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

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Abstract

We exploit the end of post-WWII sugar rationing in the UK to estimate the long-run effects of early-life sugar exposure. Using a regression discontinuity design, we find that unrestricted sugar increased adult chronic inflammation, diabetes, high cholesterol, hypertension, arthritis, and multi-morbidity, while reducing childhood cognitive development, educational attainment, skilled employment, and wealth. Benefits are larger with longer exposure to rationing and higher genetic risk of obesity. One pathway is persistent dietary habits; never-rationed adults consume more free sugar at similar caloric intake. The rationed diet closely mirrors current dietary guidelines, which our findings support.

Key words: early-life nutrition; sugar; food rationing; regression discontinuity; developmental origins; human capital, health

JEL Codes: I12, I14, I18, J13, J14, J24

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Free sugar intake, a key risk factor for obesity, diabetes, and cardiovascular disease, exceeds levels recommended by nutritional guidelines for a large share of the population (Vos et al., 2017; Ludwig, Peterson and Gortmaker, 2001; Fung et al., 2009; Ackerman et al., 2005; Larsson, Bergkvist and Wolk, 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 (total energy intake – TEI) and many more exceed the WHO strict limit of 5% of TEI, which maximizes long-term health (WHO, 2015*b*; Powell, Smith-Taillie and Popkin, 2016; Bowman et al., 2017; Knüppel et al., 2017).[2]

Sugar-rich diets begin early in life through maternal consumption of sugary foods and drinks during pregnancy and breastfeeding (Goran, Plows and Ventura, 2019; Portella et al., 2012), and through sugar rich commercially prepared beverages and foods for infants and toddlers (Walker and Goran, 2015). In the US pregnant women consume an average of 21 teaspoons of added sugars daily (Cioffi, Figueroa and Welsh, 2017) and more than half of lactating mothers exceed the maximum daily limit of 10% of TEI (Goran, Plows and Ventura, 2019). Despite nutritional guidelines that recommend zero free sugars for children below the age of two (Dewey et al., 2021), 61% of infants and 98% of toddlers consume some free sugar daily and their intake increases with age (Park et al., 2014; Fiorito et al., 2010). By age two, sugar consumption exceeds the daily limit for adults (Herrick et al., 2020; Vos et al., 2017). This 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, 2015*b*). In Mexico and Chile, most toddlers consume sweetened beverages (Essman et al., 2018; Afeiche et al., 2018*b*) and by age five consume more than 10 teaspoons of free sugar per day (Afeiche et al., 2018*a*; Herrick et al., 2020). Despite growing concern about these trends, the long-term implications of excessive sugar intake early in life are not well understood.

This paper provides 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 WWII 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). Although children's sugar intake during rationing was within the levels recommended by WHO today, it more than doubled after 1953, along with a deterioration of dental health (Jackson, 1974; Zweiniger-Bargielowska, 2000*b*; Hollingsworth, 1974, 1983; Prynne et al., 1999). Data from the National Food Survey (NFS) data from the 1950s show a sharp increase in sugar consumption, from below 40 grams per person per day to more than 80 grams. Other foods and nutrients did not show comparable jumps, changing little during the same period. There was a modest increase in total calorie consumption of approximately 5%, almost all coming from sugar (Department for Environment, Food and Rural Affairs, 2016).

[1] Free 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).

[2] Ten precent of TEI is equivalent to about six to seven teaspoons of free sugar per day.

We employ a local linear regression discontinuity design (RDD) to examine the long-run effects of the sharp increase in sugar availability after rationing ended. We compare cohorts born during rationing (1950–1953) with cohorts born after rationing ended (1955–1959), excluding cohorts around the September 1953 transition that were only partially exposed to rationing during fetal or early-postnatal periods. We analyze data from four primary sources: (i) the English Longitudinal Study of Aging (ELSA, 2002–2019) for later-life health and economic outcomes of adults ages 50–64; (ii) UK Biobank (UKB) for objectively measured (obesity), metabolic health, and genetic heterogeneity for later-life health outcomes of adults ages 53–56; (iii) Aberdeen childhood test-score data for cognitive development at ages 7-11; and (iv) the National Diet and Nutrition Survey (NDNS, 2008–2016) for dietary patterns during adulthood.

We find that greater exposure to a sugar-rich environment in early life worsened adult metabolic health more than five decades later. In ELSA, cohorts born after the end of rationing had a higher prevalence of diabetes by 4.4 percentage points (pp), equivalent to 54% of the prevalence among never-rationed cohorts, as well as elevated cholesterol (7.3pp, 30%), hypertension (6.8pp, 23%), chronic inflammation (11.2pp, 46%), arthritis (12.2pp, 48%), and having at least three chronic diseases related to diet (6.8pp, 77%). An aggregate metabolic disease index rose by 0.21 standard deviations (SD), indicating worse overall metabolic health. These findings are corroborated with UK Biobank data, where cohorts born after rationing had higher abdominal adiposity and higher prevalence of diabetes, hypertension, elevated cholesterol, and metabolic syndrome, together with worse aggregate metabolic health.

Using data from UK Biobank we examine heterogeneity by genetic susceptibility. The adverse effects of elevated sugar in early childhood are concentrated among people at a higher genetic risk of obesity, while the estimates for lower-risk individuals are small and not statistically significant. The results suggest that lowering early childhood sugar can mitigate long-term genetic risk.

The evidence on the duration of exposure to rationing is more nuanced. Point estimates generally grow when never-rationed cohorts are compared with cohorts protected by rationing for longer periods, but the differences are not always precise. In ESLA, increasing exposure to rationing by 9 months in the post natal period is statistically significant for heart disease or stroke, arthritis, and multiple chronic diseases. In UK Biobank, the clearest dose-response pattern is for adiposity.

We also examine the effect of early childhood sugar exposure on human capital and economic outcomes throughout the life course. The effects emerge well before adulthood and persist into later life in terms of education, labor market, and wealth accumulation. In the Aberdeen data, children never exposed to rationing scored 0.21 SD lower on cognitive tests at age 9 and 0.26 SD lower at age 11; both gaps are statistically significant. These early cognitive gaps persisted into educational attainment. Compared to people protected by rationing, never-rationed individuals were less likely to complete 16 or more years of education (10.0 pp, 33%) and attend college (9.2 pp, 18%). In their 50s, the never-rationed group was less likely to hold a skilled occupation (14.1 pp, 26%) and to have accumulated wealth above median (13.7 pp, 29%). Overall, the never-rationed group had a 0.26 SD lower economic sufficiency index.

What drives these long-lasting impacts of excessive sugar early in life? One pathway may be altered physiological programming due to adverse nutrition, also known as the developmental origins or Barker hypothesis (Martyn and Barker, 1994). In animal models, maternal diets rich in sugars and refined carbohydrates program metabolic changes in offspring leading to adiposity, insulin resistance, and hepatic steatosis (Samuelsson et al., 2008; Vickers et al., 2000; Bayol et al., 2008; Roncal-Jimenez et al., 2011; Rosenfeld, 2015). These conditions arise independently of total caloric intake and are epigenetic in nature.

A second pathway may be lifelong persistence in elevated sugar consumption. 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, Savage and Ventura, 2007; Jayasinghe et al., 2017; Birch, 1999). Sugar can also produce craving and withdrawal symptoms similar to opioids (DiNicolantonio, O'Keefe and Wilson, 2018; Avena, Rada and Hoebel, 2009). Early exposure to a sugar-rich environment early in life can, 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 throughout 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.[3] The end of rationing increased the likelihood of exceeding the WHO strict recommended free sugar limit of 5% of TEI by 10.2pp. Importantly, we do not observe long-term effects on total calories or other food categories (protein, carbohydrates, or fat). However, the end of rationing did decrease the intake of intrinsic sugars and fiber from whole fruits and vegetables. The evidence points to a shift in the composition of diet by substituting sweetness in healthier fruits and vegetables that have intrinsic sugars and fiber with unhealthy free sugar added sweet-tasting foods.

These findings are also consistent with the literature linking dietary exposures to cognitive development. Animal studies show that excessive sugar intake during sensitive periods can alter hippocampal structure and function, a brain system 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). Human studies link a higher prenatal and childhood sugar exposure, especially from sugar-sweetened beverages, to lower cognition in early and mid-childhood (Cohen et al., 2018), and maternal hyperglycemia or sugar-rich diets to poorer executive function, perceptual reasoning, and memory in offspring (Casas et al., 2013; Tanda et al., 2013; Hinkle et al., 2012; Neggers et al., 2003). Evidence from school-aged children similarly links poorer diet quality and increased consumption of energy-dense, nutrient-poor foods to lower academic achievement (Florence, Asbridge and Veugelers, 2008; Burrows et al., 2017). The most plausible pathways run through neuro-cognitive processes central to learning: nutrition and developmental

[3]This is comparable to the free sugar in about three Oreo cookies or half of an 8 oz soda per day.

neuroscience point to attention, memory, executive function, metabolic regulation, and hippocampal development as mechanisms through which early dietary exposures can affect school performance (Nyaradi et al., 2013).

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. In previous work, we used UK Biobank data and a reflexive before-and-after framework to show that exposure to sugar rationing in the first 1,000 days of life is associated with lower risk of type 2 diabetes and hypertension(Gracner, Boone and Gertler, 2024). Subsequent work has replicated these findings and extended them to cardiovascular, respiratory, and cancer outcomes (Zheng et al., 2025*b*; Tang et al., 2026; Zheng et al., 2025*a*; Zhu and Zhang, 2025; Tang et al., n.d.). This paper complements and extends that evidence in four ways. First, we strengthen identification by using a local linear regression discontinuity design tailored to early-life exposure timing, rather than before-and-after cohort comparisons. Second, we expand the analysis to three datasets with different sampling frames: ELSA, the UK Biobank, and the Aberdeen cohort. Third, we substantially broaden the set of outcomes. In health we expand from a few adult diseases to a more complete set of metabolic, inflammatory, and obesity outcomes. We also expand the analysis to consider human capital accumulation, labor market, and wealth. We further expand the work back in time to childhood, where we observe cognitive test scores and educational outcomes decades earlier than the prior work has looked. Unlike previous work, we rely, where possible, on objectively measured outcomes such as HbA1c, C-reactive protein, adiposity, and cognitive test scores. Third, we use nutritional surveys to assess lifelong diet as a potential mechanism. Fourth, we examine heterogeneity by genetic predisposition to obesity and by length of exposure to rationing. Together, these sharpen identification and broaden the scope to a more complete picture of health and economic well-being.

A related study examines an earlier temporary 1949 confectionery de-rationing in the UK, a brief and supply-constrained increase in sweets availability while raw sugar and most other foods remained rationed (van den Berg, von Hinke and Wang, 2025). In that setting, the first stage was sharp but short-lived: confectionery sales rose rapidly, but many shops ran out of stock within weeks. Prenatal exposure there raised education and lowered later-life BMI, which the authors read as adaptation to a still calorically constrained environment. Our setting differs: the 1953 end of rationing was permanent, followed the easing of broader caloric constraints, and raised total energy intake by only about 5%, most of it sugar. The treatment is thus a sugar-dominated increase in availability within an already adequate food environment—neither a pure compositional shift nor the relaxation of a binding constraint.

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, 1998; Almond, Currie and Duque, 2018; Roseboom, de Rooij and Painter, 2006; Ravelli et al., 2005; Barker, 1992), or on broader interventions with nutritional components, including SNAP (Hoynes, Schanzenbach and Almond, 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 sugar-rich diets specifically (Reid et al., 2016). Because later-life diet differs in sugar composition but not total daily calorie intake, our results also suggest that persistent dietary composition, and not only calorie imbalance, may shape long-run 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, Gallagher and Ritchie, 2018; Belot and James, 2011), especially at earlier ages (Lundborg, Rooth and Alex-Petersen, 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, Grosse and Sonntag, 2017; Trogdon et al., 2008; Finkelstein et al., 2010; Popkin et al., 2006; Hoynes, Schanzenbach and Almond, 2016; Bailey et al., 2020).

I. 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*b*). 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*b*). 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, 2007*a*). Because sugar and sweets were rationed, 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*b*). 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 (NFS) in 1940 (also known as the Expenditure and Food Survey today (Foster and Lunn, 2007*a*). Created for the purpose of diet tracking to prevent malnutrition 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 aggregated data from historical NFS records to describe dietary patterns during 1950 and 1960, the study period of our interest.

NFS 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 1) (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, 2015*a*; 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, 2015*a*).[4]

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% of calories came from protein compared to 15% recommended, 40% from fat compared to between 25 and 35% recommended, and 47% from carbohydrates compared to 45 to 55% recommended. This diet was healthy enough so that children were not deficient in iron, vitamin A or C (Foster and Lunn, 2007*a*), and the amount of animal protein available to children and pregnant and nursing women was deemed adequate (Hollingsworth, 1983).

Pregnant women, nursing mothers, and children received special green ration books with extra coupons and special allowances (Zweiniger-Bargielowska, 2000*b*; Sultan, 2010). Starting in 1940, they were entitled to additional food rations for priority foods such as vitamin supplements, eggs, and milk, cod-liver oil, fruits and vegetables. This provided an additional 540 calories per day and the bulk of daily calcium and vitamin needs. These priority allowances were later credited as having "done more than any other single factor in promoting the health of expectant mothers and young children during the war" (Jameson et al., 1946).

[4]Historical accounts of rationing enforcement do not provide evidence of a large sugar-specific black market. The accounts suggest that Britain's wartime and postwar black market was present, but not uniform across rationed goods. Illicit trade was more salient for goods and coupons that were scarce, valuable, transferable, or difficult to monitor, including meat, eggs, petrol, and clothing coupons (Zweiniger-Bargielowska, 2000*a*; Roodhouse, 2013). Sugar, however, was administered through the food-rationing system in which consumers registered with specific retailers and retailers' supplies were tied to their registered customer lists (Museum of English Rural Life, 2021). This design made routine off-ration purchases and resale more difficult than for goods traded through looser coupon markets or informal supply channels. Thus, informal access to sugar was possible but institutionally constrained. Aligned with this, NFS also shows no differential post-rationing increases in sugar consumption across social classes (Zheng et al., 2025*b*).

Figure A.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. During rationing access to these foods among pregnant women, nursing mothers, and young children was governed by priority entitlements, implying that de-rationing did not alter maternal and infant access to most of these foods (Gracner, Boone and Gertler, 2024; Zweiniger-Bargielowska, 2000*a*; Sultan, 2010).

Pregnant women, nursing mothers, and young children did not have priority access to sweets and sugar during rationing, which went off-ration in September 1953. This de-rationing led to an immediate expansion in the confectionery market. By the early 1950s supply channels were already capable of delivering additional sugar households demanded once rationing was removed (Moynagh, 1977; UK Parliament, 1950, 1951). Sales of sugar, sweets, and chocolate rose dramatically in the first four weeks after rationing ended (UK Parliament, 1953; Jackson, 1974). Among children aged 6–12, average sugar intake during rationing was within recommended amounts, below 115 g per week, but more than doubled, accompanied by a deterioration in dental health, after rationing ended (Jackson, 1974; Zweiniger-Bargielowska, 2000*b*; Jamel, Plasschaert and Sheiham, 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999).

We find similar dietary patterns in NFS data. Figure 1 shows changes in sugar, other food groups, total calories, and sugar calories between 1950q1 and 1959q4. Sugar intake increased sharply after rationing ended, rising from 10.2 oz per person per week in 1953q1 to 16.08 oz in 1954q1 and nearly 20 oz by 1954q3. By contrast, other foods and nutrients show no comparable break over the same period. This holds across the July 1954 end of general rationing as well: neither total calories nor the consumption of foods de-rationed at that date shifts discontinuously (e.g., Figures 1 and A.3; more in (Gracner, Boone and Gertler, 2024)).

