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THE SWEET LIFE: THE LONG-TERM EFFECTS OF A SUGAR-RICH EARLY CHILDHOOD

Paul Gertler
Tadeja Gracner

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Corresponding author: Tadeja Gracner: RAND Corporation, email: tgracner@rand.org. Paul Gertler: UC Berkeley and NBER, email: gertler@berkeley.edu. This project was supported by the National Institute on Aging (grants P30AG012815 and R01AG06548201) and Berkeley's Center for the Economics and Demography of Aging. The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication. We are grateful to Bai Liang and Kapinos Kandice for helpful discussions, and audiences at many conferences and seminars for constructive comments. We would also like to thank excellent research assistants Claire Boone and Aditya Polisetty. All errors are our own. The authors have no financial or material interests in the results discussed in this paper. The views expressed herein are those of the authors and do not necessarily reflect the views of the National Bureau of Economic Research.

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ABSTRACT

We show that sugar-rich diet early in life has large adverse effects on the health and economic well-being of adults more than fifty years later. Excessive sugar intake early in life led to higher prevalence of chronic inflammation, diabetes, elevated cholesterol and arthritis. It also decreased post-secondary schooling, having a skilled occupation, and accumulating above median wealth. We identified elevated sugar consumption across lifespan as a likely pathway of impact. Exploiting the end of the post-WWII rationing of sugar and sweets in 1953 in the United Kingdom, we used a regression discontinuity design to identify these effects.

Paul Gertler
Haas School of Business
University of California, Berkeley
Berkeley, CA 94720
and NBER
gertler@haas.berkeley.edu

Tadeja Gracner
RAND Corporation
1776 Main St.
Santa Monica, CA 90401
tgracner@rand.org

1 Introduction

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

Sugar-rich diets start early in life through maternal consumption of sugary foods and drinks during pregnancy and breastfeeding (Goran et al., 2019; Portella et al., 2012), and infant and toddler intake of commercially prepared beverages and foods (Walker and Goran, 2015). Pregnant women consume an average of 21 tsp of added sugars daily (Cioffi et al., 2017), and more than half of lactating mothers exceed the maximum daily limit of 10% of TEI (Goran et al., 2019). Despite nutritional guidelines that recommend zero free sugars for children below age of two (Dewey et al., 2021), 61% of infants and 98% of toddlers consume at least some free sugar daily. In fact, their free sugar intake increases with age (Park et al., 2014; Fiorito et al., 2010) and by the time children reach two years old, it exceeds the daily limit set for adults (Herrick et al., 2020; Vos et al., 2017).[2] Such a diet is sourced from commercially prepared infant and toddler foods, the vast majority of which are very high in free sugars.[3] These products are being aggressively advertised (Harris and Pomeranz, 2020) and supplied; the number introduced into the market has increased fourfold over the last decade alone (Romo-Palafox et al., 2020; Palafox and Harris, 2017). Despite growing concerns over these trends, the long-term implications of

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

[2] This is not unique to the US: free sugar intake among toddlers ranges from 12% of TEI in countries like Denmark, Sweden, and South Africa to a whopping 25% in Portugal (WHO, 2015b). In Mexico and Chile, the majority of toddlers consume sweetened beverages (Essman et al., 2018; Afeiche et al., 2018) and by 5 years old consume more than 10 tsp of free sugar per day (Afeiche et al., 2018; Herrick et al., 2020).

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

exposure to excessive sugar intake early in life are not well known.

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

We employ a regression discontinuity design (RDD) to examine the impact of early sugar rich diets on the health and economic well-being by those who were born during rationing (1950-1953) and just after rationing ended (1955-1959). We use data from the English Longitudinal Survey of Aging (2008-2018) to examine the impact on adults at ages 50-65.

We find that greater exposure to a sugar-rich diet early in life leads to worse adult health. The end of rationing increased the prevalence of chronic inflammation by 8.8 percentage points (pp) (or 33%), defined as C-reactive protein (CRP) blood levels above 3ml/l. Independent of total calorie intake, elevated CRP levels are consistently linked to sugar-rich diet and insulin resistance, and identified as a mechanism for chronic disease, such as diabetes, heart disease or arthritis.[4] Related, we find worsened adult metabolic health: the prevalence of diabetes increased by 3.6pp or 52%, as did prevalence of elevated cholesterol, of arthritis and of having two or more diet-related chronic diseases.

We also find that early exposure to a sugar-rich diet affects human capital accumulation and economic well-being. On average, the end of rationing reduced the probability of having at least some post-secondary education by 9.2 percentage points or 18.4%. While it did not affect the probability of being employed, it reduced the likelihood of working in a skilled profession and above median wealth accumulation.

What drives these long-lasting impacts excessive early-life intake of sugar on adult economic

[4]Elevated sugar consumption has been repeatedly associated with systemic low-grade or chronic inflammation (Calder et al., 2011), which is in turn associated with increased risk of metabolic disease, such as diabetes or arthritis (Grundy, 2003; Danesh et al., 2000; Luft et al., 2013; Lontchi-Yimagou et al., 2013).

and health outcomes? One way may be through altered physiological programming due to adverse nutrition; also known as the developmental-origins hypothesis or Barker hypothesis (Martyn and Barker, 1994). Animal studies have found that - independent of total calorie intake - diet rich in sugars and carbohydrates *in utero* establishes permanent metabolic states that elevate lifetime risk of chronic disease later in life and may be epigenetic in nature (Rosenfeld, 2015; Bateson et al., 2004; Pawlak et al., 2004; Langley-Evans and Sculley, 2005; Barker, 1998b; Martyn and Barker, 1994; Hales, 1997; Jackson, 2002; Bayol et al., 2008). Excess sugar intake early in life has also been shown to hinder their hippocampus' development in rodents - a region of the brain associated with 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), resulting in a long-term deficit of both (Noble et al., 2019). Furthermore, maternal sugar-rich diets in humans have been linked not only with a greater risk for developing metabolic disorders (Hillier et al., 2007), but also with lower cognition (Casas et al., 2013), poor performance in executive tasks (Tanda et al., 2013; Hinkle et al., 2012; Neggers et al., 2003), or lower perceptual reasoning and memory in their offspring (Li et al., 2018; Kamijo et al., 2012).

Another way may be through elevated sugar intake across lifespan, as children's food preferences correlate strongly with their diet later in life (Drewnowski, 1997; Mennella and Bobowski, 2015). This is particularly salient for sugar, which may be addictive. Although humans have an innate preference for sweetness (Drewnowski et al., 2012; Sullivan and Brumfield, 2016; Mennella and Bobowski, 2015), its degree is modified by exposure to sugar especially within the first five years of life (Birch et al., 2007; Jayasinghe et al., 2017; Birch, 1999). In contrast to other nutrients, such as fat or proteins, sugar produces characteristics of craving, bingeing, and withdrawal, and the reward for sugar intake may even surpass that of cocaine (DiNicolantonio et al., 2018; Avena et al., 2009). Such physiological responses early in life, combined with an environment rich with sugar and sweets, facilitate sugar over-consumption that increases with age and can last for a lifetime. Indeed, sugar consumption in infancy predicts higher sugar intake in later childhood and adolescence, and has been shown to quickly increase with age (Park et al., 2014; Fiorito et al., 2010).

Accordingly, we find evidence that the end of sugar and sweets rationing in the UK elevated sugar consumption in the cohorts born post-rationing across their lifespan using data from the National Diet and Nutrition Survey (NDNS, 2008-2016). Specifically, we find that five decades or more into adulthood, children born just after the end of sugar and sweets rationing consumed about 22.4% or 11 grams of sugar more than those born just before; an increase equivalent to sugar present in about three Oreo cookies or half of an 8oz soda can per day.[5] Though these

[5]Moreover, it decreased intake of intrinsic sugars and fiber, both present in whole vegetables and fruits.

calorie differences may seem small, excess of 150 calories daily alone (equivalent to three Oreos or one can of Pepsi) explained a 10-12 pounds increase in median weight observed between 1980-2000 in the US (Cutler et al., 2003). The end of rationing is associated with a 15.6 percentage points (pp) or about 20% increase in the likelihood of exceeding the WHO strict maximum recommended daily free sugar amount (i.e., 5% of TEI). Importantly, we observe no long-term effect in adult daily total calories, protein, carbohydrates, and fat intake. We do, however, find evidence of substitution of free sugars for healthier intrinsic sugars in fruits.

Our study makes several important contributions to the literature linking nutrition in-utero and during early childhood to economic and health outcomes later in life. First, we expand the literature relating early-life excessive sugar intake to long-term health. Empirical work studying *causal* impacts of early-life sugary diet on adult health largely employs animal models (Antar et al., 1970; Samuelsson et al., 2008; Pawlak et al., 2004; Walker et al., 2002; Scribner et al., 2007; Roncal-Jimenez et al., 2011). Among humans, small scale clinical trials have short follow-ups (Thangaratinam et al., 2012; Zheng et al., 2015; Aeberli et al., 2011; de Ruyter et al., 2012; Malik and Hu, 2012; Malik et al., 2010), and most non-experimental evidence linking sugar to health studies contemporaneous effects and is observational. As chronic diseases do not manifest themselves until after the fourth decade of life, sufficient follow-up time and data on outcomes from cohorts experiencing contrasting early-life conditions in sugar-rich diet at random are needed; but rare to come by. In response, a meta-analysis, on which the most recent World Health Organization (WHO) guidelines for daily free sugar intake are based, concluded that stronger evidence on effects from excessive sugar intake is needed (WHO, 2015a; Kaiser et al., 2013). We overcome data and identification obstacles in providing new robust evidence on long-term health effects of early-life sugary diet here.

