METABOLISM

Exposure to sugar rationing in the first 1000 days of life protected against chronic disease

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We examined the impact of exposure to sugar restrictions within 1000 days after conception on type 2 diabetes and hypertension, leveraging quasi-experimental variation from the end of the United Kingdom's sugar rationing in September 1953. Rationing restricted sugar intake to levels within current dietary guidelines, and consumption nearly doubled immediately after rationing ended. Using an event study design with UK Biobank data comparing adults conceived just before or after rationing ended, we found that early-life rationing reduced type 2 diabetes and hypertension risk by about 35 and 20% and delayed disease onset by 4 and 2 years, respectively. Protection was evident with in utero exposure and increased with postnatal sugar restriction, especially after 6 months, when eating of solid foods likely began. In utero sugar rationing alone accounted for about one-third of the risk reduction.

The first 1000 days after conception—from gestation until age 2 (early life)—is a critical window during which poor diet can negatively affect adult health (*1*, *2*). Current dietary guidelines recommend zero added sugars during this period (*3*), but in the United States, many children are exposed to excessive sugar through maternal diet in utero and while breastfeeding and then, directly, by consuming infant formula and solids (*4*). Pregnant and lactating women consume, on average, more than triple the recommended amount of added sugar, surpassing 80 g per day (*5*), and most infants and toddlers consume sweetened foods and beverages daily (*6*).

We estimated how exposure to added sugar restrictions in the first 1000 days after conception affects the onset of type 2 diabetes mellitus (T2DM) and hypertension decades later. Although ample causal evidence from rodent experiments demonstrated lasting adverse effects of excessive sugar intake in early life (*7*–*9*), rigorous evidence from humans is limited at best (*10*, *11*). For humans, previous research on dietary shocks early in life was primarily focused on extreme in utero caloric restrictions such as famine (*12*) or on changes in risk factors for T2DM and hypertension such as maternal gestational weight gain (*13*) and hyperglycemia (*14*). Thus, even though there is broad agreement on the correlations between excessive added sugar and metabolic dysfunction generally (*15*–*18*), the lack of cohorts randomized to varying levels of added sugar early in life limits our understanding of the causal long-term effects of excessive sugar on human health.

This study leveraged a natural experiment: the end of a decade-long sugar and sweets rationing in the United Kingdom in September 1953, which led to quasi-experimental variation in sugar exposure in early life. During rationing, the sugar allowance for everyone—including pregnant women and children—was comparable to today's dietary guidelines of around <40 g for adults or <15 g for children and assumed little to no sugar offerings to children under 2 years (*3*, *19*–*21*). The cessation of rationing in 1953 led to an immediate, nearly twofold increase in the consumption of sugar and sweets (*22*).

To assess the effects of early-life sugar exposure on T2DM and hypertension, we used an event study approach to compare adults conceived shortly before rationing ended, with probable lower early-life sugar intake, against those conceived shortly after, with likely exposure to excessive sugar early in life. We hypothesized that exposure to sugar rationing in utero and early childhood reduced the risk and delayed the onset of T2DM and hypertension, with longer exposure having stronger effects.

Results

Dietary patterns during and after sugar and sweets rationing

Figure 1A describes percentage changes in average daily consumption of sugar in the UK from quarter 1 of 1950 (1950 q1) to 1958 q4, compared with average consumption levels during rationing, along with the intake of produce (fruits and vegetables), and total protein and fats over the same period. A vertical line marks the end of sugar rationing in September 1953. For an average adult, daily sugar consumption sharply increased from 41 g in 1953 q1 to about 80 g by 1954 q3; this similarly high level was

Table 1. Characteristics of survey participants born between October 1951 and March 1956. A total of 2699 and 1667 observations were missing for parental history of rationed and never-rationed adults, respectively; their calculations of *n* are adjusted accordingly. Differences in means were expressed in percentage points for all variables except age at the latest survey and were adjusted for parents being alive. Asterisks indicate significant difference as follows: *$P < 0.05$, **$P < 0.01$, ***$P < 0.001$. Romano-Wolf step-down adjusted *P* values. Because of rounding, reported percentages may not total 100. BMI, body mass index.

