Center for Economic Institutions

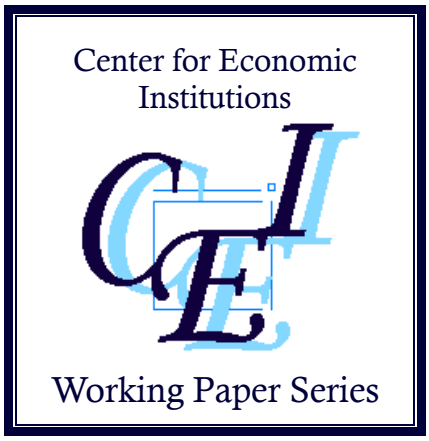
Working Paper Series

No. 2013-8

"Life Cycle Development of Obesity and Its Determinants in Six European Countries"

Sandra Cavaco, Tor Eriksson and Ali Skalli

January 2014



Institute of Economic Research
Hitotsubashi University
2-1 Naka, Kunitachi, Tokyo, 186-8603 JAPAN
http://cei.ier.hit-u.ac.jp/English/index.html
Tel:+81-42-580-8405/Fax:+81-42-580-8333

Life Cycle Development of Obesity and Its Determinants in Six European Countries

January 2014

Sandra Cavaco (LEMMA, Université Panthéon-Assas, France)

Tor Eriksson (Aarhus University, Denmark)

Ali Skalli (LEMMA, Université Panthéon-Assas, France)

Abstract

This paper empirically examines the role and relative importance of parents' and individuals' own socioeconomic status and how their impacts on the probability of overweight and obesity evolve over the life cycle. The impact of individuals' health behaviours on their obesity status later in life is also studied.

We use data from Denmark, Finland, France, Greece, the Netherlands and the U.K. in which about 6,000 individuals aged 50 to 65 are surveyed and where individuals' height and weight at different ages (25, 25, 45 and current age) are available. We perform "repeated cross-sections" analyses as well as dynamic probit analyses of the individuals' obesity histories.

Key findings are: (i) parents' socioeconomic status predicts obesity in early adulthood whereas the individual's own socioeconomic status as adult is more important in explaining obesity at later stages of the life cycle, (ii) changes in obesity status are associated with changes in health behaviours, (iii) obesity in late adulthood is strongly and positively correlated with overweight and obesity in younger ages, and (iv) cross-country differences in obesity and overweight largely remain after controlling for parental and childhood factors and individuals' health behaviours.

JEL Code: I12

Keywords: Obesity, socioeconomic status, life cycle, cross-country differences

Acknowledgements: The data used in this paper was produced by a project called SOCIOLD funded by the European Union (Contract no. QLK6-2002-02292). We are grateful to the participants in the project for the efforts and numerous discussions that resulted in this data set which covers six European countries. We are also thankful to Ian Walker, Peter Dolton, and participants at a seminar at Hitotsubashi University for very helpful suggestions.

1 Introduction

Obesity is associated with social and economic costs that have been clearly identified in the literature. These include co-morbidities and health effects that result in a lower health-related quality of life (Yancy *et al.*, 2002 and Larsson *et al.*, 2002) and most importantly, in a lower life expectancy (Thomson *et al.*, 1999, Allisson *et al.*, 1999, Fontaine *et al.*, 2003, Peeters *et al.*, 2003, Koch, 2011). This, in turn, induces huge economic costs to society. A significant proportion of health expenditures can be clearly attributed to obesity (e.g. Cawley and Meyerhoefer, 2012). Also, part of the employees' absenteeism from work is due to obesity and its health effects (e.g. Cawley *et al.*, 2007, Finkelstein *et al*., 2005). Cawley (2004) reports that for white females in the US., a difference in weight of two standard deviations (roughly 65 pounds) is associated with a difference in wages of 9 percent, which is equivalent to the wage effect of roughly one and a half years of education or three years of work experience. This wage loss likely reflects the negative correlation between weight and productivity.[1] Notably, Cawley and Spiess (2009) show that obesity among children early in the important pre-school period (2-3 years) is associated with lower skill attainment.

Thus, the growing prevalence of overweight and obesity is becoming a major concern for policy makers.[2] Combating the epidemic requires that the determinants of obesity are well understood. The two determinants that have received most attention, in particular by economists[3], are individuals' socio-economic environments (Goldblatt *et al.*, 1965, Sobal and Stunkard, 1989, Stunkard and Sorensen, 1993, Brunner *et al.*, 1997, Strauss and Knight, 1998, Paeratakul *et al.*, 2002, Zhang and Wang, 2004, Gordon-Larsen *et al.*, 2006) and the energy intake/energy consumption differential (Prentice and Jebb, 1995, Cutler *et al.*, 2003, and Drewnowski and Specter, 2004).

[1] This would also imply that obesity is harmful to economic growth as well. Although we know of no study measuring the effect of obesity on economic growth, there are good reasons to believe that such a measure might not be negligible at least because obesity is negatively correlated to health, which is in turn positively correlated to growth as suggested by endogenous growth models (see van Zon and Muysken, 2001 and Gourdel *et al.*, 2004 as well as the corresponding empirical evidence reported by Bloom and Canning, 2003 and Bloom *et al.*, 2004).

[2] In 2003, the International Obesity Task Force announced that overweight and obesity worldwide was estimated to involve 1.7 billion people. See Deitel (2003). See also Rössner (2002) as well as James *et al.* (2001). See Seidell (1995) for a European perspective and Knai *et al*., (2007) for the specific case of Eastern Europe. Also, Hedley *et al.* (2004), Rolland-Cachera *et al.* (2002) and Rennie and Jebb (2005) offer country-specific studies for the US, France and Great Britain, respectively.

[3] For overviews of the economic approach to obesity, see Acs and Lyle (2007), Grossman and Mocan (2011) and Cawley (2011).

The standard economic approach to obesity is to think of it as the consequence of technological improvements which on one hand have lowered food prices,[4] and on the other hand have made physical exercise more expensive due for instance to changes in work-life contributing to increased physical inactivity (Lakdawalla and Philipson, 2009).[5] The impact of these changes are frequently said to have been strengthened by decreased strenuousness of work, increased female labour force participation and urbanisation. For instance, Wronka and Pawlinska-Chmara (2007) report that Polish women who had attended day care centres are 2.4 cm shorter than girls brought up at home by their mothers and that mothers who work often do so at the cost of time devoted to the family which influences health and the rate of their children's development. Interestingly, the energy-needs/energy-intake balance relates to socio-economic status (henceforth: SES). Low SES individuals are not only less likely to have access to physical activity facilities, but are also more likely to resort to low quality/fat-dense food (Gorden-Larsen *et al.*, 2006, Drewnowski and Specter, 2004). Asfaw (2007) shows that, *ceteris paribus*, the odds of being overweight/obese are 80.8% higher for Egyptian micronutrient deficient mothers than for non-deficient mothers.

Yet, the observed trends in obesity suggest that public health policy recommendations for avoiding overweight and the common practice of dieting fail to prevent an increase in the prevalence of obesity (Seidell, 1995). This may be due to the curative dimension having been given more attention than prevention *per se*. Indeed, a number of authors have insisted on the idea that prevention programs are more efficient in combating obesity than weight-loss programs (Hill and Peters, 1998, Visschell and Seidell, 2001, Wang *et al.*, 2003). Prevention requires identification not only of the determinants of the current state of affairs, but also of the way overweight and obesity evolve over a person's life cycle.

While a major theme in the current research on the determinants of health is the role and importance of circumstances early in life, and during childhood in particular. This focus is, however, less

[4] The reduction in food production costs having resulted in mass preparation of food and increased availability of fast-food and calorie dense-food.

[5] Prentice and Jebb (1995) observe that while average recorded energy intake in Britain has declined substantially, obesity rates have escalated. Their conclusion is that levels of physical activity, and hence energy needs, have decreased even faster and that modern inactive lifestyles are at least as important as diet in the etiology of obesity and possibly represent the dominant factor. Using national data, Ulijasjek and Koziel (2007) find that the most plausible macro-level explanations for the obesity patterns observed in East European nations are declines in physical activity, increased real income, and increased consumption of goods that contribute to physical activity decline: cars, televisions and computers. Likewise, using the Russia Longitudinal Monitoring Survey for 1994 and 2004, Huffman and Rizov (2007) find a strong positive effect of diet/caloric intake and a strong negative effect of smoking on weight and BMI. The role of inactive lifestyles is also underlined in the East-German study by Zellner *et al.* (2007).

pronounced in research on overweight and obesity.[6] Stunkard *et al*. (1986) highlight the role of genetic factors. Whitaker *et al.* (1997) explore the importance of childhood and parental obesity as predictors of adulthood obesity. Some of the channels of inter-generational transmission have also been identified, be it via physical activity patterns (Fogelholm *et al.*, 1999) or parental feeding styles (Wardle *et al.*, 2002).

In addition, the few analyses that are based on longitudinal data are not informative enough regarding the life cycle dimension, the time span they cover in general being rather short. The same holds for economic analyses as relatively few of them have been concerned with the relative contributions of the factors influencing obesity growth; see Bleich *et al.* (2008) for a notable exception.

The aim of this paper is to empirically examine the role and relative importance of a variety of factors known to have an influence on the probability of overweight and obesity with a special focus on how these factors' impacts on the probability of overweight and obesity evolve over the life cycle. The information in our data allows us to simultaneously consider a variety of factors such as the childhood environment, parent's education and SES, own education as well as health behaviour, including diet style and physical activity. In addition, the retrospective nature of the information available to us allows us to link individuals' life-cycle weight profiles to health behaviour histories.

While the results from studies based on cross-sectional data reflect steady state or cumulative effects, exploring the life cycle dimension is likely to shed light on the dynamics of obesity. One would for instance like to know how likely an individual is to remain obese at a given age, conditional on her obesity history. For the U.S., Daouli *et al.* (2013) estimate hazard models showing that the probability to exit obesity is inversely related to the duration of the obesity spell. Equally important is the evaluation of how the respective effects of parents' and own SES evolve over individuals' life cycles as this may suggest different policy designs, depending on the targeted population's age. Likewise, while the effects of smoking or exercising are now well-established, it could be much more informative to know how they evolve over the life cycle and, most importantly, how conditional they are on one's obesity history and/or on one's past smoking or exercising behaviours.

---

[6] In their article, Must and Strauss (1999) review what they call "the small body of research on the long-term morbidity and mortality associated with childhood obesity". See also the birth cohort study by Eriksson *et al.* (2001).

Exploring the dynamics of obesity and overweight is not new. Baum and Rhum (2009) as well as Michaud *et al.* (2007) use longitudinal data to assess the effect of a variety of obesity determinants in the US and Europe, respectively. However, while the former rely on a sample of individuals aged 16-23 when they are first observed and 39-46 in the latest wave of the panel, the latter use a panel of 50 year old individuals observed thrice only. Thus, in contrast to these two studies, we can follow individuals from their twenties up to their fifties thanks to the retrospective nature of our data and hence, assess the relationship between obesity and its determinants during a longer part of individuals' life cycles.

While assessing the above mentioned effects in a life cycle perspective, we also put emphasis on two specific issues which we think are very important. The first of these is gender. For a variety of biological, cultural and/or economic reasons, not only gender-specific weight-age profiles are likely to be different, but the effects of their determinants may be different as well.

