younger than 30 is barely 0.4%, whereas the figure rises to 13.02% for respondents over 60. Interestingly, the diabetes rate for those respondents over 60 with the highest levels of job-related or leisure-time physical activity is just 1.69% or 3.36%, respectively, .

It should be noted that obesity is associated with specific characteristics such as gender, age, and income. The prevalence of obesity is higher in females (13.63%) than in males (11.85%). It rises in line with age: just 3.37% of the people younger than 30 are obese, compared to 18.76% of those aged 60 and over. Microeconomic data show that obesity is negatively related to income. Close to 19% of the lowest income bracket are obese, compared with 4.58% of the group with the highest earnings. Marital status and education are also thought to have a notable effect on body mass, and our data seem to confirm this. We find that only 6.13% of single people are obese, far below the rates for those who are married (14.71%) or widowed (19.79%). Obesity is negatively related to education in our sample. Only 5.28% of those who reached higher education and 5.46% of those with a secondary education are obese, far below

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sample (n=69,555), which had 14.66% with hypertension, 9.99% with high cholesterol, 7.21% with heart disease, 5.93% with diabetes, and 13.28% obese.

the 22.16% rate for unschooled or illiterate people and the 13% rate for those who attended only primary school.

The negative association between the consumption of cigarettes and calorie intake is well recognized in the literature. Effectively, our data confirm this: the rate of obesity among adults who smoke on a daily basis, 8.06%, is much lower than the rate for non-smokers and infrequent smokers, 14.72%. Obesity is also negatively related to leisure-time physical activity. Those who spent their spare time playing sports several times a week had an obesity rate of just 6.72%; by contrast, a sedentary life-style was associated with a 15.76% obesity rate.

It seemed possible that some of the variables included in the model might exhibit some multicollinearity problems. We investigated this issue using Tolerance Factors and Variance Inflation Factors (VIF) for the continuous variables of the estimated equations, namely age, age_square, income and income_square. The results, reported in Table A2 in the Appendix, unambiguously indicate the absence of multicollinearity. For the remaining dichotomous variables, we employed a χ^2 Pearson test. With the exception of a few specific dichotomous variables (occasional smoking; moderate and low alcohol consumption) all the remaining variables passed the χ^2 test.

4. Results

Table 3 reports the results of the joint estimation of the probability of an individual being obese and the prevalence of self-reported diabetes, high cholesterol, hypertension and heart disease using the seemingly unrelated bivariate probit model described in Section 2.4.1. As expected, the variable ρ is significant, positive, and different from zero at the conventional 5% level for all chronic illnesses examined. This indicates that a joint estimation procedure might be needed when there are common factors affecting the propensity to

chronic illness and obesity. Moreover, the positive and statistically significant coefficient indicates that obesity is likely to increase the probability of each chronic illness investigated, although to a different extent for each. The ρ is about 0.22 for arterial hypertension, 0.15 for diabetes, 0.08 for high cholesterol, and 0.06 for heart disease. To avoid endogeneity problems, we excluded eating habits, smoking, and drinking for the chronic disease equations. Although no instruments are needed at this stage, marital status variables are maintained for identification purposes.

[Insert Table 3 about here]

The results allow us to examine the joint determinants of the chronic illnesses. We find a significant gender effect in the prevalence of chronic illnesses and obesity: women are more likely than men to suffer from these conditions after controlling for other relevant covariates. This confirms previous empirical evidence from Spain (Martínez et al., 2004) which depicted a gendered pattern in the prevalence of obesity. Furthermore, the model considers possible interaction effects between gender and age, income, alcohol consumption, and smoking. The age-gender interactions suggest that older women are more likely to be obese and to suffer high cholesterol and arterial hypertension. The income-gender cross-effects unambiguously indicate that more affluent men are more likely to suffer obesity and all four chronic illnesses. Finally, males who consume alcohol daily are more likely to be obese than those who drink less, consistent with the findings of Costa-Font and Gil (2004). Age is positively associated with obesity and with all four of the chronic illnesses analyzed, although the significance and negative effect of age squared points to a likely quadratic effect in the propensities for obesity and each of the chronic illnesses.[23] This effect may be explained by the fact that survival

[23] The inflection points for the age variable are at age 81 for diabetes, 67 for high cholesterol, 79 for arterial hypertension, and over 100 for heart disease.

probabilities start to diminish significantly after age 50, after which natural selection may play a role preserving healthier individuals.