Total calorie intake rose by a small amount: roughly 100 calories per day, or about 5% relative to the rationing-period average. Calories from sugar accounted for at least 80% of this increase (Department for Environment, Food and Rural Affairs, 2016). The actual share was likely even higher: sugar-consumption data exclude purchases outside the home, whereas total calorie consumption includes food purchased outside the home.

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.

II. Data

A. The English Longitudinal Survey of Aging (ELSA)

ELSA is a biennial panel survey representative of the population aged 50 and over in the United Kingdom. It spans a 16-year period between 2002-2018 (9 waves). It was designed as a sister study to the Health and Retirement Study in the US and provides detailed information on health and socio-economic outcomes. The analysis sample includes individuals born between 1950 and 1960 who are 50-64 years old between 2002 and 2019, with all available outcome and control measures. This age range is when disability and chronic diseases emerge (Kirkman et al., 2012).[5] Those born between October 1953 and December 1954, and those born outside the UK are excluded. In the final analysis sample, there are 12,589 people aged 50-64, of whom 40% were born after rationing ended.

We examine *health outcomes* derived from physician diagnoses of chronic diseases, as reported by respondents. These include binary indicators of the presence of diabetes, elevated cholesterol, hypertension, heart disease, stroke, and arthritis. We also construct an indicator of the presence of three or more chronic conditions among the following: hypertension, diabetes, heart disease, stroke, and high cholesterol. 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. Finally, we construct a summary metabolic disease index as the weighted average of standardized z-scores for diabetes, high cholesterol, heart disease, stroke, and hypertension (Anderson, 2008). The z-scores are calculated using the mean and standard deviation of the post-rationing cohort. Higher values of the index indicate worse metabolic health.

ELSA also includes *objective biomarker* measurements of hemoglobin A1c (HbA1c) and C-reactive protein (CRP) based on blood samples for a subsample of adults.[6] We define diabetes as HbA1c $>$ 7%, consistent with clinical diagnostic thresholds. CRP is used to measure chronic inflammation, defined as CRP levels greater than 3mg/L.

Economic outcomes include indicator variables for whether the respondent has (i) 16+ years of schooling, (ii) any college education, (iii) whether the respondent was employed in the last 7 days, and (iv) had a skilled job.[7] We also consider whether family wealth is above the median wealth level.[8] Finally, we construct an index of economic sufficiency following Kling, Liebman and Katz (2007) and Hoynes, Schanzenbach and Almond (2016). The index is the average across z-scores of the above indicators. The z-scores are calculated by subtracting the mean and dividing by the standard deviation for those born after the end of rationing.

[5] More details on these data can be found at www.elsa-project.ac.uk.

[6] The subsample consists of participants who consented and were able to give blood in a clinical assessment.

[7] Skilled job is defined using the National Statistics Socio-economic Classification (NS-SEC 13), including employers in large corporations, higher managerial or professional occupations, lower professional and higher technical occupations, intermediate occupations, lower managerial or higher supervisory occupations, and employers in smaller organizations.

[8] Wealth is defined as the sum of the value of primary residence, businesses, non-housing financial wealth, secondary housing or other real estate, and total value of other physical assets (e.g., other land, or money owed by others).

ELSA also includes a "Life History Module" in Wave 3, which collects retrospective 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, whether their father's job was a 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.). There is also information on whether biological parents were ever diagnosed with diabetes, which we use as a proxy for one's genetic pre-disposition for diabetes and related disease.

B. UK Biobank

The UK Biobank (UKB) is 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 UK Biobank recruitment was staggered between 2006 and 2010, participants born in the same month were assessed at different ages, and baseline ages across this birth window range from approximately 50 to 59. Rationed and never-rationed cohorts are jointly observed only over ages 53 to 56, and we restrict the estimation sample to this common age support so that comparisons across exposure groups are not confounded by age at assessment. We exclude individuals born outside the UK, non-white participants, those from multiple births or who were adopted, participants who subsequently withdrew consent, those pregnant at the time of assessment, those partially exposed to rationing in utero only (born 1953q4–1954q2), and individuals with missing covariates, genetic information, or measures of adiposity.

UKB complements ELSA 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 estimates. Second, UKB provides objectively measured visceral adiposity (obesity) outcomes, known as a key risk factor for chronic disease (Després and Lemieux, 2006). Third, UKB includes detailed birth data so that we can use birth quarter as the running variable in the RDD analyses. Finally, UKB includes genetic data, such as polygenic scores for obesity, 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 an older age range when such conditions are more prevalent, UKB enables us to analyze early metabolic markers and gene-environment interactions with greater statistical power.

One of our primary outcomes is a visceral adiposity index constructed from (i) trunk fat percentage (% TF, field 23127) and (ii) waist-hip ratio (WHR, fields 48 and 49).[9] Each component was

[9]Trained 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

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. Other outcomes included 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 disease index using the same inverse-covariance weighting approach, as well as a metabolic syndrome indicator defined as having at least two conditions.

We assessed heterogeneity in genetic risk for obesity using a standardized polygenic index for BMI derived from GWAS summary statistics.[10] We use data on BMI PGI generated by Barcellos, Carvalho and Turley (2018) (UK Biobank return code 3586, application 1142). Using this index, we divide adults into those above vs below median genetic risk for obesity.

Unlike ELSA, UKB is not nationally representative. 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 individuals from some UK regions had lower participation rates (Fry et al., 2017). UK Biobank participants are also healthier than the general population. These sampling characteristics limit external validity and could threaten internal validity if selection into UK Biobank changed discontinuously across the exposed and comparison cohorts. We therefore examine whether cohort counts, mortality rates, and observable baseline characteristics change discontinuously around the exposure threshold. We find no evidence of differential selection by exposure status: mortality rates at ages 53–56 are very low and similar across cohorts, and observable baseline characteristics are balanced around the cutoff (Figures A.14 and A.12; Table A.1).

C. Aberdeen Children Born in the 1950s

The Aberdeen Children of the 1950s cohort comprises 12,150 children born in Aberdeen, Scotland between 1950 and 1956, 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). The data include the month of birth, which positions each cohort member relative to the September 1953 end of sugar rationing and provides the running variable for the RDD analyses.

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).

[10]The BMI polygenic score for an individual is a weighted sum of SNP genotypes. The weights are derived from GWAS statistics for BMI (Locke et al., 2015) and transformed using linkage disequilibrium (LD) prediction (Vilhjálmsson et al., 2015). In our sample, the resulting BMI PGI explains approximately 7% of the variation in BMI, aligned with data estimates elsewhere (Choi, Mak and O'Reilly, 2020; Herrera and Lindgren, 2010; Yengo et al., 2018).

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 assessment 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).

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.

D. 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, 2015*a*) 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.

E. Mortality and fertility

We construct cohort mortality for the 1950–1960 birth cohorts from the Human Mortality Database, which reports annual deaths by single year of age and live births by year of birth.[11] These data let us follow each cohort from birth and compute cumulative mortality, the share of a cohort that has died by a given age. Series by single year of age are published for England and Wales only, so we restrict these calculations to that population.

III. Empirical Methods

We employ a local linear Regression Discontinuity Design (RDD) as our main empirical approach. We estimate the following specification:

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

where Y_{it} is the outcome variable of interest and $BirthDate_i$ is the running variable, measured as time between the individual's birth date and the relevant exposure cutoff. $BornPostRation_i$ is an indicator equal to one for individuals whose fetal and early-postnatal life was after sugar rationing ended and zero for individuals whose comparable early-life period occurred under rationing. The coefficient β is the shift in the intercept of the running variable as it transitions from rationed to non-rationed states. It is the treatment effect and captures the local difference in the outcomes on either side of the transition. Away from the transition cutoff, the running-variable controls for the correlation between the timing of birth and omitted determinants of the outcomes.

The exact specifications differ by data set. In ELSA, the running variable is birth year. Controls, X_{it}, include gender, two-year age indicators, parental diabetes indicators, indicators for imputed parental diabetes information, and survey wave indicators. We include age indicators because adult

[11] Human Mortality Database, England & Wales Total Population, https://www.mortality.org.

health outcomes change mechanically with age, and the panel structure of ELSA allows us to compare respondents at the same ages across birth cohorts. We restrict the sample to a common age support of ages 50–64, on both sides of the cutoff. Parental diabetes indicators represent family health history and genetic predisposition to chronic disease.

UK Biobank's larger sample size allows a finer running variable, defined at the birth-quarter level. Because outcomes are observed once at baseline rather than repeatedly over time, the controls differ from the ELSA panel specifications: the main models adjust for sex, age-at-baseline indicators, calendar month of birth indicators, and family history of diabetes and cardiovascular conditions. As mentioned above, participants were assessed on different dates, so baseline ages across the birth window run from roughly 50 to 59. Rationed and never-rationed cohorts are jointly observed only over ages 53 to 56, and we restrict the estimation sample to this common age support.

The analysis of the Aberdeen data uses a monthly running variable. Because all children were assessed at fixed ages of 7, 9, and 11, the age support is common across cohorts without further restriction. Controls follow the UK Biobank specification, omitting the age-at-baseline indicators, which are not identified when age is fixed by design.

A. Heterogeneity

We examine heterogeneity by length of exposure to rationing and by genetic risk. These analyses are substantively important and also provide indirect evidence on identification.

Length of Exposure to Rationing: We next test whether effects increase with the length of protected exposure. If the end of rationing is what drives them, children shielded for longer should show larger effects; the reverse pattern would point to unrelated cohort differences or later policies.[12] 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 postnatally. We restrict these analyses to continuous adiposity measures and related indicators. Splitting by exposure length leaves narrow birth-quarter cells, and chronic disease is too rare at these ages to be estimated precisely, particularly in the longer-exposure specification, which is further restricted to the common age support of ages 55 to 56.

Genetic Risk of Obesity: For UK Biobank, we are able to stratify the analysis by genetic risk for obesity. If sugar rationing drives our results, individuals with high genetic risk should if anything 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 differential effects by genetic risk if our results reflect mechanisms specific to early-life sugar

[12] A flat gradient is inconclusive: it is consistent both with a critical window confined to gestation, where added postnatal months change nothing, and with limited power across narrow birth-cohort groups.

rationing exposure.

B. Analysis Samples

The exact samples vary by data set and type of analysis; Table A.3 reports the birth dates used in each. In every data set the rationed group runs from the start of the sample window through the last birth date at which a child was fully exposed to rationing. Because exposure depends on the timing of gestation and infancy rather than on birth date alone, some of the cohorts born around the cutoff were only partially exposed to rationing. Those cohorts whose cumulative fetal and early-postnatal exposure was not complete and therefore ambiguous are excluded. The never-rationed comparison group begins after the excluded window.[13]

ELSA uses a yearly running variable and two exposure windows. Under the short-exposure specification (*in utero* through 9 months postnatal), the rationed cohorts are those born between January 1950 and September 1953, births between October 1953 and December 1954 are excluded, and the never-rationed group runs from January 1955 through December 1959. Under the long-exposure specification (*in utero* plus 10 to 21 months postnatal), the cutoff moves back to June 1952, the excluded window widens to July 1952 through December 1954, and the never-rationed group is unchanged.

The UK Biobank uses a quarterly running variable, also with two exposure windows. Under the short-exposure specification (*in utero* through 3 months postnatal), the rationed cohorts are those born between January 1952 and September 1953, births between October 1953 and June 1954 are excluded, and the never-rationed group runs from July 1954 through March 1956. Under the long-exposure specification (*in utero* plus 4 to 9 months postnatal), the cutoff moves back to March 1953 and the excluded window to April 1953 through June 1954, with the same never-rationed group.

The Aberdeen sample comprises children born between October 1950 and September 1955 who were resident in Aberdeen and enrolled in primary school in December 1962 study. The running variable is monthly, and the data support a single exposure window, *in utero* only. Rationed cohorts are those born between October 1950 and September 1953, births between October 1953 and June 1954 are excluded, and the never-rationed group runs from July 1954 through September 1955.

C. Identification

The RDD treatment effect is the intercept shift (β) in the running variable due to the transition from rationed to non-rationed states in equation (1). The estimand is a local contrast between cohorts on either side of the cutoff, not a difference in means between all rationed and never-rationed cohorts.

The identification of (β) is based on the continuity assumption that among cohorts born close to the end of sugar rationing, potential outcomes evolve smoothly in the absence of a change in sugar availability (Imbens and Lemieux, 2008; Lee and Lemieux, 2010; Cattaneo, Idrobo and Titiunik,

[13] Because the running variables are aggregated to years, quarters, or months, boundaries fall on a year, quarter, or month edge rather than on the exact date implied by the exposure window.

2020). The assumption is not that observed outcomes are uncorrelated with all omitted factors nor that all rationed and never-rationed cohorts are otherwise identical. Rather, it is violated when factors correlated with outcomes other than sugar change discontinuously across the exposure cutoff. Away from the cutoff point, the running-variable controls for the correlation between the running variable (birth date) and omitted determinants of the outcomes.

Because the partially treated transition cohorts are excluded, identification requires not only the smooth evolution of underlying determinants but also that the side-specific local linear fits extrapolate smoothly across this window. Visual inspection of the cohort plots supports this approximation (e.g., Figures 2, A.8, A.9, A.10). Away from the transition window, the timing of birth is plausibly correlated with many omitted determinants of later-life outcomes; the running-variable trend absorbs these smooth cohort differences.

Identification rests on continuity (smoothness) holding for the cohorts born immediately before and after the transition window, the ones used to identify the shift in the intercept in the running variable. The running variable is grouped according to the measurement frequency and sample size of each dataset, reflecting a tradeoff between finer birth-date information, which narrows the period over which continuity must hold, and preserving enough observations around the transition to estimate local effects with adequate precision. Because ELSA has the smallest sample and low disease prevalence at finer intervals, our main ELSA specifications use birth year;[14] the larger UK Biobank sample permits birth quarters, and the Aberdeen data allow birth months. Table A.3 summarizes the definitions of the running variable, grouped birth-date intervals, excluded windows, and exposure classifications used in each data set. The continuity assumption applies to the grouped intervals immediately on either side of the excluded window. In section V, we assess the continuity assumption by checking balance in baseline characteristics and subsequent policy and economic shocks later in life for the identifying cohorts as defined in Table A.3.

D. Estimation and Inference

We estimate the main specifications as local linear RDD models using the `rdrobust` package in Stata (Calonico, Cattaneo and Titiunik, 2014). The estimating windows are pre-specified according to the exposure definition and the birth-date resolution available in each dataset (Table A.3): the main specifications use birth year in ELSA, birth quarter in UK Biobank, and birth month in Aberdeen. Within each window, triangular kernel weights place greater weight on observations closer to the cutoff, and cohort trends are modeled with a first-order polynomial in $BirthDate_i$ estimated separately on each side of the cutoff.

Data-driven MSE-optimal bandwidths are narrow in this setting, yielding comparable point estimates but limited precision; we therefore report results for the pre-specified windows.[15] Sup-

[14] We obtain similar results using birth semesters (see Supplemental Appendix).

[15] We report conventional robust standard errors rather than bias-corrected intervals. Bias correction guards against polynomial misspecification (Calonico, Cattaneo and Titiunik, 2014), but it relies on a higher-order derivative estimated from the same data; with coarse running variables and few mass points near the threshold, that estimate is imprecise and the resulting intervals are more sensitive to the pilot bandwidth.

plemental Appendix Tables A.8–A.11 report estimates across alternative bandwidths and kernel weights. Point estimates are similar in sign and magnitude across these specifications, though precision declines under the narrowest windows, where the effective sample is roughly one-third smaller.