The second dimension we consider is cross-country differences in obesity patterns. Although there is a relatively large cross-country variation in obesity rates that can be helpful in the search for explanations for the causes of obesity growth, this dimension is rarely explored. Very few attempts have been made following up on Cutler *et al.* (2003) to examine determinants of country differences. Yet, as emphasized by Wang (2001), the prevalence of obesity varies remarkably across countries with different levels of socioeconomic development. Different socio-economic groups are at different risks and the relationship between obesity and socio-economic status varies across countries.[7] Except for Michaud *et al.* (2007), Villar and Quintana-Domeque (2009) and Brunello *et al.* (2009), we know of no other studies using comparable micro-data to analyse cross-country variation in obesity patterns.

The paper is organised as follows. Section 2 presents the data. Section 3 raises some econometric issues and motivates the adopted empirical approach. Section 4 discusses the results. Section 5 consists in a general discussion of the implications one could infer from the results and contrasts these to other studies. Section 6 concludes.

[7] Of course, more remarkable is the difference between countries with different development levels. The literature review by Monteiro *et al.* (2004) shows that prior to the 1990's, obesity in the developing world was essentially considered a disease of the socioeconomic elite. As a country's gross national product increases, the burden of obesity in developing countries tends to shift towards lower SES groups.

2. Data description

The data used in our empirical analysis was constructed within an EU project called SOCIOLD within the framework of which a common detailed internet-based[8] questionnaire in six different European countries (Denmark, Finland, France, Greece, the Netherlands and the U.K.) was carried out. The questionnaire was answered by about 5,500 individuals aged 50 to 65 at the time of the survey (in 2004). Besides a host of variables describing individual traits[9] and socio-economic factors (including parents' socioeconomic status and health problems), the questionnaire collected retrospective information about individuals' weight, height, and health-related behaviour. We use this information to construct body mass index (henceforth: BMI) measures at different ages – 25, 35, 45 and current age (in the age range 50 to 65), which we correlate with a number of key obesity determinants discussed in the literature.

Table 1 shows, for each country in our sample, obesity rates calculated from the SOCIOLD data set at different ages as well as measures of the extent to which the individuals exercised or were smoking at the same ages. Not surprisingly, the likelihood of being obese increases with age. Part of this increase is of course the general trend increase in obesity during the last forty years. For those countries where the obesity is higher when the individuals were in their mid-thirties, the rate is currently (when they are in their mid-fifties) about twice as high, whereas for countries that start out with a lower average obesity rate, it is now three to four times higher. As can be seen from *Table 2*, the onsets of overweight as well as obesity problems occur relatively early: of the currently obese about ninety (seventy) per cent were already obese or overweight at age 45 (35). The cross-country variation in *Table 1* corresponds to what has been documented in previous studies; see Andreyeva *et al.* (2007). The aggregate obesity rate differentials are relatively small, while the country differences in gender specific obesity rates are larger. However, compared to the U.S., the levels are considerably lower; see Michaud *et al.* (2007) for a trans-Atlantic comparison.

[8] The survey has been conducted by private poll companies in the different countries. However, due to the lower information technology penetration in Greece, the questionnaire was carried out as a standard mail questionnaire in that country. The resulting sample size was 5,073 (897 in Denmark, 474 in Finland, 1,003 in France, 1,001 in Greece, 971 in the Netherlands and 727 in the United Kingdom) while the exploitable sample size was 4,595 (692 in Denmark, 415 in Finland, 963 in France, 961 in Greece, 964 in the Netherlands and 604 in the United Kingdom). We have compared descriptive statistics for a number of socio-demographic variables, from the SOCIOLD sample with their counterparts in the 2001 wave of the European Community Household Panel (ECHP). Only minor differences, partly due to differences in sample sizes, emerge from the comparison, which is reassuring with respect to the representativeness of our sample. A table summarizing this comparison is available from the authors upon request.

[9] In addition to conventional variables like age, gender, marital status, education, employment status, occupation, and income, the questionnaire also asks the respondents about their psychical and mental health, social networks and support.

Insert Table 1 about here

Insert Table 2 about here

Returning to *Table 1*, we may further note that with the exception of Greece, smoking is markedly more prevalent when the individuals were in their twenties after which it gradually becomes less common. The same pattern is observed for exercising, but the decline with age is less pronounced.

In the remainder of the paper we will follow Currie *et al.* (2007) and Baum and Ruhm (2009) and adopt the mother's level of education as our measure of parental socioeconomic status. Maternal education has been found to be more closely related to offspring's health. This is because mothers play a more central role in creating their children's health and diet behaviours (Eriksson *et al.*, 2001, Wardle *et al.*, 2002). From *Table 3*, we can see that the lower the maternal education is the higher is the obesity rate. The differences are not strikingly large, however, and appear small in comparison with those found in Baum and Ruhm's (2009) study for the U.S. While the obesity rate of young offspring of U.S. mothers with a low level of education is twice as high as for those whose mother has a high level of education, in the European countries studied here the relative difference at age 25 is about 13 per cent. The U.S.-Europe difference may reflect the fact that earnings differences between educational groups are considerably larger in the U.S. Note, however, that offspring of mothers with a low level of education are twice more likely to be severely obese than their counterparts whose mothers are highly educated. Perhaps equally interesting is the observation that in general, males are less likely to be obese than females irrespective of whether they were in their twenties or in their fifties and that the correlation between one's likelihood to be obese and her/his mother's education is much higher for males than for females at age 25 and lower when their age exceeds 50.

Insert Table 3 about here

In the bottom half of the table, we find the gradient with respect to the respondent's own socioeconomic status. Here we may note the larger differences in particular at younger ages.[10] Again, irrespective of their own educational level, males are in general less likely to be obese than

[10] This indicates that one should not exclude the possibility that there may be some "reverse causality" here. For an analysis of the effect of obesity on educational attainment, see Kaestner *et al.* (2011). See Stunkard and Sorensen (1993) for an analysis of the complexities of the association between obesity and socio-economic status, including reverse causality and the role of confounders.

females. However, the correlation between one's likelihood to be obese and her/his own education is systematically lower for males than for females.

As can be seen from *Table 4*, both regular physical exercise and (especially) smoking are activities the individuals in our sample are rather persistent in pursuing. Thus, about three fourths of those who exercise regularly in their fifties also did in their twenties and thirties. Most people begin smoking early and hence 95-96 per cent of the individuals in the sample who are currently smoking were already smokers 10-20 years earlier. Interestingly enough, at every age, men are more likely to exercise regularly and to smoke than women. Persistence in both exercising and smoking is, moreover, slightly higher for men than for women.

Insert Table 4 about here

As is well-known, self-reported weight and height are under- and over-reported, respectively, and the magnitude of the reporting bias is typically found to be larger for females. Cawley and Burkhauser (2006) use the National Health and Nutrition Examination Study (NHANES) for the U.S. to assess how objectively measured height and weight are related to self-reported height and weight. Respondents to the NHANES are asked to report their weight and height although these are also objectively measured. Cawley and Burkhauser (2006) compare both measures by regressing objectively measured weight and height on self-reported weight/height while controlling for demographic characteristics. Michaud *et al.* (2007) use the coefficients estimated by Cawley and Burkhauser (2006) to assess the bias in self-reported weight and height in the Health and Retirement Survey (HRS) and the 2004 wave of the Survey of Health, Ageing and Retirement in Europe (SHARE) and conclude that the applied correction has a large impact on obesity rates for the population aged 50 and above. *Table 5* below compares Michaud's *et al.* (2007) obesity rates based on self-reported and corrected height/weight to those drawn from the SOCIOLD data, the comparison being of course restricted to the four countries investigated in this study as well as by Michaud *et al.* (2007). The comparison indicates that under-reporting (over-reporting) of weight (height) is less severe in the SOCIOLD data than it is in SHARE; with the exception of Danish males and Greek females, obesity rates in SOCIOLD are even larger than Michaud *et al.* (2007)

estimates.[11]Another question is that of the extent to which these self-reporting biases vary over individuals' life cycles, a question which to the best of our knowledge, has not been examined.

Insert Table 5 about here

Of course, the best way to assess obesity determinants in a life cycle perspective would be to use panel data on objective measures of weight and height and covering the whole life period of a significant number of sampled individuals. As emphasized above, such data are rare and this is what renders the use of recall data a necessity. In this paper, we present two sets of results. One set is based on the assumption that major changes in a person's weight probably belong to those salient features of people's health histories that are well remembered and hence reported accurately. The study by Smith (2009) shows that the widespread scepticism against retrospectively collected information is a too negative view. We see these results as a benchmark to which our other results as well as those already reported in the literature can be compared. This is made possible thanks to the use of fairly frequently adopted econometric specifications to explain the probability that an individual be obese, conditional on a number of characteristics.

The second set of results we present relies on the assumption that, on the contrary, under- and over-reporting in the recall data we use might yield misleading results if the estimated specifications are not purged from measurement error bias. Below, we propose an estimation strategy where measurement error is thoroughly accounted for.

3. Empirical set-up and econometric issues

Following the standard economic approach to identify determinants of obesity, the health production function (Lakdawalla and Philipson, 2009, Lakdawalla *et al.*, 2005) we think of BMI as the outcome of a combination of different health production inputs. As we have information about individuals' weight and height and health behaviours at different ages, we are able to perform "repeated cross-section" analyses. That is, we estimate the same production functions for the same individuals when they were 25, 35, 45 as well as at their current age (between 50 and 65). We

[11] This might be due to the correction coefficients that Michaud *et al.* (2007) use for European countries being those estimated by Cawley and Burkhauser (2006) for white Americans.

believe this approach usefully sheds light on the way the weight production process evolves over individuals' life cycles.

In the health production models we estimate, we think of BMI as the health outcome of choices made during four different stages of the individual's life cycle:

$$(1)\quad \textit{Prob(obese)} = \alpha_0 + \alpha_1\, \textit{SES(mother)} + \alpha_2\, \textit{SES(own)} + \alpha_3\, \textit{illness (parents)} + \alpha_4\, \textit{health behaviours} + \alpha_5\, \textit{gender} + \textit{country dummies}$$

Mother's SES is expected to be negatively related to the probability of being obese. Several explanations have been offered for this relation.[12] Firstly, socioeconomically disadvantaged parents give their children food with a proportionally high share of low cost energy dense food. Moreover, they cannot afford to spend money on exercise activities for the children to the same extent as better off parents do. As diet and physical activity habits are established during childhood and in teenage years, the effects of parental socioeconomic status will persist also into adulthood; see Case *et al.* (2002). Secondly, parents with lower socioeconomic status are likely to have higher discount rates (Borghans and Golsteyn, 2006) or less self-control (Cutler *et al.*, 2003), and if these traits are transmitted to their offspring, this will affect their health behaviours and will result in overweight and obesity as adults.

But circumstances early in life may also affect an individual's BMI as adult only because she ended up in the same socioeconomic status category as her parents. To account for this possibility it is important to also include the person's own SES as an additional regressor. This allows us to examine what is important: only early life conditions, adult status, or both.

In addition to the SES variables, (1) also includes dummy variables for illnesses that the individual's parents suffered from and which are known to be common co-morbidities of obesity. These dummies capture the potential intergenerational transmission of obesity or the impact of intergenerational similarities in lifestyles.

As discussed above, another important determinant of overweight and obesity is health behaviour which we also control for in specification (1) by including lifestyle indicators such as exercising and smoking.

[12] See Cutler *et al.* (2011) for a comprehensive discussion of the SES-health relationship.

