The Sweet Life:
The Long-Term Effects of a Sugar-Rich Early Childhood

PRELIMINARY - DO NOT CIRCULATE OR CITE

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Abstract

Nearly all American infants and toddlers consume sugar added to foods daily and exceed the adult limit of seven teaspoons per day by the time they are two years of age. Although put forth as a key risk factor for obesity and related disease, rigorous evidence on the long-term effects of such early exposure to sugars is limited. We provide new evidence on the long-term effects of exposure to sugar-rich diet in early childhood on diet and health in later adulthood. We exploit the end of the post-war rationing of sugar and sweets in 1953 in the United Kingdom as a source of plausibly exogenous variation in exposure to sugar-rich diet early in life. Using regression discontinuity design along with the ordinary least squares, we assume that cohorts born or conceived right before and after the rationing ended are similar, except in their exposure to sugar and sweets early in life. Our approach is informed by the sharp increase at more than 100% in sugar consumption - but not other foods - soon after the rationing ended. More than five decades later, we observe no difference in total calories, or in protein or fats intake between cohorts. However, their diet composition differs: cohorts exposed to sweets rationing consume less added sugars, more fiber, fruits and vegetables, compared to cohorts conceived or born soon after the rationing ended. We also find that adults born during the rationing are less likely to be obese, to have diagnosed diabetes, cholesterol, arthritis, or to be diagnosed with at least one of these diet-related chronic disease. Preliminary evidence also shows that adults born during rationing attained more education and wealth compared to adults born after. Our results suggest that exposure to sugar-rich diet early in life affects preference for sweetness that persist into adulthood and that such dietary patterns over the life-course translate into worse health and economic outcomes; pointing at the importance of obesity prevention efforts early in life.

Key words: early childhood, added sugars, preferences, habits, diet, health

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1 Introduction

The average daily intake of sugars added to foods during processing and preparation is too high; exceeding triple the recommended daily amount (WHO, 2015b; Powell et al., 2016; Bowman et al., 2017; Knüppel et al., 2017; Johnson et al., 2009). This is concerning because excess intake of added sugars is a key risk factor for unhealthy weight-gain, cardiovascular disease, diabetes, and cancer (Vos et al., 2017; Ludwig et al., 2001; Fung et al., 2009; Ackerman et al., 2005; Larsson et al., 2006), diseases that account for nearly 70% of all deaths and a global economic burden of $2 trillion per year (Htenas et al., 2017). Mortality risk from heart disease more than doubles when a fifth or more of daily calories come from added sugars (Yang et al., 2014). Sugary drinks alone contribute to 184,000 annual deaths (Singh et al., 2015) as well as to substantial economic losses from absenteeism, reduced productivity and lost earnings (Guerrero-López and Colchero, 2018).

More troubling is that infants and toddlers also consume way too much added sugar. While nutritional guidelines recommend zero added sugars for children up to age 2 (Vos et al., 2017), American infants and toddlers consume an average of 4.2 tsp of added sugars per day, and their consumption vastly exceeds the recommended limit for *adults* by age 24 months (Herrick, Herrick; Vos et al., 2017). This is not unique to the US as added sugar consumption among children under 2 ranges from 12% of total calories in countries like Denmark, Sweden and South Africa and to a whopping 25% in Portugal (WHO, 2015b). In Mexico and Chile, two in three toddlers consume sweetened beverages by age 12 months, and four in five before they reach 24 months (Essman et al., 2018; Afeiche et al., 2018). Since nutritional intake *in utero* also has lasting metabolic effects (Almond and Currie, 2011; Barker, 1998a), excess added sugar consumption by pregnant women may lead to similar problems for their offspring.

In this paper, we provide some of the first causal evidence on the long-term effects of a sugar-rich diet *in utero* and in early childhood on diet, health and socio-economic outcomes. Intake of added sugars early in life is likely important for two main reasons. First, it can cause physiological changes with lasting adverse effects on disease risks (Rosenfeld, 2015; Bateson et al., 2004; Pawlak et al., 2004; Langley-Evans and Sculley, 2005; Barker, 1998b; Hales, 1997; Jackson, 2002; Bayol et al., 2008). A rapidly growing body of evidence suggests that key pathways that transform early nutritional experiences into adult health are epigenetic in nature (Rosenfeld, 2015; Bateson et al., 2004). Several experimental studies with animals have confirmed that nutrition rich in sugars and carbohydrates *in utero* and in infancy establish permanent physiological and metabolic states that affect the risk of diseases later in life (Pawlak et al., 2004; Bateson et al., 2004; Langley-Evans and Sculley, 2005; Barker, 1998b; Martyn and

Barker, 1994; Hales, 1997; Jackson, 2002; Bayol et al., 2008).

Second, early life intake of added sugars may also create a habit or an addiction for sugar (Drewnowski et al., 2012; Thurow, 2016; Sullivan and Brumfield, 2016; Birch et al., 2007; Domel et al., 1996; Gibson et al., 1998; DiNicolantonio et al., 2018; Avena et al., 2009). Although there is an innate human desire for sweetness (Drewnowski et al., 2012; Sullivan and Brumfield, 2016; Thurow, 2016), the degree of preference is substantially modified by the exposure to a sugar-rich diet within the first 1000 days of life (Birch et al., 2007; Jayasinghe et al., 2017). Sugar, in contrast to other nutrients such as fat or proteins, creates craving, bingeing, and withdrawal. The neurological reward for sugar intake can in some cases even surpass that of cocaine (DiNicolantonio et al., 2018; Avena et al., 2009). Such physiological responses early in life may facilitate the formation of a habit of excess sugar consumption that lasts for a lifetime. For these reasons, experts worldwide have called on countries to eliminate or at least reduce added sugar intake among young children (WHO, 2015b).

Excess early intake of added sugars may also affect learning. Substantial evidence based on animal studies has linked consumption of a diet rich in added sugars and fats with cognitive dysfunction. During critical periods of brain development, added sugars in mice adversely influenced the functioning of the hippocampus - a brain region associated with the control of learning, memory processes, as well as food intake control (Kanoski and Grill, 2017; Davidson et al., 2009, 2007; Baym et al., 2014; Meng et al., 2016; Noble and Kanoski, 2016). Sugar-rich diets in utero and early in life led to deficits in learning and memory capabilities throughout life even if intake of added sugars decreased with age (Noble et al., 2019). Evidence is emerging among humans as well. Studies have found associations between maternal obesity and poor performance of child cognitive or executive tasks (Tanda et al., 2013; Hinkle et al., 2012; Neggers et al., 2003) and that sugary beverage intake during pregnancy or early childhood is associated with on lower child cognition (Casas et al., 2013). Obesity and early-life weight status is inversely associated with children's perceptual reasoning and working memory scores, and possibly full-scale IQ scores (Li et al., 2018; Kamijo et al., 2012), likely affecting education attainment (Cawley, 2004; Goettler et al., 2017).

Both lower educational attainment and chronic disease may affect labor market outcomes and hence wealth accumulation (Cawley, 2004; Goettler et al., 2017). The obese are nearly twice as likely to take sick-leave and experience reduced work productivity (Narbro et al., 1996; Finkelstein et al., 2010). A long list of studies have documented that obesity-related chronic illness lead people to accumulate fewer assets and wealth during their working years either due to absenteeism, reduced productivity or selection into occupations with smaller returns (Trogdon et al., 2008; Finkelstein et al., 2010; Popkin et al., 2006).

To identify the causal effects of excess added sugars early in life on long-term outcomes, we exploit the United Kingdom's (UK) food rationing program that began during World War II and ended in the mid-1950s; specifically sugar and sweets rationing that ended in 1953. The program allocated a scientifically devised portion of specific foods to keep individuals healthy while assuring equal distribution of food during wartime related shortages. It focused especially on limiting processed, non-essential, sugar-rich foods; while fish, fruits and vegetables were never rationed. The ration limits were not extreme. In fact the rationing scheme afforded one a very healthy diet and conformed closely to today's nutritional guidelines (Foster and Lunn, 2007a; Fisher et al., 1977).

The lifting of rationing on sugar and sweets in 1953 seemed to mainly affect added sugar consumption. According to reports, consumption of added sugars among children age 6-12 during rationing was within today's recommended amounts, i.e. below 4oz per week, but, shortly after rationing was lifted consumption more than doubled (Jackson, 1974; Zweiniger-Bargielowska, 2000; Jamel et al., 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999). Using the data from the National Food Survey (NFS), we also observe a sharp and significant increase in the consumption of added sugars of about 100% shortly after the rationing ended, but found no change in the consumption of other nutrients. This sharp and quick response, in part, justifies the use a regression discontinuity design to study the causal effects of rationing.

We assess the long term differences in diet using data from the National Diet and Nutrition survey (2008-2016). More than five decades later, we find that cohorts born just after rationing ended consume significantly more added sugars as adults than cohorts born before rationing was lifted. Specifically, those born after rationing consume between 15 and 20 grams of added sugars more per day (equivalent to about three Oreo cookies or half an 8-ounces soda can), but consume less intrinsic sugars and fiber present in fruits and vegetables. However, we observe no difference in total calories, protein and fat intake between cohorts. This suggests that preferences for added sugars form early and persist into adulthood.

These diet effects seem to be translated into differences in health outcomes later in life. Using the English Longitudinal Survey of Aging (2008-2016), we find that more than five decades after the rationing ended, adults conceived just post rationing are 19 percent more likely to be obese, 61 percent more likely to be diagnosed with diabetes, 25 percent more likely to have high cholesterol, 50 percent to have arthritis, and 56 percent more likley to have three or more diet-related chronic diseases. We also find meaningful but insignificant effects on hypertension and heart disease. Using the same data, we find preliminary evidence that economic outcomes across birth cohorts are different as well. Specifically, we find that compared to cohorts born

during food rationing, cohorts born after were about less likely to attend high school or college, are less likely to be married or to live with a partner, and are less wealthy.

Our results are robust to several falsification tests. First, we examined changes in dietary, health and economic outcomes for people born before the rationing ended using earlier cut-off dates and for people born after the rationing ended using later cut-off dates. Second, we show that individuals born before and after the rationing cut-off date are balanced with respect to parental and childhood characteristics such as socio-economic status, parental education and occupation, parental health, and parenting practices. In addition, we control for age indicators to account for the effects of aging on health. Finally, we repeat our analyses for diseases whose incidence should be unrelated to diet or sugar consumption in particular (e.g. Type 1 diabetes, Parkinson disease, cataracts, etc.).

These results are some of the first causal evidence on the long-term effects of a sugar rich early childhood. Evaluating these relationships empirically is challenging - in large part due to the lack of high-quality data that span a period that is sufficiently long and because of selection concerns. For these reasons, the bulk of the credible empirical work estimating causal impact of excess intake of added sugars early in life on health thus far employs either animal models (Antar et al., 1970; Samuelsson et al., 2008; Pawlak et al., 2004; Walker et al., 2002; Scribner et al., 2007; Roncal-Jimenez et al., 2011) or small scale clinical studies with short follow-ups (Thangaratinam et al., 2012; Zheng et al., 2015; Aeberli et al., 2011; de Ruyter et al., 2012), for which compliance wanes with increasing study length (Malik and Hu, 2012). However, because obesity and chronic diseases, in particular, do not manifest themselves until after the fourth or fifth decade of life, data with sufficient follow-up time is needed to detect causal action. That has been difficult to achieve in human populations. Much of the empirical evidence linking excessive added sugar intake to health outcomes in humans is observational and concerns have been raised about the validity of these estimates due to selection issues; many of which we try to address here (Malik and Hu, 2012).

