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# Assessing the impact of obesity on labor market outcomes

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### ABSTRACT

We study the effect of obesity on employment, using rich data from the British National Child Development Study (NCDS). The results show a significant negative association between obesity and employment even after controlling for a rich set of demographic, socioeconomic, environmental and behavioral variables. In order to account for the endogeneity of obesity, we use and assess instruments introduced by Cawley (2004); the obesity status of biological relatives. Using parental obesity as an instrument, we show that the association between obesity and employment is no longer significant. Similar results are obtained in a model of first differences. We provide a number of different checks on the instruments, by exploiting the richness of the NCDS data. The results show mixed evidence regarding the validity of the instruments.

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## 1. Introduction

A growing literature focuses on the importance of non-traditional traits such as physical appearance, personality and birth order on labor market success (Hamermesh and Biddle, 1994; Mueller and Plug, 2006; Kantarevic and Mechoulan, 2006). The literature on physical appearance mainly considers three attributes: attractiveness, body mass and height. To date, the effect of body mass has received most attention, reflecting concerns about the sharply increasing obesity rates found in most Western countries (OECD, 2005, p. 87). Naturally, this raises the question about any adverse labor market consequences.

Obesity increases the risk of various health problems such as cancer, stroke, diabetes, asthma, hypertension, depression and arthritis (Abbott et al., 1994; Pi-Sunyer, 2002), which may affect the individual's capacity to work. There are, however, alternative explanations for the effect

of obesity on labor market outcomes. Employers may discriminate against obese workers (e.g. Hamermesh and Biddle, 1994; Baum and Ford, 2004; Rooth, 2009; Lundborg et al., 2010). Discrimination is not always clear as in some jobs, such as sales, physical appearance may be directly related to productivity. Furthermore, obesity may be related to non-desirable personality traits potentially affecting productivity. For instance, Puhl and Brownell (2001) and Sobal (2004) revealed public beliefs about obese people, they are thought to be lazier and less socially and intellectually skilled than their non-obese counterparts.

In this paper, we focus on the effect of obesity on employment. The previous literature has primarily looked at the effect of obesity on wages. The effects of obesity on employment are not necessarily the same as the effect on wages. If obese workers are less productive or if employers discriminate against obese individuals, they may receive lower wages but still be employed. However, if wages are not fully flexible or if obesity causes serious health problems, the effect of obesity on employment might be severe. In line with this, Rooth (2009) found strong indications of discrimination against obese workers by measuring employer callbacks on fictitious job applications to real jobs, where pictures of an obese or non-obese

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person were randomly assigned to similar applications. Focusing on wages alone may therefore leave out an important aspect of the effect of obesity on labor market outcomes. This may hold particularly for groups that have traditionally lower participation rates, such as females. The literature on the effect of obesity on employment is limited, however, and have reached mixed conclusions. In Cawley (2000a,b) and Norton and Han (2008), no effect of obesity on employment among US men and women is obtained. Morris (2007), on the other hand, finds a significant and negative effect for both genders in the UK.

Investigating the effect of obesity of labor market outcomes is complicated by potential reversed causality and endogeneity problems. Reversed causality may arise, for instance, because energy dense fattening food is relatively cheap, and lower wages or unemployment increases the demand for such food. Furthermore, there may be unobserved characteristics that vary systematically between obese and non-obese people, and these factors may also affect employment. For instance, people with high discount rates may be more prone to weight-gaining consumption. At the same time, high discount rates make investments in human capital (and thus future labor market outcomes) less attractive (Cawley, 2000a,b, 2004; Baum and Ford, 2004).

We contribute to the literature on the effect of obesity on labor market outcomes. For this purpose, we use data from the British National Child Development Study (NCDS), which is a longitudinal study on around 17,000 individuals born in Great Britain in the week of March 3–9, 1958, who are followed up to 2004, when they were 46 years old. The majority of studies on obesity and labor market outcomes is conducted in a US context and we thus contribute to the small literature examining the topic in a British context.

The NCDS has not often been used to study the effect of obesity on labor market outcomes. However, the data have a number of advantages. Since NCDS follows people from birth, it contains extensive information on early life conditions that could potentially affect both obesity and labor market outcomes. Even more importantly, it records the height and weight of both the respondent and the respondent's mother and father, allowing us to use the obesity status of a respondent's parents as instruments. The idea of using the obesity status of biological relatives as an instrument was introduced by Cawley (2000a,b), who argued that the strong association in body size between a parent and a child mainly reflects genetic factors, making it potentially useful as an instrument. The body mass index of biological relatives has since then been used as an instrument in a relatively large number of studies (Cawley, 2004; Burkhauser and Cawley, 2004; Cawley et al., 2005; Brunello and D'Hombres, 2007; Kline and Tobias, 2008; Atella et al., 2008; Shimokawa, 2008; Greve, 2008; Trogdon et al., 2008; Renna et al., 2008; Davey Smith et al., 2009).<sup>1</sup>

Using the obesity status of parents as an instrument, instead of that of siblings or children, has certain advantages. First, since parental obesity is available for all respondents, we do not have to rely on the obesity of a sibling or a child, which would restrict the sample to only those where information on a sibling or a child is present. Second, since we have two instruments we are able to perform overidentification tests on our instruments. We use these tests to assess the use of parental obesity as an instrument. It should be noted that the NCDS data has not been used before to estimate the causal effect of obesity by using a biological relative's weight as an instrument.