Second, we contribute to the more general literature on the effects of early nutrition on long-term well-being. Many of design-based observational studies focus on extreme caloric restrictions such as famine (Almond and Currie, 2011; Barker, 1998a; Almond et al., 2018; Roseboom et al., 2006; Ravelli et al., 2005; Barker, 1992). Other assess the exposure to broader early-life interventions that include a nutritional component such as Supplemental Nutrition Assistance Program or SNAP (Hoynes et al., 2016; Bailey et al., 2020; Maluccio et al., 2009; Stein et al., 2006), Carolina Abecedarian Project providing healthy snacks in a cognitively stimulating environment (Campbell et al., 2014), or nutritional supplementation (Stein et al., 2006). Most have found some long-term effects on metabolic health and economic outcomes,

However, we observe no difference in vegetable intake: instead, we find that the end of rationing is associated with a decrease intake of fruits, but an increase in the intake of sweets, suggesting substitutions between healthy and unhealthy sweet-tasting foods.

though exact mechanism was not identified. We extend this literature by providing new evidence on the long-term effects of pre- and postnatal exposure to sugar-rich diet specifically (Reid et al., 2016). Because we observe differences in adult outcomes despite no increase in total daily calorie intake, our findings also contribute to a long-standing debate on whether nutritional composition of diet - and not only calorie imbalance - influences health or economic outcomes (Gračner, 2021; Lustig, 2013; Basu et al., 2013; Taubes, 2007).

Third, much of prior work studying long-term effects of nutritional interventions discusses yet does not study potential pathways behind them. We provide new evidence on a potential mechanism - such as elevated sugar consumption across lifespan as preference for sweetness forms early - driving our results.

Finally, we contribute to the literature linking nutrition to economic well-being. Largely focused on short-term effects, some studies have found that higher calorie meals in schools improve learning through physical development (e.g., sight), cognition (e.g., concentration, memory), and behavior (e.g., hyperactivity, fatigue) (Sorhaindo and Feinstein, 2006), while others have found no such effects (McEwan, 2013). Evidence on diet quality - not quantity (calories) - is less mixed: poor diet quality in schools has been shown to lower test scores, largely because of more illness, missed days of school, and generally worse health (Anderson et al., 2018; Belot and James, 2011). Effects were greater at earlier ages (Lundborg et al., 2021). Such short-term adverse nutrition effects may compound over time (Heckman and Masterov, 2007; Cunha and Heckman, 2007), and can - along with direct adverse effects from diet-related chronic illness - lead to lower education attainment, selection into occupations with smaller returns, absenteeism, productivity, and accumulated wealth over lifespan (Cawley, 2004; Goettler et al., 2017; Trogdon et al., 2008; Finkelstein et al., 2010; Popkin et al., 2006; Hoynes et al., 2016; Bailey et al., 2020). Our study builds on and expands this literature further by estimating long-term effects of a particular diet (i.e., excessive sugar intake) early in life on economic outcomes more than five decades into adulthood.

Our findings also have implications for policy. First, our findings provide strong evidence of the positive and large long-term effects of compliance with WHO and USDA dietary guidelines related to limiting free sugar intake during pregnancy and early in life (Foster and Lunn, 2007a; Moynihan, 2016; Moynihan and Kelly, 2014). Second, our results support policies regulating sugar intake, such as taxes on sugar-heavy foods and drinks, and regulations on children food content and marketing.

The rest of the paper is organized as follows. In Section 2, we describe background on sugar and sweets rationing in the UK. In Sections 3 and 4, we outline our data and empirical strategy. Section 5 discusses results, and Section 6 describes sensitivity analyses. Section 7 concludes.

2 Sugar and Sweets Rationing in the UK

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

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

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

NSF data show that the diet under food rationing during this period was not extreme deprivation. In fact, it was nutritionally more balanced than during pre-war years and in many ways similar to today's WHO/USDA recommended dietary guidelines (see Table 5) (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 today's dietary guidelines (WHO, 2015a; HHS, 2015). Importantly, during our study period between 1950-1953, adults consumed on average about 40 grams of sugars per day accounting for about 6% of daily TEI (NFS, 2011). Today, 10 percent or less calories coming from sugar is strictly recommended, or 5 percent or less for long-term health benefits (Prynne et al., 1999; WHO, 2015a).

Young children's intake was also within today's daily recommended dietary allowance. An average 4-year old consumed 1445 calories per day compared to 1500 recommended today. About 13 percent of calories came from protein compared to 15 percent recommended, 40 percent from fat compared to between 25 and 35 percent recommended, and 47 percent from carbohydrates compared to 45 to 55 percent recommended. This diet was healthy enough so that children were not iron, vitamin A or C deficient (Foster and Lunn, 2007b), and amount of animal protein available for children and pregnant and nursing women was deemed adequate (Hollingsworth, 1983). Pregnant women, nursing mothers and children were given special green ration books with extra coupons and special allowances. Specifically, through the National Milk Scheme, implemented in 1940, pregnant women and nursing mothers were entitled to supplementary food rations, welfare, and priority foods such as vitamin supplements, fresh eggs, and milk, and had priority access to cod-liver oil, fruits, and vegetables. This supplied an additional 540 calories per day and the bulk of their daily requirement of calcium and vitamins.[6] Children and adolescents had similar welfare and priority foods entitlements to pregnant women and nursing mothers (Zweiniger-Bargielowska, 2000; Sultan, 2010).

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

The lifting of sugar and sweets rationing had a dramatic effect on the confectionery market – sales of sweets and chocolate increased by more than 150% within one year (Jackson, 1974). Among children (age 6-12), average consumption of sugars from sweets during rationing in the early 1950s was within recommended amounts - below 4oz (115g) per week. However, soon after the end of rationing, children's consumption of sugar and sweets more than doubled coupled

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

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

We find similar dietary patterns in NFS data. We show average daily consumption of sugar in the UK between 1950q1-1959q4 in Figure 2a, and intake of protein, fruits, vegetables, fats, and sugar for comparison over the same period in Figure 2b. We observe a sharp increase in the consumption of sugar and sweets by about 100% right after rationing ended (see Figure 2a), mapping into a corresponding increase in total calorie intake (see Figure A.1). Specifically, intake of sugars sharply increased from 10.2oz in 1953q1 to 16.08oz in 1954q1 and to nearly 20oz by 1954q3 per person per week. This contrasts with no sharp change in other foods or nutrients (e.g., protein, vegetables, fruits, fats, dairy etc.) during the same period as reflected in Figure 2b.

The sharp and quick response in the consumption of sugar and sweets, and not of other foods, soon after the rationing ended informs our research discontinuity design, which examines whether there are differences in outcomes among individuals born within only a few years of one another – specifically, those cohorts conceived or born just before the sugar and sweets rationing ended relative to those conceived and born after.

3 Data

3.1 The English Longitudinal Survey of Aging (ELSA)

ELSA is a bi-annual panel study of a representative of the population aged 50 and over in England. It spans a 16-year period between 2002-2018 (9 waves) and was designed as a sister study to the Health and Retirement Study (HRS) in the US. These data provide detailed information on economic, social, psychological, cognitive, health, biological, and genetic data, and data on health behaviors, such as physical activity, alcohol consumption, smoking as well as information on parental health, occupation, and education. The age distribution of survey participants is suited for our study as disability and chronic diseases start emerging frequently at this age (Kirkman et al., 2012).[7] Our analytic sample includes individuals born in the UK between 1950 and 1960, excluding those born in 1954 as some may or may not have been in-utero during rationing, and those born or educated outside the UK.

Data include measures of C-reactive protein (CRP) levels objectively measured from blood draws, used to determine chronic or low grade inflammation. Chronic inflammation is defined as an indicator variable for CRP levels larger than 3mg/l. Measures of CRP are available for

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

a subsample of participants who consented and were able to give blood in a clinical assessment.[8] Data also include information on objectively measured weight and height, from which we calculate one's body mass index (BMI) as kg/m^2. We define someone with obesity/severe obesity if their BMI is equal or higher than 30/35. Data also include self-reported information on whether chronic conditions were ever diagnosed by a physician. We create indicators for whether someone was diagnosed with diabetes, cardiovascular disease (i.e., elevated blood pressure, heart disease, or stroke), cancer, elevated cholesterol, or arthritis. We also create an aggregate measure for the total number of these chronic conditions reported by each participant (ranging from 0 to 5). Other conditions, such as asthma, cataracts, or lung disease are also reported and we use them to examine placebo effects, as they are unlikely related to free sugar intake early or later in life. We assume that all chronic conditions are absorbing states.