The extent to which policies aimed at improving early life conditions can improve long-term health and economic well-being is of great interest (Hoynes et al., 2016). A better understanding of these linkages is critical for more targeted intervention strategies against obesity and related disease. This study provides important evidence on the effects of early exposure to sugar-rich diet and its impact on both dietary habit formation as well as adult health and economic outcomes using rarely-available plausibly exogenous assignment to a different diet as a result of sugar rationing. At a minimum, it justifies early childhood obesity prevention efforts and efforts to update the new 2020-2025 guidelines on added sugar intake for children below two years of age. (Foster and Lunn, 2007b; Moynihan, 2016; Moynihan and Kelly, 2014).

2 Food Rationing in the UK

The limitations on sugar and sweets were a central part of the 14-year long food rationing program. The program was designed to guarantee each individual a healthy diet with fair shares for all at a time of national food shortage due to World War II. The Ministry of Food allocated a scientifically devised weekly provision of specific foods to each person. Every person was given a ration book with coupons that were required to be able to purchase rationed goods. Special allowances were made for children under six years of age, and for pregnant and nursing mothers. They were given special green ration books with extra coupons, and had priority access to cod-liver oil, fruits and vegetables. They were also given extra milk from welfare clinics. Rationing focused primarily on processed, non-essential, sugar-rich foods; fish, fruits and vegetables, for example, were never rationed. People were encouraged to eat fruits and vegetables daily, and to use of healthier alternatives in food preparation. For example, carrots were promoted as an alternative to sugar in cakes due to their natural sweetness.

The diet under food rationing was not extreme deprivation. The diet was in many ways similar to today's recommended dietary guidelines (see Figure 1). A diet based on these limits would be about 2,500 calories per day, 35% of which came from fats, 13% came from protein, and 52% from carbohydrates - all within today's dietary guidelines (WHO, 2015a; HHS, 2015). During our study period between 1950-1953, adults consumed on average about 40 grams of sugars per day or about 6.5% of total calories (NFS, 2011). Young children's intake was also within today's daily recommended dietary allowance. An average 4-year old consumed 1445 calories per day compared to 1500 recommended. About 13 percent of calories came from protein compared to 15 percent recommended, 40 percent from fat compared to between 25 and 35 percent recommended, and 47 percent from carbohydrates compared to 45 to 55 percent recommended. Calories coming from sugar and sweets accounted for about 5 percent of total calories compared to 5 percent or less recommended) (Prynne et al., 1999; WHO, 2015a). This diet was healthy enough so that children were not iron, vitamin A or C deficient (Foster and Lunn, 2007a).

Food rationing began in January 1940 and restrictions were gradually lifted starting three years after WWII had ended. The first set of restrictions lifted were on bread and flour and removed on 25 July 1948, followed by biscuits, jam, and canned and dried fruits in early 1950. Sweets and sugar went off-ration in February and September 1953, respectively. Other foods, such as bacon and other meats, butter, rice and cereals went off ration in July 1954.

To get an idea of whether the rationing was binding, we examine data the National Food Survey or NFS (NFS, 2011). The NFS was a national survey of about 10,000 households

collected quarterly from 1940 through 2000 and was used to monitor diets to make sure that were healthy during rationing. It collected detailed information on weekly individual food consumption using their daily diaries. The government used the resulting data to publish quarterly estimates of average consumption of various food groups including sugar and sweets, fats, meats, vitamins, fruits and vegetables at the national level.

Figure 2 shows average daily consumption of sugar in the UK between 1950q1-1959q4, while Figure 3 describes the intake of protein, fruits, vegetables, fats, and sugar for comparison over the same period. We observe a sharp increase in average sugar and sweets consumption in contrast to at best very small changes in other foods or nutrients during the period 1950q1-1959q4. For instance, intake of protein, vegetables and fruits either remained unchanged or increased slowly and linearly over time. The change in average consumption of fat changed continuously from 10.1oz in 1953q1 to 11.2oz in 1954q1, and of dairy from 5.3 to 5.1oz – while intake of sugars sharply increased from 10.2oz in 1953q1 to 16.08oz in 1954q1 and to more than 18oz by 1954q3 per person per week. Weekly intake of fish changed from 6.3oz in 1953 to 5.7oz in 1954, and weekly intake of meat, which stopped being rationed in July 1954, was 32.08 in 1953 and remained below 35oz through 1960. During this study period, no sharp change in vitamins (e.g., C, A, D intake), total fruit or vegetable intake was observed either (NFS, 2011).

The sweets and sugar rationing ended in February and September 1953, respectively. The lifting of sugar and sweets rationing had a dramatic effect on the confectionery market – sales of sweets and chocolate increased by more than 150% within one year (Jackson, 1974). Using the data from the NFS, we observe a sharp and significant response in the per capita average consumption of sugar and sweets by about 100% right after the rationing ended (see Figure 2). Among children (age 6-12), average consumption of sugars from sweets during rationing in the early 1950s was within recommended amounts - below 4oz (115g) per week, but soon after the end of it, their consumption more than doubled; and worse dental health is reported for children after the rationing, too (Jackson, 1974; Zweiniger-Bargielowska, 2000; Jamel et al., 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999). Some other foods, such as fats, rice, cheese, meats, milk, or cereals remained rationed until July 1954. Importantly, however, no significant changes were observed, on average, in their intake after February or September 1953, or after the end of their rationing in July 1954 (see Figure 3 and more details below) (Jackson, 1974; Zweiniger-Bargielowska, 2000; Patten, 1992; Foster and Lunn, 2007b; Davey, 2004). The sharp and quick response in the consumption of sugar and sweets right after the rationing ended informs our research design, which examines whether there are differences in outcomes among individuals born within only a few years of one another – specifically, those cohorts conceived or born just before the rationing ended relative to those conceived and born

after.

3 Data

3.1 The National Food Survey (NFS)

These data were created for the purpose of diet tracking to assure that the UK nation was fit during rationing. It provides information on the quarterly aggregate estimates of weekly per person consumption (based on a 7-day diary recordings) on various food groups, including sugar and sweets, fats, meats, vitamins, fruits or vegetables, reported at the national level. Figure 2 shows average daily consumption of sugar in the UK between 1950q1-1959q4. Figure 3 describes the intake of protein, fruits, vegetables, fats, and sugar for comparison over the same period.

We observe a sharp increase in average sugar and sweets consumption in contrast to a very small, or no sharp change in other foods or nutrients during the period 1950q1-1959q4. For instance, as seen on Figure 3, intake of protein, vegetables and fruits either remained unchanged or increased slowly and linearly over time. The change in average consumption of fat changed continously from 10.1oz in 1953q1 to 11.2oz in 1954q1, and of dairy from 5.3 to 5.1oz – while intake of sugars sharply increased from 10.2oz in 1953q1 to 16.08oz in 1954q1 and to more than 18oz by 1954q3 per person per week (author's and official analyses based on NFS). Weekly intake of fish changed from 6.3oz in 1953 to 5.7oz in 1954, and weekly intake of meat, which stopped being rationed in July 1954, was 32.08 in 1953 and remained below 35oz through 1960. During this study period, no sharp change in vitamins (e.g., C, A, D intake), total fruit or vegetable intake is observed either (NFS, 2011).

3.2 The National Diet and Nutrition Survey (NDNS)

NDNS is a continuous, nationally-representative repeated cross-sectional survey in the UK that collects detailed quantitative information on diet of about 1500 individuals every two years. It is regarded as one of the most comprehensive nutrition surveys in Europe, and it is similar in its nature to the NHANES. Its fieldwork began in 2008 and is now beginning its eleventh year. The NDNS provides the only source of high quality, nationally representative UK data on the types and quantities of more than 5000 foods consumed by individuals, from which estimates of nutrient intake (by combining food consumption data with the latest analyses in the Food Standards Agency's nutrient databank) for the population are derived. The dietary assessment

method is based on the multiple-pass 24-hour dietary recall repeated on four non-consecutive days. It includes information on several socio-demographic characteristics, as well as birth year and place of birth (Wales, Scotland, England, outside the UK). In addition to several food groups (e.g., fruits, vegetables, meats, sugar sweetened beverages), this data also provides information on nutrient intake at the individual level (i.e., X grams of protein per day). Finally, for a subsample of individuals, weight, height, waist and hip circumference is measured by a nurse. We use these to obtain several measures of obesity.

While we analyze differences in the intake of several macronutrients (e.g., proteins, carbohydrates, fats) or food groups (e.g., sweets, fruits, meats, dairy), we are particularly interested in the intake of added sugars. To define added sugars in our analyses, we use the definition of free sugars from the WHO (WHO, 2015a) and The UK Scientific Advisory Committee on Nutrition (SACN) (Swan et al., 2018), which includes: all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and unsweetened fruit juices. Under this definition, lactose (the sugar in milk) when naturally present in milk and milk products, and the sugars contained within the cellular structure of foods (particularly fruits and vegetables) are excluded. That is, added sugars in our analyses are free sugars that include all added sugars in foods and drinks in whatever form including table sugar, honey, syrups, and nectars, as well as the sugars naturally present in fruit, vegetable and pulses (e.g. soy, chickpeas) that have been juiced, pureed or made into pastes. Ingredients added such as lactose and galactose are also considered to be free sugars. We also analyze differences in intake of the intrinsic sugars. Intrinsic sugar is sugar held within the cell structure of food, as in whole fruit and vegetables.

Figure 5 shows the probability density functions (pdf) and Figure 6 the cummulative density functions (cdf) obtained from raw data on several dietary outcomes of interests by birth cohorts: daily intake of total calories, intrinsic sugars, added sugars, fats, proteins and carbohydrates. For the post-rationing cohort, we observe a right shift in consumption of added sugars (Figure 5 (b)), and a slight shift to the left in consumption of intrinsic sugars (Figure 5 (c)). Similarly, cdf for intake of added sugars is significantly higher for post-rationing cohort (Figure 6 (b)), and lower for intake of fruits (Figure 6 (c)). We do not observe such between-cohort differences for daily total calorie intake, suggesting substitution across foods (e.g., sweets to fruits and vice versa). We do not observe significant differences in raw data for other foods, such as meats or dairy either.

We present summary statistics for a selected number of variables in Table 1. Our sample consists of about 3300 observations for which we observe almost all variables except those measuring added sugar intake (710 observations). The measure of added sugars is available in

the latest two NDNS survey waves only, hence the number of observations is much smaller.[1] On average, people in our sample are 57 years old, about half of them are males and born in 1955 or after. On average, adults consume about 1800 calories daily and about 51 grams of added sugars per day on average, which is about 15 grams more than the maximum recommended amount.

3.3 The English Longitudinal Survey of Aging (ELSA)

ELSA is an bi-annual panel study of a representative of the population aged 50 and over in England, both in terms of socioeconomic profile and geographic region. It spans a 16-year period between 2002-2018 (8 waves) and was designed as a sister study to the Health and Retirement Study (HRS). These data provide detailed information on economic, social, psychological, cognitive, health, biological, and genetic data, and data on health behaviors, such as physical activity, alcohol consumption, smoking as well as information on parental health, occupation and education. The age distribution of the cohort is suited for our study as disability, chronic disease and mortality emerge at a substantial rate at this age. Important for our study, this data also includes the "Life History Module" in Wave 3 (Ward et al., 2009), which collects retrospective information about all survey participants on their childhood health and socio-economic circumstances that could affect their diet or health in adulthood irrespective of food rationing. While most outcomes are self-reported, the analyses are strengthened by the inclusion of objective markers of height and weight to obtain measure of body mass index (BMI) every 4 years as well. Our analyses include participants born in the UK between 1950 and 1960.

We present summary statistics for a selected number of variables in Table 2. Adults in our sample are on average 56 years old (between 49 and 63), and about 40 percent are born after 1954. Males represent 41 percent of the sample, about a half of adults has at least some college education. A majority of them are married. About a third of the sample is considered obese (BMI$\geq$30), and about 12 percent severely obese (BMI$\geq$35). About 7 percent of our sample is diagnosed with diabetes, about a quarter with arthritis and elevated cholesterol, and about a third with high blood pressure or hypertension. About 10 percent is diagnosed with heart disease and a similar amount with asthma.