Characteristic	Rationed (*n* = 38,155)	Never rationed (*n* = 22,028)	Total (*n* = 60,183)
	n (mean in %)	*n* (mean in %)	Difference (percentage points)
Age at the latest survey	57.24 (SD = 3.24)	55.04 (SD = 3.37)	2.19*** (years)
Sex			
Male	16,754 (43.91)	9,633 (43.73)	0.19
Female	21,401 (56.09)	12,395 (56.28)	—
Place of birth			
England	32,745 (85.82)	18,743 (85.09)	0.73*
Wales	1,927 (5.05)	1,082 (4.91)	0.14
Scotland	3,483 (9.13)	2,203 (10.00)	—
Race/ethnicity			
Non-white	2,606 (6.83)	1,507 (6.84)	−0.01
White	35,549 (93.17)	20,521 (93.16)	—
BMI polygenic z-score (SD)	0.000 (SD = 1.003)	0.002 (SD = 0.996)	−0.00
Parents diagnosed with CVD†	22,227 (62.69)	12,872 (63.22)	−0.53
Parents diagnosed with diabetes	5,967 (16.83)	3,671 (18.03)	−1.20

†Parents diagnosed with cardiovascular disease (CVD) is a self-reported indicator after respondents answered the question "Was your biological mother/father ever diagnosed with stroke/heart disease/hypertension?"

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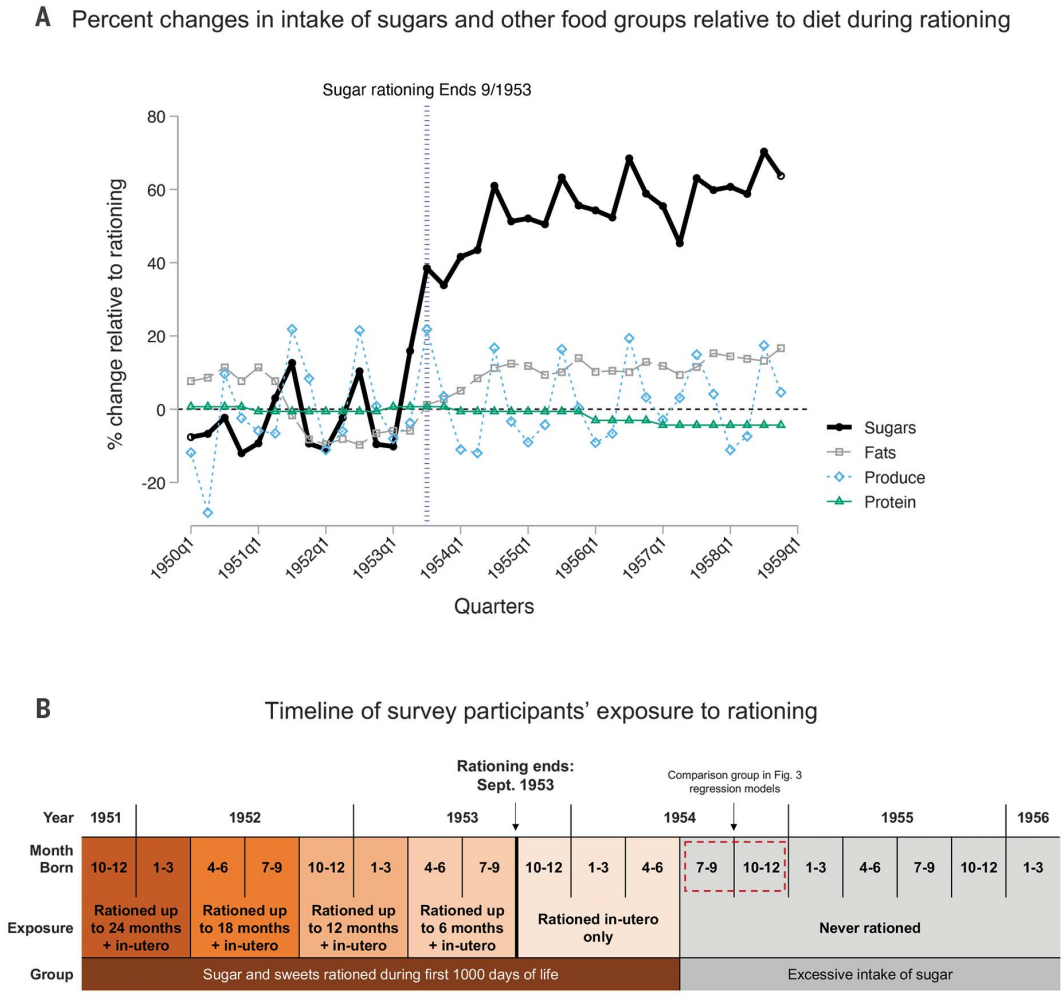

