



Supplementary Materials for

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

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Materials and Methods

Figs. S1 to S13

Tables S1 to S6

References

Other Supplementary Material for this manuscript includes the following:

MDAR Reproducibility Checklist

Correction (5 May 2025): In the first paragraph on p. 5 of the supplementary materials, the birth months listed in parentheses following the definition of a variable identifying exposure to rationing were accidentally presented in reverse order owing to an oversight during revision of the methods section prior to publication. The order has been corrected. Note that the correct birth months defining exposure are shown consistently elsewhere—specifically in Fig. 1 of the main text and table S1, both of which provide a detailed mapping of birth months to rationing exposure. No other figures, tables, or sections of the text are affected, and this correction does not affect any results, conclusions, or interpretations. The underlying Stata code is also correct and accurately reflects the analyses.

MATERIALS AND METHODS

This study was approved by RAND's (2019-0625) and University of Southern California's ethics review (UP-24-00120). The UK Biobank application number was 58599.

Background

Sugar and sweets rationing in the United Kingdom (UK) commenced in July 1942 as an integral component of a 14-year long food rationing program implemented in response to World War II. The primary objective of food rationing was to ensure a healthy diet and equitable distribution of food, thereby avoiding food shortages and starvation. The Ministry of Food implemented a scientifically calculated weekly allocation of specific foods to everyone, aimed at maintaining a minimal nutrient intake conducive to good health. Consequently, restrictions on processed, non-essential foods such as sugar and sweets were a fundamental aspect of the rationing system.

During the period of sugar and sweets rationing, every individual, including pregnant women and children aged 5 and above, received an allotment of approximately 8oz of sugar per week and 12oz of sweets per month. These provisions could be obtained by utilizing individually assigned ration books after registering with designated retailers. Children below the age of two were not specifically allotted sugar or sweets as part of their ration, as dietary practices for infants during that period focused on breastfeeding, baby formula, and age-appropriate foods, like grains, vegetables, and fruits. While during rationing, children's average consumption of sugars from sweets was within recommended amounts - below 4oz (115g) per week, soon after its end, their intake of sugar and sweets more than doubled. Evidence shows that their oral health deteriorated as well.(21, 23) Rationing of sugar concluded in September 1953, while certain food items such as fats, rice, cheese, meats, milk, and cereals remained under rationing until after but no later than July 1954 when all food rationing came to an end.(3, 19-22)

Study Design

We used an event study approach to identify the long-term health impacts of restricted exposure to added sugars during the first 1000 days since conception. We leveraged the end of sugar rationing in September 1953, which caused an immediate and sharp increase in the consumption of sugar, but not other foods (**Figure 1A**, **Figures S1 - S3**), as a natural experiment to compare adults who were restricted to limited sugar amounts during their early life to those who were not. Birth fully determined one's early-life exposure to rationing, leading to adults being quasi-experimentally assigned to either within recommended (rationed) or excessive sugar (non-rationed) amounts in utero or in early childhood. **Figure 1B** describes a timeline of sugar rationing and classifies individuals into rationed and non-rationed groups based on their year and month of birth. To identify individuals conceived within 1000 days before the end of rationing, we assumed that on average, quarters have 91 days. Since there are 11 quarters or roughly 1001 days between 1951q1 and 1954q2 (the exact number of days varies by leap years), we assumed that a roughly 1000-day window spans October 1st 1951 – June 30th 1954. Adults born on or after July 1st 1954 are considered as non-rationed or never exposed to sugar rationing.

Our analytical approach followed the intuition of a regression discontinuity design: focusing our analysis on individuals conceived within a narrow window around the end of rationing, we assumed that all other early-life determinants of adult health were similar and did not change discontinuously at the event between groups. Thus, this narrow window around the end of rationing allowed us to 1) assume that in the absence of rationing, the disease risk and onset was continuous and comparable between adults conceived before or after September 1953, and 2) anticipate that the risk of T2DM and hypertension of adults who were never subject to rationing will be similar regardless of when they were conceived after September 1953.

Study data and participants

We used National Food Survey (NSF) reports to examine quarterly dietary patterns during rationing and soon after its end between 1950 and 1960. Since 1940, NSF data has tracked weekly dietary recordings across over 10,000 households, which were then aggregated into the quarterly estimates of weekly per person consumption of various food groups, including sugar, fats, meats, vitamins, fruits, or vegetables, reported at a national level.(25) We digitized NSF reports and reported quarterly means of consumption of sugar and other foods during and after sugar rationing using these data. Data on annual sugar and sweets sales data came from (23).

Health outcomes were from the UK Biobank (2006-2022), which collected data on medical history, including International Classification of Diseases 10th Revision (ICD-10) codes retrieved from medical records through May 2022, and genetic, lifestyle, or other disease risk factors for >500,000 participants. UK Biobank data were previously described elsewhere.(48)

Our sample included $n=60,183$ adults ($n=38,155$ rationed, $n=22,028$ non-rationed) born between October 1951 and March 1956. We excluded individuals born or living outside of UK at the time of the survey, those who were part of a multiple birth or adopted, who withdrew their data from UK Biobank, were pregnant at the time of the survey, or had missing covariates.

Study variables

Early-life exposure to sugar

We created indicators based on the duration of exposure to rationing: never, in utero only, and in utero plus 6, 12, 18, or 24 months postnatally. In utero and up to 6 months duration was selected because the introduction of solid food typically starts then. To improve statistical power, we also created indicators for exposure to rationing in utero and up to age 1, in utero and up to age 2, or never. **Figure 1B** and **Table S1** display the timeline of rationing exposure and the distribution of births during our study period, respectively.

Dependent variables

Primary outcomes were T2DM and hypertension diagnosis and their age of onset. In our study, individuals experienced T2DM or hypertension if they answered affirmatively to a question about whether they were ever diagnosed with T2DM or hypertension by a doctor (data field 2443 and 1065, respectively) or if they had this disease diagnosis recorded in their primary care or hospital

data linked with UKB (data field 130708 for diabetes and 131286 or 131294 for hypertension); and if yes, at what age. Using hospital linkages, disease was defined using the codes for Internal Classification of Diseases 10 (ICD10): E11 for T2DM, and ICD10 I10 and I15 for hypertension. Time at disease onset was computed by subtracting birth date from the initial diagnosed date divided by 365.25 using self-reported or healthcare data (data field 2976 for diabetes and 2966 for hypertension). If both were available, we used the earlier age. To minimize misclassification of type 1 diabetes as T2DM, we coded those with T2DM onset before age 36 or those who initiated insulin within one year of diagnosis of diabetes (data field 2986) as missing. To ensure cohort age overlap, follow-up and disease onset ages were capped at 66.(49)

We employed a similar methodology for other outcomes. Age of obesity diagnosis in a primary care setting was determined using data fields 130792 and 30790. Our placebo outcomes were conditions unlikely to be influenced by diet in early life: type 1 diabetes, trauma-triggered depression, menarche and first prescription for glasses or myopia/hyperopia. The age of onset for type 1 diabetes was defined using data fields 130706, 130714, and 130712 (ICD10 codes E10, E13, and E14), with the condition of insulin initiation within the first year of diagnosis. We included an additional condition that diabetes was diagnosed before the age of 36 for self-reported age of diagnosis (data field 2976), where it was more challenging to differentiate between diabetes types. The age at which depression likely triggered by a traumatic event was first diagnosed was defined using data fields 20433 and 20447, age at menarche by using data field 2714, and age of first prescription for glasses used data field 2217.

Finally, we examined cross-sectional measures of waist-to-hip ratio (WHR), calculated as a ratio between waist (field 48) and hip (field 49) circumference, both in centimeters, a binary variable indicating high WHR (above 90th percentile at $\text{WHR} \geq 1$ for men and ≥ 0.85 for women), systolic blood pressure (mm Hg), and an indicator for high blood pressure (systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg (fields 4080 and 4079, respectively). Measures used were obtained from the first survey round.

Statistical Analysis

Descriptive statistics included unadjusted means with standard deviation (SD) for characteristics of rationed and non-rationed adults. We used Romano-Wolf step-down adjusted p-values to determine whether differences in these characteristics were zero. Since adults who experienced rationing likely had older parents with more chronic diseases, we adjusted our comparisons based on whether their parents were alive during the survey. Unadjusted Nelson-Aalen (NA) smoothed hazard (**Fig. 2**) and Kaplan-Meier survival curves (**Fig. S6**) were estimated to describe unadjusted associations between rationing and time to disease diagnoses. We tested the difference between survival curves using a log-rank test.

We used an event study design estimated with multivariate parametric hazard models to obtain T2DM and hypertension hazard ratios (HRs) for individuals exposed to rationing relative to those unexposed. We estimated the following equation using parametric hazard models:

$$(1) \quad h(t)_i = \exp \left(\sum_{k=-3, k \neq 0}^5 \beta_k * (\text{Birthgroup}_k = 1) + \theta' X_i + \mu_m + \delta_t \right) * \exp(\gamma t)$$

Where $h(t)$ was the hazard rate at time t , and γ was a shape parameter in Gompertz distribution. The subscripts k denoted event periods, which were intervals relative to the reference period 1954m7-1956m3 ($k=0$; birth months of adults who were the first to not be exposed to sugar and sweets rationing, omitted). The subscript $k=1, \dots, 5$ refers to five binary variables (*Birthgroup*) indicating 6-month intervals of exposure to rationing (9 months for in utero exposure): in utero (if born 1953m10–1954m6, $k=1$), in utero and through age 6 months (if born 1953m4–1953m9, $k=2$), in utero and through age 12 months (if born 1952m10–1953m3, $k=3$), in utero and through age 18 months (if born 1952m4–1952m9, $k=4$), and in utero and through age 24 months (if born 1951m10–1952m3, $k=5$). The subscript $k=-1, \dots, -3$ denoted three binary indicators for whether adults who were never exposed to sugar and sweets rationing were born between 1955m1–1955m6 ($k=-1$), 1955m7–1955m12 ($k=-2$), and 1956m1–1956m3 ($k=-3$), respectively.

This model effectively compared hazard rates in each period relative to the hazard rate of the reference, non-exposed group, that is, the first fully never-rationed cohort of adults who were conceived after September 1953 or born between July and December 1954. These relative differences were described by the coefficients β for each k as hazard ratios (HRs). We presented these coefficients in **Figure 3** and **Table S2**, along with 95% confidence intervals, adjusted for serial correlation in the outcome by clustering at the birth year-month level.

All models included time-invariant individual-level indicators (denoted by $\theta' X_i$) for whether the participant was male (vs female), non-white (vs white), was born in England (vs Wales vs Scotland), had high (vs low) risk for obesity genetically. The genetic risk indicator was based on previously generated polygenic score data (return code 3586, application 11425).(50) Results did not change if polygenic scores were excluded or included as a continuous variable. Models also included indicators for self-reported parental history of diabetes and of cardiovascular disease (stroke, hypertension or heart disease) along with an indicator for whether information on parental health history was missing (4,366 observations or 7.5% of the sample), and indicators for deciles of north and east coordinates of birth location; results were not sensitive to including or excluding these controls. Calendar birth month indicators (reference category January) were included to control for seasonality (denoted by μ_m), and nationwide outcome trends were captured by baseline survey calendar year fixed effects (denoted by δ_t).

Binary indicators for birth timing of non-rationed adults were included for two reasons: firstly, adults born after that date served as a comparison group. Secondly, comparing outcome differences for adults born in 6-months increments helped us address a potential concern that our results may be biased due to either general time trends or improved disease diagnostics leading to earlier disease detection among non-rationed adults. If this was the case, risk for chronic disease would increase with time. Our identifying assumption was therefore that, conditional on controls, the end of rationing was not associated with deviations in disease risk from the reference group for all non-rationed adults born after December 1954, keeping their HRs around 1.

We reported results as HRs with 95% confidence intervals (CIs) and p-values. */**/*** indicates significance at p value smaller than 0.05/0.01/0.001. We used Huber-White robust standard errors clustered by year-birth month and conducted the analysis with Stata SE software, version 18.0.

We applied standard tests to assess the adequacy of our models: examining the shape of unadjusted hazard rate and cumulative hazard estimates from the Nelson-Aalen estimator estimates, AIC/BIC criteria, and a relationship between Cox-Snell residuals relative to Nelson-Aalen cumulative hazard estimates. Sensitivity analyses included varying covariates, exposure duration, and adjusting models for changes in food affordability using real food prices adjusted by consumer price index in the first postnatal quarter as proxies. We also varied period baseline hazard distributions (e.g., Weibull), and estimated semi-parametric multivariate Cox-proportional hazard models. To address concerns about general trends or spurious correlations influencing our findings, we re-estimated model (1) for the outcomes of type 1 diabetes, trauma-induced depression, menarche, and the use of prescription glasses.

Our identifying assumption was that, conditional on controls, no trend in HRs among non-rationed adults should be observed, and that the end of rationing was not associated with their hazard rate deviations from the reference cohort, keeping HRs around 1. Thus, guided by the observed patterns of HRs from model (1), we estimated the before vs after regression model when testing for discontinuities in the health impacts of exposure to rationing, grouping birth groups together in several ways to increase statistical power:

$$(2) \quad h(t) = \exp(\sum_{k=1}^3 \beta_k(Birthgroup_k) + \theta'X_i + \mu_m + \delta_t) * \exp(\gamma t)$$

We created corresponding categorical variables ranging from 0 – 3. We compared all non-rationed adults (i.e., reference group are all adults born post 1954m7, k= 0) to those who were exposed to rationing in-utero (k=1), in-utero and up age 1 (k=2), or in-utero and up to age 2 (k=3) (see **Table 2**). We also examined other periods and created a variable indicating those exposed to rationing in-utero (k=1), in-utero in addition to six months postnatally (k=2), or up to age 2 (k=3) (see **Table S3**). Controls included were the same as in model (1). T-statistics were used to test for statistical differences between pre- vs pre- and postnatal effects.

