

WORKING PAPER 11-07



WP

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ISBN 9788778825346 (print)

ISBN 9788778825353 (online)

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Abstract

This paper is concerned with how obesity and some of its determinants develop over individuals' life cycles. In particular we examine empirically the role and relative importance of early life conditions (parents' education and socioeconomic status) and individuals' own education as adults and how their impacts on the probability of overweight and obesity evolves over the life cycle. As the data set includes information about the individuals' health behaviours (smoking and physical exercise) at various ages we can also examine the impact of these at different stages of the persons' life cycle.

The data used in the empirical analysis is from a common detailed questionnaire study carried out in six different European countries (Denmark, Finland, France, Greece, the Netherlands and the U.K.) and which was answered by about 6,000 individuals aged 50 to 65 at the time of the survey. Obesity indicators are constructed from information collected in the survey regarding individuals' height and weight at different ages (25, 25, 45 and current age). We perform two types of econometric analyses on data for all countries: a "repeated cross-sections" analysis where each cross-section refers to the individual's situation at a certain age and a random effects dynamic probit analysis of the individuals' obesity histories.

Key findings are: (i) controlling for parental and childhood factors, health behaviour and socioeconomic status affect country differences in overweight and obesity only marginally, (ii) parents' socioeconomic status predicts obesity in early adulthood whereas individuals' own socioeconomic status as adults is more important in explaining obesity at later stages of the life cycle, and (iii) changes in obesity status are associated with changes in 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 have also benefited from comments from participants at the IHEA congress in Beijing 2009 on an earlier version of this paper.

1 Introduction

The growing prevalence of overweight and obesity is increasingly becoming a major concern for policy makers. Explaining the development is also a challenge for research.¹ Part of the growing body of literature focuses on the societal and economic consequences of obesity, underlining the most common obesity co-morbidities and hence its health effects.² These in turn result in a lower health-related quality of life (see e.g. 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). The obesity epidemic also induces huge economic costs to society. So far, researchers have focused on the costs of obesity-related health expenditures. For instance, Wolf (1998) estimated the economic cost of obesity to represent 6% of health expenditures in the US.³ But there are other economic costs as well. Thus, Finkelstein *et al.* (2005a) estimated that for the United States approximately 30% of the total costs of obesity are due to increased absenteeism among full-time employees. 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.⁴

Thus, obesity is associated with social and economic costs and because the majority of these costs are financed by taxpayers, there is a clear motivation for governments to combat the obesity epidemic. However, this requires that the determinants of obesity can be clearly identified. So far, the research literature has explored many of these, such as genetic factors (e. g. Stunkard *et al.*, 1986), childhood and parental obesity as predictors of adulthood obesity (Whitaker *et al.*, 1997),

¹ 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). For a European perspective, see Seidell (1995). 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.

² The most commonly invoked obesity-related diseases are hypertension, dyslipidemia, type II diabetes and coronary heart diseases. See for instance Thomson *et al.* (1999) or Levy *et al.* (1995).

³ Similar figures are reported for other countries and other periods. For US studies, see for instance Colditz (1992), Wolf and Colditz (1998), Colditz (1999), Finkelstein *et al.* (2004), Finkelstein *et al.* (2005a), Finkelstein *et al.* (2005b),. For a French study, see Levy *et al.* (1995). For Canada, see Birmingham *et al.* (1999).

⁴ 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).

inter-generational transmission either via physical activity patterns (e.g. Fogelholm *et al.*, 1999) or parental feeding styles (Wardle *et al.*, 2002).⁵ The two determinants that have received most attention, in particular by economists⁶, are individuals' socio-economic environments (e.g. 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 (e. g. Prentice and Jebb, 1995 and Drewnowski and Specter, 2004).

The standard economic approach to obesity is to think of it as the consequence of technological improvements which on one hand has lowered costs of food consumption (mass preparation of food, increased availability of fast-food and calorie dense-food, 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). The impact of these changes are frequently said to have been strengthened by increased female labour force participation and urbanisation.

Interestingly, this view is shared by epidemiologists as well. 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 aetiology of obesity and possibly represent the dominant factor. Even more interesting is the way the energy-needs/energy-intake balance relates to socio-economic status (henceforth: SES). Indeed, one might reasonably argue that poor socio-economic individuals are not only less likely to have access to physical activity facilities but also more likely to resort to low quality/fat-dense food. For instance, Gordon-Larsen *et al.* (2006) provide evidence that lower-SES groups have reduced access to facilities, which in turn was associated with decreased physical activity and increased overweight. Likewise, Drewnowski and Specter (2004) demonstrate that there is an inverse relation between energy density and energy cost, such that energy-dense foods composed of refined grains, added sugars, or fats may represent the lowest-cost option to the consumer. They also show that poverty

⁵ This list is far from exhaustive. For instance, 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.