Two other relevant determinants are income (more generally, individual affluence, which we examine employing a quadratic structure) and social interactions. We find that income has a quadratic effect on obesity and on the probability of diabetes and of heart disease; this effect is not confirmed for the other illnesses, suggesting that a higher income reduces the probability of some chronic diseases consistent with the health production model presented earlier. However, the quadratic effect of income on obesity suggests the existence of a socio-economic pattern underlying obesity which does not necessarily affect low-income individuals but does affect middle-income groups. This indication that the socio-economic vector driving obesity may be less straightforward than heretofore expected is relevant in the light of the literature on inequalities in obesity (Zhang and Wang, 2004). We also find, in line with previous studies of Spain (Costa-Font and Gil, 2004), that being married increases the probability of obesity, and that those who are single or divorced are less likely to be obese. This suggests that social interactions exert some influence on the generation of body weight.

The final set of determinants are individual lifestyle choices. Table 3 specifically reports on the effect of physical exercise at work and in leisure time. As expected, the less physically active one is at work, the higher the probability of diabetes, hypertension, heart disease, and obesity. The effect of physical exercise in spare time is less evident in general; however, our findings do indicate that playing sports several times per month decreases the probability of suffering high cholesterol and heart disease. As expected, our dataset confirms that individuals with sedentary jobs who exercise not at all or only occasionally during their spare time are more likely to be obese. Interestingly, the existence of a positive interaction in the data between the number of meals and obesity contradicts the so-called “thermic effect” of food. Finally, we find that smoking

or drinking on a daily basis reduces the probability of obesity, though the interaction effects suggest that this applies mainly to men.

From these estimates we have obtained conditional probabilities that suggest that that obesity increases the probability of diabetes by 43% (from 6.06% to 8.64%), the probability of hypertension by 47% (from 14.89% to 21.94%), the probability of high cholesterol by about 20% (from 10.24% to 12.26%), and the probability of heart disease by about 15% (from 7.26% to 8.35%). Overall, the results indicate that significant morbidity could be avoided by lowering obesity rates.

Table 4 provides the estimates from the seemingly unrelated probit with endogenous obesity model described in Section 2.4.2. For all four chronic diseases the Wald test and the Likelihood-ratio test support the evidence of endogeneity. However, distinct from the coefficients obtained in Table 3, here the estimated correlation coefficient ρ is negative. This effect results from two opposed self-selection mechanisms attributed to the effect of unobservable variables that now appear as playing an opposite effect consistently with other applications (Fabbri et al., 2004). The effect of obesity is significant and positive for all chronic illnesses. This finding is consistent with previous literature (Paeratkul et al., 2002; Jung, 1997; Sowers, 2003; Hurbert et al., 1983; Murlow et al., 2000). Furthermore, it is worth noting that the coefficients indicate significant heterogeneity in the specific impact of obesity, which if not taken into account could bias the results obtained. With endogeneity accounted for, the gender-specific effects are not significant for either high cholesterol or heart diseases; this suggests that the 'gender effects' are likely channeled through the obesity-specific effect. Age displays a quadratic effect. This effect can be explained in several ways, first of all, by the presence of common unobserved features affecting the prevalence of chronic illnesses at the later stages of an individual's working life and life cycle. It can also be explained by a non-linear depreciation pattern for individual health: at retirement age,

individuals may experience a slight jump in health status consistent with the findings of Kerkhofs and Lindeboom (1997) who, using data from the Netherlands, showed that retirement improves overall health. And, as before, the non-linear effects are consistent with the 'natural selection hypothesis'. In this model, income-specific effects are significant for only one of the four chronic diseases, diabetes, indicating that some income-specific effects may be channeled through the prevalence of obesity. The positive effect for diabetes may provide evidence of a socio-economic vector for the disease after controlling for the obesity-specific effects. However, the non-linearity of income may justify dismissing this last assertion.

[Insert Table 4 about here]

The effects of lifestyle choices change once we account for endogeneity. Table 4 suggests that jobs with no or little physical exercise tend to increase the prevalence of diabetes, hypertension, and heart disease. A sedentary life in one's leisure time reduces the prevalence of diabetes and high cholesterol. A relatively intense sporting life—playing tennis, running, swimming, or cycling several times per month—reduces the prevalence of high cholesterol and heart disease, but has no significant effect on arterial hypertension. In old age, males are more likely to suffer from heart disease and females are more likely to suffer from high cholesterol and arterial hypertension. Finally, the model shows that affluent males are more likely to suffer from high cholesterol, diabetes and arterial hypertension.