Using parental obesity status (or any biological relatives obesity status) as an identification strategy depends on the assumption that there are no other pathways than via the respondent's obesity status in which parental obesity affects the respondent's labor market outcomes. Alternative pathways may be present if genetic or non-genetic factors which affect obesity also have a direct impact on labor market outcomes. There is some evidence, based on twin and adoption studies, suggesting that the association in body weight between biological relatives is due to genetics, and that shared environmental factors play no role. However, this finding is not uncontroversial. We, therefore, exploit the richness of the NCDS data, and contribute to the literature by providing some checks for the appropriateness of using a biological relative's weight as an instrument. Such tests have been rare in the literature so far.

To assess our instrument, we start by exploiting the rich data in the NCDS, and show that the strong association between the respondent's obesity status and parental obesity is virtually unaffected when we condition on environmental factors during childhood and adolescence. Conditioning on these factors at the same time makes the association between the respondent's obesity status and employment status weaker. This might be taken as evidence that it is mainly genetic factors which affect the intergenerational association in obesity. We further test this by exploiting information on adopted children in our data. If the association is only due to genetics, then one would expect no association between the obesity status of adopted children and their adoptee parents. Indeed, we find that the coefficient for adopted children is close to zero, again suggesting that environmental factors play a small role. These results, in sum, provide at least suggestive evidence that parental obesity mainly predicts genetic variation in the respondent's obesity status, making it potentially useful as an instrument.

Using parental obesity as an instruments would still be invalid, however, if the same genes that predict obesity also predict labor market outcomes. We, therefore, also exploit the panel feature of the NCDS and conduct regressions in first differences. This removes all unobserved time-invariant heterogeneity, such as genetic factors correlated with both obesity and labor market success.

In our data there is a negative correlation between obesity and the employment probability among women and men. Although the sizes of these associations become smaller after controlling for an extensive list of controls for

<sup>1</sup> Two recent studies have used specific genetic markers for obesity as instruments (Norton and Han, 2008; Ding et al., 2009). Other papers have used instruments providing non-genetic variation, such as the average BMI of individuals in the same health authority area (Morris, 2006, 2007) and sibling sex composition (Lundborg et al., 2007).

cognitive ability and parental inputs, they remain negative and significant. Using our instrumental variables strategy, no significant effects of obesity on employment remain. Similar results are obtained when we exploit the panel feature and estimate the model in first differences.

The structure of the paper is as follows. Section 2 provides a general analytical framework. Section 3 introduces the National Child Development Study data and reports on the variables used in the empirical part. Section 4 discusses the empirical results. Section 5 concludes.

## 2. Analytical framework

In this section we present an analytical framework, which includes the larger part of the frameworks used in the literature, such as, for example, Cawley (2004). However, we focus on employment rather than wages (see also Lundborg et al., 2007). This implies that we consider another margin of labor market outcomes, where responses to obesity may be different. For example, in a competitive labor market with flexible wages, there may be a very substantial wage penalty to obesity (if obese people are, for example, less productive), which hardly affects employment outcomes. On the other hand if obesity affects the propensity to work in some occupations, while being irrelevant in other occupations, one might observe that conditionally on working obesity does not affect wages. Even though our model framework has the same features as models used to study wage effect, the interpretation of the causal parameter is different.

Our outcome variable is an indicator for being employed, and we adopt a linear probability model:

$$\text{EMPLOYMENT}_{it} = \text{OBESITY}_{it}\beta_t + X_{it}\gamma + \mu_{it} + \varepsilon_{it}, \quad (1)$$

where  $\text{OBESITY}_{it}$  is the obesity status of individual  $i$  at age  $t$  and  $X_{it}$  is a vector of other variables affecting employment rates. The term  $\mu_{it}$  captures genetic and non-genetic factors which may be time-varying, and  $\varepsilon_{it}$  is the residual. This equation strongly relates to Cawley (2004), with the exception that we allow the effect of obesity to vary with age  $t$ . OLS estimation produces consistent estimates of  $\beta_t$  only in case the vector  $X_{it}$  contains all variables that both are correlated with obesity and affect employment. However, for reasons discussed in the previous section  $\text{OBESITY}_{it}$  may be correlated with  $\mu_{it}$ .

One may consider taking first differences if one is willing to believe that  $\mu_{it}$  remains constant over time ( $\mu_{it} = \mu_i$ ). This strategy was adopted by Baum and Ford (2004), Cawley (2004) and Cawley and Danziger (2005). If also the effect of obesity on employment does not vary by age, i.e.  $\beta_t = \beta$ , then the first-difference equation equals

$$\text{EMPLOYMENT}_{it} - \text{EMPLOYMENT}_{it-1} = (\text{OBESITY}_{it} - \text{OBESITY}_{it-1})\beta + (X_{it} - X_{it-1})\gamma + (\varepsilon_{it} - \varepsilon_{it-1}). \quad (2)$$

An important condition for obtaining consistent estimates from a first-difference estimator is that current

obesity should not be related to both current and past employment shocks  $\varepsilon_{it}$  and  $\varepsilon_{it-1}$ . The latter may be the case if the type of nutrient intake depends on employment status, for example, when low-income people have a higher demand for relatively cheap energy dense fattening food. Taking first differences may thus solve some endogeneity problems, but the estimates may still suffer from reversed causality.

According to Cawley (2004) obesity may be affected by the employment status

$$\text{OBESITY}_{it} = X_{it}\delta + \text{EMPLOYMENT}_{it}\alpha + Z_{it}\phi + \theta_{it} + \varepsilon_{it}. \quad (3)$$

where  $Z_{it}$  contains variables only affecting obesity,  $\theta_{it}$  captures genetic and non-genetic components, and  $\varepsilon_{it}$  contains shocks. There are potential sources for endogeneity bias of obesity in the employment Eq. (1). First, the current employment status  $\text{EMPLOYMENT}_{it}$  may affect current  $\text{OBESITY}_{it}$ . This reverse causality means that  $\alpha$  in Eq. (3) is non-zero. Second,  $\mu_{it}$  and  $\theta_{it}$  may be correlated, which implies that there are unobserved factors affecting both obesity and employment outcomes.