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

Importantly, ELSA also includes the "Life History Module" in Wave 3, which collects retrospective self-reported information for a subsample of individuals on health and socio-economic circumstances when they were 10 years old (Ward et al., 2009). These include information on whether they experienced financial hardship and whether their father's job was a high/medium skilled profession, household assets (number of books, bathrooms, rooms, or people in the residence), and parenting practices (e.g., mother was overprotective, understood my worries, was emotionally cold to me, etc.). We also use information on whether biological parents were ever diagnosed with diabetes as a proxy for one's genetic pre-disposition for diabetes and related disease.

Adults in our final analytic sample are between 50 and 65 years old; 41% percent are born after 1954. Males represent 41 percent of the sample, about a half of adults have at least some

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

post-secondary education. Among those born during(after) the rationing, 6.9(8.5) percent are diagnosed with diabetes, 38.9(36.2) with a cardiovascular condition, and about a quarter with arthritis and elevated cholesterol. About 10 percent have three or more chronic conditions.

3.2 The National Diet and Nutrition Survey

The UK National Diet and Nutrition Survey (NDNS) is a continuous, nationally-representative repeated cross-sectional survey that collects detailed quantitative information on diet of about 1500 individuals. It began in 2008 and is administered biannually. It is regarded as one of the most comprehensive nutrition surveys in Europe, and it is similar in its nature to the National Health and Nutrition Examination Survey (NHANES) in the United States (US). The NDNS provides high quality, nationally representative UK data on the types and quantities of more than 5,000 foods consumed by individuals. The dietary assessment method is based on the multiple-pass 24-hour dietary recall repeated on up to four non-consecutive days. It includes information on several socio-demographic characteristics, as well as birth quarter and place of birth (in or outside the UK). In addition to several food groups (e.g., fruits, vegetables, meats), these data also provide information on at the nutrient level (i.e., X grams of protein per day). Data on dietary intake were excluded for those born outside the UK, and when "the unusual amounts of foods consumed" was reported. To estimate intake at the nutrient level, we combine NDNS data with the latest analyses in the Food Standards Agency's nutrient databank.

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

Our final analytic sample consists of adults born in the UK before (1950-1953) and after (1954-1960) the rationing has ended. We exclude everyone born between October 1953 and June 1954, as they were born after rationing has ended, but could have been exposed to rationing at

some point while in-utero. Those born in July 1954 were likely the first cohort conceived after the end of sugar and sweets rationing in September 1953. On average, adults born during(after) the rationing consume about 1800 calories daily and about 48(51) grams of free sugars per day.

4 Empirical Approach

We employ a Regression Discontinuity Design (RDD) using ELSA as our main empirical approach and estimate the following specification:

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

where Y_{it} is the outcome variable of interest and $BirthYear_i$ is the running variable that fully determines an individual's exposure to treatment, and measures the distance between an individual's birth year and the cut-off (1954). Then, $BornPostRation_i$ is an indicator variable that takes on value 1 for individuals i who were either in-utero or born after the end of the rationing (e.g., those born between 1955 and 1960), and 0 otherwise (e.g., born between 1950 and 1953).

Each regression includes survey year fixed effects, θ_t, which capture potential differences in outcomes over time that are common across birth cohorts. We also include as controls (X_{it}): sex, age indicators, and parents' diabetes diagnoses. Parental diabetes status indicators are used as a proxy for genetic predisposition for chronic disease.

Age indicators are used to compare individuals within the same age between cohorts to control any mechanical age-driven discontinuities in adults' health, i.e., that the incidence of certain chronic illnesses may deteriorate with age. Such regressions require a common age support across cohorts - in our regression models using ELSA, adults are between 50 and 65 years old on both sides of the cut-off. We are able to control for age since we have an individual panel and therefore observe the same individual at different ages. We cluster standard errors at the individual level.

We assume that individuals born closest to the cut-off are the most exchangeable with respect to both measured and unmeasured confounders, and that exchangeability likely decreases with distance from the cut-off. We incorporate this by placing the greatest weight on observations closest to the cut-off using triangular kernel weighting. We estimate local regression models using a bandwidth of 4 years before and 5 years after the cut-off in 1954, with cohort trends being captured by a first-order polynomial of $BirthYear_i$ on each side of the cut-off. [9]

[9]We also estimate the effects using the optimal bandwidth for each outcome, produced by the algorithm

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

5 Results

We begin by investigating the extent to which cohorts born before and after the sweets and sugar rationing are balanced with respect to their observable characteristics at age 10. Table 1 shows that differences in means across several potential confounders including childhood SES, home environment, parental education, occupation, health, and parenting style are small and not statistically significantly different from zero. This suggests that individuals born before and after the end of sugar and sweets rationing began life under similar socioeconomic circumstances and their parents had similar health. While we do not include most of these variables as controls in our regression models, as they were only collected from a subsample of individuals, we do include parental diagnosis of diabetes because this information is available for about 85% of survey participants (we impute the other 15%). Exclusion of this control variable does not affect our findings.

5.1 Adult health outcomes

We first present estimates for two bio-markers of chronic disease that have been independently linked with both, excessive intake of sugar and metabolic chronic disease: (1) systemic low-grade chronic inflammation measured from C-reactive protein (CRP) in blood and (2) obesity, proxied with BMI in our data (Calder et al., 2011; Luft et al., 2013; Lontchi-Yimagou et al., 2013). They have both been objectively measured at the time of the survey, and are therefore uncorrelated to improved disease diagnostics over time.

Table 2, Cols. 1 and 2, show that the end of sugar and sweets rationing resulted in higher levels of C-reactive protein by 22.3% and in a higher prevalence of low-grade chronic inflammation (i.e., CRP$\geq$3mg/l) by 8.8pp or 33.8% on average. Figure 4 supports these results: it shows a discontinuous jump in chronic inflammation prevalence at the cut-off. Such results are supported by a growing body of work. Elevated sugar consumption has been repeatedly linked with systemic low-grade/chronic inflammation or elevated CRP levels (Calder et al., 2011). In

developed by Calonico et al.(2014), but the optimal bandwidth was very small – 2 years. Though effect sizes are comparable to alternative specifications, we are not powered to detect a significant effect with such a small bandwidth, so we do not use this approach as our main specification.

turn, independent of total calorie intake, elevated CRP levels have been shown affect insulin resistance, and have been increasingly identified as a mechanism for increased risk of chronic disease, such as diabetes, heart disease or arthritis (Grundy, 2003; Danesh et al., 2000; Luft et al., 2013; Lontchi-Yimagou et al., 2013).

Table 2, Col.3, shows that the end of sweets and sugar rationing increased severe obesity by 4.4 pp or 40%. However, our effect is imprecisely estimated at p=0.14. Without age indicators as controls, coefficient estimates remain nearly unchanged, but more precise (i.e., p<0.1). This may be because BMI is an imprecise measure of obesity, but there could be other reasons as well. It has been previously shown that obesity is not a primary cause, but rather a marker for the patho-physiology of chronic disease (e.g., insulin resistance), and that many with poor metabolic health are of healthy weight (Lustig, 2020). For instance, in our sample, nearly 40% of normal weight adults have some metabolic disease. Additionally, other obesity measures (e.g., waist circumference, body fatness) are considered better markers of metabolic health than BMI (Burkhauser and Cawley, 2008).[10]

We then examine whether the end of sugar and sweets rationing led to a higher prevalence of a poor metabolic health diagnosed by later adulthood. Figures 5 (a)-(c) show a discontinuous jump in the prevalence of diabetes, arthritis, and multiple chronic conditions defined as having 2 or more diet-related chronic conditions. Table 3 similarly shows that exposure to sugar-rich diet early in life increased adult prevalence in diagnosed diabetes (by 3.6pp or 52%), elevated cholesterol (by 7.4pp or 26.7%), and arthritis (by 11 or 44.7%). Cohorts born after the end of rationing also have 4pp or 10.5% higher prevalence in cardiovascular disease, however, the estimate is not statistically significant. We also find that the end of rationing worsened an overall metabolic health: because of this event, those born after the rationing experienced higher prevalence in at least two or three of diet-related chronic diseases by about 10.7pp (or 36.5%) or 6.1pp (or 56%), respectively.

5.2 Education and Economic outcomes

We also find that the end of the sweets and sugar rationing affected adults' human capital and economic well-being. In Figure 5 (d)-(f), we observe discontinuous jumps at the cut-off for outcomes, such as attending any higher education, having skilled occupation and top quartile wealth. Table 4 shows that on average, the lifting of the rationing caused adults born post-1954 to be 9.2pp or 18.5% less likely to have at least some higher education compared to adults

[10]While waist circumference measures are available in ELSA, they are not available in enough survey waves to allow its use in this study.

born during rationing. Though both cohorts are equally likely to be employed, the end of the rationing decreased the probability of working in a high-skilled job (by 9.8pp or 16.6%). Consistently, the end of rationing made it less likely for adults born after 1954 to accumulate wealth above the median (by 9pp or 18%) or in the top quartile (by 6.8pp or 28%).