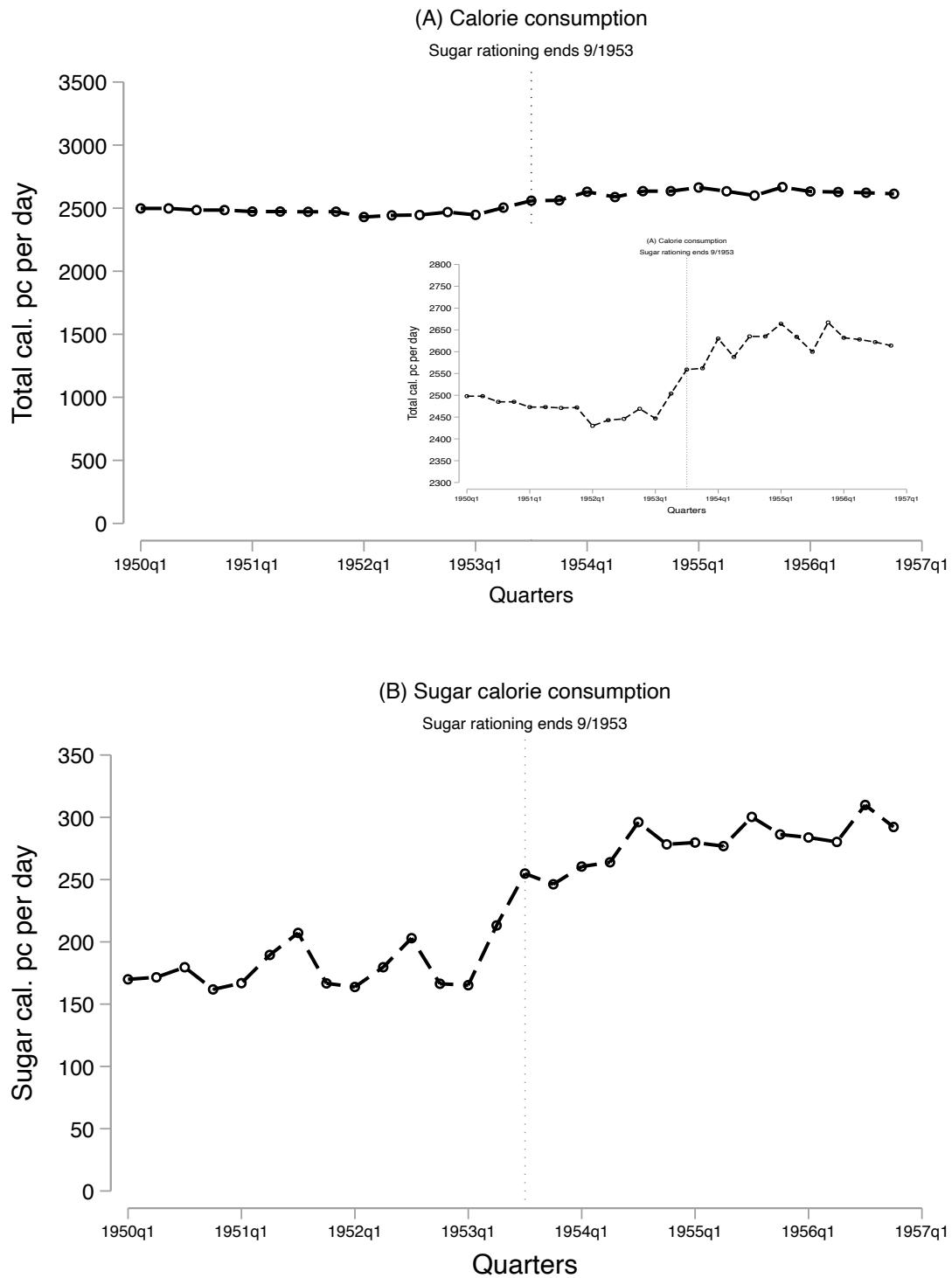
To estimate the effect of rationing on age of disease onset, we used time-to-event models assuming Weibull distribution, and again adjusted for sex, race/ethnicity, obesity and parental genetics, survey year indicators, and place of birth. We estimated the average treatment effect of early-life exposure to rationing on the age of onset of T2DM and hypertension. We estimated this average treatment effect by regression adjustment in STATA using stteffects ra command - overall and by duration of exposure to rationing.

We estimated the link between measures related to WHR and blood pressure using ordinary least squares for continuous outcomes, and linear probability regression models for binary outcomes, controlling for age, age squared, and the set of other controls listed above.

Replication files are available in the Dryad and Zenodo repositories.(46, 47)

Supplementary Figures

Figure S1.



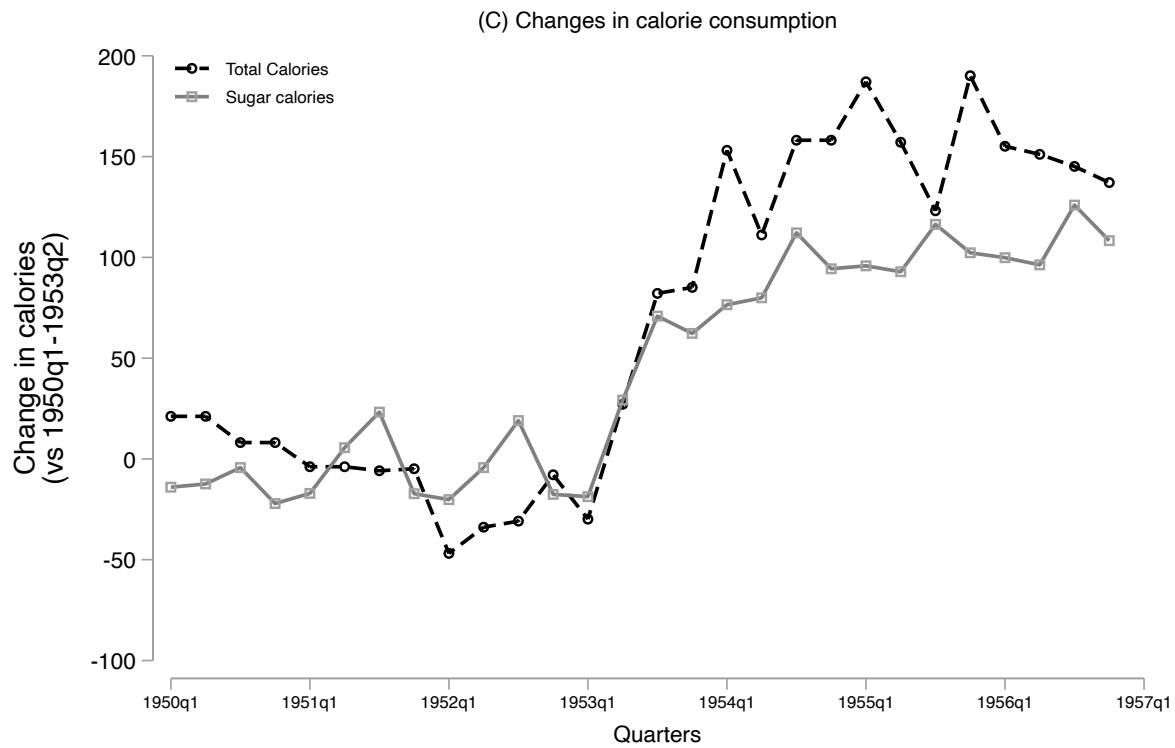


Figure S1. Total and sugar calorie intake between 1950q1 and 1956q4. Quarterly data presented are through 1956 because data for 1957-1960 are only available at a yearly level for total calories; patterns are consistent with data presented here. **(A)** Quarterly total calorie consumption, measured in calories per capita per day. The inset shows the same data with a truncated axis. **(B)** Quarterly sugar calorie (assuming 4 calories per 1 gram of sugar) intake, measured in calories per capita per day. **(C)** Comparison of quarterly total calorie or sugar calorie consumption to the average consumption between 1950q1 and 1953q2; the dashed line represents total calories, and the solid gray line represents sugar calories.

Figure S2

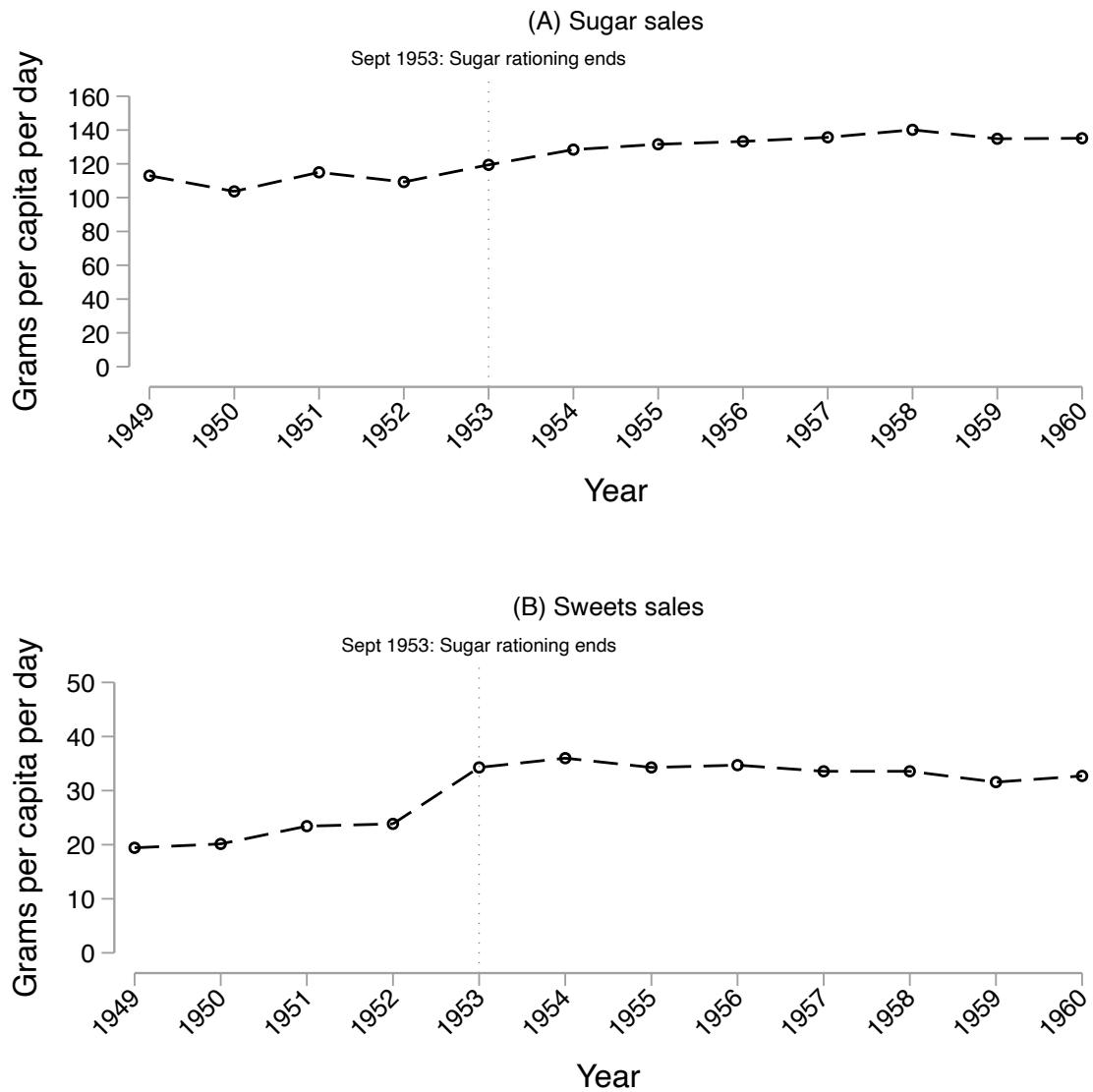
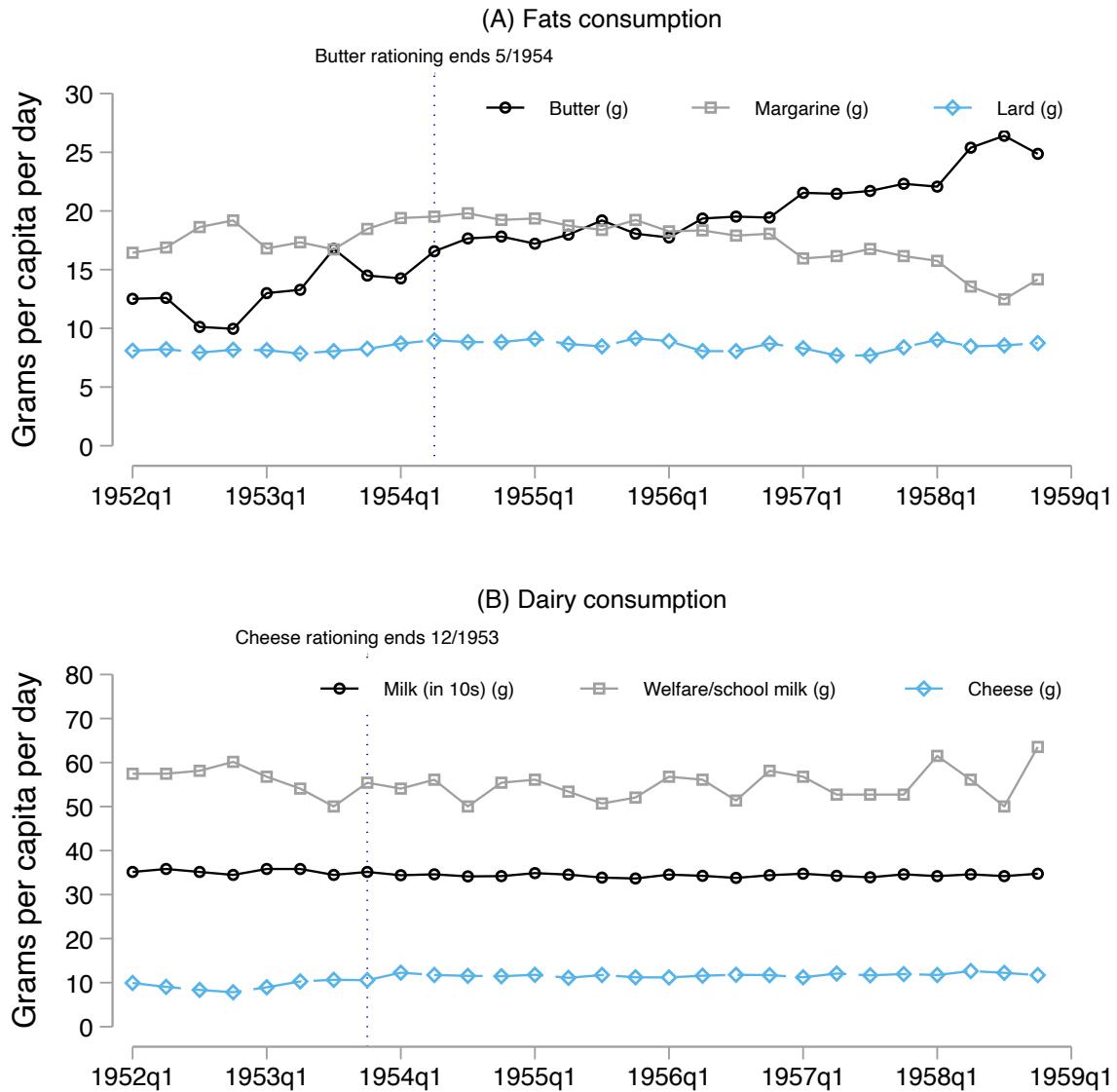


