

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

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Abstract

Exploiting the end of post-WWII sugar rationing in the United Kingdom, we use a regression discontinuity design to show that a sugar-rich diet early in life has large adverse effects on health, cognition, and economic well-being more than fifty years later. Early-life sugar exposure increased lifelong free sugar consumption and raised the prevalence of chronic inflammation, diabetes, high cholesterol, hypertension, arthritis, and multimorbidity. It also lowered childhood cognitive performance, schooling, skilled employment, and wealth accumulation. The rationed UK diet closely resembled today's USDA and WHO guidelines, suggesting that limiting free sugar early in life could yield large long-term health and economic benefits. Our findings support policies regulating sugar content and marketing in foods consumed by infants and toddlers.

Key words: early childhood, sugar, long term health and economic well-being

JEL Codes: I12, I14, J13, J14

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1 Introduction

Free sugar intake, a key risk factor for obesity, diabetes, and cardiovascular disease, exceeds nutritional guideline recommended levels for a large share of the population (Vos et al., 2017; Ludwig et al., 2001; Fung et al., 2009; Ackerman et al., 2005; Larsson et al., 2006).¹ At consumption levels as high as 150 pounds of free sugar per person per year, more than 65% of Americans exceed the World Health Organization’s (WHO) maximum recommended daily limit of 10% of calories, or total energy intake (TEI), equivalent to about six to seven teaspoons of free sugar per day (WHO, 2015b; Powell et al., 2016; Bowman et al., 2017; Knüppel et al., 2017). Many more exceed the WHO strict limit of 5% of TEI, expected to maximize long-term health benefits (WHO, 2015b).

Sugar-rich diets begin early in life through maternal consumption of sugary foods and drinks during pregnancy and breastfeeding (Goran et al., 2019; Portella et al., 2012), and through infant and toddler intake of commercially prepared beverages and foods (Walker and Goran, 2015). Pregnant women consume an average of 21 teaspoons of added sugars daily (Cioffi et al., 2017), and more than half of lactating mothers exceed the maximum daily limit of 10% of TEI (Goran et al., 2019). Despite nutritional guidelines recommending zero free sugars for children below age two (Dewey et al., 2021), 61% of infants and 98% of toddlers consume at least some free sugar daily. Their free sugar intake increases with age (Park et al., 2014; Fiorito et al., 2010), and by age two exceeds the daily limit set for adults (Herrick et al., 2020; Vos et al., 2017).² Much of this intake comes from commercially prepared infant and toddler foods, many of which are high in free sugars.³ These products are aggressively advertised (Harris and Pomeranz, 2020) and increasingly supplied; the number introduced into the market has increased fourfold over the last decade alone (Romo-Palafox et al., 2020; Palafox and Harris, 2017). Despite growing concern over these trends, the long-term implications of excessive sugar

¹Free sugars include all sugars added to foods, in addition to sugars naturally present in honey, syrups, fruit juices, fruit juice concentrates, and purees. The sugars naturally present in milk and dairy products, fresh and most types of processed fruit and vegetables, cereal grains, nuts, and seeds are excluded (Swan et al., 2018).

²This is not unique to the US: free sugar intake among toddlers ranges from 12% of TEI in countries such as Denmark, Sweden, and South Africa to 25% in Portugal (WHO, 2015b). In Mexico and Chile, the majority of toddlers consume sweetened beverages (Essman et al., 2018; Afeiche et al., 2018), and by age five consume more than 10 teaspoons of free sugar per day (Afeiche et al., 2018; Herrick et al., 2020).

³Commercially prepared foods for infants and toddlers contain high levels of free sugar: over half of baby foods report that free sugar constitutes over 20% of calories, and some report levels as high as 80% (Walker and Goran, 2015; Maalouf et al., 2017; Elliott, 2011; Hutchinson et al., 2021; WHO, 2019). Yogurt, sweet bakery products, and sweetened fruit drinks are among the top five sources of free sugars for infants and toddlers in the US (Herrick et al., 2020). Among fruit drinks, sugar-sweetened drinks are the most commonly consumed by young children (Kay et al., 2018); in 2018 alone, their sales totaled \$1.4 billion, exceeding unsweetened juices by nearly 70% (Fleming-Milici et al., 2022).

intake early in life remain poorly understood.

This paper adds evidence on how exposure to a sugar-rich diet early in life affects health, cognition, and economic well-being more than 50 years later. We exploit variation in early-life exposure to sugar-rich diets induced by the end of sugar and sweets rationing in the United Kingdom (UK) in September 1953. The end of rationing had a dramatic effect on the confectionery market and sugar intake. Shortly after rationing was lifted, sales of sweets and chocolate increased by more than 150% (Jackson, 1974). Analyses of National Food Survey (NFS) data from the 1950s show a sharp increase in sugar consumption, from below 40 grams per person per day, comparable to today’s dietary guidelines, to more than 80 grams per person per day. No discontinuous change in consumption of other foods or nutrients was observed. Similarly, while children’s sugar intake was within today’s recommended amounts during rationing, it more than doubled after 1953, alongside a noticeable deterioration in dental health (Jackson, 1974; Zweiniger-Bargielowska, 2000; Hollingsworth, 1974, 1983; Prynne et al., 1999).

We employ a regression discontinuity design (RDD) to examine the impact of early-life exposure to a sugar-rich diet on later-life outcomes. We compare cohorts born during rationing (1950–1953) with cohorts born just after rationing ended (1955–1959), excluding cohorts with ambiguous exposure around the September 1953 cutoff. Our primary analyses use the English Longitudinal Study of Ageing (ELSA, 2002–2019) to study adults ages 50–64. We complement these analyses with UK Biobank data to examine objectively measured adiposity, metabolic health and genetic heterogeneity, Aberdeen childhood test-score data to study early-life cognition, and the National Diet and Nutrition Survey (NDNS, 2008–2016) to examine adult dietary patterns.

We find that greater exposure to a sugar-rich environment early in life worsened adult metabolic health more than five decades later. In ELSA, the end of rationing increased adult prevalence of diagnosed diabetes by 4.4 percentage points (pp), elevated cholesterol by 7.3pp, hypertension by 6.8pp, chronic inflammation by 11.2pp, arthritis by 12.2pp, and having three or more diet-related chronic diseases by 6.8pp. These results are corroborated in UK Biobank, where cohorts born after rationing had higher abdominal adiposity, diabetes, hypertension, elevated cholesterol, metabolic syndrome, and worse overall metabolic health. Importantly, the UK Biobank results further show that these effects are concentrated among individuals at higher genetic risk for obesity. Overall, we find supportive evidence of an exposure gradient: adverse health effects are larger when never-rationed cohorts are compared with cohorts protected by rationing for longer periods in utero and postnatally.

We also find lasting effects on cognition, human capital, and economic well-being. In Aberdeen childhood test-score data, children born after rationing ended scored 0.21 standard

deviations (SD) lower by age 9 and 0.26 SD lower by age 12. Excess sugar intake early in life have been previously shown to hinder hippocampal development in rodents, affecting a region of the brain central to learning and memory (Kanoski and Grill, 2017; Davidson et al., 2009, 2007; Baym et al., 2014; Meng et al., 2016; Noble and Kanoski, 2016; Abbott et al., 2019; Noble et al., 2019). In humans, maternal sugar-rich diets have been linked not only to greater risk of metabolic disorders in offspring (Hillier et al., 2007), but also to lower cognition, poorer executive function, and lower perceptual reasoning and memory (Casas et al., 2013; Tanda et al., 2013; Hinkle et al., 2012; Neggers et al., 2003; Li et al., 2018; Kamijo et al., 2012).

Related, in ELSA, the end of rationing reduced the probability of completing 16 or more years of education by 10.0pp, attending any college by 9.2pp, working in a skilled occupation by 14.1pp, and accumulating above-median wealth by 13.7pp. The overall economic sufficiency index fell by 0.26 SD. Together, these findings show that early-life sugar exposure affected not only adult health, but also cognitive development and socioeconomic well-being across the life course.

These findings have implications for policy. First, they provide evidence of large long-term benefits from limiting free sugar intake during pregnancy and early childhood, consistent with WHO and USDA dietary guidelines (Foster and Lunn, 2007a; Moynihan, 2016; Moynihan and Kelly, 2014). Second, they support policies to reduce sugar intake, especially in early childhood, such as taxes on sugar-heavy foods and drinks and regulation of sugar content and marketing in foods consumed by infants and toddlers.

What drives these long-lasting impacts of excessive early-life sugar intake? One pathway may be altered physiological programming due to adverse nutrition, also known as the developmental-origins hypothesis or Barker hypothesis (Martyn and Barker, 1994). Animal studies have found that, independent of total calorie intake, diets rich in sugars and carbohydrates *in utero* can establish persistent metabolic states that elevate lifetime risk of chronic disease and may be epigenetic in nature (Rosenfeld, 2015; Bateson et al., 2004; Pawlak et al., 2004; Langley-Evans and Sculley, 2005; Barker, 1998b; Martyn and Barker, 1994; Hales, 1997; Jackson, 2002; Bayol et al., 2008).

A second pathway may be lifelong dietary persistence. Children’s food preferences strongly predict diet later in life (Drewnowski, 1997; Mennella and Bobowski, 2015), and this may be especially important for sugar. Although humans have an innate preference for sweetness (Drewnowski et al., 2012; Sullivan and Brumfield, 2016; Mennella and Bobowski, 2015), the strength of this preference is shaped by early exposure, particularly during the first five years of life (Birch et al., 2007; Jayasinghe et al., 2017; Birch, 1999). Sugar can also produce craving, bingeing, and withdrawal-like responses (DiNicolantonio et al., 2018; Avena et al., 2009). Early

exposure to a sugar-rich environment may therefore increase lifelong sugar consumption by shaping preferences when they are most malleable.

Consistent with the second mechanism, we find that the end of sugar and sweets rationing elevated sugar consumption across the life course. More than 50 years later, adults born after rationing consumed 22.4% more free sugar, or about 11 additional grams per day, than those born just before rationing ended. This increase is comparable to the free sugar in about three Oreo cookies or half of an 8oz soda can per day.⁴ The end of rationing also increased the likelihood of exceeding the WHO strict recommended free sugar limit of 5% of TEI by 10.2pp. Importantly, we observe no long-term effect on total daily calories, protein, carbohydrates, or fat intake. Instead, the evidence points to a persistent shift in diet composition: higher free sugar intake and lower intake of healthier intrinsic sugars with fiber.

Our study makes several contributions to the literature linking nutrition *in utero* and during early childhood to health, cognition, and economic well-being later in life. First, we extend the literature using the end of UK sugar rationing as a natural experiment using data from the UK Biobank to study the long-term consequences of early-life sugar exposure. In prior work, we used UK Biobank data and an event-study reflexive (before and after) framework to show that exposure to sugar rationing in the first 1,000 days of life reduced the risk of type 2 diabetes and hypertension by about 35% and 20%, respectively, and delayed disease onset by several years (Gracner et al., 2024). Subsequent work using the same data and methods has replicated and extended these findings to other cardiovascular outcomes, including cardiovascular disease, myocardial infarction, heart failure, atrial fibrillation, stroke, and cardiovascular mortality (?).

We build on this evidence in three ways. First, we move beyond reflexive designs using survival analysis of one or two disease outcomes to estimate effects across a broader set of adult metabolic, inflammatory, cognitive, human capital, and economic outcomes. Second, we extend the data beyond the UK Biobank to ELSA, Aberdeen childhood cognitive data, and NDNS dietary data, allowing us to study outcomes across the life course and examine lifelong dietary persistence as a potential pathway. Third, we use a regression discontinuity design centered on the September 1953 cutoff, which more directly exploits the sharp policy-induced change in sugar exposure than the before-versus-after event-study design used in our earlier work. This approach strengthens causal identification while substantially broadening the scope of outcomes.

Other related work uses an RDD around the temporary 1949 confectionery de-rationing and

⁴The end of rationing also decreased intake of intrinsic sugars and fiber, both present in whole fruits and vegetables. We observe no difference in vegetable intake, but find lower fruit intake and higher sweets intake, suggesting substitution from healthier to less healthy sweet-tasting foods.

finds that a short, four-month *in utero* increase in sugar availability lowered late-life education but reduced BMI and adult sugar consumption (?). Our findings are complementary: we study the permanent 1953 derationing, a broader early-life exposure window, and outcomes later in the life course, showing that greater sugar exposure worsened metabolic health and increased adult free sugar intake without increasing total calories. Together, the studies suggest that the long-term effects of sugar exposure depend on timing, duration, and nutritional context, including whether calories or diet composition are most binding.

Second, we contribute to the broader literature on early nutrition and long-term well-being. Many design-based studies focus on extreme caloric restrictions, such as famine (Almond and Currie, 2011; Barker, 1998a; Almond et al., 2018; Roseboom et al., 2006; Ravelli et al., 2005; Barker, 1992), or on broader interventions with nutritional components, including SNAP (Hoynes et al., 2016; Bailey et al., 2020; Maluccio et al., 2009; Stein et al., 2006), the Carolina Abecedarian Project (Campbell et al., 2014), and nutritional supplementation (Stein et al., 2006). Most find long-term effects on metabolic health and economic outcomes, but mechanisms are often difficult to isolate. We extend this literature by studying pre- and post-natal exposure to sugar-rich diets specifically (Reid et al., 2016). Because we find differences in adult outcomes without a corresponding increase in total daily calorie intake, our results also speak to whether diet composition, and not only calorie imbalance, shapes health and economic well-being (Gračner, 2021; Lustig, 2013; Basu et al., 2013; Taubes, 2007). We further identify a potential pathway: elevated sugar consumption across the life course, consistent with the early formation of preferences for sweetness.

Finally, we contribute to the literature linking nutrition to human capital and economic well-being. Prior work has largely focused on short-term school-based nutrition, with mixed evidence on higher-calorie meals (Sorhaindo and Feinstein, 2006; McEwan, 2013) but stronger evidence that poor diet quality lowers test scores through worse health, illness, and missed school days (Anderson et al., 2018; Belot and James, 2011), especially at earlier ages (Lundborg et al., 2021). These early effects may compound over time (Heckman and Masterov, 2007; Cunha and Heckman, 2007) and, together with diet-related chronic illness, shape educational attainment, occupational choice, absenteeism, productivity, and wealth accumulation (Cawley, 2004; Goettler et al., 2017; Trogdon et al., 2008; Finkelstein et al., 2010; Popkin et al., 2006; Hoynes et al., 2016; Bailey et al., 2020).

2 Sugar and Sweets Rationing in the UK

The UK’s 14-year long food rationing program was designed in preparation for war – to guarantee everyone a healthy diet with fair shares for all to prevent food shortage and starvation. For that reason, the Ministry of Food allocated a scientifically devised weekly provision of specific foods to each person (Zweiniger-Bargielowska, 2000). To ensure that everyone obtained their full share, every person was given a ration book with coupons that were required to be able to purchase rationed goods at a specific retailer. The aim of this policy was “...to maintain the minimal nutrient intake...compatible with health” (Collinson and Macbeth, 2014). Accordingly, rationing focused primarily on processed, non-essential, sugar-rich foods; coffee, fish, fruits, and vegetables, for example, were never rationed, and a so-called buffer of bread or potatoes was freely available to satisfy differential energy requirements (Zweiniger-Bargielowska, 2000). People were encouraged to eat fruits and vegetables daily, and to use healthier alternatives in food preparation at home (Collinson and Macbeth, 2014).

Limitations on processed, non-essential foods such as sugar and sweets were a central part of the food rationing program (Foster and Lunn, 2007b). Because sugar and sweets were rationed at 12oz monthly, carrots were promoted as an alternative to sugar in cakes due to their natural sweetness and were advertised as “Children’s best friend” (Zweiniger-Bargielowska, 2000). TV stations broadcast recipes, selected by the Ministry of Food (Bruce, 1942) and magazines regularly featured recipes with reduced sugar or creative substitutes, along with other relevant nutritional advice (Barker and Burridge, 2014).

The Nutrition Society was formed with the aim of ensuring that food rations would be based on sound science. Its central objective was surveillance of the British food supply and the establishment of the National Food Survey (NSF) in 1940 (also known as the Expenditure and Food Survey today (Foster and Lunn, 2007b). Created for the purpose of diet tracking to prevent malnourished population during rationing, the NFS surveyed about 10,000 households quarterly from 1940 through 2000 to collect detailed information on weekly food consumption of household members, using their 7-day food diaries. These weekly dietary recordings were aggregated into the quarterly estimates of weekly per person consumption of various food groups, including sugar and sweets, fats, meats, vitamins, fruits, or vegetables, reported at the national level. We obtained this data from historical NSF records and combined them with prior work to describe dietary patterns during 1950 and 1960, the study period of our interest.

NSF data show that the diet under food rationing during this period was not extreme deprivation. In fact, it was nutritionally more balanced than during pre-war years and in many ways similar to recent WHO/USDA recommended dietary guidelines (see Table 2) (Collinson

and Macbeth, 2014). A diet based on these limits was about 2,500 calories per day, 35% of which came from fats, 13% came from protein, and 52% from carbohydrates - all within modern dietary guidelines (WHO, 2015a; HHS, 2015). Importantly, during our study period between 1950-1953, adults consumed on average about 40 grams of sugar per day accounting for about 6% of daily TEI (NFS, 2011). Modern guidelines recommend that less than 10% calories should come from sugar and less than 5% for maximal long-term health benefits (Prynne et al., 1999; WHO, 2015a).

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 today. 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. This diet was healthy enough so that children were not iron, vitamin A or C deficient (Foster and Lunn, 2007b), and amount of animal protein available for children and pregnant and nursing women was deemed adequate (Hollingsworth, 1983). Pregnant women, nursing mothers and children were given special green ration books with extra coupons and special allowances. Specifically, through the National Milk Scheme, implemented in 1940, pregnant women and nursing mothers were entitled to supplementary food rations, welfare, and priority foods such as vitamin supplements, fresh eggs, and milk, and had priority access to cod-liver oil, fruits, and vegetables. This supplied an additional 540 calories per day and the bulk of their daily requirement of calcium and vitamins.⁵ Children and adolescents had similar welfare and priority foods entitlements to pregnant women and nursing mothers (Zweiniger-Bargielowska, 2000; Sultan, 2010).

Figure 1 presents the timeline of food rationing between 1939 and 1954. Restrictions on food rationing were gradually lifted starting three years after the WWII had ended. The first set of restrictions lifted were on bread and flour in July 1948, followed by biscuits, jam, and canned and dried fruits in early 1950. Though some other foods, such as fats, rice, cheese, meats, milk, or cereals remained rationed until July 1954, sweets and sugar went off-ration by September 1953.