Table 5 reports the results of the two-stage probit least squares model introduced in Section 2.4.3, which uses the body mass index (BMI) instead of the dichotomous variable for obesity used in the first two models. Again, changing the specification of the model affects the robustness of the results. The effect of (log) BMI systematically exhibits a positive and statistically significant coefficient in explaining the prevalence of each chronic disease. The significance of the gender effect disappears. The non-linear effect of age

remains for high cholesterol, hypertension, and heart disease but is absent for diabetes.[24] Income is only significant for hypertension, which suggests that income influences body mass rather than directly impacting specific chronic illnesses, and that inequalities in the prevalence of chronic illnesses are likely to be channeled through the effect of income on obesity rather than being the result of pure health inequality. As expected, lifestyle choices are important factors associated with the prevalence of chronic illnesses but exert heterogeneous effects. A sedentary job exerts a significant and positive effect on the prevalence of diabetes, arterial hypertension, and heart disease, but a sedentary life during spare time reduces the probability of suffering diabetes, high cholesterol and arterial hypertension. In explaining the determinants of chronic illnesses, certain cross-effects are again key determinants. Older males are more inclined to suffer from diabetes and heart disease while older females are more likely to suffer from cholesterol. Other interaction effects were not significant.

[Insert Table 5 about here]

Table 6 reports the marginal effects of obesity over each of the chronic illnesses examined using the seemingly unrelated probit model accounting for endogeneity (2.4.2). We find that the marginal effect is close to 0.01 for arterial hypertension and considerably smaller for the other chronic conditions. The elasticity of obesity evaluated at the mean indicates that a 10% increase in obesity causes increments of 6.7% in diabetes, 2.4% in heart disease, 1.7% in high cholesterol, and 1.4% in arterial hypertension. On the basis of these results, we projected the marginal effects of age-gender obesity onto the latest population projections for Spain for 2005-2030. We estimate that over the next 25 years the male obese population will increase by 33% and the female obese population by 37%. (See Figure A1 in the Appendix.)

[Insert Table 6 about here]

[24] For this econometric specification the age inflection points are 67 years old for high cholesterol, 98 for arterial hypertension, and just 37 for heart disease, which has a U-shaped

Figure 1 presents the marginal effects of each chronic illness by age group, accounting for the effect of obesity (adjusted) and without such an adjustment. The prevalence of diabetes without accounting for obesity displays a downward age effect, but when the effects of obesity are incorporated we find that the diabetes rate increases up to age 60 and then declines. There is a significant gap between the obese and the non-obese in the probability of suffering hypertension and heart diseases. For high cholesterol the effect of obesity is less noticeable, with the greatest effect at around 60 years. On the basis of these marginal effects, we estimate that by 2030, the number of non-obese diabetics will decrease by 5.3% while the number of obese diabetics will increase by 40.6%. We expect increases in the obese population suffering from hypertension and cholesterol of 35% and 38%, respectively, while for the total population we project smaller increases of 12.7% and 21%, respectively. The obese population suffering from heart disease is expected to increase by 57% while the total population with heart disease will grow by only 51%. In relative terms, the overall effect of obesity is less remarkable for heart disease than for the other chronic illnesses examined.

[Insert Figure 1 about here]

5. Conclusion

This paper examines the influence of obesity jointly with other determinants on the prevalence of four chronic conditions, type 2 diabetes, cardiovascular disease, hypertension, and high cholesterol, using Spanish micro data. We accounted for potential unobserved heterogeneity in the propensity to develop these chronic conditions and for potential endogeneity issues concerned with obesity-specific effects by using a simultaneous seemingly unrelated probit model. Our findings provide suggestive evidence of the role of obesity as a

path, affecting the very old at a greater rate.

cause of several chronic diseases. This justifies the concern, currently being voiced in many Western countries, that obesity is an important cause of morbidity with a significant impact on health care costs. In our empirical model, we found evidence that supports the need for correcting for the endogeneity of obesity in explaining the prevalence of chronic conditions. Indeed, accounting for obesity as an endogenous variable had a significant effect on the determinants of chronic diseases. Moreover, when the body mass index (BMI) is employed instead of a dichotomous variable for obesity we found that the effect on three out of four chronic conditions—diabetes, hypertension, and high cholesterol, but not heart disease—was statistically significant and positive, suggesting that even a slight degree of overweight may affect these chronic conditions. We found evidence of a gender-specific effect as well as a gender interaction effect with income and age. An income-specific effect appeared to be specification-dependent. Physical exercise, both at work and in one's spare time, appeared to be a strong predictor of the prevalence of chronic disease.