Fig. 1. Sugar and sweets rationing timeline and diet during and after rationing. (**A**) Calculations using National Food Survey data. (**B**) Rationed groups are shown in shades of orange, and never-rationed group is shown in gray. The red dashed rectangle represents adults born between July and December in 1954, which constitutes the first group that never experienced sugar and sweets rationing. This group of adults serves as the reference or comparison group in regression models.

sustained for several years. These data are for adults, but other research has shown that sugar intake for children more than doubled postrationing and that their oral health deteriorated as well (*21*, *23*). Relatively higher added sugar consumption for those conceived postrationing was maintained into the sixth decade of life (*24*).

After sugar rationing ended, total calorie intake sharply increased. Figure S1, A and B, shows quarterly fluctuations in sugar and total calorie consumption, and fig. S1C details their absolute changes relative to baseline levels during rationing (1950 q1 to 1953 q2). These data indicate that, on average, 77% of the increase in calorie intake postrationing was due to higher sugar consumption. The actual percentage was likely even higher: While data on sugar consumption exclude the intake of sweets and purchases outside the home, total calorie consumption includes a 10% adjustment for potential wastage and food bought outside the home (*25*). Figure S2 describes annual changes in sugar and sweets sales, which increased sharply after rationing ended.

Although sugar was not the only food derationed during our study period, and all rationing had ended by July 1954, the consumption of foods and nutrients other than sugar remained largely unchanged or showed only minor changes during our study period (Fig. 1A). Butter was derationed in May 1954. This caused households to largely revert to butter from margarine, likely leading to only a minor adjustment in overall fats consumption (Fig. 1A and fig. S3A). Similarly, despite the end of cheese rationing in December 1953, fig. S3B shows a relatively constant consumption of cheese and milk, including that provided to children in schools or on welfare. Figure S3C indicates a steady consumption of fresh fruits and vegetables but a notable increase in canned and dried fruit intake, especially after September 1953. This may have contributed to higher added sugar intake, as a 15-oz can of fruit in syrup contains between 30 and 40 g of added sugars and processed dried fruits often have added sugar (*26*). Cereals were derationed in July 1954, yet their consumption remained relatively stable (fig. S3D). We observed a minor decline in fish consumption and only a gradual increase in meat consumption despite its derationing in July 1954 (fig. S3E). This is consistent with protein substitution, as protein intake remained steady throughout our study (Fig. 1A). Figure S4 shows relatively constant food affordability over time, proxied with food prices adjusted for general inflation and wage growth.

In summary, the quick and sharp response in consumption and sales of sugar and sweets immediately after derationing, unlike for other foods, strengthens our research design. We examined whether there are differences in outcomes among individuals born within a few months of each other—specifically, cohorts conceived or born just before versus just after sugar and sweets rationing ended.