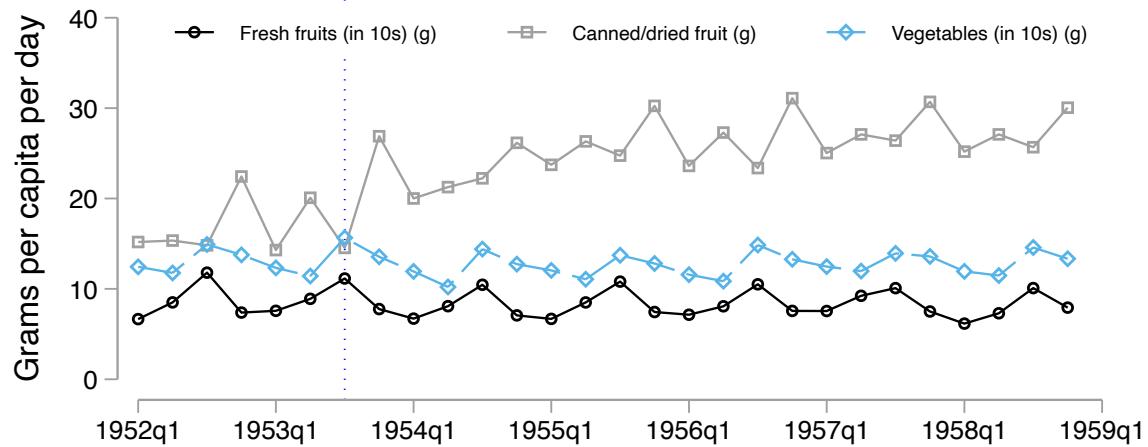
Figure S2. Annual sales of sugar and sweets between 1949 and 1965. **(A)** Annual sales of sugars. **(B)** Annual sales of sweets. Annual data on sales of sweets and sugar obtained from (23).

Figure S3



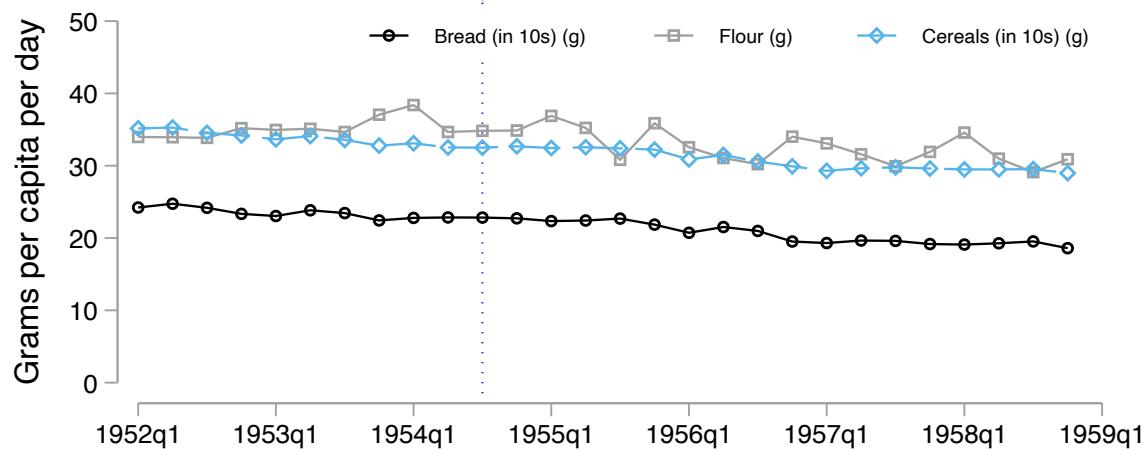
(C) Fruits and vegetables consumption

Sugar rationing ends 9/1953



(D) Cereal consumption

Cereal rationing ends 7/1954



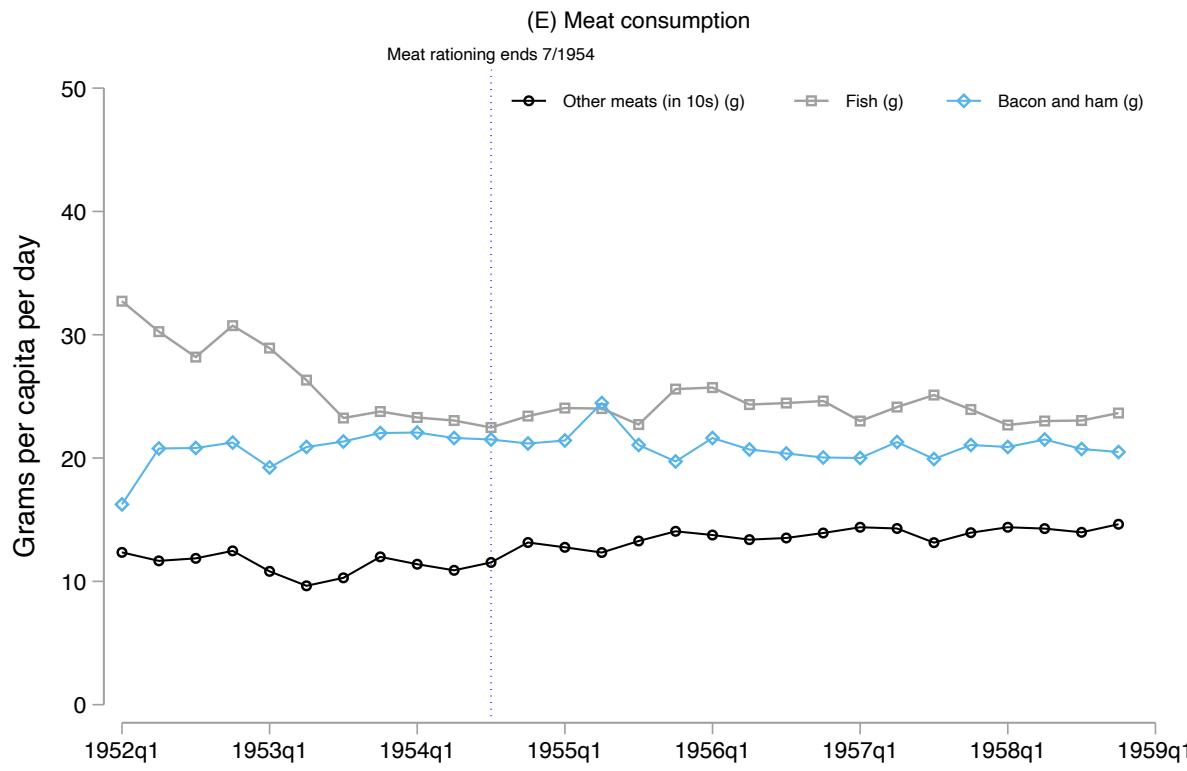


Figure S3. Quarterly consumption of fats, dairy, fruits and vegetables, cereal, and meat between 1952q1 and 1958q4. **(A)** Quarterly consumption of fats (butter, margarine, and lard). **(B)** Quarterly consumption of dairy (milk, cheese, milk on welfare/in schools). **(C)** Quarterly consumption of produce (fresh fruit, fresh vegetables, and canned vegetables and fruit). **(D)** Quarterly consumption of cereal (bread, flour, and cereals). **(E)** Quarterly consumption of meats (fish, bacon and ham, and other meats). Quarterly data on detailed food sub-categories is consistently available from 1952q1 through 1958q4. All quantities were originally reported in oz (or pints for milk) per head per week, which we converted into grams per day. Milk, fresh fruits, vegetables, bread and cereal are reported in 10s to simplify visualization.

Figure S4.

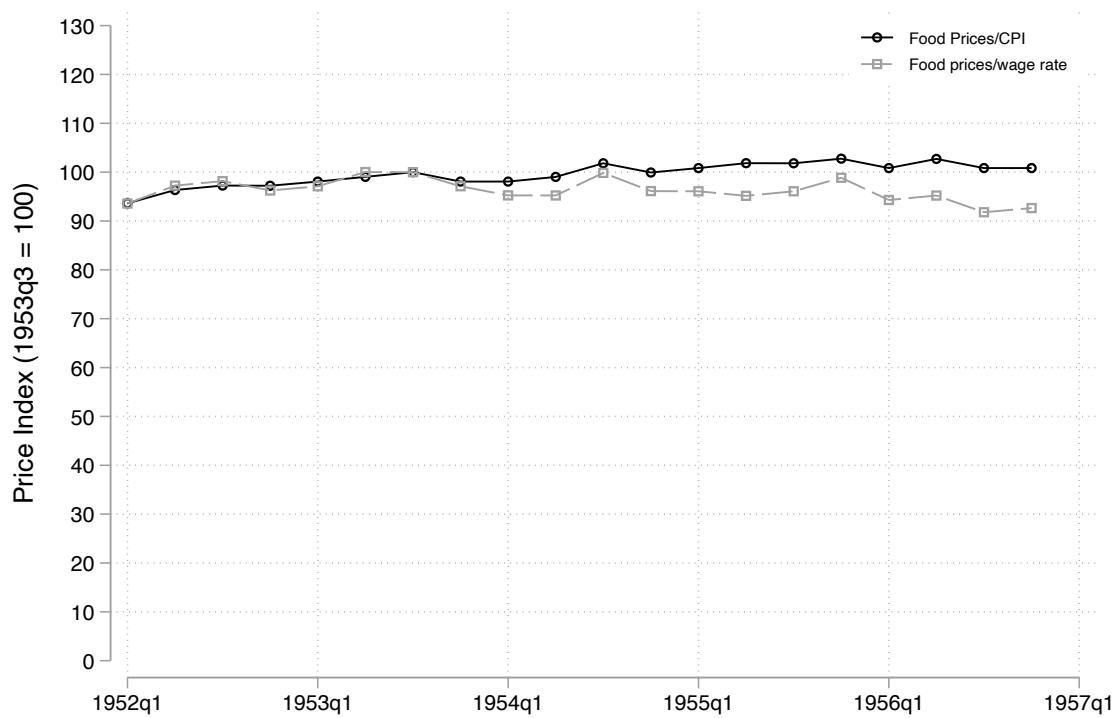


Figure S4. Food prices relative to general inflation and wage growth between 1952q1 and 1956q4 (1953q3=100). Quarterly data on prices were collected from historic NFS reports, where they were consistently available from 1952q1 through 1956q4.(25)

Figure S5.

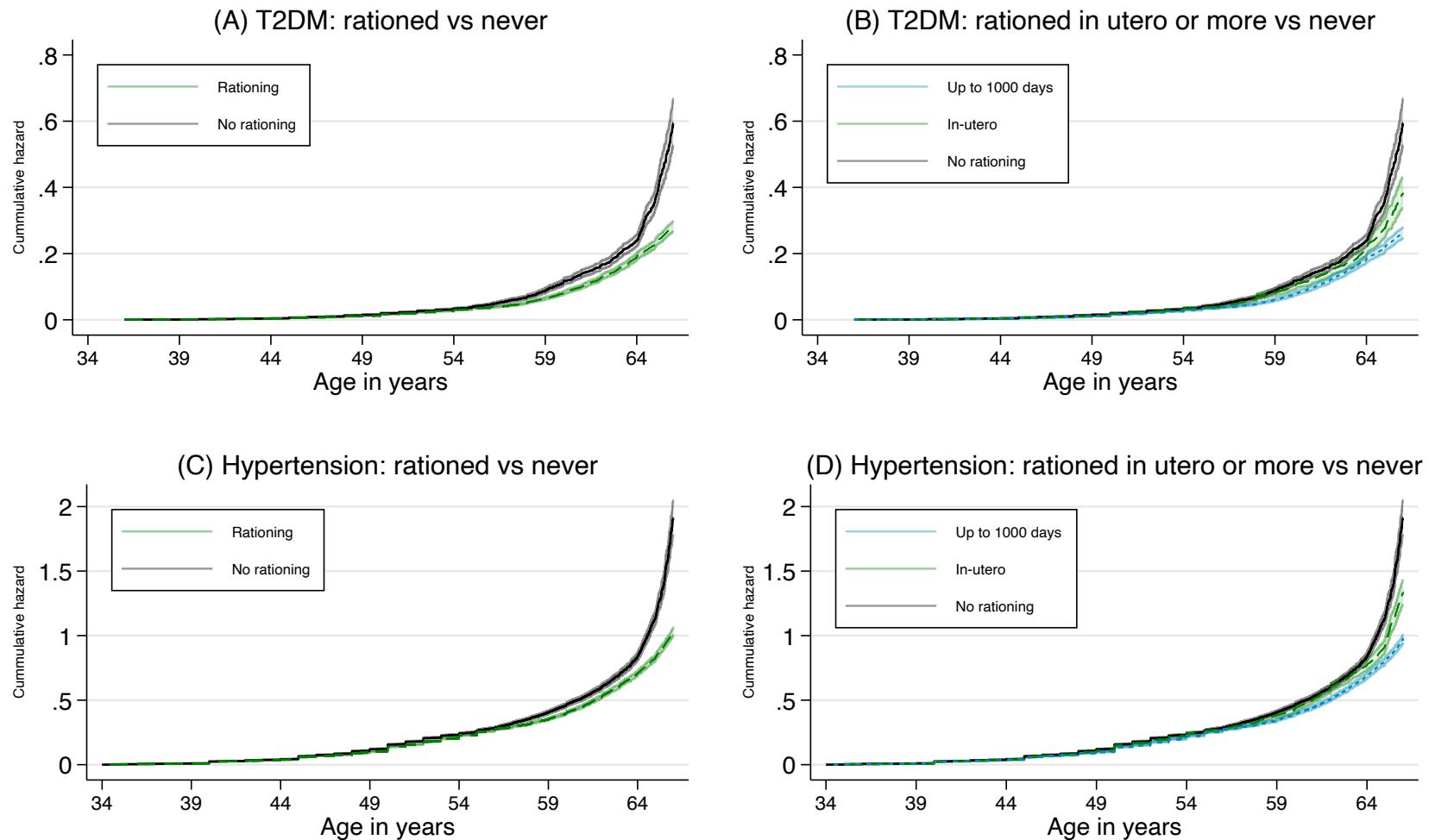


Figure S5. Cumulative hazard for type 2 diabetes and hypertension. Cumulative hazard for **(A)** type 2 diabetes by ever vs. never exposed to rationing, **(B)** type 2 diabetes by rationed in utero or more vs. never rationed, **(C)** hypertension by ever vs. never exposed to rationing, **(D)** Hypertension by rationed in utero or more vs. never rationed. Tables supporting these figures can be found on Dryad repository.(47)

Figure S6.