⁶ For an overview of the economists' approach to obesity, see for instance, Acs and Lyle (2007) and Grossman and Mocan (2009).

and food insecurity are associated with lower food expenditures, low fruit and vegetable consumption, and lower-quality diets.

However, the observed trends in obesity suggest that public health policies recommending avoidance of overweight and the common practice of dieting fail to prevent an increase in the prevalence of obesity (Seidell, 1995). Perhaps this is due to the curative dimension having been given more attention than prevention *per se*. Yet, a number of authors have insisted on the idea that prevention programs are more efficient in combating obesity than weight-loss programs (see e. g. 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 one's life cycle. 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 less pronounced in the research on overweight and obesity.⁷ This likely reflects the dearth of longitudinal data that would allow researchers to shed light on the development of obesity at the individual level and its determinants. Thus, most previous studies have been based on cross-sectional data and these results of the analyses have been interpreted as reflecting steady-state or cumulative outcomes. The same holds for economic analyses as relatively few ones have been concerned with the relative contributions of the factors influencing obesity growth; see Bleich *et al.* (2008) for a notable exception.

Another distinguishing feature of the literature is that 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, it rather seldom explores this dimension. 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 socioeconomic development levels. Different socio-economic groups are at different risks, and the relationship between obesity and socio-economic status varies across countries.⁸ Except for Michaud *et al.* (2007) and Brunello *et al.* (2009), we know of no other studies using comparable nationally representative micro-data to analyse cross-country variation in obesity patterns.

⁷ 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).

⁸ Of course, more remarkable is the difference between countries with different development levels. For instance, 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.

The aim of this paper is twofold. First, we examine empirically the role and relative importance of a variety of factors known to have an influence on the probability of overweight and obesity as well as how these factors' impacts on the probability of overweight and obesity evolve over the life cycle. The fairly rich 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. Second, we examine to what extent these same factors can explain cross-country differences in obesity. To be more specific, we estimate these differences across six EU countries and examine how sensitive are those estimates to differences in the determinants of obesity.

The paper is organised as follows. Section 2 briefly discusses analyses that are comparable to the current study. Section 3 describes the data as well as the general patterns they highlight. Section 4 presents the empirical strategy and discusses the results. Section 5 concludes.

2. Previous research

In the following we briefly discuss studies that are most relevant for comparisons with the current study. As mentioned above, 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 SHARE project, the aim of which is to build a European data set corresponding to the HRS in the United States. The sample analysed includes individuals aged 50 or above and the focus is on cross-country differences.

The authors document significant differences in the SES-obesity gradient across countries. A large portion of the cross-country variation can according to the study be attributed to differences in physical activity. Another prime suspect, the expenditure spent on food consumed outside home, turned out not to contribute to the explanation of country differences in obesity. Unlike for the U.S., where the amount of food eaten outside home is positively correlated with obesity, the results for European countries point to a negative relationship, which is interpreted as reflecting US-Europe differences in the quality of food eaten away from home. Notably, a considerable part of the cross-country differences in obesity remains after differences in physical activity and patterns of food intake are accounted for. This indicates that SES differences could be due to other factors such as

family background and early childhood/adolescence events which the authors do not control for. Also, as the SHARE data used is from a cross-section, the authors could not adopt a dynamic empirical approach and have had to rely on a steady-state or cumulative interpretation of the estimated energy-accounting equations.

Baum and Ruhm's (2009) study is closer to ours.⁹ They make use of panel data from the National Longitudinal Survey of Youth (NLSY) during the period 1981-2004. Owing to the specific character of the data source 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.

Baum and Ruhm (2009) also find a clear SES-obesity gradient for both genders. Parental socioeconomic status is measured by mother's level of education. When both maternal and the individual's own education are included as regressors, both effects differ significantly from zero. Adding family income, marital status, number of children and health behaviour variables does not affect the estimated impacts of own and mother's education, hence 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 (see e. g., Cutler *et al.*, 2008), the authors find that the SES-obesity gradient widens with age.

A common feature of Baum and Ruhm's (2009) study and ours is the dynamic nature of the examined relationships. However, in contrast to them, we focus on individuals aged 50 and above 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.