Our estimates suggest that obesity explains a (heterogeneous) increase in the prevalence in all four chronic diseases studied with elasticities that vary from 0.67 to 0.14 depending on the specific illness. This means that a reduction of obesity rates could lead to remarkable reductions in the morbidity and mortality resulting from these diseases, with consequent savings for the health system and for the human capital of the country under examination. One of the diseases studied, cardiovascular disease, is the main cause of death in Spain (52% of all deaths), and all are important causes of premature mortality. Accordingly, significant indirect improvements in life expectancy could be obtained by reducing the prevalence of obesity. In addition, the possibility of co-morbidities among survey respondents should be acknowledged, which indicates that reducing obesity might have simultaneous effects on the reduction of several chronic illnesses at once. The effects of obesity are especially prevalent among the 40-to-69 age groups for both men and women, which indicates that unless

health promotion policies specifically tackle obesity we should expect it to rise considerably. Such an increase will have a dramatic effect on the probability of diabetes and a lead to less dramatic although sizeable rise in other chronic illnesses.

Major caveats need to be taken into account. First, our results are based on self-reported data, which could bring their validity into question. Although we have found that our estimates are, in aggregate, quite consistent with previous observational studies, some bias might be still present due to underreporting of weight and height. Second, possible unobserved heterogeneity could limit the validity of our estimates, as could the fact that the survey is a cross-section. However, assuming that the results hold valid, they suggest that policies fostering healthier individual lifestyles could positively influence obesity rates, which could in turn be expected to indirectly reduce the prevalence of chronic diseases.

[Insert Tables A1 and A2 and Figure A1 at the end of the article, in an Appendix]

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Table 1. Variable definition

Variable	Definition
Diabetes	Dichotomous variable that equals 1 if respondent is diabetic
Heart disease	Dichotomous variable that equals 1 if respondent has heart disease
Hypertension	Dichotomous variable that equals 1 if respondent has hypertension
High cholesterol	Dichotomous variable that equals 1 if respondent has high cholesterol
Obese	Dichotomous variable that equals 1 if Body Mass Index $\geq$30 kg/m^2
BMI	Body Mass Index (i.e., weight in kilograms divided by the square of height in meters, kg/m^2)
Male	Dichotomous variable that equals 1 if respondent is male
Age	Age of respondent
Age squared	Square of age of respondent
Income	Logarithm of total net equivalent income
Income squared	Square of logarithm of total net equivalent income
University education	Dichotomous variable that equals 1 if respondent attained university education or equivalent
Secondary education	Dichotomous variable that equals 1 if respondent attained secondary education [a]
Primary education	Dichotomous variable that equals 1 if respondent attained primary education (includes the first stage of secondary level education)
Unschooled	Dichotomous variable that equals 1 if respondent is unschooled or illiterate
Sedentary job	Dichotomous variable that equals 1 if respondent is sitting down for most of the day in his/her main job or occupation
Job with little movement	Dichotomous variable that equals 1 if respondent is standing for most of the day, with little movement in his/her main job or occupation
Job with frequent movement.	Dichotomous variable that equals 1 if respondent is walking, carrying loads or moving about frequently in his/her main job or occupation
Hard work	Dichotomous variable that equals 1 if respondent is involved in hard work or a job that call for considerable physical exertion
Sedentary life	Dichotomous variable that equals 1 if respondent does sedentary activities in spare time
Occasional exercise	Dichotomous variable that equals 1 if respondent practices some physical exercise occasionally in spare time (i.e., walking, gardening,…)
Some physical exercise	Dichotomous var. that equals 1 if respondent plays a sport several times a month in spare time (i.e. tennis, running, swimming, cycling,…)
Intensive phys. exercise	Dichotomous variable that equals 1 if respondent plays a sport several times a week in spare time
Eating habits	Dichotomous variable that equals 1 if respondent ate small amounts of food many times each day in the last 6 months
Daily smoking	Dichotomous variable that equals 1 if respondent smokes daily at present
Daily alcohol consumption	Dichotomous variable that equals 1 if respondent drinks alcohol on a daily basis at present
Single	Dichotomous variable that equals 1 if respondent is single
Widowed	Dichotomous variable that equals 1 if respondent is widowed
Divorced	Dichotomous variable that equals 1 if respondent is divorced or separated

[a] Secondary education includes the second stage of secondary education and the medium and higher grade vocational training cycle.