[1]Obtaining this measure from previous waves of data is in progress and as new waves of NDNS become available, we will update the time series and re-run the regressions accordingly.

4 Empirical Approach

Ideally, individuals would be randomly assigned sugar-rich diet early in life and then their diet, health and related outcomes would be observed many decades later. That is often infeasible. We approximate such setting by leveraging the end of rationing of sugar and sweets in February 1953 in the UK as a natural experiment inducing plasubily exogenous variation in the availability of sugar and sweets early in life. That is, we assume that cohorts conceived or born right before (1950-1953) and those conceived or born after the rationing ended (1955-1960) are similar, except in their exposure to sugar and sweets early in life.

We use the Regression Discontinuity Design (RDD) along with the Ordinary Least Square (OLS) approach as our preferred approaches to estimate the following regression models:

$$\log \mathrm{Y}_{it} = \alpha + \beta BornPostRation_i + \gamma f(BirthDate_i) + X_i'\delta + \theta_t + \varepsilon_{it} \tag{1}$$

where Y_{it} is the dependent variable of interest, such as individual $i's$ diet (measured in grams per day) or health (measured as an indicator variable if diagnosed with the disease) in time t (some time in adulthood). $BornPostRation_i$ is an indicator variable that takes on value 1 for individuals in-utero or born after the end of the rationing (e.g., those born between January 1955 and December 1959), and 0 otherwise (e.g., born between January 1950 and December 1953). The sweets and sugar rationing ended in September and February 1953, respectively. In the RDD approach, we define birth year as the factor determining exposure to treatment (known as the "running or forcing variable"). θ is a time fixed effect (e.g., survey year), which capture potential differences in dietary habits over time that are common across birth cohorts. Including X_{it}, the regression specification controls for a vector of socio-demographic characteristics during childhood (e.g., parental occupation, education, and participant' socio-economic status). Note that in all regressions, we exclude individuals born in 1954, as these individuals may have been exposed to sugar and sweets rationing at least to a certain extent (e.g., in utero in 1953). Intuitively, we can think of the coefficient of interest, β, as being estimated from local regressions using data points within a given bandwidth around the cut-off. β is positive if early-life exposure to rationing increased intake of added sugars later in life.

We assume that individuals born closest to the cut-off are the most exchangeable with respect to both measured and unmeasured confounders, and that exchangeability likely decreases with distance from the cut-off. We incorporate this in our analysis by placing the greatest weight on observations closest to the cut-off (e.g., by triangular kernel weighting). Identification in this

model comes from assuming that the underlying, potentially endogenous relationship between ϵ_{it} and $BirthDate_i$ is fully captured by the flexible function $f(BirthDate_i)$, and that the relationship does not change around 1954 discontinuously. If this assumption holds, our estimate will be consistent, and can therefore be interpreted as the causal impact of early exposure to sugar-rich food environments on long-term dietary habits (Moscoe et al., 2015; Imbens and Lemieux, 2008).

We complement the RDD approach with the OLS approach to improve precision in our estimates, as the RDD estimates are local and evaluate the changes in outcomes within a narrow window of the cut-off (i.e., LATE) and the number of observations in survey data is small. We estimate the following regression model:

$$\log \mathrm{Y}_{it} = \alpha + \beta BornPostRation_i + \gamma Dev1954_i + Dev1954_i * BornPostRation_i + X_i'\delta + \theta_t + \varepsilon_{it} \quad (2)$$

In addition to the variables describedin the equation 1 above, we add the variable $Dev1954_i$ to this model, which is the difference between i's birth year and 1954 to control for trends of birth cohort born before and after 1954, as well as adjust for secular trends and seasonality that might influence especially diet by including survey year indicators, and to control for an uneven distribution of years before and after the cut-off. We cluster standard errors at the individual level.

We estimate equations 1 and 2 separately for each dietary outcome Y_{it}, for individual i at time t. These are continuous log-transformed measures of grams of added sugars eaten per day, total calories per day, daily intake of total grams of fiber,intrinsic sugars, meats, protein, fats, carbohydrates, as well as grams of sweets, fruits, vegetables red meats, poultry or dairy; all recorded for 4 non-consecutive days. Sweets include sugars, preserves, sweet spreads, and chokolate and sugar confectionery.

We repeat this estimation for several health outcomes at the individual level as well. Data we use include objectively measured waist and hip circumference (in cm), weight (in kg) and height (in cm) and BMI. Our continuous outcome variables are log-transformed waist circumference and BMI measures. We also define several indicators of obesity, such as an indicator variable for obesity based on BMI (obesity with BMI$\geq$30 or severe obesity with BMI$\geq$35); abdominal obesity (waistline larger than 102/88 cm for men/women); obesity based on waist-to-hip ratio (obesity if ratio is more than 0.95). We also examine other self-reported indicators for whether or not one has ever been diagnosed with diabetes, heart disease, stroke, arthritis, cancer, and elevated blood pressure or cholesterol. We also create an indicator variable for being diagnosed with at least one, two or three or more of these diet-related chronic diseases. When studying

chronic conditions, we include age indicators in our regressions to ensure that we are comparing individuals between cohorts within the same age and address possible age-driven discontinuities in adult health.

5 Results

Based on our RDD and OLS regression analyses, we find that cohorts exposed to rationing consume less added sugars and sweets in their later adulthood compared to cohorts that were not. Specifically, relative to an adult born pre-1954, those born post-1954 consume between 15 and 20 grams of added sugars (about 60 calories) per day more; equvivalent to about the amount of added sugar present in a half of an 8-ounces soda can (see Table 3, column (1)). Those born post-1954 also consume significantly less intrinsic sugar, and fiber (see Table 3, columns (2-3)). Despite the differences in added sugar intake, we find that total calorie intake between cohorts is not statistically significantly different between cohorts, suggesting substitution across foods (e.g., added sugars for sugars naturally present in fruits). We observe no statistically significant difference in consumption of carbohydrates, proteins or fats (see Table 3, columns (4-7)).

We also look at the cohort differences in consumption of food groups (see Table 4). We see that cohorts exposed to rationing consume significantly less sweets, but more fruits; on average, equvivalent to about one apple a day more. We observe no difference across cohorts in consumption of vegetables, red meats, poultry or dairy.

These diet effects are economically meaningful and seem to translate into differences in health outcomes in later adulthood (see Tables 5, 6, and 7). Table 5 shows that more than fice decades after the rationing, cohorts born after 1954 are on average more likely to have obesity, as measured with either body mass index (BMI, obesity if BMI $\leq$30), waist circumference (obesity if $\leq$102cm for men or 80 cm for women), or waist-to-hip ratio (WHR, obesity if $\leq$0.95). For instance, cohorts born after 1954 are more likely to have bigger waistlines by 3.4 percent, and have obesity between 12 and 20 percent, depending on the obesity measure. They are 5.5 percentage points more likely to be severely obese (BMI$\leq$35). Our results show that adults born after 1954 also suffer from more chronic conditions (see Table 6). They are more likely, on average, to be diagnosed with diabetes, high cholesterol, arthritis, or have at least one of these diet-related chronic disease by about 4, 6, 12, and 8 percentage points, respectively. We do not find statistically significant difference for diseases such as hypertension, heart disease or cancer.

We also find some preliminary evidence that cohorts born after the rationing have worse economic outcomes compared to the cohorts born during the rationing (see Table 8). Specifi-

cally, we find that on average, they are less likely to attend high school or college (by about 10 percent), be married (by about 7 percent), and are more likely to have above median wealth.

Figure 9 is a visual representation of the RDD regressions for several outcomes. Consistent with the regresion results, there is no significant jump at the cut-off for daily intake of total calories, fats, or proteins, but a significant one for added sugars. Similarly, we observe a significant jump at the cut-off birth year for outcomes, such as diabetes, cholesterol, arthritis and for being diagnosed with at least three chronic diseases, and for economic outcomes like education, marital status and wealth.

6 Robustness checks

There are two key threats to the validity of our empirical approach. These are related to the following identifying assumptions: 1) factors affecting diet or health outcomes must not change sharply for birth cohorts born around the cut-off (February 1953), and 2) no other event than the end of rationing is causing any observed cohort differences in outcomes of today's older adults. We carry out a number of falsification tests, designed to test whether our identifying assumptions are valid.

First, we check whether individuals conceived or born around the cut-off date are balanced with respect to independent outcome predictors (e.g. SES, parental education, occupation and parental health during childhood), so their outcome probabilities would be the same conditional on the same exposure to rationing. Table 9 addressess this concern by showing that differences in means across several potential confounders are not statistically significantly different from zero. Childhood SES score at age 10 was statisticaly significanly worse among individuals born between 1955-1960, however the difference is not economically meaningful. Additionally, we include early childhood circumstances and parental health outcomes as controls in our regressions - findings remain nearly unchanged (see Tables 15 and 16.

Second, we examine changes in dietary and health outcomes for people born several years before rationing ended, using earlier cut-off dates. If, for instance, some other unobserved events (either before or after the end of rationing) are driving our results, similar effects may also appear for these older cohorts. Tables 11 and 10 show that cohort differences in diet and health outcomes are not statistically significantly different at other cut-off years.

We also repeat the analyses for diseases whose incidence should be unrelated to diet in general and sugar consumption in particular (e.g. Type 1 diabetes, Parkinson disease, cataract). Given our empirical strategy, exposure to rationing should not affect these placebo outcomes. We do not observe any statistically significant difference in such diseases between cohorts (see

Table 12).

Third, we may be concerned about mechanical age-driven discontinuities in adults' health outcomes. This would be a concern if all of the outcome data were from a single year, and the incidence of certain chronic illnesses discontinuously increasesed with age. To address this concern, we include age indicators in our regressions to compare individuals within the same age between cohorts. We can do that as we observe each individual repeatedly at various ages in our longitudinal surveys. Common age support between cohorts is visually presented in Figure 10 and shows that adults in our regression models using ELSA are between 51 and 61 years old. Including or excluding these indicators does not change our findings.

While we observe today's older adults' diet, born before or after the rationing ends, we do not observe differences in their diet composition during childhood around the time when the rationing ended. We address this limitation by providing evidence on sharp increases in consumption of sugar and sweets (in contrast to other foods and vitamins) in late 1953 (see Figure 2), and provide evidence from published studies showing associations between significant increase in intake of sweets and worse dental health among children after the rationing has ended (Jackson, 1974; Zweiniger-Bargielowska, 2000; Jamel et al., 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999).

Finally, there are limitations in the fact that, even if the rationing ended, sugar and sweets may not be available immediately and evenly across the country (e.g., in rural areas). We address this concern in part by applying some "donut" regressions - excluding birth cohorts in years around the cut-off, for instance – to establish the robustness of our findings. When excluding those birth cohorts, however, results remain nearly unchanged.

[Note: in progress - multiple hypotheses adjustment]

7 Conclusion

Nearly all American infants and toddlers consume added sugar daily, and exceed its daily adult limit by the time they are two years of age. Although put forth as a key risk factor for obesity and related disease, rigorous evidence on the long-term health impacts of such early exposure to sugar-rich diet is limited and the pathways through which early nutrition may have lasting effects are poorly understood. In this paper, we study long-term effects of exposure to sugar-rich diet in early childhood on diet and health in later adulthood.

We exploit the end of the rationing of sugar and sweets in 1953 in the United Kingdom as a natural experiment inducing variation in exposure to sugar-rich diet in early childhood.

Using a RDD along with the OLS, we assume that cohorts born right before (1950-1953) and after the rationing ended (1955-1960) are similar, except in their exposure to sugar and sweets early in life. The research design is informed by the sharp increase in consumption of sugary foods immediately after the rationing ended. We observe diets and health among today's older adults in the Dietary and Nutrition Survey and the English Longitudinal Study of Ageing.