Identification of the causal effect of obesity on employment should come from independent variation in obesity status. In particular, the variable  $Z_{it}$  should have a non-trivial effect on obesity. If we use  $Z_{it}$  to instrument  $\text{OBESITY}_{it}$  in the levels Eq. (1), the identifying assumption is that  $Z_{it}$  should be uncorrelated with  $\mu_{it}$  and also to employment shocks  $\varepsilon_{it}$  in the employment regression.

We follow Cawley (2004) and use obesity status of biological relatives as instrumental variables. In particular, we use obesity of the parents of the respondent. It is well established, through twin, adoption and family studies that an individual's risk of obesity is substantially increased when he or she has relatives who are obese. A number of studies have provided evidence, suggesting that 40–70% of the variation in obesity-related phenotypes, such as body mass index, skinfold thickness, fat mass and leptin levels, is inheritable (see Grilo and Pogue-Geile, 1991, for an overview).

The crucial assumption for parental obesity to be a valid instrument for the respondent's obesity is that it should not have an independent effect on the respondent's employment outcomes. One could, however, think of a number of pathways through which parental obesity could affect the respondent's employment outcomes.

First, genetic factors determining weight may be the same, or are correlated with, genetic factors determining labor market success. In the estimations we include several variables correlated with parental labor market outcomes as regressors. These variables should control for such genetic factors, if they exist. Furthermore, estimating the model in first differences as in Eq. (2) may solve some of

these issues as it removes time-invariant genetic effects from the employment regression.

Second, one could imagine the existence of household environment factors common to respondents and their parents that may affect both the respondent's employment status and obesity. Cawley (2004) argues forcefully that all correlation in weight between biological relatives is due to genetic factors, and cites evidence from several studies all pointing to the non-importance of such common household effects. It has been found, for instance, that the correlation between the body size of parents and their children is no different for twins reared apart or together (e.g. Maes et al., 1997). Moreover, it has been found that there is no significant correlation between the body mass of unrelated adoptees reared in the same family (Grilo and Pogue-Geile, 1991). Reviewing the literature on genetic and environmental influences on obesity, Grilo and Pogue-Geile (1991) concluded that the only important environmental experiences are those not shared among family members and that experiences shared among family members appear largely irrelevant in determining individual differences in weight and obesity.

However, this is not uncontroversial. There is a strand of literature that argues that the family environment plays the main role in the development of children's food preferences. The idea is that parents shape the eating environment of their children by making food available and by their own eating habits and food choices (see Birch, 1999, for a review). We have access to an exceptionally rich dataset that allows us to deal with this to some extent. The data has detailed information on parental choices regarding investments in their children's well-being and health, such as whether the child was breastfed, whether the parents took their child out for walks, outings, whether the parents went to swimming pools with their child and whether the mother and father read frequently to the child. Furthermore there is information on mother's smoking behavior during pregnancy, whether there were domestic tensions in the family and whether there were alcohol problems in the family. Such variables are likely to be strongly correlated to parental preferences towards food and eating habits. We will examine the sensitivity of our estimates to the inclusion of these parental input variables. Furthermore, it is not necessarily so that any non-genetic factors potentially reflected in the relationship in obesity between a parent and child renders the instrument invalid. If non-genetic eating preferences are transmitted from the parent to the child, this is no problem as long as these eating preferences are not directly related to the labor market outcomes of the child. It should also be noted that the food rationing implemented in Great Britain during the second world war did not end until 1954, when the last restrictions on the sales of meat and bacon were lifted (Huxley et al., 2000). The parents of the NCDS respondents thus grew up in an environment with food rationing, where fatness could be expected to be more dependent on genetic factors than on excessive eating habits. Finally, our data has a subsample of adoptees. If environmental factors are important one would also expect to see an association in obesity between the adopted child and their adoptee parents.

### 3. Data

In the empirical analyses we use data from the National Child Development Study (NCDS), which is a longitudinal study of about 17,000 individuals born in Great Britain in the week of March 3–9, 1958. Originally, the NCDS started out as the "Perinatal Mortality Survey", with the aim of surveying economic and obstetric factors associated with stillbirth and infant mortality. Since the first survey in 1958, cohort members have been traced on seven other occasions, in 1965 (age 7), 1969 (age 11), 1974 (age 16), 1981 (age 23), 1991 (age 33), 1999/2000 (age 42), and 2004 (age 46). It should be noted, however, that we do not use the information from the 2004 wave, since it does not include information on height and weight.

In the 1958 wave, information was gathered from the mother and from medical records, whereas interviews were carried out with parents, teachers, and the school health service in waves 1–3. In the latter waves, ability tests were administered to the cohort members. In subsequent surveys, information on employment and income, health and health behavior, citizenship and values, relationships, parenting and housing, education and training of the respondents was included.

Since the NCDS is a long panel, attrition may be of serious concern. Case et al. (2005) investigated attrition in the NCDS by comparing low birth weight and father's occupation across the different waves. No evidence for any non-random attrition with respect to these variables was obtained. Moreover, advisory and user support groups of the NCDS compared respondents and non-respondents in the later surveys in terms of social and economic status, education, health, housing and demography. Again, it was found that the sample survivors did not differ from the original sample to any great extent (National Child Development Study User Support Group, 1991). In yet another study, the 1981 sample was compared to the United Kingdom 1981 Population Censuses in terms of key variables such as marital status, gender, economic activity, gross weekly pay, tenure and ethnicity (Ades, 1983). The author concluded that the sample appeared to be representative with respect to these variables.