3 Data description

The data used in our empirical analysis is constructed within an EU project called SOCIOLD within the framework of which a common detailed internet-based¹⁰ questionnaire in six different European countries (Denmark, Finland, France, Greece, the Netherlands and the U.K.) was carried out. The

⁹ Brunello *et al.* (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 highly unrepresentative.

¹⁰ Due to the low IT penetration in Greece, the questionnaire was carried out as a standard mail questionnaire in that country.

questionnaire was answered by about 6,000 individuals aged 50 to 65 at the time of the survey (in 2004). Besides a host of variables describing individual traits¹¹ 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 BMI measures at different ages – 25, 35, 45 and current age (in the age range 50 to 65). In section 4 below, we correlate our measures of overweight ($25 < \text{BMI} < 30$) and obesity ($\text{BMI} > 30$) with their determinants.

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 NHANES (National Health and Nutrition Examination Study) 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 (e.g., age). 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 HRS (Health and Retirement Study) and SHARE 2004 and conclude that the applied correction has a large impact on obesity rates for the population aged 50 and above. *Table 1* 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).

Table 1 shows that under-reporting (over-reporting) of weight (height) is clearly 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.¹²

¹¹ In addition to conventional variables like age, gender, marital status, education, employment status, occupation, and income, the questionnaire also asks the respondents about their psychological and mental health, social networks and support.

¹² 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.

Table 1. Obesity rates in SOCIOLD and from Michaud *et al.* (2007).¹³

Countries	Females			Males			Total		
	Sociold	SHARE		Sociold	SHARE		Sociold	SHARE	
	Self-reported	Correc.	Self-reported	Correc.	Self-reported	Correc.			
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

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. A presumably major concern is that the information available to us is of retrospective nature. Again, we know of no evidence that there would be systematic changes or biases in how individuals perceive their height and weight over their lives. Our assumption is 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.

Table 2 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 3, the onset of overweight as well as obesity problems occur relatively early: of the currently obese about ninety (seventy) per cent already were obese or overweight at age 45 (35). The cross-country

¹³ See Appendix B, Table B.2 in Michaud *et al.* (2007).

variation in *Table 2* corresponds to what has been documented in previous studies; see e.g., 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 U.S. the levels are considerably lower; see e. g. Michaud *et al.* (2007) for a transatlantic comparison.

Returning to *Table 2*, 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.

Table 2. 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

Table 3. 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

Table 4. Obesity and socioeconomic status (%)

	Obese (BMI > 30) at 25	Obese (BMI > 30) now	Severely obese (BMI > 40) now
<i>Mother's education</i>			
Low	5.4	23.1	2.1
Medium	5.2	20.5	1.3
High	4.7	18.1	1.1
<i>Own education</i>			
Low	8.5	25.9	2.1
Medium	6.4	23.1	2.0
High	3.8	19.2	1.6

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 (see e.g. Eriksson *et al.*, 2001, Wardle *et al.*, 2002). From Table 4 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 US 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.

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.¹⁴

As can be seen from *Table 5*, 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 sample who are currently smoking were already smokers 10-20 years earlier.

Table 5. Persistence of exercising and smoking (%)

Exercises regularly	Of these, proportion of individuals who also exercised regularly at age :		
Now	25	35	45
54.7	75.2	76.9	83.1
Smokes regularly	Of these, proportion of individuals who also smoked regularly at age :		
Now	25	35	45
53.1	79.7	95.1	95.8

4. Empirical analysis

The standard economic approach to identifying the determinants of obesity is the health production function (see e.g., Lakdawalla and Philipson, 2009, Lakdawalla *et al.*, 2005). We therefore think of the body mass index as the outcome of the combination of different inputs. However, as we have information about individuals' body mass index, the output, and health style behaviours at different

¹⁴ 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.* (2009). 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.

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 for their current age (between 50 and 65). We believe this approach usefully sheds light on the way the weight production process evolves over one's life cycle.

But we also exploit the longitudinal nature of the data and estimate random effects dynamic models of the individuals' obesity histories. The aim of the dynamic analyses is to assess the extent to which obesity at a certain age relates to obesity at younger ages.

4.1. Repeated cross-section estimates

Beginning with the repeated cross-sections, these are simple health production models where we think of the body mass index as the health outcome of choices made during four different stages of the individual's life cycle:

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

Mother's SES is expected to be negatively related to the probability of being obese. Several explanations have been offered for this relation; see Cutler *et al.* (2008) for a comprehensive discussion of the SES-health relationship. 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 more well 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 affect an individual's body mass index 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.