Basically, a separate equation should be estimated for each gender as there are biological and cultural factors that may make men and women react differently to any of these same obesity determinants. Hence, equation (1) is a benchmark specification where data for both genders are pooled, but where a gender dummy is included in order to measure average gender differentials in obesity. To control for cultural, institutional and economic cross-country differences in obesity patterns, specification (1) also includes country dummies.

As mentioned above, direct estimation of (1) might yield misleading results if under- and over-reporting of weight and height lead to severe measurement errors in the data. To overcome this problem, we proceed in the following way. Let w_i and h_i denote the weight and height of individual *i*, respectively. We have:

$$BMI_i = \frac{w_i}{h_i^2}$$

and $\ln(BMI_i) = \ln(w_i) - 2\ln(h_i)$, which implies that $\Delta\ln(BMI_i) = \Delta\ln(w_i) - 2\Delta\ln(h_i)$. Given that individuals stop growing before their twenties, in our data $\Delta\ln(h_i) = 0$, and hence:

$$\Delta\ln(BMI_i) = \Delta\ln(w_i)$$

Thus, an alternative specification of (1) would be the following:

(2) $\Delta\ln(w_{it})$ = a_0 + a_1 *SES(mother)* + a_2 *SES(own)* + a_3 *illness (parents)* + a_4 *health behaviours* $_{(t-1)}$ + a_5 *gender* + *country dummies*

This has several advantages. First, although the only time-varying right-hand side variables are those describing health behaviours, (2) includes some dynamics as the a_4 parameter measures the effect of health behaviours ten years earlier on weight variation over the subsequent ten years. Second, it allows us to avoid estimating non-linear models and adoption of ad hoc assumptions concerning the likelihood function (normality in the case of a probit model). Third, it avoids classifying marginal individuals as being non obese (or the other way around), due to reporting bias. Most importantly, suppose individuals under-report their weight and let w_{it} denote the reported weight when the actual one is w_{it}^*. That is, $\ln(w) = \ln(w^*) + v$ where v denotes measurement error. Differencing of the left-hand side variable in (2) implies that $\Delta\ln(w) = \Delta\ln(w^*) + \Delta v$ and, therefore, leads to two different scenarios. The optimistic scenario is one where measurement error is constant over time so that $\Delta v = 0$, that is, differencing eliminates the measurement error. A more pessimistic

scenario would be one where $\Delta v \neq 0$ and where measurement error is still an issue. In this case, however, measurement error affects the left-hand side of the estimated specification, which means that it is the variance-covariance matrix of the estimated coefficients which would be biased, not the estimated coefficients themselves. What actually happens in this case is that the residuals become dependent on the extent of measurement error in the left-hand side variable and the problem translates into a heteroskedasticity problem. We address this in *Table 6b* which reports heteroskedasticity corrected estimates using individuals' weight a decade ago as the weighting variable, the underlying assumption being that reporting bias in weight is correlated with one's weight itself.

Of course, some of the other right-hand side variables of interest in (2) may also be measured with error; health behaviour indicators, in particular. Note, however, that unlike (1), (2) is linear and is therefore estimable by use of standard methods (e.g. OLS or FGLS). In this case, for any (recall data) variable, say *x*, on the right-hand side, let α be the corresponding coefficient estimate had there been no measurement error and *a*, the biased estimate (say, OLS). It is well-known that:

$$\operatorname{plim} a = \alpha\left(1 - \frac{\sigma_v^2}{\sigma_x^2}\right) < \alpha$$

where *v* denotes the measurement error (i.e. $x = x^* + v$). This suggests that measurement error results in downward biased estimates, understating the true effects of the corresponding right-hand side regressors on the dependent variable: They thus measure lower bounds of the effects of interest. Put differently, positive effects are understated while negative ones are overstated.

We also exploit the longitudinal nature of the data and estimate random effects dynamic models of individuals' obesity histories.[13] The aim of the dynamic analyses is to assess the extent to which obesity at a given age is related to obesity at younger ages. Thanks to the retrospective questions regarding the individual's height, weight and some health behaviours, we have a panel consisting of four waves with ten year intervals (except the last one for which this is an approximation). The model to be estimated is:

[13] Estimation of probit models with fixed effects encounters two shortcomings that have been well-documented in the literature: a practical one due to likelihood maximization being highly cumbersome (e.g. Lancaster, 2000, Hsiao, 2003) and a methodological one due to the so called incidental parameter problem (e.g. Greene, 2004) The latter issue is even more problematic when it comes to dynamic probit models with fixed effects (see Nerlove, 1967, 1971) and Nickell (1981).

$$(3) \qquad y_{it}^{*} = \gamma y_{it-1} + \mathbf{x}_{it}\beta + \alpha_i + u_{it}$$

where y_{it}^{*} is the latent dependent variable, $\mathbf{x}_{it}$ is a vector of exogenous explanatory variables, α_i are individual-specific random effects, and the u_{it} are assumed to be normally distributed. The coefficient γ is the state dependence parameter. The observed binary outcome variable (obesity) is defined as $y_{it} = 1$ if $y_{it}^{*} \geq 0$ and $y_{it} = 0$ otherwise.

For estimation of (3), we have to address two problems: (i) the treatment of initial conditions (y_{i1}) and (ii) persistence and unobserved individual heterogeneity (u_i). Estimation of the model requires an assumption about the initial observations, y_{i1}, and in particular about their relationship with the individual random effects, α_i. Making the assumption that the initial conditions, y_{i1} are exogenous allows us to estimate the model. It should, however, be noted that if the y_{it-1} are correlated with the α_i, this estimator will overstate the extent of state dependence.

Table 7a presents the outcome from applying three different estimators. The estimates in the first column are simple pooled probit estimates. This specification ignores heterogeneity and is likely to overestimate the coefficient on the lagged variable. The random effects probit specification (second column) allows for unobserved heterogeneity, but treats the initial conditions as exogenous. The third estimator uses Wooldridge's (2005) specification, which allows for the endogeneity of the initial conditions, but assumes no autocorrelation in the error term.[14] Wooldridge (2005) proposed a Conditional Maximum Likelihood estimator that considers the distribution conditional on the initial period value (and exogenous variables).

Reliability of the results from specification (3) that are reported in *Table 7a* depends on how reliable is the assumption that individuals accurately recall and (retrospectively) report their weight and height. To account for the possibility that this is a too strong assumption, we propose an alternative testing strategy, in line with the one we apply in estimating (2). That is, we consider an alternative specification where the log of BMI is differenced once. Therefore, the main variable of interest is $\Delta\ln(BMI_{it}) = \Delta\ln(w_{it})$, the idea being that differencing reduces measurement error. Thus, the alternative specification we estimate is:

$$(4) \qquad \Delta\ln(w_{it}) = \gamma\,\Delta\ln(w_{it-1}) + \mathbf{x}_{it}\beta + \alpha_i + u_{it}$$

[14] Another solution is the two-step estimation method proposed by Heckman (1981) which introduces a set of exogenous instruments. Stewart (2007) proposes an application of these estimators in the context of an investigation of the dynamics of the conditional probability of unemployment. He found similar results for both estimators.

Specifications (2) and (4) share a number of advantages which we described above. However, specification (4) includes $\Delta \ln(w_{it-1})$ as a right-hand side variable and so, any remaining measurement error after differencing will translate into two econometric problems. Measurement error in the left-hand side variable ($\Delta \ln(w_{it})$) will generate heteroskedasticity, which should be accounted for. Again, we correct for heteroskedasticity using individuals' weight ten years earlier as the weighting variable. Measurement error in the right-hand side variable ($\Delta \ln(w_{it-1})$) will yield measurement error bias. We deal with the latter issue by instrumenting $\Delta \ln(w_{it-1})$ by its lagged value as one would naturally do in a generalized method of moments framework. Thus, the results we report in *Table 7b* are purged from measurement error thanks to the correction for heteroskedasticity as well as to the use of the instrumental variable method we just described.

Before turning to the results, a few additional remarks are in order. First, as males and females are physiologically different they may react differently to early life conditions and/or to own SES variables. Although inclusion of a gender dummy in equations (2), (3) and (4) is likely to capture such differences, it may not be informative enough as to how different early life conditions, different life styles and/or different SES are in determining later obesity for males and females.[15] Thus, below we will report both regressions including a gender dummy and separate regressions by gender. The same holds for cross-country differences as there might be cultural, institutional or economic (e.g. food prices) specificities influencing the sensitivity of individuals' weight to some or all of its determinants. Although we have conducted country-specific estimations, to save space we do not report these results.[16] In all of the tables below, we report cluster-correlated Huber-White robust standard errors, clustering being based on country affiliation. The idea is that not only is it likely that country-specific unobserved determinants may play a role, but reporting behaviour might also be country-specific.

Applying a life-cycle perspective, one should also account for the possibility that overweight at a certain age might later translate into obesity. This is not accounted for in specifications 1 (*Table 6a*) and 3 (*Table 7a*) the focus of which is on the likelihood of obesity *per se*. In contrast, specifications 2 (*Table 6b*) and 4 (*Table 7b*) analyze the determinants of growth rates in weight in general with no

[15] Using data on children and adolescents from Cracow, Poland over the 1971-2000 period, Chrzanowska *et al*., (2007) observe the absence of a positive secular trend in BMI among adolescent females relative to males. They argue that this may be due to sociocultural pressures associated with transition to a free market economy in Poland and the fact that the extent to which girls attempt to achieve the ideal body, as portrayed by media and society more generally, increases across adolescence.

[16] The results are available from the authors upon request.

specific emphasis on the potential effect of overweight. This is why we also complement the results in *Tables 6* and *7* by reporting in *Table 8* the results from three different specifications. In the first four columns we estimate for each category an equation similar to specification 1 but where the dependent variable is a binary indicator of overweight (25 < BMI < 30). In the three subsequent columns, we replicate (3), again replacing obesity indicators by overweight ones. In the last three columns of *Table 8*, the latter specification is extended to include lagged obesity status.

Finally, it is worth noting that although we do not report their coefficients, time fixed effects have been included in all of the dynamic specifications reported in *Table 7a* as well as in those reported in *Table 7b* where instrumental variables are not used. Indeed, in *Table 7b*, the dependent variable is differenced once and the lagged value of the difference is entered as an explanatory variable. Because the data are treated as a 4-wave panel, using the lagged difference of the lagged difference of the dependent differenced variable leaves no room for inclusion of time dummies. Thus, the IV specifications in *Table 7b* should be seen as being specific to individuals in their 50s the obesity status of whom is explained by their weight history as well as by the other suspected determinants.

4. Results

4.1. Repeated cross-section estimates

According to the probit estimates displayed in *Table 6a*, there is a SES(mother)-obesity gradient when the individuals are in their mid-twenties and -thirties and this gradient is more pronounced for males.[17] As in Baum and Ruhm's (2009) study, the disparity is increasing in age.[18] However, as from when the respondents are in their forties, the results from the pooled sample as well as from the males sample show that there is still an inverse relationship between obesity and maternal SES, but it is no longer statistically significant. This pattern of first increasing and later narrowing health-SES disparities has also been found for other health outcomes; see Smith (2004). The maternal SES-obesity gradient is, however, more persistent among females as it is associated with a negative and significant coefficient even for women in their fifties. *Table 7b* provides highly interesting complementary results. The coefficients estimated for the whole sample are in general not significant, hence suggesting that weight gain is not associated with mother's education. They even

[17] In presenting the estimation results, in Tables 6a – 8, we have, to save space, omitted the standard errors. Tables including them are available from the authors. We have also carried out estimations of the same probit equations but replacing mother's education with that of the father. The same pattern of results was obtained.