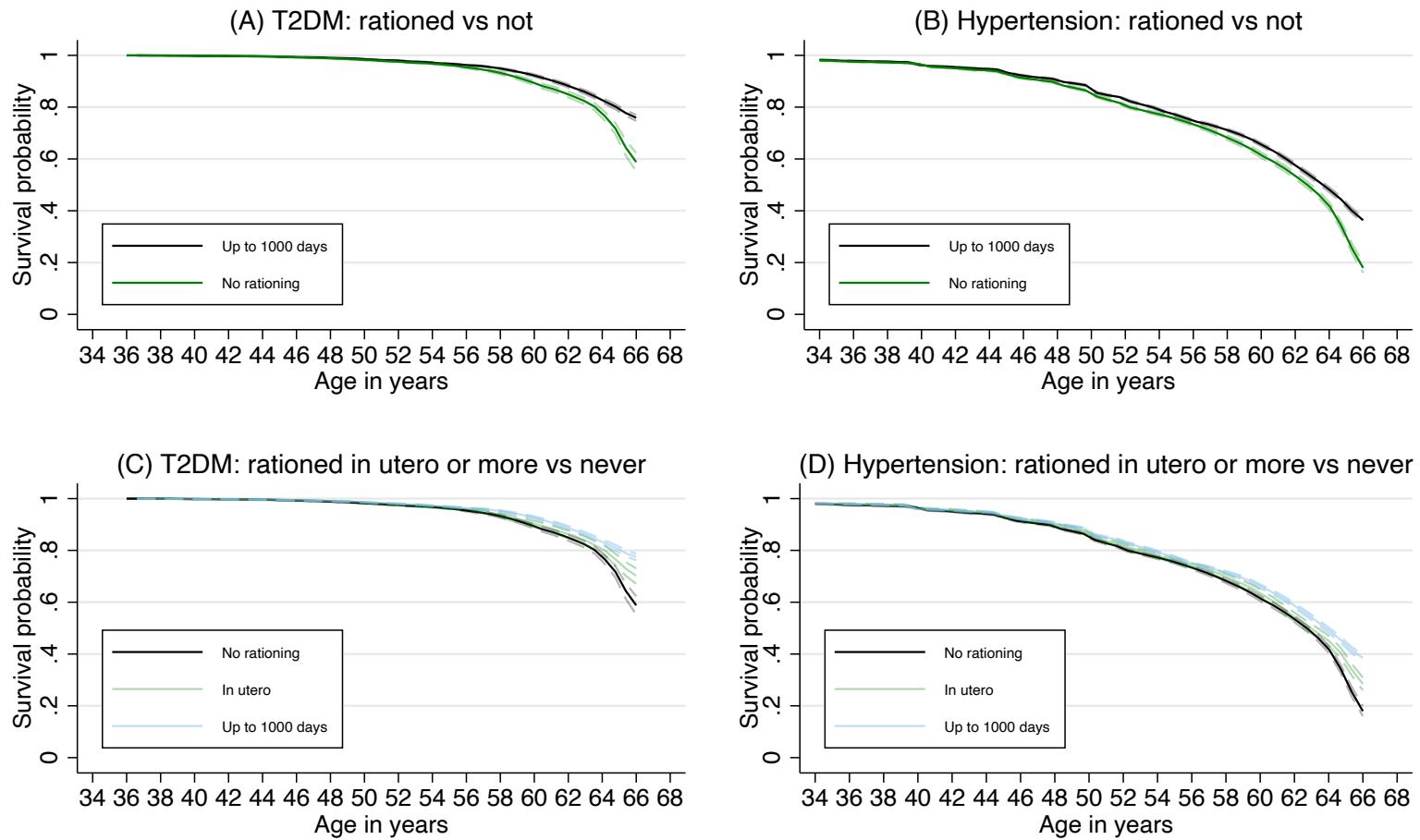
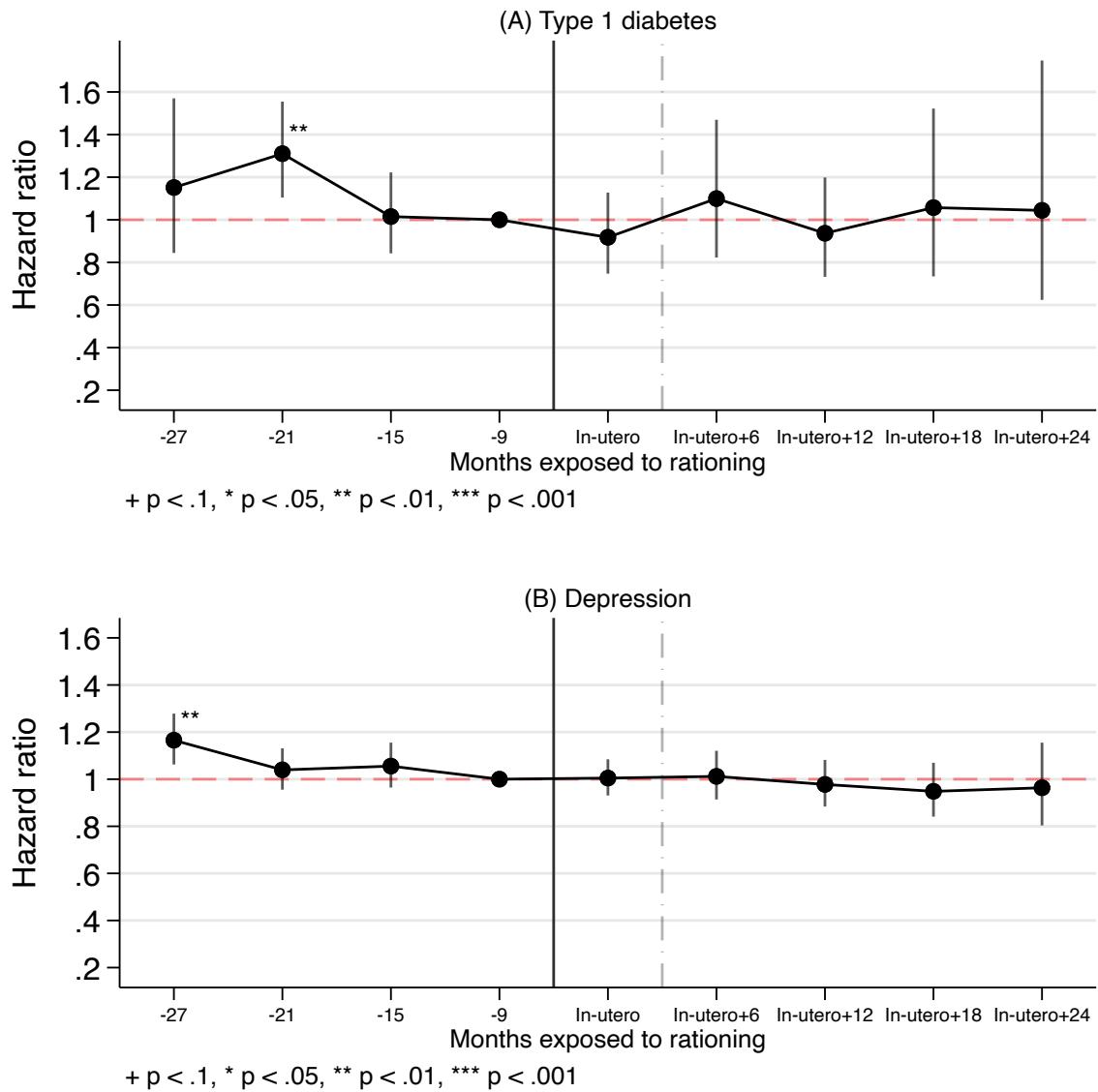


Figure S6. Survival functions for type 2 diabetes and hypertension. Survival functions show the probability of remaining free from **(A)** type 2 diabetes for those ever vs. never exposed to rationing, **(B)** hypertension for those ever vs. never exposed to rationing, **(C)** type 2 diabetes for those ever rationed in utero or more vs. never rationed, and **(D)** hypertension for those rationed in utero or more vs. never rationed. Log-rank test to test statistical differences between survival curves was employed separately for subgroups. At $p < 0.001$, survival curves were statistically significantly different between subgroups. Tables supporting these figures can be found in Dryad repository.(47)

Figure S7.



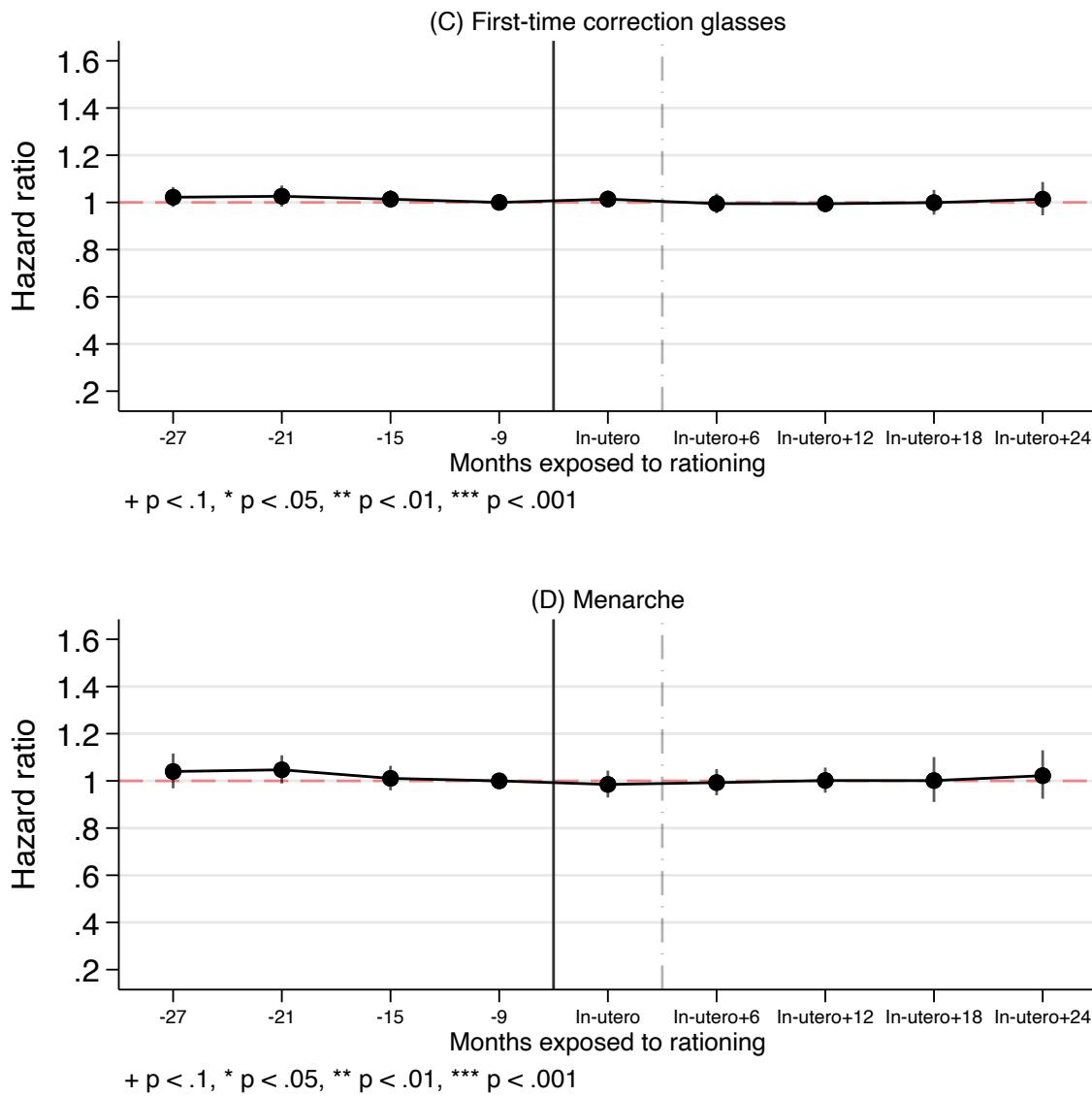


Figure S7. Hazard ratios comparing the hazard rates at various levels of rationing exposure to the hazard rate of never-rationed adults born July-December 1954 for placebo outcomes. Placebo outcomes are **(A)** Type 1 Diabetes, **(B)** Depression, **(C)** First-time correction glasses, and **(D)** Menarche. 95% confidence intervals are reported. Type 1 diabetes diagnosis is based on ICD-10 code E10 (insulin-dependent diabetes), unspecified (E14) and other specified diabetes (E13) if insulin therapy was initiated within the first year of diagnosis, and general diabetes diagnosis before age 36 without insulin treatment within the first year of diagnosis. The diagnosis of depression is restricted to instances where it is reported to have been triggered by trauma. +/*/**/*** indicates significance at p smaller than 0.1/0.05/0.01/0.001.

Figure S8.