Inference is matched to the structure of each dataset. In ELSA, where respondents are observed repeatedly across survey waves, we cluster standard errors at the individual level to account for within-person correlation across waves. In Aberdeen, where exposure is assigned by birth month, we cluster standard errors at the birth-month level. In UK Biobank, where exposure is assigned by birth quarter and the number of quarter-level clusters within the estimation window is limited, we use the heteroskedasticity-robust nearest-neighbor variance estimator implemented in `rdrobust`; estimates are similar when standard errors are clustered at the birth-quarter level.[16] We assess robustness to alternative bandwidths, weighting choices, and birth-date groupings in the Supplemental Appendix. For each outcome family, we report Romano–Wolf adjusted p-values based on the step-down procedure in Romano and Wolf (2005) to account for multiple hypothesis testing.

IV. Results

A. Adult health outcomes

In *ELSA*, early-life exposure to the post-rationing sugar environment increased adult prevalence of diagnosed diabetes by 4.4 pp(54%) ; elevated cholesterol by 7.3 pp (30%); and hypertension by 6.8 pp (23%), and no change in heart disease or stroke (Table 2, Panel A). These results are robust to measurement and specification. For example, elevated diabetes prevalence persists whether diabetes is measured through self-report or objectively using HbA1c thresholds of 6.5% or 7%, and across alternative running variables including birth year and six-month birth periods. The consistency among subjective and objective measures suggests that reporting artifacts or general diagnostic trends are unlikely to explain the observed effects.

We also find evidence of higher inflammatory burden in ELSA. Objectively measured C-reactive protein above 3 mg/L increased by 11.2 pp, and arthritis increased by 12.2 pp. These findings are consistent with evidence linking elevated sugar consumption to low-grade systemic 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 the risk of chronic diseases such as 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 in ELSA. Individuals born after rationing had a 6.8 pp higher prevalence of comorbidities, defined as having three or more diet-related chronic diseases, and a 0.21 SD higher metabolic disease index. Figures A.8 and A.9 visually support these results, showing discontinuous jumps at the cutoff across the main health outcomes.

[16] This choice is consistent with concerns that clustering at the assignment-variable level can perform poorly when the number of support points is limited (Kolesár and Rothe, 2018).

Table 3 reports the results for *UK Biobank*. It confirms the ELSA findings and adds objectively measured abdominal adiposity outcomes. Never-rationed adults experienced a 1.1 pp (a 30%) increase in diabetes; a 2.5 pp increase in hypertension; a 1.9 pp increase in elevated cholesterol; a 0.11 SD increase in the metabolic disease index; and a 2.8 pp increase in the prevalence of metabolic syndrome (Panel A). The prevalence of chronic disease among never-rationed adults in UKB is lower than in ELSA because UKB participants are ages 53–56 compared to ages 50–64 in ELSA.

Together, the results from the analysis of ELSA and UK Biobank data indicate that exposure to the post-rationing sugar environment during fetal life and infancy increased metabolic risk later in life. The consistency of these effects across two independent adult datasets, across running variables measured in years and quarters, and across both diagnosed conditions and objectively measured biomarkers strengthens the interpretation that early-life sugar exposure had lasting consequences for adult health.

1. Length of Exposure to Rationing

These analyses test whether longer exposure to rationing was associated with larger protective effects. In *ELSA*, Table 2, Panel B, presents suggestive evidence that health effects do, in fact, increase with the duration of exposure. Treatment effects increased by about 50% for all outcomes except for hypertension as the exposure to rationing increased by 12 months from *in utero* plus 0 to 9 months postnatally to *in utero* plus 10 to 21 months. However, the differences are only statistically significant ($p < 0.10$) for heart disease or stroke, arthritis, and multiple comorbidities.

In *UK Biobank*, we explore dose-response effects using continuous adiposity measures rather than binary disease outcomes. [17] Table 4 shows that a six-month increase in exposure to rationing had larger protective effects concentrated at the upper tail of the adiposity distribution. The effect on mean adiposity does change significantly between those rationed *in utero* plus 0 to 3 months postnatally and those rationed *in utero* plus 4 to 9 months postnatally. However, abdominal obesity, defined as exceeding the 90th percentile of the never-rationed distribution, shows a clearer dose-response pattern: an effect 3 pp lower prevalence of abdominal obesity increases to a 6 pp pp effect as exposure increases 6 months.

2. Genetic Risk for Obesity

Table 3 separates respondents by above- and below-median genetic risk for obesity, measured using a polygenic index for BMI. The average health effects are driven almost entirely by adults at higher genetic risk. For low-risk adults, differences between never-rationed and rationed adults are generally smaller than those for high-risk adults. Formal tests comparing high- and low-risk groups show a statistically significant difference ($p<0.10$) for abdominal adiposity. Differences for diabetes, hypertension, and the metabolic health syndrome or index follow the same pattern but are not statistically significant at conventional levels in the current specification. These results suggest that

[17] This choice reflects the lower chronic disease prevalence at ages 53–56 in UKB compared with ELSA's older sample, which limits statistical power when restricting to narrow birth-quarter groups.

genetic predisposition shaped vulnerability to early-life sugar exposure, with the adverse metabolic effects concentrated among individuals at higher inherited risk of obesity.

B. Human Capital and Economic Welfare

1. Cognitive development in childhood

Table 6 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 scored 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. By age 9, the gap widens to −0.214 standard deviations, and by age 11 it reaches −0.256 standard deviations.

This monotonic pattern suggests that the cognitive cost of early-life sugar exposure was not fully expressed at school entry but emerged and intensified over the course of primary schooling, as test content shifted from picture-based reasoning at age 7 to verbal reasoning and attainment at later ages. Figure 3 displays the underlying reduced-form relationship between birth month, centered at the September 1953 end of rationing, and standardized cognitive scores at each age. Together, the table and figure indicate that children protected by early-life sugar rationing scored higher, with effects that were modest and imprecisely estimated at school entry but substantial and statistically robust by later childhood.

2. Education, labor supply, and wealth

Figure A.10 shows discontinuous changes at the cutoff for outcomes including completing 16 or more years of education, working in a skilled occupation, and accumulating above-median wealth by the mid-60s. Table 5, Panel A, shows that the end of rationing reduced the probability of completing 16 or more years of education by 10 pp (33%) and the probability of attending any college by 9.2 pp (18%), relative to adults exposed to rationing *in utero* and up to nine months postnatally.

Although both cohorts were equally likely to be employed, the end of rationing reduced the probability of working in a high-skilled occupation by 14.1 pp (26%). Consistent with these human capital and occupational effects, adults born after rationing were about 14 pp less likely to accumulate wealth above the median. Finally, the economic sufficiency index was 0.26 SD lower among those never rationed. As with health, estimates are larger for cohorts protected longer: the gap widens to 0.35 SD under the longer exposure window, about a third more. That difference between panels is not statistically significant, however, so we treat the duration evidence as suggestive.

The economic results indicate that the end of rationing affected both early cognitive development and socioeconomic achievement later in life. The childhood evidence from Aberdeen suggests that differences in cognitive skills emerged early, while the adult evidence from ELSA shows persistent consequences for education, occupation, wealth, and overall economic sufficiency.

C. Benchmarking

Figure 4 benchmarks our ELSA and UK Biobank estimates against prior evidence from Hoynes, Schanzenbach and Almond (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 postnatal, the effect sizes are comparable to those reported by Hoynes, Schanzenbach and Almond (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, Schanzenbach and Almond (2016)), and broader socioeconomic contexts, which should be considered when interpreting these comparisons.

D. 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 include total energy intake per day and grams of fats, proteins, and carbohydrates, the latter divided into fiber, intrinsic sugars, and free sugars. Binary outcomes indicate whether individuals ate any sweets (sugar, preserves, sweet spreads, chocolate, and sugar confectionery), fruits, or vegetables on a given day, and whether free sugar intake exceeded 5 or 10 percent of total energy intake.

We estimate the same discontinuity specification used for our other outcomes, over the pre-specified window of UK-born respondents born between 1950 and 1960, excluding those born between 1953q4 and 1954q2. Each outcome is regressed on an indicator for birth after the end of rationing, a linear trend in year of birth estimated separately on each side of the excluded window, weighting all observations equally. This is equivalent to a local linear estimator with a uniform kernel and a bandwidth equal to the full estimating window, implemented by ordinary least squares. Standard errors are robust and clustered at the individual level, since respondents are observed on multiple recall days.[18]

Figure A.5 describes diet outcomes across cohorts of today's older adults. Panels (a)–(c) show a sharp increase at the cutoff in the share exceeding the recommended free sugar limit of 5% of TEI, in daily free sugar intake, and in the probability of consuming any sweets. Intrinsic sugar intake and the probability of consuming any fruit decrease discontinuously at the cutoff. In contrast, neither total calorie intake nor other macronutrients changes discontinuously.

The regression estimates in Table 7 confirm this pattern. Panel A shows that the end of rationing raised the share of adults exceeding the 5% TEI recommendation by 10.2 pp (13%) and increased free sugar intake by 12 grams per day. Panel B shows no difference in the probability of consuming any vegetables, but never-rationed adults were more likely to consume sweets (8.0 pp,

[18] We do not use data-driven bandwidth selection here: with 2,853 observations across eleven birth years, the bandwidth selector and its bias-correction term rest on very few effective observations.

13%) and less likely to consume fruit (14.2 pp, 22%). They also consumed 17% less intrinsic sugar and 9% less fiber. Panel C shows no effect on total calories, fats, proteins, or carbohydrates.

V. Threats to Identification and Robustness

We perform several analyses designed to assess whether factors other than the end of sugar rationing could explain the estimated effects. The identifying assumption is that, among nearby cohorts with clearly classified exposure status, potential outcomes would have evolved smoothly across the rationing transition in the absence of the change in sugar availability. This assumption would be violated if other determinants of later-life outcomes changed discontinuously across the same exposure threshold, either early in life or through subsequent policies and events. The relevant birth-date unit varies by dataset: ELSA uses birth year, UK Biobank uses birth quarter, and Aberdeen uses birth month. In each case, the identifying comparison comes from cohorts nearest the transition whose early-life exposure can be cleanly classified.

Early Life Circumstances: 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 A.1, Panel A, shows that the differences in means in several potential confounders including childhood SES, home environment, parental education, occupation, health, and parenting style, are small and not statistically significantly different from zero. We observe similarly balanced childhood characteristics in UK Biobank (Panel B) and Aberdeen data (Panel C). 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.

Figure A.12 plots binned means of the same characteristics by birth cohort and shows that they evolve smoothly through the threshold, so the balance in Table A.1 reflects continuity at the cutoff rather than offsetting trends on either side. Additionally, Figure A.4 shows no break in food prices at the threshold, indicating that the end of rationing changed the quantity of sugar available rather than its cost. Cohorts born just after September 1953 therefore faced a sharply different sugar environment under a similar food budget constraint. Together, these patterns indicate that our estimates are not driven by unobserved differences in family background, early-life environment, or food affordability.

Public Policy and Economic Shocks Later in Life: It is possible that health, education and other social policies or large macro-economic shocks differentially affected the two cohorts. We addressed this possibility in several ways. First, we systematically reviewed UK policies and major economic events from the end of WWII through 2000. We then investigated whether these policies and shocks could have in principle differentially affected rationed versus never-rationed cohorts nearest the transition used to identify the intercept shift in the running variable around the September 1953 cutoff and found that none of them did so (Table A.15).

Second, we test for discontinuities in the outcomes of interest at cutoffs at different times other than when sugar rationing ended (see Table A.7). 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 the end of rationing versus subsequent policies or events drove the observed effects, we should find no discontinuities at these later dates. Estimated treatment effects in health and economic outcomes are small and not statistically significant at these alternative cutoffs for all outcomes. These null results support the interpretation that the end of rationing, rather than other policies or events before or after 1953q3, drives the observed differences.

Third, while rationing is expected to have a differential effect by genetic risk for obesity, differential effects of public policy and economic shocks should affect health outcomes regardless of genetic risk. In UK Biobank, Table 3 Panel B presents results for adults at above-median genetic risk for obesity, while Panel C presents results for adults at below-median genetic risk. The health effects are driven almost entirely by adults at higher genetic risk. For low-risk adults, differences between never-rationed and rationed groups are not statistically different from zero.

We also conduct a genetic risk falsification test using a placebo for genetic risk; migraine—a condition unrelated to diet. Table A.12 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 for migraine groups.

Fourth, 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 differential policies and events might have affected them. The analysis shows that there are no statistically significant differences in such diseases between cohorts (see Table A.6).

Improved Medical Technology: 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 nearest the transition differentially. This is a very tight window of at most a year and it is unlikely that improved technology differentially affected the two cohorts over a 50+ year period. 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 Table 2. Similarly, exposure to rationing should not have affected the placebo outcomes, but improved diagnostics might have. However, we find no effects on the placebo outcomes (see Table A.6).

Aging: 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. The panel structure of ELSA lets us address this concern: because we observe the same individuals at multiple ages, we have rationed

and never-rationed respondents at the same age, and can include age indicators in the regressions. We further restrict the ELSA sample to adults aged 50–64, where the rationed and never-rationed cohorts overlap in age, and the UK Biobank sample to ages 53–56 at baseline.

Mortality Selection: If rationed cohorts were more likely to die before entering ELSA or the UK Biobank, surviving rationed respondents would be more selected than their never-rationed counterparts. Figure A.14, panels (a) and (b), shows that cumulative mortality is low and similar across exposure groups over the relevant age ranges: about 7.5 percent of our analysis cohorts have died by age 50, and in the ELSA ranges 12.5 percent of rationed versus 11.3 percent of never rationed cohorts by age 60. Panel (c) shows this level difference is a secular trend, not a break at the threshold. Cumulative mortality declines monotonically across single birth cohorts at every horizon—infant mortality from 29.7 to 21.9 per 1,000, and mortality by age 60 from 12.8 to 10.9 percent—and evolves smoothly through the end of rationing. Local linear fits on either side of the excluded window imply discontinuities at the threshold of -0.02 percentage points by age one and -0.04 by age 60, against a trend of -0.14 to -0.21 points per cohort-year. Smooth cohort differences of this kind are absorbed by the running-variable trend; a discontinuous change in survival would not be, and we find none.

Fertility Selection: Selective fertility would bias the estimates only if it changed the composition of births at the threshold, and it would operate through exactly the family attributes shown to be balanced above. Aggregate fertility also evolved smoothly through the transition (Figure A.13). Cohort sizes vary by only a few percent during rationing and immediately after; births rise only from 1956, reaching 14.9 percent above the rationing-period average by 1960 with the onset of the baby boom. Because our outcomes are not counts, cohort size does not mechanically affect the estimates; controlling for annual live births nonetheless leaves our conclusions unchanged (Tables A.13 and A.14). The UK Biobank window ends in the first quarter of 1956, before cohort sizes move, so an analogous control there is uninformative.

Robustness to Specification: Finally, we provide evidence that our results are not sensitive to the choice of bandwidth, or running variable grouping (see Tables A.9, A.8, A.11, A.10, and A.4 and A.5).

VI. Conclusion

This paper studies the long-term effects of exposure to a sugar-rich diet early in life on health, cognition, and economic well-being. We exploit the end of sugar and sweets rationing in the United Kingdom in September 1953, which generated a sharp increase in sugar and sweets consumption, while other foods and nutrients changed little or only gradually over the same period. Using a local linear regression discontinuity design across ELSA, UK Biobank, Aberdeen childhood records, and NDNS dietary data, we compare nearby cohorts born around the end of rationing, excluding cohorts with ambiguous early-life exposure.

We find that early-life exposure to the post-rationing sugar environment worsened adult metabolic

health more than five decades later. In ELSA, cohorts born after rationing ended had higher prevalence of diabetes, elevated cholesterol, hypertension, chronic inflammation, arthritis, multiple diet-related chronic diseases, and worse overall metabolic health. UK Biobank corroborates these findings using a narrower birth-cohort window and objectively measured metabolic outcomes, including abdominal adiposity and metabolic syndrome. Effects are consistently larger among individuals at higher genetic risk for obesity, and the difference is statistically detectable for abdominal adiposity, suggesting that early-life sugar exposure may compound existing biological susceptibility. Remaining interaction tests are imprecise, so we view this as a direction for further work on gene–environment interplay in early-life nutrition.