[18] Here as well as in the sequel, we are comparing estimates, not the parameters *per se* as the cross-sectional structure of our data does not allow us to test whether the observed differences are statistically significant.

suggest that individuals whose mother is highly educated gain more weight after their forties, although the coefficient is only slightly significant. The gender-specific results, in contrast, suggest that mother's education is negatively correlated with weight gain over the twenties to thirties period. Overall, these results suggest that not only are individuals with poorly educated mothers more likely to be obese as early as in their twenties, but they may also gain weight before their thirties faster than their counterparts with more educated mothers. But because the results in the first part (four first columns) of *Table 8* also highlight a negative correlation between mother's education and the likelihood of overweight, one could conjecture that individuals whose mothers are only poorly educated and who are not yet obese in their twenties are more likely to become so in their thirties.

Insert Tables 6a and 6b about here

The questionnaire provides no direct income information for the parents when the respondent was a child. However, some indirect questions regarding economic conditions during youth can be used. From *Table 6a* we may see that a variable describing the number of persons per room at age 14 is positively and significantly correlated with obesity when the individuals are in their thirties. The gender-specific estimations show that this is in fact only true for females, and not for males. We may also see from *Table 6b* that it is also positively correlated with weight gain over the twenties-thirties period although no such correlation emerges from the gender-specific samples. This suggests that either these childhood living conditions variable are not really associated with the likelihood of obesity and with weight gain or that they are correlated with some other SES indicator included in these regressions; like respondent's own education. This is confirmed by the results in *Table 8* (columns 1 to 4) which documents no correlation between this variable and the likelihood of obesity.

The likelihood of obesity is decreasing in the respondent's own level of education.[19] The coefficient in *Table 6a* is increasing in age but differs significantly from zero only when the individual is in her fifties or early sixties. However, these estimates mask a large difference between the genders. From *Table 6a*, we may notice, that for males, their own education is not correlated with the probability of obesity at any age. But as a woman becomes older, not only does the association with her parents' socioeconomic background become stronger, but also the importance of her own

[19] Excluding the respondent's own education from the equation leads to only tiny changes in the other coefficient estimates. In particular, it should be pointed out that the insignificance of maternal education when the respondent is in her forties and fifties is also found when the person's own education is omitted.

socioeconomic status grows.[20] This result, which has also been found in Michaud et al. (2007) and Baum and Ruhm (2009), implies that, unlike for men, for women, intergenerational socioeconomic mobility has an impact on obesity which increases in importance later in adulthood. Interestingly, combining this result with the gender-specific ones in *Table 6b* suggests that while the likelihood of obesity is related to a person's own SES among females, the same does not hold for weight gain, although the coefficients from the overall sample displays a positive correlation between own SES and weight gain over the thirties-forties period and for a negative correlation over the subsequent age periods. Interestingly, *Table 8* suggests that for all life cycle stages considered, a person's own SES is uncorrelated with her likelihood of being overweight.

Insert Tables 7a and 7b about here

In addition to mother's education, we also entered two other parental variables: two dummy variables for whether any of the parents had suffered from diabetes or high blood pressure, respectively. Both illnesses are associated with substantially increased probability of overweight (*Table 8*, columns 1-4) and obesity (*Table 6a*). The association with obesity is more pronounced for females, and is hence picking up an intergenerational propensity of becoming obese transmitted by genetic factors or similarities in life style between generations within the family. Both dummies attach positive coefficients and the marginal effects are in general of a magnitude that is larger than those associated with mother's and respondent's own education. It is also worth noting that the two parents' diseases we consider are also associated with individuals' weight gain (*Table 6b*). For women, weight growth is higher over the twenties-thirties period when the parents suffered from blood pressure; for men, it is lower after their forties when their parents suffered from diabetes.

Turning next to the association between the respondent's health related behaviours and her probability of obesity, it should be noted that the questions in the survey regarding smoking and physical exercise are retrospective. More precisely, the questionnaire asked: "Do/did you regularly (minimum two times per week) exercise for at least 30 minutes" and "Do/did you smoke regularly (a positive number of cigarettes or equivalent every day)".[21]

[20] Note that we do not have information about changes in the respondent's SES over her life cycle.

[21] The questionnaire also included questions regarding the respondents' food and alcohol consumption. This information is only available for the current period, however.

As can be seen from *Table 6a*, for both genders regular physical activities are associated with a considerably lower likelihood of obesity.[22] The marginal effect increases strongly with age. Regular physical activities are also negatively correlated with the likelihood of overweight until individuals reach their fifties (*Table 8*).[23] As in other studies, we also find that the correlation between smoking and the probability of obesity is negative and that the marginal effects are quite sizable. The gender-specific estimations suggest, however, that such a correlation is more pronounced for women and for the oldest ages. *Table 6b* confirms this result as it shows that, for both males and females, smoking is also negatively correlated with the weight gain when individuals are beyond their thirties. The results in *Table 8* (Columns 1-4) go in the same direction as they show that it is only when individuals are in their forties or older that the correlation between smoking and the probability of overweight becomes negative, although it is statistically significant for individuals in their forties only, not when they are older.

Insert Table 8 about here

A final observation worth making from the results in *Table 6a* is that the country fixed effects are relatively large in the estimations for the mid-twenties and the mid-thirties but are substantially lower when the respondents are in their fifties and early sixties. When the respondents are in their twenties or thirties the "raw" differences (not shown) are in fact smaller than those observed after controlling for cross-country differences in SES and other factors. On the other hand, the raw differences observed for the respondents in their fifties do not differ much from those remaining after controlling for SES and other explanatory variables in *Table 6a*. Thus, cross-country differences for this age group are not due to differences between countries in the prevalence of diabetes or high blood pressure among parents, maternal education and physical exercise behaviour. In other words, the higher obesity among the elderly in the UK and the Netherlands are due to factors not accounted for in our study and which will be discussed in section 5.

[22] As emphasized by McInnes and Shinogle (2011), physical activity as a factor contributing to obesity growth has received proportionately little attention in economic research. Even if this would be justified – that is, the main causes of obesity would be found elsewhere – it could nevertheless be that part of the solution to the obesity problem is to be found here. See also Gordon-Larsen *et al.* (2006).

[23] The fourth column in *Table 8* indicates that individuals exercising while in their fifties are on average more likely to be overweight. This is in all likelihood due to reverse causation.

Interestingly, although the patterns are similar, the gender-specific results seem to suggest that there are much more cross-country differences in the prevalence of obesity among women than there are among men. Even more interesting is the comparison of the results in the first four columns in *Table 6a* and in *Table 8*. The latter shows that there are cross-country differences in the prevalence of overweight as well. Yet, the patterns of obesity and overweight are not the same. In Denmark and Finland the patterns are quite similar. In Greece, the prevalence of obesity is higher (compared to France, the omitted group) for individuals in their twenties or their thirties whereas the prevalence of overweight is higher for individuals in their forties or older. In the Netherlands, overweight is much more prevalent than obesity for every age group. For the UK, the opposite pattern emerges. It is like if in this country, individuals tend to be either normal-weight or obese and rather seldom in between.

Perhaps equally noteworthy are the results in *Table 6b* as they indicate the way individuals' weight tends to evolve over time. Roughly speaking, individuals' age-weight profile seems to be steeper in France than it is in Denmark or Greece and to a lesser extent, in Finland and the Netherlands. But, compared to France, these countries also show a higher prevalence of obesity, especially in the early stages of individuals' life-cycle, which suggests that their slower weight gain profile is the explanation of why they also show no higher prevalence of obesity at later stages of the life-cycle. In contrast, in the UK, the prevalence of obesity is higher for all ages and, at the same time, individuals in their thirties gain much more weight than their French counterparts.

4.2. Dynamic model estimates

Next we report results from estimations exploiting the "longitudinal" character of the questionnaire data. As mentioned above, three specifications are considered: the pooled probit model, the random effects probit model and the Wooldridge random effects model with endogenous initial conditions. In both *Table 7a* and *Table 8*, we performed likelihood ratio tests to assess the value added of the random effects model compared to the pooled probit model. These tests show systematically that the value added is zero, suggesting the two specifications are roughly equivalent. Yet, we report both models in *Tables 7a* and *Table 8* as the random effects dynamic specification is the one to which Wooldridge's specification is to be compared. Indeed, not only does Wooldridge's specification account for the endogeneity of initial conditions, but it also systematically results in the highest likelihood values.

Turning to the estimates in *Table 7a*, we may first notice that early life conditions as measured by maternal education are associated with later obesity. In particular, we find that having a mother with a high educational level carries a negative and significant coefficient in the pooled ordered probit and random effects probit models. In addition, the gender-specific estimates highlight no such relationship for females. This is probably due to the lagged obesity indicator having captured all of the parental SES effect. Indeed, *Table 6a* suggested that only in early life stages does the latter effect matter. Likewise, none of the coefficients associated with maternal education in *Table 7b* is significant, hence suggesting the latter does not influence weight gains either. Again, this is likely to be due to the lagged variation in weight having captured the expected effect of mother's SES. According to *Table 8*, however, the latter is strongly correlated with the probability of overweight despite the estimated specifications include previous obesity and overweight statuses in their right-hand side. Combining the three sets of results, we may conclude that mother's SES is highly negatively correlated with the probability of overweight which is in turn highly correlated with later obesity statuses. The latter correlation is clearly detectable in columns 5-7 of *Table 8*.

The other variable describing economic circumstances during childhood, the extent to which the individual was living in overcrowded conditions, remains important and shows a sizable positive correlation with the probability of being obese. Note that living in overcrowded housing can reflect two circumstances: small economic means, but also, a large number of siblings. The gender-specific estimates in *Table 7a* suggest, however, that this relation is statistically significant only for females, a result in line with the one found in *Table 6a*. Controlling for previous overweight status in *Table 8* (Columns 5-7) results in the coefficients associated with early living conditions becoming insignificant, suggesting that previous overweight status captures the latter effect. *Table 8* shows that previous overweight status is strongly correlated with current overweight (columns 8-10) as well as with obesity (columns 5-7) status. In addition, when previous overweight as well as previous obesity statuses are controlled for, the association between early crowded living conditions and current overweight status becomes positive and highly significant. Lastly, *Table 7b* shows that living circumstances during childhood are not associated with weight growth over one's life.

Notably, the person’s own SES is strongly correlated with the likelihood of being obese. Again, in line with the results in *Table 6a*, this seems to be true for females but not for males. There also seems to be an own SES-weight gain relationship, although this is statistically insignificant in the gender-specific estimations reported in *Table 7b*. Note also that controlling for previous overweight status, as in columns 5-7 of *Table 8*, results in the coefficient on own SES turning statistically

insignificant. Since for none of the age groups do columns 1-4 of *Table 8* document an SES-overweight gradient (nor does it in the overweight dynamic models in columns 8-11), one cannot infer that previous overweight status is capturing the SES effect in columns 5-7.