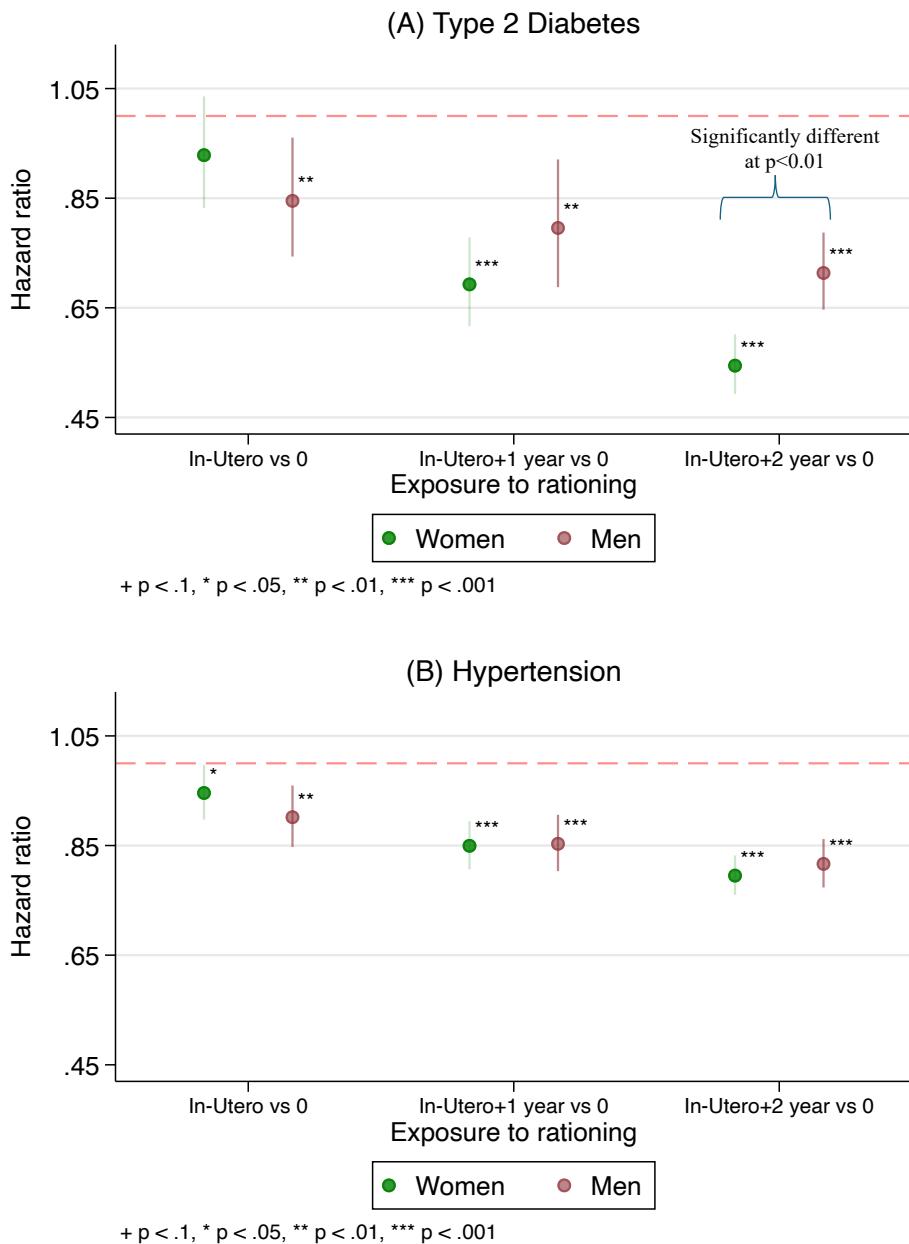


Figure S8. Hazard ratios comparing the hazard rates at various levels of rationing exposure to the hazard rate of adults never exposed to rationing, by sex. **(A)** Hazard ratios describing hazard rates for type 2 diabetes at various durations of rationing exposure relative to the hazard rate of adults never exposed to rationing. **(B)** Hazard ratios describing hazard rates for hypertension at various durations of rationing exposure relative to the hazard rate of adults never exposed to rationing. 95% confidence intervals are reported. +/*/**/*** indicates significance at p smaller than 0.1/0.05/0.01/0.001.

Figure S9.

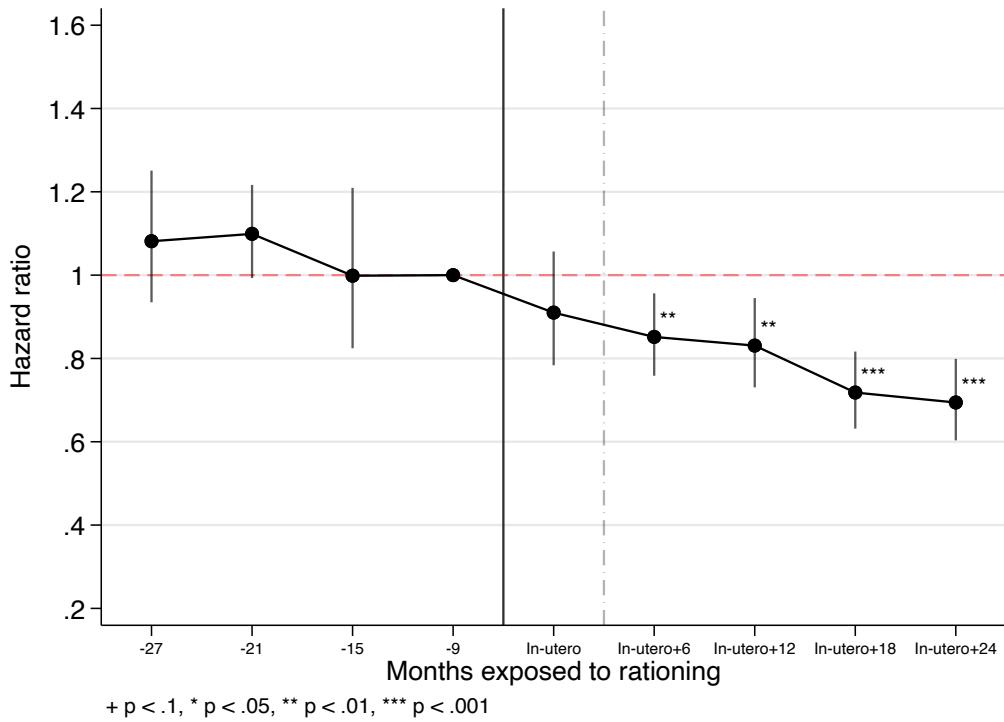
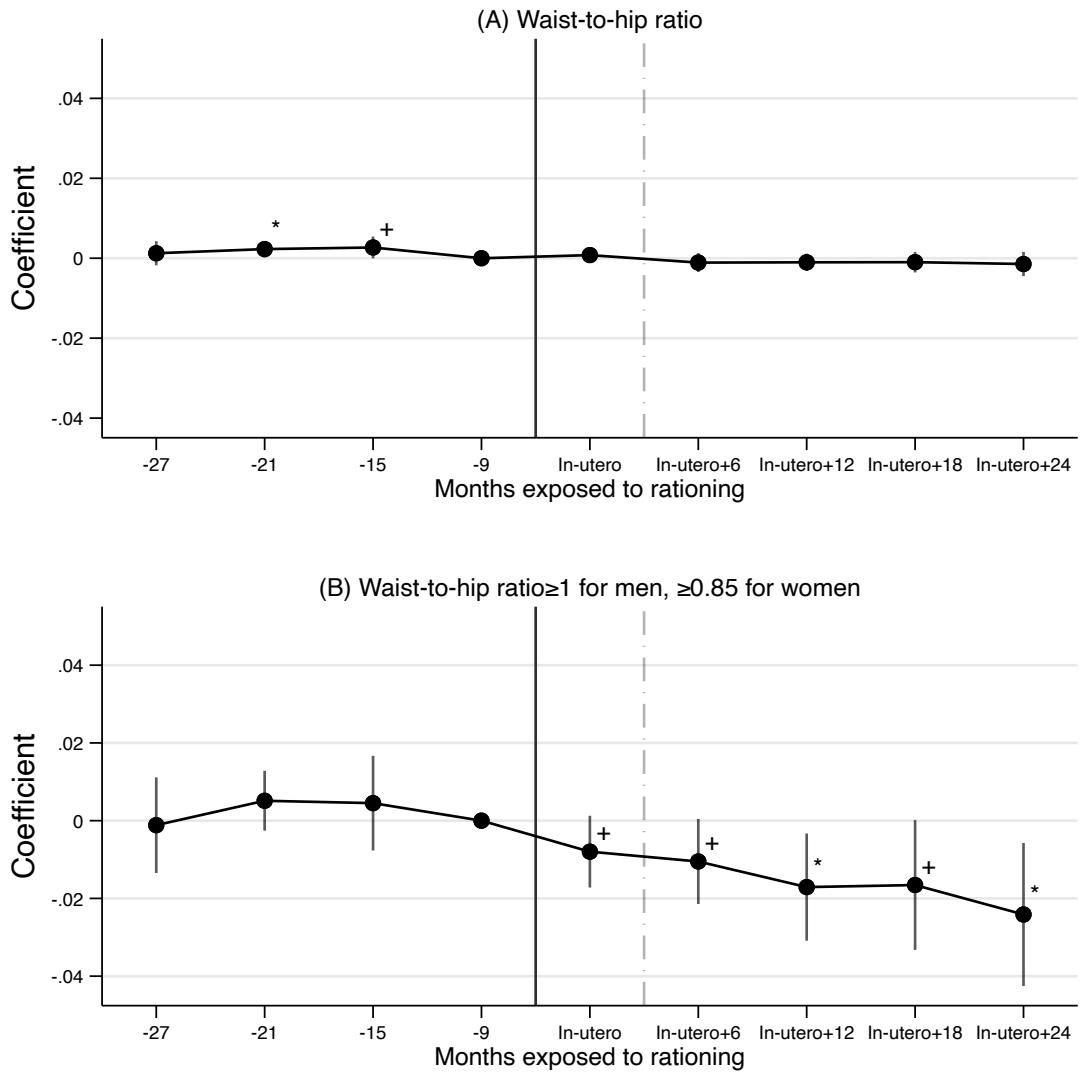


Figure S9. Hazard ratios of obesity first diagnosed in primary care comparing hazard rates at various durations of rationing exposure to the hazard rate of never-rationed adults born July–December 1954. 95% confidence intervals are reported. First diagnosis of obesity reported in primary care clinical records, using ICD-10 code E66 for obesity and E65 for local adiposity. Each point represents a hazard ratio, comparing the hazard rate of adults exposed to rationing in utero, in utero plus 6/12/18/24 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 (more than 9 months after rationing). Adults never exposed to rationing were also those born between January and June 1955 (more than 15 months after rationing), July to December 1955 (more than 21 months after rationing), and January to March 1956 (more than 27 months after rationing). The HR estimates for adults born between January 1955 and April 1956, who were never rationed, were not significantly different from the HR estimate of adults born in the reference group of July to December 1954 (at $p = 0.25$). +/*/**/** indicates significance at p smaller than 0.1/0.05/0.01/0.001.

Figure S10



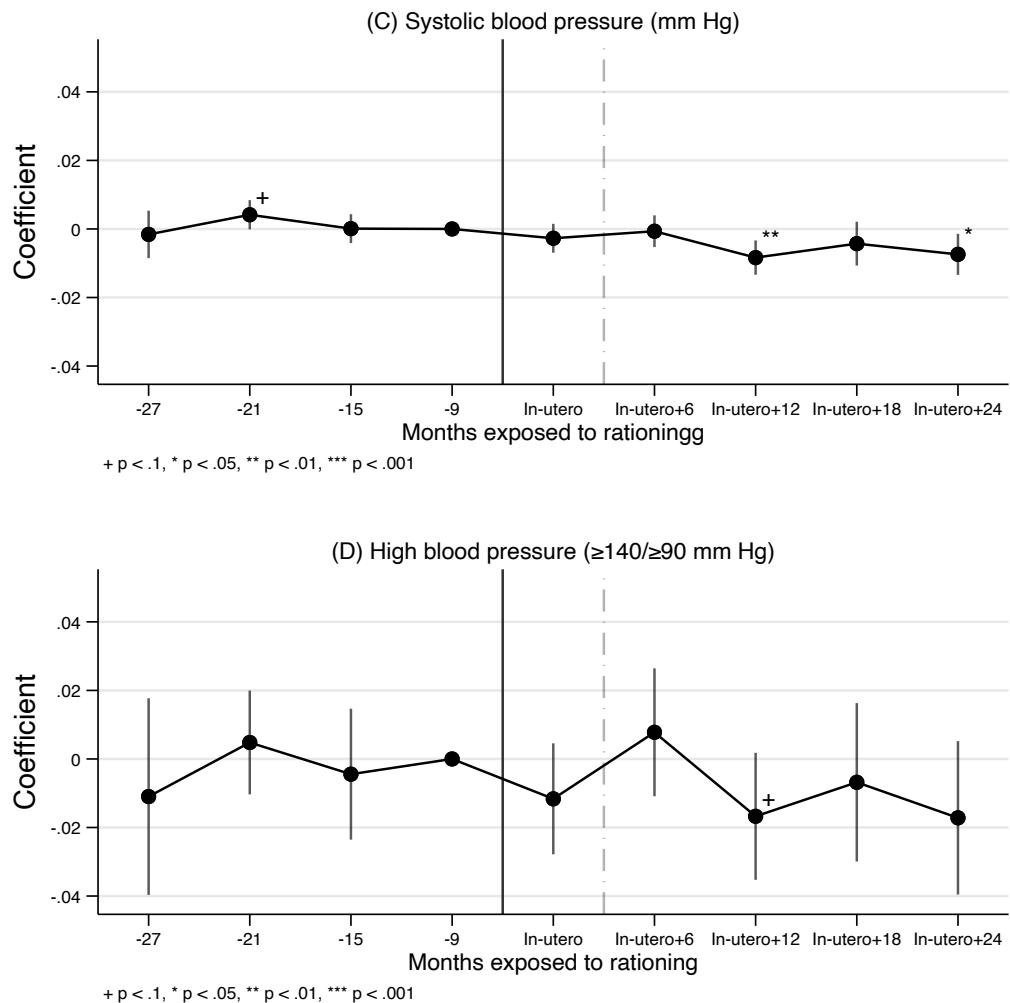


Figure S10: Outcome differences at various durations of rationing exposure relative to the outcomes of the cohort of never-rationed adults born between July and December 1954. **(A)** Differences in WHR between cohorts relative to the never-rationed adults born between July and December 1954. **(B)** Differences in high WHR, defined as $\text{WHR} \geq 1$ for men and $\text{WHR} \geq 0.85$ for women, between cohorts relative to the never-rationed adults born between July and December 1954. **(C)** Differences in systolic blood pressure (log-transformed) between cohorts relative to the never-rationed adults born between July and December 1954. **(D)** Differences in hypertension (≥ 140 mm Hg systolic or ≥ 90 mm Hg diastolic blood pressure) between cohorts relative to the never-rationed adults born between July and December 1954. In each figure, points represent outcome differences for adults exposed to rationing in utero, in utero plus 6/12/18/24 months post-birth, or not at all, relative to the reference group of never-rationed adults born between July and December 1954 (more than 9 months after rationing). Adults never exposed are also those born between January and June 1955 (more than 15 months after rationing), July to December 1955 (more than 21 months after rationing), and January to March 1956 (more than 27 months after rationing). All regressions included month of birth, birth place (England, Wales, Scotland), deciles of birthplace coordinates, self-reported sex, race (white vs. non-white), parental cardiovascular disease (hypertension, heart disease, stroke), or diabetes indicators, age, and age squared. +/*/**/*** indicates significance at p smaller than 0.1/0.05/0.01/0.001.