5.3 Diet

Finally, we use the NDNS data to explore whether elevated sugar consumption across lifespan was a potential mechanism behind the long-term health and economic effects of early sugar-rich diet. Outcomes of interest are either continuous log-transformed measures of total calories per day, grams of fats, proteins, and carbohydrates, which are further divided into daily intake (in grams) of fiber, intrinsic and free sugars. Binary outcomes of interest are indicators whether individuals ate any sweets (i.e., sugar, preserves, sweet spreads, chocolate, and sugar confectionery), fruits or vegetables on a given day, and whether sugar intake was above the 5% or 10% percent of TEI recommended limit per day.

For these analyses, we employ the Ordinary Least Squares (OLS) regression models, mainly because the NDNS sample size was too small to estimate RDD models above.[11] We follow the RDD setup in that we regress each outcome on an indicator variable for whether survey participant was born in and after the third quarter of 1954 (1954q3).[12] In all our regressions, we also control for cohort-specific linear trend and its interaction with an indicator for whether one was born post-rationing. As controls, we include calendar month and day of the week, and survey year indicators to adjust for secular trends and seasonality, or for an uneven distribution of years before and after the cut-off. We also control for participant's sex, height, and race. We do not include age indicators in these regressions: food preferences are unlikely to change sharply at an older age, so the concern of non-linear changes in diet is much smaller than for health outcomes. However, we control for age indicators in our sensitivity analyses: our point estimates remain unchanged with a slight loss in precision. We cluster standard errors at the individual level.

Figure 3 visually describes diet outcomes between cohorts of today's older adults. Panels a-c show a sharp increase at the cut-off for share of people exceeding the recommended levels of free sugar intake at 5% of TEI, daily intake of free sugar and for consuming any sweets. Similarly, a discontinuous decrease at the cut-off is observed for intake of intrinsic sugars and any

[11] We re-estimated OLS regression models using ELSA for health and economic outcomes in a similar fashion and results are very similar to our results estimated using RDD.

[12] Unlike ELSA, NDNS included information on quarter of birth. Individuals born in 1954q3 or after were not exposed to sugar rationing neither in-utero nor later in life.

fruits. In contrast, we observe a discontinuous change in neither total calorie intake nor other macronutrients, such as protein, carbohydrates, proteins or fats. These descriptive analyses are consistent with our regression results, which similarly show that the end of rationing did not affect adults' total daily calorie intake, but their diet composition in free sugar intake. Specifically, Table 6, panel A, shows that the lifting of sugar rationing increased the share of adults exceeding the recommended free sugar intake amount set at 5% TEI by 10.2pp or by 12%, and increased free sugar intake by 22.4% or about 11 grams of free sugar pay day. This additional free sugar amount is comparable to the grams of free sugars found in about 3 Oreo cookies or in half of an 8oz soda can. Though no difference is observed in any vegetable intake (Panel B), the end of rationing resulted in adults being more likely to consume some sweets (by 8pp or 13.3%), no fruits (by 14.2pp or 21.2%), and less intrinsic sugars (by 18.4%) or fiber (by 9.0%); corresponding to about a half of an apple. We find no effect on the daily intake of total calories, or on the daily intake of macronutrients, such as fats, proteins and carbohydrates (Panel C).

6 Sensitivity Analyses

We carry out several analyses designed to rule out alternative explanations of the estimated effects. First, as reported above, we assessed whether the cohorts conceived and born right before (1950-1953) and those conceived and born after the rationing ended (1955-1960) are similar in terms of family background and enviornment early in life - except in their exposure to sugar and sweets. We checked whether individuals born before and after 1954 are balanced with respect to characteristics such as childhood SES and home environment, parental education, occupation, health, and parenting style. Table 1 shows that differences in means across these potential confounders are small and not statistically significantly different from zero. This suggests that our results are not driven by unobserved differences in family background and environment early in life.

Second, we also test for discontinuities in the outcomes of interest at cutoffs at different times than the year rationing ended both before and after 1954 (see Table B.3). We consider 2 different cutoffs: (1) 1950-51 compared to 1952-53 and (2) 1955-56 compared to 1957-60. Table B.3 shows that cohort differences in health and economics outcomes are small and not statistically significantly different at these other cut-off years across almost all of the outcomes.[13]

[13]This falsification test helps to address a specific concern, related to a potential discontinuity in the relationship between outcomes and birth year due to an educational 1972 reform, which increased the minimum age at which students could drop out of school from 15 to 16 years. The reform affected only students born on or after

These results are consistent with end of rationing driving the observed differences in outcomes rather than some other policy or event either before or after the rationing cutoff.

Third, there is the possibility that improved disease diagnostics over time is driving our results as younger cohorts would then be more likely to be diagnosed at an earlier age than older cohorts. We address this in a number of ways. First, we conduct a number of falsification tests by repeating the main analyses for diseases whose incidence should be unrelated to diet in general and sugar consumption but for which diagnostics have improved over the same period (e.g. lung disease, asthma, cataract). Exposure to rationing should not have affected these placebo outcomes but improved diagnostics might have. The analysis shows that there are no statistically significant difference in such diseases between cohorts (see Table B.2). Additionally, if improved diagnostic trends were driving our results, we would expect a positive significant effect for an 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 chronic inflammation. In fact, we show the opposite in Tables 2 and 3.

Fourth, within survey years, the cohort born after the end of rationing is younger than the cohort born right before it, which might attenuate the estimates. We address this concern by age indicators in the regressions. We are able to do so because the panel nature of our data allows us to observe the same individual at multiple times.

Fifth, we address the concern of positive selective fertility during rationing by controlling for the annual number of live births in new set of regressions (see eTable B.8); our conclusions remain unchanged.

Finally, we provide evidence that our results are not sensitive to the choice of bandwidth, or to the order of polynomial chosen (see Tables B.5, B.4, B.7 and B.6).

There are a few limitations to our study that we cannot fully address. First, while we observe today's older adults' diet, born before or after the rationing ends, we do not observe differences in their diet composition during childhood around the time when the sugar and sweets rationing ended. We address this by providing evidence of sharp increases in consumption of sugar and sweets (in contrast to other foods and vitamins) in late 1953 for everyone (see Figure 2), and cite published studies showing associations between significant increase in intake of sweets and deteriorating dental health among children after the rationing has ended (Jackson, 1974; Zweiniger-Bargielowska, 2000; Jamel et al., 2004; Toverud, 1949; Hollingsworth, 1974, 1983; Prynne et al., 1999).

September 1, 1957. While we do observe a small discontinuity in skilled employment for those born after 1957 vs those born before (but after 1955), the coefficient is negative, which is the wrong sign.

Second, cohort difference in added sugars at younger ages (e.g., adolescence or younger adulthood) might have been larger than what we observe here. It is possible that older adults reduced sugar intake following some disease diagnosis. This would imply that our estimates are a lower bound.

Finally, sugar and sweets may not have been available immediately and evenly across the country (e.g., in rural areas) soon after the rationing has ended. We address this concern in part by excluding birth cohorts in years around the cut-off (e.g., 1955) but our conclusions remain largely unchanged.

7 Conclusion

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

These findings are concerning, given the increased attention food companies are paying to infants and toddlers. In 2018, sweetened children's drink sales totalled $1.4 billion, exceeding unsweetened children's juices by nearly 70% (Fleming-Milici et al., 2022). Studies have found that more than 50% of commercially prepared baby foods feature more than 20% (and some even 80%) of calories coming from sugar (Walker and Goran, 2015). A good example of this is a relatively new food for young children called "toddler" milk, consisting primarily of powdered milk and added sugars such as corn syrup, sucrose, cane juice or other caloric sweeteners (Romo-Palafox et al., 2020; Palafox and Harris, 2017). Despite being considered inadequate for young children (Palafox and Harris, 2017), new heavily sweetened drinks and foods are being introduced rapidly and advertised heavily. There were four times more product launches in the baby and toddler food aisle in 2018 than in 2005 (Romo-Palafox and Harris, 2021), and $56.5 million was spent on advertising in 2015 alone (Harris et al., 2016; Harris and Pomeranz, 2020).

A major issue is also the often misleading marketing claims that position sugar-rich infant and toddler foods as beneficial for their nutrition and development (Duffy et al., 2021) (Dallacker et al., 2018; Cattaneo et al., 2015; Fleming-Milici et al., 2022).

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

Our findings are also in line with the literature studying the health impact of higher prices of sugar-sweetened beverages (SSBs) or sugar-rich foods. Though long-term implications of such policies are unknown as they have only been implemented recently, (Gračner et al., 2022) shows that a ten percent SSBs tax leads to about 0.4kg weight loss among heavier teenage girls (corresponding to a daily 7.8 calorie reduction) already within two years of a tax. Similarly, (Gračner, 2021) shows that higher prices of foods rich in sugar (and not in other nutrient) lead to less obesity and diabetes. Several modeling studies predict larger improvements in obesity and metabolic health over time; with largest disease reductions expected for populations exposed to the SSBs tax at a younger age (Sánchez-Romero et al., 2016; Basto-Abreu et al., 2019; Dubois et al., 2020). For instance, a 20% SSBs tax was projected to reduce prevalence of obesity between 1.5% and 10%, and of diabetes cases by up to 3.4% annually over a decade (Mytton et al., 2014; Wang et al., 2012; Smith, 2010). Similarly, a 20% reduction in added sugar intake (about 14 grams of free sugars at the current mean intake among US adults (Ervin and Ogden, 2013)) was projected to reduce incidence of diabetes by about 20 cases per 100,000 people over two decades (Vreman et al., 2017; Basu et al., 2014).