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). 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. However, soon after the end of rationing, children’s consumption of sugar and sweets more than doubled coupled

⁵The priority allowance was later credited as having “done more than any other single factor to promote the health of expectant mothers and young children during the war” (Jameson et al., 1946).

with a deterioration in dental health (Jackson, 1974; Zweiniger-Bargielowska, 2000; Jamel et al., 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999).

We find similar dietary patterns in NFS data. We show average daily consumption of sugar in the UK between 1950q1-1959q4 in Figure 2a, and intake of protein, fruits, vegetables, fats, and sugar for comparison over the same period in Figure 2b. We observe a sharp increase in the consumption of sugar and sweets by about 100% right after rationing ended (see Figure 2a), mapping into a corresponding increase in total calorie intake (see Figure A.4). Specifically, intake of sugars sharply increased from 10.2oz in 1953q1 to 16.08oz in 1954q1 and to nearly 20oz by 1954q3 per person per week. This contrasts with no sharp change in other foods or nutrients (e.g., protein, vegetables, fruits, fats, dairy etc.) during the same period as reflected in Figure 2b.

The sharp and quick response in the consumption of sugar and sweets, and not of other foods, soon after the rationing ended informs our research discontinuity 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 sugar and sweets rationing ended relative to those conceived and born after.

3 Data

3.1 The English Longitudinal Survey of Aging (ELSA)

ELSA is a bi-annual panel study of a representative of the population aged 50 and over in England. It spans a 16-year period between 2002-2018 (9 waves) and was designed as a sister study to the Health and Retirement Study (HRS) in the US. 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 survey participants is suited for our study as disability and chronic diseases start emerging frequently at this age (Kirkman et al., 2012).⁶ Our analytic sample includes individuals born in the UK between 1950 and 1960 who are 50-64 years old between 2002 and 2019, with all available outcome and control measures, excluding those born between October 1953 and in 1954, and those born outside the UK.

Our analysis examines several health outcomes derived from self-reported physician diagnoses of chronic conditions. We construct binary indicators for diabetes, elevated cholesterol,

⁶More details on this data can be found at www.elsa-project.ac.uk.

cardiovascular conditions (hypertension, heart disease, or stroke), and arthritis. We define a multimorbidity indicator as the presence of three or more conditions among the following: hypertension, diabetes, heart disease, stroke, and high cholesterol.

Following (Anderson, 2008) and (Hoynes et al., 2016), we construct a metabolic disease index as the weighted average of standardized z-scores for each metabolic component, where z-scores are calculated using the mean and standard deviation of the post-rationing cohort (born 1954 and later). The metabolic disease index aggregates adverse outcomes including diabetes, high cholesterol, heart disease, stroke, and hypertension. Higher values of the index indicate worse metabolic health.

For falsification tests, we examine conditions unlikely to be influenced by early-life sugar exposure, including self-reported cataracts, asthma, and lung disease. These serve as negative controls to assess the specificity of our findings to metabolically-relevant outcomes.

We treat all chronic conditions as absorbing states: once an individual reports a diagnosis in any wave, we code them as having that condition in all subsequent waves.

ELSA also collects biomarker data. It collects hemoglobin A1c (HbA1c) measurements for a subsample of adults. We define diabetes as $\text{HbA1c} \geq 7\%$, consistent with clinical diagnostic thresholds. We treat diabetes as an absorbing state—once an individual meets the diagnostic threshold. That is, they are classified as having diabetes in all subsequent periods, mirroring our approach with self-reported diagnoses. Data also include measures of C-reactive protein (CRP) levels objectively measured from blood draws, used to determine chronic or low grade inflammation. Chronic inflammation is defined as an indicator variable for CRP levels larger than 3mg/l. Measures of CRP are available for a subsample of participants who consented and were able to give blood in a clinical assessment.⁷

Economic outcomes of interest include indicator variables for whether survey participant has any college education, whether within the last 7 days of the survey they were employed full or part-time, had a skilled job (defined using the Standard Occupation Code SEC-13 referring to employers in large corporations, higher managerial or professional occupations, lower professional and higher technical occupations, intermediate, lower managerial or higher supervisory occupations, or employers in smaller organizations). We also construct an indicator for whether participant’s family wealth is above the median or in the top quartile. Total family wealth is defined as the sum between net value of primary residence, net value of business, net value of non-housing financial wealth, net value of secondary residence, and total value of other physical

⁷Missing biomarker data was largely because participants did not consent to give blood or were ineligible (people with clotting/bleeding disorders or taking anti-coagulant medication). Survey participants with missing CRP data were not significantly different across observable characteristics between cohorts.

assets (e.g., other land, or money owed by others). We construct economic sufficiency index follow (Kling et al., 2007) and (Hoynes et al., 2016) in calculating average across standardized z-score measures of "positive" outcomes like attending having over 16 years of education, working in medium/high-skilled occupation, being employed, having above-median wealth, and log-transformed wealth. The z-score is calculated by subtracting the mean and dividing by the standard deviation for those born after the end of rationing.

Importantly, ELSA also includes the "Life History Module" in Wave 3, which collects retrospective self-reported information for a subsample of individuals on health and socio-economic circumstances when they were 10 years old (Ward et al., 2009). These include information on whether they experienced financial hardship and whether their father's job was a high/medium skilled profession, household assets (number of books, bathrooms, rooms, or people in the residence), and parenting practices (e.g., mother was overprotective, understood my worries, was emotionally cold to me, etc.). We also use information on whether biological parents were ever diagnosed with diabetes as a proxy for one's genetic pre-disposition for diabetes and related disease. Data also include information on year and quarter of birth and whether participants were born in the UK or not.

Adults in our final analytic sample are between 50 and 65 years old; approximately 40 percent were born after rationing ended in 1954. Males represent 41 percent of the sample, and about half have at least some post-secondary education. Among those born during (after) rationing, 6.8 (8.2) percent are diagnosed with diabetes, 24.0 (24.0) percent with elevated cholesterol, 6.7 (7.6) percent with heart disease or stroke, and approximately one-third and one-quarter with hypertension and arthritis, respectively. About 10 percent have three or more chronic conditions.

3.2 UK Biobank

We use UK Biobank (UKB), a population-based cohort study launched by the UK National Health Service (NHS). Between 2006 and 2010, approximately 9.2 million adults aged 40-69 years registered in the NHS living within 25 miles of 22 assessment centers throughout the UK received study invitations, of which 503,325 individuals were enrolled (5.47% response rate) (Sudlow et al., 2015). We restricted our sample to participants born between October 1951 and March 1956; conceived within a 1000-day window around the end of sugar and sweet rationing in September 1953. Because UKB enrollment occurred between 2006 and 2010, this birth cohort restriction yields participants aged 52-56 at baseline, ensuring age overlap between rationed and never-rationed cohorts. We exclude individuals born outside England, those from

multiple births or who were adopted, participants who withdraw consent, those pregnant at the time of the survey, those exposed to rationing in utero only (i.e. born between 1953 q4-1954 q2), and individuals with missing covariates, genetic information or measures of adiposity. Our final analytic sample comprises approximately 46,914 adults, although this varies by outcome studied. The time-invariant characteristics of the baseline participants are presented in Table A A.1.

UKB complements our ELSA analyses in several ways. First, its larger sample size enables us to focus on a narrower window around the end of rationing, improving precision in our treatment definition. Second, UKB provides objectively measured visceral adiposity outcomes, known as key risk factor for chronic disease (Després and Lemieux, 2006). Third, UKB includes detailed birth data—we use quarter of birth as the running variable and information on birth location at the coordinate level. Finally, UKB includes genetic data, such as polygenic scores for BMI, which we use to explore heterogeneity in rationing effects by genetic predisposition. Thus, while ELSA’s longitudinal design allows us to examine longer-term chronic disease and economic outcomes across a wider age range when such conditions are more prevalent, UKB enables us to analyze early metabolic markers and gene-environment interactions with greater statistical power.

Our primary outcome is a visceral adiposity index constructed from trunk fat percentage (% TF, field 23127) and waist-hip ratio (WHR, fields 48 and 49).⁸ Each component was standardized using the never-rationed population (born after July 1954) as the reference group. We applied inverse-covariance weighting to create a summary index, reducing dimensionality while accounting for correlation across outcomes (Anderson, 2008). We analyzed the visceral adiposity index both continuously and as a binary indicator for abdominal obesity, defined as values at or exceeding the 90th percentile of the never-rationed distribution. We also created binary indicators for type 2 diabetes, hypertension, and hypercholesterolemia (proxied by cholesterol-lowering medication use or ICD-10 diagnosis E78) at baseline). Using these chronic disease indicators and abdominal obesity, we constructed a metabolic health index using the same inverse-covariance weighting approach, as well as a metabolic syndrome indicator defined as having at least two conditions at baseline.

We assessed heterogeneity in genetic risk for obesity using a standardized polygenic index

⁸Trained healthcare practitioners collected all measurements using standardized protocols across assessment centers. Waist and hip circumferences were measured directly in centimeters. Body and trunk fat percentages were assessed using bioelectrical impedance analysis (BIA) with the Tanita BC-418MA body composition analyzer, which measures electrical resistance through different tissues (fat tissue has higher resistance due to lower water content than lean tissue). Participants stood barefoot on the analyzer while holding handles, enabling calculation of regional fat percentages using proprietary algorithms incorporating impedance values, height, weight, age, and sex (UK Biobank Coordinating Centre, 2011).

for BMI derived from GWAS summary statistics. We use data on BMI PGI generated by Barcellos et al. (Barcellos et al., 2018) (UK Biobank return code 3586, application 1142). Using this index, we divide adults into those above vs below median genetic risk for obesity.

The BMI polygenic score for individual i is calculated as a weighted sum of SNP genotypes:

$$S_i = \sum_j x_{ij} \times w_j$$

where $x_{ij} \in \{0, 1, 2\}$ is the count of reference alleles for individual i at SNP j , and w_j is the weight associated with SNP j . The weights are derived from GWAS summary statistics for BMI (Locke et al., 2015) and transformed using linkage disequilibrium (LD) prediction (Vilhj  lmsson et al., 2015). LDpred requires both the GWAS results and a reference sample to estimate the linkage disequilibrium (correlation) structure of the genetic data. The 1000 Genomes Project, a catalog of genetic variation from over 2,500 individuals across diverse populations was used as the reference sample. The GWAS itself consists of regressions of BMI on individual SNP genotypes, controlling for sex, age, and principal components of genetic ancestry. To avoid overfitting, samples independent from the prediction sample were used to estimate the weights. In our sample, the resulting BMI PGI explains approximately 7% of the variation in BMI, aligned with data estimates elsewhere (Choi et al., 2020; Herrera and Lindgren, 2010; Yengo et al., 2018).

Unlike ELSA, UKB is not nationally representative. As mentioned earlier, recruitment was limited to individuals residing within 25 miles of one of the 22 assessment centers, all located in urban areas. Males, residents of socioeconomically deprived areas, and those from particular UK regions had lower participation rates.(?) UK Biobank participants are also healthier than the general population. Although these sampling characteristics affect external validity and limit generalizability to broader populations, they do not compromise internal validity. Crucially, there is no differential selection into the study between rationed and never-rationed cohorts. Mortality rates at ages 52-56 are very low and equivalent across both cohorts (Figure A8), ensuring balanced representation. The absolute levels of health outcomes in our sample may not generalize to the full UK population, but the causal effect of early-life sugar rationing that we estimate remains internally valid.

3.3 Aberdeen Children Study of Children Born 1950-56

The Aberdeen Children of the 1950s cohort comprises 12,150 children born in Aberdeen, Scotland between 1950 and 1956, linked to contemporaneous obstetric records from the Aberdeen

Maternity and Neonatal Databank (Leon et al., 2006). We observe month of birth, which positions each cohort member relative to the September 1953 end of sugar rationing and provides the running variable for our analyses.

Importantly, birth records are linked to cognitive test scores from routine school testing at ages 7, 9, and 11, administered within six months of each child’s birthday (Batty et al., 2004; Lawlor et al., 2006). At age 7, children completed the Moray House Picture Intelligence Test (form 1 or 2), a non-verbal group test of general reasoning based on pictorial analogies, classification, and pattern recognition, designed for children at the start of formal schooling before reading proficiency is established. At age 9, children completed the Schonell and Adams Essential Intelligence Test (form A or B), a group test covering verbal reasoning, vocabulary, and numerical problem solving. At age 11, children completed the Moray House Test battery used for secondary school placement under the Scottish tripartite system, comprising two verbal reasoning ability tests and two attainment tests in arithmetic and English; these components were combined with a teacher’s estimate to form a total transfer score (Batty et al., 2004; Leon et al., 2006). The age-11 Moray House verbal reasoning tests belong to the same family used in the Scottish Mental Surveys of 1932 and 1947, facilitating comparison with the Lothian Birth Cohorts (Deary et al., 2004).

The original scores were standardized to a mean of 100 and standard deviation of 15 using national norms at ages 7 and 9 and local Aberdeen norms at age 11 (Batty et al., 2004). For comparability across ages and with our analyses, we re-standardize each score to mean zero and standard deviation one within the never-rationed reference population, defined as cohort members with no early-life exposure to sugar rationing. All cognitive outcomes are therefore expressed in standard deviation units relative to never-rationed children.

3.4 The National Diet and Nutrition Survey

The UK National Diet and Nutrition Survey (NDNS) is a continuous, nationally-representative repeated cross-sectional survey that collects detailed quantitative information on diet of about 1500 individuals. It began in 2008 and is administered biannually. It is regarded as one of the most comprehensive nutrition surveys in Europe, and it is similar in its nature to the National Health and Nutrition Examination Survey (NHANES) in the United States (US). The NDNS provides high quality, nationally representative UK data on the types and quantities of more than 5,000 foods consumed by individuals. The dietary assessment method is based on the multiple-pass 24-hour dietary recall repeated on up to four non-consecutive days. It includes information on several socio-demographic characteristics, as well as birth quarter and place of

birth (in or outside the UK). In addition to several food groups (e.g., fruits, vegetables, meats), these data also provide information on at the nutrient level (i.e., X grams of protein per day). Data on dietary intake were excluded for those born outside the UK, and when “the unusual amounts of foods consumed” was reported. To estimate intake at the nutrient level, we combine NDNS data with the latest analyses in the Food Standards Agency’s nutrient databank.

Our main outcomes of interest are daily total energy intake measured in calories, and the intake of several macronutrients (e.g., proteins, carbohydrates, fats), measured in grams eaten per day. We divide carbohydrates into fiber and sugars. Sugars are further divided into free and intrinsic sugars. We follow the WHO (WHO, 2015a) and The UK Scientific Advisory Committee on Nutrition (SACN) (Swan et al., 2018) definition of free sugars. We also examine extrinsic sugars, often referred to as added sugars, which are free sugars excluding sugars naturally present in juiced or pressed foods. Intrinsic sugars are defined by the WHO as the sugars incorporated within the cell structure of food, as in intact fruit and vegetables. On average, fruits contain a higher amount of intrinsic sugars than vegetables (Ruiz et al., 2017; Hess et al., 2012). We also examine daily intake across food groups (e.g., sweets, fruits, vegetables) and construct indicators for whether someone eats any sweets (i.e., sugar, preserves, sweet spreads, chocolate, and sugar confectionery), fruits or vegetables on a given day.

Our final analytic sample consists of adults born in the UK before (1950-1953) and after (1954-1960) the rationing has ended. We exclude everyone born between October 1953 and June 1954, as they were born after rationing has ended, but could have been exposed to rationing at some point while in-utero. Those born in July 1954 were likely the first cohort conceived after the end of sugar and sweets rationing in September 1953. On average, adults born during(after) the rationing consume about 1800 calories daily and about 48(51) grams of free sugars per day.

3.5 Mortality

We collected data on live births⁹ and total deaths for cohorts born between 1950 and 1960. Data was collected between 1965-2019 by the Office for National Statistics, National Records of Scotland, Northern Ireland Statistics, and Research Agency. We had live births and mortality data by sex and age available for these years for England and Wales only, thus, we only present such calculations here.

⁹Office for National Statistics (2024). Births in England and Wales: 2023. Available at: <https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/livebirths/bulletins/birthsummarytablesenglandandwales/2023> (Accessed: January 13, 2026).

4 Empirical Approach

We employ a Regression Discontinuity Design (RDD) as our main empirical approach. The empirical specification is adapted to the measurement frequency and structure of each dataset. In all cases, the coefficient of interest is the discontinuous change in the outcome at the rationing cutoff (September 1953), so estimates should be interpreted as local comparisons of cohorts born close to the end of rationing.

We estimate the following specification:

$$\log Y_{it} = \alpha + \beta \text{BornPostRation}_i + \gamma f(\text{BirthDate}_i) + X_i' \delta + \theta_t + \varepsilon_{it} \quad (1)$$

where Y_{it} is the outcome variable of interest and BirthDate_i is the running variable. The running variable measures the time between the individual’s birth date and the cutoff date when rationing ended. BornPostRation_i is an indicator equal to one for individuals conceived after rationing ended and zero for individuals exposed to rationing in utero or early postnatally. The coefficient β is the discontinuous shift in the intercept at the cutoff in the relationship between the outcome and the running variable, and is the treatment effect of interest. Individuals with ambiguous exposure status, such as those conceived during rationing but born after rationing ended, are excluded. The exact excluded birth window differs across analyses depending on whether the comparison is designed to capture exposure in utero only or exposure in utero plus early postnatal months.

In ELSA, our main adult health and economic analyses use birth year as the running variable. The controls, X_{it} , follow the controls used in the corresponding outcome tables. All main ELSA specifications include sex, age indicators, parental diabetes indicators, indicators for imputed parental diabetes information, and survey wave/year indicators. We include age indicators because adult health outcomes change mechanically with age; the panel structure of ELSA allows us to compare respondents at the same ages across birth cohorts. We therefore restrict the sample to a common age support, ages 50–64, on both sides of the cutoff. The parental diabetes indicators proxy for family health history and genetic predisposition to chronic disease. Depending on the exposure window and outcome family, we additionally include birth-season controls. Standard errors are clustered at the individual level. For each outcome family, we report Romano-Wolf adjusted p -values to account for multiple hypothesis testing.

