Intergenerational Nutrition Patterns and How to Break Them *

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First Version: October 2022
This Version: February 9, 2023

Abstract

Budget limitations cannot explain why poorer households eat unhealthier. Instead, I study how your family shapes your diet with unique grocery transactions and administrative records and find strong intergenerational persistence of food habits. Then, I identify the distributional effects of life-changing family events on eating habits (namely, the birth of children and the death of parents). The birth of a first child increases fruit and vegetable intake by one percentage point. The death of a parent increases consumption by four percentage points, but only for high-income households. Hence, high-income households assimilate health-related information shocks better, enforcing dietary patterns over generations.

Keywords: consumption inequality, intergenerational mobility, health behaviors
JEL-codes: D15, D83, I12, J12, L14.

*I thank Martina Pons and Maximilian von Ehrlich for all their invaluable help. I thank Dino Collalti, Lukas Hauck, Marcel Henkel, and Pierre Magontier for their critical thoughts and suggestions.

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1 Introduction

The widespread consumption of an unhealthy diet is one of the main global health challenges, shortening quality-adjusted life years and generating high economic costs.[1] Accordingly, the Sustainable Development Goals aim to reduce the mortality rate of cancer, diabetes, and cardiovascular diseases by one-third. Yet, no world region currently meets the recommendations for a healthy diet, adult obesity, or salt intake, and an improvement in diet could reduce healthcare spending and generate a labor market income of 5.7 trillion USD annually by 2030 (Global Nutrition Report, 2021). Appropriate policies are essential to achieve this goal, and as individual household decisions ultimately determine dietary choices, policymakers must understand the factors shaping individual consumption behavior.

But diet is very heterogeneous. Allcott et al. (2019a) document considerable variation in dietary choices within the United States. They find that lower-income households consume an unhealthier diet, which a lack of access to healthy food cannot explain. Instead, two hypotheses could account for this observation: the direct lack of income or social environments. The former constrains your food choices if healthy products cost more per calorie. Budget limitations may be relevant for the United States, where a healthy diet is too expensive for 1.5% of the population (FAO, 2020). However, they cannot explain nutritional inequality in a small and prosperous country like Switzerland, where less than 0.1% cannot afford a healthy diet. Yet, Figure 1 displays the substantial difference in healthy food intake along the Swiss income distribution. Low-income households allocate 17% of their food expenditures to fruits and vegetables. This share increases continuously over the distribution to 22% for the best-earning households.[2] Hence, budget limitations cannot explain nutritional inequality.

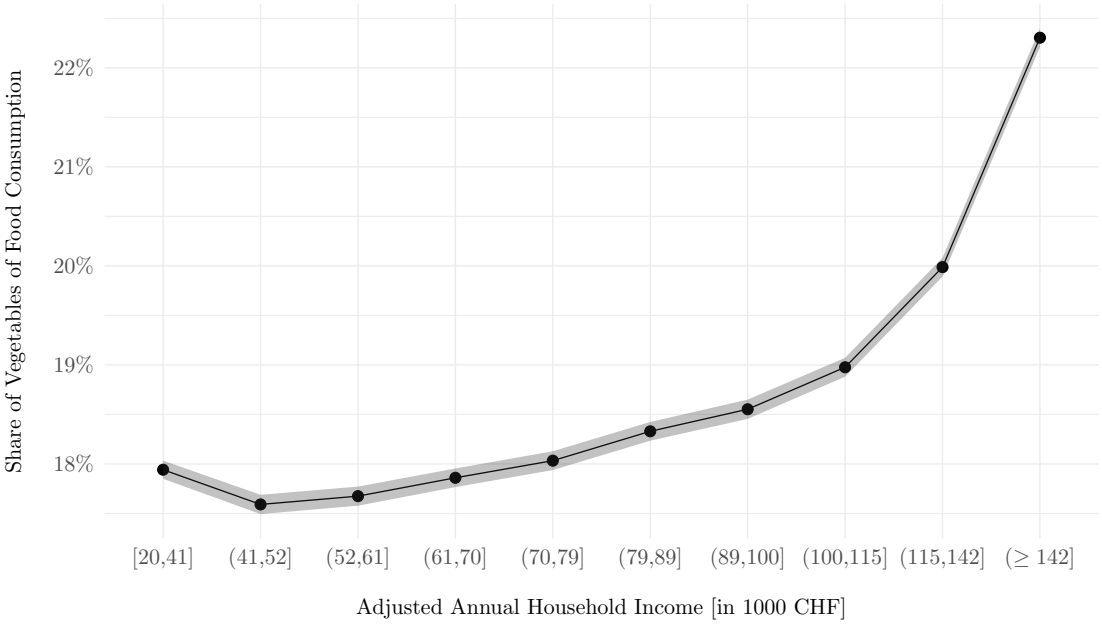
In this paper, I study the alternative hypothesis that your social environment shapes your dietary behavior at an early age. I analyze the intergenerational persistence of diet in Switzerland and how life-changing events break these patterns. Switzerland is an insightful case to study consumption, as income inequality is low and everyone has sufficient access to healthy food. This setting allows me to study if poorer households assimilate health information shocks within their social network differently and how they respond through their nutritional choices.

I achieve this by exploiting unique consumption data from the largest Swiss retailer, Migros. The data tracks the consumption behavior of 3 million Swiss households over time and provides information about the product groups purchased (fresh produce, convenience products, meat, etc.) in 1.5 billion daily transactions between 2019 and 2020. The share of fresh fruits and vegetables in a diet approximates its healthiness. I argue that this is a good proxy because of three reasons. First, Allcott et al. (2019a) report a high correlation between their health index and fruits, vegetables, and greens of 0.57, 0.41, and 0.47, respectively. Second, a diet low in

[1] 22% of all global deaths in 2017 were attributable to an inappropriate nutritional intake, mainly due to cardiovascular diseases. The leading causes are too high salt intake in high-income regions and a diet low in whole grains and fruit in low-income countries (Afshin et al., 2019).

[2] This pattern is also consistent with variation across space. Figure A1 shows that vegetable consumption strongly varies across space, with the highest rates in urban and higher-income areas.

Figure 1: Nutritional Inequality



Notes: The figure shows regression results for the share of total food expenditures at Migros for fruit and vegetables on income. Annual gross household labor market income in 2020 is adjusted for the square root of household size and binned into deciles. Consumption data is averaged over all periods. Confidence bands are on the 95% level.

fruits and vegetables is the third and fifth most frequent reason for nutrition-related mortality and loss in disability-adjusted life-years (DALYs) listed in Afshin et al. (2019). Third, the proxy is transparent and objective as it requires no weighting of different nutrients or products. I complement the consumption data with administrative data from the Federal Statistical Office, adding individual-level labor market income, household characteristics, and family linkages for all Swiss citizens. The registry of death records, including the final cause of passing and underlying health conditions, links customers to their deceased relatives.

The first part of the paper documents the intergenerational persistence of diet. Our diet strongly correlates with our parents', indicating a long-lasting persistence of food patterns across generations. Also, low-income households are more often trapped in an unhealthy diet. This means they are less likely to eat better than their parents, which is also true for households living geographically close to their parents. Looking at cohabiting couples, nutrition is stronger linked to the parents of the female and second-earning partners.