Figure S11.

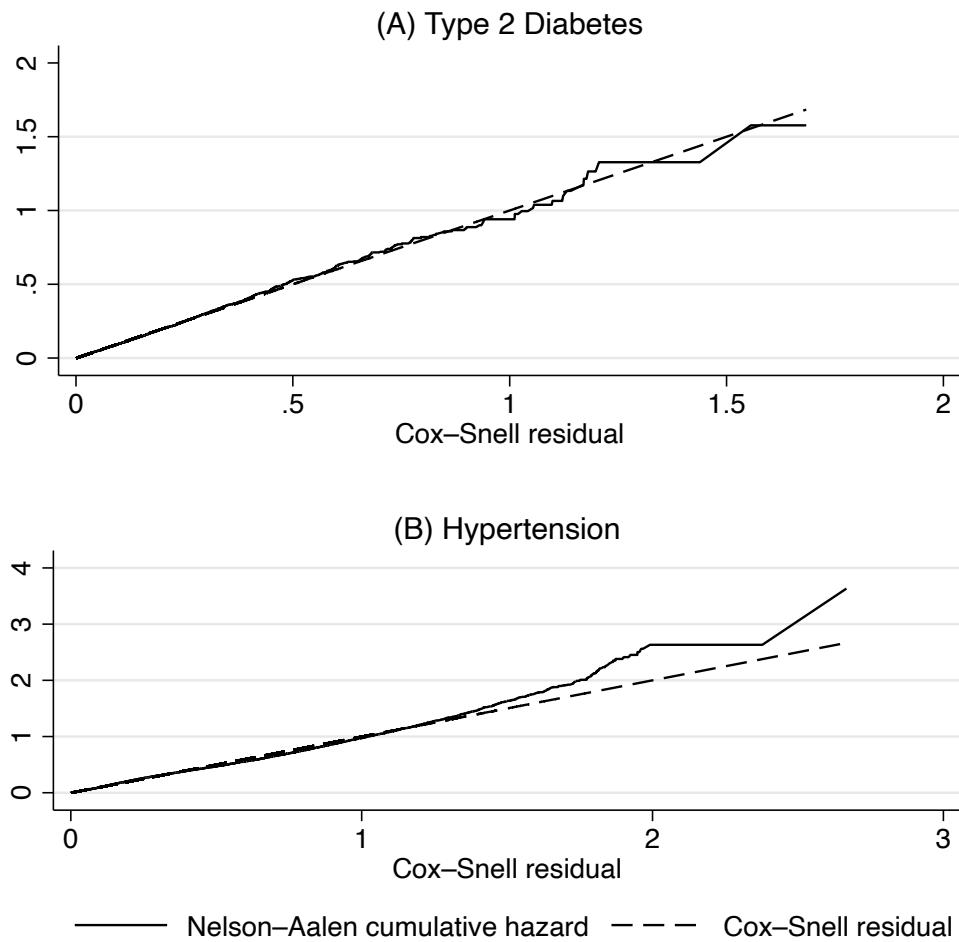


Figure S11. Goodness of fit for parametric hazard models with Gompertz baseline hazard parametrization. **(A)** Goodness of fit for parametric hazard models with Gompertz baseline hazard parametrization for type 2 diabetes. **(B)** Goodness of fit for parametric hazard models with Gompertz baseline hazard parametrization for hypertension.

Figure S12.

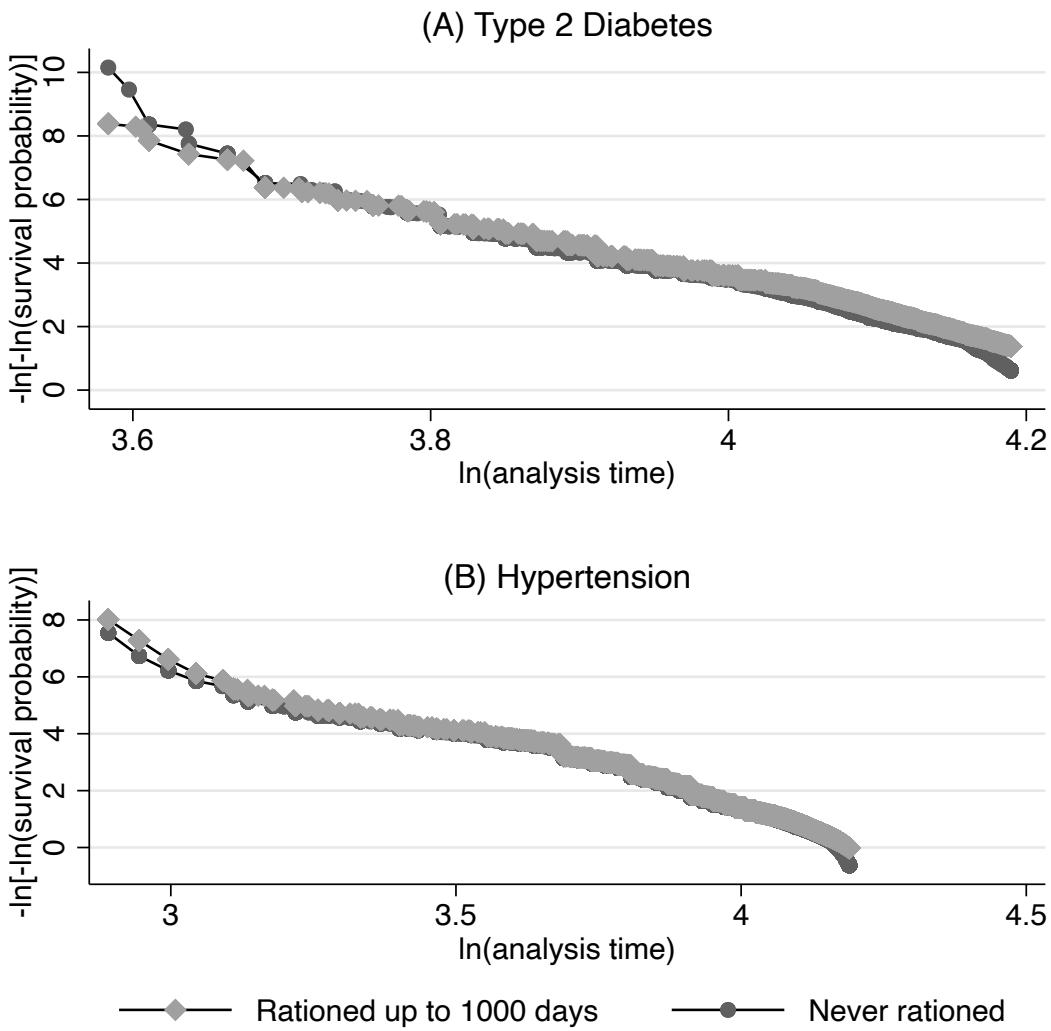


Figure S12. Testing hazards proportionality assumption between adults exposed to rationing vs. not using Cox model. **(A)** Proportionality assumption test for rationed vs. non-rationed adults for type 2 diabetes. **(B)** Proportionality assumption test for rationed vs. non-rationed adults for hypertension.

Figure S13

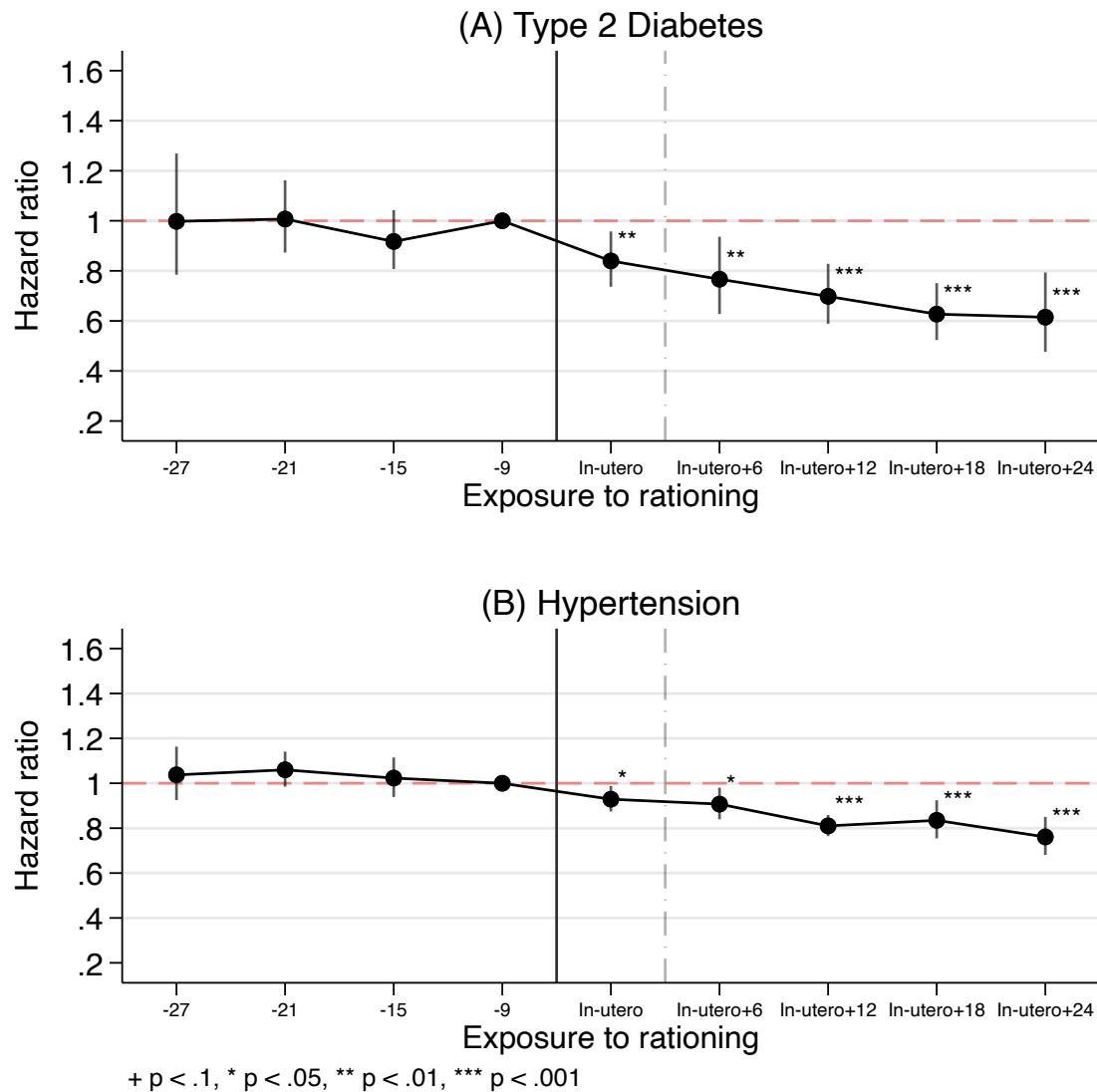


Figure S13: Hazard ratios comparing hazard rates at various durations of rationing exposure to the hazard rate of never-rationed adults born July–December 1954, adjusted for food affordability. **(A)** Hazard ratios at various durations of rationing exposure, adjusted for food affordability for type 2 diabetes. **(B)** Hazard ratios at various durations of rationing exposure, adjusted for food affordability for hypertension. Each point represents a hazard ratio, comparing the hazard rate of adults exposed to rationing in utero, in utero plus 6/12/18/24 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 (more than 9 months after rationing). Adults never exposed to rationing were also those born between January and June 1955 (more than 15 months after rationing), July to December 1955 (more than 21 months after rationing), and January to March 1956 (more than 27 months after rationing). All regressions include indicators for calendar month of birth, baseline survey year, birth place (England vs Wales vs Scotland), deciles of north and east place-of-birth coordinates, self-reported sex, race (white vs not), and parental cardiovascular disease (hypertension, heart

disease, stroke), or diabetes indicator, and real food prices adjusted for consumer price index.
+/*/**/*** indicates significance at p smaller than 0.1/0.05/0.01/0.001.

Supplementary Tables

Table S1.

Birth month	N	Percent	Cum.	EXPOSURE TO RATIONING	
1951m10	1,081	1.80	1.80	up to 24 months + in-utero	EXPOSED TO RATIONING
1951m11	1,071	1.78	3.58		
1951m12	1,168	1.94	5.52		
1952m1	1,181	1.96	7.48		
1952m2	1,162	1.93	9.41		
1952m3	1,312	2.18	11.59		
1952m4	1,195	1.99	13.58	up to 18 months + in-utero	EXPOSED TO RATIONING
1952m5	1,350	2.24	15.82		
1952m6	1,229	2.04	17.86		
1952m7	1,179	1.96	19.82		
1952m8	1,148	1.91	21.73		
1952m9	1,135	1.89	23.61		
1952m10	1,129	1.88	25.49	up to 12 months + in-utero	EXPOSED TO RATIONING
1952m11	1,033	1.72	27.21		
1952m12	1,102	1.83	29.04		
1953m1	1,155	1.92	30.96		
1953m2	1,140	1.89	32.85		
1953m3	1,238	2.06	34.91		
1953m4	1,256	2.09	36.99	up to 6 months + in-utero	EXPOSED TO RATIONING
1953m5	1,317	2.19	39.18		
1953m6	1,193	1.98	41.16		
1953m7	1,170	1.94	43.11		
1953m8	1,149	1.91	45.02		
1953m9	1,127	1.87	46.89		
1953m10	1,051	1.75	48.64	In-utero	EXPOSED TO RATIONING
1953m11	1,010	1.68	50.31		
1953m12	1,011	1.68	51.99		
1954m1	1,105	1.84	53.83		
1954m2	1,085	1.80	55.63		
1954m3	1,202	2.00	57.63		
1954m4	1,090	1.81	59.44	Conceived post-rationing	EXPOSED TO RATIONING
1954m5	1,249	2.08	61.52		
1954m6	1,132	1.88	63.40		
1954m7	1,133	1.88	65.28		
1954m8	1,117	1.86	67.14		
1954m9	1,024	1.70	68.84		
1954m10	1,047	1.74	70.58		
1954m11	962	1.60	72.18		

1954m12	1,016	1.69	73.86	
1955m1	1,032	1.71	75.58	
1955m2	997	1.66	77.24	
1955m3	1,139	1.89	79.13	
1955m4	1,075	1.79	80.91	
1955m5	1,175	1.95	82.87	
1955m6	1,110	1.84	84.71	
1955m7	1,092	1.81	86.53	
1955m8	967	1.61	88.13	
1955m9	969	1.61	89.74	
1955m10	1,006	1.67	91.41	
1955m11	976	1.62	93.04	
1955m12	1,018	1.69	94.73	
1956m1	1,015	1.69	96.41	
1956m2	973	1.62	98.03	
1956m3	1,185	1.97	100.00	
Total	60,183	100		

**NOT
EXPOSED TO
RATIONING**

Table S1. Sample distribution of births by calendar months and their exposure to sugar rationing.