The effects are not limited to adult health. In Aberdeen childhood records, cognitive gaps emerge during primary school: children born after rationing ended do not differ sharply at age 7, but score substantially lower by ages 9 and 11. In ELSA, cohorts born after rationing also complete less schooling, are less likely to work in skilled occupations, accumulate less wealth, and have lower overall economic sufficiency. These patterns suggest that early-life sugar exposure may affect socioeconomic outcomes both through early cognitive development and through later-life health.

The NDNS results point to persistence in diet composition as one possible pathway. Adults born after rationing consume more free sugar even more than five decades after rationing ended, while total daily calories and other macronutrients do not change discontinuously at the cutoff. This pattern suggests that early exposure to a sugar-rich environment may shift long-run dietary habits, consistent with evidence that food preferences and habits form early in life.

These findings contribute to the literature on early-life conditions and adult outcomes by examining a change in the composition of early-life nutrition rather than its overall adequacy. Much of the existing evidence on early nutrition studies famine, broad nutritional supplementation, or food assistance programs, where the treatment is a change in the level of nutrition. Our setting identifies a large and persistent change in sugar availability in an otherwise nutritionally adequate environment, allowing us to study whether exposure to a specific diet composition, rather than caloric deprivation or supplementation, has long-term effects. The results indicate that early-life exposure to sugar-rich environments can have lasting consequences for metabolic health, cognitive development, and socioeconomic attainment.

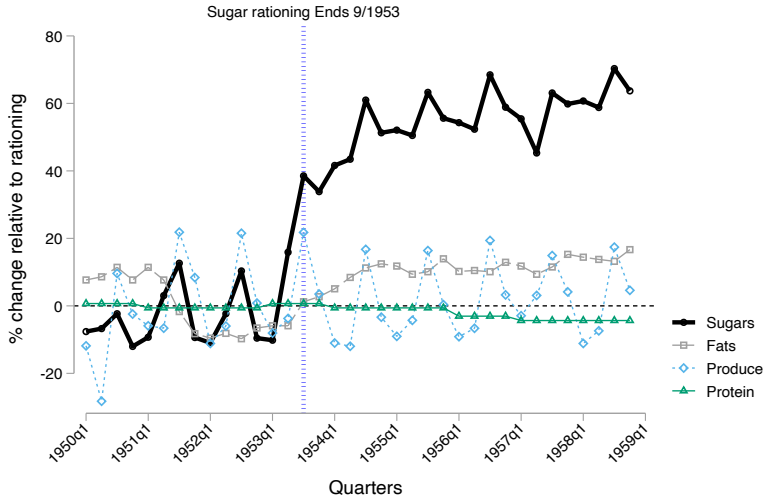
These findings have implications for policies aimed at reducing early-life free sugar exposure. They provide evidence to support WHO and USDA guidance limiting free sugar intake during pregnancy and early childhood (Foster and Lunn, 2007*b*; Moynihan, 2016; Moynihan and Kelly, 2014). Although the historical rationing setting differs, the results suggest that policies reducing sugar exposure early in life, such as limits on sugar content and marketing foods consumed by infants and toddlers, may have long-run health and economic benefits.

Several questions remain open. First, while the evidence is consistent with both persistent dietary habits and biological susceptibility, we cannot fully distinguish how these channels interact. Future work should examine whether genetically susceptible individuals are more affected because early sugar exposure has stronger developmental effects, because later-life sugar consumption is

more harmful for them, or because both mechanisms reinforce each other. Second, we cannot trace the complete timing of divergence over the life course. Aberdeen allows us to observe cognitive differences by childhood, but most health and economic outcomes are measured only in adulthood. Understanding when health and socioeconomic gaps first emerge, and how they evolve from childhood through later life, is an important direction for future research.

Figures

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



(b) Changes in total and sugar calorie consumption

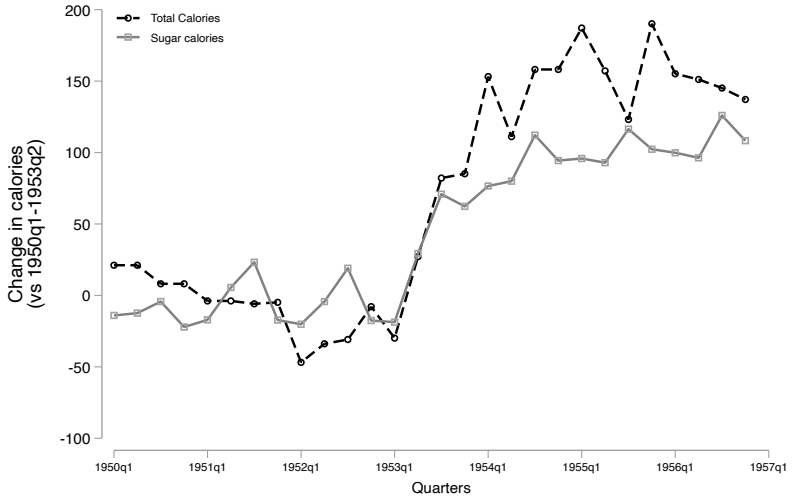
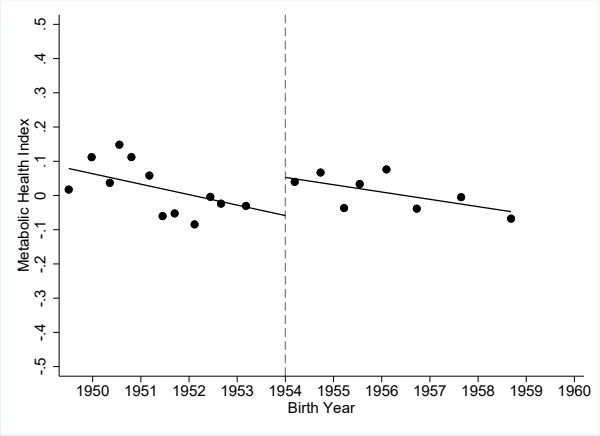
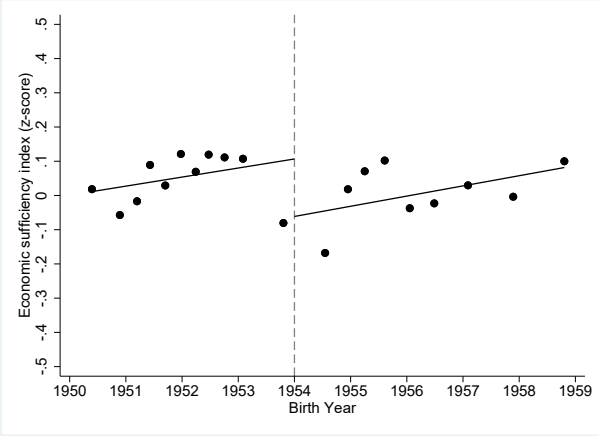


Figure 1: Changes in Sugar, Total Calories, and Other Foods during 1950–1960

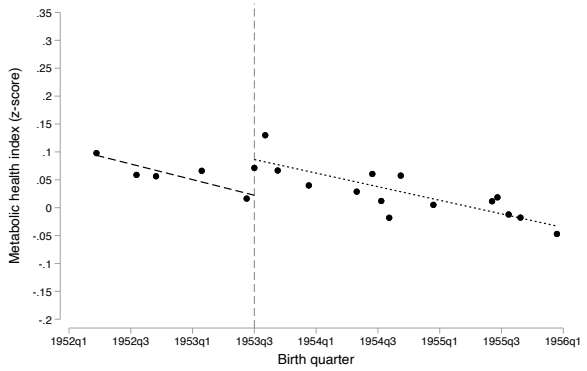
(a) ELSA: Metabolic disease index (z-score)



(b) ELSA: Economic sufficiency index (z-score)



(c) UK Biobank: Metabolic disease index (z-score)



(d) UK Biobank: Abdominal obesity (1, 0)

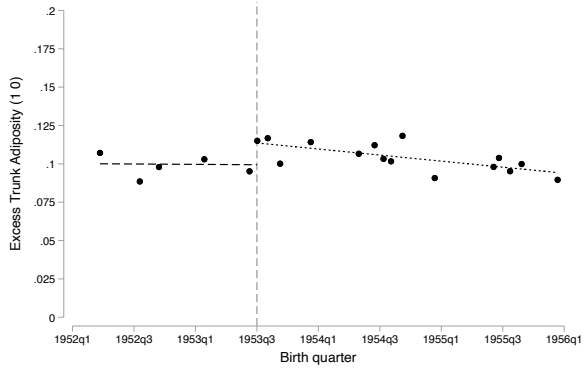


Figure 2: Metabolic Disease and Economic Sufficiency Indices across Birth Cohorts

Notes: Figures show cohort differences in health and economic well-being. (a) The ELSA metabolic disease index includes adverse health outcomes: diabetes, cholesterol, heart disease, stroke, and hypertension. Obesity-related measures are available only for a smaller sample and are not included in the ELSA index. (b) The ELSA economic sufficiency index includes positive economic outcomes: 16 or more years of education, medium- or high-skilled occupation, employment, above-median wealth, and log-transformed wealth. Indices follow Anderson (2008) and Hoynes, Schanzenbach and Almond (2016) in calculating weighted averages across standardized z-score measures of each component. (c) The UK Biobank metabolic disease index includes adverse health outcomes: diabetes, hypertension, cholesterol proxied by cholesterol medication use, and abdominal adiposity measures based on trunk fat percentage and waist-to-hip ratio. 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 or above the 90th percentile of the UK Biobank distribution among never-rationed adults. The vertical line marks the end of sugar and sweets rationing in September 1953.

(a) Age 7 **(b)** Age 9 **(c)** Age 11

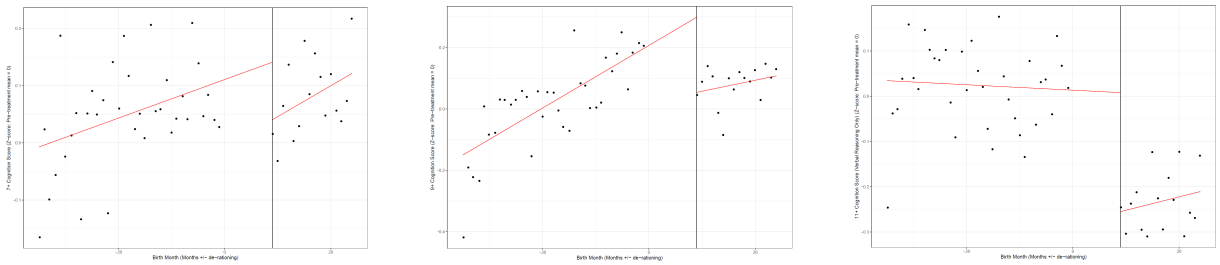


Figure 3: Childhood Cognitive Test Scores at Ages 7, 9, and 11 across Birth Cohorts

Notes: 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 in December 1962. Children born from October 1953 to June 1954 are excluded. Markers show covariate-adjusted binned means by birth month; lines show local linear fits estimated separately on each side of the exposure threshold. Outcomes are standardized, so coefficients are interpreted as standard deviation changes.

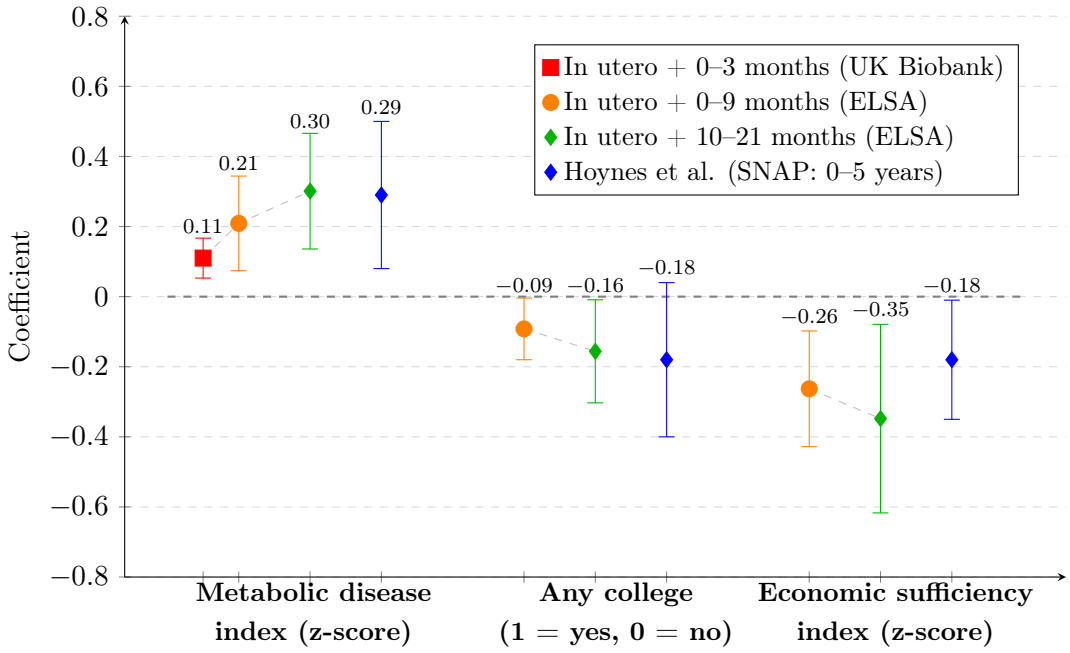


Figure 4: Benchmarking Against Prior Evidence in Hoynes, Schanzenbach and Almond (2016)

Notes: Estimates are benchmarked for scale only; populations, interventions, and ages at outcome measurement differ across studies. Metabolic disease and economic sufficiency indices follow Anderson (2008) and Hoynes, Schanzenbach and Almond (2016) in calculating weighted averages across standardized z-score measures of each component. For our estimates, z-scores are calculated using the mean and standard deviation among those born after the end of rationing. The metabolic disease index includes diabetes, high cholesterol, heart disease, stroke, and hypertension in ELSA, and also includes abdominal adiposity in UK Biobank. The economic sufficiency index includes attending 16 or more years of education, medium- or high-skilled occupation, employment, above-median wealth, and log wealth. Orange and green estimates are from ELSA; the red metabolic disease estimate is from UK Biobank. The Hoynes, Schanzenbach and Almond (2016) estimates show the long-run effects of Food Stamp access before age five.

Tables

Table 1: Diet under Rationing (1950–1953) and Modern Healthy Diet Recommendations

	UK diet in 1950–1953	Today's diet recommendation
Total calorie intake	2,500 kcal	1,800–3,000 kcal
Fats	35%	20–35%
Protein	13%	10–30%
Carbohydrates	52%	45–55%
Free sugars	6.5% (40 g)	<10% (strong) <5% (conditional)

Notes: Fat, protein, and carbohydrate ranges reflect Acceptable Macronutrient Distribution Ranges. WHO recommends limiting free sugars to below 10% of total energy intake, with a conditional recommendation for health benefits below 5%. *Sources:* Department of Environment, Food and Rural Affairs, National Food Survey (2011); World Health Organization (2023*b*); World Health Organization (2023*a*); World Health Organization (2015); U.S. Department of Agriculture and U.S. Department of Health and Human Services (2020); U.S. Department of Agriculture and U.S. Department of Health and Human Services (2026).