Table 2. Means and standard deviations (n=54,159 observations)

Variable	Mean	Standard deviations
Diabetes	0.0606	(0.238)
Heart disease	0.0726	(0.259)
Hypertension	0.1489	(0.356)
High cholesterol	0.1024	(0.303)
Obese	0.1281	(0.334)
Log. of BMI	3.2191	(0.160)
Male	0.4592	(0.498)
Age	50.166	(20.05)
Age squared	2918.61	(2054.9)
Income	11.384	(0.540)
Income squared	129.89	(12.27)
University education	0.1088	(0.311)
Secondary education	0.1867	(0.390)
Primary education	0.4765	(0.499)
Unschooled	0.2281	(0.420)
Sedentary job	0.3415	(0.472)
Job with little movement	0.5066	(0.500)
Job with frequent movement	0.1159	(0.320)
Hard work	0.0360	(0.186)
Sedentary life	0.4538	(0.498)
Occasional exercise	0.3667	(0.482)
Some physical exercise	0.0614	(0.240)
Intensive physical exercise	0.1181	(0.323)
Eating habits	0.0229	(0.149)
Daily smoking	0.2581	(0.438)
Daily alcohol consumption	0.1787	(0.383)
Single	0.2929	(0.455)
Widowed	0.1384	(0.345)
Divorced	0.0290	(0.168)

Table 3. Seemingly unrelated bivariate probit model (n=54,159)

	Diabetes	High Cholesterol	Arterial Hypertension	Heart Disease	Obese
Male	-1.9619 (-4.10)	-0.7390 (-1.97)	-1.3182 (-3.61)	-0.9574 (-2.19)	-1.3674 (-4.00)
Age	0.0967 (15.84)	0.1297 (32.10)	0.1294 (30.32)	0.0442 (11.34)	0.0772 (26.47)
Square of age (10^{-3})	-0.5954 (-12.48)	-0.9610 (-29.10)	-0.8226 (-24.05)	-0.1504 (-4.66)	-0.6018 (-23.15)
Log of equivalent income	1.8365 (3.30)	0.7207 (1.71)	0.7728 (1.90)	1.1373 (2.26)	2.1658 (5.44)
Square of log equivalent income	-0.0918 (-3.70)	-0.0371 (-1.99)	-0.0414 (-2.30)	-0.0582 (-2.60)	-0.1069 (-6.05)
Sedentary job	0.3152 (3.96)	0.0532 (1.07)	0.2394 (4.24)	0.3519 (4.88)	0.1171 (2.69)
Job with little movement	0.1720 (2.17)	0.0116 (0.24)	0.1941 (3.47)	0.1318 (1.83)	0.0391 (0.91)
Job with frequent movement	0.2074 (2.51)	0.0183 (0.35)	0.1573 (2.66)	0.1000 (1.32)	-0.0075 (-0.16)
Sedentary life	-0.0377 (-0.98)	-0.0557 (-1.92)	0.0402 (1.37)	0.0432 (1.22)	0.3171 (11.26)
Occasional exercise	0.0283 (0.74)	-0.0194 (-0.67)	0.0587 (2.00)	0.0249 (0.70)	0.1552 (5.41)
Some physical exercise	-0.1271 (-1.71)	-0.1295 (-2.60)	-0.1016 (-1.86)	-0.1340 (-2.01)	-0.0492 (-1.06)
Eating habits					0.0956 (2.04)
Daily smoking					-0.2332 (-6.97)
Daily alcohol consumption					-0.1103 (-3.06)
Single					-0.1155 (-5.23)
Widowed					0.0362 (1.58)
Divorced					-0.0850 (-1.94)
Male*age	-0.0025 (-1.67)	-0.0115 (-9.94)	-0.0085 (-7.50)	0.0017 (1.42)	-0.0082 (-8.84)
Male*income	0.1859 (4.67)	0.1167 (3.73)	0.1461 (4.83)	0.0869 (2.35)	0.1638 (5.64)
Male*daily smoking					0.0492 (1.20)
Male*daily alcohol consumption					0.0922 (2.16)
Constant	-14.0794 (-4.50)	-8.5873 (-3.61)	-9.0558 (-3.94)	-9.1035 (-3.22)	-14.2380 (-6.35)
Log pseudo-likelihood	-29,595.56	-35,045.30	-37,365.31	-31,020.46	
Rho (ρ)	0.1506 (11.22)	0.0777 (6.61)	0.2225 (21.21)	0.0588 (4.43)	
Wald test	121.13	43.41	420.28	19.46	

Note: All regressions include regional (17 Autonomous Communities) dummies. The *t-ratios* (based on Huber/White or robust standard errors) are in brackets. For reasons of ease of explanation, one obese regression is shown.