More than five decades later, cohorts born before the rationing ended consume less added sugars (about 15 grams or 60 calories from added sugars daily), more fiber, fruits and vegetables, compared to cohorts born soon after the rationing ended. We observe no difference in total calorie intake, however, suggesting substitution across foods (e.g., fruits vs sweets). We also do not find a statistically significant difference across cohorts in intake of protein, fats or carbohydrates. These diet effects are economically meaningful and seem to translate into differences in health outcomes in later adulthood. We find that more than five decades after the rationing, cohorts born after 1954 have bigger waistlines and higher BMI, and are more likely to be severely or abdominally obese. We also find that those adults are more likely to be diagnosed with chronic conditions, such as diabetes, high cholesterol, arthritis, or with at least one these diet-related chronic diseases. These health differences exist despite the fact that on average, total daily calorie intake is the same across cohorts - but their diet composition differs in how much intrinsic or added sugars they eat.

The extent to which policies aimed at improving early life conditions can improve long-term health and economic well-being is of great interest (Hoynes et al., 2016), as a better understanding of these linkages is critical for more targeted intervention strategies against obesity and related disease. This study provides important evidence on the effects of early exposure to sugar-rich diet and its impact on eating patterns and dietary habit formation, as well as on adult health using rarely-available plausibly exogenous assignment to a different diet as a result of sugar and sweets rationing. It, at a minimum, informs the design of early childhood obesity prevention efforts, as well as the dietary guidelines for children below age of two. Today's dietary guidelines on added sugar intake are based on short-term clinical trials and reviews of data largely coming from population-based observational studies on the associations between intake of free sugars and dental caries (55 out of 68 in the WHO report), and not unhealthy weight or related chronic-disease long-term (Foster and Lunn, 2007b; Moynihan, 2016; Moynihan and Kelly, 2014). In fact, most are limited to SSBs intake alone - even though added sugars in other foods in the US represent nearly 40% of daily calories (Yang et al., 2014). Finally, there are currently no guidelines for daily recommended limits for added sugar for children under the age of two - as evidence on their impact is lacking - but the upcoming 2020-2025 edition of the Dietary Guidelines for Americans is set to include them. As diets

during post-war rationing in our setting were similar to dietary guidelines for children and adults between ages 2 and 18 today, our results may serve as one of the first references on the long-term effect of dietary guidelines in general and especially children below age of 2.

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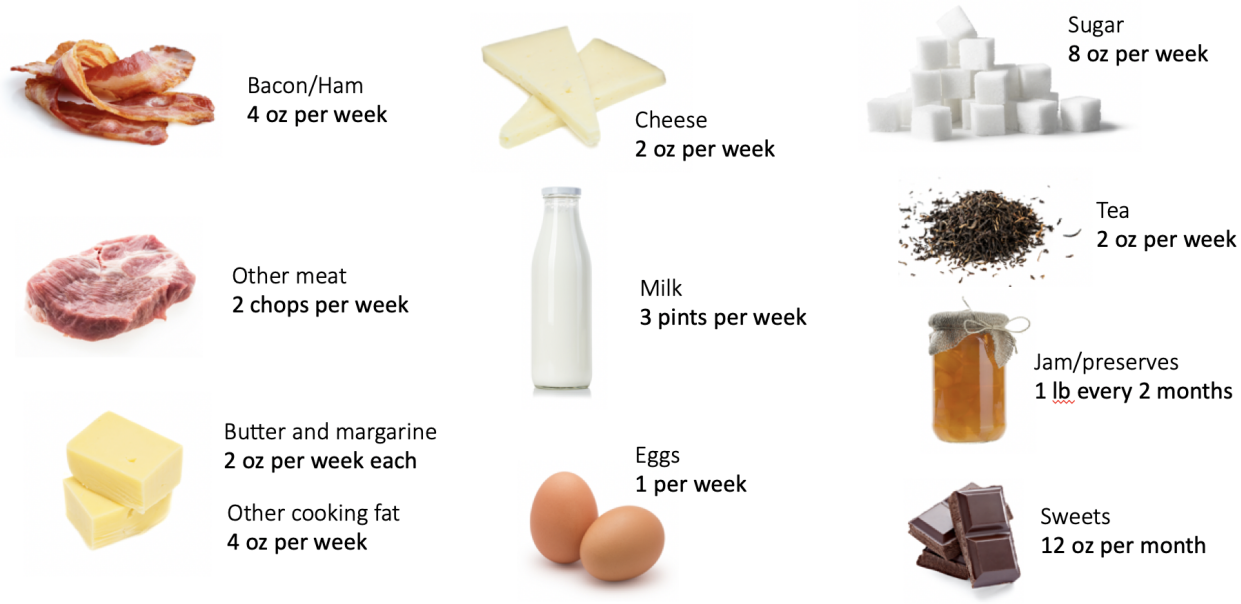
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8 Results: Figures

Figure 1: Food ration limits during 1950-1953

(a) Rations 1950-1953

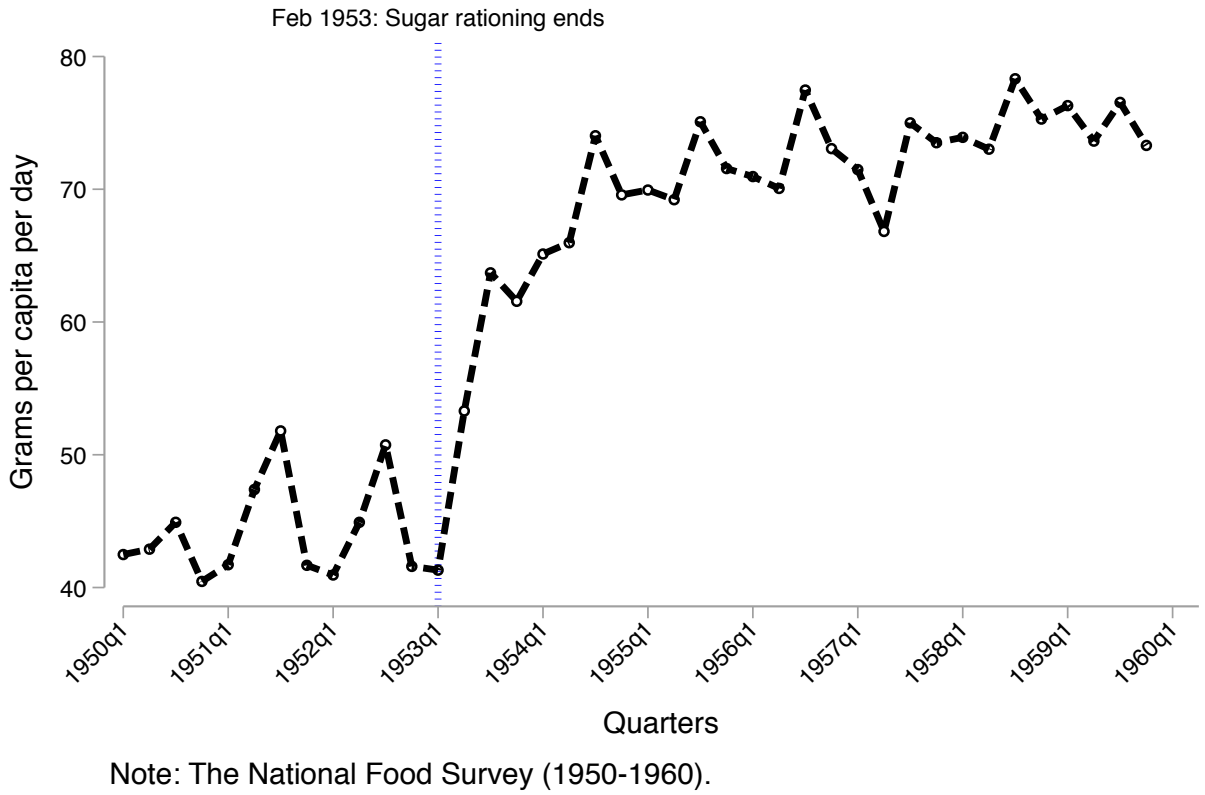


(b) Daily intake 1950-1953

	Estimated daily consumption in the UK, 1950–1953	Today's recommended consumption
Calories	2,500 kcal	1,800–3,000 kcal
Fat	35%	20–35%
Protein	13%	10–30%
Carbohydrates	52%	45–55%
Sugar	**6.5 % (40 g)**	**<10% (strict) or <5 % (cond.)**

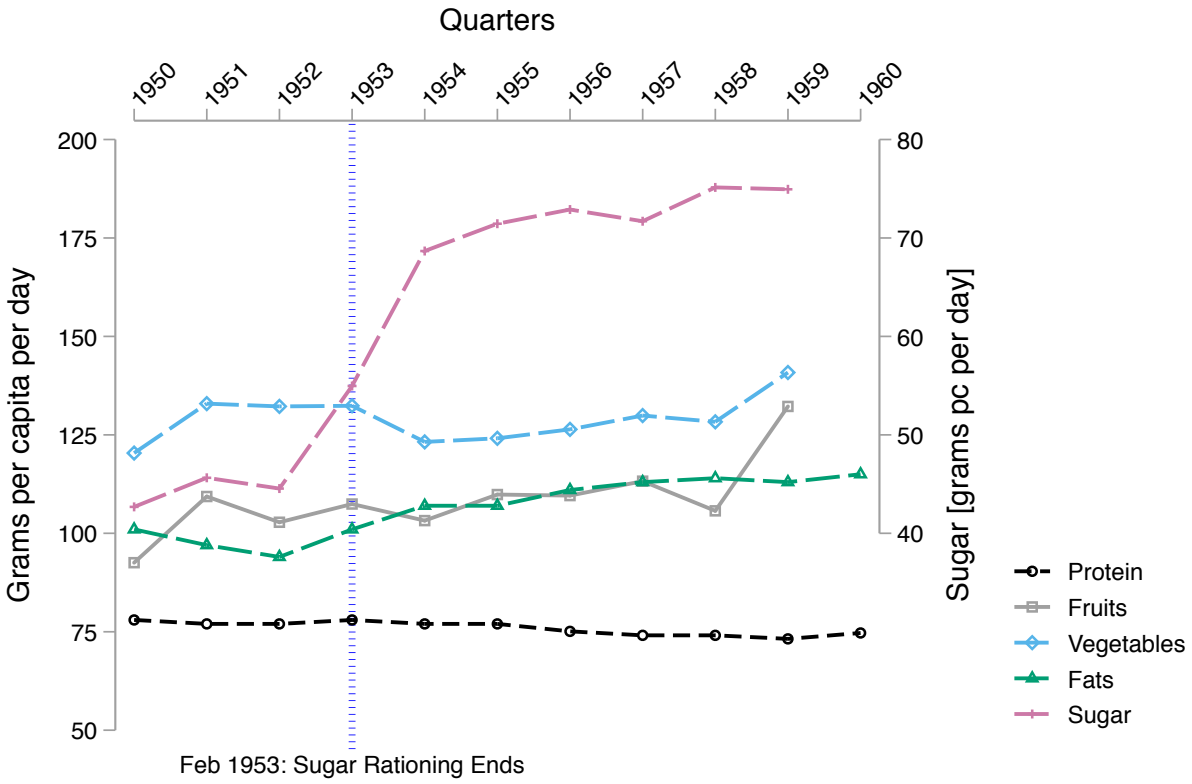
Note: Department of Environment, Food and Rural Affairs, National food survey. 2011; Dietary guidelines 2015-2020. Rationed portions were similar to today's dietary guidelines.