Finally, as diets during post-war rationing in our setting were similar to dietary guidelines

for free sugar today, our results may serve as one of the first references on the long-term effect of compliance to dietary guidelines early in life. Today's dietary guidelines on added sugar intake are based on short-term clinical trials and reviews of data largely coming from population-based observational studies on the associations between intake of free sugars and dental caries (55 out of 68 in the WHO report), and not unhealthy weight or related chronic-disease long-term (Foster and Lunn, 2007a; Moynihan, 2016; Moynihan and Kelly, 2014). In fact, most are limited to SSBs intake alone - even though added sugars in other foods in the US represent nearly 40% of daily calories and similarly large shares have been found in the UK (Yang et al., 2014; Griffith et al., 2020). There are currently no strict guidelines for daily recommended limits for added sugar intake for pregnant women. Only the most recent 2015-2020 dietary guidelines have been updated to recommending zero free sugar for children under the age of two.

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

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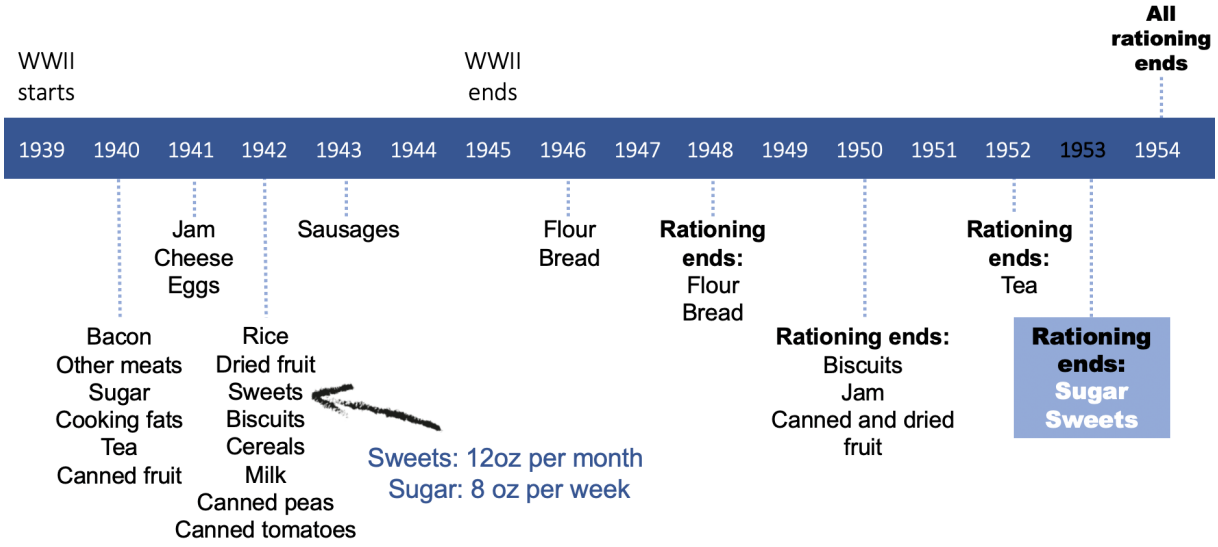
Figure 1: Food rationing timeline in the UK: 1939-1954

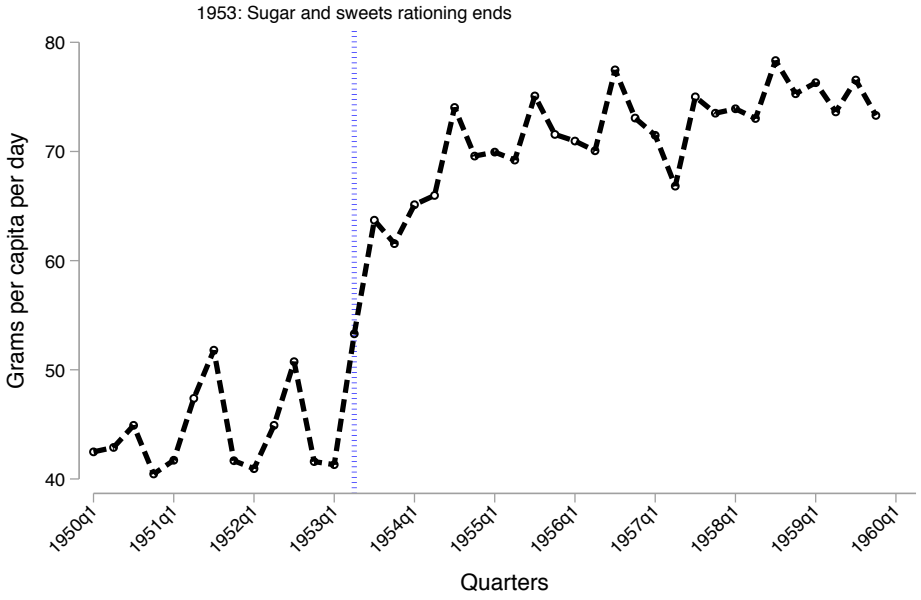


Note: Author's presentation of years during which different foods went off-ration.