In the second part, I show that income raises have no positive effect on diet. Instead, I estimate the causal effect of life-changing events on fresh fruit and vegetable consumption, providing evidence for the potential to change intergenerational patterns. I consider the birth of a firstborn child, and a parent's death due to an unexpected stroke or heart attack.[3] I identify the effects

[3]In this context, an *unexpectedly* deceased person had no underlying health conditions that likely contributed to the death.

of these shocks on the nutritional expenses of households by comparing them to households that experience the same shock later. Applying a changes-in-changes model allows me to estimate the quantile treatment effect along the distribution of vegetable consumption. I look at the entire distribution because two channels may particularly affect individuals at the tails: households that initially eat few vegetables have the highest gains of a behavioral change, while the ones already eating healthily may assimilate shocks better.

I find that after the death of a parent, affluent households consume four percentage points more fruits and vegetables, but low-income households do not react. To pin down the underlying mechanisms, I analyze the response to the death of a step-parent. The missing genetic relation separates a learning mechanism about a potential health predisposition from a salience effect that temporarily raises awareness. I find no effect for step-parents, indicating a dominating learning effect. The birth of a first child increases produce intake by one percentage point, independent of income. Two channels may explain this: either the birth induces parents to collect diet-related information for their child, which spill over to themselves, or they value their health higher due to their new responsibility. To isolate these channels, I look at the second child's birth, where I expect no additional information collection. As I find no effect, I argue that the information channel is the driving factor. These results provide policymakers with important information for targeted information campaigns to stimulate healthy eating and reduce nutritional inequality. Additional results extend the social network from the family to neighbors. Moving to an unhealthy neighborhood reduces the quality of your diet, while a healthy surrounding has no effect. This shows that the role of social networks for diet extends beyond your close family, while the exact mechanisms, in this case, are unclear.

This paper relates to several strands of the literature. First, I contribute to intergenerational mobility. Arguably, living in a society with equal opportunities is desired, which Lara and Shores (2022) quantify by calculating the marginal willingness to pay for income equality. Yet, Chetty et al. (2014, 2016, 2020), and Chetty and Hendren (2018) document persistent patterns in income (non-)mobility across the United States, with strong spatial variation and disproportional disadvantages for non-white groups. Chuard and Grassi (2020) follow their approach for Switzerland and detect easier mobility in income between generations compared to the United States. So far, most papers focus on income or wealth. An exception is Waldkirch (2004), who uses PSID survey data to show a positive intergenerational correlation in food consumption after controlling for income.

Second, this paper connects to the recent advances on diet-related health and mortality inequalities. Deryugina and Molitor (2021) and Couillard et al. (2021) document extensively the pronounced geographic differences in life expectancy between US counties, and lifestyle-related survivability evolved in favor of the rich (Dahl et al., 2021). One determinant of this divergence may be inequality in nutrition, as low-income households typically eat unhealthier (Allcott et al., 2019a). These dietary patterns are highly persistent: disease diagnoses or household shocks may only lead to limited improvements (Oster, 2018; Hut and Oster, 2022) and migrants often adapt

only after decades to their new surroundings.[4] Alternatively, active policy interventions or individual shocks may help to improve diets, but positive findings are scarce. Potential policies include, among others, store openings in food deserts (Allcott et al. 2019a), subsidies (Hastings et al. 2021; Goldin et al. 2022), food labels (Barahona et al. 2021; Araya et al. 2018; Cook et al. 2005), sin taxes (Allcott et al. 2019b; Dubois et al. 2020; Aguilar et al. 2021), carbon pricing of nutrition (Springmann et al., 2018), or school-food programs (Berry et al., 2021; Handbury and Moshary, 2021).

Third, I contribute to the effect of social networks on individual consumption behavior. Chetty et al. (2022a,b) use Facebook data to show that the share of high-income friends strongly correlates with upward mobility for low-income individuals. Regarding consumption, De Giorgi et al. (2020) use Danish employer-employee data to analyze how peers affect our consumption. But there is also a focus on the role of social networks for health. Fadlon and Nielsen (2019, 2021) estimate the effect of health shocks on family members' behavior. They find a persistent increase in preventive care consumption that even extends to coworkers and identify learning about one's own health and a salience channel as the main drivers.

This paper is structured as follows. Section 2 introduces the data and Section 3 delineates the patterns of intergenerational mobility and persistence in diet. Section 4.1 describes the empirical identification for the diet-related shocks and Section 4.2 provides the associated results. Section 5 concludes.

2 Data

I combine unique consumption data with administrative data on a spatial resolution of 100 × 100 m. The main ingredient for this paper are customer-store-linked grocery expenditures collected through the loyalty program of the largest Swiss retailer, Migros. The program allows participating households to record their expenditures for exclusive discounts. It is Switzerland's most successful loyalty program, capturing 79% of total Migros sales. Its 3 million registered households (85% of the population) make it also the most inclusive (GfK, 2021). I focus on in-store expenditures for food, where Migros' 621 supermarkets achieved a market share of 32.7% in 2020. Importantly, Migros charges the same prices throughout the country, independently of local purchasing power, wages, and costs. Stores of similar size also generally offer a similar assortment of goods, except for local products.

I use the universe of 1.5 billion daily transaction records in this program for the period 2019-2020. The data groups individual bar codes into 41 broader categories, including the food categories *fruit and vegetables*, *meat and fish*, *milk products*, *convenience*, and *other food*. Household characteristics include the location of the residence on the 100×100m grid, the cardholder's

[4] See Hut (2020) and Hut and Oster (2022) for the United States and Atkin (2013, 2016) for India, where individuals even reduce caloric intake to stick to their original diet.

Table 1: Household Summary Statistics

	Matched Data					Population				
Variable	Mean	SD	p1	p50	p99	Mean	SD	p1	p50	p99
Household Head										
Age	55.4	18.3	21	55	92	47.6	22.3	1	49	90
Female	0.65	0.48	0	1	1	0.51	0.5	0	1	1
Married	0.56	0.5	0	1	1	0.36	0.48	0	0	1
Swiss	0.86	0.35	0	1	1	0.76	0.43	0	1	1
Household										
Size	3.1	9.6	1	2	8	2.2	3.5	1	2	6
Income total	132	149	3	115	512	116	136	3	97	461
Income p.c. adjusted	78	76	2	70	275	77	83	2	68	285
Geographic regions										
Urban	0.17	0.37	0	0	1	0.31	0.46	0	0	1
German	0.7	0.46	0	1	1	0.7	0.46	0	1	1
French	0.23	0.42	0	0	1	0.23	0.42	0	0	1
Italian	0.07	0.25	0	0	1	0.07	0.25	0	0	1
Food Consumption										
Expenditures	204	182	10	148	831					
% Fruit & Vegetables	0.21	0.12	0.04	0.19	0.6					
% Meat & Fish	0.29	0.12	0.06	0.28	0.65					
% Convenience	0.2	0.12	0.04	0.17	0.64					
Observations	912'780					3'888'413				

Notes: The table shows summary statistics. I compare characteristics of the final data, matching transaction and administrative data, to the entire Swiss population in the administrative data.

age, gender, and household type.[5] I disregard refunds and those above 2'000 CHF, as they are likely professional customers. I aggregate the individual shopping trips to monthly baskets and calculate the food expenditure share of each category. The average Swiss one-person household spends around 300 CHF per month on food.[6] Thus, I restrict my analysis to frequent consumers with average monthly expenditures of at least 100 CHF. The average household then spends 204 CHF on food products per month, with a share of 21.4% for fruits and vegetables and 29.0% for meat and fish (see Table 1).