#### 3.1. Dependent variable: labor market outcomes

Our main dependent variable is employment. We consider employment at age 33 and 42, defined as having a full-time or part-time job, or being self-employed. Table 1 shows that at both ages the employment rates for men are about 92%. For women the employment rate increased from 69% at age 33 to 80% at age 42.

#### 3.2. Independent variables

##### 3.2.1. Obesity status

The NCDS records the height and weight of the respondents at all waves, except for the last one in 2004. In the 1981 and 2000 waves, weight and height were self-reported, while they were measured by interviewers in the other waves. Using the measures on height and weight, we construct the body mass index (BMI), which, in

**Table 1**  
Descriptive statistics.

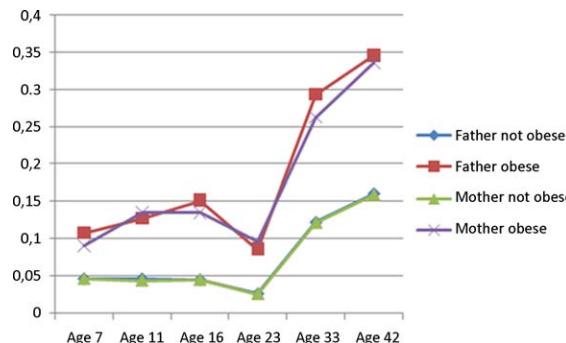
	Males		Females	
	Mean	SD	Mean	SD
Dependent variables				
Employed at age 42	0.916	0.277	0.798	0.401
Employed at age 33	0.924	0.265	0.685	0.465
Obesity				
Obese 42	0.167	0.373	0.169	0.375
Obese 33	0.113	0.316	0.115	0.319
Mother obese	0.039	0.195	0.039	0.194
Father obese	0.069	0.254	0.060	0.237
Demographic variables				
Less than O-level	0.228	0.420	0.238	0.426
O-level equivalent	0.189	0.391	0.281	0.450
A-level equivalent	0.299	0.458	0.263	0.440
Degree equivalent	0.133	0.339	0.110	0.313
White	0.920	0.271	0.923	0.266
Married or cohabitating	0.806	0.396	0.810	0.392
Father's years of schooling	7.502	4.510	7.530	4.490
Mother's years of schooling	7.592	4.384	7.655	4.397
Number of children	1.468	1.349	1.763	1.364
Early life conditions				
Mother's age at birth	26.543	7.521	26.536	7.624
Financial difficulties at age 7	0.040	0.195	0.053	0.224
Financial difficulties at age 11	0.078	0.268	0.086	0.281
Financial difficulties at age 16	0.063	0.243	0.063	0.244
Domestic tension in family	0.027	0.163	0.032	0.176
Parental alcohol problems	0.005	0.071	0.005	0.071
Parental investments				
Mother smoked during pregnancy	0.299	0.458	0.316	0.465
Mother breastfed	0.642	0.479	0.658	0.474
Mother read	0.464	0.499	0.457	0.498
Father read	0.337	0.473	0.338	0.473
Often walked with mother (age 11)	0.520	0.500	0.577	0.494
Often walked with father (age 11)	0.515	0.500	0.520	0.500
Often went swimming with parents (age 11)	0.443	0.497	0.468	0.499
Outings with mother (age 7)	0.778	0.416	0.803	0.398
Outings with father (age 7)	0.674	0.469	0.655	0.475
Low birth weight	0.080	0.272	0.103	0.304
Cognitive ability				
Math score (age 7)	4.990	2.796	4.857	2.685
Math score (age 11)	17.221	11.110	16.622	10.480
Reading score (age 7)	21.431	9.172	23.391	8.635
Reading score (age 11)	15.693	7.342	15.644	6.896
BSAG score (age 7)	8.282	8.679	6.205	7.445
BSAG score (age 11)	8.178	9.026	5.711	7.158
Other				
Number of cigarettes	4.486	9.145	3.898	7.844

turn, is used to construct an indicator of being obese. We follow the convention and label someone as obese if having a BMI of 30 or above. Since the definition of obesity varies for children and teenagers, we use age-specific thresholds of obesity and overweight, as provided by Cole et al. (2000).

Previous research has shown substantial measurement errors in self-reported height and weight (e.g. Rowland, 1989). For instance, underweight people tend to over-report their weight, while the opposite is the case for overweight people. We correct for such errors in self-reported height and weight in the 1981 and 2000 waves by using the results from Burkhauser and Cawley (2008), where prediction equations for actual weight and height

were provided.<sup>2</sup> Applying their formulas, average BMI for females at age 42 increase from 25.29 to 25.68 and from 26.58 to 26.75 for males. This, in turn, increases the fraction of obese females from 15.1% to 16.9% and the fraction of obese males from 15.7% to 16.7%. The correction for systematic measurement error might be more important in OLS regressions than in regressions in which obesity is instrumented. It is well known that classical measure-

<sup>2</sup> The results from Burkhauser and Cawley (2008) were obtained by comparing self-reported weight with measured weight in the NHANES data in the US. It should be noted that the relation between self-reported weight and measured weight may differ between the UK and the US.



**Fig. 1.** Obesity status at various ages by parental obesity status.

ment errors in regressors cause OLS estimates to be biased towards 0, which can be solved by instrumenting the regressors. Descriptive statistics on obesity status are shown in Table 1.