Table 2: RDD Estimates of the Effect of Lifting Rationing on Health Outcomes (ELSA)

		Metabolic disease			Cardiovascular		Inflammation			
		Diabetes	Diabetes HbA1c≥7%	Cholesterol	Hypertension	Heart disease or stroke	C-reactive protein ≥3 mg/l	Arthritis	Chronic diseases 3+	Metabolic disease index
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A. In utero + [0, 9 months]:										
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	0.064	0.150	0.064	0.150	0.720	0.021	0.003	0.012	0.021
N		12589	2435	6351	12588	12589	2481	12589	12589	12589
Panel B. In utero + [10, 21 months]:										
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	0.051	0.045	0.013	0.447	0.020	0.389	0.000	0.001	0.014
	RW p-val	0.071	0.175	0.017	0.462	0.026	0.332	0.001	0.001	0.004
N		10480	2027	5082	10479	10480	2091	10480	10480	10480
Never-rationed mean		0.082	0.048	0.240	0.297	0.077	0.245	0.253	0.088	-0.000
Panel B > Panel A (p-value)		0.284	0.322	0.132	0.588	0.043	0.698	0.092	0.093	0.178

Notes: Sample includes individuals born between 1950 and 1960, excluding those born between 1953q4 and 1954q4, who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Each model adjusts for sex, age, parental health history, and survey year indicators. Semester of birth is included for all outcomes except cholesterol and C-reactive protein, 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+ indicates three or more of hypertension, diabetes, heart disease, stroke, and cholesterol. The metabolic disease index equals the average of standardized z-scores for diabetes, high cholesterol, heart disease, stroke, and hypertension, calculated using the mean and standard deviation of the post-rationing cohort, so higher values indicate worse health. Mean is reported for never-rationed adults so remains unchanged across panels. Standard errors clustered at the individual level are reported in parentheses. Romano–Wolf adjusted p-values account for multiple hypothesis testing. The final row reports one-sided tests of whether Panel B estimates exceed Panel A estimates. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 3: RDD Estimates of the Effect of Lifting Rationing on Health Outcomes (UK Biobank)

		Abdominal adiposity (z-score) (1)	Abdominal obesity ≥90th percentile (2)	Diabetes (3)	Hypertension (4)	Cholesterol (5)	Metabolic disease index (z-score) (6)	Metabolic syndrome (7)
Panel A. Overall:								
Born after rationing	β	0.081***	0.028***	0.011**	0.025**	0.019**	0.105***	0.028***
	SE	(0.027)	(0.008)	(0.006)	(0.012)	(0.010)	(0.028)	(0.009)
N		27519	27519	27494	27023	27553	27553	26934
Never-rationed mean		-0.000	0.100	0.037	0.206	0.119	-0.000	0.106
Panel B. High genetic obesity risk:								
Born after rationing	β	0.115***	0.024**	0.016*	0.035**	0.025*	0.119***	0.029**
	SE	(0.038)	(0.012)	(0.009)	(0.017)	(0.014)	(0.040)	(0.014)
N		13755	13755	13732	13453	13775	13775	13394
Never-rationed mean		-0.000	0.100	0.049	0.238	0.138	-0.000	0.125
Panel C. Low genetic obesity risk:								
Born after rationing	β	0.040	0.014	0.006	0.013	0.013	0.080**	0.008
	SE	(0.039)	(0.012)	(0.006)	(0.016)	(0.013)	(0.040)	(0.012)
N		13764	13764	13762	13570	13778	13778	13540
Never-rationed mean		-0.000	0.100	0.026	0.174	0.100	0.000	0.086
Panel B > Panel C (p-value)		0.086	0.270	0.174	0.167	0.260	0.248	0.128

Notes: Robust standard errors from the local linear RDD specification are reported in parentheses. Non-white adults and those born in 1953q4–1954q2 are excluded for the in-utero exposure window. The abdominal adiposity index is the average of z-standardized trunk fat percentage and waist-to-hip ratio. Abdominal obesity equals 1 for individuals at or above the 90th percentile of never-rationed adults. The metabolic disease index is a weighted average of z-standardized abdominal adiposity, diabetes, hypertension, and cholesterol. Metabolic syndrome indicates at least two of four conditions: abdominal obesity, diabetes, hypertension, and cholesterol. Genetic obesity risk is defined by the BMI polygenic index, split at the sample median. Outcomes are standardized using the never-rationed group mean and standard deviation overall for Panel A and within genetic-risk subgroup for Panels B and C. The final row reports p-values for tests comparing high- and low-genetic-risk estimates. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 4: RDD Estimates of the Effect of Lifting Rationing on Adiposity by Exposure Duration (UK Biobank)

		Abdominal adiposity (z-score) (1)	Abdominal obesity ≥75th percentile (2)	Abdominal obesity ≥90th percentile (3)
Panel A. In utero + [0, 3 months]:				
Born after rationing	β	0.081***	0.025**	0.028***
	SE	(0.027)	(0.012)	(0.008)
N		27519	27519	27519
Panel B. In utero + [4, 9 months]:				
Born after rationing	β	0.065*	0.013	0.046***
	SE	(0.038)	(0.017)	(0.012)
N		22711	22711	22711
Y(=0) Mean		-0.003	0.251	0.099
Panel B > Panel A (p-value)		0.498	0.189	0.017

Notes: Robust standard errors from the local linear RDD specification are reported in parentheses. Non-white adults are excluded. Panel A excludes those born in 1953q4–1954q2 for the in-utero and up to 3 months postnatal exposure window. Panel B excludes those born in 1953q2–1954q2 for the in-utero and up to 9 months postnatal exposure window. Outcomes are standardized using the never-rationed group mean and standard deviation. Mean is reported for never-rationed adults so remains unchanged across panels. The final row reports p-values for tests comparing the longer exposure window with the shorter exposure window. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 5: RDD Estimates of the Effect of Lifting Rationing on Economic Outcomes (ELSA)

		Education years 16+ (1)	Any college (2)	Employed (3)	Skilled occupation (4)	Above median wealth (5)	Economic sufficiency index (6)
Panel A. In utero + [0, 9 months]:							
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	0.041	0.042	0.896	0.000	0.000	0.000
N		12589	12438	12512	10203	11116	12589
Panel B. In utero + [10, 21 months]:							
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	0.021	0.050	0.744	0.050	0.050	0.036
N		10480	10329	10415	8395	9251	10480
Never-rationed mean		0.303	0.502	0.700	0.550	0.476	0.000
Panel B < Panel A (p-value)		0.800	0.767	0.630	0.570	0.470	0.701

Notes: Sample includes individuals born between 1950 and 1960, excluding those born between 1953q4 and 1954q4, who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Each model adjusts for sex, age, semester of birth, parental health history, and survey year indicators. Economic sufficiency indices follow Kling, Liebman and Katz (2007) and Hoynes, Schanzenbach and Almond (2016), averaging standardized z-scores across components. Z-scores use the mean and standard deviation among those born after rationing ended. The economic sufficiency index includes 16 or more years of education, medium- or high-skilled occupation, employment, above-median wealth, and log-transformed wealth. Standard errors clustered at the individual level are reported in parentheses. Mean is reported for never-rationed adults so remains unchanged across panels. Romano–Wolf adjusted p-values account for multiple hypothesis testing. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 6: RDD Estimates of the Effect of Lifting Rationing on Childhood Cognitive Outcomes

		Cognition age 7 (1)	Cognition age 9 (2)	Cognition age 11 (3)
Born after rationing	β	−0.081	−0.214***	−0.256***
	SE	(0.046)	(0.047)	(0.045)
N		9,436	9,336	9,241

Notes: Robust standard errors clustered at the birth-month level 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 in December 1962. Children born from October 1953 to June 1954 are excluded. Outcomes are standardized, so coefficients are interpreted as standard deviation changes. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Table 7: RDD Estimates of the Effect of Lifting Rationing on Diet

Panel A. Sugars					
		Free sugars≥5% of energy intake	Log(Free sugars)	Log(Intrinsic sugars)	Log(Fiber)
		(1)	(2)	(3)	(4)
Born after rationing	β	0.102**	0.224**	−0.185**	−0.090**
	SE	(0.044)	(0.096)	(0.079)	(0.041)
	Single p-value	0.020	0.020	0.019	0.028
	RW p-value	0.001	0.001	0.000	0.002
N		2,853	2,853	2,853	2,853
Rationed mean		0.79	48.41	31.98	14.69
Panel B. Food groups					
		Any vegetables	Any fruits	Any sweets	Any meats
		(5)	(6)	(7)	(8)
Born after rationing	β	−0.007	−0.142***	0.080*	0.044
	SE	(0.042)	(0.052)	(0.041)	(0.046)
	Single p-value	0.863	0.007	0.052	0.339
	RW p-value	0.802	0.000	0.011	0.413
N		2,853	2,853	2,853	2,853
Rationed mean		0.78	0.66	0.60	0.52
Panel C. Macronutrients					
		Log(Total energy intake)	Log(Fats)	Log(Protein)	Log(Carbohydrates)
		(9)	(10)	(11)	(12)
Born after rationing	β	−0.001	0.042	−0.027	−0.019
	SE	(0.041)	(0.029)	(0.028)	(0.025)
	Single p-value	0.981	0.151	0.348	0.445
	RW p-value	—	0.102	0.413	0.413
N		2,853	2,853	2,853	2,853
Rationed mean		1,768.26	65.94	75.45	209.99

Notes: Standard errors clustered at the individual level are reported in parentheses. Unadjusted p-values are reported alongside Romano–Wolf p-values adjusted for multiple hypothesis testing. The sample includes UK-born individuals born between 1950 and 1960 and surveyed in NDNS at least once between 2008 and 2016, excluding those born between 1953q4 and 1954q2. The sample excludes respondents reporting unusual food or beverage intake and observations above the 99th percentile of free sugar intake. Log-transformed continuous outcomes are measured in grams, except total energy intake, which is measured in kcal. Intrinsic sugars exclude lactose. Free sugars ≥5% indicates whether free sugar calories are at least 5% of total energy intake. Food-group outcomes indicate any consumption in the last 24 hours; sweets include sugar preserves, sweet spreads, sugar confectionery, and chocolate confectionery. All outcomes are adjusted for total energy intake except total energy intake itself, cohort-specific linear trends, sex, height, race, calendar month, day of week, and survey-year fixed effects. * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

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Supplemental Appendix

A. Tables

Table A.1: Baseline Childhood Characteristics

Characteristic	Rationed (%)	Never Rationed (%)	Diff.
Panel A: ELSA			
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	
Panel B: UK Biobank			
High genetic risk for obesity	49.8	49.5	0.3
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	
Panel C: Aberdeen data			
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
Father's occupation at birth			
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. = difference in means. 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. Adults born between October 1953 and June 1954 are excluded. 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 coded as unskilled. Significant difference is marked at * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A.2: Baseline Childhood Characteristics by Genetic Risk in UK Biobank

	Low Genetic Risk			High Genetic Risk		
	Rationed %	Never-rationed %	Difference (p.p.)	Rationed %	Never-rationed %	Difference (p.p.)
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 the median of the BMI polygenic score distribution) and high genetic risk (above the median of the 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 because these questions were asked of a subset of respondents. Reported means are unadjusted, but 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. Significance is based on Romano–Wolf adjusted p-values: * $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table A.3: Analysis Specifications Across Datasets

			Included birth dates		Exposure length		
Dataset	**Running variable**	**Excluded birth dates****	**Rationed**	**Never rationed**	**Minimum**	**Maximum**	**Tables**
ELSA	Birth years*	1953q4–1954q4	1950m1–1953m9	1955m1–1959m12	*In utero (IU)*	IU + 9 months postnatal	Table 2A, Table 5A
	Birth years	1952q3–1954q4	1950m1–1952m6	1955m1–1959m12	IU + 10m	IU + 21m	Table 2B, Table 5B
UK Biobank	Birth quarters	1953q4–1954q2	1952m1–1953m9	1954m7–1956m3	IU	IU + 3m	Table 3, Table 4A
	Birth quarters	1953q2–1954q2	1952m1–1953m3	1954m7–1956m3	IU + 4m	IU + 9m	Table 4B
Aberdeen	Birth months	1953m10–1954m6	1950m10–1953m9	1954m7–1955m9	IU	IU	Table 6

Notes: *Six-month birth cohorts are used for robustness analyses reported in Tables A.4, A.5, and A.7. **Excluded birth dates are cohorts dropped because fetal or early-postnatal exposure cannot be cleanly classified. After the first row, *in utero* plus postnatal months is abbreviated IU + Xm; for example, IU + 9m denotes exposure *in utero* plus the first nine months after birth. Running variables indicate the temporal aggregation used in the main analyses. Included birth dates give the ranges for rationed and never-rationed cohorts. Exposure length reports the minimum and maximum sugar-rationing exposure among individuals born at the rationing cutoff. Letters appended to table numbers in the final column refer to panels within the referenced tables.

Table A.4: RDD Estimates of Lifting Rationing on Health Outcomes (Running Variable: Six-Month Birth Cohorts)

		Metabolic health		Cardiovascular conditions		Inflammation		Comorbidity		
		Diabetes	Cholesterol	Hypertension	Heart disease & stroke	C-reactive protein >3	Arthritis	Chronic diseases 3+	Metabolic syndrome	Metabolic disease index
		(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
Panel A: In utero + [0, 9 mths]:										
Born after rationing	β	0.042***	0.052***	0.064***	0.064*	0.006	0.098***	0.118***	0.066***	0.195***
	SE	(0.016)	(0.012)	(0.021)	(0.033)	(0.033)	(0.033)	(0.045)	(0.017)	(0.069)
N		12589	2435	6351	12588	12589	2481	12589	12589	12589
Panel B: In utero + [10, 21 mths]:										
Born after rationing	β	0.058**	0.068***	0.140**	0.045	0.069***	0.087	0.223***	0.126***	0.305***
	SE	(0.023)	(0.013)	(0.060)	(0.035)	(0.025)	(0.086)	(0.044)	(0.017)	(0.084)
N		10480	2027	5082	10479	10480	2091	10480	10480	10480
Never-rationed mean		0.076	0.045	0.231	0.293	0.064	0.230	0.252	0.085	-0.000

Notes. Robust standard errors clustered at the six-month birth cohort level are reported in parentheses; results are similar when standard errors are clustered at the individual level. Adults born in 1953q4 and 1954 are excluded. The sample includes individuals born between 1950 and 1960 who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Each model adjusts 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 Lifting 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***	-0.086***	-0.022	-0.119***	-0.132***	-0.241***
	SE	(0.020)	(0.026)	(0.026)	(0.031)	(0.025)	(0.052)
N		12589	12438	12512	10203	11116	12589
Panel B: In-utero+ [10, 21mths]:							
Born after rationing	β	-0.165***	-0.161***	-0.057***	-0.145**	-0.129***	-0.345***
	SE	(0.032)	(0.034)	(0.019)	(0.068)	(0.037)	(0.108)
N		10480	10329	10415	8395	9251	10480
Never-rationed mean		0.303	0.502	0.700	0.550	0.476	0.000

Notes. Robust standard errors clustered at the six-month birth cohort level are reported in parentheses; results are similar when standard errors are clustered at the individual level. The sample includes individuals born between 1950 and 1960 who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019; adults born in 1953q4 and 1954q4 are excluded. Each model adjusts for sex, age, seasonality at birth, and survey year indicators. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.6: 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 + 1 yr:				
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
Never-rationed mean		0.040	0.145	0.036