I enrich this information with individual-level administrative records for the entire Swiss population (roughly 8 million people). The Population and Households Statistics (STATPOP) includes basic characteristics like gender, age, or marital status and links individuals to house-

[5]These household types include the categories *small households*, *young families*, *established families*, *golden agers*, and *pensioners*.

[6]575 CHF for couples without children, 825 CHF for couples with children (BFS, 2022).

holds, spouses, and family trees over time.[7] Hence, I can infer from this the birth of newborn children. The Old-Age and Survivors Insurance (AHV) adds gross labor market income and contribution periods for every citizen from tax records.[8] This allows the identification of monthly changes in income and unemployment periods. Finally, the Statistics on natural population movements (BEVNAT) feature all fatalities, including the ICD-10 codes of disease causes and underlying health conditions for the period of interest. Among the 360'000 individuals who died between 2016 and 2020, 17'412 died of a stroke and 11'790 of a sudden heart attack. Of these, 2'156 died suddenly without any underlying health conditions.[9]

I combine consumers and citizens by identifying unique combinations of households using their grid cell, age, gender, and household form. This yields 1.1 million unique consumer-citizen combinations, which accounts for 28% of Swiss households and 37% of regular customers. Table 1 compares summary statistics for the final data set to the entire Swiss population. The household head denominates the card owner in the matched data and the oldest person in the population. The average household head is 55.4 years old, lives in a household of 3.1 inhabitants with a total household income of 132'000 CHF or 78'000 CHF per person (adjusted by the square root of household size).[10] 65% of the matched card owners are female, 56% are married and 86% have a Swiss passport. Further, 70% live in a German-speaking municipality, compared to 24% and 7% in French- and Italian-speaking municipalities, respectively. Yet, fewer consumers and residents can be uniquely matched in dense urban areas. Only 17% of matched households live in dense urban areas, compared to 31% in the entire population.[11] Therefore, my data links grocery consumption to 41% of all residents in the canton of Appenzell Innerrhoden, but only 8% for the case of Basel and 11% for Geneva. Overall, the final data slightly overrepresents older Swiss families in less-dense areas.

3 The Intergenerational Transmission of Diet

In the first part of this paper, I describe the intergenerational persistence of diet by linking individuals to their parents and comparing their diets. To do so, I identify pairs of children and

[7] Anonymized pseudo-identifiers for parents and children are collected in the INFOSTAR register. This register was introduced in 2005 and contains 99.8% of all Swiss residents and 45.5% of the foreign residents in Switzerland. Missing individuals had either never lived in Switzerland or died before 2005. The intergenerational links are then available if children or their parents have changed their civil status since the 1990s (wedding, divorce, birth). Hence, the STATPOP data has the following coverage of family linkages: 98.8% for Swiss under 20 years old (52.7% for foreigners), 84% for Swiss under 60 years old, 22% for those above 60.

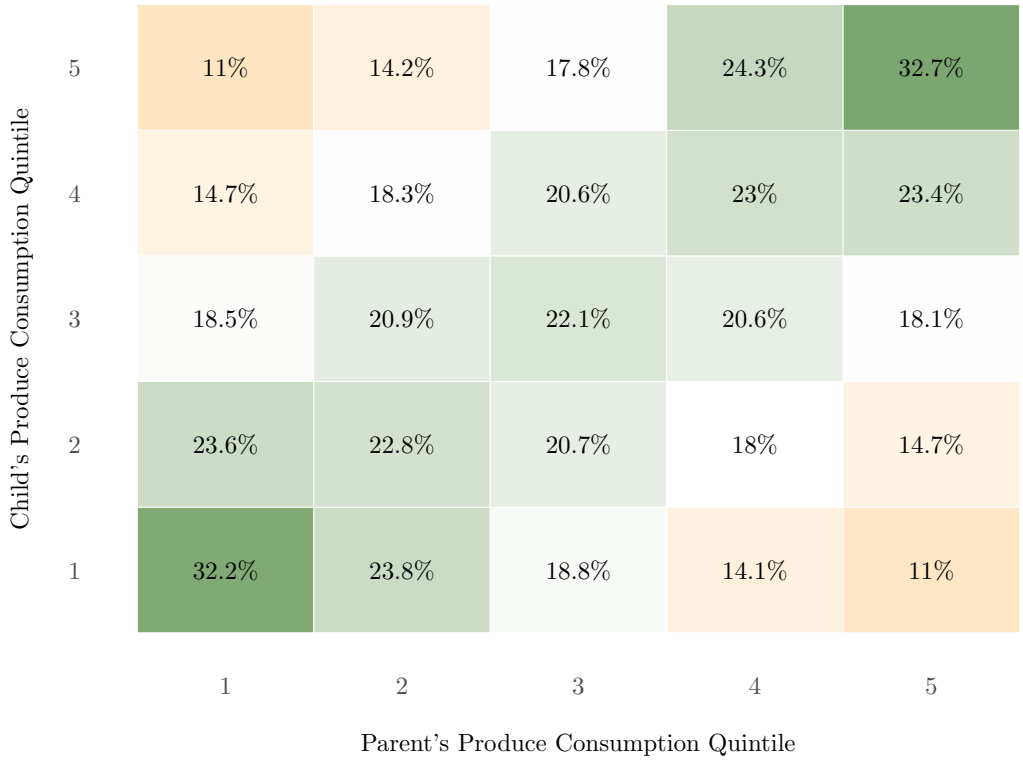
[8] This includes also official awards, gifts, and boni. Everyone except for workers younger than 25 with a gross labor market income below 750 CHF is recorded.

[9] The ten leading causes of decease are mental health (including dementia and Alzheimer's), ischaemic heart diseases, stroke, diseases of the nervous systems, lung cancer, symptomatic heart diseases, hypertension, heart attack, other heart diseases, and Covid-19.

[10] I use the OECD's equivalence scales to adjust total household income for household size. Thus, the square-root-scale adjusts income as follows: $adj.income = \frac{income}{\sqrt{HHsize}}$.

[11] As the observed characteristics used in the matching process simply fit to more individuals living in a denser area.

Figure 2: Intergenerational Diet



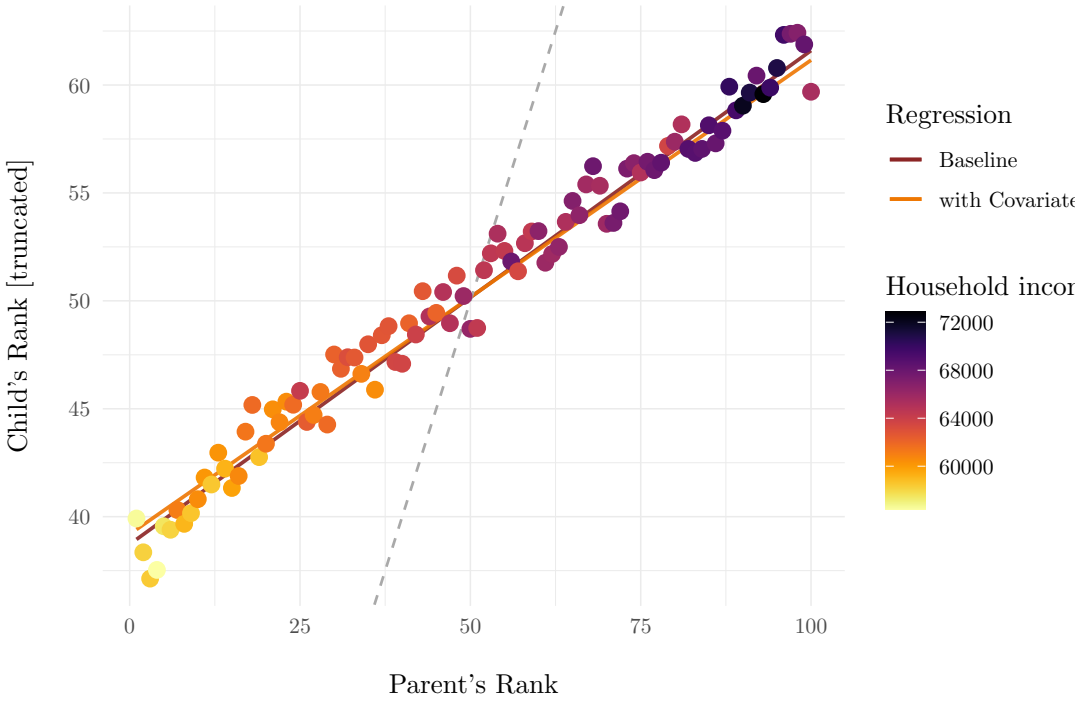
Notes: The figure shows the transition matrix for all households that could uniquely be matched to their parents in both data sets. Quintiles are then plotted against each other for children and parents.