UK Biobank’s larger sample size and exact month of birth allow us to use a finer running variable than in ELSA, defined at the birth-quarter level. Because UK Biobank outcomes are observed at baseline rather than repeatedly over time, the controls differ from the ELSA panel

specifications. The main UK Biobank models adjust for sex, age-at-baseline indicators, calendar month of birth indicators, and family history of diabetes and cardiovascular conditions. For the main, shorter exposure specification, the cutoff is 1953q3. We exclude cohorts born in the quarters immediately after the end of rationing, whose early-life exposure is ambiguous. For the longer exposure adiposity specification, the cutoff shifts to 1953q1, and we exclude a wider set of ambiguous birth quarters around the end of rationing, restricting the sample to the tighter common age support of ages 55–56 at baseline. This longer-exposure specification is used for continuously measured adiposity outcomes, where the larger effective sample preserves enough precision to examine dose-response. The UK Biobank RDDs estimate local linear models using triangular kernel weights.

The Aberdeen analysis uses the same RDD logic but adapts it to childhood data and a monthly running variable. The sample includes children born between October 1950 and September 1955 who were resident in Aberdeen and enrolled in primary grades 3–7 in a state school in Aberdeen City at the time of the December 1962 study. We exclude children born immediately after the end of rationing because their exposure status around the cutoff is ambiguous. Because outcomes are observed in childhood in a single study setting, the Aberdeen specification does not include adult survey-year fixed effects or adult age indicators as in ELSA.

4.1 Identification

Identification relies on the standard RDD continuity assumption: cohorts born just before and after the end of rationing in September 1953 differ only in their exposure to sugar consumption, not other factors affecting long-term health outcomes (Imbens and Lemieux, 2008; ?; ?). It is violated if factors other than sugar exposure differentially affected rationed versus never-rationed cohorts, either early in life or through subsequent policies and events throughout the life course.

The treatment effect is the shift in the outcome at the cutoff point for the running variable, not the difference in means between all rationed and never-rationed cohorts regardless of distance from the cutoff. Therefore, the continuity assumption must hold only for the birth dates used to identify the intercept shift. Away from the cutoff date, the running variable is likely correlated with many omitted variables that also affect outcomes, except at the cutoff point where it is assumed to be "as if" randomized. The trend function for the running variable controls for these omitted variables in observations away from the cutoff. Hence, the continuity identification assumption applies specifically to birth cohorts immediately before and after the cutoff that identify the intercept shift.

The cohorts just before and just after the cutoff are defined by the time period used for the running variable. The running variable is a count variable where dates are grouped into intervals. The continuity assumption only applies to the intervals just before and just after the cutoff date. The interval lengths used include year, semester, quarter, and month depending the dataset. The interval choice is a tradeoff between the length of time the continuity assumption needs to hold and having a large enough sample around the cutoff to have well powered estimates of the intercept shift at the cutoff point. As a result, we are able to have shorter periods in larger dates sets. For ELSA, the smallest sample size, we used birth year as the running variable interval because of the low disease prevalence with finer intervals.¹⁰ We group births into birth quarter intervals in UK Biobank, and birth months in the Aberdeen data. See Table A.2 for details on the specifications of the running variable and associated birth date grouping across datasets.

4.2 Heterogeneity

We examine heterogeneity in the length of exposure to rationing and genetic risk. Not only are these analyses substantive, but they also help address potential identification concerns.

First, we test whether longer exposure to rationing produces larger effects. If sugar rationing drives our results, the effect should increase with exposure duration; if other factors (childhood circumstances, subsequent policies) drive results, effect sizes should be similar across exposure lengths. In ELSA, we compare effects for *in utero* plus up to 4 to 9 months of postnatal exposure versus *in utero* plus up to 10 to 21 months postnatal. In UK Biobank, which offers both larger sample sizes and a narrower birth cohort window, we estimate separate effects for *in utero* plus 0 to 3 months of postnatal exposure and for *in utero* plus up to 4 to 9 months. We restrict the analysis of UK Biobank data to visceral adiposity due to the small prevalence of other diseases because the age range is younger than ELSA.¹¹

For the UK Biobank, we stratify the analysis by genetic risk for obesity. If sugar rationing drives our results, individuals with high genetic risk should be more affected than low-risk individuals, as genetic predisposition moderates nutritional exposures (Qi et al., 2012). Importantly, both groups should be equally affected by differences in childhood circumstances or subsequent policies. As a falsification test, we examine genetic traits unrelated to metabolic health or economic outcomes (e.g., migraine genetic risk), where we should observe no differen-

¹⁰We check the robustness of this decision using birth-semester. The results reported in the appendix show that there is little change in the point estimates, although there is a small loss in precision for some outcomes.

¹¹For diabetes, hypertension, elevated cholesterol, metabolic disease index, and metabolic syndrome, we estimate effects for *in utero* plus up to 3 months exposure, but not for the longer window.

tial effects by genetic risk if our results reflect mechanisms specific to early-life sugar rationing exposure.

4.3 Estimation and Inference

We estimate the models using optimal RDD methods proposed in (Calonico et al., 2014) using the software in STATA. We assume that individuals born in the periods 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 by placing the greatest weight on observations closest to the cut-off using triangular kernel weighting. We estimate local regression models using a bandwidth of 4 years before and 5 years after the cut-off in 1954, with cohort trends being captured by a first-order polynomial of *BirthDate_i* on each side of the cut-off. We report conventional robust standard errors, clustered at the individual level.¹² We also conduct a series of robustness analyses varying the weighting and bandwidth, reporting the results in the appendix).

For each set of outcomes, we address the concern of arbitrarily selecting statistically significant treatment effects in the presence of multiple outcomes by performing multiple hypothesis testing based on the step-down algorithm proposed in (Romano and Wolf, 2005) (RW). We report RW p-values accounting for multiple hypotheses for each outcome.

5 Results

We begin by investigating the extent to which cohorts born before and after the sweets and sugar rationing are balanced with respect to their observable characteristics earlier in life. Table 1 shows that the differences in means in several potential confounders in ELSA, including childhood SES, home environment, parental education, occupation, health, and parenting style, are small and not statistically significant from zero. This suggests that individuals born before and after the end of sugar and sweets rationing began life under similar socioeconomic circumstances and that their parents were of similar health. While we do not include most of these variables as controls in our regression models, as they were only collected from a subsample of individuals, we do include parental diagnosis of diabetes because this information is available

¹²We report conventional robust standard errors rather than bias-corrected standard errors. Bias correction addresses potential polynomial misspecification (Calonico et al., 2014), but visual inspection confirms linear trends fit our data well. With a yearly running variable and modest sample size resulting in sparse observations per bin, bias-corrected procedures can be unstable. We also explored optimal bandwidths following (Calonico et al., 2014), which produced very small bandwidths (approximately 2 years), yielding comparable effect sizes but insufficient statistical power.

for about 85% of survey participants (we impute the other 15%). The exclusion of this control variable does not affect our findings. We observe similarly balanced childhood characteristics in UK Biobank and Aberdeen data (see Tables A.1 and 8).

5.1 Adult health outcomes

5.1.1 ELSA

We find that the end of sugar and sweets rationing led to higher prevalence of poor metabolic health in adulthood. Early-life exposure to a sugar-rich environment—in utero and during the first nine months postnatally—increased adult prevalence of diagnosed diabetes by 4.4 percentage points (54%), elevated cholesterol by 7.3 percentage points (30%), and hypertension by 6.8 percentage points (23%), with no observed change in heart disease or stroke (Table 4, Panel A). These results are robust to measurement approach and specification. As seen in Table ??, the elevated diabetes prevalence persists whether measured through self-report (Column 1) or objectively via HbA1c thresholds of 6.5% (Column 2) or 7% (Column 3), and across alternative running variables including birth year (panel A) and six-month birth periods (panel B). The consistency across subjective and objective measures rules out both reporting artifacts and general diagnostic trends as explanations for the observed effects.

We also observe higher prevalence of inflammatory conditions measured both objectively and through self-report: C-reactive protein above 3mg/l increased by 11.2 percentage points and arthritis by 12.3 percentage points. These findings are consistent with evidence linking elevated sugar consumption to systemic low-grade inflammation and elevated CRP levels (Calder et al., 2011). Independent of total calorie intake, elevated CRP has been shown to affect insulin resistance and increase risk of chronic diseases including diabetes, heart disease, and arthritis (Grundy, 2003; Danesh et al., 2000; Luft et al., 2013; Lontchi-Yimagou et al., 2013).

The end of rationing also affected overall metabolic health: those born after rationing experienced a 6.1 percentage point higher prevalence of comorbidities, defined as having at least three chronic diseases related to diet, and 0.21 standard deviations higher metabolic health index scores, indicating worse metabolic health. Figures A.6 and A.7 visually support these results, showing a discontinuous jump at the cut-off across all outcomes.

Table 4, Panel B, presents suggestive evidence that health effects increased with duration of rationing exposure. The difference in disease prevalence between those never-rationed and those exposed to rationing grew larger with longer rationing exposure. Comparing never-rationed individuals to those exposed in utero and up to 18 months postnatally, we find diabetes was 6.5 percentage points higher, elevated cholesterol 15.6 percentage points higher, heart disease

or stroke 7.6 percentage points higher, arthritis 21.3 percentage points higher, having at least three comorbidities 12.3 percentage points higher, and the metabolic health index 0.30 standard deviations higher. When comparing exposure in utero and up to 9 months versus in utero and up to 18 months, we find statistically significant differences ($p < 0.10$) for heart disease or stroke, arthritis, and comorbidities.

5.1.2 UKB

We next turn to UK Biobank data to test the hypotheses from ELSA and examine whether individuals with higher genetic predisposition to obesity experience different treatment effects. Table 5 confirms the ELSA findings on diabetes, hypertension, elevated cholesterol, and overall metabolic health, and adds objectively measured abdominal adiposity outcomes. We find that never-rationed adults experienced a 1.3 percentage point increase in diabetes prevalence (52% relative increase), a 3 percentage point increase in hypertension, a 2 percentage point increase in elevated cholesterol, a 0.11 standard deviation increase in the metabolic health index, and a 3 percentage point (21%) increase in metabolic syndrome prevalence (Panel A).¹³

These average effects mask substantial genetic heterogeneity. Panel B presents results for adults at above-median genetic risk for obesity (measured by polygenic index for BMI), while Panel C shows those at below-median risk. The overall health effects are driven almost entirely by high-risk adults. For low-risk adults, differences in outcomes between never-rationed and rationed groups are not statistically different from zero, with point estimates consistently smaller than those for high-risk adults. Formal tests comparing high- versus low-risk groups show statistically significant differences ($p < 0.10$) for abdominal adiposity and metabolic health index. Differences for diabetes, hypertension, and metabolic syndrome show the same pattern but reach only marginal significance ($p < 0.15$).

Next, we explore dose-response effects as in ELSA, but focus on continuous adiposity measures rather than binary disease outcomes. This choice reflects the lower chronic disease prevalence at ages 53-56 in UKB compared to ELSA’s older sample, which limits statistical power when further restricting to narrow birth quarters for dose-response comparisons. Table 6 shows that differences between never-rationed and rationed adults increase with rationing exposure duration, though this relationship seems to be concentrated at the upper tail of the adiposity distribution. The mean adiposity score does not differ significantly between those rationed in utero and up to 3 months versus in utero and up to 9 months. However, abdominal obesity prevalence when defined as exceeding the 90th percentile shows a dose-response: 3 percentage

¹³Chronic disease prevalence among never-rationed adults in UKB is lower than in ELSA because UKB participants are ages 53-56 compared to 50-64 in ELSA.

points higher for up to 3-month postnatal exposure versus 6 percentage points higher for up to 9-month exposure, relative to never-rationed adults.

5.2 Child Cognitive development

Table 8 reports RDD estimates of the effect of early-life sugar rationing on childhood cognitive test scores at ages 7, 9, and 11, with outcomes standardized to mean zero and standard deviation one within the never-rationed reference population. Across all three ages, children never exposed to rationing score lower than those whose early-life diets were constrained by sugar rationing. At age 7, the point estimate is -0.081 standard deviations and is not statistically distinguishable from zero ($SE = 0.046$). By age 9, the gap widens to -0.214 standard deviations ($SE = 0.047$, $p < 0.01$), and by age 12 it reaches -0.256 standard deviations ($SE = 0.045$, $p < 0.01$). The monotonic pattern indicates that the cognitive cost of early-life sugar exposure is not fully expressed at school entry but emerges and intensifies over the course of primary schooling, as test content shifts from picture-based reasoning at age 7 to verbal reasoning and attainment at ages 9 and 12. Figure 5 displays the underlying reduced-form relationship between birth month (centered at the September 1953 end of rationing) and standardized cognitive scores at each age. Taken together, the table and figure indicate that the removal of sugar rationing produced cognitive benefits among the generation of British children exposed to restricted sugar diets early in life, with effects that were modest and imprecisely estimated at school entry but substantial and statistically robust by ages 9 and 12.

5.3 Human capital, Labor Supply and Wealth

We find that the end of the sweets and sugar rationing affected adults' human capital and economic well-being. In Figure A.8, we observe discontinuous jumps at the cut-off for outcomes such as completing 16 or more years of education, working in a skilled occupation and accumulate above median wealth by mid-60s. Table 7, panel A, shows that on average, the lifting of the rationing caused adults born post-1954 to be 10pp or about 30% less likely to have 16 or more years of education, and 9.2pp or about 18% less likely to attend any college compared to adults who were exposed to rationing in utero and up to 9 months postnatally. Though both cohorts are equally likely to be employed, the end of the rationing decreased the probability of working in a high-skilled job (by 14.1pp or about 25%). Consistently, the end of rationing made it less likely for adults born after 1954 to accumulate wealth above the median (by about 14pp). Finally, we observe that economic sufficiency index for those never rationed was 0.26SD lower than for those who were rationed in utero and up to 9 months postnatally.

As with health outcomes, we find suggestive evidence of dose-dependent effects, though differences between groups are not statistically significant. Compared to adults rationed in utero and up to 18 months postnatally, never-rationed adults were about 15 percentage points less likely to attend college and had 0.35 SD lower economic sufficiency index.

5.4 Benchmarking

Figure 6 benchmarks our ELSA and UK Biobank estimates against prior evidence from Hoynes et al. (2016), who examined the long-term effects of the U.S. Food Stamp Program rollout (1961-1975). We find that: (1) metabolic protective effects increase with longer exposure to rationing, and at exposures up to 21 months, the effect sizes are comparable to those reported by Hoynes et al. (2016) for Food Stamp exposure; (2) similar patterns emerge for economic outcomes. However, important differences exist between the studies, including the populations (1940s-1950s UK versus 1960s-1970s U.S.), baseline nutritional status, the nature of interventions (universal sugar rationing versus means-tested food assistance), the ages at outcome measurement (50-70 years in our study versus 30-60 years in Hoynes et al. (2016)), and broader socioeconomic contexts, which should be considered when interpreting these comparisons.

5.5 Diet

Finally, we use the NDNS data to explore whether elevated sugar consumption over the lifespan was a potential mechanism behind the long-term health and economic effects of rationing. Outcomes total calories per day, grams of fats, proteins, and carbohydrates, which are further divided into daily intake (in grams) of fiber, intrinsic and free sugars. Binary outcomes are indicators of whether individuals ate any sweets (i.e., sugar, preserves, sweet spreads, chocolate, and sugar confectionery), fruits or vegetables on a given day, and whether sugar intake was above the 5% or 10% percent of TEI recommended limit per day. We estimated the RDD specifications using Ordinary Least Squares (OLS), because the NDNS sample size was too small to use the optimal methods in RDD software.¹⁴

Figure A.3 visually describes diet outcomes between cohorts of today’s older adults. Panels a-c show a sharp increase at the cut-off for share of people exceeding the recommended levels of free sugar intake at 5% of TEI, daily intake of free sugar and for consuming any sweets. Similarly, a discontinuous decrease at the cut-off is observed for intake of intrinsic sugars and any fruits. In contrast, we observe a discontinuous change in neither total calorie intake nor other

¹⁴We re-estimated the models for ELSA health and economic outcomes using OLS and found results very similar to those estimated using RDD software.

macronutrients, such as protein, carbohydrates, proteins or fats. These descriptive analyses are consistent with our regression results, which similarly show that the end of rationing did not affect adults' total daily calorie intake, but their diet composition in free sugar intake. Specifically, Table 3, panel A, shows that the lifting of sugar rationing increased the share of adults exceeding the recommended free sugar intake amount set at 5% TEI by 10.2pp or by 12%, and increased free sugar intake by 22.4% or about 11 grams of free sugar per day. This additional free sugar amount is comparable to the grams of free sugars found in about 3 Oreo cookies or in half of an 8oz soda can. Though no difference is observed in any vegetable intake (Panel B), the end of rationing resulted in adults being more likely to consume some sweets (by 8pp or 13.3%), no fruits (by 14.2pp or 21.2%), and less intrinsic sugars (by 18.4%) or fiber (by 9.0%); corresponding to about a half of an apple. We find no effect on the daily intake of total calories, or on the daily intake of macronutrients, such as fats, proteins and carbohydrates (Panel C).

6 Threats to Identification and Robustness

We carry out several analyses designed to rule out alternative explanations of the estimated effects. Identification relies on the cohorts born just before and after the end of rationing that are used to identify the intercept shift in the relationship between the outcome and the running variable differ only in their exposure to sugar consumption, not other factors affecting long-term health outcomes. It is violated if factors other than sugar exposure differentially affected rationed versus never-rationed cohorts, either early in life or through subsequent policies and events throughout the life course. The identification assumption applies specifically to birth cohorts immediately before and after the cutoff that identify the intercept shift as defined by the interval length of the running variable.

First, we assessed whether the cohorts conceived and born right before (1950-1953) and those conceived and born after the rationing ended (1955-1960) are similar in terms of family background and environment early in life - except in their exposure to sugar and sweets. Specifically, we checked whether individuals born before and after 1954 are balanced with respect to characteristics such as childhood SES and home environment, parental education, occupation, health, and parenting style. Table ?? shows that differences in means across these potential confounders are small and not statistically significantly different from zero. This suggests that our results are not driven by unobserved differences in family background and environment early in life.

Second, we test for discontinuities in the outcomes of interest at cutoffs at different times

than when sugar rationing ended (see Table A.6). Specifically, we conduct placebo tests by re-estimating our main specification using six-month birth periods as the running variable and applying artificial cutoffs at dates well after rationing ended. Specifically, we test for discontinuities at 1956q3, 1957q3, and 1958q3—corresponding to 3, 4, and 5 years after the end of sugar and sweets rationing in 1953q3. Under the null hypothesis that rationing ending drove the observed health effects, we should find no discontinuities at these later dates.¹⁵ Cohort differences in health and economic outcomes are small and not statistically significant at these placebo cutoffs across all outcomes. These null results support the interpretation that end of rationing, rather than other policies or events before or after 1953q3, drive the observed differences.