Notes. Robust standard errors in parentheses, clustered at the individual level. Each model adjusts for sex, age, seasonality at birth, and survey year indicators. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.7: 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, cutoff Apr.–Sept. 1953)						
Born after 1954	0.044**	0.068***	0.209***	-0.100**	-0.127***	-0.138***
	(0.021)	(0.022)	(0.077)	(0.043)	(0.044)	(0.041)
N	12589	12589	12589	12589	10203	11116
Y(=0) Mean	0.076	0.085	-0.000	0.253	0.565	0.460
B: 1955–1956q3 vs. 1956q4–1960 (after rationing, cutoff Apr.–Sept. 1956)						
Born after 1956q3	-0.022	-0.014	-0.108	0.023	-0.026	-0.010
	(0.035)	(0.033)	(0.136)	(0.052)	(0.062)	(0.064)
C: 1955–1957q3 vs. 1957q4–1960 (after rationing, cutoff Apr.–Sept. 1957)						
Born after 1957q3	0.019	-0.002	0.138	0.057	-0.047	0.033
	(0.028)	(0.027)	(0.123)	(0.042)	(0.055)	(0.058)
D: 1955–1958q3 vs. 1958q4–1960 (after rationing, cutoff Apr.–Sept. 1958)						
Born after 1958q3	-0.003	-0.005	0.059	0.005	-0.040	0.004
	(0.028)	(0.025)	(0.119)	(0.043)	(0.055)	(0.058)
N	6537	6537	5042	6537	5703	4415
Never-rationed mean	0.076	0.081	-0.000	0.244	0.570	0.460

Notes. 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.8: Varying RDD Bandwidth: RDD Estimates of the Effect of Lifting Rationing on Health Outcomes

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

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

Table A.9: Varying RDD Bandwidth: RDD Estimates of the Effect of Lifting Rationing on Economic Outcomes

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

Notes. Robust standard errors in parentheses, clustered at the individual level. These estimates are not adjusted for multiple hypothesis testing. The sample includes individuals born between 1950 and 1960 who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Bandwidth used for regression discontinuity varies by panel; N shows the effective estimation sample size. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.10: Varying Kernel: RDD Estimates of the Effect of Lifting Rationing on Health Outcomes

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

Notes. Robust standard errors in parentheses, clustered at the individual level. The sample includes individuals born between 1950 and 1960 who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Cardiovascular is an indicator for hypertension, stroke, or heart disease compared with diagnosis with none of these diseases. Chronic 2+ indicators for at least two of the following conditions: hypertension, stroke, heart disease, diabetes, arthritis, and high cholesterol. Each model adjusts for age. Kernel used for regression discontinuity varies by panel. Bandwidth is 4 years to the left of the cutoff and 6 years to the right in all models. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.11: Varying Kernel: RDD Estimates of the Effect of Lifting Rationing on Economic Outcomes

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

Notes. Robust standard errors in parentheses, clustered at the individual level.The sample includes individuals born between 1950 and 1960 who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Any higher education indicates whether the respondent attended at least some post-secondary schooling. Skilled job is conditional on current employment and classified using the UK Standard Industrial Classification 8-code system, compared with low-skilled occupations. Each model adjusts for age. Kernel used for regression discontinuity varies by panel. Bandwidth is 4 years to the left of the cutoff and 6 years to the right in all models. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.12: Falsification Test: Health Outcomes by Migraine Genetic Risk

		Obesity 90th pct (1)	Metabolic syndrome (1, 0) (2)	Metabolic health index (z-score) (3)
A) Low migraine genetic risk:				
Born after rationing	β	0.024**	0.026**	0.094**
	SE	(0.011)	(0.012)	(0.040)
N		13768	13492	13784
Never rationed mean		0.100	0.103	-0.010
B) High migraine genetic risk:				
Born after rationing	β	0.032***	0.031**	0.103**
	SE	(0.012)	(0.013)	(0.040)
N		13751	13442	13769
Never rationed mean		0.100	0.108	0.010

Notes. Robust standard errors from the local linear RDD specification are reported in parentheses. Coefficients for binary outcomes are in probability units. The sample includes white UK Biobank respondents, excluding those born in 1953q4–1954q2. Low and high migraine genetic risk are defined using the sample median of the polygenic score for migraine susceptibility. Obesity 90th pct indicates abdominal obesity at or above the 90th percentile of the never-rationed group. Metabolic syndrome indicates diagnosis with at least two metabolic syndrome components. The metabolic health index is a z-score index of metabolic health outcomes. All models use a triangular kernel, a first-order polynomial, and covariates. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.13: RDD Estimates of the Effect of Lifting Rationing on Health Outcomes (ELSA), adjusted for number of live births

		Metabolic disease			Cardiovascular		Inflammation			
		Diabetes	Diabetes HbA1c≥7%	Cholesterol	Hypertension	Heart disease or stroke	CRP≥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	0.039*	0.048	0.087**	0.068*	0.023	0.121***	0.177***	0.096***	0.222***
	SE	(0.021)	(0.030)	(0.039)	(0.039)	(0.024)	(0.043)	(0.036)	(0.022)	(0.077)
N		12589	2435	6351	12588	12589	2481	12589	12589	12589
B) In utero + [10, 21 mths]:										
Born after rationing	β	0.068**	0.080**	0.163***	0.046	0.079**	0.053	0.200***	0.114***	0.301**
	SE	(0.034)	(0.040)	(0.063)	(0.067)	(0.032)	(0.076)	(0.059)	(0.036)	(0.123)
N		10480	2027	5082	10479	10480	2091	10480	10480	10480
Never-rationed mean		0.082	0.048	0.240	0.297	0.077	0.245	0.253	0.088	-0.000

Notes. Sample includes individuals born between 1950 and 1960, excluding those born between 1953q4 and 1954q4, who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Each model adjusts for sex, age, parental health history, survey year indicators and number of live births. Semester of birth is included for all outcomes except cholesterol and CRP/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+ indicates three or more diseases: hypertension, diabetes, heart disease, stroke, and cholesterol. The 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, so higher values indicate worse health. Estimates are reported with standard errors clustered at the individual level. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.14: RDD Estimates of the Effect of Lifting Rationing on Economic Outcomes (ELSA), adjusted for number of live births

		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	-0.117***	-0.092**	-0.035	-0.132***	-0.138***	-0.279***
	SE	(0.043)	(0.045)	(0.033)	(0.044)	(0.041)	(0.084)
N		12589	12438	12512	10203	11116	12589
B) In utero + [10, 21 mths]:							
Born after rationing	β	-0.172**	-0.168**	-0.052	-0.172**	-0.137**	-0.365***
	SE	(0.072)	(0.075)	(0.051)	(0.075)	(0.068)	(0.137)
N		10480	10329	10415	8395	9251	10480
Never-rationed mean		0.303	0.502	0.700	0.550	0.476	0.000

Notes. Sample includes individuals born between 1950 and 1960, excluding those born between 1953q4 and 1954q4, who are ages 50–64 and surveyed in ELSA at least once between 2002 and 2019. Each model adjusts for sex, age, semester of birth, parental health history, survey-year indicators, and number of live births. Economic sufficiency indices follow Kling, Liebman and Katz (2007) and Hoynes, Schanzenbach and Almond (2016), averaging standardized z-scores across components. Z-scores use the mean and standard deviation among those born after rationing ended. The economic sufficiency index includes 16 or more years of education, medium- or high-skilled occupation, employment, above-median wealth, and log-transformed wealth. Estimates are reported with standard errors clustered at the individual level. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A.15: Policy Changes Around the Rationing Cutoff: Differential Effects at the RDD Threshold

Reform/Policy	Timing	Assessment at the 1953Q4/1954Q1 Cutoff	Discontinuity
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

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Table A.15 – *Continued from previous page*

Reform/Policy	Timing	Assessment at the 1953Q4/1954Q1 Cutoff	Discontinuity
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
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

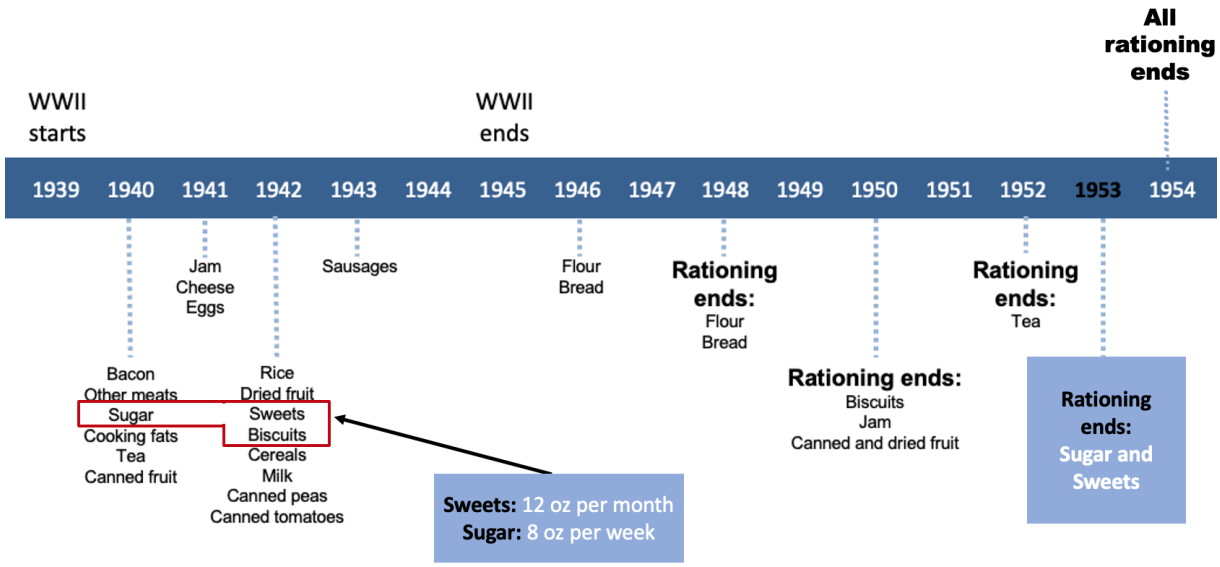
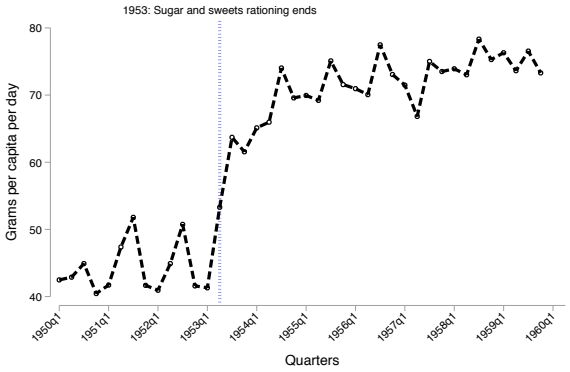


Figure A.1: Food Rationing Timeline in the UK: 1939–1954

Notes: Sugar and sweets rationing ended in September 1953; remaining food rationing ended in July 1954.

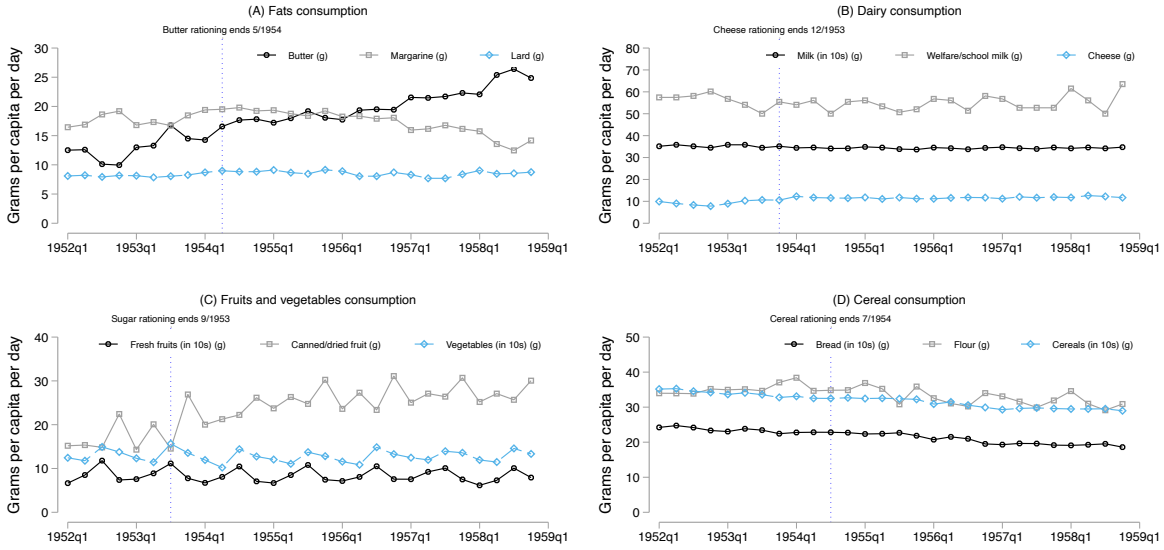
Figure A.2: Daily Per Capita Sugar Intake for Adults Between 1950 and 1960

(a) Daily per capita intake of sugar



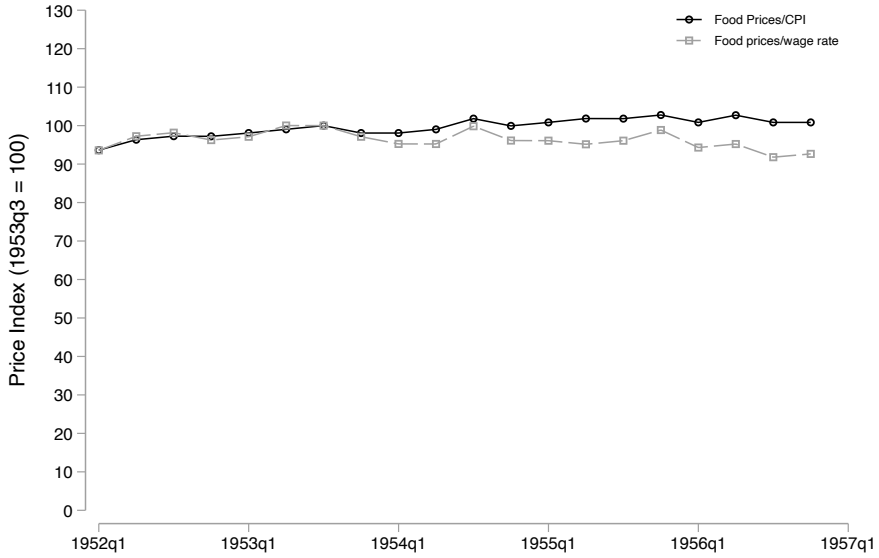
Notes. Sourced from Gracner, Boone and Gertler (2024).