parents that are both regular customers at the retailer Migros, but live in separate households. This results in roughly 108'000 matched intergenerational pairs. For each household, I calculate the average monthly share of fresh produce consumption 2019-2020 and calculate its rank in the entire population. Then, I plot children against their parents in the form of transition matrices between quintiles.

To begin with, Figure 2 shows the unconditional results for intergenerational mobility in diet across all households. There is strong persistence — most children are in the same quintile as their parents. For example, 32.2% of children growing up in a household with the lowest vegetable intake remain in the lowest quintile, while only 11% move to the highest. On the other hand, if an individual grows up with a healthy diet, she stays at the top in 32.7% of the cases. A Q1 to Q5 transition, happens in 11% of the cases.

One may challenge this result and suggest that income differences mainly drive this pattern. I provide three arguments against this. First, the persistence in diet outpasses previous results for income mobility in Switzerland. For example, Chuard and Grassi (2020) calculate values of 23.66%, 30.30%, and 11.87% for the Q1Q1, Q5Q5, and Q1Q5 cells, respectively. Hence, persistence in the lowest quintile is 8.6 percentage points higher for diet than for income (Figure A2 shows the difference for the entire matrix).
Second, Figure 3 plots the children's ranks in the vegetable distribution against their parents' ranks. The corresponding rank-rank slope (RSS) is then the estimated slope-coefficient of the

Figure 3: Rank-Rank Regressions



Notes: The figure plots the parents' ranks in the vegetable consumption distribution against the children's ranks. The color indicates average household income adjusted by the square root of household size. The dashed line shows the 45-degree line as the y-axis is cut. The rank-rank model regresses the child's rank on the parent's rank (slope: 21.9). The residual model first regresses the child's rank on her income and then the residuals on the parents' rank (slope: 22.9).

following regression:

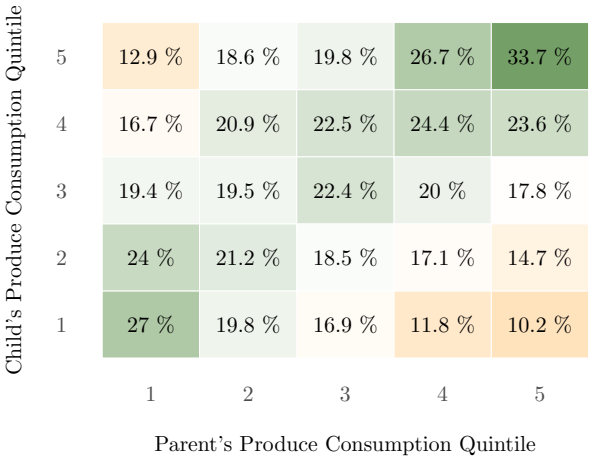
$$R_c = \delta + \beta R_p + \epsilon, \tag{1}$$

where R_c is the child's rank, and R_p is the parents' rank. To eliminate the income channel, I run an alternative specification, where I regress R_c on a measure of income in the first step and regress the residuals on R_p in the second step.[12] Both approaches lead to a similar RSS of 0.22 and 0.23, compared to the RSS for income of 0.14 in Chuard and Grassi (2020). Therefore, the results are robust to controlling for income.

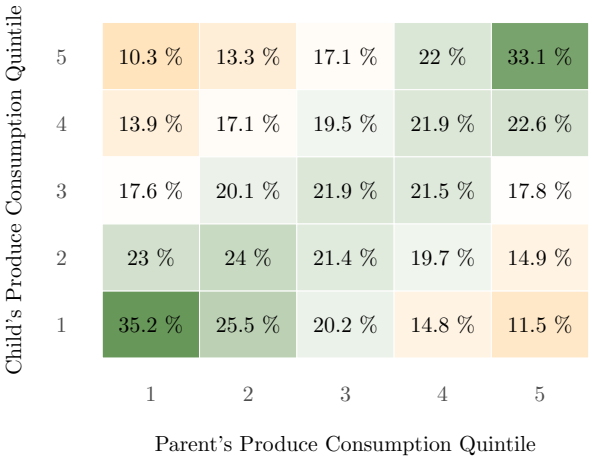
Third, I display the transition matrices separately for low- and high-income households and the absolute difference between the transition probabilities (Figure 4). The two figures show a similar pattern. Thus, my findings seem not driven by one of the two groups. Nonetheless, high-income households are likelier to "move up" - meaning, following a better diet than their parents - while poor households are often trapped at the bottom. For example, the share of low-income children staying in the Q1Q1-cell is 8.2 percentage points higher.

[12] Table A 1 shows the regression results for different measures of income. First, I add parents' income in the first step. Second, I use the average income within 500 m instead of individual income. This provides a broader measure of socio-economic status based on a household's neighborhood, and thus also includes retired parents. The RSS remains stable across all specifications.

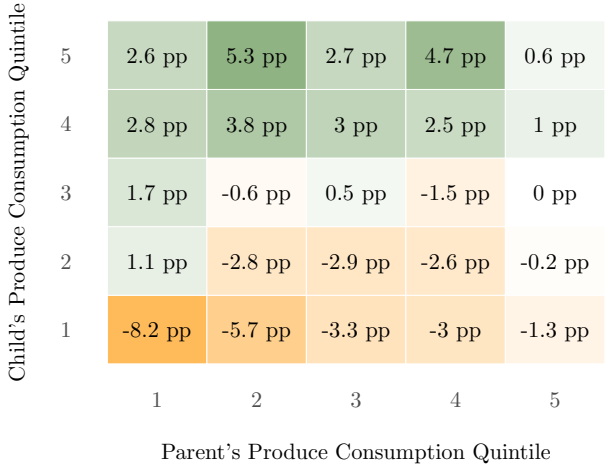
Figure 4: Intergenerational Diet: The role of income



(a) High-income Children



(b) Low-income Children



(c) High-/low-income difference

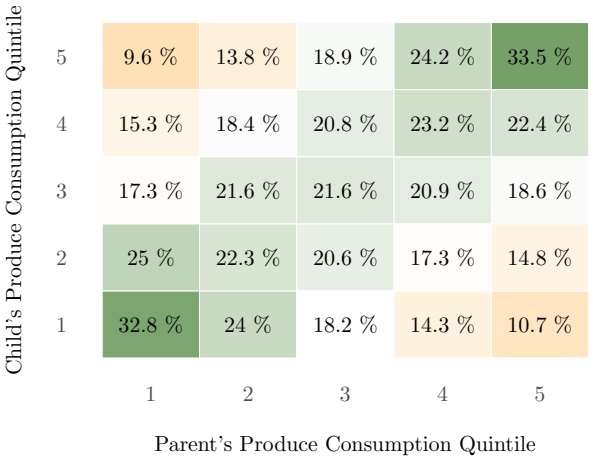
Notes: The figure shows the transition matrix for all households that could uniquely be matched to their parents in both data sets for the highest (Figure 4a) and lowest income quartile (Figure 4b). Quintiles are then plotted against each other for children and parents. Figure 4c takes the absolute differences between each quintile-quintile combination in the previous two plots.