Study population characteristics

The sample included 60,183 participants born between October 1951 and March 1956, aged 51 to 66 when surveyed. Adults conceived in the 1000 days before September 1953 were classified as "rationed" (born October 1951 to

Table 2. The effect of rationing on disease risk and age of disease onset by duration of exposure to rationing. (Top) Hazard ratios are expressed relative to the hazard rate of those never exposed to rationing (born after July 1954). Parametric hazard models were estimated using the Gompertz distribution, although results were not sensitive to the choice of the distribution function, like Weibull. (Bottom) Time-to-event models assume Weibull distribution and are estimated using the "stteffects, ra" command in STATA. Asterisks indicate significance as follows: *P < 0.05, **P < 0.01, ***P < 0.001.

Rationing exposure	Type 2 diabetes (n = 60,183)	Hypertension (n = 60,183)
	1954 m7–1956 m3 (reference group)	1954 m7–1956 m3 (reference group)
Disease hazard ratio		
In utero versus 0	**0.87*** **	**0.92** **
SE	(0.03)	(0.02)
95% CI	0.81–0.94	0.88–0.97
P value	(0.000)	(0.001)
In utero + up to 1 year versus 0	**0.75*** **	**0.85*** **
SE	(0.04)	(0.02)
95% CI	0.68–0.84	0.81–0.89
P value	(0.000)	(0.000)
In utero + up to 2 years versus 0	**0.64*** **	**0.81*** **
SE	(0.02)	(0.02)
95% CI	0.60–0.69	0.77–0.84
P value	(0.000)	(0.000)
Delay in age of onset (years)		
In utero versus 0	**1.46** **	**0.53***
SE	(0.52)	(0.24)
95% CI	0.45–2.48	0.07–1.00
P value	(0.005)	(0.028)
In utero + up to 1 year versus 0	**2.80*** **	**1.47*** **
SE	(0.61)	(0.28)
95% CI	1.61–4.00	0.93–2.01
P value	(0.000)	(0.000)
In utero + up to 2 years versus 0	**4.17*** **	**2.12*** **
SE	(0.55)	(0.24)
95% CI	3.10–5.24	1.65–2.60
P value	(0.000)	(0.000)

June 1954, n = 38,155), and adults conceived after were classified as "never rationed" (born July 1954 to March 1956, n = 22,028). Figure 1B shows a timeline of exposure to rationing by calendar birth month. See table S1 for a sample distribution of births.

Table 1 shows balance in time-invariant characteristics between rationed and never-rationed cohorts, including sex (male: 43.91% versus 43.73%), race (white: 93.17% versus 93.16%), family history of diabetes (16.83% versus 18.03%), cardiovascular disease (62.69% versus 63.22%), and calculated genetic risk for obesity. Rationed adults were slightly more likely to be born in England (85.82% versus 85.09%, P < 0.05).

Association between early-life sugar-rich diet and risk for T2DM and hypertension

A total of 3936 participants were diagnosed with T2DM and 19,644 with hypertension. The risk of disease diagnosis increased with age for everyone, but it increased faster among adults with little or no exposure to rationing. Disease risk started to diverge when participants were in their mid 50s, and the largest differences were observed after age 60 (Fig. 2, A and B). See fig. S5 for estimated cumulative hazard. Unadjusted Kaplan-Meier survival curves (fig. S6) show that 92.2 and 65.4% of adults exposed to rationing in utero and postnatally had a higher chance of living without T2DM and hypertension to age 60, respectively, compared to those unexposed (89.1 and 61.4%, respectively). Log-rank tests confirmed significant survival curve differences at P < 0.001.