Similar to the "repeated cross-section" estimates, we find that having a parent who suffered from diabetes or high blood pressure is associated with a considerably higher probability that the offspring is obese and this finding survives inclusion of previous overweight status among the explanatory variables (*Table 8*, columns 5-7). As mentioned earlier, individuals whose parents suffered from diabetes are more likely to be obese during their twenties or their thirties whereas those whose parents suffered from high blood pressure are more likely to be obese during their forties or later (*Table 8*, columns 1-4). However, adding previous overweight and obesity statuses to the right-hand side variables of the dynamic overweight models (see columns 8-11 of *Table 8*) suggests that only when her parents suffered from diabetes is the offspring more likely to be overweight and the relationship is not precisely estimated. This probably means that obesity/overweight at younger ages is the channel through which parents' diseases influence obesity/overweight at older ages.

Regular physical exercise and smoking are accompanied by a significantly lower obesity risk for both genders (*Table 7a*). However, this reduction is not significant when previous overweight status is controlled for (*Table 8* columns 5-7). This indicates that the latter is capturing the life style effects on obesity risk. Likewise, the corresponding IV estimates in *Table 7b* show no significant association with weight gain either. This is not a counter-intuitive result since these specifications include the lagged variation in weight, the coefficient of which is relatively large and highly significant. Again, this suggests that the latter is picking up the life style effects on the age-weight profile. Note finally, that even when we cater for previous obesity and overweight statuses (columns 8-10 of *Table 8*), there remains a substantially lower probability of being overweight for individuals who are smoking or regular doing physical exercise.

As can be seen from *Table 7a* and columns 8-10 of *Table 8*, the estimate of the lagged obesity variable, which is likely upward biased due to unobserved heterogeneity, is reduced once the latter is accounted for.[24] When we, following Wooldridge (2005)[25], estimate the average partial effect

[24] As pointed out by Stewart (2007), the dynamic random effects probit model and the pooled probit model involve different normalizations, a modification of the coefficient is needed for comparison.

[25] Wooldridge (2005) defines the average partial effect of a regressor for dynamic limited dependent panel data models, where the partial effects on the mean outcome are averaged over the distribution of unobserved heterogeneities.

(APE) with respect to the lagged dependent variable we can see that the APEs of the pooled model probit and of the random effects probit model are very close. However, the Wooldridge specification of the random effects probit model reduces the APE by about a third. Thus, taking into account the endogeneity of the initial conditions, the degree of persistence in obesity is considerably reduced, but remains statistically significant.

Things are different regarding overweight. The coefficient on the lagged overweight variable in the dynamic obesity models in columns 5-7 of *Table 8* is only slightly reduced in Wooldridge's model, suggesting that the bias was not important. The same holds for the corresponding APEs as they are only reduced a little. The association between previous overweight status and current obesity status is therefore insensitive to unobserved heterogeneity and is relatively large and highly significant. This is confirmed by the results in columns 8-10 of *Table 8* where both the lagged overweight and the lagged obesity variables are included. In this model, the coefficients on the lagged overweight variable are even larger than when unobserved heterogeneity is controlled for. However, the corresponding APE in Wooldridge's model lies between its pooled probit and random effects probit counterparts.

Persistence effects also emerge from the results in *Table 7b* where the dependent variable is the wage gain over a decade. We may see that whatever sample is considered, the IV estimate of the persistence effect is systematically larger than its FGLS counterpart, hence confirming the prediction we have made in section 2.2 that failure to correct for measurement error in the data is likely to result in downward biased estimates. Nevertheless, the effect of the lagged variation in weight is systematically large and significant.

5. Discussion

In this section we sum up the results reported in the previous section in an attempt to evaluate their main implications. We begin with the gender dimension, noticing that while *Table 6a* and *Table 8* show that, ceteris paribus, women are less likely than men to be overweight or obese, *Table 6b* suggests that over the life-cycle, they gain weight faster than men. This gender-specific difference in age-weight profiles implies that females and males may be reacting differently to the weight determinants. Consequently, women’s sensitivity to obesity combating policy initiatives may be different, too. For instance, our results in *Table 6a* suggest that while encouraging physical activity may be beneficial for both genders, anti-smoking policies may increase the prevalence of obesity among women, but not among men.

Our results also confirm the existence of an SES-obesity gradient in health that has been documented in other studies.[26] Thus, Baum and Ruhm (2009) find a clear SES-obesity gradient for both genders in their study based on panel data from the National Longitudinal Survey of Youth (NLSY) during the period 1981-2004. When they include both maternal and the individual's own education as regressors, both effects are statistically significant. Adding family income, marital status, number of children and health behaviour variables does not affect the estimated impacts of own and mother's education, suggesting that these factors are not the channels via which the intergenerational transmission of education takes place. Similarly to research on other health outcomes like mortality and self-reported health status (Cutler *et al.*, 2003), the authors find that the SES-obesity gradient widens with age.

Owing to the specific character of the data source used in Baum and Ruhm (2009), the individuals included are relatively young; the average age of the persons in the sample is 31.2 years (in the first year the individuals are between 16 and 23 years old and in the last between 39 and 46). This means that the study is concerned with the development of obesity among people from their early twenties until they turn forty. A common feature of Baum and Ruhm's (2009) study and ours is the dynamic nature of the examined relationships. However, in contrast to Baum and Ruhm, we can also follow individuals beyond the age of forty into their fifties and, given the retrospective nature of the data we use, we are also able to assess the relationship between obesity and its determinants during a longer part of individuals' life cycles.

Our results also document an SES-obesity gradient. However, we find that while maternal SES is associated with obesity in the first half of one's life-cycle (as in Baum and Ruhm's study), the relationship later weakens and is replaced by an association between the individual's own SES and obesity. This is important as it implies that because of the high degree of persistence in obesity patterns, policies targeting adult individuals are likely to reduce the prevalence of obesity among their children whereas initiatives towards the socioeconomic environment of children are likely to prevent obesity among these same children during their adulthood.

As for the individual's own SES, we find it to be much more strongly correlated among with women's probability to be obese, and this correlation is, moreover, increasing with the woman's age. The stronger obesity and own SES relationship was also observed in Michaud et al. (2007) and

[26] Villar and Quintana-Domeque (2009) make use of the European Community Household Panel to examine the obesity-SES relationship. However, as the ECHP only allows linking offspring to parents when these are cohabiting, the samples analysed are likely to be rather unrepresentative.

Baum and Ruhm (2009).[27] Although we do not address the issue of reverse causality in this paper, we find it interesting to note that a number of studies exploring the effect of obesity on economic outcomes show that such an effect is significant for women, but not for men (see e.g., Conley and Glauber (2005) and references cited therein). Our data show that men and women with comparable SES are equally likely to be obese as adults and that although males and females from disadvantaged SES may show similar energy intake behaviours, females energy consumption was probably lower. The people we observe are all born sometime between 1938 and 1953 and at that time, female labour market participation rates were much lower. In addition, female jobs in the least skill-demanding occupations were most of the time rather sedentary and less energy-demanding than they are nowadays. Combined with the significant persistence effect our study highlights, this may explain why the obesity-SES gradient is much stronger for women than it is for men. This is perhaps what underlies the descriptive statistics in *Table 3*: the gender differential in obesity rates increases much faster over age for low educated individuals and/or for individuals whose mother is low educated then it does for the highly educated and/or for offspring of highly educated mothers.

Another dimension we have addressed in this study is that of cross-country differences in the prevalence of obesity and overweight, an issue that is also well documented in the literature. For instance, Michaud *et al.* (2007) make use of comparable micro-data from ten different European countries. The data set has been collected in 2004 as part of the ongoing Survey of Health, Ageing and Retirement in Europe (SHARE) project, the aim of which is to build a European data set corresponding to the Health and Retirement Study (HRS) in the United States. The sample analysed includes individuals aged 50 or above and the focus is on cross-country differences.

Our results also highlight the presence of cross-country differences in the prevalence of obesity and overweight which persist despite the variation in SES, childhood environment, lifestyle indicators we have controlled for. Although not reported, the country-specific regressions we have conducted also reveal some cross-country differences in the sensitivity of the obesity risk to these variables. For example, they confirm the observation of Michaud *et al.* (2007) that there are significant differences in the SES-obesity gradient across countries. These differences are, however, unlikely to be the key explanation of the quite sizeable cross-country differences in obesity/overweight that emerge from our results. Michaud *et al.* (2007) argue that a large portion of the cross-country variation can be attributed to differences in physical activity. Despite the high significance of this

[27] In fact, Michaud et al. (2007), like our study, find no relationship for males.

variable in our study, as in Michaud et al. (2007), quite large differences remain also after controlling for it.

The patterns in the estimates indicate that unobserved country-specific characteristics play an important role. Examples of these are the expenditure spent on food consumed outside home, the relative price of good quality food as compared to fat-dense food and/or the accessibility of sport facilities. There might also be cultural or social factors.[28] Countries differ also in their obesity combating policies. They also differ in their specific histories. The individuals we observe in our data are born around and just after World War II. If parental SES as well as childhood environment are important determinants of early life BMI as the many available studies, including our own, suggest, then it is likely that the BMI distributions we observe today partly reflect these initial conditions.

6. Concluding remarks

The analysis in this paper confirmed the role of a number of obesity determinants, the importance of which has often been stressed in the literature. Socio-economic status, childhood environments, genetic factors, health behaviours show a significant association with the likelihood of individuals being obese. In addition, there are significant cross-country differences in obesity rates which are mostly due to unobserved characteristics and only marginally to differences in observed elements such as socio-economic status or health behaviours.

The main value added of the present analysis is its focus on how the highlighted associations evolve over the life cycle. Thus, our results show that while parents' socio-economic status predicts the probability that individuals will be obese when they are in their twenties or thirties, this effect dissipates at older ages, and is, especially for females, replaced by individuals' own socio-economic status playing a more important role. Likewise, and not surprisingly, the effect of physical activity increases with age, demonstrating its cumulative beneficial influence on BMI. A final example relates to the relative importance of observed and unobserved characteristics in explaining cross-country differences in obesity: the explanatory power of observed differences between the countries with respect to socio-economic status and health behaviours for understanding country differences in obesity among individuals over fifty years of age is quite limited.

[28] Sanchez-Johansen *et al.* (2004) explored the effect of self-image and showed that although Latin-American women weighed less than black women, they perceived their current body image as heavier and reported greater body image dissatisfaction than black women. Christakis and Fowler (2007) highlighted the effect of social networks and concluded that network phenomena are relevant to the biologic and behavioural trait of obesity and that obesity spreads through social ties. See also Ulijaszek (2007) on the relationship between socio-cultural factors and obesogenic environments.

Our life cycle oriented study sheds some new light on the policy implications for obesity combating/prevention programs. For instance, while a short-term obesity prevention policy may aim at widening access to physical activity facilities, our analysis suggests the long-run effects of such a policy would be stronger if the effort is primarily targeted at young people. Likewise, a policy aiming at reducing socio-economic differences in obesity would be more efficient if primarily targeted at young parents with children.

Equally important in this respect are the results from our dynamic analysis. In particular they show how influential and highly significant the past experience of being obese is. Again, this has important policy implications for obesity-prevention programs. The fact that obesity today is strongly and positively correlated with obesity at younger ages may be given two alternative explanations. On the one hand, past experience of being obese may alter the individual's cost of obesity prevention. This may be due to obesity-related metabolic disorders, to obese people becoming less prone to engage in physical activity or simply to living in an obesogenic environment. This means that the past obesity experience generates a behavioural impact on the current body mass. On the other hand, obese individuals may be prone to be obese simply because they possess time-invariant unobservable characteristics affecting their body masses. This would be the case if obesity is genetically inherited or if it is the outcome of unchanged time discounting behaviours.