These patterns should grow weaker over time if households move away from their family and the social network in which they grew up. Therefore, I look next at the role of the distance between the children's and parents' residences.[13] We see in Figure A3 that nutritional mobility remains low even if children live far away from their parents. However, the further the children move away from their parents, the lower the patterns' persistence. Striking is especially that households are ten percentage points less likely to be trapped at the bottom if they live far away.

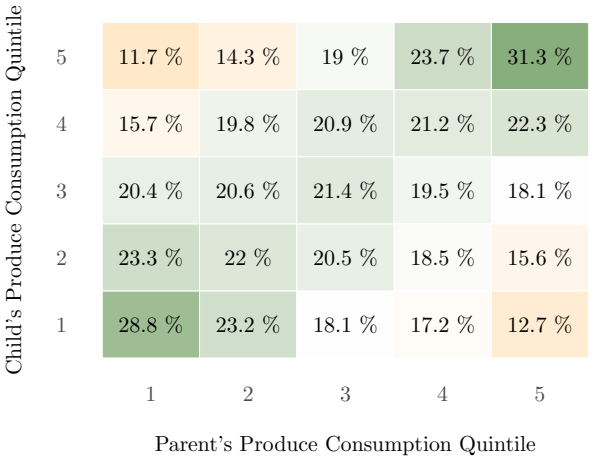
Nonetheless, 80% of Swiss citizens live in a multi-person household and dietary decisions are most likely taken jointly. But then, for a cohabiting couple, which partner's background will influence

[13] I use the API of *search.ch*, a Swiss phone book directory, to calculate travel times by car between grid cells.

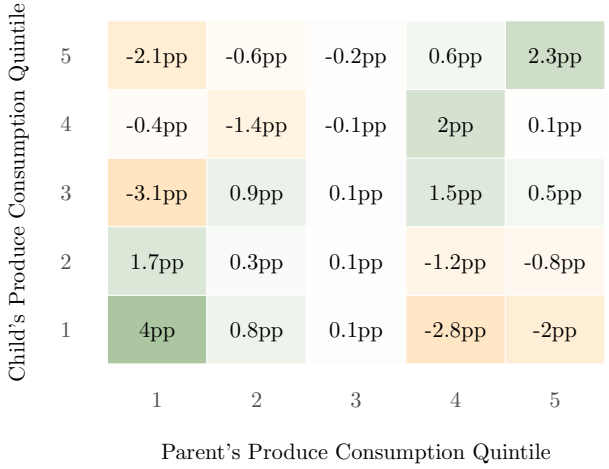
Figure 5: Within a couple: Which gender dominates?



(a) Parents of the Female Partner



(b) Parents of the Male Partner



(c) Female/Male Differences

Notes: The figure shows the transition matrix for all couples that could uniquely be matched to their parents in both data sets for the woman's parents (Figure 5a) and the man's parents (Figure 5b). Quintiles are then plotted against each other for children and parents. Figure 5c takes the absolute differences between each quintile-quintile combination in the previous two plots.

what the couple eats? I select the sub-sample of 20'000 genetically non-related two-person households for which I can match both partners of a cohabiting household across generations to their parents for each of the partners. I observe the following patterns. First, a couple's nutrition is closer related to the woman's parents (see Figure 5). Women from a household eating little vegetables are especially prone to follow their parents' consumption patterns in their relationship. Yet, this gender difference may primarily be driven by a story of first- and second-earners, which I show in Figure A4b. The general message remains the same, but the difference between first and second earners is smaller. Thus, this likely only explains a part of the observed gender difference. Overall, intergenerational dietary patterns are very persistent in Switzerland and exceed similar patterns documented for income.

4 How to Break the Patterns

In this part, I analyze how life-changing individual shocks can induce major adjustments in a household's behavior toward a healthier diet. To this end, I analyze a first child's birth and a parent's death due to a lifestyle-related disease.
These shocks likely affect households differently depending on their initial vegetable intake. On the one hand, those eating poorly have the highest marginal gain of an adjustment. On the other hand, households already eating healthily may have more initial information or stronger preferences regarding diet. Hence, I am interested in the variation of the causal treatment effect along the distribution and apply a changes-in-changes model to estimate the quantile treatment effect (QTE) for different quantiles τ. The Athey and Imbens (2006) model is a generalization of the standard difference-in-differences approach, estimating effects for the entire distribution rather than for a simple average. Also, compared to a difference-in-differences strategy, the CIC approach does not rely on additive separability, and the assumptions are invariant to the scaling of the outcome (say, taking levels or logarithms).

4.1 Identification

In the two-period changes-in-changes model, a treatment group receives a binary treatment in the second period, while both groups are untreated in the initial period. Then, the conditional QTE for quantile τ is given by

$$\Delta^{QE}(\tau|x) = F^{-1}_{Y^I|11x}(\tau) - F^{-1}_{Y^N|11x}(\tau), \tag{2}$$

where Y^N and Y^I are the potential outcomes for non-treated and treated units, respectively. The conditional distribution function is denoted by $F_{Y^N|gtx}$ for treatment status $g \in \{0,1\}$, period $t \in \{0,1\}$, and a set of (potential) covariates x. Integrating over x gives the unconditional quantile treatment effect for quantile τ:

$$\Delta^{QE}(\tau) = F^{-1}_{Y^I|11}(\tau) - F^{-1}_{Y^N|11}(\tau). \tag{3}$$

While $F_{Y^I|11x}$ is observed, I need to construct the counterfactual $F_{Y^N|11x}$ from observed distributions, which relies on the following assumptions:

Assumption 1 (Potential Outcomes) *The potential outcome without treatment can be written as $Y^N = h(X,T,U)$, where U is the unobservable part of Y.*

Assumption 2 (Strict Monotonicity) *$h(\cdot)$ is strictly increasing in the realization of the unobservable, u.*

Assumption 3 (Time Invariance) $U \perp\!\!\!\perp T|G, X$.