The risk of T2DM or hypertension decreased with the duration of exposure to rationing (Fig. 3). Each point in Fig. 3 is a hazard ratio (HR) estimate: the cohort's HR relative to the reference group of never-rationed adults born between July and December 1954. Compared with this reference group, adults exposed to rationing only in utero had about a 15% lower risk of T2DM [HR: 0.84; 95% confidence interval (CI): 0.75 to 0.95] (Fig. 3A and table S2). Risk was further reduced with longer exposure to rationing, particularly beyond 6 and 12 months postnatally. HRs remained relatively stable at around 0.62 (95% CI: 0.55 to 0.69) for adults exposed to rationing for at least 19 months. A similar pattern was observed for hypertension, although with a smaller risk reduction. The HR decreased from 0.94 (95% CI: 0.88 to 1.00), for adults exposed in utero only, to 0.79 (95% CI: 0.74 to 0.85), for adults exposed from conception through at least 19 months postnatally (Fig. 3B and table S2).

The HR estimates for T2DM and hypertension in never-rationed adults remained constant after December 1954, with all postrationing HRs statistically indistinguishable from 1 (P value at 0.498 for T2DM and 0.511 for hypertension) (Fig. 3). This supported our assumption of a similar disease risk trajectory between rationed and never-rationed cohorts independent of rationing. It also mitigated concerns about improvements in diagnostics or other events, such as improved access to medical care, over time, influencing the age of initial diagnosis, or regarding simultaneous exposure to sugar or other foods among adults who were never exposed to sugar rationing. For instance, butter went off-ration in May 1954. If fat rather than sugar alone affected these adults' health, we would expect that adults conceived after butter derationing (after May 1954) would fare differently than adults conceived prior (between October 1953 and April 1954) and that disease risk would increase for younger cohorts. However, we find no significant differences in T2DM and hypertension risk among adults conceived at different points after sugar rationing ended.

We further addressed potential concerns about time trends influencing our findings by analyzing conditions unlikely to be affected by sugar intake: type 1 diabetes, trauma-triggered depression, menarche, and myopia or hyperopia. Despite having similar diagnostic trends and requirements to T2DM or hypertension, these conditions showed no significant differences in HRs between cohorts (fig. S7).

Guided by HRs suggesting a similar disease risk between cohorts in the absence of rationing, and to increase statistical power, we combined never-rationed adults into one group and compared them with adults exposed to rationing in utero, in utero and up to age 1, and in utero and beyond age 1. In utero exposure alone explained about a third of the total reduction in T2DM and hypertension risk compared with exposure in utero and beyond age 1 (Table 2, top). T2DM risk reduction due to in utero exposure (HR: 0.87; 95% CI: 0.81 to 0.94) differed from in utero and postnatal

exposure of up to a year ($P < 0.01$) and beyond ($P < 0.001$). Hypertension risk reduction due to in utero exposure (HR: 0.92; 95% CI: 0.88 to 0.97) differed from postnatal exposure of up to a year ($P < 0.001$) and beyond ($P < 0.001$). Table S3 shows that disease risk especially declines with exposure beyond 6 months postnatally (at $P < 0.001$ for T2DM and hypertension). Among those with >1 year of postnatal exposure to rationing, the T2DM risk reduction was significantly larger for women than for men ($P < 0.01$) (fig. S8).

Early-life exposure to sugar rationing increased the time to the first T2DM or hypertension diagnosis (Table 2, bottom). Adults rationed in utero experienced a 1.46 (95% CI: 0.45 to 2.48) year delay in T2DM onset and a 0.53 (95% CI: 0.07 to 1.00) year delay in hypertension onset. Those exposed in utero and beyond 1 year postnatally experienced approximately a 4- and 2-year delay in diagnosis of T2DM and hypertension, respectively.

Figure S9 illustrates a roughly 30% decrease in risk of obesity as first diagnosed in primary care from exposure to rationing in utero and beyond the first year (HR: 0.69; 95% CI: 0.60 to 0.80). Figure S10 and table S4 also show that adults exposed to in utero and early postnatal rationing had about 2 percentage points lower prevalence of high waist-to-height ratios (WHRs), with no meaningful differences in continuous WHR or blood pressure outcomes between cohorts. The lack of differences may be due to improved disease management after long-standing diagnoses of T2DM or hypertension and because sugar rationing delayed but did not prevent them.