Third, we conduct a number of falsification tests by repeating the main analyses for diseases whose incidence should be unrelated to diet in general and sugar consumption but for which diagnostics have improved over the same period (e.g. lung disease, asthma, cataract). Exposure to rationing should not have affected these placebo outcomes but improved diagnostics might have. The analysis shows that there are no statistically significant differences in such diseases between cohorts (see Table A.5). In addition, Table A.11 shows no differential effects of sugar rationing on health outcomes when stratifying by genetic risk for migraine—a condition unrelated to diet. Effects are similar in magnitude and not significantly different between high versus low genetic risk groups.

Fourth, there is the possibility that improved disease diagnostics over time are driving our results as younger cohorts would then be more likely to be diagnosed at an earlier age than older cohorts. However, the improved diagnostics would have to affect the cohorts just before and just after the cutoff date differentially. However, this is a very tight window. Additionally, if improved diagnostic trends were driving our results, we would expect a positive significant effect for outcomes like hypertension or diabetes that are diagnosed by a medical care provider, but no significant effect on objective outcomes measured with biomarkers at the time of the survey such as CRP or HbA1c. In fact, we show the opposite in Tables ?? and ?? .

Fifth, within survey years, the cohort born after the end of rationing is younger than the cohort born right before it, which might attenuate the estimates. We address this concern by age indicators in the regressions. We are able to do so because the panel nature of our data allows us to observe the same individual at multiple times. In ELSA, we limit the sample on adults with age overlap between cohorts to 50-64 in ELSA. In UK Biobank, we limit our sample

¹⁵This falsification test also addresses concerns about potential confounding from the 1972 educational reform, which raised the minimum school-leaving age from 15 to 16 for students born on or after September 1, 1957. While we observe a small discontinuity in skilled employment for those born after 1957 versus before, the coefficient is negative—opposite the sign predicted if the reform explained our health findings.

to ages 52-56 at baseline.

Sixth, we address the concern of positive selective fertility during rationing by controlling for the annual number of live births in new set of regressions (see eTable ??); our conclusions remain unchanged.

Seventh, selection from differential mortality between cohorts by the time of survey could bias the estimates. We show that mortality rates at ages 50-64 (corresponding to ELSA sample) and at ages 52-56 (corresponding to UK Biobank sample) are very low and not significantly different across cohorts at any age (Figure A.10), ensuring balanced representation.

Eight, we used several AI programs to systematically reviewed UK policies and major economic events from the end of WWII through 2000 to identify factors that could have differentially affected rationed versus never-rationed cohorts around the September 1953 cutoff (Table A.12) and found no policy that could explain our results.

Finally, we provide evidence that our results are not sensitive to the choice of bandwidth, or to the order of polynomial chosen, or running variable choice (see Tables A.8, A.7, A.10, A.9, and A.3 and A.4). We also re-estimate our main specification in ELSA using a running variable being six-month birth periods and our findings remain unchanged (Tables A.3, A.4).

7 Conclusion

We study the long-term effects of exposure to sugar-rich diet in early childhood on health and economic well-being in later adulthood, and identify diet as a possible pathway. We exploit the end of sugar and sweets rationing in 1953 in the UK as a natural experiment inducing variation in exposure to sugar-rich diet in early childhood. We find that the end of rationing increased the adult prevalence of chronic inflammation, an important marker of chronic disease. We also find increases in poor metabolic health; particularly diabetes, cholesterol and arthritis. Finally, we find that excessive intake of sugar also results in worse economic outcomes; specifically, in reduced human capital accumulation, a lower likelihood of having a skilled occupation, and a lower accumulation of wealth. While the effects of sugar-rich diet early in life may have affected these outcomes directly via physiological programming, we find that sugar-rich diet early in life elevated free sugar consumption across lifespan; consistent with evidence that life-long dietary preferences and habits form early, and that sugar may be addictive.

These findings are concerning, given the increased attention food companies are paying to infants and toddlers. In 2018, sweetened children’s drink sales totalled \$1.4 billion, exceeding unsweetened children’s juices by nearly 70% (Fleming-Milici et al., 2022). Studies have found that more than 50% of commercially prepared baby foods feature more than 20% (and some

even 80%) of calories coming from sugar (Walker and Goran, 2015). A good example of this is a relatively new food for young children called “toddler” milk, consisting primarily of powdered milk and added sugars such as corn syrup, sucrose, cane juice or other caloric sweeteners (Romo-Palafox et al., 2020; Palafox and Harris, 2017). Despite being considered inadequate for young children (Palafox and Harris, 2017), new heavily sweetened drinks and foods are being introduced rapidly and advertised heavily. There were four times more product launches in the baby and toddler food aisle in 2018 than in 2005 (Romo-Palafox and Harris, 2021), and \$56.5 million was spent on advertising in 2015 alone (Harris et al., 2016; Harris and Pomeranz, 2020). A major issue is also the often misleading marketing claims that position sugar-rich infant and toddler foods as beneficial for their nutrition and development (Duffy et al., 2021) (Dallacker et al., 2018; Cattaneo et al., 2015; Fleming-Milici et al., 2022).

Our results suggest the need for public intervention to reduce the consumption of free sugar by pregnant and lactating women and young children. Both imperfect nutrition knowledge and potentially addictive properties of sugar are rationales for intervention (Griffith et al., 2020; Allcott et al., 2019; Gertler et al., 2022). Public health campaigns similar to the anti-smoking advertising may be warranted. Another could be stronger food labeling warnings. The US has largely relied on self-regulation in the food industry, though limits on sugar content in foods marketed to infants and toddlers could also be imposed. That is because a growing body of medical evidence shows that excessive sugar intake can trigger processes leading to a wide range of negative health outcomes (some of which we also show here), including liver toxicity, chronic inflammation, insulin resistance, and other diseases (Lustig, 2010). Importantly, this has been shown to happen regardless of total calorie intake, suggesting that there is a *type* of consumables (and not diet generally) that can be uniquely harmful (Lustig, 2020, 2010; Taubes, 2007). Additionally, while quantity may be determined by the end user, and therefore a personal responsibility issue; food content and quality is determined by manufacturers, and thereby becomes public health issue (Lustig, 2020). Our results are consistent with these findings in that we show long-term sugar adverse effects regardless of total calorie intake.

Our findings are also in line with the literature studying the health impact of higher prices of sugar-sweetened beverages (SSBs) or sugar-rich foods. Though long-term implications of such policies are unknown as they have only been implemented recently, (Gračner et al., 2022) shows that a ten percent SSBs tax leads to about 0.4kg weight loss among heavier teenage girls (corresponding to a daily 7.8 calorie reduction) already within two years of a tax. Similarly, (Gračner, 2021) shows that higher prices of foods rich in sugar (and not in other nutrient) lead to less obesity and diabetes. Several modeling studies predict larger improvements in obesity and metabolic health over time; with largest disease reductions expected for populations exposed to

the SSBs tax at a younger age (Sánchez-Romero et al., 2016; Basto-Abreu et al., 2019; Dubois et al., 2020). For instance, a 20% SSBs tax was projected to reduce prevalence of obesity between 1.5% and 10%, and of diabetes cases by up to 3.4% annually over a decade (Mytton et al., 2014; Wang et al., 2012; Smith, 2010). Similarly, a 20% reduction in added sugar intake (about 14 grams of free sugars at the current mean intake among US adults (Ervin and Ogden, 2013)) was projected to reduce incidence of diabetes by about 20 cases per 100,000 people over two decades (Vreman et al., 2017; Basu et al., 2014).

Finally, as diets during post-war rationing in our setting were similar to dietary guidelines for free sugar today, our results may serve as one of the first references on the long-term effect of compliance to dietary guidelines early in life. 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, 2007a; 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 and similarly large shares have been found in the UK (Yang et al., 2014; Griffith et al., 2020). There are currently no strict guidelines for daily recommended limits for added sugar intake for pregnant women. Only the most recent 2015-2020 dietary guidelines have been updated to recommending zero free sugar for children under the age of two.

There are still many questions left open to study. For instance, we are not able to identify the importance of developmental periods in early exposure to sugar, such as prenatal vs postnatal period, in long-term effects. Additionally, while we observe health and economic differences in older adulthood, we are unable to observe when these disparities start emerging. Finally, while we find suggestive evidence that health and economic effects are in part driven by excessive free sugar intake across lifespan, we are unable to observe the precise mechanisms and the extent of diet effects. These are all important questions part of our ongoing research.

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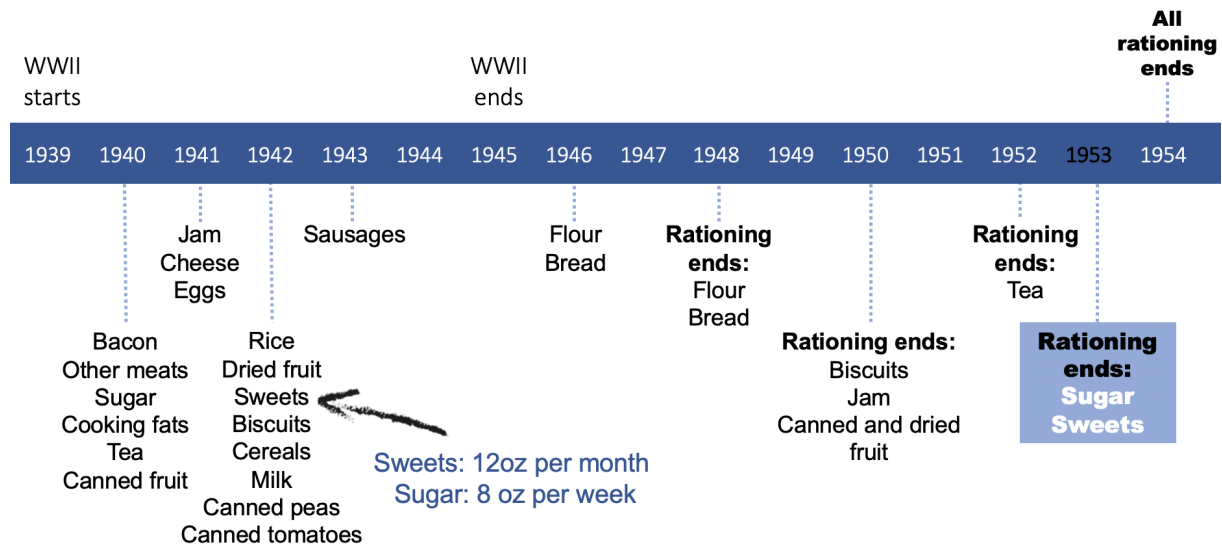
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8 Figures

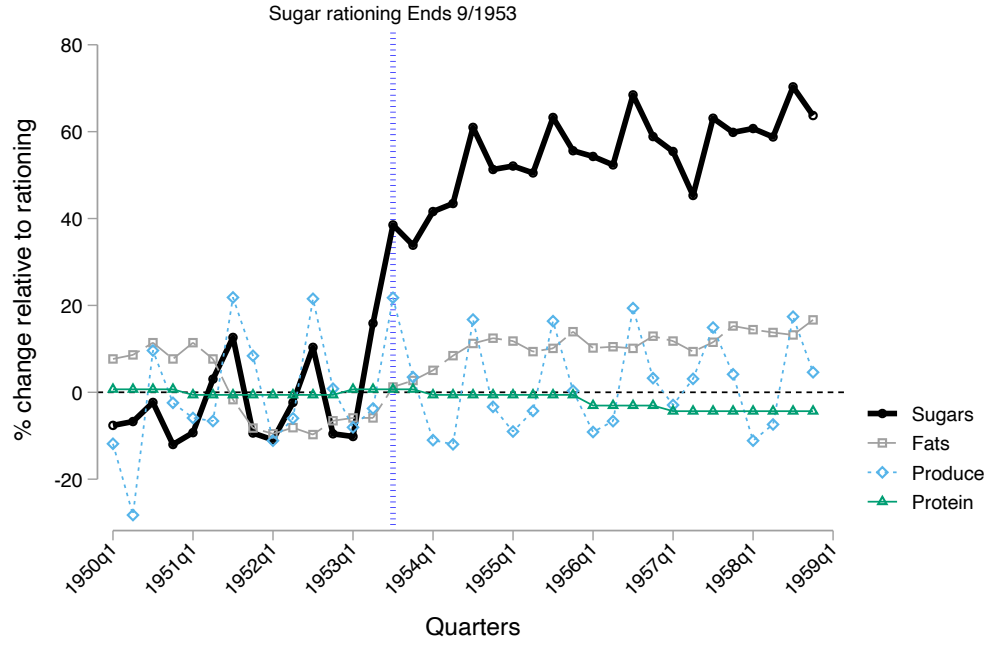
Figure 1: Food rationing timeline in the UK: 1939-1954



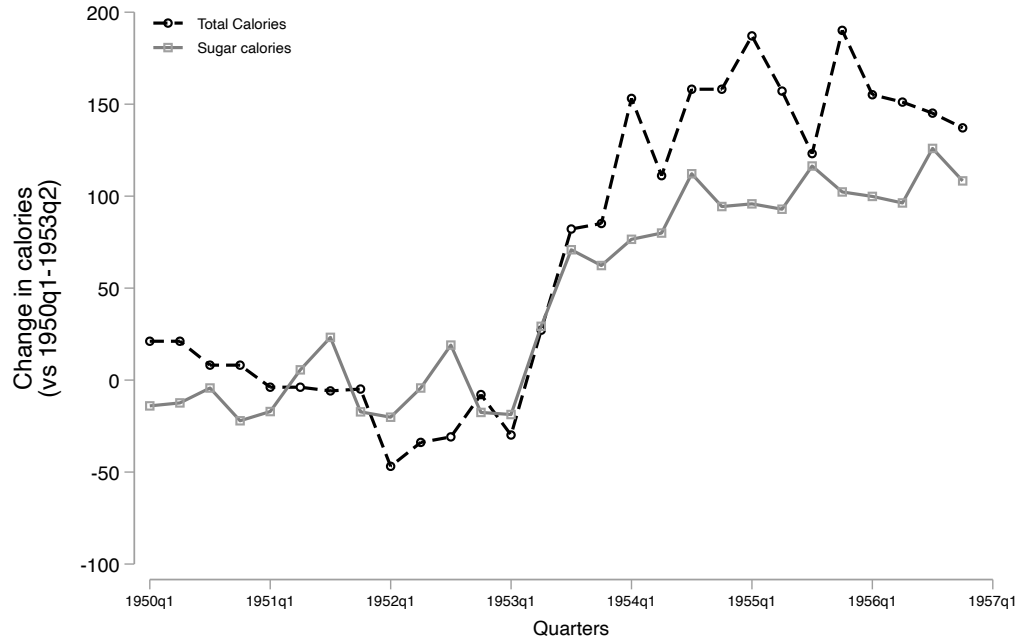
Note: Author's presentation of years during which different foods went off-ration.

Figure 2: Change in sugar intake for adults during 1950-1960

(a) Change in daily per capita consumption of sugar and other foods

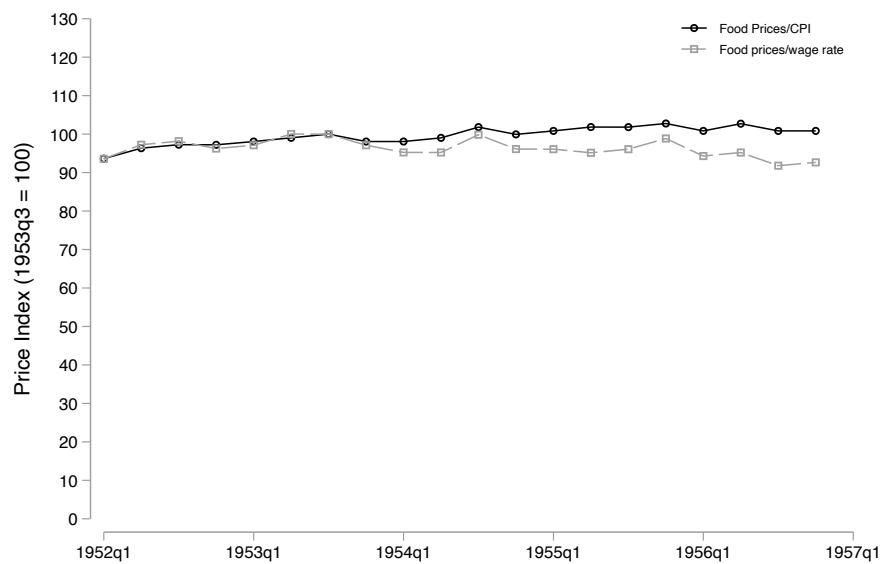


(b) Changes in total and sugar calorie consumption



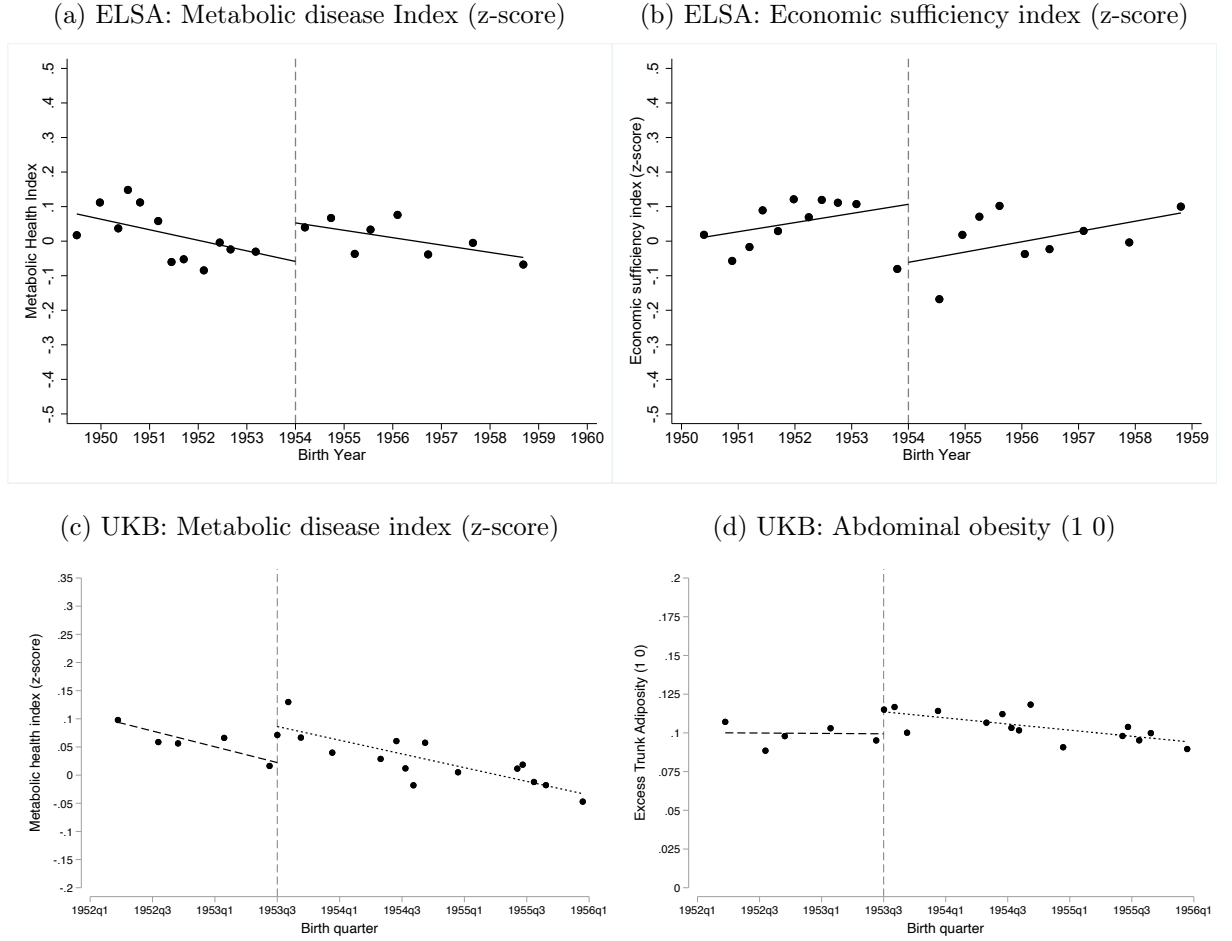
Note: Graphs based on data published by the National Food Survey 1950-1960. These figures have been previously published in (Gracner et al., 2024).