Assumption 4 (Common Support) *The QTE is only defined over the common support.*

Assumption 1 implies that Y^N does not depend on group assignment g. In the absence of treatment, households in both groups would observe the same Y. Further, Assumption 2 means that a higher unobservable u always leads to a higher outcome y. Assumption 3 is the counterpart to the parallel trend assumption in a difference-in-differences model, meaning that the distribution of the unobservables is stable over time within a group. Importantly, this still allows for different distributions between groups, and individuals can change their rank over time. Finally, Assumption 4 holds mechanically as the changes-in-changes model does not extrapolate beyond the support. Under these assumptions, Athey and Imbens (2006) proof that the counterfactual for the treatment group in $t = 1$ can be constructed from the observable CDFs and the QTE is

$$\begin{aligned}\Delta^{QE}(\tau|x) &= F^{-1}_{Y^I|11x}(\tau) - F^{-1}_{Y^N|11x}(\tau) \\ &= F^{-1}_{Y|11x}(\tau) - F^{-1}_{Y|01x}\left(F_{Y|00x}\left(F^{-1}_{Y|10x}(\tau)\right)\right). \end{aligned} \tag{4}$$

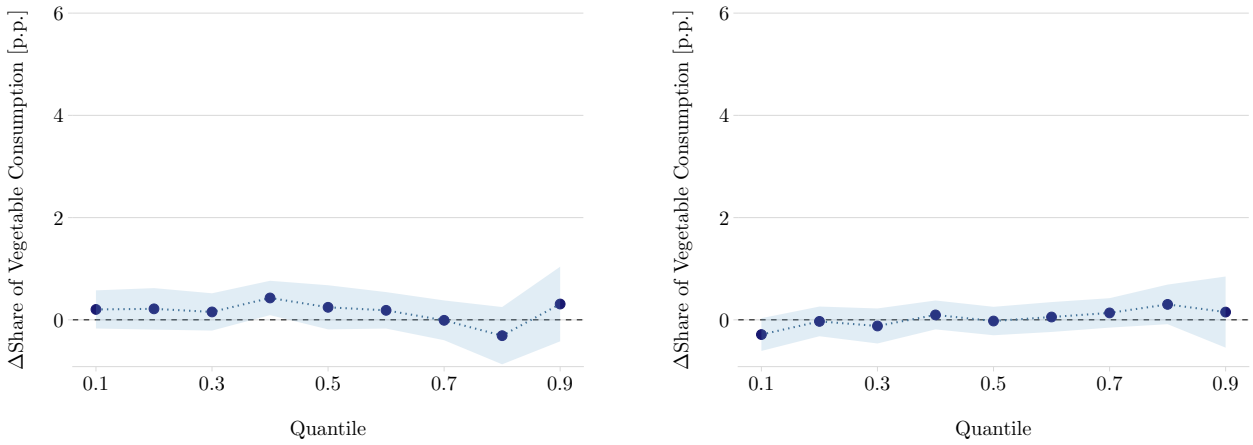
I extend the basic two-by-two CIC-model as suggested in Athey and Imbens (2006) and Melly and Santangelo (2015) to multiple groups and periods, where $t \in \{1, \ldots, T\}$ and $g \in \{2, \ldots, G\}$. Households receive a binary staggered treatment in any period and stay treated thereafter. For a given group-period combination $\{g, t\}$, I estimate the changes-in-changes model in Equation 4 using a valid comparison $\{g_0, t_0\}$, where $t_0 < t$, $g_0 > g$, and $t >= g$.[14] I apply this procedure to every valid tuple $j = \{g, t, g_0, t_0\} \in \{1, \ldots, J\}$. Following the plug-in principle, I estimate and store the empirical CDF $\hat{F}_n(t) = \frac{1}{n}\sum_{i=1}^{n} \mathbb{1}_{X_i \leq t}$ for $\hat{F}^j_{Y^I|gtx}(\tau)$ and the counterfactual distribution $\hat{F}^j_{Y^N|gtx}(\tau)$. To construct the sample analogs of $F_{Y^I|11x}(\tau)$ and $F_{Y^N|11x}(\tau)$, I average all empirical CDFs for the treated and control groups separately, weighting by the size of group g:

$$\hat{F}_{Y^I|11x}(\tau) = \sum_{\{g,t,g_0,t_0\} \in J} \hat{F}^j_{Y^I|gtx}(\tau) \cdot \omega_g \quad \text{and} \quad \hat{F}_{Y^N|11x}(\tau) = \sum_{\{g,t,g_0,t_0\}} \hat{F}^j_{Y^N|gtx}(\tau) \cdot \omega_g,$$

where $\sum_{g=1}^{G} \omega_g = 1$. Then, Equation 2 identifies the quantile treatment effect (QTE). Bootstrapping this procedure 1'000 times adds the standard errors. Additionally, I aggregate the empirical CDFs for every post-treatment period separately to analyze the evolution of the QTE over time in an event-study fashion. Ultimately, never-treated households may differ from treated units in my specifications and may not be a good comparison group. Thus, I exploit the random timing

[14] This implies that in the baseline period t_0 none of the two groups is treated. Further, the control group needs to be treated after the treatment group (meaning, it is a *not-yet-treated* comparison group), and QTEs are only identified for post-treatment periods. QTEs for the first and last group ($g \in \{1, G\}$) are not identified as the first has no baseline period, and the last has no later-treated group. This results in $\sum_{g=2}^{T-1}\sum_{k=1}^{T-g} k \cdot (g-1)$ valid two-by-two comparisons.

Figure 6: Changes-in-Changes Estimates: Income Increase



(a) Effect on low-income households

(b) Effect on high-income households

Notes: The figure shows the average quantile treatment effect of an income increase of at least 10% on the share of vegetables of total food consumption. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications. Figure 6a shows the results for households with a below-median household income, adjusted for the square root of household size. Figure 6b shows the same results for households above the median.

of the shocks and use as a control group only households that receive the same shock later.

4.2 Results

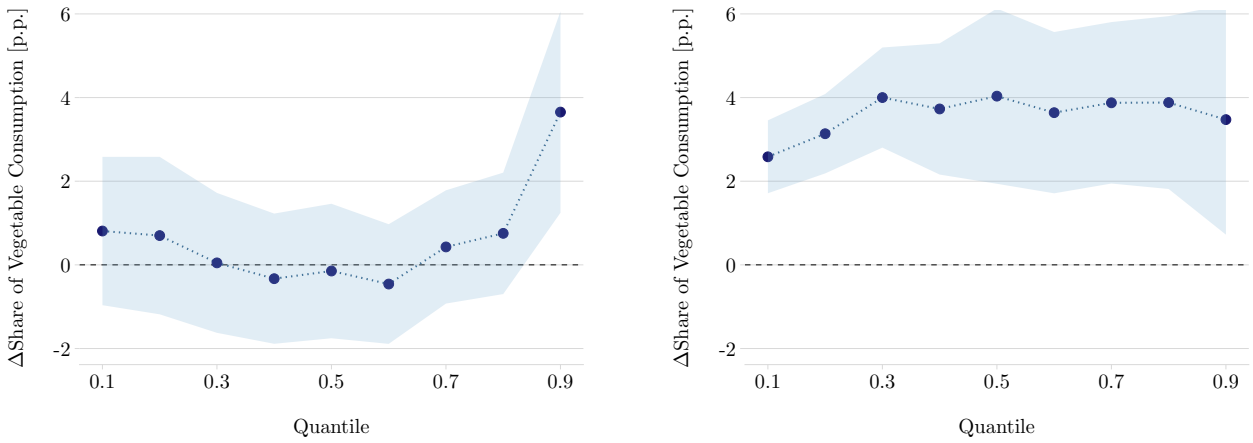
This subsection presents the estimation results for the changes-in-changes models. I discuss the effects of an income raise before analyzing the life-changing events of a first child's birth and a parent's death due to lifestyle-related strokes and heart attacks.