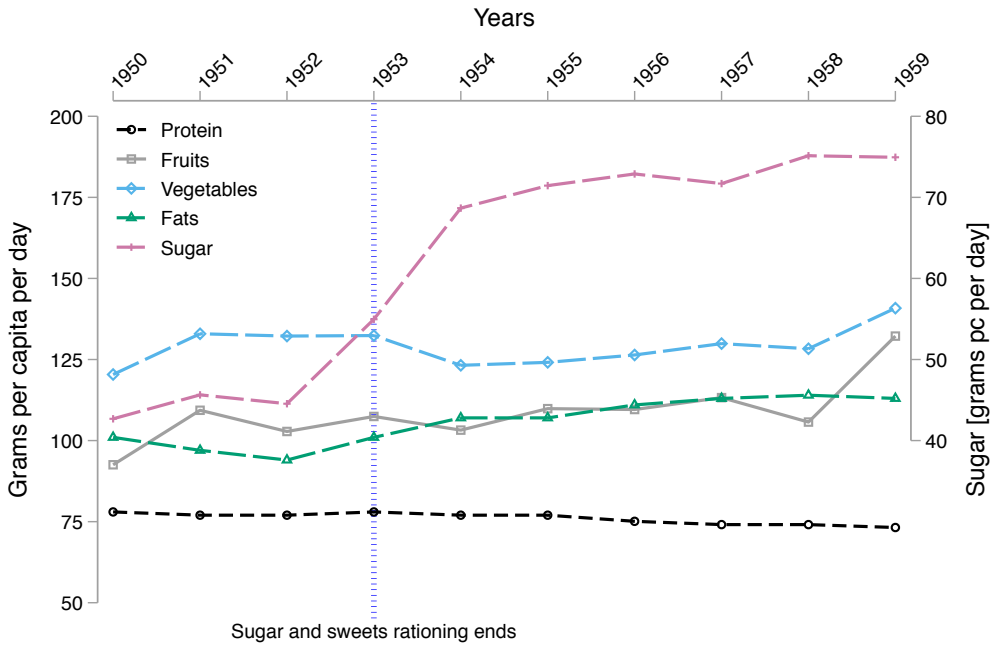
8 Results: Figures

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

(a) Daily per capita intake of sugar

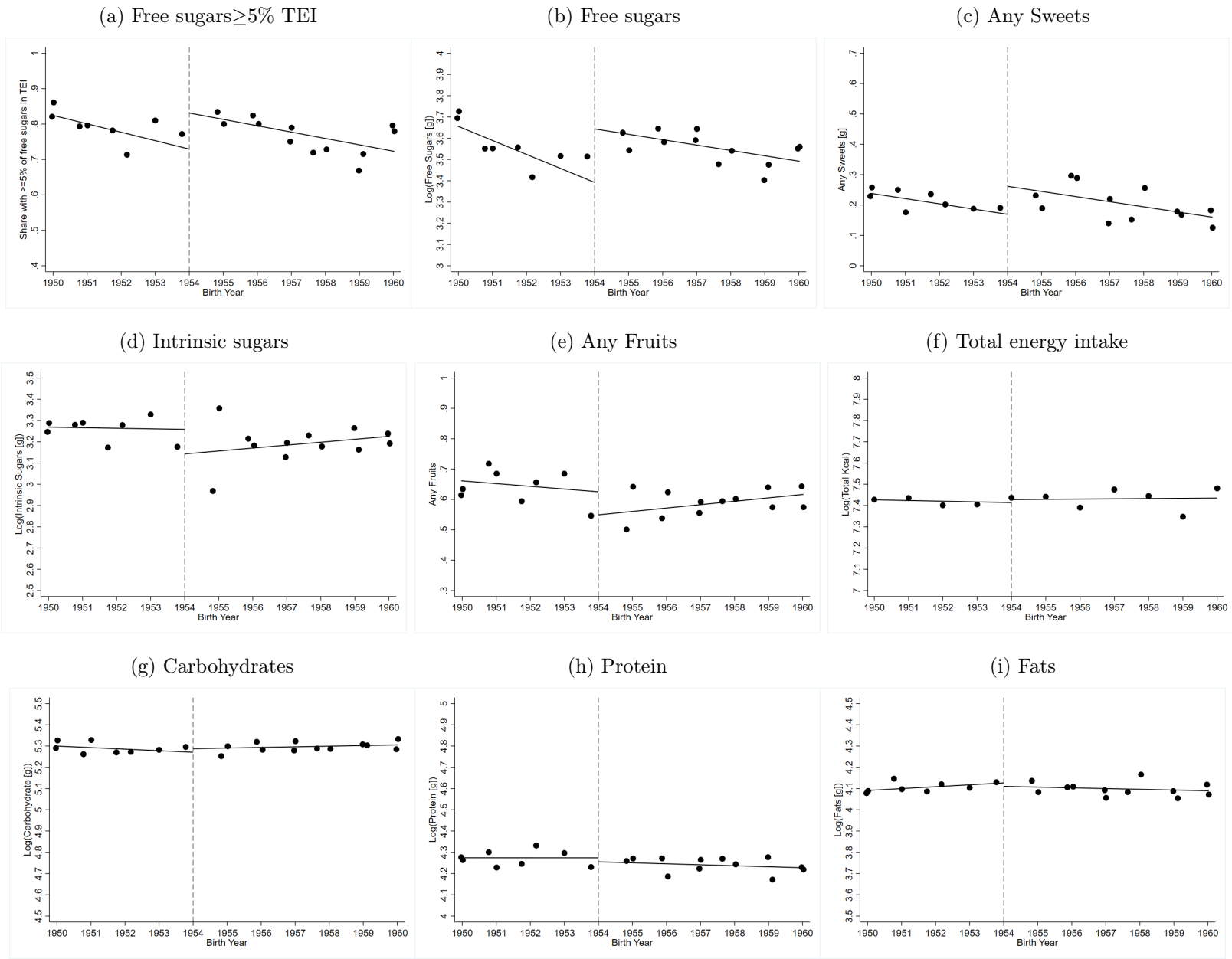


(b) Daily per capita intake of sugar and other foods



Note: Authors' calculations based on data published by the National Food Survey 1950-1960.

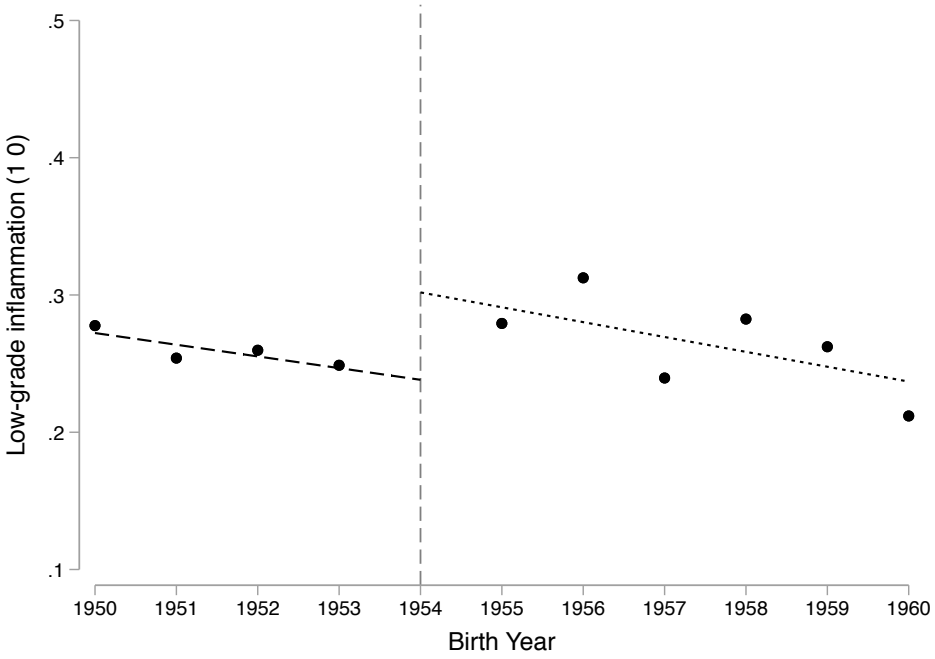
Figure 3: Current diet among today's older adults born between 1950-1960



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

Figure 4: Low-grade inflammation (CRP>3) across birth cohorts

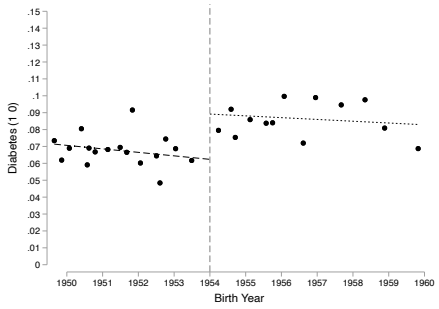
(a)



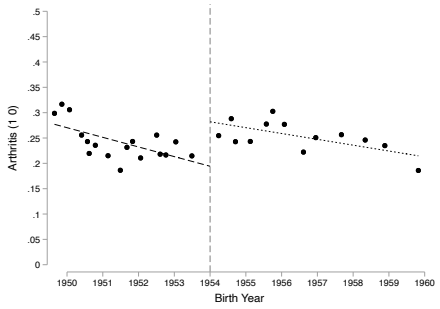
Note: Cohort differences in probability of experiencing low-grade inflammation as measured by CRP. Authors' calculations based on ELSA (1998-2019).

Figure 5: Current health and economic outcomes across birth cohorts

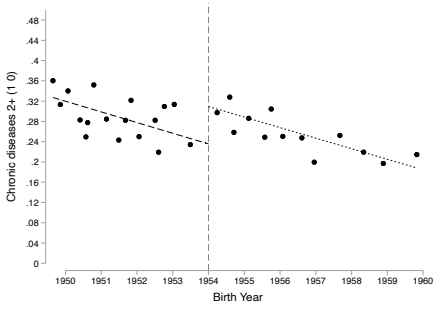
(a) Diabetes (1 0)



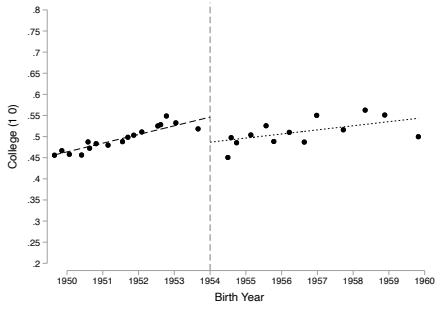
(b) Arthritis (1 0)



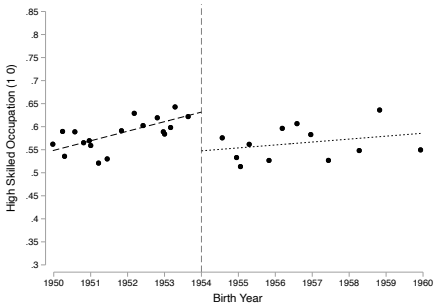
(c) Chronic diseases 2+ (1 0)



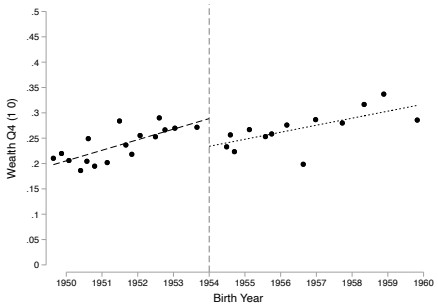
(d) Any higher education (1 0)



(e) High skilled occupation (1 0)



(f) Wealth Q4 (1 0)



Note: Cohort difference in probability of being diagnosed with diet-related disease and in selected economic outcomes. Adjusted for age indicators. Data: The English Longitudinal Survey of Aging (2002-2019). Chronic 2+ is an indicator for whether one is diagnosed with at least two or three of: hypertension, stroke, heart disease, diabetes, cancer, arthritis, high cholesterol.