The NCDS also includes information on the weight and height of the respondent's father and mother. This information was assessed in wave 3, when the respondents were 11 years old, and is self-reported by the mother and father. Since we are mainly interested in the obesity of a biological parent, we make use of information on family relations, allowing us to discriminate between biological parents and adoptee parents, stepparents and foster parents. In the empirical analyses we only focus on individuals for which both the father's and mother's obesity status are recorded at age 11.

Fig. 1 shows obesity rates at various ages by the obesity status of the mother and the father for the NCDS respondents. Clearly, the obesity rates are greater for those having an obese mother or father at all ages considered. At age 7, the obesity rate of those with obese parents is about twice as high as for those with non-obese parents. At ages 11 and 16 the difference becomes even greater. From age 23 and onwards, the picture suggests that the increase in obesity is greater for those having obese parents. However, the dip in obesity at age 23 is also greater for those with obese parents. This dip may suggest that the self-reports on height and weight at age 23 are unreliable and that our measurement error correction does not address this adequately. In the following, we will, therefore, not use information on obesity at age 23. Another reason for not using the data at age 23 is that at this age some individuals were still in full-time education. At age 42, which is the age at which we evaluate labor market outcomes, the obesity rate of those having obese parents is about 33%, whereas the corresponding rate for those not having obese parents is about 15%. The figure suggest that there are substantial differences in the probability of being obese by the obesity status of the parents. This also gives us the predictive power needed from our instrument.

### 3.2.2. Other background variables

The NCDS contains rich information on issues such as the individual's initial health assets, the socioeconomic status during early childhood and cognitive ability during

childhood. We follow Llena Nozal (2007) and Case et al. (2005) when constructing our relevant background variables. In Table 1, sample means are shown. Since many variables have some item non-response we follow Case et al. (2005), and construct dummy variables that indicate if the information on a variable is missing to avoid losing many observations.

To measure the family's socioeconomic status, we include information on the number of years of education of the mother and the father, a measure of permanent family income at age 16, and a measure of financial problems in the family at ages 11, 14, and 16. As to family income, the NCDS only records it when the child is 16. Since this measure might not reflect living standards earlier in childhood or persistent poverty problems, the data holders have developed a measure of family income, which we will make use of. Since the permanent income measure is dependent on the estimation technique and data availability, however, we will use this measure in combination with the measures of whether or not the family had serious financial difficulties when the child was aged 7, 11, and 16.<sup>3</sup>

We created a number of different measures of parental inputs and early life conditions. In order to capture mother's smoking during pregnancy, we created a dummy variable indicating whether the mother smoked after the fourth month of pregnancy. Smoking during pregnancy has been found to be related with cognitive deficiencies and other health problems, such as low birth weight, and may thereby affect both obesity and labor market outcomes (see, for instance, Blair et al., 1996; Williams et al., 1998). Moreover, the mother's age at the child's birth may affect child's health through, for instance, nutritional deficiencies if the mother is very young, or delivery complications if the mother is older (Llena Nozal, 2007). In addition, we include an indicator of having low birth weight and whether the respondent was breastfed.

A number of indicators of parental activities together with their child were created as well. These indicated whether the mother or the father often took the child for walks, to swimming pools, to outings and were assessed when the child was 11. In addition, we constructed variables indicating whether the mother and the father frequently did read books to the respondent at age 7. Finally, two variables measuring the presence of any alcohol problems among the parents and the presence of any domestic tension during the upbringing of the child were created.

To measure cognitive ability at early ages, we used the results from test scores on math and reading tests at ages 7 and 11. In the math test, which was designed for the NCDS, the score ranges from 0 to 10. Prior studies have established that test scores at the age of 7 show a significant impact on later education attainments and

<sup>3</sup> Family income in the NCDS consists of father's earnings, mother's earnings, and other sources of family income. In order to obtain a prediction of permanent parental income, the data holders used information on individual characteristics to predict permanent wages for a sample of NCDS respondents and their fathers. For details, see Taylor (2008).

**Table 2**

Marginal effects and robust standard errors from OLS regression for employment at age 42 (separate for males and females).

	Employment									
	Males					Females				
	(I)	(II)	(III)	(IV)	(V)	(I)	(II)	(III)	(IV)	(V)
Obese 42	-0.023*	-0.019*	-0.017	-0.013	-0.013	-0.049***	-0.039**	-0.040**	-0.038**	-0.038**
n	4185	4185	4185	4185	4185	4147	4147	4147	4147	4147
Obese 33	-0.045***	-0.030**	-0.030**	-0.025*	-0.027*	-0.061***	-0.046**	-0.047**	-0.045**	-0.046**
n	(0.016)	(0.014)	(0.014)	(0.014)	(0.015)	(0.022)	(0.022)	(0.022)	(0.022)	(0.022)

I: no controls; II: basic controls included (marital status, ethnicity, education, smoking, number of children); III: controls for parental background added (permanent income, parental education financial difficulties, mother's age at birth); IV: controls for ability added (test scores on math, reading scores scores at ages 7 and 11, and BSAG scores); V: controls for parental inputs added (going for walks, outings, going to swimming pools, smoking during pregnancy, low birth weight, breastfed, alcohol problems, domestic tension, mother's smoking during pregnancy, and reading to child).

\*  $p < 0.1$ .\*\*  $p < 0.05$ .\*\*\*  $p < 0.01$ .

labor market outcomes (Currie and Thomas, 2001). Reading skills were assessed by the Southgate Reading Test.

Social maladjustment was assessed at ages 7 and 11 with the Bristol Social Adjustment Guide (BSAG) study. The BSAG consists of a large number of behavioral items, such as 'attitudes to teacher', 'attitudes towards other children', evaluated by the child's teacher. Higher scores indicate higher maladjustment.