Positive income shocks

The most obvious potential explanation for nutritional inequality is that eating healthily is too expensive for low-income households. Then, the relationship between income and vegetable consumption would not be due to differences in tastes or network effects but simply because of a binding budget constraint for the less affluent. Therefore, if this is the limiting factor for low-income households to follow a healthier diet, an unexpected positive income shock should arguably improve the quality of their diet. Hence, I look at the response of a household's monthly vegetable intake after a permanent increase in household income of at least 10%, separately for above- and below-median income households.[15]

[15]I omit households that receive multiple treatments within my sample periods. Further, I do not analyze negative income shocks because they will often neither be exogenous nor random. Households can reduce their labor supply voluntarily (e.g., for parental leave). Then, the shock does not necessarily tighten their budget constraint.

Figure 7: Changes-in-Changes Estimates: Death due to a lifestyle-realted disease



(a) Effect on low-income children

(b) Effect on high-income children

Notes: The figure shows the average quantile treatment effect of the unexpected lifestyle-related death of a parent (heart attack or stroke) on the share of vegetables of total food consumption. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications. Figure 7a shows the results for households with a below-median household income, adjusted by the square root of household size. Figure 7b shows the same results for households above the median.

Figure 6 demonstrates the average quantile treatment effects for the changes-in-changes model with multiple periods. Vegetable consumption does not significantly increase after the income shock, neither for the poorer nor the richer households, and this conclusion holds for the entire distribution. Thus, an income increase has no impact on produce intake, independent of income and previous consumption levels. This is consistent with previous evidence and supports the initial motivation that almost no households in Switzerland should be budget-constrained (FAO, 2022). These findings suggest that the role of income for diet in Switzerland is negligible for all income groups, and other mechanisms must cause nutritional inequality.

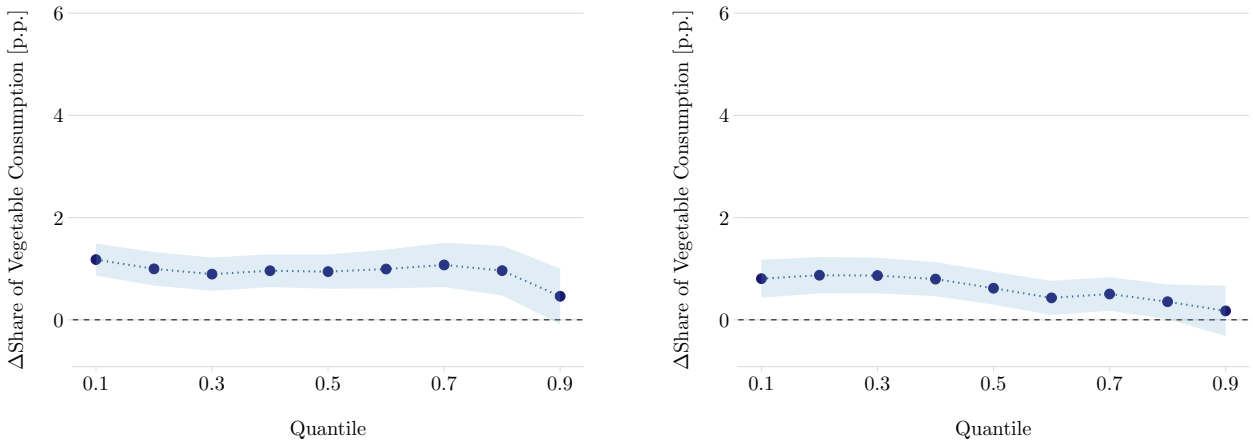
The death of a parent

Instead, nutritional patterns may be shaped early on through one's upbringing and the influence of the close family. Motivated by Fadlon and Nielsen (2019), I exploit the quasi-experimental variation where a family member dies due to a lifestyle-related disease. These deaths are statistically more likely if an individual follows an unhealthy lifestyle. I focus on parents that die from a stroke or heart attack but exclude those suffering from underlying health conditions that increase the likelihood of a stroke or heart attack because then households may already have incorporated the shock, and no new information would be revealed.[16]

Figure 7 shows the quantile treatment effects separately for below- and above-median income

[16]The underlying accompanying diseases leading to an exclusion are diabetes, hypertension, ischemic, symptomatic and other heart diseases, dementia, and Alzheimer's.

Figure 8: Changes-in-Changes Estimates: Birth of a first child



(a) Effect on low-income children

(b) Effect on high-income children

Notes: The figure shows the average quantile treatment effect of the birth of a first child on the share of vegetables of total food consumption. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications. Figure 8a shows the results for households with a below-median household income, adjusted by the square root of household size. Figure 8b shows the same results for households above the median.

households. Looking at the less affluent, I find a positive effect only for the last decile and a U-shape for the distribution, although insignificant. This observed U-shape of the effect makes intuitively sense: The left tail includes households that follow an unhealthy diet and have the highest marginal gains. The right tail consists of those that already eat healthily. Thus, these groups may better access nutritional information or assimilate the information shock. Switching to wealthy households, I find that a parent's death increases average vegetable consumption by up to four percentage points. Compared to the mean share of 21%, this is an increase of 19 percent. Although the effect is smaller at the lower tail, it is significant along the entire distribution. Hence, I find a strong positive reaction from high-income individuals but no response from the less affluent. This positive effect could either originate from learning about one's health risk and a potential predisposition or from a temporary shift of awareness. The response to the death of a step-parent isolates the latter channel, as there is no genetic relationship to the children. In this case, the results show no significant response in produce consumption. Thus, I argue that the learning channel drives the mechanism. This conclusion contrasts Fadlon and Nielsen (2019), who find that the effect of a parent's death on the consumption of preventive medicine mainly goes through a salience channel. To assess the robustness of these findings, I conduct a placebo test by looking at the response to deceased that are not linked to an unhealthy lifestyle. Figure A5 shows for no decile a significant change in diet after a parent's death due to pneumonia, influenza, bronchitis, or an infectious disease.

Overall, the low-income households' lack of response may be either caused by insufficient knowledge about the links between diet and health or systematically different assimilation of the shock. In both cases, low-income households should be sensitized by their parents' doctors about genetic predispositions and the potentially beneficial role of a healthy diet.

The birth of a child

Next, I analyze the nutritional response to the birth of your first child. Figure 8 provides the quantile treatment effects. For both income groups, households at the bottom of the distribution face a significant increase in their produce intake of around 1%. The effect gradually declines and becomes insignificant at the top of the distribution, with no apparent difference between the two income groups. Finally, I explore the potential channels driving this finding. The birth of your first child confronts parents with the question of how to nurture the child. Hence, they will likely collect nutritional information that may have spillover effects on their own diet. Alternatively, the birth may increase the parents' valuation of their own health in response to the new responsibility they face. To separate the channels, I repeat the analysis for the birth of the second child. In this case, I expect no additional information collection, and the parents' valuation of their health would explain the effect. As all the results are insignificant in this case, I argue that the information collection mechanism determines the response for the first child. To analyze the dynamics of the response, Figure A6 shows the average effect over time. In the first months after the child's birth, households eat 1-2 percentage points less fresh produce. Subsequently, households across all quantiles gradually increase their intake of fruits and vegetables, reaching their previous level after half a year. Eventually, the effect becomes significantly positive in the long run and stabilizes around 1-2 percentage points. The only exception are households at the top deciles, whose positive effect vanishes after one year. Hence, the average QTE is higher if we exclude the negative short-term effects.

My findings suggest that an important share of households at the left tail of the vegetable distribution lacks decisive information about a healthy diet. Providing further guidance to young couples may be a beneficial policy.