Figure 3: Food affordability between 1950-1960



Note: These data have been previously published in (Gracner et al., 2024).

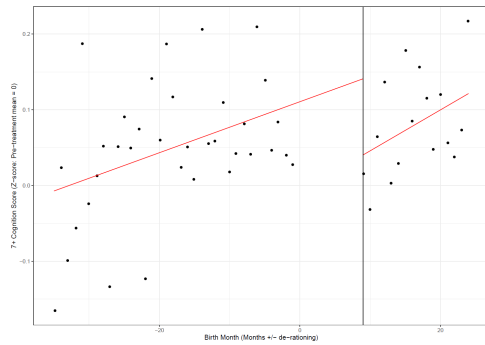
Figure 4: Metabolic disease and economic self-sufficiency index across birth cohorts



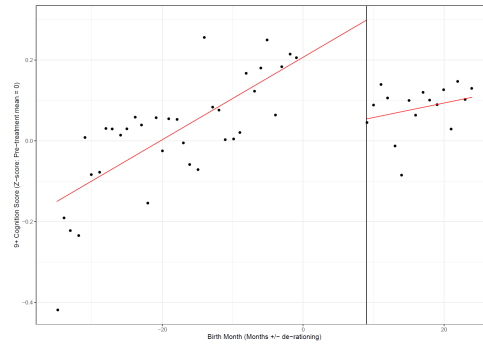
Note: Cohort difference health and economic well-being. (a) Metabolic health index includes "bad" outcomes, like diabetes, cholesterol, heart disease, stroke, or hypertension. We only have obesity-related measures for a smaller sample of individuals, so we do not include them here. (b) Economic sufficiency index (ESI) includes "positive" outcomes like attending having over 16 years of education, working in medium/high-skilled occupation, being employed, having above-median wealth, and log-transformed wealth; based on ELSA data. Indices follows Anderson (2008) and Hoynes et al. 2016 (for ESI) in calculating weighted average across standardized z-score measures of each component. (c) Metabolic health index includes "bad" outcomes like diabetes, hypertension, cholesterol (proxied with cholesterol medication use), and abdominal adiposity measures (i.e., trunk fat percentage, waist-to-hip ratio); using UKB data. Z-scores are calculated by subtracting the mean and dividing by the standard deviation for those born after the end of rationing. (d) Abdominal obesity identifies those at above the 90th percentile within UKB distribution among never rationed adults. Cut-off determining exposure is September 1953.

Figure 5: Childhood cognitive test scores at age 7, 9 and 11 across birth cohorts

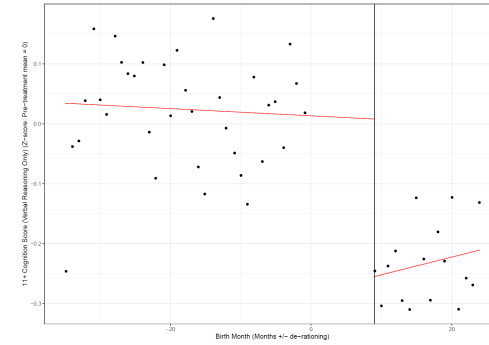
(a) Age 7



(b) Age 9

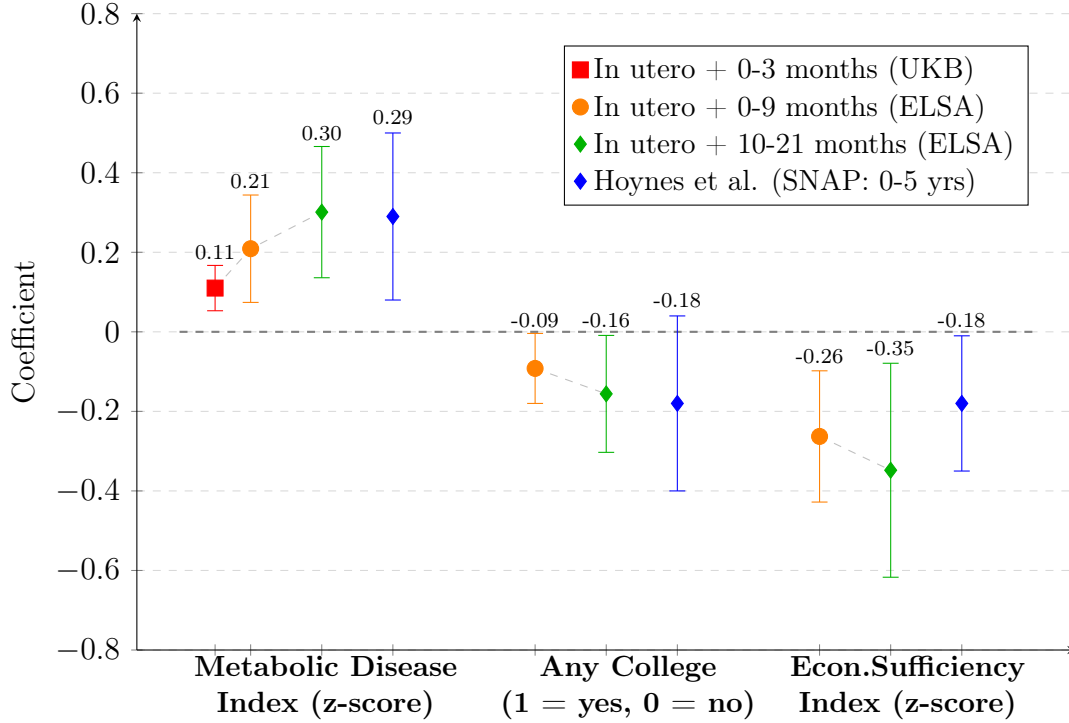


(c) Age 11



Note: The sample comprises children born between October 1950 and September 1955 who were resident in Aberdeen and enrolled in primary grades 3–7 in a state school in Aberdeen City at the time of study (December 1962). Children born from October 1953 to June 1954 are excluded. Outcome variables are standardised against the pre-ratifying mean, therefore coefficients are interpreted as standard deviation changes.

Figure 6: Benchmarking Against Prior Evidence in Hoynes et al.(2016)



Notes: Metabolic disease and economic self-sufficiency indices follow Anderson (2008) and Hoynes et al. (2016) in calculating a weighted average across standardized z-score measures of each component. The z-score for each component is calculated by subtracting the mean and dividing by the standard deviation for those born during rationing (1950–1953). The metabolic disease index includes adverse outcomes such as diabetes, high cholesterol, heart disease, stroke, and hypertension in ELSA, as well as abdominal obesity marker in UKB. The economic sufficiency index includes outcomes such as attending any college, medium- or high-skilled occupation, employment status, above-median wealth, and wealth (in logs). Orange and green estimates are from ELSA (English Longitudinal Study of Ageing), while the red estimate for metabolic disease is from UK Biobank. Hoynes et al. (2016) find that children with access to greater economic resources (food stamps) before age five have better outcomes as adults.

9 Tables

Table 1: Cohort Differences in Childhood and Parent Characteristics

Characteristic	Rationed (%)	Never Rationed (%)	Diff.
<i>Panel A: ELSA</i>			
Mother had diabetes	9.0	10.0	1.1
Father had diabetes	9.0	10.0	0.8
Father occupation – high	32.0	30.0	–1.9
SES score at age 10, 0–5 (cont.)	3.3	3.2	–0.1
Had bath in-house at age 10	82.0	81.0	–1.0
Had toilet in-house at age 10	77.0	77.0	–0.3
No or few books at age 10	19.0	17.0	–1.1
N of rooms at age 10 (cont.)	3.0	3.1	0.1
N of people in residence at age 10 (cont.)	5.0	5.1	0.0
Mother let me do the things I liked doing	63.0	60.0	–3.2
Mother was emotionally cold to me	12.0	13.0	0.8
Mother understood my worries	57.0	51.0	–6.5
Mother liked me to make my own decisions	63.0	61.0	–2.2
Mother was overprotective	15.0	13.0	–2.2
Observations	851	376	
<i>Panel B: UK Biobank</i>			
High genetic risk for obesity	66.3	66.4	–0.1
Family history: diabetes	17.3	18.3	–0.9
Family history: CVD	63.4	62.8	0.6
Often/very often felt loved as a child	77.0	76.4	0.5
Never sexually molested as a child	89.8	89.6	0.2
Had someone take me to a doctor as a child	84.6	85.1	–0.6
Long-term antibiotic therapy as a child	15.5	16.3	–0.8
Observations	20,748	18,377	
<i>Panel C: Aberdeen data</i>			
Mother married at birth	96.7	97.0	–0.3
Number of siblings (cont.)	3.2 (SD 1.6)	3.2 (1.6)	0.1
Mother’s age at birth (cont.)	26.9 (SD 5.3)	26.9 (5.5)	0.0
Attends private school	1.7	1.7	0.0
Attends selective school	6.8	7.0	–0.2
<i>Father’s occupation at birth</i>			
Skilled	78.2	79.8	–1.6
Unskilled	16.5	15.1	1.4
Unemployed	5.4	5.2	0.2
Observations	6,529	2,907	9,436

Notes: Diff. = simple difference in means; superscripts *, **, and *** denote significance at the 10%, 5%, and 1% levels, respectively. Differences without stars are not statistically significant. All values are percentages unless marked (cont.), which indicates a continuous variable reported in its natural units. Panel A uses ELSA data; childhood variables are measured in wave 3 only. Panel B uses UK Biobank data. Family history variables have slightly smaller samples due to missing data and are imputed and controlled for in all regressions. Variables relating to childhood experiences (feeling loved, being taken to the doctor, antibiotic use, and sexual molestation) are based on a subset of respondents to whom these questions were administered. Significance tests for parental health variables adjust for whether each parent was alive at the time of survey, as rationed individuals are older and thus more likely to have parents who were diagnosed with or died from age-related conditions. Panel C comprises children born between October 1950 and September 1955 who were resident in Aberdeen and enrolled in primary grades 3–7 in a state school in Aberdeen City in December 1962; children born between October 1953 and June 1954 are excluded. Father’s occupation follows the UK Registrar General’s classification, with SES groups 1–3 coded as skilled and groups 4–5 as unskilled.

Table 2: Diet under rationing (1950-1953) and today’s recommendations on healthy diet

	UK diet in 1950-1953	Today’s diet recommendation
Total calorie intake	2,500 Kcal	1800-3000 Kcal
Fats	35%	20-35%
Protein	13%	10-30%
Carbohydrates	52%	45-55%
Sugar	6.5% (40g)	<10% (strict) <5%(conditional)

Sources. Department of Environment, Food and Rural Affairs, National Food Survey (2011). Today’s recommendations are drawn from ?, ?, and ? for macronutrient and sugar targets, and from ? and ? for U.S. dietary guidelines. Fat, protein, and carbohydrate ranges reflect the Acceptable Macronutrient Distribution Ranges (AMDRs), which remained unchanged across the 2020–2025 and 2025–2030 editions of the U.S. Dietary Guidelines. The sugar thresholds follow WHO guidance: a strong recommendation to limit free sugars to below 10% of total energy intake, and a conditional recommendation to limit intake further to below 5%. The 2025–2030 U.S. Dietary Guidelines adopt a stricter stance, recommending that no amount of added sugars be considered part of a healthy diet (?).

Table 3: The Effect of Lifting Rationing on Diet

<u>Panel A:</u>		Sugars			
		Free sugars $\geq$ 5% TEI (1)	Log(Free Sugars) (2)	Log(Intrinsic Sugars) (3)	Log(Fiber) (4)
Born after rationing	β	0.102**	0.224**	-0.185**	-0.090**
	SE	(0.044)	(0.096)	(0.079)	(0.041)
	Single p -val	0.020	0.020	0.019	0.028
	RW p -val	0.001	0.001	0.000	0.002
N		2,853	2,853	2,853	2,853
Y(=0) Mean		0.79	48.41	31.98	14.69
<u>Panel B:</u>		Food Groups			
		Any Vegetables (5)	Any Fruits (6)	Any Sweets (7)	Any Meats (8)
Born after rationing	β	-0.007	-0.142***	0.080*	0.044
	SE	(0.042)	(0.052)	(0.041)	(0.046)
	Single p -val	0.863	0.007	0.052	0.339
	RW p -val	0.802	0.000	0.011	0.413
N		2,853	2,853	2,853	2,853
Y(=0) Mean		0.78	0.66	0.60	0.52
<u>Panel C:</u>		Macronutrients			
		Log(TEI) (9)	Log(Fats) (10)	Log(Protein) (11)	Log(Carbohydrates) (12)
Born after rationing	β	-0.001	0.042	-0.027	-0.019
	SE	(0.041)	(0.029)	(0.028)	(0.025)
	Single p -val	0.981	0.151	0.348	0.445
	RW p -val	-	0.102	0.413	0.413
N		2,853	2,853	2,853	2,853
Y(=0) Mean		1768.26	65.94	75.45	209.99

Note. Robust standard errors in parentheses, clustered at the individual level. Single p -values are unadjusted, and RW P -values are adjusted for multiple hypothesis testing using the Romano & Wolf correction. Sample includes individuals who were born in the UK between 1950-1960, and who are surveyed in NDNS at least once between 2008-2019. Born after rationing is an indicator for those born after September 1953 (excluding those born between 1953q4 and 1954q2). Sample excludes those who reported eating an unusual amount of food or beverages on a particular day or above 99th percentile of free sugar. All log-transformed continuous outcomes are measured in grams; except TEI, which is measured in kcal. Intrinsic sugars exclude lactose. Recommended 5% of TEI is an indicator if the share of free sugar calories in total energy intake is $\geq$ 5%. Any fruits/vegetables/meat is an indicator for whether an individual consumed any amount of fruits/vegetables/meat in the last 24 hours. Any sweets is an indicator for whether an individual consumed any amount of sugar preserves, sweet spreads, sugar confectionery or chocolate confectionary in the last 24 hours. All outcomes are adjusted for TEI (except TEI outcome), cohort-specific linear trend, sex, height, race, and for calendar month and day of the week, and survey year fixed effects. Results are not sensitive to including/excluding these individual characteristics. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: RDD Estimate of Lifted Rationing on Health Outcomes (ELSA)

		Metabolic disease			Cardiovascular		Inflammation			
		Diabetes	Diabetes HbA1c $\geq$ 7%	Cholesterol	Hypertension	Heart disease or stroke	CRP $\geq$ 3 mg/l	Arthritis	Chronic diseases 3+	Metabolic disease index
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
A) In utero + [0, 9 mths]:										
Born after rationing	β	0.044**	0.057*	0.073*	0.068*	0.007	0.112**	0.122***	0.068***	0.209***
	SE	(0.021)	(0.030)	(0.039)	(0.039)	(0.024)	(0.043)	(0.036)	(0.022)	(0.077)
	Single p -val	0.036	0.061	0.060	0.082	0.765	0.010	0.001	0.002	0.007
	RW p -val	0.000	0.000	0.001	0.000	0.001	0.000	0.000	0.000	0.000
N		12589	2435	6351	12588	12589	2481	12589	12589	12589
B) In utero + [10, 21 mths]:										
Born after rationing	β	0.066*	0.080**	0.156**	0.051	0.076**	0.066	0.214***	0.124***	0.301**
	SE	(0.034)	(0.040)	(0.063)	(0.067)	(0.032)	(0.077)	(0.059)	(0.036)	(0.123)
	Single p -val									
	RW p -val	0.000	0.000	0.001	0.000	0.001	0.200	0.000	0.000	0.000
N		10480	2027	5082	10479	10480	2091	10480	10480	10480
Y(=0) Mean		0.082	0.048	0.240	0.297	0.077	0.245	0.253	0.088	-0.000
$B > A$ (p-value)		0.284	0.322	0.132	0.588	0.043	0.698	0.092	0.093	0.178

Note. Sample: Born 1950-1960 (excl. 1953q4-1954q4), ages 50-64, ELSA 2002-2019. Sample includes individuals born between 1950-1960 (excluding those born between 1953q4-1954q4) who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Each model adjusts for sex, age, parental health history, and survey year indicators. Semester of birth is included for all outcomes except cholesterol, C and inflammation, where limited sample size reduced precision but not effect magnitude. C-reactive protein above 3 mg/l indicates moderate or high inflammation. Chronic diseases 3+ indicate three or more diseases (hypertension, diabetes, heart disease, stroke, cholesterol). Metabolic disease index equals the average of standardized z-scores for each component measure, where z-scores are calculated using the mean and standard deviation of the post-rationing cohort. Components include diabetes, high cholesterol, heart disease, stroke, and hypertension (i.e., higher value means worse health). We report estimates with standard errors clustered at the individual level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Romano-Wolf adjusted p-values account for multiple hypothesis testing. We conducted one-sided t-tests to evaluate whether Panel B estimates significantly exceeded Panel A estimates, testing the hypothesis that effects increase with exposure to rationing.