Education is measured through four dummy variables, indicating national vocational qualification levels. The following categories were included: less than O-levels, O-level equivalent, A-level equivalent, and degree equivalent. This follows the definition used by Llena Nozal (2007). To capture marital status, we created a dummy variable indicating whether the respondent was married or cohabitating. Ethnicity was measured through a dummy variable, indicating whether the respondent belonged to the European Caucasian group. Additional covariates were the number of children and the number of cigarettes smoked per day.

## 4. Results

### 4.1. Baseline OLS results

We start by examining whether there exist any evidence suggesting different employment levels by obesity status in our data. Table 2 summarizes the results from regressions on the association between obesity and employment at age 42. The first column of the tables shows the results when we include only the individual's obesity status as covariate. In columns (II) to (V) we then add various sets of control variables. It should be noted that the results in the different rows reflect separate regressions on the effect of obesity at various ages on the outcomes at age 42.

Starting with only obesity as a covariate (columns (I)), the results show that both obese males and females face significantly lower employment probabilities. Being obese at age 42 is associated with a 4.9 percentage points reduction in the employment probability for females and a 2.3 percentage points reduction for males. Even stronger

results are obtained when measuring obesity at age 33, with the obesity penalty doubling for males and increasing by about 20% for females.

### 4.2. Accounting for heterogeneity

#### 4.2.1. Controlling for socioeconomic characteristics

The results discussed so far did not control for other characteristics of the individual that may be correlated with obesity. In the columns (II) of Table 2, we, therefore, add some basic control variables, including marital status, ethnicity, education, number of children and smoking. For both males and females, the negative association between obesity and employment is still significant for both males and females, although it is somewhat reduced in magnitude. The pattern is the same, irrespective if obesity is measured at age 33 or age 42.

#### 4.2.2. The influence of family background

Obese persons may come from other types of family backgrounds than their non-obese counterparts. Coming from a background with lower economic and human capital, for instance, may also affect later labor market outcomes. Thus, the previously found employment "gap" by obesity status may simply reflect such differences. In order to account for differences in family background, we include controls for parental education, permanent income, and financial difficulties in the household at ages 7, 11, and 16. As shown in columns (III) of Table 2, the association between obesity and employment does not change when controlling for family background.

#### 4.2.3. What role does cognitive ability play?

There may still be other important differences between obese and non-obese people that we have not accounted for. One may suspect that obesity is simply picking up some differences in cognitive ability between the obese and non-obese. We, therefore, add controls for cognitive ability, measured through results on test scores in math and reading at ages 7 and 11. In addition, we control for social maladjustment at ages 7 and 11. The results are shown in the columns (IV) of Table 2. Adding cognitive

**Table 3**

Correlation in obesity status between the child and the child's mother and father.

	(I) Raw association				(II) Association with controls			
	Males		Females		Males		Females	
	Mother	Father	Mother	Father	Mother	Father	Mother	Father
Age 7	0.012*	0.043***	0.048***	0.029**	0.013**	0.044***	0.050***	0.028**
Age 11	0.042***	0.036***	0.041***	0.030***	0.042***	0.036***	0.040***	0.029***
Age 16	0.028***	0.033***	0.046***	0.049***	0.026***	0.031***	0.047***	0.045***
Age 23	0.030***	0.063***	0.101***	0.033**	0.025***	0.059***	0.094***	0.029**
Age 33	0.059***	0.199***	0.151***	0.108***	0.044**	0.192***	0.133***	0.098***
Age 42	0.131***	0.187***	0.193***	0.140***	0.124***	0.176***	0.165***	0.128***

(II) Basic controls, parental background, cognitive ability, and parental inputs.

\*  $p < 0.1$ .\*\*  $p < 0.05$ .\*\*\*  $p < 0.01$ .

ability only has a slight effect on the employment estimates. For males, however, the employment penalty obtained when obesity is measured at age 33 is now reduced in magnitude and is only significant at the 10% level.

#### 4.2.4. Is it the parental inputs?

Finally, we consider the importance of a number of parental inputs, that may not have been picked up by the family background variables. We consider leisure activities, such as taking the child frequently for walks, to swimming pools, and on outings at age 11. Moreover, we include controls for parental educational inputs, measured through whether the mother and the father read frequently to the child. Finally, we control for mothers smoking during pregnancy, whether or not the mother breastfed, the existence of alcohol problems within the family, low birth weight, and the existence of any domestic tension within the household.

As seen in the columns (V) of Table 2, the associations between obesity and employment do hardly change. It should be noted that very few of these early parental inputs show any significant effect on later employment (results on request). In sum, differences in family background, cognitive ability, and parental inputs by obesity status do not seem to fully explain why obese people fare worse on the labor market at later ages.

#### 4.3. Instrumental variable estimation

Even though the richness of the NCDS data allow us to control for many previously unmeasured factors, such as cognitive abilities and parental investments, important factors may still remain unmeasured. Moreover, as previously discussed, reverse causality running from labor market outcomes to obesity and BMI may bias our results. We will, therefore, resort to instrumental variables estimation, using our indicator of the obesity status of the mother and the father of the respondent as instruments.

#### 4.3.1. Parent-child association in obesity and the role of family background

First, we will examine to what extent the association in obesity between the parent and the child is affected when

accounting for non-genetic factors pertaining to the family background of the child. If the association in obesity is unaffected when accounting for potentially important environmental influences during childhood and adolescence, this provides at least suggestive evidence that the association is mainly due to genetic factors.