Figure 2: Grams of Daily Intake of Sugars per Person



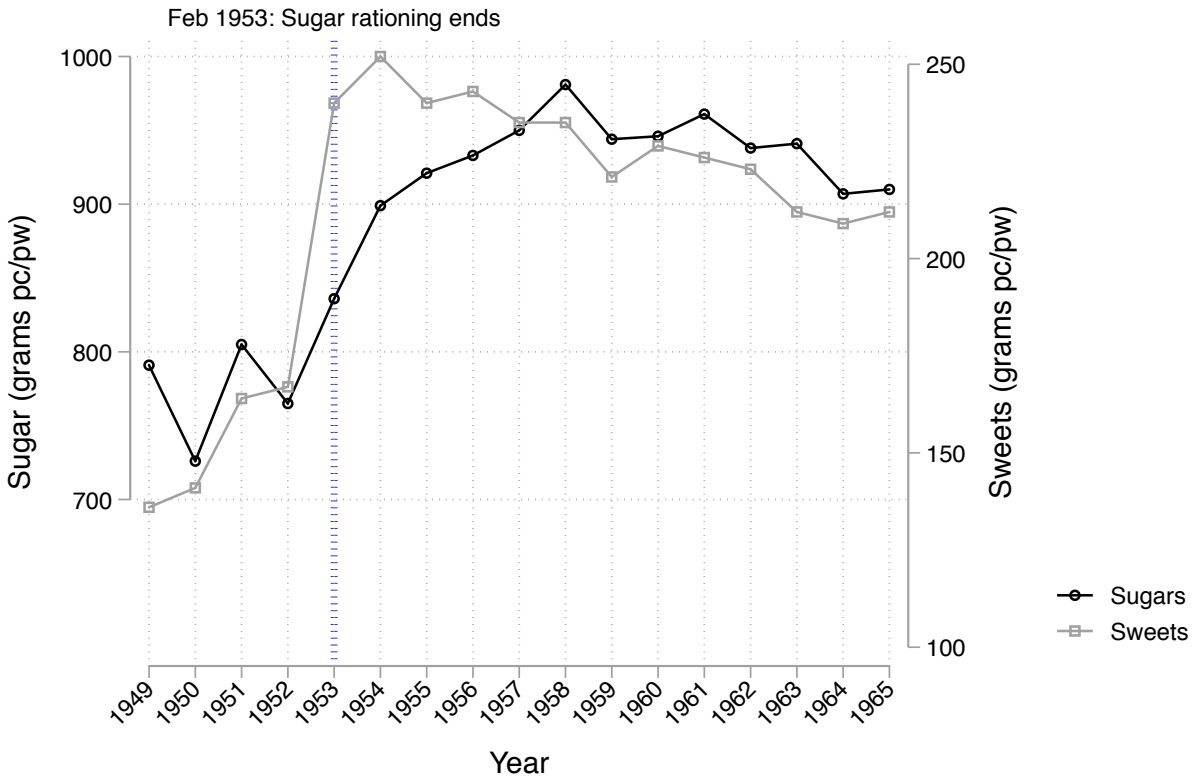
Note: National Food Survey, data based on household level food dairies. Estimated average intake of food per day (in grams).

Figure 3: Fruits, Vegetables and Meat Intake per Person per Day (in grams)



Note: National Food Survey, data based on household level food dairies. Estimated average intake of food per day (in grams).

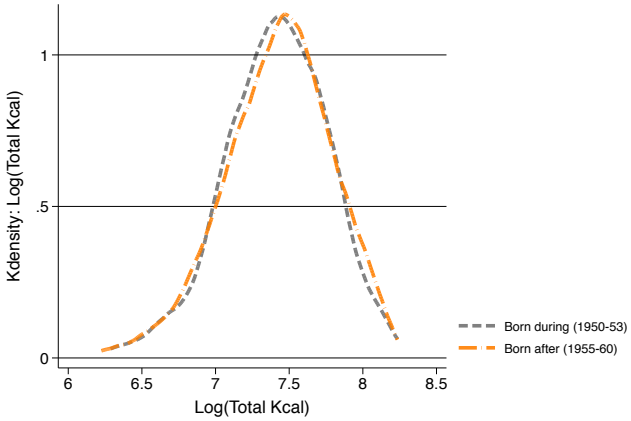
Figure 4: Estimated sales of sugar and sweets per person sold per Week



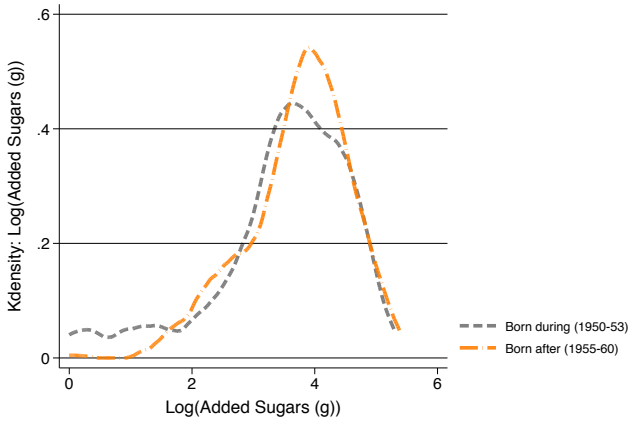
Note: *Caries experience in English children and young adults during the years 1947-1972 (British Dental Journal, 91-98, August 1974.* Data on Sweets sales supploed by the Cocoa Alliance. Data on Sugar sales supplied by the Board of Trade Journal, and Trade and Industry.

Figure 5: Cohort differences in the distribution of daily dietary intake

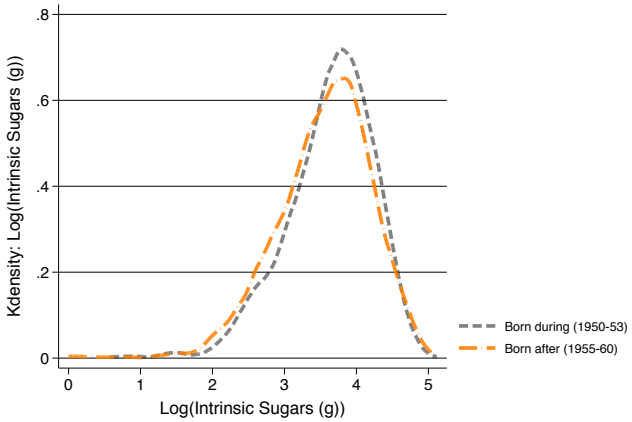
(a) Log(Total Kcal)



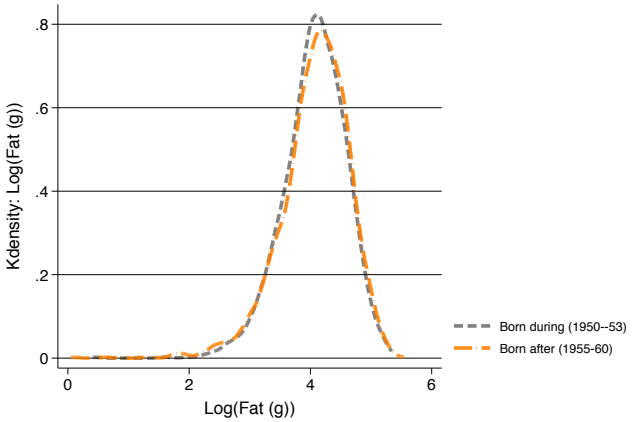
(b) Log(Added Sugars)



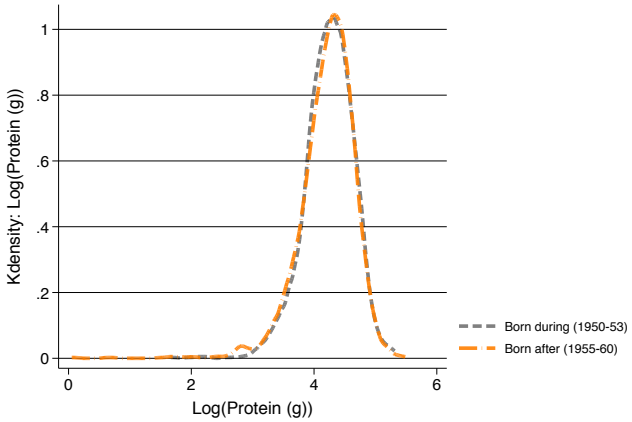
(c) Log(Intrinsic Sugars)



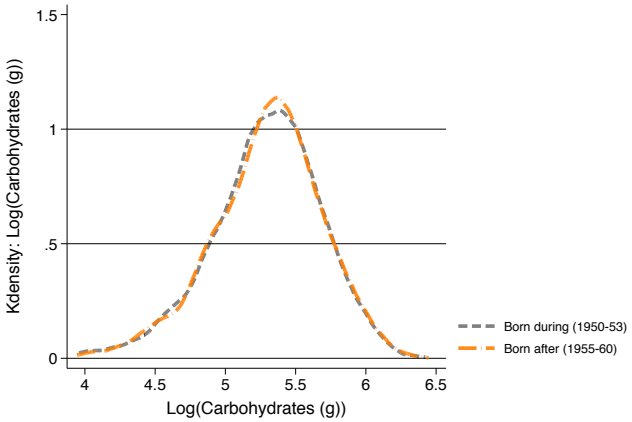
(d) Log(Fats)



(e) Log(Protein)



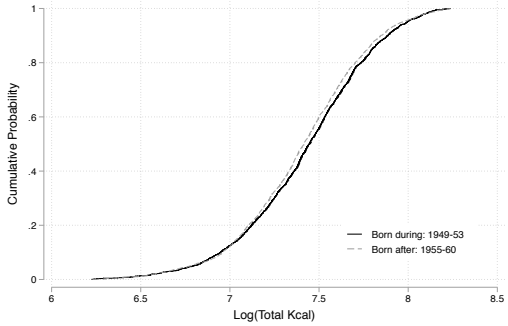
(f) Log(Carbohydrates)



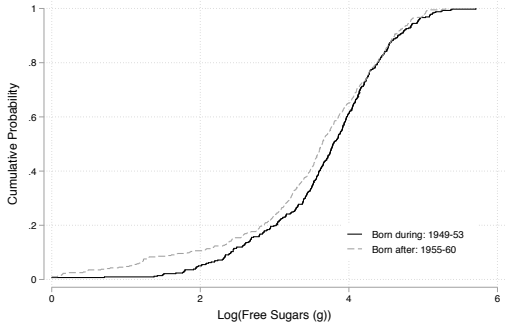
Note: Kernel density for daily total calorie intake and the intake of added sugars, intrinsic sugars present mostly in whole foods and vegetables, fats, proteins and carbohydrates in grams per day between adults born in (1950-1953) and (1955-1960).