9 Results: Tables

Table 1: Cohort Differences in Childhood and Parent Characteristics

Panel A: Childhood Characteristics	Born 1950-1953	Born 1955-1960	Diff	RW p-value
Mother had diabetes	0.09	0.10	0.014	0.99
	(0.29)	(0.30)		
Father had diabetes	0.09	0.10	0.009	1.00
	(0.29)	(0.30)		
Father's job at age 10: High-Med skill	0.90	0.74	-0.167	0.00
	(0.30)	(0.44)		
SES score at age 10 (0-5)	3.28	3.22	-0.060	0.98
	(1.11)	(1.17)		
Had bath in-house at age 10	0.82	0.81	-0.005	1.00
	(0.39)	(0.39)		
Had toilet in-house at age 10	0.76	0.77	0.004	1.00
	(0.43)	(0.42)		
No or few books at age 10	0.18	0.17	-0.009	1.00
	(0.39)	(0.38)		
N of rooms at age 10	2.98	3.05	0.078	0.43
	(0.70)	(0.68)		
N of people in residence at age 10	5.02	5.06	0.035	1.00
	(1.50)	(1.47)		
Mother let me do the things I liked doing	0.63	0.60	-0.031	0.99
	(0.48)	(0.49)		
Mother was emotionally cold to me	0.13	0.13	0.006	1.00
	(0.33)	(0.34)		
Mother understood my worries	0.57	0.51	-0.061	0.62
	(0.50)	(0.50)		
Mother liked me to make my own decisions	0.62	0.61	-0.012	1.00
	(0.49)	(0.49)		
Mother was overprotective	0.15	0.13	-0.019	0.99
	(0.36)	(0.34)		
Observations	929	377		

Note. Childhood variables are measured only in ELSA wave 3. Parental diabetes is measured in all waves of ELSA. The Diff column is the coefficient of a simple regression of birth cohort on the variable. The last column is the Romano-Wolf stepdown p-value for multiple hypothesis testing. Data: The English Longitudinal Survey of Aging (2002-2019).

Table 2: RDD Estimate of Lifted Rationing on Chronic Inflammation

		Log(CRP)	Low-grade inflammation ($CRP > 3$)	Severe Obesity ($BMI \geq 35$)
		(1)	(2)	(3)
Born after rationing	β	0.223**	0.088**	0.044
	SE	(0.113)	(0.041)	(0.030)
	Single p-val	0.049	0.035	0.140
N		3304	3304	4868
Y(=0) Mean		3.08	0.26	0.11

Note. Robust standard errors in parentheses, clustered at the individual level. Adults born in 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. C-reactive protein levels (CRP) (ml/l) are measured by a nurse with a blood draw. Low-grade inflammation is defined as CRP>3. Each model is adjusted for sex, age, survey participant and survey year indicators. Results for severe obesity are significant at p=0.065 when age indicators are removed for precision and controlled for linearly. * p<0.1, ** p<0.05, *** p<0.01.

Table 3: RDD Estimates of the Effect of Lifted Rationing on Metabolic Health Outcomes

DV (1 0)		Diabetes	Cholesterol	Arthritis	Cardiovascular	Chronic 2+	Chronic 3+
		(1)	(2)	(3)	(4)	(5)	(6)
Born after rationing	β	0.036*	0.074**	0.110***	0.041	0.107***	0.061***
	SE	(0.020)	(0.034)	(0.033)	(0.038)	(0.033)	(0.022)
	Single p-val	0.063	0.031	0.001	0.291	0.001	0.006
	RW p-val	0.000	0.000	0.000	0.012	0.000	0.000
N		13792	10138	13792	13792	13792	13792
Y(=0) Mean		0.069	0.277	0.246	0.389	0.293	0.109

Note. Robust standard errors in parentheses, clustered at the individual level. Single p-values are unadjusted, and RW P-values are adjusted for multiple hypothesis testing using the Romano & Wolf correction. Adults born in 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. All conditions are self-reported ("Have you ever been diagnosed by a physician...?") Cardiovascular is an indicator if diagnosed with at least one of these conditions: hypertension, stroke, or heart disease. Chronic 2+ and chronic 3+ are indicators for at least two or three of: hypertension, stroke, heart disease, diabetes, cancer, arthritis, high cholesterol (vs none). Each model is adjusted for sex, age indicators, mother and father type 2 diabetes, and for imputation of mother's or father's diabetes (13% and 14% respectively). * p<0.1, ** p<0.05, *** p<0.01.

Table 4: RDD Estimates of the Effect of Lifted Rationing on Economic Outcomes

DV: 1 0		Any Higher Education (1)	Employed (part/full) (2)	Skilled Job (3)	>Median Wealth (4)	Wealth Q4 (5)
Born after rationing	β	-0.092**	-0.018	-0.098**	-0.090**	-0.068**
	SE	(0.041)	(0.030)	(0.040)	(0.037)	(0.032)
	Single p-val	0.025	0.558	0.014	0.015	0.033
	RW p-val	0.000	0.219	0.000	0.000	0.000
N		13632	13700	11205	13537	13537
Y(=0) Mean		0.497	0.650	0.589	0.500	0.242

Note. Robust standard errors in parentheses, clustered at the individual level. Single p-values are unadjusted, and RW P-values are adjusted for multiple hypothesis testing using the Romano & Wolf correction. Adults born in 1954 are excluded. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Any higher education is an indicator for if respondent attended at least some post-secondary schooling. Skilled job is conditional on current employment, and was classified using the UK Standard Industrial Classification 8-code system and is compared to low-skilled occupations. Continuous wealth in GBP was categorized into quartiles over all survey waves. Each model is adjusted for sex, age indicators, both parents' type 2 diabetes status, and for imputation of mother's or father's diabetes (13% and 14% respectively). * p<0.1, ** p<0.05, *** p<0.01.

Table 5: Diet under rationing (1950-1953) and today's recommendations on healthy diet

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

Source. Department of Environment, Food and Rural Affairs, National food survey. 2011; Dietary guidelines 2015-2020 (HHS, 2015).

Table 6: The Effect of Lifting Rationing on Diet

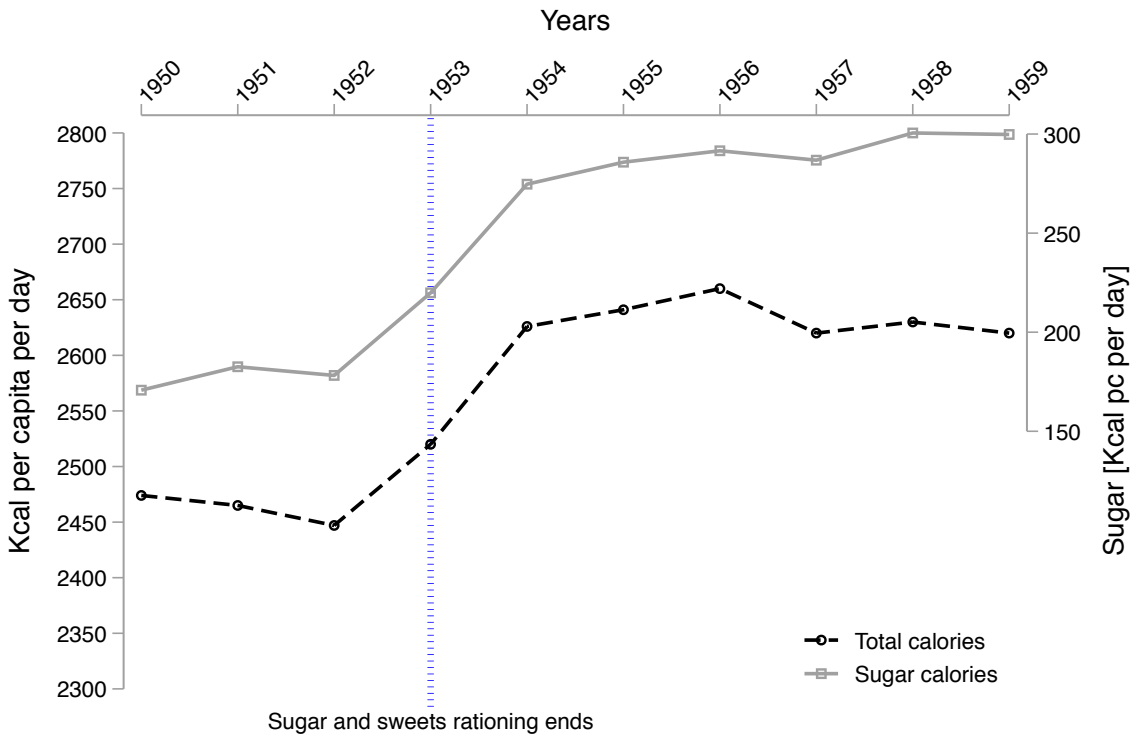
Panel A:		Sugars			
		Free sugars≥5% TEI (1)	Log(Free Sugars) (2)	Log(Intrinsic Sugars) (3)	Log(Fiber) (4)
Born after rationing	β	0.102**	0.224**	-0.185**	-0.090**
	SE	(0.044)	(0.096)	(0.079)	(0.041)
	Single p-val	0.020	0.020	0.019	0.028
	RW p-val	0.001	0.001	0.000	0.002
N		2,853	2,853	2,853	2,853
Y(=0)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

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

10 Appendix

A Figures

Figure A.1: Total calorie and sugar calorie daily intake per capita (1950-1960)



Note: Authors' calculations based on data published by the National Food Survey 1950-1960. We assume that 1 gram of sugar equals 4 Kcal.