Table 5: RDD Estimates of Lifted Rationing on Health Outcomes (UK Biobank)

		Abdominal adiposity (z-score) (1)	Abd. Obesity ≥ 90 th pct (1, 0) (2)	Diabetes (1, 0) (3)	Hypertension (1, 0) (4)	Cholesterol (1, 0) (5)	Metabolic disease index (z-score) (6)	Metabolic syndrome (1, 0) (7)
A) Overall:								
Born after rationing	β	0.079***	0.029***	0.013**	0.025**	0.020**	0.109***	0.027***
	SE	(0.028)	(0.009)	(0.006)	(0.012)	(0.010)	(0.029)	(0.009)
N		24924	24924	24904	24469	24954	24954	24393
$Y(=0)$ Mean		0.000	0.100	0.037	0.204	0.117	0.000	0.105
B) High genetic obesity risk:								
Born after rationing	β	0.117***	0.028**	0.019**	0.038**	0.025*	0.128***	0.040***
	SE	(0.039)	(0.012)	(0.009)	(0.018)	(0.015)	(0.042)	(0.014)
N		12461	12461	12442	12186	12479	12479	12135
$Y(=0)$ Mean		0.000	0.100	0.050	0.238	0.136	-0.000	0.137
C) Low genetic obesity risk:								
Born after rationing	β	0.035	0.013	0.005	0.011	0.014	0.080*	0.008
	SE	(0.041)	(0.012)	(0.007)	(0.016)	(0.014)	(0.042)	(0.012)
N		12463	12463	12462	12283	12475	12475	12258
$Y(=0)$ Mean		-0.000	0.100	0.025	0.171	0.098	0.000	0.084
$B > C$ (p-value)		0.074	0.185	0.113	0.136	0.304	0.079	0.129

Note. Robust standard errors in parentheses, clustered at the birth quarter level. Non-white adults and those born in 1953q4 - 1954q2 are excluded (in-utero exposure). Abdominal adiposity index is constructed as the average of z-standardized measures of visceral adiposity: trunk fat percentage and waist to hip ratio. Obesity indicator equals 1 for those at or above 90ths percentile of this score. The metabolic disease index is constructed as the weighted average of z-standardized measures of abdominal adiposity, a history of diabetes or hypertension diagnosis, and cholesterol. Metabolic syndrome indicates those being diagnosed with at least 2 out of 4 conditions (abdominal obesity, diabetes, hypertension, cholesterol). Genetic risk for obesity is defined using the individual's polygenic index (PGI) for obesity, split at the sample median (high - at or above, low - below). Outcomes are standardized using the mean and standard deviation of the never-rationed group (A), and separately within genetic and never-rationed subgroups (B, C). p-value < 0.1. * p<0.1, ** p<0.05, *** p<0.01.

Table 6: RDD Estimates of Lifted Rationing on Adiposity by Exposure Duration (UK Biobank)

		Abdominal adiposity (z-score) (1)	Abd. Obesity $\geq 75^{\text{th}}$ pct (2)	Abd. Obesity $\geq 90^{\text{th}}$ pct (3)
A) In utero + [0, 3 mths]:				
Born after rationing	β	0.079***	0.023*	0.029***
	SE	(0.028)	(0.012)	(0.009)
N		24924	24924	24924
B) In utero + [4, 9 mths]:				
Born after rationing	β	0.081**	0.044**	0.057***
	SE	(0.039)	(0.018)	(0.012)
N		9537	9537	9537
Y(=0) Mean		0.000	0.250	0.100
$B > A$ (p-value)		0.481	0.158	0.030

Note. Robust standard errors in parentheses, clustered at the birth quarter level. Non-white adults and those born in 1953q4 - 1954q2 are excluded for panel A (in-utero and up to 3 months postnatal exposure), and those born between 1953q2-1954q4 for panel B (in-utero and up to 9 months postnatal exposure). Outcomes are standardized using the mean and standard deviation of the never-rationed group. Increases in abdominal adiposity at or above the 90th percentile are significantly larger for those exposed up to 9 months postnatally compared to exposure limited to up to 3 months postnatally at $p < 0.1$. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 7: RDD Estimate of Lifted Rationing on Economic Outcomes (ELSA)

		Education Years 16+ (1)	Any College (2)	Employed (3)	Skilled Job (4)	>Median Wealth (5)	Economic sufficiency Index (6)
A) In utero + [0, 9 mths]:							
Born after rationing	β	-0.100**	-0.092**	-0.030	-0.141***	-0.137***	-0.263***
	SE	(0.043)	(0.045)	(0.033)	(0.044)	(0.041)	(0.084)
	Single p -val	0.019	0.040	0.358	0.001	0.001	0.002
	RW p -val	0.000	0.000	0.056	0.000	0.000	0.000
N		12589	12438	12512	10203	11116	12589
B) In utero + [10, 21 mths]:							
Born after rationing	β	-0.171**	-0.156**	-0.050	-0.156**	-0.131*	-0.348**
	SE	(0.072)	(0.075)	(0.051)	(0.075)	(0.068)	(0.137)
	Single p -val	0.018	0.038	0.326	0.037	0.052	0.011
	RW p -val	0.000	0.000	0.000	0.000	0.000	0.000
N		10480	10329	10415	8395	9251	10480
$Y(=0)$ Mean		0.303	0.502	0.700	0.550	0.476	0.000
$B > A$ (p-value)		0.800	0.767	0.630	0.570	0.470	0.701

Note. Sample includes individuals born between 1950-1960 (excluding those born between 1953q4-1954q4) who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Each model is adjusted for sex, age, semester of birth (calendar months 1-6, vs 6-12), parental health history, and survey year indicators. Economic sufficiency indices follow Kling, Liebman, and Katz (2007) and Hoynes et al. 2016 in calculating average across standardized z-score measures of each component. The z-score is calculated by subtracting the mean and dividing by the standard deviation for those born after the end of rationing. Economic sufficiency index includes "positive" outcomes like attending having over 16 years of education, working in medium/high-skilled occupation, being employed, having above-median wealth, and log-transformed wealth. We report estimates with standard errors clustered at the individual level. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$. Romano-Wolf adjusted p-values account for multiple hypothesis testing.

Table 8: RDD Estimate of Lifted Rationing on Childhood Cognitive Outcomes

		Cognition age 7 (1)	Cognition age 9 (2)	Cognition age 12 (3)
Born after rationing	β	-0.081	-0.214***	-0.256***
	SE	(0.046)	(0.047)	(0.045)
	Single p -val			
N		9436	9336	9241

Note. Robust standard errors are reported in parentheses. The sample comprises children born between October 1950 and September 1955 who were resident in Aberdeen and enrolled in primary grades 3–7 in a state school in Aberdeen City at the time of study (December 1962). Children born from October 1953 to June 1954 are excluded. Outcome variables are standardised against the pre-rationing mean, therefore coefficients are interpreted as standard deviation changes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

10 Appendix

A Tables

Table A.1: Summary statistics on survey participants' time-invariant characteristics by genetic risk in UKB

	Low Genetic Risk			High Genetic Risk		
	Rationed %	Never-rationed %	Difference (p.p.)	Rationed %	Never-rationed %	Difference (p.p.)
Age at baseline survey (SD)	55.63 (0.968)	55.06 (0.750)	0.581***	55.62 (0.967)	55.06 (0.735)	0.563***
Male	43.533	44.237	−0.704	43.153	44.536	−1.383
Non-white	6.856	6.859	−0.003	6.761	7.151	−0.391
Family history: diabetes	17.751	16.922	0.829	18.764	17.772	0.992
Family history: CVD	62.751	63.247	−0.496	62.851	63.495	−0.644
Often/very often felt loved as a child	77.420	77.956	−0.536	75.268	75.733	−0.465
Never sexually molested as a child	90.292	90.681	−0.389	88.718	88.708	0.009
Had someone take me to a dr as a child	86.377	85.383	0.994	83.705	83.570	0.135
Long-term antibiotic therapy as a child	15.844	14.089	1.755	16.796	17.164	−0.368
Observations	9,218	10,480	19,698	9,159	10,266	19,425

Notes: This table presents summary statistics separately for individuals with low genetic risk (below median of BMI polygenic score distribution) and high genetic risk (above median of BMI polygenic score distribution). Family history variables have slightly smaller samples due to missing data; these are imputed and controlled for in all regressions. Childhood-related variables such as feeling loved, being taken to the doctor, taking antibiotics as a child, and experiences of sexual molestation are based on smaller samples as these questions were asked of a subset of respondents. Reported means are unadjusted. Statistical significance tests adjust for whether each parent was alive at the time of survey, as rationed individuals are older and thus more likely to have parents who were diagnosed with or died from age-related conditions. Differences between rationed and never-rationed groups are presented in percentage points (except for age presented in years), with statistical significance indicated at Roman-Wolf * $p < 0.05$, ** $p < 0.01$, or *** $p < 0.001$.

Table A.2: Analysis specifications across datasets

Dataset	Running Variable	Excluded	Exposure Windows		Exposure Length at Cut-off		Tables
			Rationed	Never Rationed	Minimum	Maximum	
ELSA	Birth years*	1953q4–	1950m1–	1955m1–	IU (born 1953q3)	IU + 9m	4A, 7A
		1954q4	1953m9	1955m12		(born 1953q1)	
	Birth years	1952q4–	1950m1–	1955m1–	IU + 10m (born 1952q2)	IU + 21m	4B, 7B
		1954q4	1952m6	1955m12		(born 1952q1)	
UK Biobank	Birth quarters	1953q4–	1952m1–	1954m10–	IU	IU + 3m	5, 6A
		1954q2	1953m9	1956m3	(born in Sept 1953)	(born in July 1953)	
	Birth quarters	1953q2–	1952m1–	1954m10–	IU+4m	IU + 6m	5, 6B
		1954q2	1953m3	1956m3	(born in Sept 1953)	(born in March 1953)	
Aberdeen	Birth months	1953m9–	1950m10–	1954m7–	IU (born in Sept 1953)	IU (born in Sept	8
		1954m6	1953m9	1955m9		1953)	

Notes: *Birth quarters for robustness (Table A3, A4, A6). IU = in utero. Running variables indicate temporal aggregation used in main analyses. Exclusions define birth cohorts dropped to avoid ambiguous treatment assignment. Exposure windows show birth date ranges for rationed vs. never-rationed cohorts. Exposure length columns show minimum and maximum sugar rationing exposure (in utero + postnatal) for individuals born at the rationing cutoff, with example birth dates in parentheses.

Table A.3: RDD Estimate of Lifted Rationing on Health (running variable: six-month birth cohorts)

		Metabolic health		Cardiovascular conditions		Inflammation		Comorbidity		Metabol inc (
		Diabetes	Cholesterol	Hypertension	Heart disease & stroke	C-reactive protein>3	Arhtritis	Chronic diseases 3+	Metabolic syndrome	
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	
Panel A: In-utero + [0, 9mths]:										
Born after rationing	β	0.042***	0.052***	0.064***	0.064*	0.006	0.098***	0.118***	0.066***	0.19
	SE	(0.016)	(0.012)	(0.021)	(0.033)	(0.033)	(0.033)	(0.045)	(0.017)	(0.0
N		12589	2435	6351	12588	12589	2481	12589	12589	12
Panel B: In-utero+ [10, 21mths]:										
Born after rationing	β	0.058**	0.068***	0.140**	0.045	0.069***	0.087	0.223***	0.126***	0.30
	SE	(0.023)	(0.013)	(0.060)	(0.035)	(0.025)	(0.086)	(0.044)	(0.017)	(0.0
N		10480	2027	5082	10479	10480	2091	10480	10480	10
Y(=0) Mean		0.076	0.045	0.231	0.293	0.064	0.230	0.252	0.085	-0.

Note. Robust standard errors in parentheses, clustered at the six-month birth cohort level. Clustering at the treatment assignment level yields somewhat smaller standard errors than clustering at the individual level, likely reflecting stronger within-individual serial correlation and limited within-cohort variation. Results remain very similar: estimates that are statistically significant with cohort-level clustering generally remain significant or borderline significant when clustering at the individual level. Adults born in 1953q4 and 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Each model is adjusted for sex, age, seasonality at birth and survey year indicators. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.4: RDD Estimate of Lifted Rationing on Economic Outcomes (running variable: six-month birth cohorts)

		Any College (1)	Education Years 16+ (2)	Employed (3)	Skilled Job (4)	>Median Wealth (5)	Economic sufficiency Index (6)
Panel A: In-utero + [0, 9mths]:							
Born after rationing	β	-0.092*** (0.020)	-0.086*** (0.026)	-0.022 (0.026)	-0.119*** (0.031)	-0.132*** (0.025)	-0.241*** (0.052)
	SE						
<i>N</i>		12589	12438	12512	10203	11116	12589
Panel B: In-utero+ [10, 21mths]:							
Born after rationing	β	-0.165*** (0.032)	-0.161*** (0.034)	-0.057*** (0.019)	-0.145** (0.068)	-0.129*** (0.037)	-0.345*** (0.108)
	SE						
<i>N</i>		10480	10329	10415	8395	9251	10480
Y(=0) Mean		0.303	0.502	0.700	0.550	0.476	0.000

Note. Robust standard errors in parentheses, clustered at the six-month birth cohort level. Clustering at the treatment assignment level yields somewhat smaller standard errors than clustering at the individual level, likely reflecting stronger within-individual serial correlation and limited within-cohort variation. Results remain very similar: estimates that are statistically significant with cohort-level clustering generally remain significant or borderline significant when clustering at the individual level. Adults born in 1953q4 and 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Adults born in 1953q4 and 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Each model is adjusted for sex, age, seasonality at birth and survey year indicators. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.5: RDD Estimates of the Effect of Lifting Rationing on Placebo Health Outcomes

DV: 1 0		Lung disease (1)	Asthma (2)	Cataracts (3)
Panel A: In-utero:				
Born after rationing	β	0.006	0.016	0.007
	SE	(0.017)	(0.032)	(0.016)
	Single p -val	0.730	0.625	0.664
	RW p -val	0.607	0.607	0.607
N		12589	12589	10617
Panel B: In-utero+1yr:				
Born after rationing	β	0.027	0.032	-0.001
	SE	(0.023)	(0.052)	(0.027)
	Single p -val	0.239	0.539	0.966
	RW p -val	0.200	0.400	1.000
N		10480	10480	8767
Y(=0) Mean		0.040	0.145	0.036

Table A.6: Placebo cutoffs for health and economic outcomes

DV: 1 0	Diabetes (1 0)	Chronic disease 3+ (1 0)	Metabolic index (z-score)	Any college (1 0)	Skilled job (1 0)	>Median wealth
	(1)	(2)	(3)	(4)	(5)	(6)
A: 1950-1953 vs. 1955-1960 (main model, cut-off Apr-Sept 1953)						
Born after 1954	0.044** (0.021)	0.068*** (0.022)	0.209*** (0.077)	-0.100** (0.043)	-0.127*** (0.044)	-0.138*** (0.041)
<i>N</i>	12589	12589	12589	12589	10203	11116
<i>Y</i> (=0) Mean	0.076	0.085	-0.000	0.253	0.565	0.460
B: 1955-1957q3 vs. 1957q4-1960 (After rationing, cut-off Apr-Sep 1956)						
Born after 1956q3	-0.022 (0.035)	-0.014 (0.033)	-0.108 (0.136)	0.023 (0.052)	-0.026 (0.062)	-0.010 (0.064)
C: 1955-1958q3 vs. 1958q4-1960 (After rationing, cut-off Apr-Sep 1957)						
Born after 1957q3	0.019 (0.028)	-0.002 (0.027)	0.138 (0.123)	0.057 (0.042)	-0.047 (0.055)	0.033 (0.058)
D: 1955-1958q3 vs. 1958q4-1960 (After rationing, cut-off Apr-Sep 1958)						
Born after 1958q3	-0.003 (0.028)	-0.005 (0.025)	0.059 (0.119)	0.005 (0.043)	-0.040 (0.055)	0.004 (0.058)
<i>N</i>	6537	6537	5042	6537	5703	4415
<i>Y</i> (=0) Mean	0.076	0.081	-0.000	0.244	0.570	0.460

Note. Robust standard errors in parentheses, clustered at the individual level. The running variable in Models B–D is defined at the six-month birth cohort level. Model A presents results from Tables 3 and 4 for comparison. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.7: Vary RDD Bandwidth: RDD estimates of the effect of lifting rationing on health

DV: 1 0	Diabetes (1 0)	Cholesterol (1 0)	Heart disease or stroke	Arthritis (1 0)	Inflammation (1 0)	Chronic diseases (2+)	Metabolic index (z-score)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: left 3, right 3 year bandwidth							
Born after 1954	0.046 (0.029)	0.052 (0.054)	-0.010 (0.033)	0.045 (0.051)	0.072 (0.060)	0.070 (0.050)	0.189* (0.110)
<i>N</i>	8904	4778	8904	8904	1796	8904	8904
Panel B: left 4, right 4 year bandwidth							
Born after 1954	0.041* (0.024)	0.070 (0.045)	0.008 (0.027)	0.096** (0.042)	0.089* (0.050)	0.098** (0.041)	0.199** (0.091)
<i>N</i>	11662	6014	11662	11662	2342	11662	11662
Panel C: left 4, right 5 year bandwidth							
Born after 1954	0.042* (0.023)	0.069 (0.042)	0.006 (0.026)	0.097** (0.039)	0.101** (0.046)	0.102*** (0.038)	0.201** (0.084)
<i>N</i>	12168	6250	12168	12168	2435	12168	12168

Note. Robust standard errors in parentheses, clustered at the individual level. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Bandwidth used for regression discontinuity varies by panel; *N* shows effective estimation sample size. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.8: Vary RDD Bandwidth: RDD estimates of the effect of lifting rationing on economic outcomes

	Education Years 16+ (1 0)	Employed (1 0)	Skilled Job (1 0)	>Median Wealth (1 0)	Economic sufficiency Index (z-score)
	(1)	(2)	(3)	(4)	(5)
A: left 3, right 3 year bandwidth					
Born after rationing	-0.088 (0.060)	-0.034 (0.047)	-0.138* (0.062)	-0.136* (0.059)	-0.267* (0.120)
<i>N</i>	8904	8845	7361	7818	8904
B: left 4, right 4 year bandwidth					
Born after rationing	-0.100* (0.049)	-0.032 (0.038)	-0.132** (0.051)	-0.141** (0.048)	-0.273** (0.098)
<i>N</i>	11662	11591	9447	10313	11662
C: left 4, right 5 year bandwidth					
Born after rationing	-0.101* (0.046)	-0.034 (0.036)	-0.130** (0.048)	-0.148** (0.045)	-0.281** (0.092)
<i>N</i>	12168	12095	9865	10754	12168

Note. Robust standard errors in parentheses, clustered at the individual level. Not adjusted for multiple hypothesis testing. Sample includes individuals born between 1950-1960 aged 50-64 years, surveyed in ELSA at least once between 2002-2019. Bandwidth used for regression discontinuity varies by panel; ; *N* shows effective estimation sample size. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.9: Vary Kernel: RDD estimates of the effect of lifting rationing on health

DV: 1 0	Diabetes (1 0)	Cholesterol (1 0)	Heart disease or stroke (1 0)	Arthritis (1 0)	Inflammation (1 0)	Chronic diseases (2+)	Metabolic index (z-score)
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
A: Triangular kernel, first order polynomial (main specification)							
Born after rationing	0.044** (0.021)	0.073* (0.039)	0.007 (0.024)	0.122*** (0.036)	0.112** (0.043)	0.114*** (0.035)	0.209*** (0.077)
B: Uniform kernel, first order polynomial							
Born after rationing	0.044** (0.021)	0.075** (0.038)	0.008 (0.023)	0.133*** (0.035)	0.115*** (0.043)	0.120*** (0.035)	0.212*** (0.077)
C: Epanechnikov kernel, first order polynomial							
Born after rationing	0.044** (0.021)	0.075* (0.038)	0.008 (0.023)	0.129*** (0.036)	0.113*** (0.043)	0.117*** (0.035)	0.210*** (0.077)

Note. Robust standard errors in parentheses, clustered at the individual level. Not adjusted for multiple hypothesis testing. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Cardiovascular is an indicator for hypertension, stroke, or heart disease compared to diagnosis with none of these diseases. Chronic 2+ and chronic 3+ are indicators for at least two or three of: hypertension, stroke, heart disease, diabetes, cancer, arthritis, high cholesterol, compared to diagnosis with none of these diseases. Each model is adjusted for age. Kernel used for regression discontinuity varies by panel. Bandwidth is 4 years to the left of cut off, and 6 years to the right in all models. * p<0.1, ** p<0.05, *** p<0.01.