Table S2.

	Type 2 Diabetes n=60,183 <i>Col 1</i>	Hypertension n=60,183 <i>Col 2</i>
Months exposed to rationing:		
-27 mths since Sept 1953	0.995	1.027
<i>SE</i>	(0.118)	(0.054)
<i>95% CI</i>	0.789 - 1.255	0.926 - 1.139
<i>p-value</i>	(0.968)	(0.613)
-21 mths since Sept 1953	1.004	1.048
<i>SE</i>	(0.059)	(0.033)
<i>95% CI</i>	0.895 - 1.126	0.986 - 1.115
<i>p-value</i>	(0.943)	(0.129)
-15 mths since Sept 1953	0.918	1.023
<i>SE</i>	(0.060)	(0.045)
<i>95% CI</i>	0.807 - 1.043	0.939 - 1.115
<i>p-value</i>	(0.187)	(0.603)
In-Utero	0.842**	0.939*
<i>SE</i>	(0.050)	(0.029)
<i>95% CI</i>	0.749 - 0.947	0.884 - 0.999
<i>p-value</i>	(0.004)	(0.046)
In-Utero through 6 months	0.771**	0.924**
<i>SE</i>	(0.071)	(0.028)
<i>95% CI</i>	0.643 - 0.924	0.872 - 0.980
<i>p-value</i>	(0.005)	(0.009)
In-Utero through 12 months	0.701***	0.822***
<i>SE</i>	(0.054)	(0.025)
<i>95% CI</i>	0.603 - 0.815	0.774 - 0.874
<i>p-value</i>	(0.000)	(0.000)
In-Utero through 18 months	0.633***	0.863***
<i>SE</i>	(0.040)	(0.026)
<i>95% CI</i>	0.559 - 0.716	0.813 - 0.916
<i>p-value</i>	(0.000)	(0.000)
In-Utero through 24 months	0.622***	0.793***
<i>SE</i>	(0.035)	(0.024)
<i>95% CI</i>	0.556 - 0.695	0.747 - 0.842
<i>p-value</i>	(0.000)	(0.000)
Sex = 1	1.947***	1.491***
<i>SE</i>	(0.066)	(0.018)
<i>95% CI</i>	1.821 - 2.081	1.457 - 1.527
<i>p-value</i>	(0.000)	(0.000)
Month of birth = 2	0.868*	0.970
<i>SE</i>	(0.059)	(0.038)
<i>95% CI</i>	0.760 - 0.992	0.899 - 1.046
<i>p-value</i>	(0.037)	(0.428)

Month of birth = 3	0.942	0.948
<i>SE</i>	(0.061)	(0.031)
<i>95% CI</i>	0.830 - 1.070	0.889 - 1.012
<i>p-value</i>	(0.357)	(0.107)
Month of birth = 4	0.853*	0.940
<i>SE</i>	(0.056)	(0.041)
<i>95% CI</i>	0.751 - 0.970	0.862 - 1.025
<i>p-value</i>	(0.015)	(0.159)
Month of birth = 5	1.029	0.954
<i>SE</i>	(0.066)	(0.032)
<i>95% CI</i>	0.907 - 1.168	0.894 - 1.018
<i>p-value</i>	(0.660)	(0.157)
Month of birth = 6	0.831	0.917*
<i>SE</i>	(0.102)	(0.037)
<i>95% CI</i>	0.654 - 1.057	0.849 - 0.992
<i>p-value</i>	(0.132)	(0.030)
Month of birth = 7	0.869	0.930*
<i>SE</i>	(0.102)	(0.032)
<i>95% CI</i>	0.690 - 1.093	0.870 - 0.995
<i>p-value</i>	(0.229)	(0.035)
Month of birth = 8	0.880	0.920
<i>SE</i>	(0.064)	(0.039)
<i>95% CI</i>	0.763 - 1.015	0.846 - 1.000
<i>p-value</i>	(0.079)	(0.050)
Month of birth = 9	1.101	0.959
<i>SE</i>	(0.088)	(0.037)
<i>95% CI</i>	0.941 - 1.287	0.889 - 1.035
<i>p-value</i>	(0.231)	(0.286)
Month of birth = 10	1.057	0.920**
<i>SE</i>	(0.063)	(0.026)
<i>95% CI</i>	0.941 - 1.187	0.870 - 0.972
<i>p-value</i>	(0.348)	(0.003)
Month of birth = 11	0.983	0.971
<i>SE</i>	(0.074)	(0.031)
<i>95% CI</i>	0.847 - 1.140	0.912 - 1.035
<i>p-value</i>	(0.816)	(0.368)
Month of birth = 12	0.972	0.953
<i>SE</i>	(0.074)	(0.031)
<i>95% CI</i>	0.837 - 1.129	0.895 - 1.015
<i>p-value</i>	(0.714)	(0.137)
1 if born in Wales	1.398***	1.175**
<i>SE</i>	(0.134)	(0.058)
<i>95% CI</i>	1.159 - 1.686	1.066 - 1.295
<i>p-value</i>	(0.000)	(0.001)
1 if born in Scotland	0.947	0.967
<i>SE</i>	(0.143)	(0.075)

<i>95% CI</i>	0.704 - 1.275	0.832 - 1.125
<i>p-value</i>	(0.722)	(0.667)
Non-white	0.949	1.049
<i>SE</i>	(0.063)	(0.031)
<i>95% CI</i>	0.833 - 1.080	0.989 - 1.112
<i>p-value</i>	(0.425)	(0.113)
High genetic risk for obesity	2.377***	1.454***
<i>SE</i>	(0.106)	(0.023)
<i>95% CI</i>	2.178 - 2.593	1.410 - 1.500
<i>p-value</i>	(0.000)	(0.000)
Parent had diabetes	2.137***	1.223***
<i>SE</i>	(0.068)	(0.024)
<i>95% CI</i>	2.008 - 2.275	1.178 - 1.270
<i>p-value</i>	(0.000)	(0.000)
Parent had CVD	0.763***	1.224***
<i>SE</i>	(0.022)	(0.019)
<i>95% CI</i>	0.720 - 0.809	1.187 - 1.263
<i>p-value</i>	(0.000)	(0.000)
Missing (indic.): parents' health	1.096	0.867***
<i>SE</i>	(0.068)	(0.019)
<i>95% CI</i>	0.970 - 1.239	0.831 - 0.905
<i>p-value</i>	(0.141)	(0.000)
Missing (indic.): coordinate	1.266	1.165
<i>SE</i>	(0.267)	(0.113)
<i>95% CI</i>	0.837 - 1.915	0.963 - 1.409
<i>p-value</i>	(0.265)	(0.116)
North coordinate – decile 1	0.887	0.933
<i>SE</i>	(0.134)	(0.064)
<i>95% CI</i>	0.660 - 1.191	0.815 - 1.068
<i>p-value</i>	(0.424)	(0.314)
North coordinate – decile 2	1.184	1.062
<i>SE</i>	(0.182)	(0.085)
<i>95% CI</i>	0.876 - 1.600	0.908 - 1.243
<i>p-value</i>	(0.271)	(0.449)
North coordinate – decile 3	1.011	0.996
<i>SE</i>	(0.156)	(0.075)
<i>95% CI</i>	0.747 - 1.369	0.858 - 1.155
<i>p-value</i>	(0.943)	(0.954)
North coordinate – decile 4	0.838	0.924
<i>SE</i>	(0.120)	(0.071)
<i>95% CI</i>	0.633 - 1.109	0.796 - 1.073
<i>p-value</i>	(0.217)	(0.302)
North coordinate – decile 5	0.934	0.980
<i>SE</i>	(0.134)	(0.073)
<i>95% CI</i>	0.705 - 1.236	0.847 - 1.134
<i>p-value</i>	(0.631)	(0.785)

North coordinate – decile 6	0.993	0.957
<i>SE</i>	(0.150)	(0.074)
<i>95% CI</i>	0.738 - 1.335	0.822 - 1.113
<i>p-value</i>	(0.962)	(0.567)
North coordinate – decile 7	0.915	1.009
<i>SE</i>	(0.139)	(0.077)
<i>95% CI</i>	0.680 - 1.231	0.869 - 1.172
<i>p-value</i>	(0.558)	(0.908)
North coordinate – decile 8	0.892	0.930
<i>SE</i>	(0.125)	(0.067)
<i>95% CI</i>	0.677 - 1.174	0.808 - 1.070
<i>p-value</i>	(0.414)	(0.309)
North coordinate – decile 9	0.779	0.881
<i>SE</i>	(0.114)	(0.060)
<i>95% CI</i>	0.586 - 1.037	0.772 - 1.007
<i>p-value</i>	(0.088)	(0.063)
East coordinate – decile 1	1.316**	1.239***
<i>SE</i>	(0.128)	(0.050)
<i>95% CI</i>	1.087 - 1.592	1.145 - 1.340
<i>p-value</i>	(0.005)	(0.000)
East coordinate – decile 2	1.211	1.170***
<i>SE</i>	(0.121)	(0.041)
<i>95% CI</i>	0.995 - 1.474	1.092 - 1.253
<i>p-value</i>	(0.056)	(0.000)
East coordinate – decile 3	1.118	1.164***
<i>SE</i>	(0.098)	(0.034)
<i>95% CI</i>	0.941 - 1.328	1.099 - 1.233
<i>p-value</i>	(0.204)	(0.000)
East coordinate – decile 4	1.119	1.169***
<i>SE</i>	(0.095)	(0.042)
<i>95% CI</i>	0.947 - 1.323	1.090 - 1.255
<i>p-value</i>	(0.185)	(0.000)
East coordinate – decile 5	1.146	1.214***
<i>SE</i>	(0.099)	(0.042)
<i>95% CI</i>	0.968 - 1.357	1.134 - 1.300
<i>p-value</i>	(0.114)	(0.000)
East coordinate – decile 6	1.139	1.230***
<i>SE</i>	(0.115)	(0.041)
<i>95% CI</i>	0.935 - 1.389	1.151 - 1.314
<i>p-value</i>	(0.197)	(0.000)
East coordinate – decile 7	1.055	1.184***
<i>SE</i>	(0.084)	(0.042)
<i>95% CI</i>	0.903 - 1.232	1.104 - 1.269
<i>p-value</i>	(0.501)	(0.000)
East coordinate – decile 8	1.139	1.212***
<i>SE</i>	(0.088)	(0.036)

<i>95% CI</i>	0.978 - 1.326	1.143 - 1.284
<i>p-value</i>	(0.093)	(0.000)
East coordinate – decile 9	0.907	1.061
<i>SE</i>	(0.063)	(0.039)
<i>95% CI</i>	0.791 - 1.040	0.987 - 1.142
<i>p-value</i>	(0.162)	(0.109)
2007 survey year FE	1.252***	1.132***
<i>SE</i>	(0.080)	(0.039)
<i>95% CI</i>	1.104 - 1.420	1.058 - 1.212
<i>p-value</i>	(0.000)	(0.000)
2008 survey year FE	1.182***	1.151***
<i>SE</i>	(0.057)	(0.026)
<i>95% CI</i>	1.075 - 1.298	1.101 - 1.203
<i>p-value</i>	(0.001)	(0.000)
2009 survey year FE	1.069	1.088***
<i>SE</i>	(0.058)	(0.022)
<i>95% CI</i>	0.961 - 1.189	1.046 - 1.131
<i>p-value</i>	(0.218)	(0.000)

Table S2. Hazard ratio estimates between rationed and non-rationed cohorts using parametric hazard models corresponding to Figure 3. Hazard ratios are expressed relative to the hazard rate for chronic disease among those born July to December 1954 and therefore never exposed to rationing. Coefficients of exposure are plotted visually in Figure 3. */**/*** indicates significance at p value smaller than 0.05/0.01/0.001.

Table S3.

	Col 1 Type 2 diabetes (n=60,183)	Col 2 Type 2 diabetes (n=60,183)	Col 3 Hyper- tension (n=60,183)	Col 4 Hyper- tension (n=60,183)
Panel A: Hazard ratio				
<i>Rationing exposure:</i>				
In-utero vs 0	0.87**		0.92**	
SE	(0.04)		(0.02)	
95% CI	0.80 - 0.94		0.88 - 0.97	
p-value	(0.001)		(0.001)	
In-utero&6 mths vs 0		0.84***		0.91***
SE		(0.04)		(0.02)
95% CI		0.77 - 0.91	<i>Diff.sig. at p<0.001</i>	0.87 - 0.95
p-value		(0.000)	<i>Diff.sig. at p<0.001</i>	(0.000)
In-utero through 24mths vs 0	0.69***	0.66***	0.83***	0.81***
SE	(0.03)	(0.03)	(0.02)	(0.02)
95% CI	0.64 - 0.76	0.62 - 0.72	0.79 - 0.86	0.78 - 0.84
p-value	(0.000)	(0.000)	(0.000)	(0.000)
Panel B: Delay in age of onset (yrs)				
<i>Rationing exposure:</i>				
In-utero vs 0	1.46**		0.54*	
SE	(0.52)		(0.24)	
95% CI	0.45 - 2.48		0.07 – 1.00	
p-value	(0.005)		(0.024)	
In-utero & 6 mths vs 0		2.38***		0.79**
SE		(0.56)		(0.24)
95% CI		1.28 - 3.47		0.33 - 1.26
p-value		(0.000)		(0.001)
In-utero through 24mths vs 0	3.47***	3.32***	1.79***	2.00***
SE	(0.49)	(0.55)	(0.23)	(0.24)
95% CI	2.50 - 4.44	2.25 - 4.39	1.34 - 2.24	1.55 - 2.46
p-value	(0.000)	(0.000)	(0.000)	(0.000)

Table S3. Hazard ratio and delay in disease onset by duration of exposure to rationing. Hazard ratios are expressed relative to the hazard rate of those never exposed to rationing (i.e., those born after July 1954). Difference in hazard ratios for those exposed in-utero only vs those exposed in-utero and beyond is significant at $p<0.001$ for both conditions. Parametric hazard models in panel A were estimated using the Gompertz distribution, though results were not sensitive to the choice of the distribution function, like Weibull. Time-to-event models in panel B assume Weibull distribution and are estimated using the stteffects, ra command in STATA. */**/*** indicates significance at p value smaller than 0.05/0.01/0.001.