Figure A.3: Consumption of Various Food Groups Between 1950 and 1960



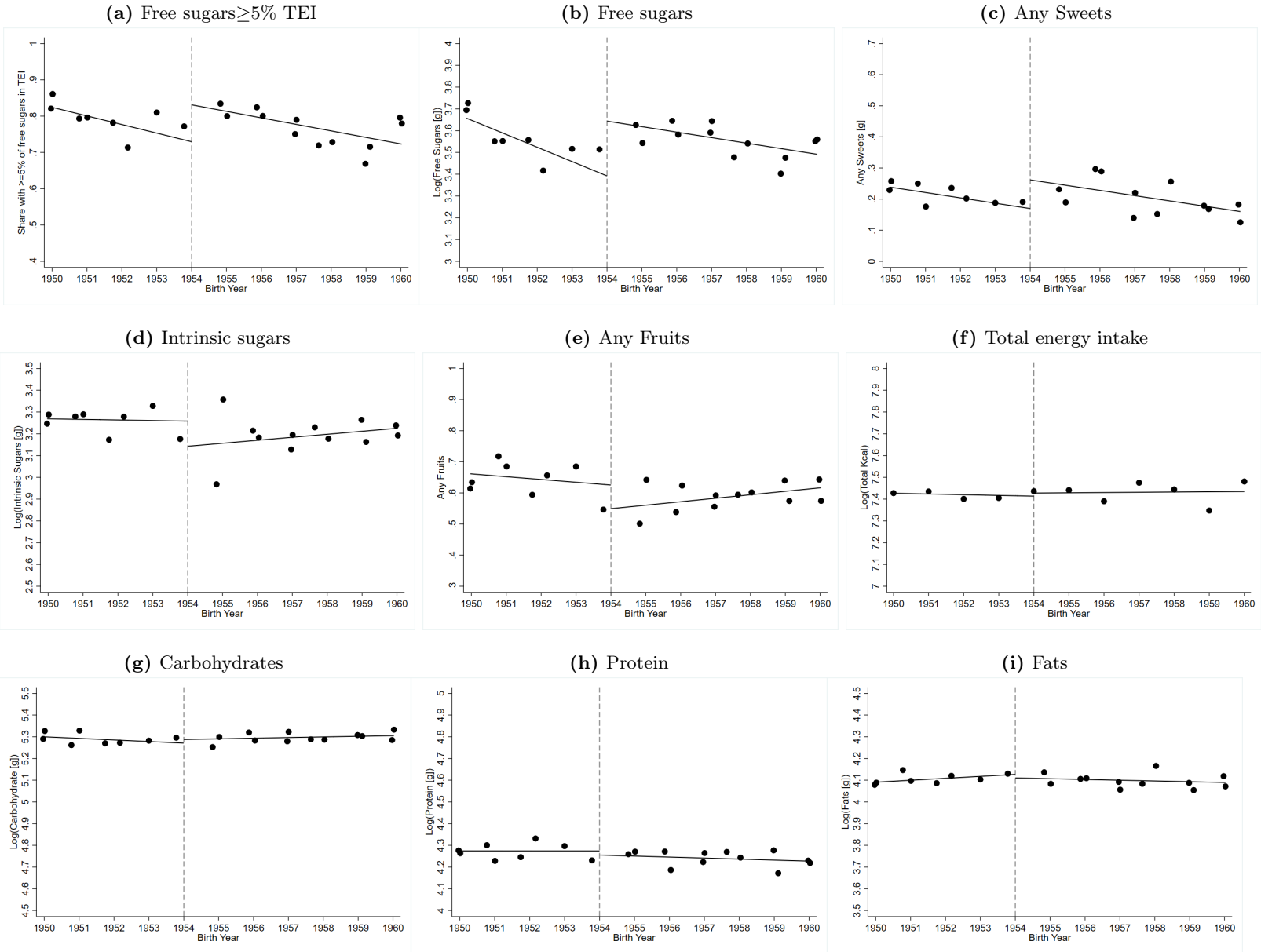
Notes. Sourced from Gracner, Boone and Gertler (2024).

Figure A.4: Food Affordability Between 1950–1960



Notes. We compiled quarterly food-price series, expressed relative to general inflation and wage growth, for 1952q1–1956q4 and normalized to 1953q3 = 100. Price data were drawn from historical National Food Survey reports, which provide consistent quarterly series over this period. Figure sourced from Gracner, Boone and Gertler (2024).

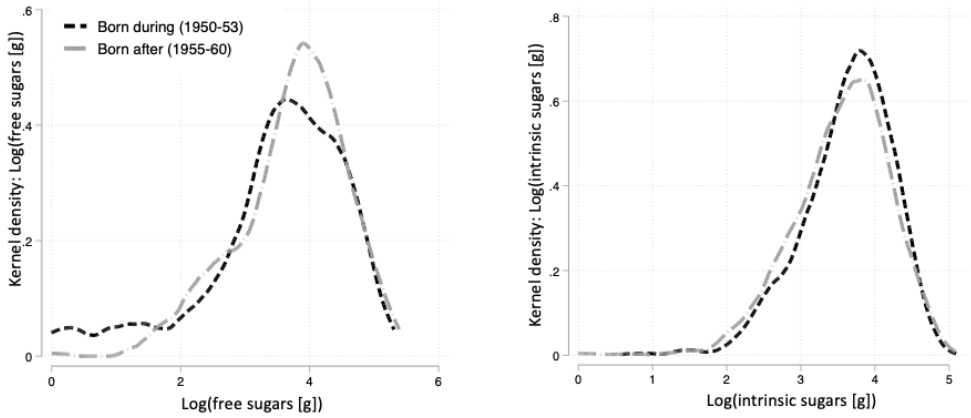
Figure A.5: Diet in 2008–2019 Among Older Adults Born Between 1950 and 1960



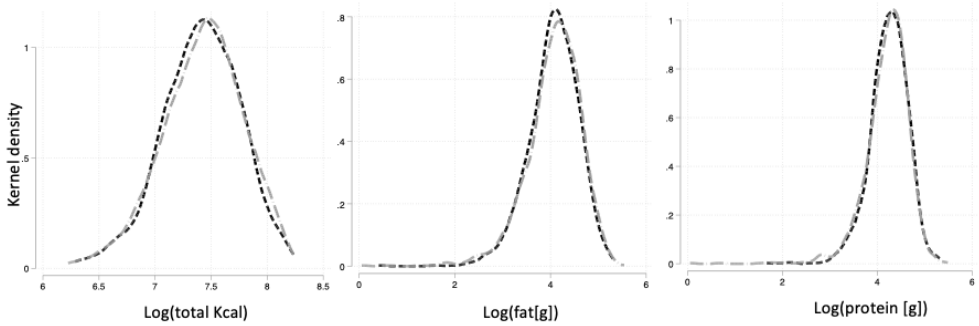
Notes. Estimates based on the National Diet and Nutrition Survey, 2008–2016. Markers show binned means by birth year; lines show local linear fits estimated separately on each side of the exposure threshold.

Figure A.6: Kernel Densities of Current Diet Outcomes

(a) Sugars: free and intrinsic



(b) Total calories, fats, and protein



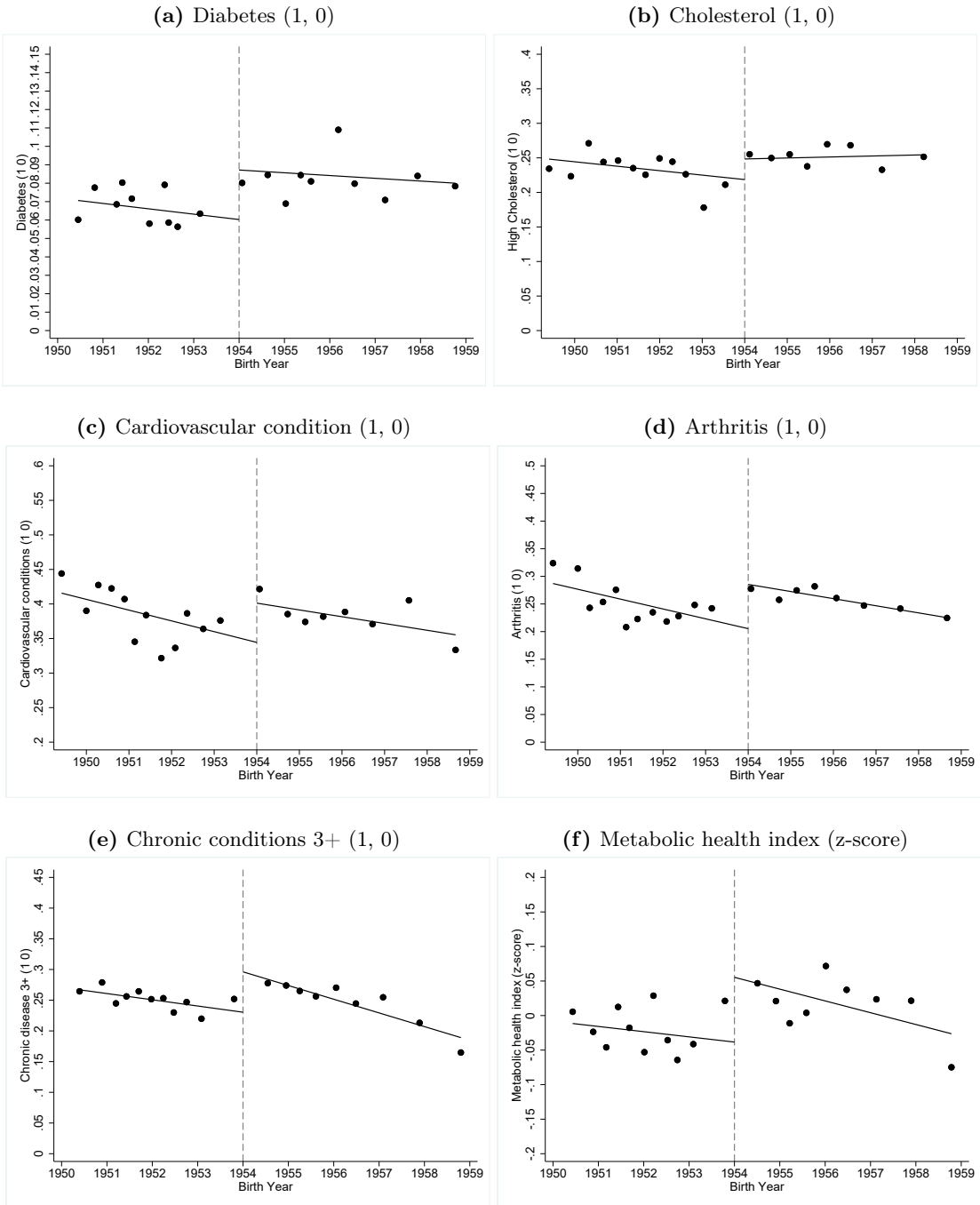
Notes. Authors' calculations using the National Diet and Nutrition Survey, 2008–2016.

Figure A.7: Age Overlap Between Cohorts Born Between 1950 and 1960 in ELSA, 2002–2018

	Birth year (common support: ages <65)										
Survey year	1950	1951	1952	1953	1954	1955	1956	1957	1958	1959	1960
2002	52	51	50	49	48	47	46	45	44	43	42
2004	54	53	52	51	50	49	48	47	46	45	44
2006	56	55	54	53	52	51	50	49	48	47	46
2008	58	57	56	55	54	53	52	51	50	49	48
2010	60	59	58	57	56	55	54	53	52	51	50
2012	62	61	60	59	58	57	56	55	54	53	52
2014	64	63	62	61	60	59	58	57	56	55	54
2016	66	65	64	63	62	61	60	59	58	57	56
2018	68	67	66	65	64	63	62	61	60	59	58

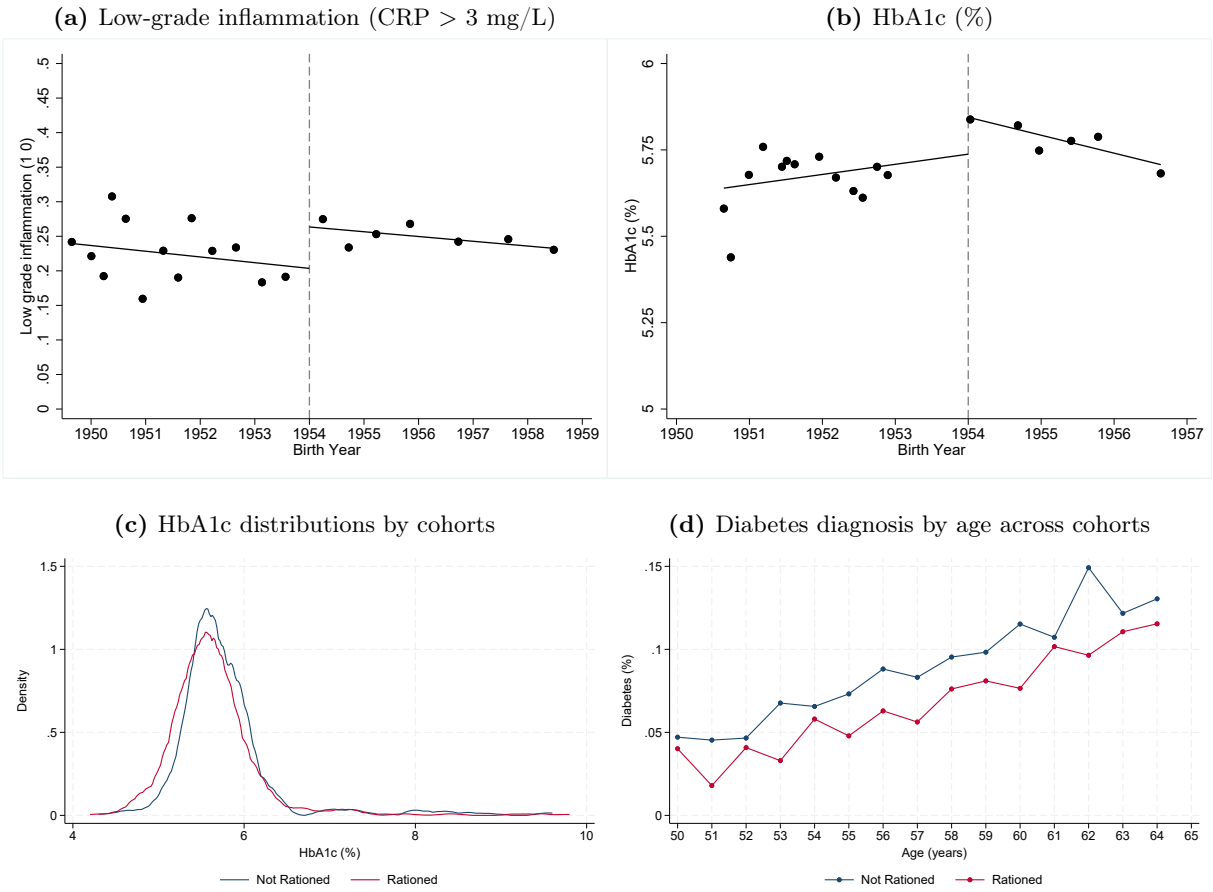
Notes. Based on nine waves of ELSA, 2002–2018.

Figure A.8: ELSA: Current Health Outcomes Across Birth Cohorts



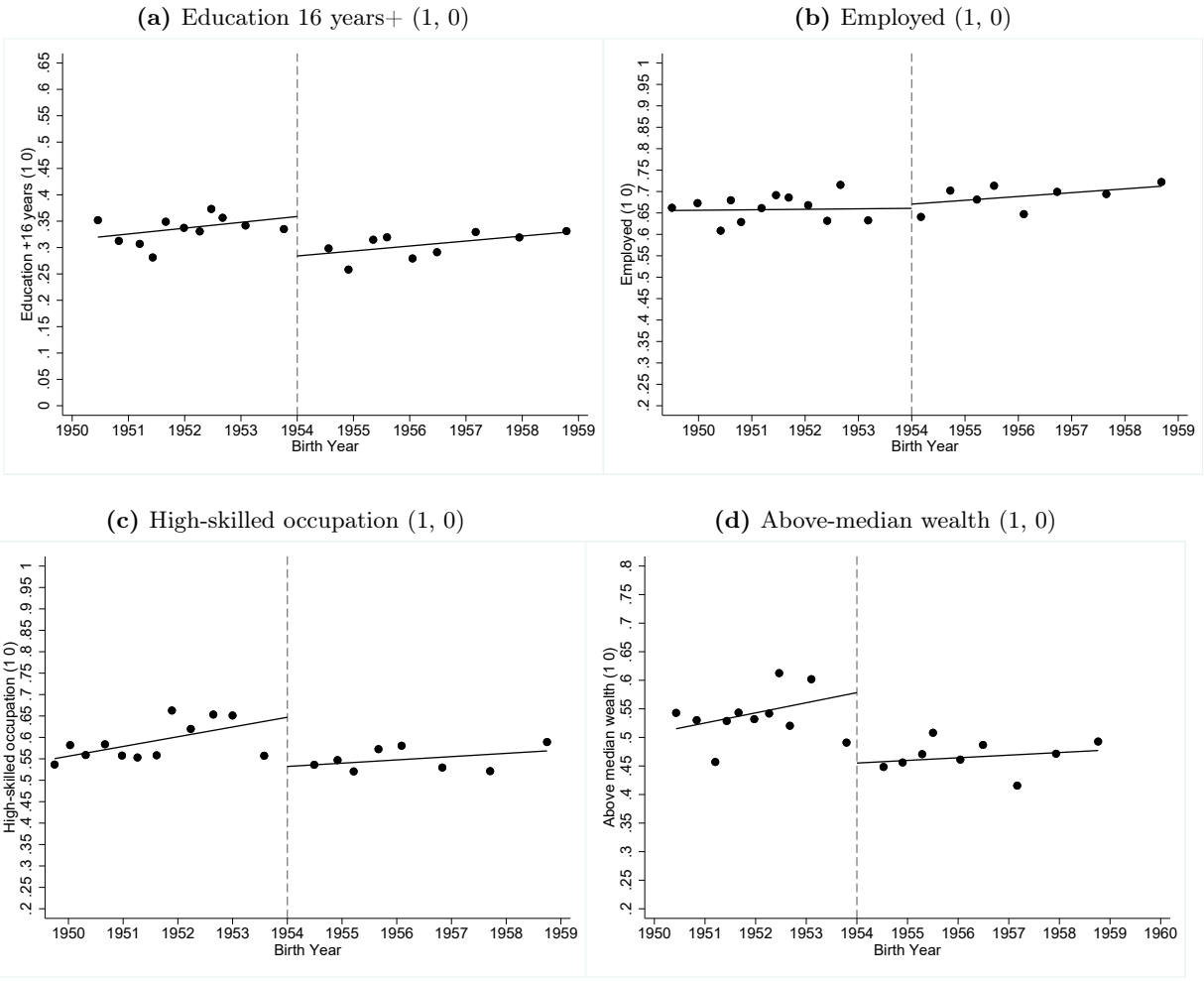
Notes. Figures show cohort differences in the probability of diet-related disease diagnosis. Markers show covariate-adjusted binned means by birth year; lines show local linear fits estimated separately on each side of the exposure threshold. Estimates are adjusted for age, sex, birth quarter, and parental disease history. Cardiovascular condition indicates diagnosis of hypertension, stroke, or heart disease. Chronic conditions 3+ indicates diagnosis of three or more conditions: diabetes, hypertension, high cholesterol, heart disease, and arthritis. The metabolic health index is the average of z-scores, standardized to the post-rationing cohort, for diabetes, high cholesterol, heart disease, stroke, and hypertension.