Table A.10: Vary Kernel: RDD estimates of the effect of lifting rationing on economic outcomes

	Education Years 16+ (1 0)	Employed (1 0)	Skilled Job (1 0)	>Median Wealth (1 0)	Economic sufficiency Index (z-score)
	(1)	(2)	(3)	(4)	(5)
A: Triangular kernel, first order polynomial (main specification)					
Born after rationing	-0.100** (0.043)	-0.026 (0.033)	-0.127*** (0.044)	-0.138*** (0.041)	-0.259*** (0.084)
B: Uniform kernel, first order polynomial					
Born after rationing	-0.100** (0.042)	-0.022 (0.032)	-0.125*** (0.043)	-0.134*** (0.041)	-0.249*** (0.083)
C: Epanechnikov kernel, first order polynomial					
Born after rationing	-0.100** (0.042)	-0.025 (0.032)	-0.126*** (0.043)	-0.137*** (0.041)	-0.256*** (0.083)

Note. Robust standard errors in parentheses, clustered at the individual level. Not adjusted for multiple hypothesis testing. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Any higher education is an indicator for if respondent attended at least some post-secondary schooling. Skilled job is conditional on current employment, and was classified using the UK Standard Industrial Classification 8-code system and is compared to low-skilled occupations. Each model is adjusted for age. Kernel used for regression discontinuity varies by panel. Bandwidth is 4 years to the left of cut off, and 6 years to the right in all models. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.11: Falsification test: health outcomes by migraine genetic risk

	Obesity 90th pct	Metabolic syndrome (1 0)	Metabolic health index (z-score)
	(1)	(2)	(3)
A: Low migraine genetic risk (below median PGS)			
Born after rationing β	0.025**	0.024*	0.094**
SE	(0.012)	(0.013)	(0.041)
N	12480	12227	12493
$Y(=0)$ Mean	0.100	0.103	-0.011
B: High migraine genetic risk (above median PGS)			
Born after rationing β	0.033***	0.029**	0.095**
SE	(0.013)	(0.013)	(0.042)
N	12444	12166	12461
$Y(=0)$ Mean	0.100	0.107	0.011

Note. Robust standard errors in parentheses. Sample includes white individuals born between birth years excluding 1916-1919. Low and high risk refer to below and above median polygenic scores for migraine susceptibility. Body Size p90 indicates top 10% of body size distribution. Met Syn 2+ indicates presence of 2+ metabolic syndrome components. Met Syn Abd indicates abdominal obesity component. All models use triangular kernel with bandwidth of 12 years, first-order polynomial, and include covariates. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table A.12: Policy changes around rationing cutoff: Differential effects at the RDD threshold

Reform/Policy	Timing	Assessment at the 1953Q4/1954Q1 Cutoff	Discont.?
EDUCATIONAL POLICIES			
School leaving age raised to 15	1-Apr-47	Applied to all cohorts near the cutoff; no change in compulsory schooling at 1953Q4/1954Q1.	No
11+ examination (Tripartite)	Late 1940s-1950s	Operated continuously across cohorts near the cutoff; local variation existed but not discontinuously at 1953Q4/1954Q1.	No
Comprehensive school reorganization (DES Circular 10/65)	From 1965; staggered by LEA	Introduced years later and staggered locally; exposure changes gradually by cohort and region, not at the birth cutoff.	No
School leaving age raised to 16 (ROSLA)	1-Sep-72	Affected later cohorts, mainly Sept 1957+ births; no discontinuity for cohorts born immediately around 1953Q4/1954Q1.	No
HEALTH POLICIES			
NHS prescription charges introduced	1952	In place before the cutoff; cohorts born immediately before and after 1953Q4/1954Q1 faced the same regime.	No
NHS prescription charges abolished	1965	Occurred later and affected ages smoothly; no change in exposure at the birth cutoff.	No
NHS prescription charges reintroduced	1968	Occurred later with exemptions; exposure varies by age and health status, not discontinuously at the cutoff.	No
Diphtheria immunization	Routine by early 1950s	Routine before the cutoff; no discrete change in vaccine access for cohorts born around 1953Q4/1954Q1.	No
Polio vaccination (IPV then OPV)	1956 onward; OPV 1962	Introduced after the cutoff and rolled out by age eligibility; exposure changes smoothly with birth cohort, not sharply at 1953Q4/1954Q1.	No
NHS Health Check (CVD screening)	2009 pilot; 2012-13 rollout	Implemented decades later for broad adult age ranges; no birth-cohort discontinuity at 1953Q4/1954Q1.	No
ECONOMIC AND ENVIRONMENTAL SHOCKS			
Korean War rearmament & price shock	1950-1952	Pressures were already subsiding before the cutoff; no discrete economic shock at 1953Q4/1954Q1.	No
1970s oil shocks	1973 and 1979	Cohorts near the cutoff experienced these shocks at nearly identical ages; no discontinuity at birth cutoff.	No

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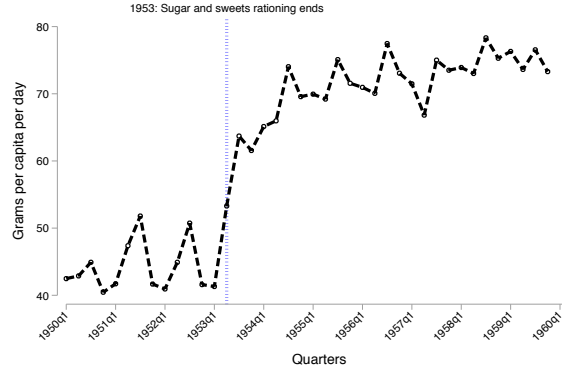
Table A.12 – *Continued from previous page*

Reform/Policy	Timing	Assessment at the 1953Q4/1954Q1 Cutoff	Discont.?
Clean Air Act following Great Smog	Act 1956; phased rollout	Implemented after the cutoff and phased in gradually; cohorts near the cutoff had similar early-life exposure.	No
SOCIAL POLICIES			
School milk changes	Secondary 1968; Primary 1971	Changes occurred much later and applied by school stage; no discontinuity for births around 1953Q4/1954Q1.	No
National Service ends	Wind-down 1957-1963	Applied to older male cohorts only; all cohorts near the cutoff were too young.	No
Postwar housing reconstruction	1950s-1960s	Housing improved gradually over time; no sharp change at the birth cutoff.	No

A Figures

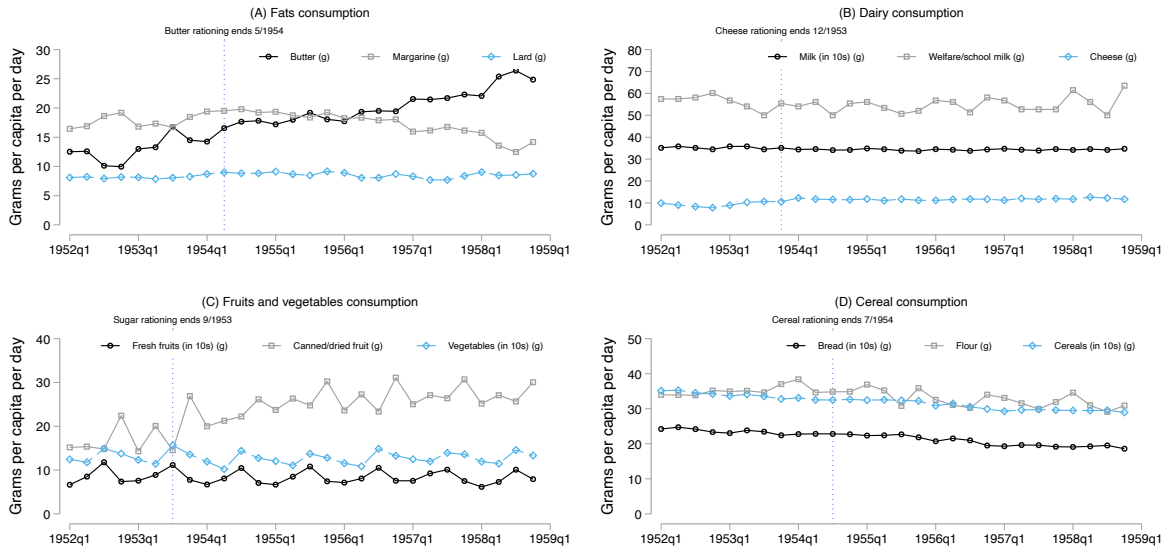
Figure A.1: Grams of daily sugar intake per capita for adults between 1950 and 1960

(a) Daily per capita intake of sugar



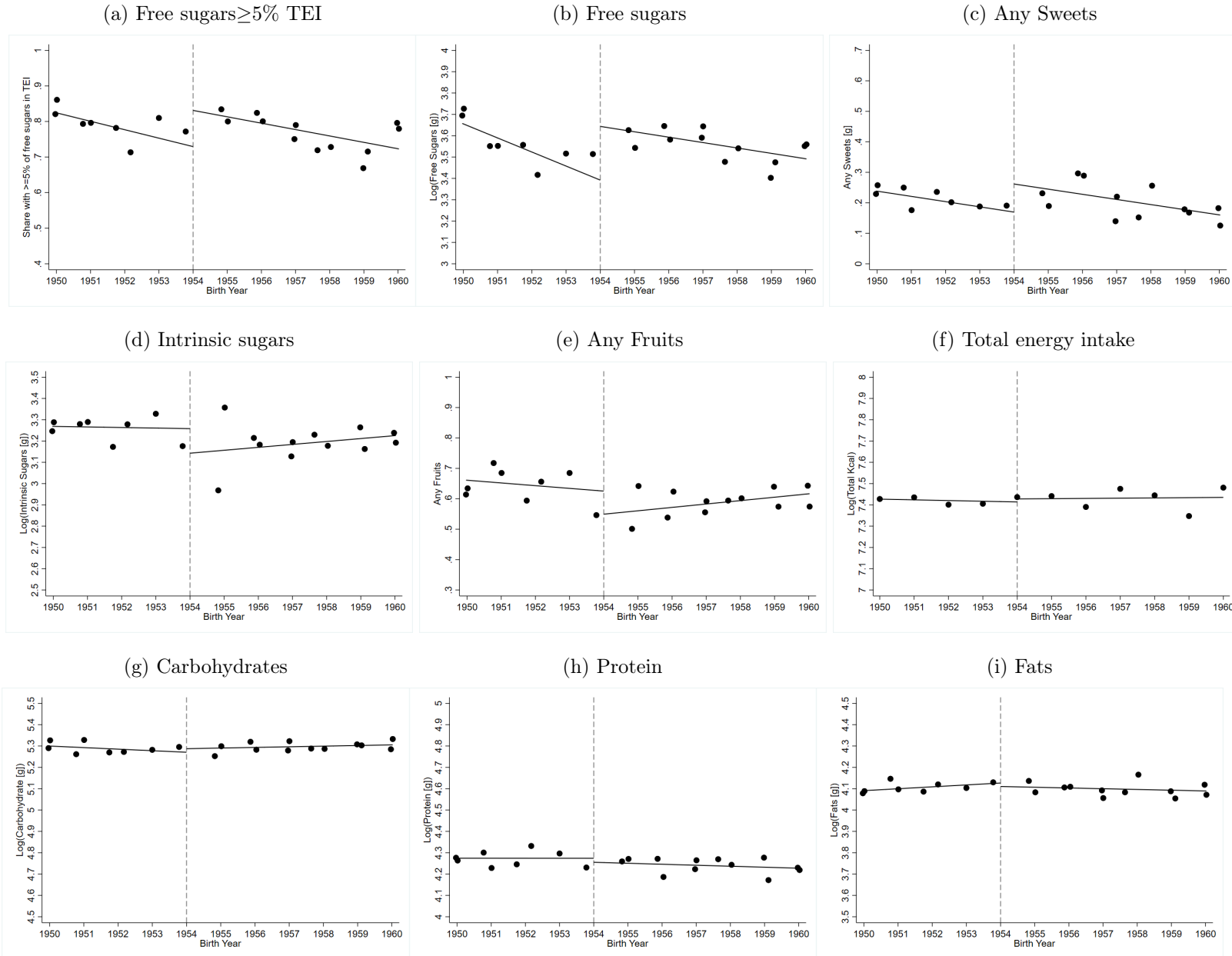
Note: These figures have been previously published in (Gracner et al., 2024).

Figure A.2: Consumption of various food groups between 1950-1960



Note: These figures are replications from already published work in Gracner et al. (2024).

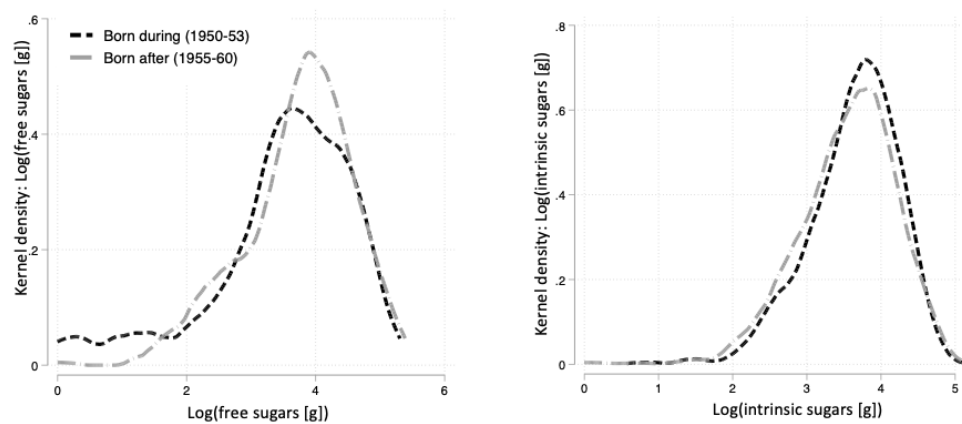
Figure A.3: 2008-2019 diet among today's older adults born between 1950-1960



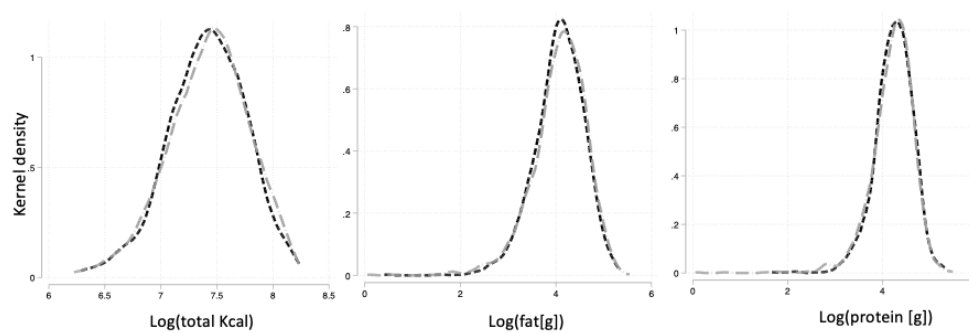
Note: Authors' calculations using the National Diet and Nutrition Survey (2008-2019).

Figure A.4: Changes in total calorie and sugar calorie daily intake per capita between 1950 and 1960

(a) (A) Sugars: free and intrinsic



(b) (B) Total calories, fats and protein



Note: Author's calculations using National diet and nutritional survey.