Obviously, these two potential sources of state dependency have different policy implications. Short-term obese-prevention policies will have significant impacts on the long-term obese status of individuals only in the case of genuine persistency in obesity, not if it is mainly driven by unobserved heterogeneity. Our results show that most of the observed state-dependency reflects the effect of past obesity on individuals' behaviours towards preventing later obesity status. Indeed, even when unobserved heterogeneity is controlled for, the association between obesity status and its behavioural determinants remains highly significant.

Overall, our study shows that a better understanding of obesity patterns necessitates a dynamic approach and especially an analysis of how the effects of the various determinants of obesity and overweight evolve over individuals' life cycle. Efforts should, therefore, be exerted towards the collection of longitudinal or cohort data covering a sufficiently long period of time. This, we believe, should be high on the agenda for future research.

References

Acs, J., Lyles, A., 2007. Obesity, Business and Public Policy. Edward Elgar Publishing.

Allisson, D. B., Fontaine, K. R., Manson, J. E., Stevens, J., van Itallie, T. B., 1999. Annual deaths attributable to obesity in the United States. Journal of the American Medical Association 282(16), 1530-1538.

Andreyeva, T., Michaud, P.-C., van Soest, A., 2007. Obesity and health in Europeans aged 50 years and older, Public Health 121, 497-509.

Asfaw, A., 2007. Micronutrient deficiency and the prevalence of mothers' overweight/obesity in Egypt. Economics and Human Biology 5, 471–483.

Baum, C., Ruhm, C., 2009. Age, socioeconomic status and obesity growth. Journal of Health Economics 28(3), 635-48.

Bleich, S., Cutler, D., Murray C., Adams, A. 2008. Why is the developed world obese? Annual Review of Public Health 29, 273-295.

Bloom, D. E., Canning, D. 2003. Health as a human capital and its impact on economic performance. Geneva Papers on Risk and Insurance: Issues and Practice 28, 304-315.

Bloom, D. E., Canning, D., Sevilla, J., 2004. The effect of health on economic growth: A production function approach. World Development 32, 1-13.

Borghans, L., Golsteyn, B., 2006. Time discounting and the body mass index: Evidence from the Netherlands. Economics and Human Biology 4(1), 39-61.

Brunello, G., Michaud, P.-C., Sanz-de-Galdeano, A., 2009. The rise in obesity in Europe: An economic perspective. Economic Policy. 57, 551-596.

Brunner, E. J., Marmot, M. G., Nanchahal, K. Shipley, M. J., Stansfeld, S. A., Juneja, M., Alberti, K. G. M., 1997. Social inequality in coronary risk: Central obesity and the metabolic syndrome. Evidence from the Whitehall II study. Diabetologia 40, 1341-1349.

Case, A., Darren, L., Paxson, C., 2002. Economic status and health in childhood: The origin of the gradient. American Economic Review 92(5), 1308-34.

Cawley, J., 2004. The impact of obesity on wages. Journal of Human Resources 39(2), 451-474.

Cawley, J., 2011. The economics of obesity. In: Cawley, J. (Eds.), The Oxford Handbook of the Social Science of Obesity. New York: Oxford University Press, 120-137.

Cawley, J., Burkhauser, R., 2006. Beyond BMI: The value of more accurate measures of fatness and obesity -in social science research. National Bureau of Economic Research Working Paper #12291. June.

Cawley, J., Meyerhoefer, C. 2012. The medical care costs of obesity: An instrumental variables approach. Journal of Health Economics 31, 219-230.

Cawley, J., Rizzo, J., Haas, K., 2007. Occupation-specific absenteeism costs associated with obesity and morbid obesity. Journal of Occupational and Environmental Medicine 49, 1317-1324

Cawley, J., Spiess, K., 2008. Obesity and skill attainment in early childhood. Economics and Human Biology 6(3), 388-397.

Chrzanowska, M., Koziel, S., Ulijaszek, S. J., 2007. Changes in BMI and the prevalence of overweight and obesity in children and adolescents in Cracow, Poland, 1971–2000. Economics and Human Biology 5, 370–378.

Christakis, N. A., Fowler, J. H., 2007. The Spread of obesity in a large social network over 32 years. New England Journal of Medicine 357, 370-379.

Conley, D. and R. Glauber (2005), *Gender, Body Mass and Economic Status*, NBER Working Paper #11343, May.

Currie, J., Shields, M., Price, S., 2007. The child health/family income gradients: Evidence from England. Journal of Health Economics 26 (2), 213-32.

Cutler, D., Glaeser, E., Shapiro, J., 2003). Why have Americans become more obese?. Journal of Economic Perspectives 17 (3), 93-118.

Cutler, D., Lleras-Muney, A., Vog, T., 2011. Socioeconomic status and health: Dimensions and mechanisms. In: Glied, S., Smith, P. (Eds.). Oxford Handbook of Health Economics.

Daouli, J., Davillas, A., Desmoussis, M., Giannakopoulos, N., 2013. Obesity persistence and duration dependence: Evidence from a cohort of US adults (1985-2010). Economics and Human Biology, http://dx.doi.org/10.1016/j.ehb.2013.07.002.

Deitel, M., 2003. Overweight and obesity worldwide now estimated to involve 1.7 billion people. Obesity Surgery 13(3), 329-330.

Drewnowski, A., Specter, S.E., 2004. Poverty and obesity: The role of energy density and energy costs. The American Journal of Clinical Nutrition 79(1), 6-16.

Eriksson, J., Forsen, T., Tuomilehto, J., Osmond, C., Barker, D., 2001. Size at birth, childhood growth and obesity in adult life. International Journal of Obesity 25(5), 735-740.

Finkelstein, E. A., Fiebelkorn, I. C., Wang, G., 2005. The costs of obesity among full-time employees. *A*merican Journal of Health Promotion 20(1), 45-51.

Fogelholm, M., Nuutinen, O., Pasanen, M., Myöhänen, E., Säätelä, T., 1999. Parent-child relationship of physical activity patterns and obesity. International Journal of Obesity 23(12), 1262-1268.

Fontaine, K. R., Redden, D. T., Wang, C., Westfall, A. O., Allisson, D. B. 2003. Years of life lost due to obesity. Journal of the American Medical Association 289(2), 187-193.

Goldblatt, P. B., Moore, M. E., Stunkard, A. J., 1965. Social factors in obesity. New England Journal of Medicine 192(12),1039-1044.

Gordon-Larsen, P., Nelson, M. C., Page, P. and Popkin, B. M., 2006. Inequality in the built environment underlies key health disparities in physical activity and obesity. Pediatrics 117(2), 417-424.

Gourdel, P., Hoang-Ngoc, L., le Van, C., Mazamba, T., 2004. Health care and economic growth. Annales d'Economie et de Statistiques 75/76, 257-272.

Greene, W., 2004. The behaviour of the maximul likelihood estimator of limited dependent variable models in the presence of fixed effects. The Econometrics Journal 7(1), 98-119.

Grossman, M., Mocan, N., 2009. (Eds.). Economic Aspects of Obesity. University of Chicago Press.

Heckman, J.J., 1981. The incidental parameters problem and the problem of initial conditions in estimating a discrete time-discrete data stochastic process. Manski, C. F., McFadden, D. L.. (Eds.). Structural Analysis of Discrete Panel Data with Econometric Applications. Chap. 4, Cambridge, MA: MIT Press, 179-195.

Hedley, A. A., Ogden, C. L., Johnson, C. L., Carroll, M. D., Curtin, L. R., Flegal, K. M., 2004. Prevalence of overweight and obesity among US children, adolescents and adults, 1999-2002. Journal of the American Medical Association 291(23), 2847-2850.

Hill, J. O., Peters, J.C., 1998. Environmental contributions to the obesity epidemic. Science 280, 1371-1374.

Hsiao, C., 2003. Analysis of Panel Data. Cambridge: Cambridge University Press.

Huffman, S. K., Rizov, M., 2007. Determinants of obesity in transition economies: The case of Russia. Economics and Human Biology 5, 379–391.

James, P., Leach, R., Kalamara, E., Shayeghi, M., 2001. The worldwide obesity epidemic. Obesity Research 9, S228–S233.

Kaestner, R., Grossman, M., Yarnoff, B., 2011. Effects of weight on adolescent educational attainment. In: Grossman, M., Mocan, N. (Eds.). Economic Aspects of Obesity. University of Chicago Press, 283-311

Knai, C., Schurcke, M., Lobstein, T., 2007. Obesity in Eastern Europe: An overview of its health and economic implications. Economics and Human Biology 5, 392–408.

Koch, D. (2011), Waaler Revisited: The Anthropometrics of Mortality. *Economics and Human Biology*, 9(1), 106-117.

Lakdawalla, D., Philipson, T., 2009. The growth of obesity and technological change: A theoretical and empirical examination. Economics and Human Biology 7(3), 283-293.

Lakdawalla, D., Philipson, T., Bhattacharya, J., 2005. Welfare-enhancing technological change and the growth of obesity. American Economic Review 95(2), 253-7.

Lancaster, T., 2000. The incidental parameter problem since 1984. Journal of Econometrics 95, 391-413.

Larsson, U., Karlsson, J., Sullivan, M., 2002. Impact of overweight and obesity on health-related quality of life: A Swedish population study. International Journal of Obesity 26(3), 417-424.

McInnes, M., Shinogle, J., 2011). Physical activity: Economic and policy factors. In: Grossman, M., Mocan, N. (Eds.). Economic Aspects of Obesity. University of Chicago Press, 249-282.

Michaud, P-C., van Soest, A., Andreyeva, T., 2007. Cross-country variation in obesity patterns among older Americans and Europeans. Forum for Health Economics & Policy 10(2), Article 8.

Monteiro, C. A., Moura, E. C., Conde, W. L., Popkin, B. M., 2004. Socioeconomic status and obesity in adult populations of developing countries: A review. Bulletin of the World Health Organization 82(12), 940-946.

Must, A., Strauss, R. S., 1999. Risks and consequences of childhood and adolescent obesity. International Journal of Obesity 23(2), 2-11.

Nerlove, M., 1967. Experimental evidence on the estimation of dynamic economic relations from time series of cross-sections. Economic Studies Quarterly 18, 42-74.

Nerlove, M., 1971. Further evidence on the estimation of dynamic economic relations from time series of cross-sections. Econometrica 39, 359-387.

Nickell, S., 1981. Biases in dynamic models with fixed effects. Econometrica 49(6), 1417-1426.

Paeratakul, S., Lovejoy, J. C., Ryan, D. H., Bray, G. A., 2002. The relation of gender, race and socioeconomic status to obesity and obesity comorbidities in a Sample of US adults. International Journal of Obesity Related Metabolic Disorders 26(9), 1205-1210.

Peeters, A., Barendregt, J. J., Willekens, F., Mackenbach, J. P., Al Mamun, A., L. Bonneux, L., 2003. Obesity in adulthood and its consequences for life expectancy: A life-table analysis. Annals of Internal Medicine 138(1), 24-32.

Prentice A. M., Jebb, S. A., 1995. Obesity in Britain: Gluttony or sloth? British Medical Journal 311, 437-439.

Rennie, K. L., Jebb, S. A., 2005. Prevalence of obesity in Great Britain. Obesity Reviews 6(1), 11-12.

Rolland-Cachera, M. F., Castetbon, K., Arnault, N., Bellisle, F., Romano, F. C., Lehingue, Y., Frelut, M. L., Hercberg, S., 2002. Body mass index in 7-9-y-old French children: Frequency of obesity, overweight and thinness. International Journal of Obesity 26(12), 1610-1616.