As can be seen from (1), in addition to the SES variables we also include a number of other variables the motivation for and explanation of will be provided below in connection with the discussion of the estimation results.

According to the probit estimates displayed in *Table 6*, there is a SES(mother)-obesity gradient when the individuals are in their mid-twenties and -thirties.¹⁵ As in Baum and Ruhm's (2009) study, the disparity is increasing in age. However, as from when the respondents are in their forties, 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 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. In the table we may see that a variable describing the number of persons per room at age 14 has a positive and significant effect when the individuals are in their thirties and forties. The respondent's own level of education affects the likelihood of obesity negatively.¹⁶ The coefficient is increasing in age but differs significantly from zero only when the individual is in her fifties or early sixties. Thus, as the individual ages, the influence of her parents' socioeconomic background dissipates, while the importance of her own socioeconomic status increases.¹⁷ This implies that intergenerational socioeconomic mobility has an impact on obesity which appears later in adulthood.

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 and obesity and are 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 of about the same magnitude as for mother's and respondent's own education. Moreover, the marginal effects of the two parental illnesses are considerably larger when the respondents are in their forties and fifties.

¹⁵ 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.

¹⁶ 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.

¹⁷ Note that we do not have information about changes in the respondent's SES over her life cycle.

Turning next to the association between the respondent's health related behaviours and her probability of obesity, it should be noted that variables for the questions regarding smoking and physical exercise are like the BMI constructed from answers to retrospective questions. 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)".¹⁸

As can be seen from *Table 6*, regular physical activities greatly reduce the likelihood of obesity.¹⁹ The marginal effect increases strongly with age. As in other studies, we also find that smoking lowers the probability of obesity and that the corresponding marginal effects are quite sizable.

A final observation worth making from the results in *Table 6* is that the country effects are relatively large in the estimations for the mid-twenties 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 6*. 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.

¹⁸ The questionnaire also included questions regarding the respondents' food and alcohol consumption. This information is only available for the current period, however.

¹⁹ As emphasized by McInnes and Shinogle (2009), 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 (2006).

Table 6. Probit estimations of obesity determinants at different ages.

Variables	Age: mid-20s		Age: mid-30s		Age: mid-40s		Now (age 50-65)	
	Marg. Effect.	St dev	Marg. Effect	St dev	Marg. Effect	St dev	Marg. Effect	St dev
Female	0.008	0.006	-0.003	0.008	-0.003	0.010	0.030*	0.013
Medium maternal education	-0.004	0.007	-0.011	0.010	-0.007	0.013	0.001	0.016
High maternal education	-0.011*	0.007	-0.025*	0.011	-0.013	0.017	-0.016	0.021
Number of persons per room at 14 years	-0.001	0.004	0.011*	0.004	0.010	0.006	0.013	0.008
Respondent low education	0.010	0.008	0.013	0.011	0.018	0.014	0.035*	0.017
Parents' disease: diabetes	0.035**	0.010	0.014	0.011	0.047**	0.015	0.043*	0.017
Parents' disease: blood pressure	0.013	0.007	0.018*	0.009	0.021*	0.012	0.036*	0.014
Sport activity	-0.009	0.006	-0.044***	0.009	-0.069***	0.011	-0.123***	0.013
Smoker	-0.001	0.006	-0.016*	0.009	-0.024*	0.011	-0.072**	0.013
Denmark	0.070**	0.026	0.046*	0.020	0.014	0.019	-0.016	0.022
Finland	0.063*	0.030	0.077**	0.026	0.067**	0.026	0.026	0.028
Greece	0.169***	0.031	0.098***	0.021	0.016	0.018	-0.012	0.021
Netherlands	0.048*	0.020	0.037*	0.017	0.015	0.017	0.045*	0.022
UK	0.322***	0.047	0.227***	0.033	0.141***	0.028	0.159***	0.029
Number obs	4,595		4,595		4,595		4,595	

* $p < 0.10$; ** $p < 0.05$; *** $p < 0.01$

4.2. Dynamic model estimates

Next we report results from estimations where we have exploited the “longitudinal” character of the questionnaire data. 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 for the last one for which this is an approximation). Three different estimators for the model are presented in *Table 7*. The first estimates presented 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 allows (second column) 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.²⁰ Wooldridge (2005) proposed a Conditional Maximum Likelihood estimator that considers the distribution conditional on the initial period value (and exogenous variables)²¹.