Figure S11 and table S5 describe goodness of fit for parametric hazard models with Gompertz baseline parametrization and the criteria for these findings. Figure S12 presents a test of proportionality assumptions. Results remained robust across various model specifications (table S6), including adjusting models for changes in food affordability (fig. S13).

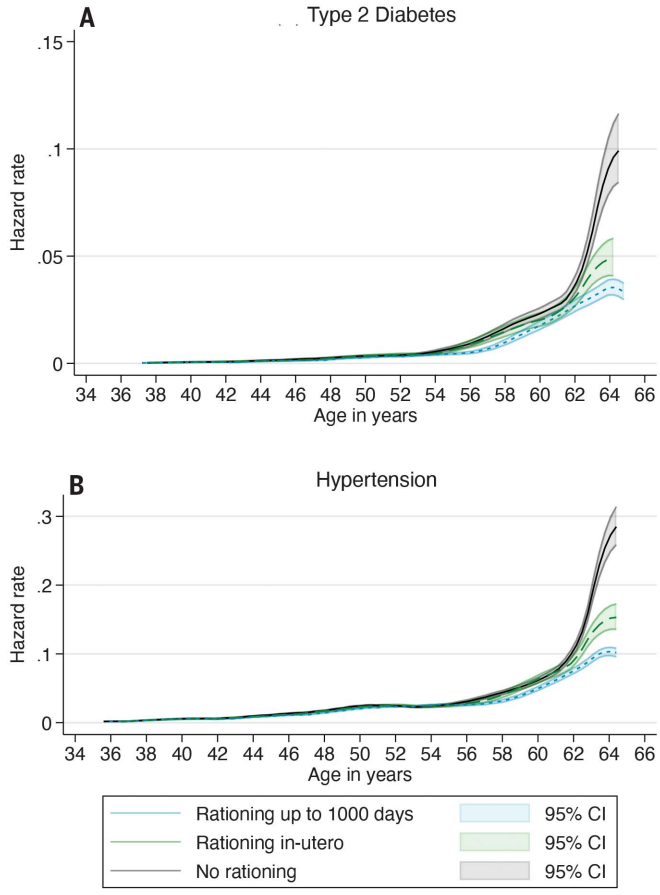

Fig. 2. Smooth hazard functions for type 2 diabetes and hypertension by exposure to rationing. (**A**) Hazard function for T2DM by rationed in utero or longer versus never rationed. (**B**) Hazard function for hypertension by rationed in utero or longer versus never rationed. Log-rank test confirmed significant difference between survival curves at $P < 0.001$. 95% confidence intervals are shown.

Discussion

We examined the impact of exposure to sugar restrictions during the first 1000 days after conception on T2DM and hypertension, leveraging quasi-experimental variation from the UK's end of sugar and sweets rationing in September 1953. Derationing nearly doubled sugar consumption. We found that early-life exposure to sugar rationing led to a reduction in T2DM and hypertension risk by about 35 and 20% and delayed the onset of these diseases by about 4 and 2 years, respectively. In utero sugar rationing alone was protective, but most risk reduction occurred when rationing lasted beyond the age of 6 months.

Restricting early-life sugar intake could affect health by altering physiological programming in utero, as supported by evidence from the "fetal origins hypothesis" (*2*, *11*, *27*). We found that exposure to in utero rationing alone contributed to about one-third of the overall reduction in T2DM and hypertension risk and accounted for approximately a quarter of the delay in disease onset. Our findings on in utero sugar effects are consistent with animal studies demonstrating that high-sugar diets during pregnancy increase risk factors for T2DM and hypertension, such as insulin resistance and glucose intolerance, in adulthood (*8*, *9*) and with studies on humans demonstrating an association between sugar-rich diet during pregnancy and lactation with increased obesity risk in offspring (*28*).