Figure A.9: ELSA: Biomarker Outcomes Across Birth Cohorts



Notes. Figures show cohort differences in diet-related disease outcomes measured via biomarkers in ELSA. Models adjust for age, sex, and parental disease history indicators. Low-grade inflammation is defined as C-reactive protein >3 mg/L at the time of survey. CRP values >10 mg/L are excluded because they likely reflect acute infection rather than chronic inflammation. The HbA1c distribution, limited to overlapping ages 52–56, differs significantly between rationed and never-rationed individuals (Kolmogorov–Smirnov D = 0.149, p<0.001).

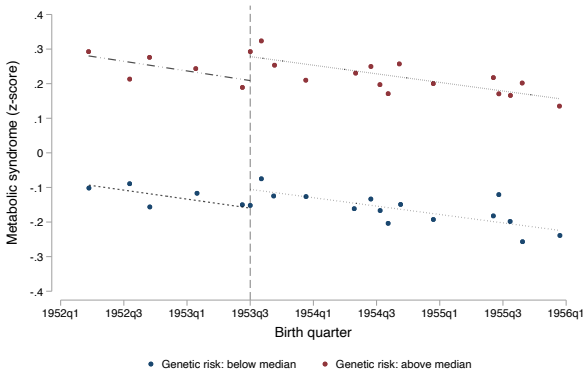
Figure A.10: ELSA: Current Economic Outcomes Across Birth Cohorts



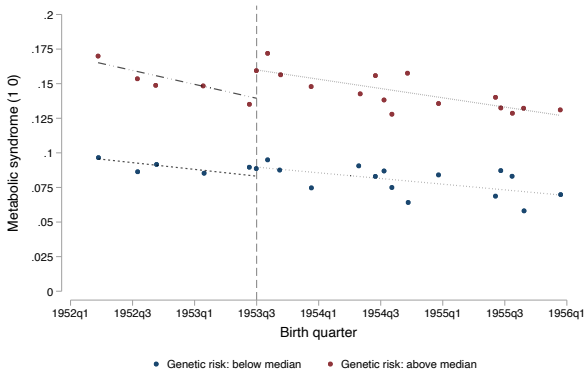
Notes. Figures show cohort differences in selected economic outcomes using English Longitudinal Study of Ageing, 2002–2019. Markers show covariate-adjusted binned means by birth year; lines show local linear fits estimated separately on each side of the exposure threshold. Estimates are adjusted for age, sex, first calendar quarter of birth, and parental health indicators.

Figure A.11: UK Biobank: Health Outcomes by Genetic Risk for Obesity

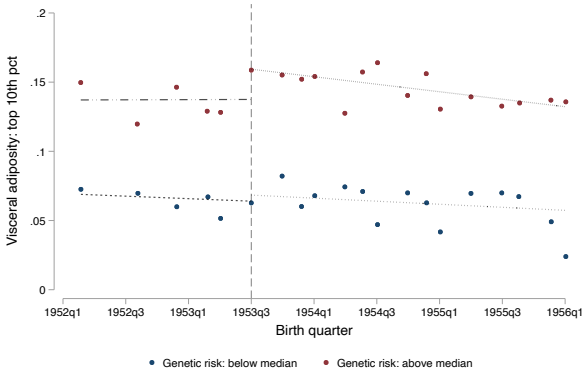
(a) Metabolic Disease Index (z-score)



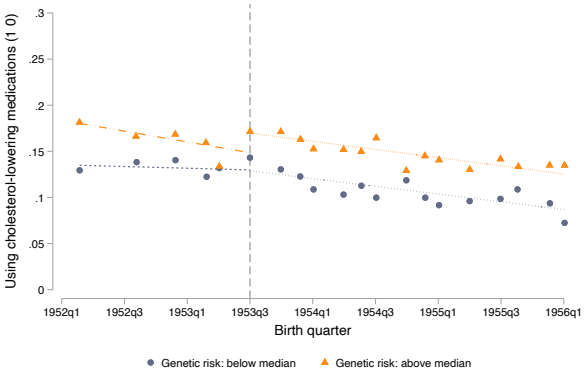
(b) Metabolic syndrome (1, 0)



(c) Abdominal obesity, 90th pct

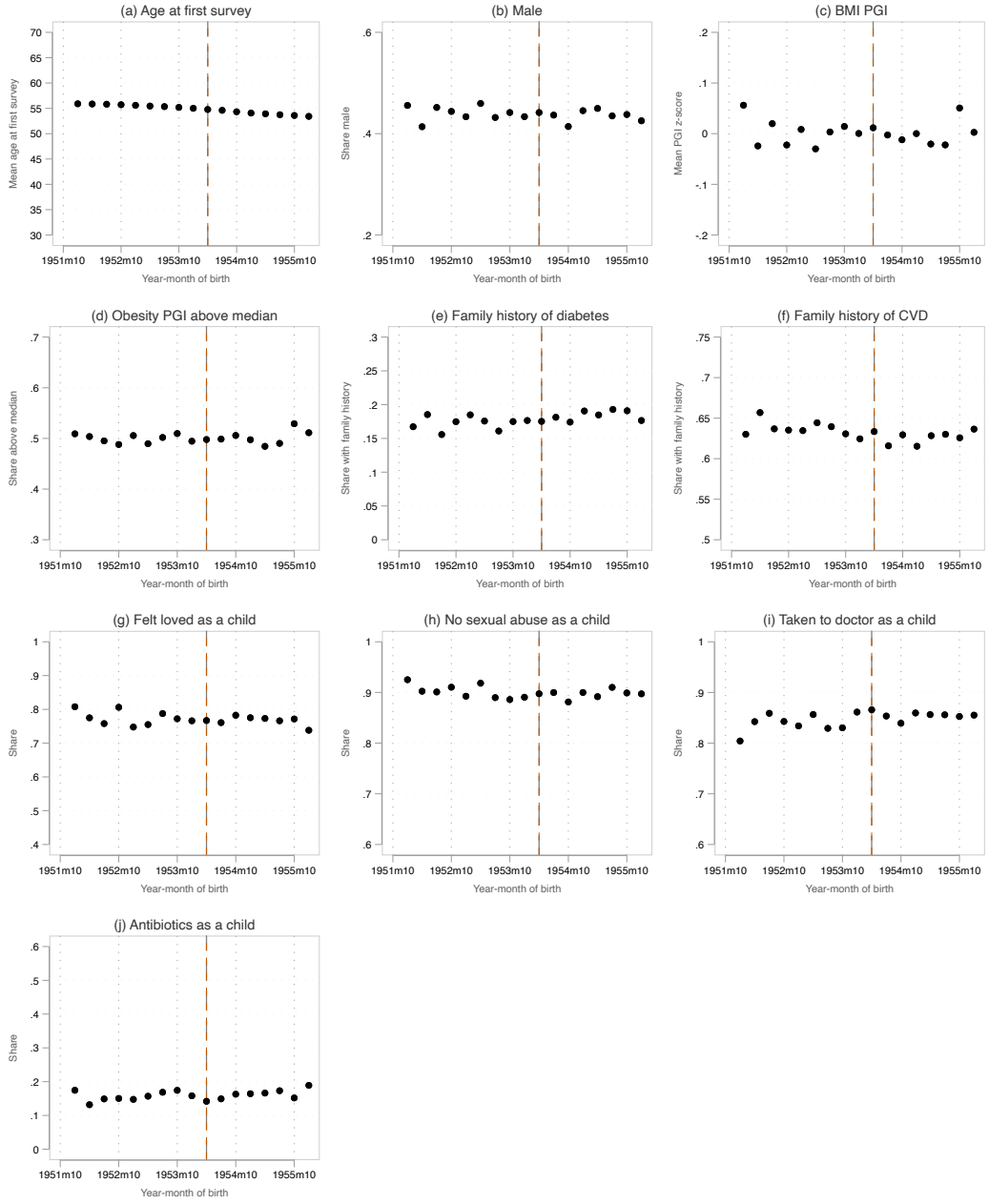


(d) Cholesterol-lowering medication use



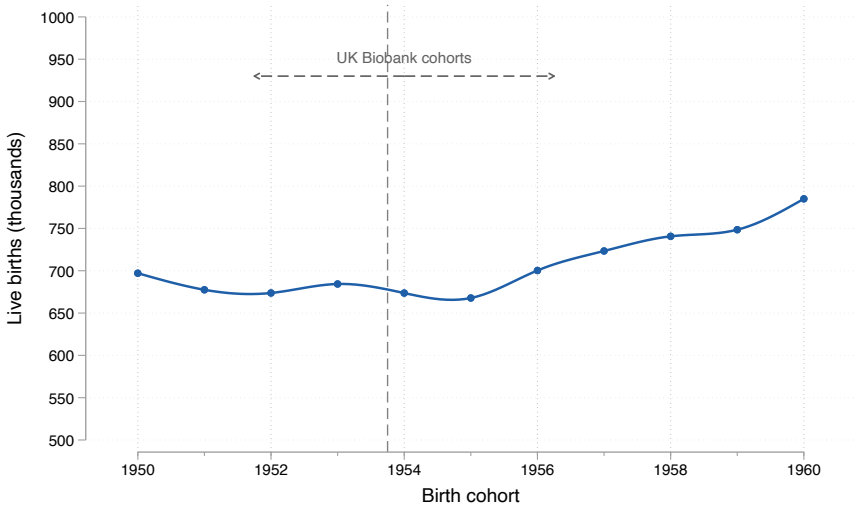
Notes. Figures based on UK Biobank data. Markers show covariate-adjusted binned means by birth quarter; lines show local linear fits estimated separately on each side of the exposure threshold. Genetic risk is defined using the BMI polygenic index, split at the sample median.

Figure A.12: Baseline Characteristics Across Cohorts of Birth, UK Biobank



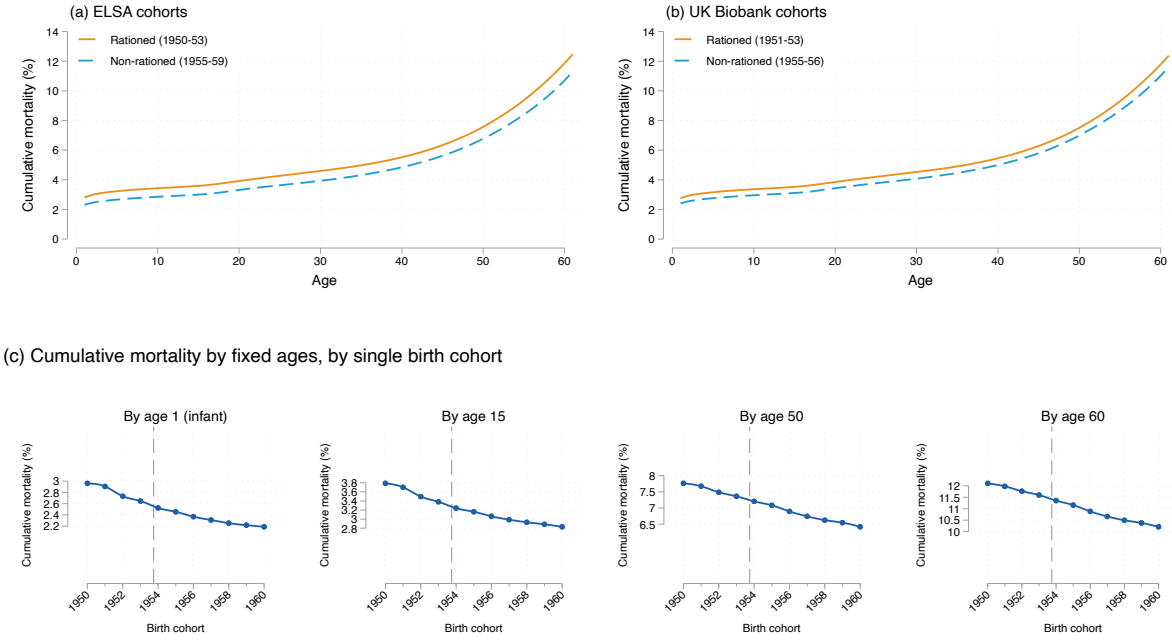
Notes. Binned means of baseline characteristics by cohort of birth, with bins constructed separately on each side of the exposure threshold; the vertical line marks the end of rationing in September 1953. Sample includes white, English-born participants born October 1951–March 1956. Age is measured at baseline assessment (2006–2010) and so varies across cohorts by construction. Panels (g)–(j) come from retrospective childhood questions asked of a subset of participants. No characteristic changes discontinuously at the threshold: regression-discontinuity estimates range from −0.047 to 0.015 and none approaches conventional significance.

Figure A.13: Live Births by Birth Cohort, England and Wales



Notes. Annual live births by year of birth, England and Wales, from the Human Mortality Database (England & Wales). The vertical dashed line marks the end of sugar and sweets rationing in September 1953; the horizontal dashed arrow marks the birth cohorts included in the UK Biobank sample (October 1951–March 1956).

Figure A.14: Cumulative Mortality for Rationed and Never-Rationed Cohorts, England and Wales



Notes. Cumulative mortality is the share of a birth cohort that died before the stated age, computed as cumulative cohort deaths divided by live births in the cohort's year of birth. Data are for England and Wales, both sexes, from the Human Mortality Database (England & Wales). Deaths are taken from the Lexis-triangle series, which reports year of birth alongside age and calendar year of death. Panels (a) and (b) plot cohort-size weighted averages for the cohort ranges used in the ELSA and UK Biobank analyses. Panel (c) plots cumulative mortality by fixed ages against single birth cohort. The vertical dashed line marks the end of sugar and sweets rationing in September 1953. Denominators are live births, so the series measures mortality within the cohort as born and does not net out migration.