In the first four columns of Table 3, the raw parent-child association in obesity at various ages are shown, separately for females and males and for mothers and fathers. Each row thus represents a separate regression clearly, there is a strong association in obesity between the parent and the child at all ages, for both males and females. The associations tend to be stronger at older ages, which is not that surprising, since at these ages, the main respondent is closer to the age at which the height and weight of the mother and the father were assessed.

In columns five to eight of Table 3, the same associations are again examined but this time controlling for all observed demographic, socioeconomic, environmental and behavioral characteristics described in the preceding section. The results show that the parent-child associations remain very similar. Moreover, most of the included control variables are not significant in explaining obesity at later ages (results available on request). This is in line with the results from previous studies, suggesting that the association in obesity between biological relatives is mainly due to genetic factors and that factors related to a common environment plays a small or no role.

#### 4.3.2. Is the mother-child relationship in obesity different for adopted children?

Next, we turn our attention to differences in the parent-child association in obesity between adopted and non-adopted children. The NCDS records whether the main respondent was adopted. If the parent-child association is only due to genetics, we would expect no relationship in the obesity status of adopted children and their adoptive parents. For the analysis to make sense, we must first make sure that the allocation of adopted children is made in a close to random manner. For the NCDS this has been established by Sacerdote (2002).

The sample of adopted children is small, only 79, and the results are, therefore, not likely to be very strong. Yet, they may provide at least some suggestive evidence, pointing in the same direction as our previous results. We

**Table 4**

Correlation in body size between adopted and biological children obesity status and the parents obesity status.

	Ages		
	Age 7	Age 11	Age 16
Mother obese	−0.017 (0.082)	−0.029 (0.065)	−0.019 (0.120)
Father obese	−0.021 (0.081)	−0.028 (0.074)	−0.010 (0.085)
Non-adopted (mother)	0.012 (0.029)	−0.003 (0.025)	−0.019 (0.028)
Non-Adopted (father)	−0.013 (0.023)	−0.014 (0.020)	0.010 (0.023)
Non-adopted (mother) <sup>*</sup> mother obese	0.045 (0.082)	0.071 (0.065)	0.057 (0.119)
Non-adopted (father) <sup>*</sup> father obese	0.056 (0.081)	0.058 (0.074)	0.051 (0.085)
Observations	10,565	11,487	8827
R2	0.01	0.01	0.01

Robust standard errors in parentheses. \*\* $p < 0.05$ , \*\*\* $p < 0.01$ .\*  $p < 0.1$ 

run regressions, pooling the samples of adopted and natural children, and include interaction terms between the dummy variable indicating not being an adopted child and the variable measuring the obesity status of the parent. If the parent–child association is due to genetics, we would expect the interaction effects to be positive, but the main effect, measuring the relationship for adopted children, to be close to zero or negative for adopted children.

To maximize the number of adopted children, we will only consider the respondents at ages 7, 11, and 16. At later ages, due to attrition, the sample of adopted children becomes very small. The early ages are the most relevant since this is the period where most children and parents share a common environment.

In Table 4 OLS results for the full sample at the ages 7, 11, and 16 are shown. The table shows the coefficients of the variables indicating parental obesity, the dummy variables indicating not being an adopted child, and the interaction terms between the former and the latter variables.<sup>4</sup> The results show that the interaction terms are positive at ages 7, 11, and 16. The magnitude of the main effect of mother's and father's obesity suggests an association that is either close to zero or negative for adopted children. This is found for all the ages considered in the analysis. This is in line with what we would expect if genetics play the major role for the association in obesity between parents and children. The sample of adopted children is small, however, and the interaction effects never reach statistical significance. We are, therefore, not able to draw firm conclusions from this exercise, but we note that the results at least point to the direction of no association or a negative association in obesity between adopted children and their adoptee parents.

The exercises above, together with the evidence from previous twin and adoption studies, gives some credibility to the claim that the obesity of a parents predicts genetically induced variation in the obesity of the child. If true, non-genetic factors that are common to the child and the parent play little role for obesity, meaning also that those factors do not predict both obesity and labor market success, which would invalidate our instrument.

<sup>4</sup> We ran the analysis both with and without controls for parental background and environmental influences but the results were similar. The results presented in Table 4 are without controls.

**Table 5**

Marginal effects and standard errors from IV employment regressions for males and females.

	Employment	
	Males	Females
Obese 42	0.113 (0.086)	0.014 (0.129)
F-stat. 1st stage	35.41	34.79
Sargan test ( <i>p</i> -val)	0.11	0.19
<i>n</i>	4185	4147
Obese 33	0.149 (0.103)	0.082 (0.176)
F-stat. 1st stage	32.08	24.60
Sargan test ( <i>p</i> -val)	0.77	0.13
<i>n</i>	3543	3694

Basic controls, parental background, cognitive ability, and parental inputs. \* $p < 0.1$ , \*\* $p < 0.05$ , and \*\*\* $p < 0.01$ .

#### 4.3.3. IV results

Next, we perform instrumental variables analyses. The results are summarized in Table 5. To preserve space, we only show the marginal effects of the obesity variable.

As shown in the table, the IV estimates are very different from the OLS estimates. For males, the association between obesity and employment is now positive but imprecisely measured and insignificant. The instruments predict well, with *F*-statistics above 10 in the first-stage regressions. Similar results are found for females, where the coefficients of obesity at various ages are now positive and insignificant. The previously found negative and significant association between obesity and employment of women has now completely vanished. Also among women, the instruments predicts obesity well. While we cannot exclude that our exclusion restrictions are valid in the employment regressions, based on the Sargan tests, it should be noted that the *p*-values are rather low in two out of the four specifications, which may be a cause of concern.