Furthermore, early-life sugar exposure may affect health by intensifying a lifelong preference for sweetness. Infancy and toddlerhood in particular are critical periods for developing a taste for sweets (or even addiction) that can elevate sugar consumption throughout life (*24*, *29*–*31*). More than 70% of foods marketed to infants or toddlers, including formula, foods, and beverages, contain added sugars (*4*). By age 2, many children consume the adult-recommended 7 teaspoons of sugar daily, and this amount nearly triples by their teens (*6*). We identified improved health from restricting sugar beyond gestation; postnatal rationing accounted for the majority of the reduction in disease risk, especially for T2DM. Its protective effects especially increased when extended past 6 months—the typical age for introducing solids (*32*). Women showed greater postnatal health benefits than men in terms of reduced T2DM risk, reflecting findings from animal studies that females in high-sugar settings are more susceptible to sugar addiction and disrupted glucose regulation, both of which elevate T2DM risk (*33*, *34*). Postnatal rationing also reduced obesity risk and high-WHR prevalence, which are known markers for increased cardiometabolic risk (*35*). Although not necessarily a primary cause (*35*), obesity suggests a biological pathway, as it correlates with elevated sugar intake and metabolic disease (*36*, *37*). Disease effects stabilized by age 1; the benefits of restricting sugar intake beyond age 2 remains uncertain.

Our findings provide insights beyond the context of rationing, because the sugar allowance then aligned with today's limits set by the United States, World Health Organization, and American Heart Association dietary guidelines (*17*, *38*). Although we did not directly evaluate these guidelines, our findings imply that adhering to them early in life could provide substantial health benefits. The magnitudes of our results are comparable to the 2.8-year-long