Table S4

	Col 1 WHR (n=60,083)	Col 2 High WHR (≥1 M, ≥0.85 W) (n=60,083)	Col 3 Log(systolic BP) (n=56,153)	Col 4 High BP (≥140/≥90) (n=56,153)
<i>Rationing exposure:</i>				
In-utero vs 0	-0.001	-0.01*	-0.003	-0.008
SE	(0.001)	(0.004)	(0.002)	(0.007)
95% CI	-0.002 – -0.001	-0.019 - -0.001	-0.006 – -0.009	-0.021 – -0.006
p-value	(0.397)	(0.031)	(0.147)	(0.252)
In-utero & through 1 year vs 0	-0.002*	-0.016**	-0.005*	-0.001
SE	(0.001)	(0.005)	(0.002)	(0.008)
95% CI	-0.004 - -0.000	-0.027 - -0.005	-0.009 - -0.000	-0.017 – -0.013
p-value	(0.023)	(0.006)	(0.038)	(0.847)
In-utero + through 24mths vs 0	-0.002 ⁺	-0.02*	-0.006*	-0.008
SE	(0.001)	(0.008)	0.003	(0.010)
95% CI	-0.005 – -0.000	-0.038 - -0.005	-0.011 - -0.000	-0.027 – -0.011
p-value	(0.058)	(0.012)	(0.044)	(0.381)
<i>Mean for those never exposed:</i>	0.87	22.78%	136.96 mm Hg	45.21%

Table S4. Changes in obesity and blood pressure related outcomes by duration of exposure to rationing relative to those never exposed. WHR refers to waist-to-hip ratio. High WHR is an indicator for WHR ≥ 1 for males and ≥ 0.85 for women. BP refers to blood pressure (measured automatically in mm Hg). High BP is an indicator for systolic or diastolic blood pressure ≥ 140 or ≥ 90 mm Hg, respectively. All measures are obtained in the first survey round. Each regression includes age, age squared, month of birth, birthplace (England, Wales, Scotland), deciles of birthplace coordinates, self-reported sex, race (white vs. non-white), parental cardiovascular disease (hypertension, heart disease, stroke), or diabetes indicators. */**/*** indicates significance at p value smaller than 0.05/0.01/0.001.

Table S5.

Baseline hazard parametrized with a distribution:	Type 2 Diabetes		Hypertension	
	AIC	BIC	AIC	BIC
Gompertz	13551.12	14010.39	38808.92	39268.18
Weibull	13619.41	14078.67	39594.05	40053.31
Logistic	13693.70	14152.96	40718.31	41177.57
Log normal	14103.03	14562.29	44196.92	44656.18
Exponential	28206.54	28656.79	85614.20	86064.45

Table S5. Assessing the best fitted distribution using the Akaike and Bayesian Information Criterion. Based on this criterion and Figure S11, we proceeded with Gompertz distribution as our preferred one.

Table S6.

	Diabetes (n=60,183)		Hypertension (n=60,183)	
	Parametric estimation: Weibull	Semi-parametric estimation: Cox model	Parametric estimation: Weibull	Semi-parametric estimation: Cox model
<i>Comparison group born:</i>	<i>1954 m7-m12</i>	<i>1954 m7-m12</i>	<i>1954 m7-m12</i>	<i>1954 m7-m12</i>
Months exposed to rationing:	(1)	(2)	(3)	(4)
-27 mths since Sept 1953	0.996	0.965	1.019	1.016
<i>SE</i>	(0.118)	(0.115)	(0.054)	(0.052)
<i>95% CI</i>	0.790 - 1.255	0.764 - 1.219	0.919 - 1.130	0.919 - 1.124
<i>p-value</i>	(0.973)	(0.767)	(0.717)	(0.754)
-21 mts since Sept 1953	1.007	0.984	1.043	1.041
<i>SE</i>	(0.059)	(0.057)	(0.032)	(0.032)
<i>95% CI</i>	0.898 - 1.128	0.880 - 1.102	0.983 - 1.107	0.980 - 1.106
<i>p-value</i>	(0.911)	(0.785)	(0.165)	(0.188)
-15 mths since Sept 1953	0.920	0.907	1.021	1.015
<i>SE</i>	(0.059)	(0.059)	(0.044)	(0.045)
<i>95% CI</i>	0.811 - 1.044	0.799 - 1.030	0.939 - 1.110	0.931 - 1.107
<i>p-value</i>	(0.198)	(0.133)	(0.629)	(0.735)
In-Utero	0.848**	0.849**	0.945	0.938*
<i>SE</i>	(0.050)	(0.051)	(0.028)	(0.029)
<i>95% CI</i>	0.755 - 0.952	0.756 - 0.954	0.892 - 1.001	0.883 - 0.997
<i>p-value</i>	(0.005)	(0.006)	(0.054)	(0.039)
In-Utero+ 6 mths	0.778**	0.784**	0.937*	0.926**
<i>SE</i>	(0.071)	(0.074)	(0.027)	(0.028)
<i>95% CI</i>	0.650 - 0.931	0.652 - 0.943	0.885 - 0.992	0.873 - 0.982
<i>p-value</i>	(0.006)	(0.010)	(0.024)	(0.010)
In-Utero + 12 mths	0.707***	0.721***	0.836***	0.828***
<i>SE</i>	(0.054)	(0.056)	(0.025)	(0.025)
<i>95% CI</i>	0.609 - 0.820	0.619 - 0.839	0.789 - 0.886	0.780 - 0.879
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
In-Utero + 18 mths	0.637***	0.659***	0.878***	0.876***
<i>SE</i>	(0.040)	(0.043)	(0.026)	(0.027)
<i>95% CI</i>	0.563 - 0.721	0.580 - 0.748	0.828 - 0.930	0.825 - 0.930
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
In-Utero + 24 mths	0.627***	0.651***	0.812***	0.812***
<i>SE</i>	(0.035)	(0.038)	(0.023)	(0.024)
<i>95% CI</i>	0.561 - 0.700	0.581 - 0.730	0.767 - 0.859	0.766 - 0.861
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
Female = 1	1.955***	1.945***	1.498***	1.492***
<i>SE</i>	(0.066)	(0.066)	(0.018)	(0.018)
<i>95% CI</i>	1.830 - 2.089	1.819 - 2.078	1.464 - 1.533	1.457 - 1.528
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
Month of birth = 2	0.874*	0.862*	0.973	0.966
<i>SE</i>	(0.059)	(0.059)	(0.037)	(0.039)
<i>95% CI</i>	0.766 - 0.997	0.754 - 0.985	0.903 - 1.048	0.893 - 1.044
<i>p-value</i>	(0.045)	(0.029)	(0.471)	(0.381)
Month of birth = 3	0.943	0.938	0.946	0.944

<i>SE</i>	(0.060)	(0.062)	(0.031)	(0.031)
<i>95% CI</i>	0.833 - 1.068	0.825 - 1.068	0.887 - 1.009	0.886 - 1.006
<i>p-value</i>	(0.358)	(0.334)	(0.092)	(0.074)
Month of birth = 4	0.855*	0.851*	0.941	0.942
<i>SE</i>	(0.055)	(0.056)	(0.040)	(0.041)
<i>95% CI</i>	0.753 - 0.971	0.748 - 0.968	0.866 - 1.022	0.865 - 1.026
<i>p-value</i>	(0.015)	(0.014)	(0.146)	(0.172)
Month of birth = 5	1.030	1.028	0.956	0.955
<i>SE</i>	(0.065)	(0.066)	(0.030)	(0.032)
<i>95% CI</i>	0.909 - 1.166	0.906 - 1.165	0.898 - 1.017	0.895 - 1.019
<i>p-value</i>	(0.644)	(0.671)	(0.154)	(0.164)
Month of birth = 6	0.834	0.828	0.920*	0.919*
<i>SE</i>	(0.102)	(0.102)	(0.036)	(0.037)
<i>95% CI</i>	0.656 - 1.060	0.651 - 1.053	0.852 - 0.993	0.849 - 0.996
<i>p-value</i>	(0.138)	(0.124)	(0.033)	(0.038)
Month of birth = 7	0.869	0.869	0.930*	0.930*
<i>SE</i>	(0.101)	(0.102)	(0.031)	(0.032)
<i>95% CI</i>	0.692 - 1.092	0.690 - 1.094	0.871 - 0.994	0.870 - 0.995
<i>p-value</i>	(0.228)	(0.231)	(0.031)	(0.035)
Month of birth = 8	0.884	0.878	0.921*	0.919
<i>SE</i>	(0.064)	(0.063)	(0.039)	(0.040)
<i>95% CI</i>	0.767 - 1.019	0.762 - 1.011	0.849 - 1.000	0.843 - 1.001
<i>p-value</i>	(0.089)	(0.071)	(0.050)	(0.052)
Month of birth = 9	1.102	1.097	0.961	0.959
<i>SE</i>	(0.087)	(0.088)	(0.036)	(0.037)
<i>95% CI</i>	0.944 - 1.286	0.937 - 1.285	0.893 - 1.035	0.888 - 1.035
<i>p-value</i>	(0.219)	(0.248)	(0.297)	(0.284)
Month of birth = 10	1.061	1.056	0.922**	0.921**
<i>SE</i>	(0.062)	(0.063)	(0.025)	(0.026)
<i>95% CI</i>	0.946 - 1.189	0.940 - 1.188	0.875 - 0.973	0.872 - 0.973
<i>p-value</i>	(0.312)	(0.358)	(0.003)	(0.003)
Month of birth = 11	0.986	0.982	0.974	0.974
<i>SE</i>	(0.074)	(0.075)	(0.030)	(0.031)
<i>95% CI</i>	0.851 - 1.142	0.844 - 1.141	0.917 - 1.036	0.915 - 1.037
<i>p-value</i>	(0.849)	(0.808)	(0.404)	(0.416)
Month of birth = 12	0.976	0.967	0.956	0.953
<i>SE</i>	(0.073)	(0.074)	(0.029)	(0.030)
<i>95% CI</i>	0.843 - 1.131	0.832 - 1.123	0.900 - 1.015	0.895 - 1.014
<i>p-value</i>	(0.751)	(0.660)	(0.140)	(0.131)
1 if born in Wales	1.379***	1.389***	1.154**	1.165**
<i>SE</i>	(0.131)	(0.134)	(0.057)	(0.058)
<i>95% CI</i>	1.144 - 1.663	1.150 - 1.678	1.047 - 1.272	1.057 - 1.285
<i>p-value</i>	(0.001)	(0.001)	(0.004)	(0.002)
1 if born in Scotland	0.941	0.944	0.952	0.961
<i>SE</i>	(0.141)	(0.144)	(0.073)	(0.074)
<i>95% CI</i>	0.702 - 1.262	0.700 - 1.273	0.820 - 1.105	0.826 - 1.119
<i>p-value</i>	(0.684)	(0.705)	(0.518)	(0.608)
Non-white	0.946	0.952	1.047	1.051
<i>SE</i>	(0.062)	(0.063)	(0.031)	(0.031)
<i>95% CI</i>	0.832 - 1.076	0.836 - 1.084	0.988 - 1.109	0.992 - 1.115
<i>p-value</i>	(0.400)	(0.459)	(0.122)	(0.094)
High genetic risk for obesity	2.345***	2.386***	1.432***	1.452***

<i>SE</i>	(0.104)	(0.106)	(0.022)	(0.023)
<i>95% CI</i>	2.151 - 2.557	2.187 - 2.603	1.389 - 1.476	1.407 - 1.498
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
Parent had diabetes	2.149***	2.139***	1.233***	1.227***
<i>SE</i>	(0.068)	(0.068)	(0.023)	(0.024)
<i>95% CI</i>	2.019 - 2.288	2.009 - 2.276	1.188 - 1.280	1.181 - 1.275
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
Parent had CVD	0.774***	0.759***	1.238***	1.225***
<i>SE</i>	(0.023)	(0.022)	(0.019)	(0.019)
<i>95% CI</i>	0.730 - 0.820	0.717 - 0.805	1.201 - 1.277	1.187 - 1.263
<i>p-value</i>	(0.000)	(0.000)	(0.000)	(0.000)
Missing indicator: parent health	1.090	1.093	0.891***	0.896***
<i>SE</i>	(0.068)	(0.068)	(0.029)	(0.029)
<i>95% CI</i>	0.964 - 1.232	0.968 - 1.235	0.837 - 0.949	0.841 - 0.955
<i>p-value</i>	(0.168)	(0.152)	(0.000)	(0.001)
2007 survey year FE	1.258***	1.165*	1.130***	1.090*
<i>SE</i>	(0.081)	(0.077)	(0.039)	(0.038)
<i>95% CI</i>	1.110 - 1.426	1.024 - 1.326	1.057 - 1.208	1.019 - 1.167
<i>p-value</i>	(0.000)	(0.021)	(0.000)	(0.013)
2008 survey year FE	1.185***	1.119*	1.147***	1.114***
<i>SE</i>	(0.056)	(0.054)	(0.025)	(0.025)
<i>95% CI</i>	1.080 - 1.301	1.018 - 1.229	1.099 - 1.197	1.067 - 1.164
<i>p-value</i>	(0.000)	(0.019)	(0.000)	(0.000)
2009 survey year FE	1.071	1.047	1.081***	1.069***
<i>SE</i>	(0.058)	(0.057)	(0.021)	(0.021)
<i>95% CI</i>	0.963 - 1.190	0.940 - 1.165	1.040 - 1.124	1.029 - 1.110
<i>p-value</i>	(0.205)	(0.406)	(0.000)	(0.001)

Table S6. Alternative specifications estimating hazard ratios for disease between rationed and non-rationed adults. Hazard ratios for adults exposed to rationing in-utero, in-utero + 6/12/18/24 months after birth or not at all) are expressed relative to the hazard rate of the first cohort of adults never exposed to rationing (born between July and December 1954). Cohorts of adults never exposed to rationing were those born between July and December 1954 (reference group), January to June 1955 (more than 15 months after rationing), July 1955 to December 1955 (more than 21 months after rationing), and after through March 1956 (more than 27 months after rationing). Indicators for birth coordinates in the east and north directions are included among the same list of controls presented in tables above but are omitted from this table for the sake of brevity. */**/*** indicates significance at p-value smaller than 0.05/0.01/0.001.

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