Rössner, S., 2002. Obesity: The disease of the twenty-first century. International Journal of Obesity 26(4), 2-4.

Sanchez-Johansen L. A. P. , Fitzgibbon, M. L., Martinovich, Z., Stolley, M. R., Dyer, A. R., Van Horn, L., 2004. Ethnic differences in correlates of obesity between Latin-American and Black women. Obesity Research 12, 652–660.

Seidell, J. C., 1995. Obesity in Europe: Scaling an epidemic. International Journal of Obesity Related Metabolism Disorders 19, S1-4.

Smith, J., 2004. Unravelling the SES-health connection. In: J. Linda (Eds.). Aging, Health, and the Public Policy: Demographic and Economic Perspectives 30 (Supplement to Population and Development Studies), 108-32.

Smith, J., 2009. Re-constructing childhood health histories. Demography 46(2), 387-403.

Smith, J.P., Kington, R., 1997. Demographic and economic correlates of health in old age. Demography 34(1), 159-170.

Sobal, J., Stunkard, A. J., 1989. Socioeconomic status and obesity: A review of the literature. Psychological Bulletin 105(2), 260-275.

Stewart, M.B., 2007. The interrelated dynamics of unemployment and low-wage employment. Journal of Applied Econometrics 22(3), 511-531.

Strauss, R. S., Knight, J., 1998. Influence of the home environment on the development of obesity in children. Pediatrics 103(6), e85.

Stunkard, A. J., Sorensen, T. I. A., Hanis, C., Teasdale, T. W., Chakraborty, R., Schull, W. J., Schulsinger, F., 1986. An adoption study of human obesity. New England Journal of Medicine 314, 193-198.

Stunkard, A. J., Sorensen, T. I. A., 1993. Obesity and socioeconomic status - A complex relation. New England Journal of Medicine 329, 1036-1037.

Thompson, D., Edelsberg, J., Colditz, G. A. Bird, A. P., Oster, G., 1999. lifetime health and economic consequences of obesity, Archives of Internal Medicine 159, 2177-2183.

Ulijaszek, S. J., 2007. Frameworks of population obesity and the use of cultural consensus modelling in the study of environments contributing to obesity. Economics and Human Biology 5, 443-457.

Ulijaszek, S. J., Koziel, S., 2007. Nutrition transition and dietary energy availability in Eastern Europe after the collapse of communism. Economics and Human Biology 5, 359-369.

van Zon, A., Muysken, J., 2001. Health and economic growth. Journal of Health Economics 20, 169-185.

Villar, J.G., Quintana-Domeque, C., 2009. Income and body mass index in Europe. Economics and Human Biology 7(1), 73-83.

Visschell, T. R., Seidell, J. C., 2001. The public health impact of obesity. Annual Review of Public Health. 22, 355-375.

Wang, Y., 2001. Cross-national comparison of childhood obesity: The epidemic and the relationship between obesity and socioeconomic status. International Journal of Epidemiology 30(5), 1129-1136.

Wang, L. Y., Yang, Q., Lowry, R., Wechsler, H., 2003. Economic analysis of a school-based obesity prevention program. Obesity Research 11, 1313-1324.

Wardle, J., Sanderson, S., Guthrie, C. A., Rapoport, L. and Plomin, R., 2002. Parental feeding style and the inter-generational transmission of obesity risk. Obesity Research 10(6), 453-62.

Whitaker, R. C., Wright, J. A., Pepe, M. S., Seidel, K. D., Dietz, W. H., 1997. Predicting obesity in young adulthood from childhood and parental obesity. The New England Journal of Medicine 337, 869-873.

Wooldridge, J.M., 2005. Simple solutions to the initial conditions problem in dynamic, nonlinear panel data models with unobserved heterogeneity. Journal of Applied Econometrics 20(1), 39-54.

Wronka, I., Pawlinska, R., 2007. Childcare, height and BMI among female Polish university students, 2005. Economics and Human Biology 5, 435–442.

Yancy Jr., W. S., Olsen, M. K., Westman, E. C., Bosworth, H. B., Edelman D., 2002. Relationship between obesity and health-related quality of life in men. Obesity Research 10, 1057–1064.

Zellner, K., G. Ulbricht, Kromeyer-Hauschild, K., 2007. Long-term trends in body mass index of children in Jena, Eastern Germany. Economics and Human Biology 5, 426-434.

Zhang, Q., Wang, Y., 2004. Trends in the association between obesity and socioeconomic status in U.S. adults: 1971 to 2000. Obesity Research 12(10), 1622-1632.

Table 1. Obesity, smoking and physical exercise at different ages in six European countries (%)

	Denmark	Finland	France	Greece	Netherlands	United Kingdom
Obese at 25	3.3	2.8	1.1	10.3	3.1	8.3
Obese at 35	6.9	8.7	4.0	11.4	6.7	14.0
Obese at 45	12.1	16.1	10.9	14.5	13.0	20.1
Obese now	19.5	22.2	21.2	22.8	25.8	22.1
Males	15.4	21.0	21.4	20.1	24.3	20.6
Females	22.4	24.1	20.9	25.0	27.1	25.0
Smoking at 25	62.6	51.1	62.8	24.3	68.8	94.1
Smoking at 35	59.7	40.7	70.3	50.2	60.2	81.3
Smoking at 45	50.1	28.2	57.6	48.4	58.4	76.0
Smoking now	32.9	19.0	51.2	41.8	29.2	52.6
Exercising at 25	62.5	78.8	62.3	29.8	72.4	68.0
Exercising at 35	60.9	81.2	60.9	26.2	70.6	64.4
Exercising at 45	56.7	82.7	53.7	20.3	67.5	55.9
Exercising now	53.0	73.3	42.4	19.1	64.9	45.2

Reading: 19.5% of Danish respondents were obese at the time of interview and 12.1% of Danish respondents were obese when they were in their forties. 15.4% (22.4%) of Danish male (female) respondents were obese at the time of interview.

Table 2. Obesity dynamics of the currently obese (%)

	Obese now (%)	Of these, obese at age 45 (%)	Of these, obese at age 35 (%)	Of these, overweight at age 45 (%)	Of these, overweight at age 35 (%)
All countries	22.4	43.2	31.3	44.7	40.2
Denmark	19.5	47.1	26.1	42.0	46.4
Finland	22.2	65.2	31.5	29.3	47.8
France	21.2	41.1	14.8	45.5	47.8
Greece	22.8	46.2	21.3	43.6	44.9
Netherlands	25.8	39.0	18.9	49.4	51.0
UK	22.1	47.0	18.3	37.5	61.2

Reading: 19.5% of Danish respondents were obese at the time of interview and, among them, 47.1% were already obese when they were in their forties.

Table 3. Obesity and socioeconomic status (%)

	Obese (BMI > 30) at 25			Obese (BMI > 30) now			Severely obese (BMI > 40) now		
	Males	Females	All	Males	Females	All	Males	Females	All
Mother's education									
Low	5.2	5.6	5.4	21.1	25.1	23.1	1.6	2.6	2.1
Medium	4.9	5.5	5.2	19.0	21.9	20.5	1.2	1.4	1.3
High	4.0	5.5	4.7	17.9	18.4	18.1	0.9	1.3	1.1
Own education									
Low	7.6	9.3	8.5	23.0	29.0	25.9	1.7	2.5	2.1
Medium	6.5	6.3	6.4	22.8	23.4	23.1	1.9	2.1	2.0
High	3.6	4.0	3.8	17.5	20.5	19.2	1.0	2.1	1.6

Reading: Among the respondents with low educated mothers, 5.4% were obese when they were in their twenties. Among male (female) respondents with low educated mothers, 5.2% (5.6%) were obese when they were in their twenties.

Table 4. Persistence of exercising and smoking (%)

Exercises regularly			Of these, proportion of individuals who also exercised regularly at age :								
Now			25			35			45		
Males	Females	All	Males	Females	All	Males	Females	All	Males	Females	All
49.0	45.6	47.2	79.0	71.3	75.2	78.3	75.5	76.9	83.7	82.5	83.1

Smokes regularly			Of these, proportion of individuals who also smoked regularly at age :								
Now			25			35			45		
Males	Females	All	Males	Females	All	Males	Females	All	Males	Females	All
41.7	41.4	41.5	82.7	76.8	79.7	96.3	94.0	95.1	96.5	95.1	95.8

Reading: 41.7% of male respondents smoked regularly at the time of interview. Of these, 82.7% (96.5%) also smoked when they were in their twenties (forties).

Table 5. Obesity rates in SOCIOLD and from Michaud et al. (2007).

Countries	Females			Males			Total		
	Sociold	SHARE		Sociold	SHARE		Sociold	SHARE	
	Self-reported		Corrected	Self-reported		Corrected	Self-reported		Corrected
Denmark	22.4	13.1	18.2	15.4	14.2	17.5	19.5	13.6	17.9
France	20.9	15.1	20.3	21.4	15.0	16.2	21.2	15.0	18.5
Greece	25.0	22.3	31.2	20.1	16.9	19.2	22.8	19.8	25.6
Netherlands	27.1	16.5	23.2	24.3	13.1	15.3	25.8	14.9	19.5

*Sources: SOCIOLD and Appendix B.2 in Michaud et al. (2007).

Reading: While 22.4% of Danish female SOCIOLD respondents reported they were obese, only 13.1% of female Danish SHARE respondents did. Correction of the latter figure for self-reporting bias *à la* Michaud et al. (2007), suggests the obesity rate among Danish females is 18.2% .

Table 6a. Obesity determinants at different ages: Marginal effects from Probit estimations

	All				Females				Males			
	Mid-20s	Mid-30s	Mid-40s	50-65	Mid-20s	Mid-30s	Mid-40s	50-65	Mid-20s	Mid-30s	Mid-40s	50-65
Female	0.008	-0.003	-0.003	**0.030**								
Medium maternal education	-0.004	-0.011	-0.007	0.001	0.009	0.006	0.010	-0.001	**-0.015**	**-0.029**	-0.024	0.003
High maternal education	*-0.011*	*-0.025*	-0.013	-0.016	-0.002	*-0.026*	-0.006	*-0.053*	**-0.016**	-0.024	-0.018	0.021
Nb of persons per room at 14	-0.001	*0.011*	*0.010*	0.013	0.001	**0.012**	0.012	0.017	-0.002	0.006	0.010	0.009
Respondent: low education	0.010	0.013	0.018	*0.035*	0.016	*0.024*	*0.032*	*0.048*	0.004	0.001	0.000	0.019
Parents' disease: diabetes	**0.035**	0.014	**0.047**	*0.043*	**0.035**	*0.029*	**0.078**	**0.086**	**0.033**	-0.007	0.009	-0.007
Parents' disease: high blood pressure	0.013	*0.018*	*0.021*	*0.036*	**0.025**	*0.023*	**0.034**	**0.049**	-0.001	0.013	0.009	0.026
Regular physical activity	-0.009	**-0.04**	**-0.08**	**-0.12**	-0.015	**-0.05**	**-0.08**	**-0.13**	-0.006	**-0.04**	**-0.06**	**-0.11**
Smoker	-0.001	*-0.016*	*-0.024*	**-0.072**	-0.013	-0.014	**-0.035**	**-0.09**	0.007	-0.021	-0.016	**-0.06**
Denmark	**0.070**	*0.046*	0.014	-0.016	**0.086**	**0.075**	*0.048*	0.025	0.059	0.016	-0.021	**-0.062**
Finland	*0.063*	**0.077**	**0.067**	0.026	*0.090*	**0.115**	0.061	0.052	0.055	0.050	**0.062**	0.003
Greece	**0.169**	**0.098**	0.016	-0.012	**0.146**	**0.104**	0.025	0.005	**0.194**	**0.085**	0.006	-0.032
Netherlands	*0.048*	*0.037*	0.015	*0.045*	*0.042*	*0.042*	0.024	*0.059*	**0.064**	0.037	0.010	0.034
United Kingdom	**0.322**	**0.227**	**0.141**	**0.159**	**0.334**	**0.250**	**0.209**	**0.239**	**0.321**	**0.205**	**0.093**	**0.091**
Nb of obs.	4,595	4,595	4,595	4,595	2,373	2,373	2,373	2,373	2,222	2,222	2,222	2,222

Source: Authors' own calculations. Marginal effects estimates in *italics* and as **bold** are significant at the ten and five per cent levels, respectively.