Table 4: Seemingly unrelated bivariate probit model with endogeneity (n=54,159)

	Diabetes	High Cholesterol	Arterial Hypertension	Heart Disease
Obese	1.2946 (15.85)	0.5642 (4.22)	0.8430 (9.36)	0.9280 (9.76)
Male	-1.3383 (-2.89)	-0.5062 (-1.35)	-0.9669 (-2.64)	-0.5666 (-1.32)
Age	0.0776 (13.70)	0.1208 (27.06)	0.1147 (25.58)	0.0334 (8.78)
Square of age (10^{-3})	-0.4551 (-10.26)	-0.8938 (-24.92)	-0.7105 (-19.94)	-0.072 (-2.29)
Log of equivalent income	1.2183 (2.29)	0.4801 (1.16)	0.3641 (0.90)	0.7282 (1.49)
Square of log equivalent income	-0.0608 (-2.57)	-0.0250 (-1.36)	-0.0210 (-1.17)	-0.0378 (-1.74)
Sedentary job	0.2583 (3.37)	0.0348 (0.70)	0.2105 (3.74)	0.3139 (4.45)
Job with little movement	0.1494 (1.96)	0.0050 (0.10)	0.1838 (3.30)	0.1203 (1.71)
Job with frequent movement	0.1980 (2.48)	0.0186 (0.36)	0.1579 (2.69)	0.0988 (1.34)
Sedentary life	-0.1079 (-2.93)	-0.0879 (-2.99)	-0.0169 (-0.57)	-0.0056 (-0.16)
Occasional exercise	-0.0015 (-0.04)	-0.0321 (-1.10)	0.0347 (1.19)	0.0060 (0.17)
Some physical exercise	-0.1190 (-1.68)	-0.1264 (-2.56)	-0.0973 (-1.80)	-0.1304 (-2.01)
Male*age	-0.0005 (-0.39)	-0.0106 (-9.00)	-0.0071 (-6.24)	0.0027 (2.30)
Male*income	0.1224 (3.18)	0.0922 (2.93)	0.1088 (3.57)	0.0483 (1.34)
Constant	-10.5749 (-3.53)	-7.2194 (-3.05)	-6.7177 (-2.93)	-6.8444 (-2.49)
Log pseudo-likelihood	-29,548.81	-35,039.65	-37,345.95	-31,000.83
Rho (ρ)	-0.5376 (13.27)	-0.2298 (3.20)	-0.2428 (4.86)	-0.4313 (9.10)
Likelihood-ratio test	69.99	6.82	12.89	32.24
Wald test	116.60	9.53	21.77	63.49

Note: All regressions include regional (17 Autonomous Communities) dummies. The *t-ratios* (based on Huber/White or robust standard errors) are in brackets.

Table 5: Two-stage probit least squares model (n=54,159)

	Diabetes	High Cholesterol	Arterial Hypertension	Heart Disease
Log. BMI_hat	6.3676 (8.65)	1.7490 (3.31)	5.2776 (9.67)	5.5079 (8.04)
Male	-0.1879 (-0.35)	-0.2557 (-0.65)	0.1442 (0.36)	0.5717 (1.17)
Age	0.0119 (1.09)	0.1061 (13.24)	0.0591 (7.20)	-0.0302 (-3.04)
Square of age (10^{-3})	0.0343 (0.41)	-0.7859 (-12.79)	-0.3005 (-4.80)	0.4038 (5.34)
Log of equivalent income	-0.4579 (-0.74)	0.0814 (0.18)	-1.1225 (-2.45)	-0.8738 (-1.52)
Square of log equivalent income	0.0194 (0.69)	-0.0062 (-0.30)	0.0506 (2.45)	0.0390 (1.50)
Sedentary job	0.3192 (3.92)	0.0540 (1.09)	0.2415 (4.19)	0.3488 (4.82)
Job with little movement	0.1784 (2.21)	0.0122 (0.25)	0.1966 (3.45)	0.1330 (1.85)
Job with frequent movement	0.2344 (2.76)	0.0249 (0.48)	0.1758 (2.91)	0.1222 (1.60)
Sedentary life	-0.1734 (-4.05)	-0.0913 (-2.93)	-0.0718 (-2.22)	-0.0723 (-1.81)
Occasional exercise	-0.0387 (-0.95)	-0.0369 (-1.24)	0.0032 (0.11)	-0.0324 (0.85)
Some physical exercise	-0.1172 (-1.56)	-0.1268 (-2.52)	-0.0935 (-1.71)	-0.1251 (-1.83)
Male*age	0.0947 (4.76)	-0.0082 (-5.52)	0.0014 (0.95)	0.0120 (6.85)
Male*income	-0.0496 (-1.00)	0.0521 (1.43)	-0.0485 (-1.32)	-0.1162 (-2.54)
Constant	-20.3733 (-6.30)	-10.2503 (-4.31)	-14.3020 (-6.02)	-14.3446 (-4.79)
Log likelihood	-10,537.11	-15,983.11	-18,455.51	-11,914.75
Likelihood-ratio test	3,683.26	3,810.04	8,660.86	4,362.29
Pseudo R2	0.1488	0.1065	0.1900	0.1547