However, even a rejection of the overidentification test statistic does not necessarily mean that the instruments are invalid. If there is heterogeneity in the effects, different instruments may give different causal effects. IV measures the treatment effect for the compliers, which may be different subsamples for different instruments. Thus, if the compliers are different when using fathers' obesity as an instruments compared to when using mothers' obesity status, the overidentification test statistic may be rejected (see for instance, Angrist and Pischke, 2009, for a discussion). One should, therefore, be careful in interpret-

**Table 6**

Marginal effects and robust standard errors from first-difference employment regressions for males and females.

ΔEmployment		
	Males	Females
ΔObesity	0.026* (0.015)	−0.033 (0.028)
Observations	3470	3578

Controls for change in marital status and smoking status. Robust standard errors in parentheses. \* $p < 0.1$ , \*\* $p < 0.05$ , \*\*\* $p < 0.01$ .

ing the results from an overidentification test, such as the Sargan test. Our result raises the question why one should expect different effects when using fathers' or mothers' obesity status as instruments if the instruments only pick up genetic information. The parents of the child both provide 50% of the child's genes and this alternative interpretation of the overidentification test may, therefore, also suggest that our instruments pick up something more than pure genetics. In relation to this, it could also be noted that the results in Table 3, i.e. the associations between parental and child obesity differ between mothers and fathers.

#### 4.4. Changes in obesity and employment

If the genes that determine obesity are the same or are correlated with the genes that determine employment, both our OLS and IV estimates may still be biased. To the extent that cognitive ability, for instance, is genetically determined it is certainly not farfetched to assume that both employment and obesity may be affected by it. In the case of cognitive ability, we are able to control for it by including education and test scores at childhood, but for other potentially genetically determined characteristics we may be less successful.

We, therefore, utilize the panel feature of the NCDS in order to examine changes in employment status and obesity between the waves. Estimating our regressions in first differences, we are able to remove some of the unobserved heterogeneity that may plague both the OLS and IV estimates. Only time-invariant unobserved heterogeneity will cancel out. It should be noted that some of the important unmeasured factors in our case are indeed time-invariant, such as genetic upset.

Table 6 shows the results from the first-difference equation. In this specification, we are analyzing changes in employment between age 42 and 33 as a function of changes in obesity between ages 42 and 33. In our sample of males, 9% changes employment status across the waves. The corresponding percentage of females is 30%. Since most of the control variables are constant over time, they will drop out from the analysis, but we allow for changes in marital status and in smoking. For males, the results show a slight positive association between changes in obesity and employment that is significant at the 10% level. For females, the association is negative but not significant.

#### 5. Discussion and conclusions

We had two main aims with this paper. The first was to study the causal effect of obesity on employment using (1)

parental obesity as an instrument and (2) a fixed effects approach. The second was to carefully assess the validity of using the body size of a biological relative as an instrument for one own's body size. This latter aim was motivated by the growing literature that uses instruments supposed to carry genetic information.

Starting with our first aim, we obtained no results suggesting a strong causal effect of obesity on employment. We started by showing that there exist large differences in employment probabilities between obese and non-obese men and women. We also showed that these associations did not disappear when controlling for a wealth of potentially important factors pertaining to family background, cognitive ability, and inputs during childhood and adolescence. When we instrumented the individual's obesity status with that of the respondent's mother and father, no significant effect remained, however. This result was confirmed in panel data regressions on first differences in employment and obesity. With this strategy, we removed unmeasured time-invariant heterogeneity, such as the influence of common genes.

Our results confirm those of Cawley (2000a,b) and Norton and Han (2008), where no effect of obesity on employment among US men and women was obtained, but contrast to those obtained by Morris (2007) for the UK. One potential reason for the diverging findings is that genetically induced variation in obesity was used to identify the effect in this study and in the studies by Cawley (2000a,b) and Norton and Han (2008), whereas Morris (2007) relied on variation in obesity induced by local area variation in average BMI. Since different instruments are being used, the compliant subpopulation may very well differ between the studies and different Local Average Treatment Effects will be produced.

Regarding our second aim, assessing the validity of so called "genetic" instruments, we obtained some mixed results. Our tests of the instruments suggested that it is mainly genetic variation which causes intergenerational correlation in obesity, while environmental factors play less of a role. This is in line with evidence from a large number of studies in medicine, where no common household effects are obtained. The instruments passed our tests for overidentifying restrictions, but the  $p$ -values were somewhat low in some specifications. This does not need to imply that our instruments were not valid, since a rejection of the Sargan test statistic could also result from treatment heterogeneity between the instruments. Such an interpretation would, on the other hand, raise the question as to why the compliers would be different when using fathers' obesity or mothers' obesity as instruments. Since both parents provide 50% of the child genes, one would expect similar results irrespective of what instrument is used. Future studies should further assess the validity of using the weight of a biological relative as an instrument.

To conclude, if obesity has a causal effect on some labor market outcomes, there may be large gains at both the individual and societal level from more efficient obesity treatments. Indeed, previous studies have suggested relatively large penalties from being obese. If obesity is simply picking up some other, unmeasured trait, treat-

ments and/or policies aimed at reducing obesity will not have the intended effects on labor market outcomes. It is, therefore, of great importance to increase the knowledge about the causal effect of obesity on labor market success. While our results do not imply that obesity affects employment, these results were obtained in a UK context and using a particular type of instrument. Since the literature on the effect of obesity on employment is rather limited, it should be of great interest to analyze the corresponding effects in other countries, where the labour markets look different, and using different sources of variation in obesity.

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