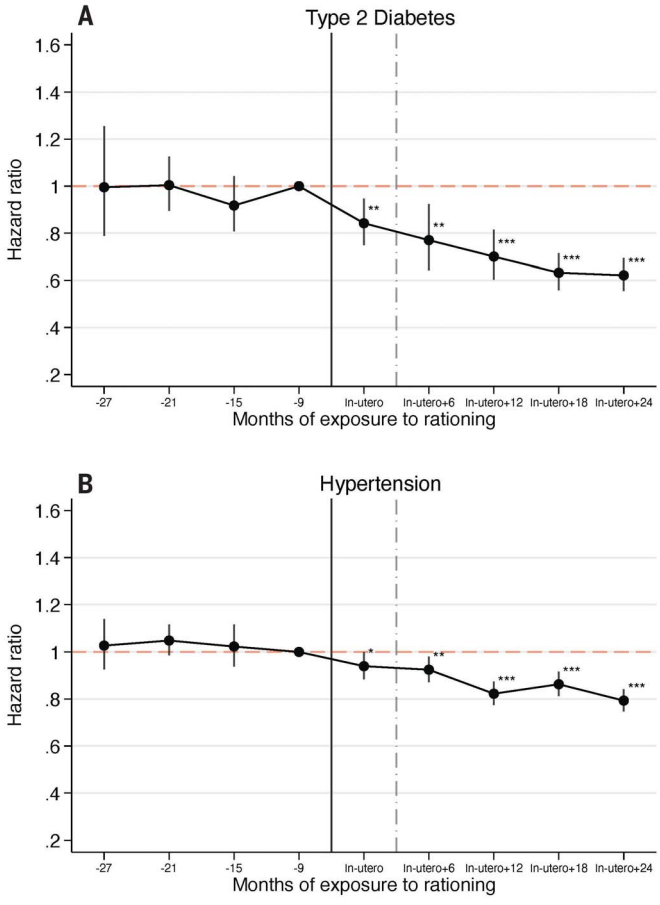

Fig. 3. Hazard ratios comparing the hazard rates at various levels of rationing exposure to the hazard rate of never-rationed adults born between July and December 1954. (**A**) Hazard ratios at various durations of rationing exposure for T2DM. (**B**) Hazard ratios at various durations of rationing exposure for hypertension. Each point represents a hazard ratio, comparing the hazard rate of adults exposed to rationing in utero, in utero plus 6, 12, 18, or 24 months after birth, or not at all, to the hazard rate of the reference group of never-rationed adults born between July and December 1954 (>9 months after rationing). Adults never exposed to rationing were also those born between January and June 1955 (>15 months after rationing), July to December 1955 (>21 months after rationing), and January to March (>27 months after rationing). The HR estimates for adults born between January 1955 and April 1956, who were never rationed, were neither individually nor jointly significantly different from the HR estimate of adults born in the reference group of July to December 1954 (at $P = 0.498$ for T2DM and $P = 0.511$ for hypertension). All regressions include indicators for calendar month of birth, survey year, birth place (England versus Wales versus Scotland), deciles of north and east place-of-birth coordinates, self-reported sex, race (white versus not), and parental cardiovascular disease (hypertension, heart disease, stroke), or diabetes indicator. Asterisks indicate significance as follows: $^{+}P < 0.1$, $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$. See table S2 for results supporting this figure.

National Diabetes Prevention Program that decreased T2DM risk by 34% and delayed its onset by about 4 years a decade later (*39*), potentially extending life expectancy by about 1 to 1.5 years (*40*).

Dietary guidelines on added sugar intake have been criticized for relying on moderate-to-low-quality short-term evidence focused on obesity and dental health, on observational studies, and on short-term clinical trials that studied T2DM or hypertension or their markers (*17*, *41*). None were focused on the first 1000 days after conception. As conversations intensify regarding policies such as taxing sugar or sugar-sweetened beverages (*42*) or the regulation of added sugars in infant or toddler foods and their marketing (*43*), understanding the direct link between early-life sugar consumption and chronic disease is critical. Our results add to this discussion by not only linking sugar to health but also underscoring the importance of early-life diet in managing long-term metabolic disease risk.

This study has limitations, but we have taken various steps to ensure the robustness of our results. One concern was the potential for unobservable differences between cohorts conceived during and after rationing (aside from early-life sugar exposure), particularly without a contemporaneous control group. We addressed this in several ways: (i) We used a narrow time window around the end of rationing and observed similar early-life and parent disease characteristics between cohorts. (ii) We provided evidence that diet in the 1950s, aside from sugar, was similar between cohorts. (iii) Our results controlled for several early-life characteristics and food prices and are robust to excluding them. Additionally, we observed no differences in disease risk between cohorts for conditions likely unrelated to diet. Finally, equivalent to testing pretrends in outcomes between rationed and never-rationed adults, HRs among older and younger never-rationed adults showed no significant differences irrespective of birth date.

Another limitation was that UK Biobank is not nationally representative and skews toward wealthier, healthier individuals, although it is useful for studying exposure-outcome relationships (*44*) and offers a larger sample than comparable surveys. The likelihood of volunteering for surveys was likely unrelated to the end of rationing, as both the rationed and never-rationed cohorts were recruited using the same protocols. However, if sugar rationing improved economic well-being (*24*), wealthier adults might have received earlier disease diagnostics owing to improved access to health care. The single-payer health care system in the UK helps lessen these concerns, as do our results showing no impacts on placebo conditions. A final limitation was right censoring and no prestudy mortality information; the latter concern is small given the similarly low mortality rates in rationed and never-rationed adults (*45*).

This study has several strengths. A quasi-experimental assignment to higher or lower early-life sugar exposure strengthens the likely causal nature of our findings. We accounted for confounding factors and consistently observed a protective effect of sugar rationing across various analytical specifications. We provided strong evidence that the health impacts observed were due to changes in sugar calorie consumption rather than shifts in consumption of other nutrients. Finally, our large sample enabled separate identification of in utero and in utero/postnatal effects, while still using a narrow analytic window, and subgroup analyses by sex.

We conclude that limiting exposure to sugar in utero and in early life can protect against T2DM and hypertension. Further research is needed to understand the optimal levels of added sugar consumption during pregnancy, lactation, and after the introduction of solids, as well as their pathways to influencing long-term health.

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SUPPLEMENTARY MATERIALS

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