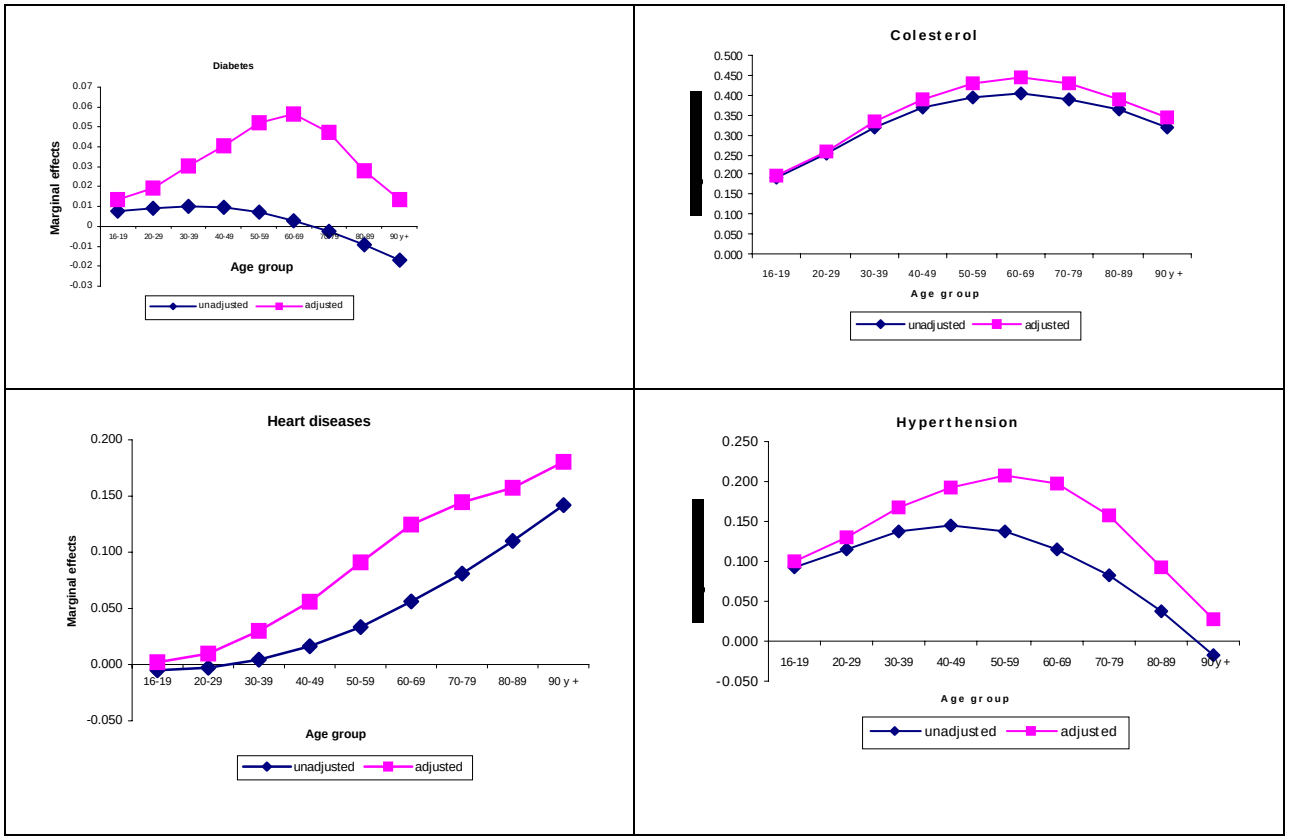
Note: All regressions include regional (17 Autonomous Communities) dummies and incorporate corrected standard errors.