B Tables

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

	Born 1950-1953	Born 1955-1960
Age (years)	57.33	56.51
	(4.055)	(3.763)
Male	0.456	0.370
	(0.498)	(0.483)
Diabetes	0.0690	0.0848
	(0.253)	(0.279)
Cholesterol	0.277	0.214
	(0.448)	(0.410)
Arthritis	0.246	0.247
	(0.431)	(0.432)
Cardiovascular disease	0.389	0.362
	(0.488)	(0.481)
Chronic Disease: 2+	0.293	0.251
	(0.455)	(0.433)
Chronic Disease: 3+	0.109	0.0956
	(0.311)	(0.294)
At least some college	0.497	0.508
	(0.500)	(0.500)
Employed	0.650	0.681
	(0.477)	(0.466)
Skilled job	0.589	0.561
	(0.492)	(0.496)
Above median wealth	0.500	0.498
	(0.500)	(0.500)
Wealth Q4	0.242	0.260
	(0.428)	(0.439)
Observations	8120	5672

Data: The English Longitudinal Survey of Aging (2002-2019).
Means and standard deviations in parentheses.

Table B.2: RDD Estimates of the Effect of Lifting Rationing on Placebo Health Outcomes

DV: 1 0		Lung disease (1)	Asthma (2)	Cataracts (3)
Born after rationing	β	0.006	0.017	0.002
	SE	(0.015)	(0.029)	(0.012)
	Single p-val	0.670	0.559	0.875
	RW p-val	0.645	0.511	0.781
N		13792	13792	13792
Y(=0) Mean		0.038	0.129	0.048

Table B.3: Placebo cutoffs for key health and economic outcomes

DV: 1 0	Diabetes (1)	Chronic 2+ (2)	Chronic 3+ (3)	College (4)	Skilled job (5)	>Median wealth (6)
Panel A: 1950-1953 vs. 1955-1960 (Rationing cut off)						
Born after 1954	0.036	0.107	0.061	-0.092	-0.098	-0.090
	(0.020)	(0.033)	(0.022)	(0.041)	(0.040)	(0.037)
N	13792	13792	13792	13632	11205	13537
Y(=0) Mean	0.069	0.293	0.109	0.497	0.589	0.500
Panel B: 1950-1951 vs. 1952-1953						
Born after 1951	-0.004	0.015	0.024	0.004	0.089	0.057
	(0.027)	(0.047)	(0.031)	(0.063)	(0.062)	(0.054)
N	8120	8120	8120	7990	6361	7958
Y(=0) Mean	0.054	0.248	0.081	0.460	0.556	0.401
Panel C: 1955-1956 vs. 1957-1960						
Born after 1956	0.001	0.001	-0.012	-0.012	-0.132*	-0.008
	(0.039)	(0.056)	(0.040)	(0.070)	(0.070)	(0.062)
N	5672	5672	5672	5642	4844	5579
Y(=0) Mean	0.073	0.270	0.105	0.486	0.534	0.438

Note. Robust standard errors in parentheses, clustered at the individual level. Not adjusted for multiple hypothesis testing. Sample includes individuals who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Panel C: RDD cut off is 1957, corresponding to the educational reform in the UK. Cardiovascular is an indicator for hypertension, stroke, or heart disease compared to diagnosis with none of these diseases. Chronic 2+ and chronic 3+ are indicators for at least two or three of: hypertension, stroke, heart disease, diabetes, cancer, arthritis, high cholesterol, compared to diagnosis with none of these diseases. College is an indicator for if respondent attended at least some college or higher education, compared to less than college education. Skilled job is conditional on current employment, and was classified using the UK Standard Industrial Classification 8-code system and is compared to low-skilled occupations. Each model is adjusted for sex, age indicators, both parents' type 2 diabetes status, and for imputation of mother's or father's diabetes (13% and 14% respectively). * p<0.1, ** p<0.05, *** p<0.01.

Table B.4: Vary RDD Bandwidth: RDD estimates of the effect of lifting rationing on health

DV: 1 0	Diabetes (1)	Cholesterol (2)	Arthritis (3)	Cardiovascular (4)	Chronic 2+ (5)	Chronic 3+ (6)
Panel A: left 3, right 3 year bandwidth						
Born after rationing	0.038	0.063	0.046	0.030	0.086	0.045
	(0.027)	(0.049)	(0.046)	(0.054)	(0.047)	(0.031)
Panel B: left 4, right 4 year bandwidth						
Born after rationing	0.033	0.073	0.090*	0.043	0.103**	0.057*
	(0.023)	(0.040)	(0.038)	(0.045)	(0.039)	(0.026)
Panel C: left 4, right 5 year bandwidth						
Born after rationing	0.033	0.071	0.089*	0.038	0.101**	0.057*
	(0.021)	(0.037)	(0.036)	(0.042)	(0.036)	(0.024)

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

Table B.5: Vary RDD Bandwidth: RDD estimates of the effect of lifting rationing on economic outcomes

DV: 1 0	Any Higher Education (1)	Employed (part/full) (2)	Skilled Job (3)	>Median Wealth (4)	Wealth Q4 (5)
Panel A: left 3, right 3 year bandwidth					
Born after rationing	-0.120*	-0.023	-0.120*	-0.092	-0.051
	(0.058)	(0.042)	(0.056)	(0.052)	(0.045)
Panel B: left 4, right 4 year bandwidth					
Born after rationing	-0.112*	-0.022	-0.113*	-0.096*	-0.059
	(0.048)	(0.035)	(0.047)	(0.043)	(0.037)
Panel C: left 4, right 5 year bandwidth					
Born after rationing	-0.108*	-0.025	-0.108*	-0.099*	-0.066
	(0.045)	(0.033)	(0.044)	(0.040)	(0.035)

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

Table B.6: Vary Kernel: RDD estimates of the effect of lifting rationing on health

DV: 1 0	Diabetes (1)	Cholesterol (2)	Arthritis (3)	Cardiovascular (4)	Chronic 2+ (5)	Chronic 3+ (6)
Panel A: Triangular kernel, first order polynomial (main specification)						
Born after rationing	0.036*	0.074**	0.110***	0.041	0.107***	0.061***
	(0.020)	(0.034)	(0.033)	(0.038)	(0.033)	(0.022)
Panel B: Uniform kernel, first order polynomial						
Born after rationing	0.038*	0.076**	0.119***	0.042	0.110***	0.062***
	(0.019)	(0.034)	(0.032)	(0.038)	(0.032)	(0.022)
Panel C: Epanechnikov kernel, first order polynomial						
Born after rationing	0.037*	0.076**	0.115***	0.042	0.109***	0.062***
	(0.019)	(0.034)	(0.033)	(0.038)	(0.033)	(0.022)

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

Table B.7: Vary Kernel: RDD estimates of the effect of lifting rationing on economic outcomes

DV: 1 0	Any Higher Education (1)	Employed (part/full) (2)	Skilled Job (3)	>Median Wealth (4)	Wealth Q4 (5)
Panel A: Triangular kernel, first order polynomial (main specification)					
Born after rationing	-0.092**	-0.018	-0.098**	-0.090**	-0.068**
	(0.041)	(0.030)	(0.040)	(0.037)	(0.032)
Panel B: Uniform kernel, first order polynomial					
Born after rationing	-0.085**	-0.015	-0.094**	-0.085**	-0.068**
	(0.041)	(0.030)	(0.039)	(0.036)	(0.031)
Panel C: Epanechnikov kernel, first order polynomial					
Born after rationing	-0.089**	-0.017	-0.096**	-0.088**	-0.068**
	(0.041)	(0.030)	(0.040)	(0.037)	(0.032)

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

Table B.8: RDD Estimates of the Effect of Lifted Rationing on Health and Economic Outcomes

DV (1 0)	Diabetes	Cholesterol	Arthritis	Cardiovascular	Chronic 2+	Chronic 3+
	(1)	(2)	(3)	(4)	(5)	(6)
Born after rationing	0.035*	0.089***	0.172***	0.077*	0.152***	0.096***
	(0.020)	(0.034)	(0.034)	(0.039)	(0.034)	(0.023)
N	13423	10138	13423	13423	13423	13423
Y(=0) Mean	0.070	0.277	0.248	0.392	0.296	0.110
DV: 1 0	Any Higher Education	Employed (part/full)	Skilled Job	>Median Wealth	Wealth Q4	
	(1)	(2)	(3)	(4)	(5)	
Born after rationing	-0.097**	-0.038	-0.109***	-0.106***	-0.096***	
	(0.042)	(0.031)	(0.041)	(0.038)	(0.033)	
N	13268	13340	10898	13170	13170	
Y(=0) Mean	0.495	0.653	0.587	0.505	0.247	

Notes: Robust standard errors in parentheses, clustered at the individual level. Single p-values are unadjusted. Sample includes individuals born between 1950-1960 who are 50-64 years old and surveyed in ELSA at least once between 2002-2019. Each model is adjusted for sex, age indicators, both parents' type 2 diabetes status, and for imputation of mother's or father's diabetes (13% and 14% respectively), and the number of live births each year. * p<0.1, ** p<0.05, *** p<0.01.