Figure 6: Cummulative density function across foods by birth cohorts

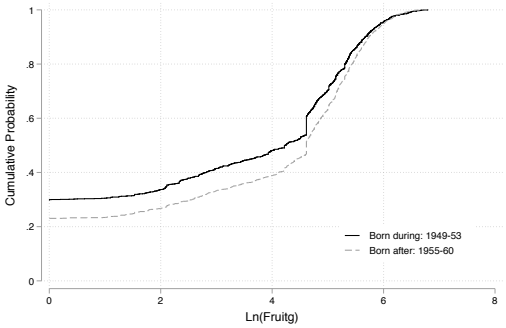
(a) Log(Total Kcal)



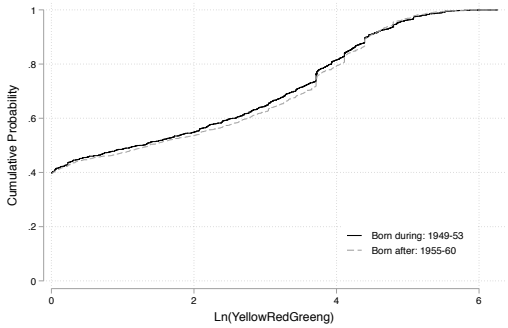
(b) Log(Added sugars)



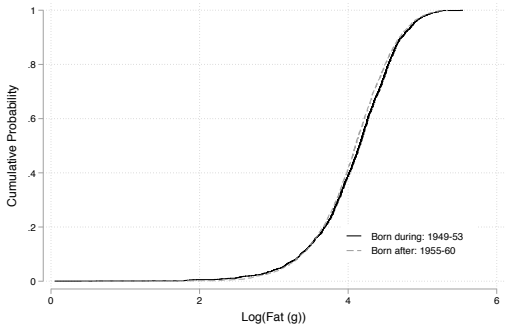
(c) Log(Fruits)



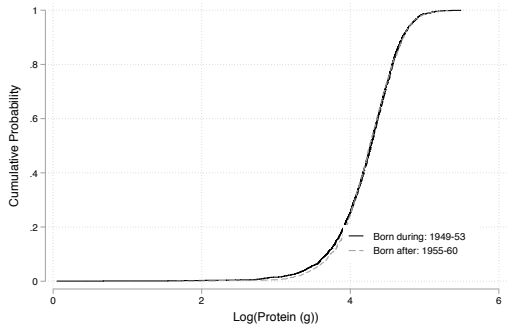
(d) Log(Vegetables)



(e) Log(Fats)

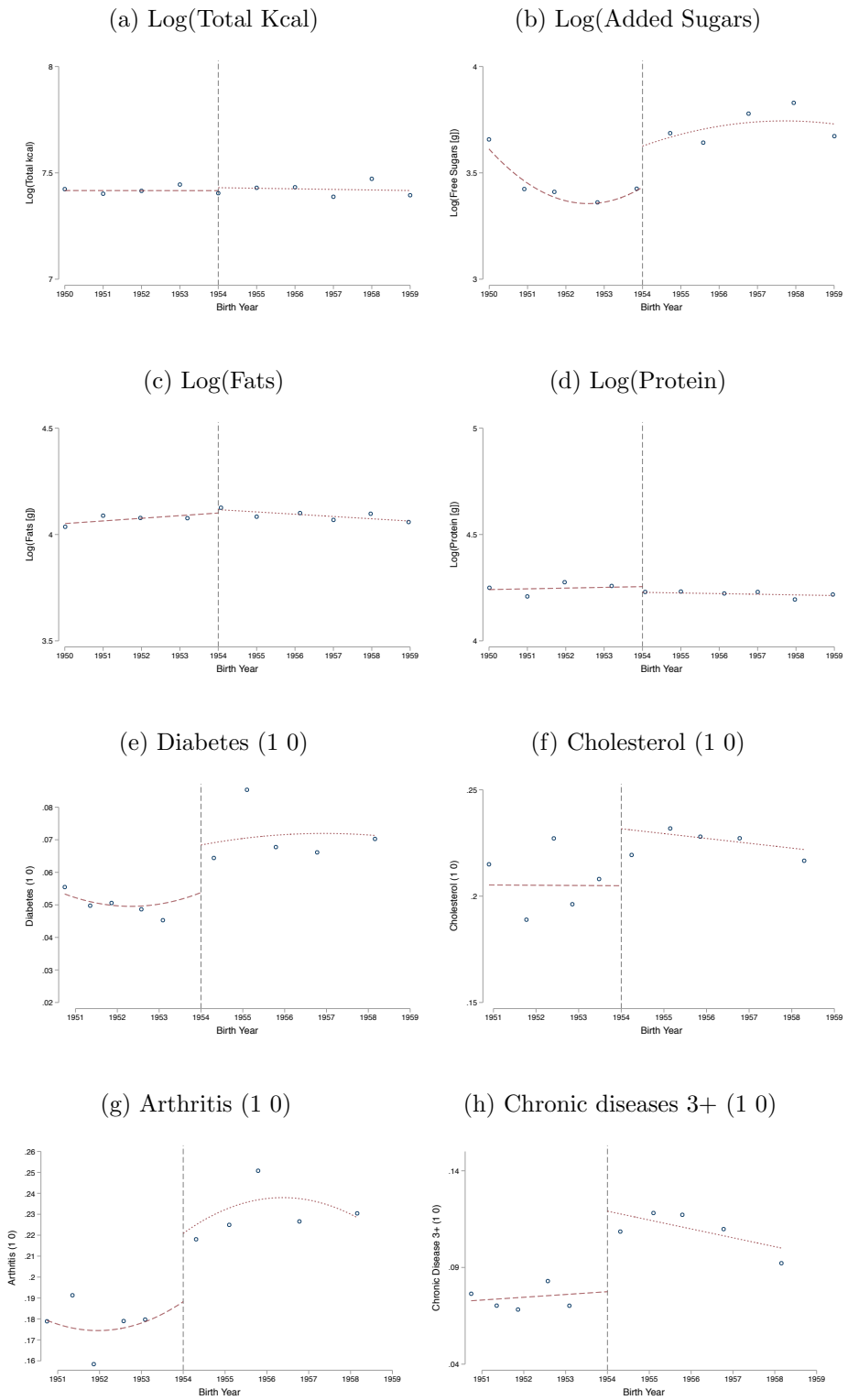


(f) Log(Protein)



Note: Cummulative density function across various foods by birth cohorts (1950-1953) and (1955-1960). All dietary intake is express in logharitm of grams per day. Kolmogorov-Smirnov test show that cdfs for added sugars in panel B and fruits in panel C are statistically significanlty different between cohorts.

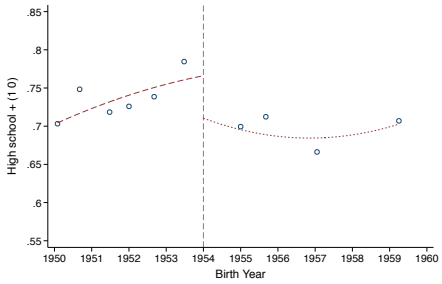
Figure 7: Cohort differences in diet and health outcomes among today's older adults



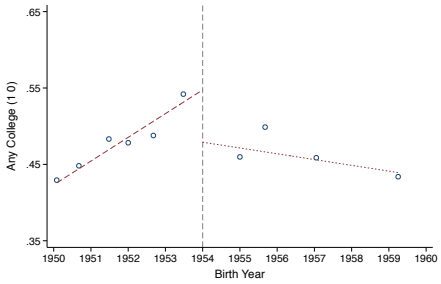
Note: Cohort difference in diet and probability of being diagnosed with diet-related disease.

Figure 8: Cohort differences in economic outcomes among today's older adults (1)

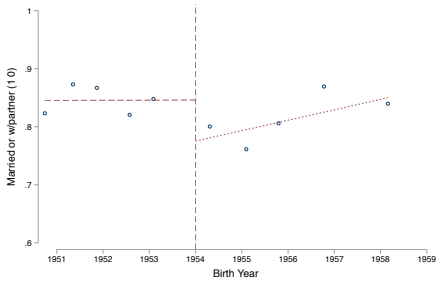
(a) High school or more



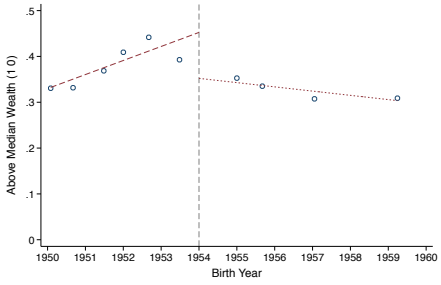
(b) At least some college



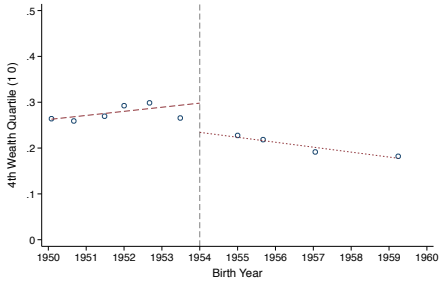
(c) Married



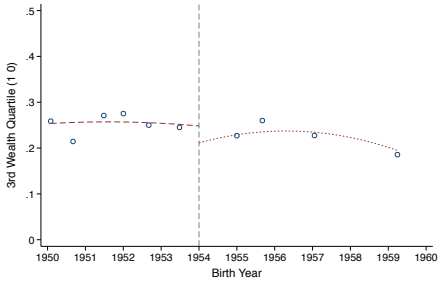
(d) More than median wealth



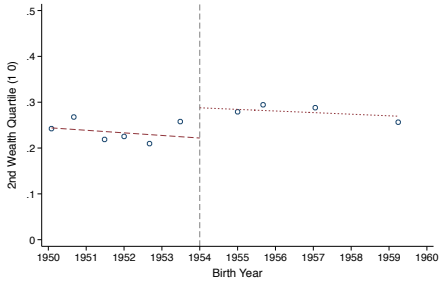
(e) 4th Wealth quartile



(f) 3rd Wealth quartile



(g) 2nd Wealth quartile



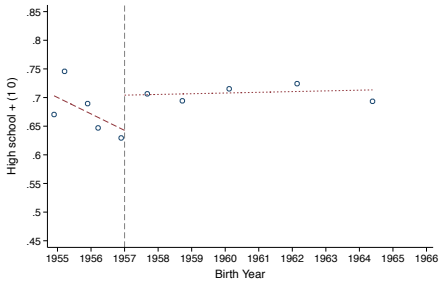
(h) 1st Wealth quartile



Note: Cohort differences in economic outcomes.

Figure 9: Cohort differences in economic outcomes among today's older adults (2)

(a) High school or more - 1957 reform



Note: Cohort differences in economic outcomes.

Figure 10: Overlapping ages between cohorts: ELSA

	Birth year (common support: ages 49–62)										
Survey year	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960
2002	52	51	50	49	48	47	46	45	44	43	42
2004	54	53	52	51	50	49	48	47	46	45	44
2006	56	55	54	53	52	51	50	49	48	47	46
2008	58	57	56	55	54	53	52	51	50	49	48
2010	60	59	58	57	56	55	54	53	52	51	50
2012	62	61	60	59	58	57	56	55	54	53	52
2014	64	63	62	61	60	59	58	57	56	55	54
2016	66	65	64	62	62	61	60	59	58	57	56

Note: The English Longitudinal Survey of Aging, 2002-2016.

9 Results: Tables

Table 1: Summary Statistics for Adults Born in 1950-1960.

	N	Mean	SD	Min	Max
Born After 1955	3379	0.52	0.50	0	1
Age	3379	57.15	4.03	49	66
Sex 1-M	3379	0.44	0.50	0	1
Married	2299	0.60	0.49	0	1
Age left school: 14 and under	3363	0.02	0.15	0	1
Age left school: 15-18	3363	0.73	0.45	0	1
Age left school: 19+	3363	0.25	0.43	0	1
Total Kcal	3320	1768.26	610.98	504	3773
Fat (g)	3379	65.70	32.87	0	253
Protein (g)	3379	73.24	28.54	0	240
Carbohydrates (g)	3379	211.69	81.60	0	630
Free Sugars (g)	710	51.40	39.51	0	217
Intrinsic Sugars (g)	3379	42.05	24.14	0	166
Fiber (g)	3379	14.32	6.58	0	49
Sweets (g)	3379	19.55	31.04	0	575
Fruits (g)	3379	121.83	141.23	0	890
Vegetables (g)	3379	28.63	49.15	0	519

Note: The National Diet and Nutrition Survey (2008-2016).

Table 2: Summary Statistics for Adults Born in 1950-1960.

	N	Mean	SD	Min	Max
Born After 1954	12352	0.38	0.48	0	1
Age yrs	12352	56.06	3.75	49	63
Sex - 1 M	12352	0.41	0.49	0	1
At least some college	12352	0.47	0.50	0	1
Married or with partner	12352	0.81	0.39	0	1
Obese	4332	0.34	0.47	0	1
Severly obese	4332	0.12	0.33	0	1
Ever smoked	12352	0.60	0.49	0	1
Active weekly	12352	0.69	0.46	0	1
Diabetes	12352	0.07	0.25	0	1
Arthritis	12352	0.24	0.43	0	1
Cholesterol	11386	0.26	0.44	0	1
Heart disease	12352	0.12	0.32	0	1
Cancer	12352	0.05	0.22	0	1
Hypertension	12352	0.30	0.46	0	1
Stroke	12352	0.01	0.12	0	1
Diet-related chronic disease 2+	12352	0.29	0.45	0	1
Lung disease	12352	0.04	0.19	0	1
Asthma	12352	0.13	0.34	0	1
Cataract	12352	0.05	0.21	0	1

Note: The English Longitudinal Survey of Aging (2002-2016).
Data is limited on adults born between 1950-1960 (ages 49-63).