The latent equation for the random effects dynamic probit model is specified as:

$$(2) \quad y_{it}^* = \gamma y_{it-1} + \mathbf{x}_{it} \beta + \alpha_i + u_{it}$$

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 dynamic models, we have to solve two problems: (i) the treatment of initial conditions (y_{it-1}) and (ii) persistence and unobserved individual heterogeneity (u_i). Estimation of the model requires an assumption about the initial observations, y_{it} , 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_{i1} are correlated with the α_i , this estimator will overstate the extent of state dependence.

²⁰ 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.

²¹ This method has the advantage that it can be implemented with standard random effects probit (see Wooldridge, 2005).

Turning to the estimates in *Table 7*, we may begin with notice that early life conditions as measured by maternal education affect later obesity. In particular, we find that having a mother with a high educational level carries a negative and significant coefficient. This result is also obtained when we exclude the person's own educational level as an explanatory variable. The other variable describing economic circumstances during childhood, the extent to which the individual was living in overcrowded conditions, remains important and has a sizable positive impact on 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.

Notably, the person's own SES has an important impact on the likelihood of being obese. Similarly to the "repeated cross-section" estimations, having a parent who suffered from diabetes or high blood pressure strongly increases the probability of the offspring being obese. Likewise, regular physical exercise and smoking contribute to a significant reduction in the individual's risk of obesity.

As can be seen from *Table 7* (comparing columns 1 and 2²² with 3), the estimate of the lagged obesity variable, which was surely upward biased because of the unobserved heterogeneity, is reduced once the latter is accounted for. When, following Wooldridge (2005)²³, we estimate the average partial effect (APE) with respect to the lagged dependent variable we can see that the APEs of the pooled model probit and of the random effect 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 reduced considerably, but remains statistically significant. As was shown earlier in *Table 5*, there is strong persistency in physical exercise and smoking. Still, there is likely sufficient variation over time in these behaviours to influence the estimates.

²² 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.

²³ 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.

Table 7: Estimation results from models with dynamics

Variables	Pooled probit		Random effects probit		Wooldridge's dynamic probit	
	Coeff.	SE	Coeff.	SE	Coeff.	SE
Obese (t-1)	1.448***	0.037	1.415***	0.039	0.965***	0.045
Female	0.006	0.031	0.007	0.033	0.008	0.034
Medium maternal education	-0.028	0.055	-0.028	0.041	-0.021	0.043
High maternal education	-0.107**	0.054	-0.111**	0.055	-0.082	0.059
Number of persons per room at 14 years	0.045**	0.018	0.046**	0.019	0.049**	0.019
Respondent low education	0.072*	0.040	0.076*	0.042	0.072*	0.042
Parents' disease: diabetes	0.122***	0.040	0.127***	0.042	0.067	0.043
Parents' disease: blood pressure	0.069**	0.035	0.073**	0.036	0.059	0.037
Sport activity	-0.291***	0.033	-0.299***	0.034	-0.207***	0.052
Smoker	-0.156***	0.033	-0.162***	0.034	-0.289***	0.061
Denmark	0.071	0.057	0.074	0.059	0.025	0.059
Finland	0.174***	0.066	0.179***	0.068	0.184***	0.069
Greece	0.162***	0.054	0.169***	0.055	-0.045	0.058
Netherlands	0.123**	0.052	0.127**	0.054	0.101*	0.055
UK	0.523***	0.061	0.543***	0.063	0.258***	0.066
Average Partial Effect for Obese (t-1)	0.567		0.553		0.375	
Observations	13679		13679		13679	

*p<0.05; **p<0.01; ***p<0.001; time dummies are not reported

5. 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 have a significant influence on 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 resides in its focus on how the highlighted influences evolve over the life cycle. Thus, our results show that while parents' socio-economic status influences the probability that individuals will be obese when they are in their twenties or thirties, this effect dissipates at older ages, and is replaced by individuals' own socio-economic status playing the most important role. Likewise, not surprisingly, the effect of physical activity increases with age. 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.

Compared to cross-sectional or short panel analyses, our life cycle oriented study sheds more 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 mostly oriented towards youngsters. 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 is informative as to the policy implications of obesity-prevention programs. The fact that obesity today is strongly and positively correlated with obesity at a younger age 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 showed that although the part of the correlation between past experience in obesity and current obesity status is due to unobserved heterogeneity, most of the observed state-dependency reflects the effect of past obesity on individuals' behaviours towards preventing later obesity status.

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