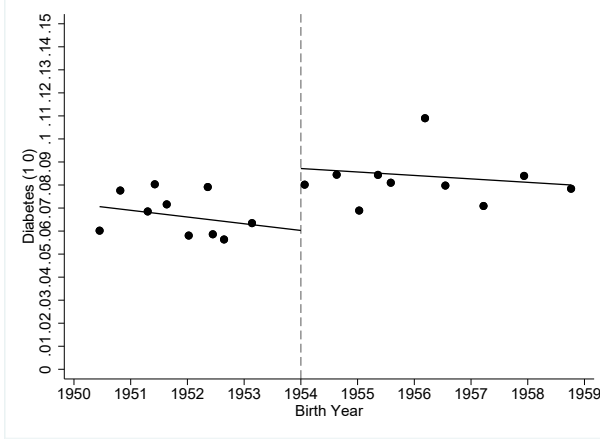
Figure A.5: Age overlap between cohorts born between 1950-1960 in ELSA 2002-2018

Survey year	Birth year (common support: ages <65)										
	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	63	62	61	60	59	58	57	56
2018	68	67	66	65	64	63	62	61	60	59	58

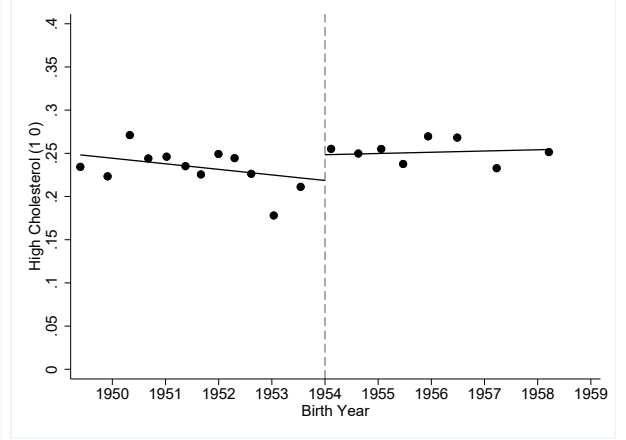
Note: Based on nine waves of ELSA 2002-2018.

Figure A.6: ELSA: Current health outcomes across birth cohorts

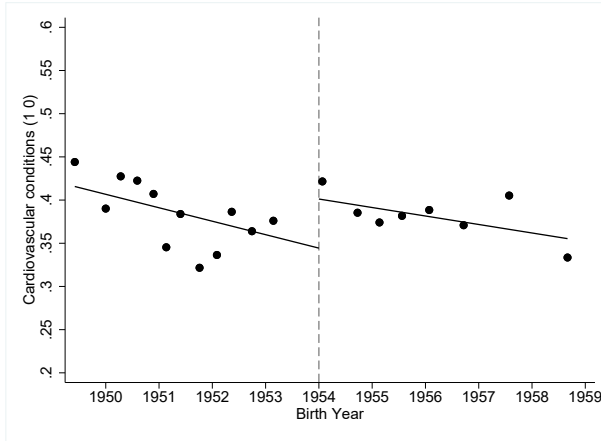
(a) Diabetes (1 0)



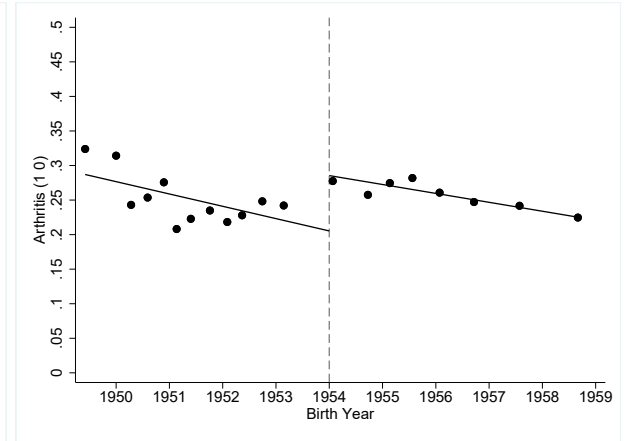
(b) Cholesterol (1 0)



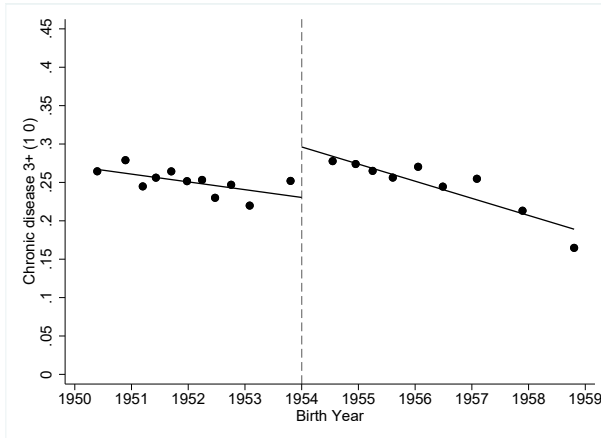
(c) Cardiovascular cond. (1 0)



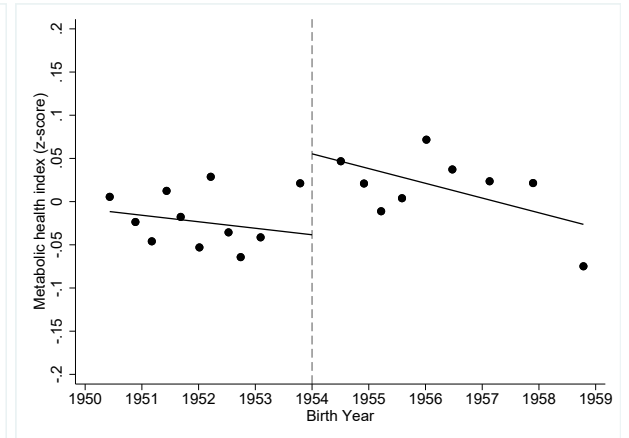
(d) Arthritis (1 0)



(e) Chronic cond. 2+ (1 0)

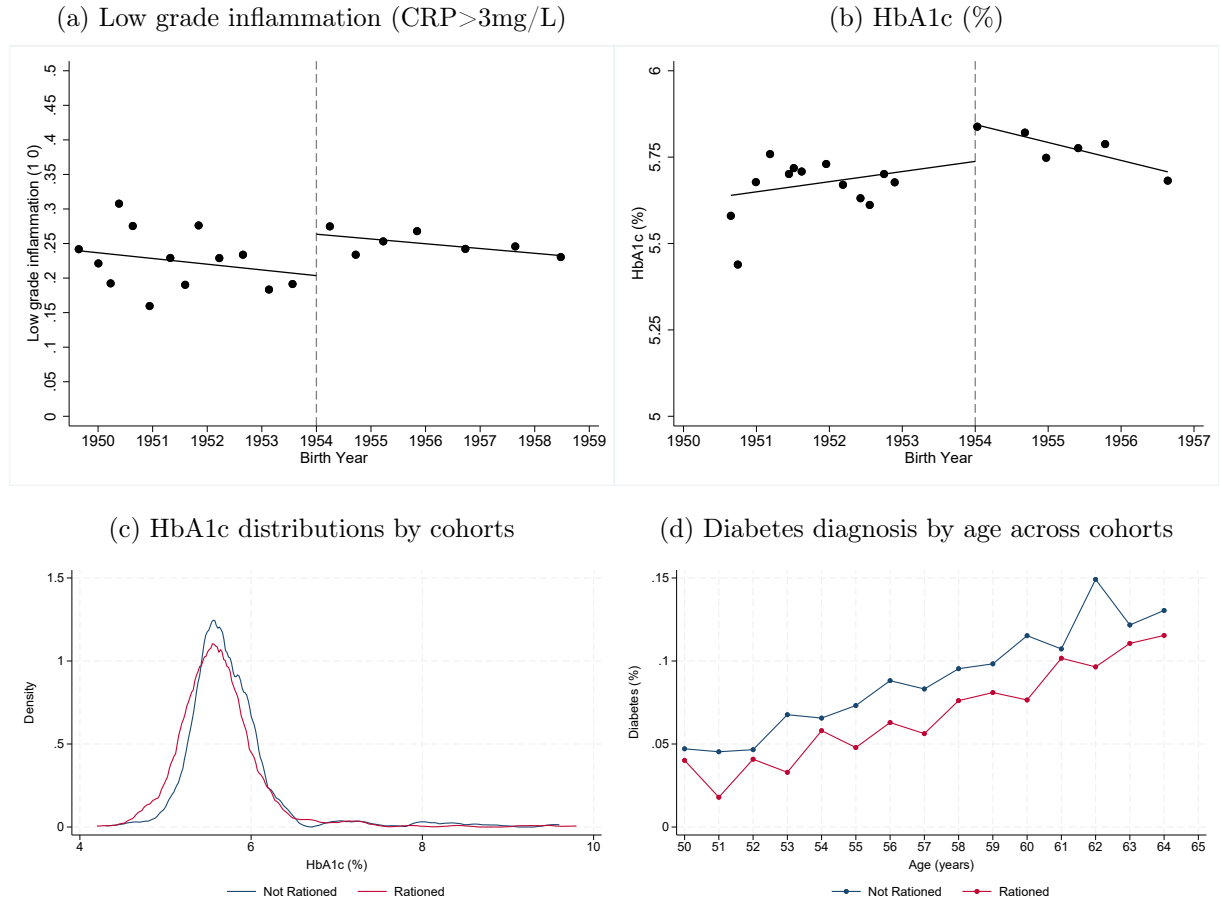


(f) Metabolic health index (z-score)



Note: Cohort difference in probability of diet-related disease diagnosis. Adjusted for age, sex, birth quarter, and parental disease history. Cardiovascular condition indicates diagnosis of hypertension, stroke, or heart disease. Chronic condition 2+ indicates diagnosis of two or more conditions (diabetes, hypertension, high cholesterol, heart disease, arthritis). Metabolic health index is the average of z-scores (standardized to post-rationing cohort) for diabetes, high cholesterol, heart disease, stroke, and hypertension.

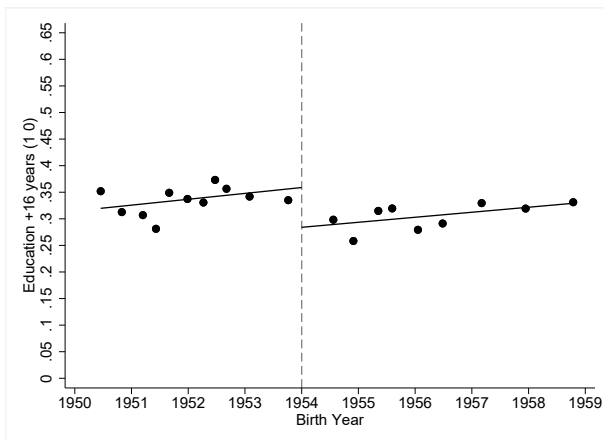
Figure A.7: ELSA: Current health outcomes across birth cohorts - biomarkers.



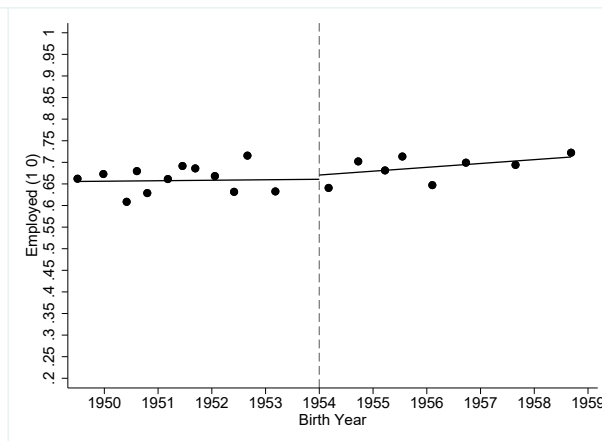
Note: Cohort difference in diet-related disease outcomes, measured via biomarkers in ELSA. Models adjusted for age, sex, and parental disease history indicators. Low-grade inflammation is defined as C-reactive protein >3 mg/L at time of survey. CRP values >10 mg/L are excluded as they likely reflect acute infection rather than chronic inflammation. The distribution of HbA1c, limited to 52-56 before many people get diagnosed, differed significantly between rationed and non-rationed individuals (Kolmogorov-Smirnov $D = 0.149$, $p < 0.001$).

Figure A.8: ELSA: Current economic outcomes across birth cohorts

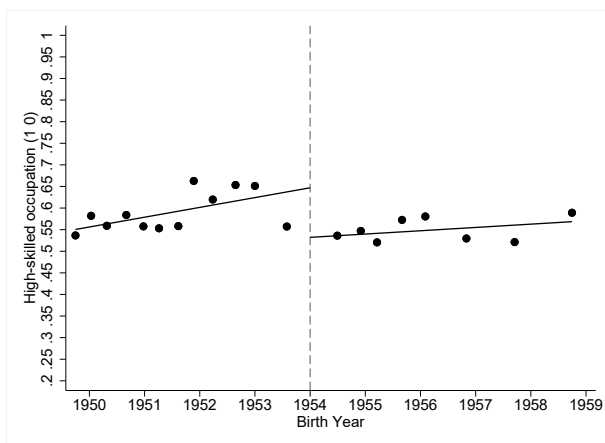
(a) Education 16yrs+ (1 0)



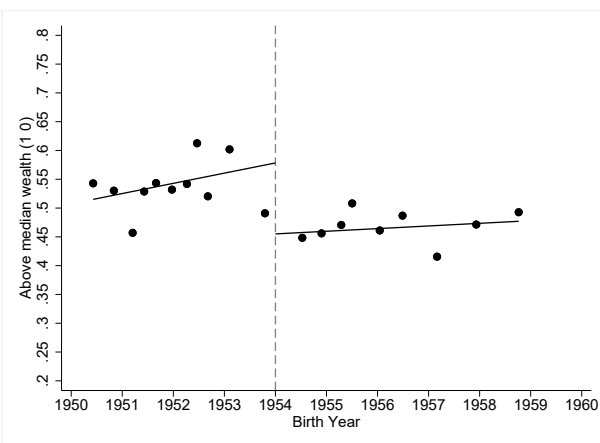
(b) Employed (1 0)



(c) High skilled occupation (1 0)



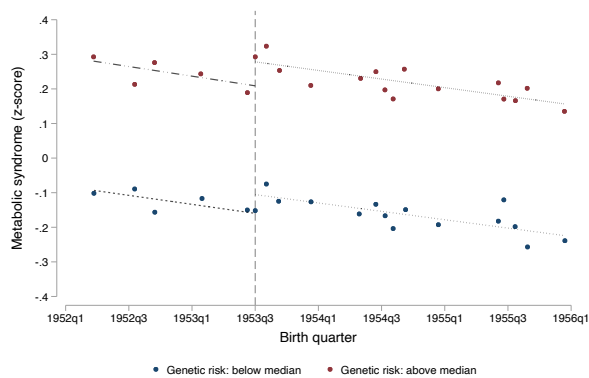
(d) Above median wealth (1 0)



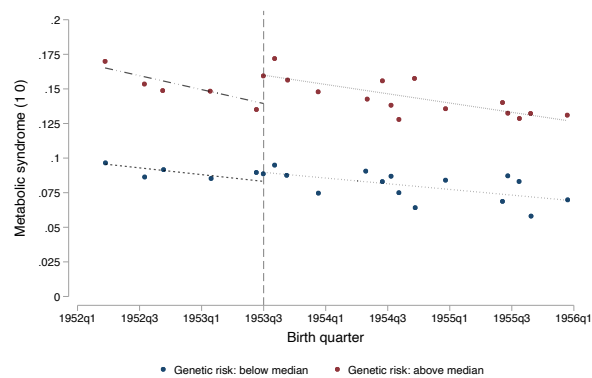
Note: Cohort difference in selected economic outcomes. Adjusted for age, sex, first calendar quarter of birth, and parental health indicators. Data: The English Longitudinal Survey of Aging (2002-2019).

Figure A.9: UK Biobank: Health outcomes by genetic risk for obesity

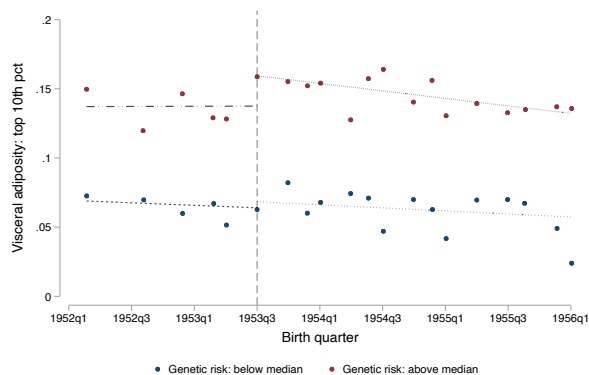
(a) Metabolic Disease Index (z-score)



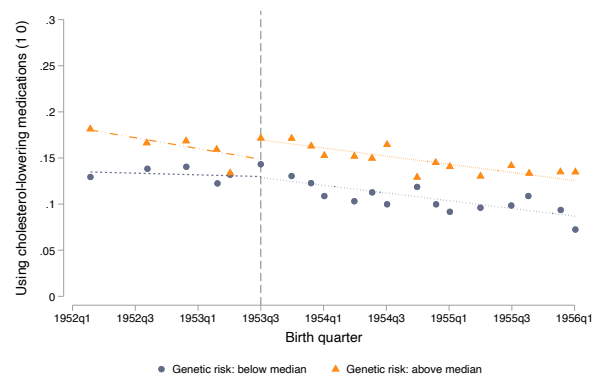
(b) Metabolic Syndrome (1 0) pct



(c) Abd.Obesity 90th pct



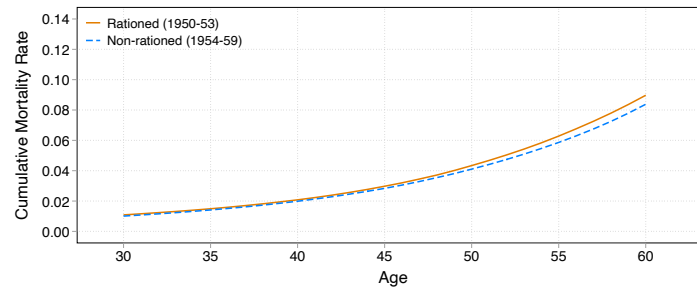
(d) Cholesterol-lowering medication use



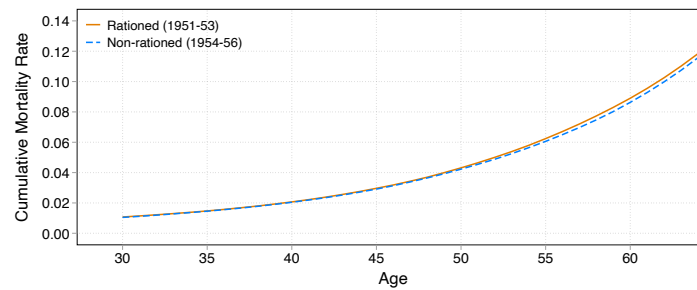
Note: Authors' calculations based on UK Biobank data.

Figure A.10: Mortality rates for rationed and never rationed cohorts

(a) Cumulative mortality rates (ELSA)



(b) Cumulative mortality rates (UKB)



Note: Based on data on live births and total deaths in each cohort (1950-1959) collected between calendar years 1965-2019 by the Office for National Statistics, National Records of Scotland, Northern Ireland Statistics and Research Agency. Mortality rates are calculated for England and Wales.