Table 3: Cohort Differences in Diet: RD and OLS

Log(DV)	**Added Sugars** **(1)**	**Intrinsic Sugars** **(2)**	**Fiber** **(3)**	**Total Calories** **(4)**	**Protein** **(5)**	**Fats** **(6)**	**Carbohydrates** **(7)**
Panel A: RD Estimate							
RD_Estimate	0.407*	-0.112**	-0.069**	-0.032	0.031	-0.056	-0.006
	(0.223)	(0.057)	(0.035)	(0.037)	(0.029)	(0.034)	(0.027)
Observations	5409	27266	27266	27266	27266	27266	27266
Panel B: OLS Estimate							
Born After 1955	0.439*	-0.198***	-0.128**	-0.021	0.026	-0.029	-0.025
	(0.234)	(0.075)	(0.050)	(0.047)	(0.035)	(0.034)	(0.030)
N	703	3320	3320	3320	3320	3320	3320
Y(=0) Mean (g/day)	48.47	43.30	14.53	1754.59	64.83	73.91	211.47

Note: Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. In all regressions, adults born in 1954 are excluded. Study period for the RD regressions includes all adults born between 1930 and 2000. All regressions except (4) control for total calorie intake per day. OLS regressions also include survey wave fixed effects.

Table 4: Cohort Differences in Diet: RD and OLS

Log(DV)	**Sweets (1)**	**Fruits (2)**	**Vegetables (3)**	**Red Meats (4)**	**Poultry (5)**	**Dairy (6)**
Panel A: RD Estimate						
RD_Estimate	0.371*	-0.599***	0.027	0.087	-0.032	-0.079
	(0.219)	(0.231)	(0.185)	(0.163)	(0.139)	(0.183)
Observations	27266	27266	27266	27266	27266	27266
Panel B: OLS Estimate						
Born After 1955	0.403*	-0.930***	-0.061	0.127	-0.038	-0.061
	(0.229)	(0.283)	(0.198)	(0.183)	(0.192)	(0.215)
N	2955	3320	3320	3320	3320	3320
Y(=0) Mean (g/day)	20.34	131.35	29.06	18.27	27.68	213.05

Note: Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. In all regressions, adults born in 1954 are excluded. Study period for the RD regressions includes all adults born between 1930 and 2000. All regressions control for total calorie intake per day. OLS regressions also include survey wave fixed effects.

Table 5: Cohort Differences in Obesity (1)

	NDNS						ELSA		
DV:	**Log(BMI)**	**Log(Waist)**	**Obese Any**	**Obese BMI≥30**	**Obese Waist≥ 102(88)**	**Obese WHR>0.95**	**Log(BMI) All**	**Log(BMI) if BMI≥30**	**Sev.Obese BMI≥35**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**	**(9)**
Panel A: RDD									
Born After 1954	0.026	0.034**	0.113**	0.055	0.108**	0.136***	0.016	0.037*	0.055*
	(0.017)	(0.015)	(0.045)	(0.045)	(0.051)	(0.051)	(0.019)	(0.019)	(0.030)
Panel B: OLS									
Born After 1954	0.021	0.035**	0.086*	0.043	0.090*	0.117**	0.021	0.042**	0.061**
	(0.018)	(0.016)	(0.049)	(0.048)	(0.054)	(0.054)	(0.019)	(0.020)	(0.031)
N	2263	1874	2343	2263	1874	1872	4976	1695	4976
Y(=0) Mean	28.51	96.51	0.60	0.34	0.53	0.53	28.41	34.51	0.11

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Obesity - any equals 1 if an individual is obese as defined by BMI, waist circumference or waist-to-hip ratio.

Table 6: Cohort Differences in Chronic Conditions (2)

DV: 1 0	**Diabetes (1)**	**Cholesterol (2)**	**Arthritis (3)**	**Hypertension (4)**	**Heart (5)**	**Chronic 1+ (6)**	**Chronic 2+ (7)**	**Chronic 3+ (8)**
Panel A: RDD								
Born After 1954	0.032*	0.064*	0.118***	0.026	0.025	0.077**	0.108***	0.067***
	(0.019)	(0.034)	(0.033)	(0.037)	(0.026)	(0.037)	(0.033)	(0.023)
Panel B: OLS								
Born After 1954	0.043**	0.068*	0.120***	0.027	0.025	0.080**	0.116***	0.076***
	(0.020)	(0.037)	(0.036)	(0.040)	(0.028)	(0.040)	(0.036)	(0.025)
Panel C: OLS & controls								
Born After 1954	0.042**	0.068*	0.121***	0.037	0.027	0.088**	0.117***	0.077***
	(0.020)	(0.037)	(0.035)	(0.040)	(0.028)	(0.040)	(0.036)	(0.025)
N	12344	11379	12344	12344	12344	12344	12344	12344
Y(=0) Mean	0.07	0.27	0.24	0.32	0.11	0.61	0.29	0.12

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. OLS regression is limited to those born between 1950-1960, 49-63 years old. Each OLS model includes controls for survey wave and age indicators.

Table 7: Cohort Differences in Other Conditions (3)

DV: 1 0	**Lunge Disease (1)**	**Asthma (2)**	**Parkinson (3)**	**Cataract (4)**
Panel A: RDD				
RD_Estimate	0.007	0.019	-0.002	0.010
	(0.014)	(0.029)	(0.002)	(0.015)
Panel B: OLS				
Born After 1954	0.002	0.034	0.001	0.006
	(0.016)	(0.031)	(0.002)	(0.015)
Panel C: OLS & controls				
Born After 1954	0.003	0.031	0.001	0.007
	(0.016)	(0.031)	(0.002)	(0.015)
N	12344	12344	12344	12344
Y(=0) Mean	0.04	0.13	0.00	0.05

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960, 49-63 years old. Each model includes controls for survey wave and age indicators.

Table 8: Cohort Differences in Economic Outcomes

DV: 1 0	**High School+**	**Married**	**in Wealth q1**	**in Wealth q2**	**in Wealth q3**	**in Wealth q4**	**Above Median Wealth**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**
Panel A: RDD							
RD_Estimate	-0.080**	-0.064**	0.005	0.066**	-0.009	-0.061*	-0.076**
	(0.037)	(0.032)	(0.033)	(0.031)	(0.028)	(0.033)	(0.035)
Panel B: OLS							
Born After 1954	-0.129***	-0.050	0.005	0.065**	-0.007	-0.063*	-0.089**
	(0.041)	(0.032)	(0.036)	(0.033)	(0.031)	(0.036)	(0.039)
Panel C: OLS & controls							
Born After 1954	-0.124***	-0.049	-0.005	0.068**	-0.003	-0.059*	-0.081**
	(0.039)	(0.032)	(0.033)	(0.033)	(0.030)	(0.034)	(0.036)
N	13845	13845	13578	13578	13578	13578	13578
Y(=0) Mean	0.73	0.82	0.23	0.24	0.26	0.28	0.38

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. OLS regression is limited to those born between 1950-1960, 49-63 years old. Each OLS model includes controls for survey wave and age indicators.

Table 9: Cohort differences in childhood characteristics

	Born 1950-1953		Born 1955-1960		
	Mean	Std. Dev.	Mean	Std. Dev.	Difference
Mother had diabetes	0.13	0.34	0.15	0.35	-0.02
Father had diabetes	0.13	0.33	0.14	0.34	-0.01
SES score at age 10 (0-5)	3.15	0.84	3.07	0.78	0.08***
Had bath in-house at age 10	0.82	0.39	0.80	0.40	0.02
Had toilet in-house at age 10	0.76	0.43	0.76	0.43	0.00
No or few books at age 10	0.19	0.39	0.17	0.38	0.02
N of rooms at age 10	2.96	0.52	2.98	0.43	-0.02
N of people in residence at age 10	5.00	1.13	5.00	0.92	0.00
Mother let me do the things I liked doing	0.64	0.48	0.61	0.49	0.02
Mother was emotionally cold to me	0.12	0.33	0.13	0.33	-0.00
Mother understood my worries	0.57	0.50	0.53	0.50	0.04
Mother liked me to make my own decisions	0.62	0.49	0.62	0.49	0.00
Mother was overprotective	1.01	4.07	0.88	4.24	0.13

Note: * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level.

Table 10: Other cutoffs varies

Cut-off year	**1952**	**1958**	**1975**
Log(Free Sugars)	-0.211	-0.015	0.037
	(0.253)	(0.227)	(0.200)
Log(Sweets)	-0.141	-0.024	0.114
	(0.222)	(0.240)	(0.208)
Log(Intrinsic Sugars)	-0.023	0.031	-0.017
	(0.081)	(0.088)	(0.078)
Log(Fiber)	0.047	0.069	0.038
	(0.052)	(0.058)	(0.050)
Log(Total Kcal)	0.003	0.082	0.033
	(0.049)	(0.050)	(0.050)
Log(Fats)	-0.007	0.018	-0.029
	(0.040)	(0.038)	(0.032)
Log(Protein)	0.053	-0.036	-0.001
	(0.036)	(0.034)	(0.040)
Log(Carbs)	-0.011	0.007	0.009
	(0.029)	(0.029)	(0.027)

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regressions are limited to those born between 1950-1960. OLS regressions also control for survey wave.

Table 11: Other cutoffs: health in adulthood

Cut-off year :	**1952**	**1958**
	(1)	**(2)**
Health		
Sev.Obese	-0.008	0.013
	(0.025)	(0.056)
Diabetes	-0.003	0.000
	(0.017)	(0.028)
Arthritis	-0.009	-0.017
	(0.028)	(0.051)
Cholesterol	-0.045	-0.012
	(0.030)	(0.047)
Chr.Diseases 3+	-0.000	-0.007
	(0.019)	(0.032)

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regressions are limited to those born between 1950-1960. OLS regressions also control for survey wave.

Table 12: Cohort Differences in Health Outcomes (2)

DV: 1 0	**Lung Disease (1)**	**Asthma (2)**	**Mental Health (3)**	**Parkinson's (4)**	**Cataract (5)**
Panel A: RD Estimate					
RD_Estimate	0.009	0.022	0.044	-0.004*	0.006
	(0.015)	(0.030)	(0.029)	(0.002)	(0.014)
Observations	9097	9097	9097	9097	9096
Panel B: OLS Estimate					
Born After 1954	0.002	0.034	0.029	0.001	0.006
	(0.016)	(0.031)	(0.030)	(0.002)	(0.015)
Dev1954	0.004	-0.027	-0.028*	-0.005*	-0.006
	(0.008)	(0.017)	(0.017)	(0.003)	(0.008)
(Dev1954) x (Born After 1954)	-0.004	0.014	0.020	0.003	0.003
	(0.008)	(0.016)	(0.016)	(0.002)	(0.008)
N	12344	12344	12344	12344	12344
Y(=0) Mean if born 1950-1953	0.04	0.13	0.12	0.00	0.07

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960. Each model includes survey wave and age indicators.