Table 6b. Determinants of growth rate in weight at different ages: Heteroskedasticity corrected estimations

	All			Females			Males		
	20s to 30s	30s to 40s	40s to 50s	20s to 30s	30s to 40s	40s to 50s	20s to 30s	30s to 40s	40s to 50s
Female	0.000	**0.009**	**0.0171**						
Medium maternal education	-0.005	-0.002	0.002	-0.006	-0.006	-0.002	**-0.003**	0.002	0.004
High maternal education	-0.001	-0.003	**0.012**	**-0.015**	-0.003	0.010	**0.013**	-0.003	0.012
Number of persons per room at 14	**0.004**	0.001	0.003	0.003	0.000	0.004	0.007	0.004	0.001
Respondent: low education	0.002	**-0.008**	**0.018**	0.000	-0.008	**0.013**	0.004	-0.007	0.023
Parents' disease: diabetes	0.002	**0.007**	-0.006	0.006	0.009	-0.001	-0.004	0.004	**-0.011**
Parents' disease: high blood pressure	**0.008**	0.003	0.000	**0.011**	0.004	-0.001	0.004	0.001	-0.001
Regular physical activity	-0.005	**-0.012**	**-0.014**	-0.003	**-0.009**	**-0.016**	-0.007	**-0.015**	**-0.014**
Smoker	-0.001	**-0.008**	**-0.014**	0.003	**-0.011**	**-0.014**	-0.007	**-0.007**	**-0.014**
Denmark	-0.008	**-0.021**	**-0.01**5	0.001	**-0.03**1	**-0.041**	**-0.020**	-0.008	**0.019**
Finland	0.009	-0.004	**-0.030**	**0.028**	-0.010	**-0.039**	-0.007	0.001	**-0.019**
Greece	**-0.015**	**-0.024**	**-0.025**	-0.005	**-0.03**2	**-0.032**	**-0.027**	**-0.013**	**-0.017**
Netherlands	-0.001	**-0.011**	**-0.010**	0.006	**-0.016**	**-0.017**	-0.011	-0.006	-0.002
United Kingdom	-0.007	0.004	**0.013**	-0.008	**0.020**	0.004	-0.010	-0.006	**0.024**
Number of observations	4,595	4,595	4,595	2,373	2,373	2,373	2,222	2,222	2,222

Source: Authors' own calculations. Estimates in *italics* and as **bold** are significant at the ten and five per cent levels, respectively.

Table 7a. Obesity determinants: Marginal effects estimated from models with dynamics

	All			Females			Males		
	Pooled Probit	Random effects Probit	Wooldridge's dynamic probit	Pooled Probit	Random effects Probit	Wooldridge's dynamic probit	Pooled probit	Random effects Probit	Wooldridge's dynamic probit
Obese (*t* - 1)	**1.448**	**1.415**	**0.965**	**1.534**	**1.505**	**0.987**	**1.370**	**1.330**	**0.961**
Female	0.006	0.007	0.008						
Medium maternal education	-0.028	-0.028	-0.021	0.019	0.020	-0.010	-0.080	-0.083	-0.030
High maternal education	**-0.107**	**-0.111**	-0.082	-0.077	-0.083	-0.093	*-0.134*	*-0.136*	-0.066
Number of persons per room at 14	**0.045**	**0.046**	**0.049**	**0.048**	**0.050**	**0.057**	0.040	0.041	0.045
Respondent: low education	*0.072*	*0.076*	*0.072*	**0.134**	**0.140**	**0.134**	0.014	0.015	0.002
Parents' disease: diabetes	**0.122**	**0.127**	0.067	**0.212**	**0.220**	**0.181**	0.015	0.014	-0.069
Parents' disease: high blood pressure	**0.069**	**0.073**	0.059	**0.130**	**0.136**	**0.103**	0.008	0.011	0.027
Regular physical activity	**-0.291**	**-0.299**	**-0.207**	**-0.331**	**-0.339**	**-0.161**	**-0.263**	**-0.272**	**-0.282**
Smoker	**-0.156**	**-0.162**	**-0.289**	**-0.184**	**-0.190**	**-0.229**	**-0.160**	**-0.166**	**-0.401**
Denmark	0.071	0.074	0.025	**0.173**	**0.179**	0.105	-0.054	-0.057	-0.055
Finland	**0.174**	**0.179**	**0.184**	**0.214**	**0.219**	0.160	**0.136**	**0.140**	**0.193**
Greece	**0.162**	**0.169**	-0.045	**0.149**	**0.156**	-0.087	**0.170**	**0.176**	-0.006
Netherlands	**0.123**	**0.127**	**0.101**	**0.149**	**0.153**	0.127	0.108	0.112	0.093
United Kingdom	**0.523**	**0.543**	**0.258**	**0.653**	**0.677**	**0.403**	**0.424**	**0.441**	*0.155*
Average Partial Effect for Obese (*t*–1)	0.567	0.553	0.375	0.599	0.588	0.384	0.535	0.519	0.374
Number of obs.	13,679	13,679	13,679	7,060	7,060	7,060	6,619	6,619	6,619
Log Likelihood	-4079.38	-4079.38	-3736.48	-2061.61	-2061.61	-1872.39	-1996.04	-1996.04	-1836.32

Source: Authors' own calculations. Marginal effects estimates in *italics* and as **bold** are significant at the ten and five per cent levels, respectively.

Table 7b. Determinants of the growth rate in weight: Models with dynamics corrected for heteroskedasticity (FGLS and IV) and for measurement error in weight (IV)

	All		Females		Males	
	FGLS	IV	FGLS	IV	FGLS	IV
Δln(weight($t-1$))	**0.475**	**0.605**	**0.466**	**0.597**	**0.492**	**0.625**
Female	**-0.001**	0.002				
Medium maternal education	**-0.0003**	-0.0001	-0.0003	0.0003	-0.0004	-0.0001
High maternal education	-0.001	0.001	-0.002	-0.001	0.000	0.002
Respondent: low education	***-0.001***	***0.001***	-0.001	0.001	-0.001	0.001
Parents' disease: diabetes	0.000	**-0.001**	0.000	-0.001	0.000	-0.001
Parents' disease: high blood pressure	0.001	0.000	0.001	0.001	0.000	0.000
Regular physical activity a decade earlier	**-0.001**	-0.000	-0.001	-0.001	-0.001	-0.000
Smoker a decade earlier	**-0.001**	-0.000	-0.000	0.001	**-0.001**	-0.001
Denmark	0.000	**0.003**	**0.002**	**0.005**	**-0.003**	-0.001
Finland	**0.002**	**0.001**	**0.004**	**0.004**	0.000	-0.001
Greece	0.000	**0.002**	0.001	**0.004**	-0.001	-0.001
Netherlands	0.001	**0.002**	0.001	**0.003**	-0.001	0.000
United Kingdom	-0.001	**-0.001**	-0.000	-0.003	-0.002	0.001
Number of observations	9,084	4,489	4,687	2,314	4,397	2,175

Source: Authors' own calculations. Estimates in *italics* and as **bold** are significant at the ten and five per cent levels, respectively.

Table 8. Marginal effects from other dynamic specifications (with neither heteroskedasticity nor measurement bias corrections)

	Overweight determinants at different ages Probit estimations				Dynamic obesity models with control for previous obesity/overweight statuses			Dynamic overweight models		
	Mid-20s	Mid-30s	Mid-40s	50-65	Pooled Probit	Random effects Probit	Wooldridge's dynamic probit	Pooled probit	Random effects probit	Wooldridge's dynamic probit
Obese ($t-1$)								**1.468**	**1.444**	**0.799**
Overweight ($t-1$)					**0.982**	**0.968**	**0.944**	**0.534**	**0.553**	**0.760**
Female	**-0.050**	**-0.097**	**-0.117**	**-0.125**	**-0.252**	**-0.257**	**-0.252**	*0.061*	*0.064*	**0.092**
Medium maternal education	**-0.024**	-0.013	**-0.035**	**-0.042**	**-0.072**	**-0.075**	**-0.077**	-0.018	-0.018	0.001
High maternal education	-0.005	-0.009	*-0.045*	-0.032	*-0.070*	*-0.075*	**-0.084**	**-0.129**	**-0.133**	-0.106
Number of persons per room at 14	0.001	0.003	-0.006	-0.003	-0.006	-0.006	-0.006	**0.048**	**0.050**	**0.066**
Respondent: low education	0.013	0.012	-0.003	0.020	0.020	0.023	0.031	0.068	0.072	0.088
Parents' disease: diabetes	**0.035**	**0.058**	0.030	0.019	**0.064**	**0.065**	**0.083**	*0.082*	*0.087*	0.035
Parents' disease: high blood pressure	0.010	0.018	**0.043**	*0.028*	**0.065**	**0.069**	**0.078**	0.054	0.057	0.061
Regular physical activity	**-0.020**	-0.012	**-0.049**	*0.031*	-0.003	-0.004	0.007	**-0.315**	**-0.325**	**-0.256**
Smoker	0.016	0.019	**-0.032**	-0.019	-0.016	-0.019	0.046	**-0.147**	**-0.154**	**-0.320**
Denmark	**0.061**	0.026	0.027	0.041	0.063	0.065	0.063	0.036	0.038	-0.009
Finland	**0.052**	**0.066**	*0.057*	0.008	0.078	0.072	0.076	**0.168**	**0.172**	0.191
Greece	0.016	0.037	**0.068**	**0.063**	**0.129**	**0.133**	**0.178**	0.030	0.037	**-0.177**
Netherlands	**0.052**	**0.070**	**0.093**	0.018	**0.109**	**0.114**	**0.105**	0.085	0.089	0.077
United Kingdom	**-0.035**	**0.076**	0.035	0.034	**0.123**	**0.123**	**0.199**	**0.440**	**0.464**	**0.227**
Average Partial Effect, obese ($t-1$)								0.574	0.564	0.315
Average Partial Effect, overweight ($t-1$)					0.382	0.377	0.367	0.227	0.300	0.233
Log Likelihood					-7716.7	-7716.7	-7654.08	-3407.5	-3406.9	-3184.32
Number of observations	4,595	4,595	4,595	4,595	13,679	13,679	13,679	10,222	10,222	10,222

Source: Authors' own calculations. Marginal effects estimates in *italics* and as **bold** are significant at the ten and five per cent levels, respectively.