Table 6. Marginal effects and elasticity of obesity on chronic diseases

	Marginal Effect of Obesity	Elasticity of Obesity
Diabetes	0.0031 (9.87)	0.6704 (7.66)
High Cholesterol	0.0057 (8.30)	0.1706 (3.25)
Arterial Hypertension	0.0092 (6.84)	0.1450 (3.29)
Heart Diseases	0.0036 (12.87)	0.2431 (3.19)

Note: Marginal effects and elasticities were computed using the model in Table 4. Marginal effect refers to a discrete change of dummy variable obesity from 0 to 1. Elasticities are calculated in the form of d(lny)/d(lnx). The *t-ratios* are in brackets.

Figure 1. Marginal effects of each chronic illness by age group, adjusted and unadjusted by obesity



Appendix. Table A1. Interval regression for net household income (n=54,159)

Variables	Coefficient	Std. Error	t-Student
Age	1,543.43	129.26	11.94
Age squared	−18.79	1.23	−15.24
Male	2,736.61	894.09	3.06
University education	123,422.50	1,657.43	74.47
Secondary School	61,260.63	1,487.08	41.19
Primary School	23,446.65	1,110.86	21.11
Region 2 (Aragon)	13,077.34	2,183.93	5.99
Region 3 (Asturias)	30,998.58	2,439.91	12.71
Region 4 (Balearic Is.)	24,901.09	2,650.24	9.40
Region 5 (Canary Is.)	10,409.82	2,216.22	4.70
Region 6 (Cantabria)	19,460.57	2,862.00	6.80
Region7 (Castile-Leon)	9,822.35	1,459.25	6.73
Reg.8 (Castile-Mancha)	5,822.25	1,764.64	3.3
Region 9 (Catalonia)	25,553.04	1,546.95	16.52
Regi.10 (C. of Valencia)	13,546.46	1,689.10	8.02
Reg.11 (Extremadura)	5,784.02	2,341.07	2.47
Region 12 (Galicia)	14,554.42	1,652.39	8.81
Region 13 (Madrid)	38,107.57	1,795.06	21.23
Region 14 (Murcia)	3,334.74	2,503.45	1.33
Region 15 (Navarre)	52,419.44	2,824.99	18.56
Regi.16 (Basque Country)	21,950.07	2,266.98	18.56
Region 17 (Rioja)	3,517.90	3,985.07	0.88
Reg.18 (Ceuta-Melilla)	56,033.82	3,366.33	16.64
Private insurance	36,403.38	1,455.71	25.00
Employed	35,545.17	1,351.76	26.30
Unemployed	−28,725.23	1,869.39	−15.37
Pensioner	−5,303.13	1,307.40	−4.06
Student	41,301.65	2,246.20	18.39
Constant	77,838.31	3,658.42	21.28
σ (sigma)	88,861.24	287.99	
Log likelihood = −100,880.17	LR chi2(28) = 19,056.12		

Note: Cf. Table 1 for definitions of variables. The omitted categories are: female, unschooled and illiterate, Andalusia, public health insurance, and inactive.

Table A2. Estimates of the Tolerance and Variance Inflation factors

Dep. Var.	Independent variables	Tolerance	VIF
Age	Male, income, income2, physical exercise at work, physical exercise in spare time, eating habits, daily tobacco consumption, daily alcohol consumption, civil status, 18 regional variables	0.5105	1.96
Age2	Male, income, income2, physical exercise at work, physical exercise in spare time, eating habits, daily tobacco consumption, daily alcohol consumption, civil status, 18 regional variables	0.5325	1.88
Income	Male, age, age2, physical exercise at work, physical exercise in spare time, eating habits, daily tobacco consumption, daily alcohol consumption, civil status, 18 regional variables	0.8819	1.13
Income2	Male, age, age2, physical exercise at work, physical exercise in spare time, eating habits, daily tobacco consumption, daily alcohol consumption, civil status, 18 regional variables	0.8798	1.14

Note: Typically a VIF value greater than 10 is a matter of concern.

Figure A1. Obese population projections 2005-2030