Table 13: Cohort differences in health outcomes with childhood controls (1): OLS

DV: 1 0	**BMI≥30**	**BMI≥35**	**Diabetes**	**Cholesterol**	**Hypertension**	**Heart Disease**	**Arthritis**	**Cancer**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
Born After 1954	0.027	0.057*	0.042**	0.068*	0.037	0.027	0.121***	0.014
	(0.017)	(0.032)	(0.020)	(0.037)	(0.040)	(0.028)	(0.035)	(0.021)
Dev1954	-0.002	-0.011	-0.033***	-0.038*	-0.062***	-0.018	-0.032	-0.000
	(0.009)	(0.019)	(0.012)	(0.023)	(0.023)	(0.015)	(0.020)	(0.010)
(Dev1954) x (Born After 1954)	0.016*	0.018	0.010	0.009	0.042*	0.014	0.043**	-0.009
	(0.009)	(0.018)	(0.011)	(0.020)	(0.022)	(0.015)	(0.019)	(0.009)
mom_diabe_impute	0.036*	0.039	0.028	0.124***	0.075*	0.004	-0.110***	0.009
	(0.020)	(0.039)	(0.020)	(0.037)	(0.044)	(0.031)	(0.041)	(0.017)
Mother had diabetes	0.028**	0.063**	0.091***	0.051**	0.073**	0.017	0.032	-0.010
	(0.012)	(0.025)	(0.019)	(0.025)	(0.028)	(0.019)	(0.025)	(0.012)
dad_diabe_impute	-0.029	-0.022	0.007	-0.068**	0.012	0.018	0.152***	0.001
	(0.019)	(0.035)	(0.019)	(0.034)	(0.042)	(0.031)	(0.043)	(0.017)
Father had diabetes	0.020*	0.045*	0.075***	0.051*	0.027	0.050**	-0.021	-0.006
	(0.012)	(0.024)	(0.018)	(0.026)	(0.029)	(0.022)	(0.024)	(0.012)
dad_occupation==Intermediate	0.012	0.020	0.030**	0.025	0.008	-0.009	0.036	0.013
	(0.010)	(0.018)	(0.013)	(0.022)	(0.025)	(0.018)	(0.022)	(0.012)
dad_occupation==Low	0.033***	0.064***	0.030**	0.069***	0.043*	-0.013	0.041*	-0.010
	(0.011)	(0.021)	(0.013)	(0.023)	(0.026)	(0.017)	(0.023)	(0.012)
dad_occupation==Sick/Disabled	-0.006	-0.012	-0.038***	0.073	-0.068	-0.111***	0.014	0.049
	(0.043)	(0.083)	(0.015)	(0.091)	(0.094)	(0.021)	(0.101)	(0.069)
dad_occupation==Unemployed	0.046	0.096	0.054	0.113	0.128	0.005	0.051	0.012
	(0.033)	(0.083)	(0.051)	(0.081)	(0.085)	(0.057)	(0.085)	(0.045)
N	3034	4331	12344	11379	12344	12344	12344	12344
Y(=0) Mean if born 1950-1953	28.46	0.12	0.07	0.29	0.33	0.12	0.26	0.06

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960, 49-59 years old. Each model includes controls for survey wave and age indicators.

Table 14: Cohort differences in health outcomes with childhood controls (2): OLS

DV: 1 0	**Lunge** (1)	**Asthma** (2)	**Mental Health** (3)	**Parkinson** (4)	**Cataract** (5)	**Chronic1+** (6)	**Chronic2+** (7)	**Chronic3+** (8)
Born After 1954	0.003	0.031	0.027	0.001	0.007	0.088**	0.117***	0.077***
	(0.016)	(0.031)	(0.030)	(0.002)	(0.015)	(0.040)	(0.036)	(0.025)
Dev1954	0.002	-0.026	-0.026	-0.005*	-0.006	-0.049**	-0.071***	-0.051***
	(0.008)	(0.017)	(0.017)	(0.003)	(0.008)	(0.023)	(0.020)	(0.014)
(Dev1954) x (Born After 1954)	-0.001	0.017	0.019	0.003	0.002	0.036*	0.048**	0.030**
	(0.008)	(0.016)	(0.016)	(0.002)	(0.008)	(0.021)	(0.019)	(0.013)
mom_diabe_impute	0.008	-0.026	0.032	0.000	-0.016	0.055	0.012	0.047*
	(0.021)	(0.039)	(0.038)	(0.000)	(0.016)	(0.040)	(0.038)	(0.025)
Mother had diabetes	0.020	0.012	-0.043**	0.002	0.030**	0.091***	0.068***	0.067***
	(0.013)	(0.022)	(0.017)	(0.003)	(0.013)	(0.025)	(0.025)	(0.019)
dad_diabe_impute	0.007	0.055	0.019	-0.001	0.021	0.049	0.054	0.011
	(0.020)	(0.038)	(0.036)	(0.001)	(0.017)	(0.038)	(0.038)	(0.024)
Father had diabetes	0.018	0.067***	-0.029	-0.002*	0.006	0.034	0.063**	0.052***
	(0.013)	(0.025)	(0.018)	(0.001)	(0.012)	(0.027)	(0.026)	(0.019)
dad_occupation==Intermediate	0.005	-0.017	0.026	-0.003	0.011	0.012	0.045**	0.022
	(0.009)	(0.019)	(0.018)	(0.002)	(0.010)	(0.025)	(0.022)	(0.015)
dad_occupation==Low	0.011	-0.003	0.034*	-0.003	-0.003	0.071***	0.068***	0.024
	(0.010)	(0.020)	(0.019)	(0.002)	(0.010)	(0.024)	(0.022)	(0.015)
dad_occupation==Sick/Disabled	-0.027***	0.089	0.121	-0.004	0.004	-0.015	0.010	-0.056
	(0.010)	(0.100)	(0.077)	(0.003)	(0.035)	(0.100)	(0.087)	(0.037)
dad_occupation==Unemployed	-0.051***	-0.117***	-0.019	-0.003*	0.021	0.117	0.115	0.086
	(0.012)	(0.037)	(0.049)	(0.002)	(0.036)	(0.079)	(0.074)	(0.057)
N	12344	12344	12344	12344	12344	12344	12344	12344
Y(=0) Mean if born 1950-1953	0.04	0.13	0.12	0.00	0.07	0.63	0.32	0.14

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960, 49-59 years old. Each model includes controls for survey wave and age indicators.

10 Appendix

Table 15: Cohort differences in health hutcomes with childhood controls (1): OLS

DV: 1 0	**BMI≥30**	**BMI≥35**	**Diabetes**	**Cholesterol**	**Hypertension**	**Heart Disease**	**Arthritis**	**Cancer**
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**	**(8)**
Born After 1955	0.010	0.063*	0.043**	0.063*	0.049	0.021	0.111***	0.016
	(0.056)	(0.038)	(0.020)	(0.037)	(0.041)	(0.029)	(0.036)	(0.020)
Dev1954	-0.005	-0.019	-0.034***	-0.018	-0.058**	-0.004	-0.039*	0.003
	(0.032)	(0.022)	(0.012)	(0.024)	(0.024)	(0.016)	(0.020)	(0.009)
(Dev1954) x (Born After 1955)	0.026	0.013	0.005	-0.004	0.033	0.014	0.043**	-0.008
	(0.031)	(0.022)	(0.011)	(0.021)	(0.023)	(0.015)	(0.019)	(0.009)
Sex - 1 M	-0.038*	-0.047***	0.038***	0.051***	0.048***	0.021*	-0.072***	-0.025***
	(0.023)	(0.015)	(0.010)	(0.016)	(0.018)	(0.012)	(0.016)	(0.008)
Mother had diabetes	0.106***	0.076***	0.088***	0.049**	0.099***	0.031	0.051**	-0.009
	(0.035)	(0.028)	(0.020)	(0.025)	(0.030)	(0.020)	(0.026)	(0.012)
Father had diabetes	0.070**	0.031	0.066***	0.051*	0.032	0.056**	-0.027	-0.009
	(0.035)	(0.026)	(0.018)	(0.027)	(0.030)	(0.023)	(0.024)	(0.012)
dad_occupation==Intermediate	0.070**	0.025	0.030**	0.033	0.012	-0.009	0.051**	0.011
	(0.030)	(0.020)	(0.013)	(0.022)	(0.026)	(0.018)	(0.022)	(0.012)
dad_occupation==Low	0.150***	0.066***	0.027**	0.076***	0.025	-0.001	0.059***	-0.012
	(0.032)	(0.022)	(0.013)	(0.023)	(0.026)	(0.018)	(0.023)	(0.011)
dad_occupation==Sick/Disabled	0.101	0.035	-0.049***	0.097	-0.026	-0.056	0.015	0.031
	(0.105)	(0.098)	(0.015)	(0.081)	(0.090)	(0.050)	(0.096)	(0.056)
dad_occupation==Unemployed	0.097	0.080	0.041	0.060	0.117	0.018	0.028	0.022
	(0.112)	(0.090)	(0.042)	(0.072)	(0.091)	(0.063)	(0.090)	(0.050)
N	2709	2709	9096	8099	9096	9096	9097	9097
Y(=0) Mean if born 1950-1953	0.33	0.11	0.05	0.23	0.30	0.10	0.21	0.04

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960, 49-59 years old. Each model includes controls for survey wave and age indicators.

Table 16: Cohort differences in health hutcomes with childhood controls (2): OLS

DV: 1 0	**Lunge** (1)	**Asthma** (2)	**Mental Health** (3)	**Parkinson** (4)	**Cataract** (5)	**Chronic1+** (6)	**Chronic2+** (7)	**Chronic3+** (8)
Born After 1955	0.004	0.038	0.024	-0.001	0.005	0.087**	0.122***	0.078***
	(0.016)	(0.031)	(0.031)	(0.001)	(0.014)	(0.042)	(0.037)	(0.025)
Dev1954	-0.005	-0.043**	-0.018	-0.003	-0.003	-0.045*	-0.063***	-0.040***
	(0.008)	(0.018)	(0.017)	(0.003)	(0.007)	(0.024)	(0.020)	(0.015)
(Dev1954) x (Born After 1955)	0.004	0.030*	0.017	0.001	-0.001	0.032	0.040**	0.022*
	(0.008)	(0.017)	(0.016)	(0.001)	(0.008)	(0.024)	(0.019)	(0.012)
Sex - 1 M	-0.005	-0.043***	-0.035***	0.000	-0.007	0.001	0.029*	0.007
	(0.007)	(0.014)	(0.013)	(0.001)	(0.007)	(0.019)	(0.016)	(0.011)
Mother had diabetes	0.033**	0.021	-0.039**	0.003	0.021*	0.123***	0.090***	0.076***
	(0.014)	(0.024)	(0.018)	(0.004)	(0.012)	(0.027)	(0.027)	(0.020)
Father had diabetes	0.017	0.066**	-0.021	-0.002*	-0.002	0.020	0.068**	0.054***
	(0.013)	(0.026)	(0.019)	(0.001)	(0.010)	(0.030)	(0.027)	(0.020)
dad_occupation==Intermediate	0.005	-0.008	0.039**	-0.003	0.010	0.021	0.052**	0.030**
	(0.009)	(0.020)	(0.019)	(0.002)	(0.009)	(0.027)	(0.023)	(0.014)
dad_occupation==Low	0.010	0.004	0.046**	-0.003	0.001	0.074***	0.066***	0.025*
	(0.010)	(0.021)	(0.020)	(0.002)	(0.009)	(0.026)	(0.023)	(0.015)
dad_occupation==Sick/Disabled	-0.034***	0.095	0.069	-0.003	0.016	0.036	0.035	-0.036
	(0.010)	(0.099)	(0.069)	(0.003)	(0.030)	(0.091)	(0.080)	(0.033)
dad_occupation==Unemployed	-0.041***	-0.106***	0.009	-0.002	0.021	0.108	0.071	0.054
	(0.012)	(0.037)	(0.068)	(0.001)	(0.032)	(0.087)	(0.075)	(0.053)
N	9097	9097	9097	9097	9096	9097	9097	9097
Y(=0) Mean if born 1950-1953	0.03	0.13	0.11	0.00	0.03	0.57	0.24	0.09

Robust standard errors in parentheses, clustered at the individual level. * denotes significant at the 10% level, ** denotes significant at the 5% level, and *** denotes significant at the 1% level. Adults born in 1954 are excluded. Regression is limited to those born between 1950-1960, 49-59 years old. Each model includes controls for survey wave and age indicators.