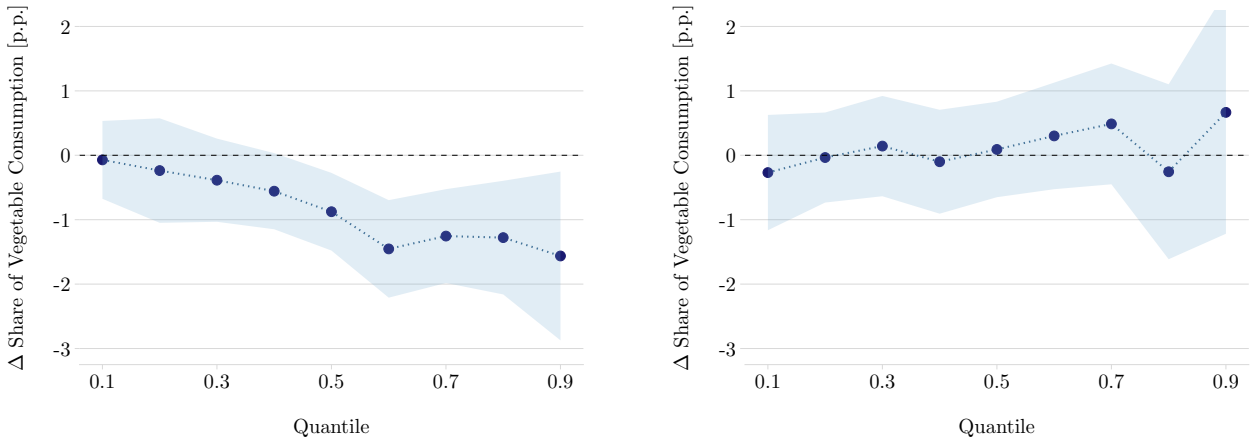
4.3 Additional Results

Broader social networks may also influence diet outside of the close family. Therefore, I look at neighborhood effects by exploiting the strong spatial variation in produce intake (Figure A1) and following movers between municipalities.

Moving between municipalities

To this end, I calculate the average produce share in overlapping neighborhoods with 500-meter radii around the household's residence and define neighborhoods with above-median fruit and vegetable consumption as "healthy" and those below the median as "unhealthy". This allows me to compare households moving from a healthy to an unhealthy neighborhood to those who move later in the same direction.Yet, the location choice of movers is endogenous, and households may sort into specific neighborhoods due to food supply and individual preferences. Although the shock is not random, estimation still provides causal effects if I control for all nutrition-related

Figure 9: Changes-in-Changes Estimates: Movers



(a) Move to a worse place

(b) Move to a better place

Notes: The figure shows the average quantile treatment effect of moving on the share of vegetables of total food consumption. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications. Figure 9a shows the results for households moving from a healthy to an unhealthy neighborhood. Figure 9b shows the results for households moving from an unhealthy to a healthy neighborhood.

factors that would influence the location choice.[17] Hence, I control for income, household size, and the neighborhood's number of supermarkets and takeaways. This eliminates any channels going through key socio-economic variables and grocery stores or out-of-home food providers.

Figure 9 shows the estimation results. On the one hand, diet does not change if a household moves to a healthier municipality. Hence, I find no evidence for positive spatial spillovers or *moving to opportunity* in terms of nutrition. This result aligns with Allcott et al. (2019a), who find no dietary response after moving to a place with broader availability of healthy shops. On the other hand, I find a significant negative effect of moving to a bad place that increases along the vegetable distribution. This means households that previously ate plenty of vegetables experience the most significant drop after the move into an unhealthier surrounding of up to two percentage points. Yet, this analysis does not separate the effects of the new neighborhood from the move itself and the underlying mechanisms driving the results are unclear.

Lastly, I repeat this analysis on the municipality level and find for both groups insignificant results. This suggests that spatial spillovers in diet work within a small neighborhood and do not extend to an entire municipality.

[17] See Deryugina and Molitor (2021) discussing the difficulties in estimating causal place effects.

4.4 Sensitivity and Robustness

The rank invariance assumption is testable. In the two-period case, this requires observing an additional period $t = -1$. Then, the rank invariance is not violated if

$$F_{Y,0,-1}\left(F^{-1}_{Y,1,-1}(\tau)\right) = F_{Y,0,0}\left(F^{-1}_{Y,1,0}(\tau)\right) \forall \tau \in (0,1), \tag{5}$$

where $Z = F^{-1}_{Y,1,j}(\tau)$ is the treatment group's quantile function in period j and $F_{Y,0,j}(\cdot)$ returns the rank of Z in the control group.[18] The Kolmogorov-Smirnov test, for example, checks this condition by testing whether two samples may originate from the same theoretical distribution (Melly and Santangelo, 2015; Athey and Imbens, 2006).

With multiple pre-periods, Equation 5 naturally extends to all observed $t < 0$:

$$F_{Y,0,-j}\left(F^{-1}_{Y,1,-j}(\tau)\right) = F_{Y,0,0}\left(F^{-1}_{Y,1,0}(\tau)\right) \forall \tau \in (0,1) \text{ and } \forall j \in (t_0, \dots, -1), \tag{6}$$

where t_0 is the initial period in the data. A k-sample Anderson-Darling test tests these $|t_0|$ conditions jointly by checking the equality of multiple samples. I still have to implement these tests.

5 Conclusions

The fact that wealthier households eat healthier attracted the interest of many economists recently. The natural hypothesis is that a healthy diet is too expensive for low-income households. In response, I analyze nutrition in Switzerland to shut down this income channel, as it is a homogeneous, wealthy country where everyone can afford a healthy diet. Rather than income, I show that social networks and intergenerational patterns shape how we eat and that informational health shocks going through this network can alter behavioral patterns. Moreover, I find that low-income and immobile households are more likely stuck in a poor lifestyle over generations, and they either assimilate health information shocks worse or have insufficient access to them. Thus, increasing shop density through zoning policies or substituting healthy products may be ineffective policies to improve the diet of low-income households. Instead, information campaigns like school-food programs or food labeling policies may be more helpful, and the strong intergenerational persistence of diet would only enhance the benefits of successful campaigns by generating spillovers throughout the family. Also, doctors should sensibilize their patients at risk

[18] Assume, for example, that the median in the treatment group in period $t = -1$ is 100 and that a control individual with this value of 100 would be at the seventh decile. Then, Equation 5 requires that if this median increases in period $t = 0$ to 110, a control individual with a value of 110 still needs to be at the seventh decile in $t = 0$.

of lifestyle diseases and their relatives about the health benefits of a balanced diet. Successfully targeting low-income households would generate particularly high welfare gains.

Nevertheless, it is important to be aware of the limitations of my approach. First, although arguably a good proxy on average, the consumption of fresh fruits and vegetables does not necessarily capture a complete picture of a household's healthiness. It especially ignores all healthy frozen or processed products and food waste. Second, this paper focuses on consumption at the largest Swiss retailer Migros, which may bias the results if there is some sorting between chains. Furthermore, I cannot account for any out-of-home consumption, self-supply, or local farmers' markets, which may underestimate vegetable consumption in rural areas. Third, the administrative data does not have complete coverage for foreign-born individuals. Hence, their share of the population is underrepresented. Fourth, I cannot account for previous non-fatal heart attacks in the death records, and the death of a parent may cause adverse effects (e.g., depression) on an individual's health that influence her diet.

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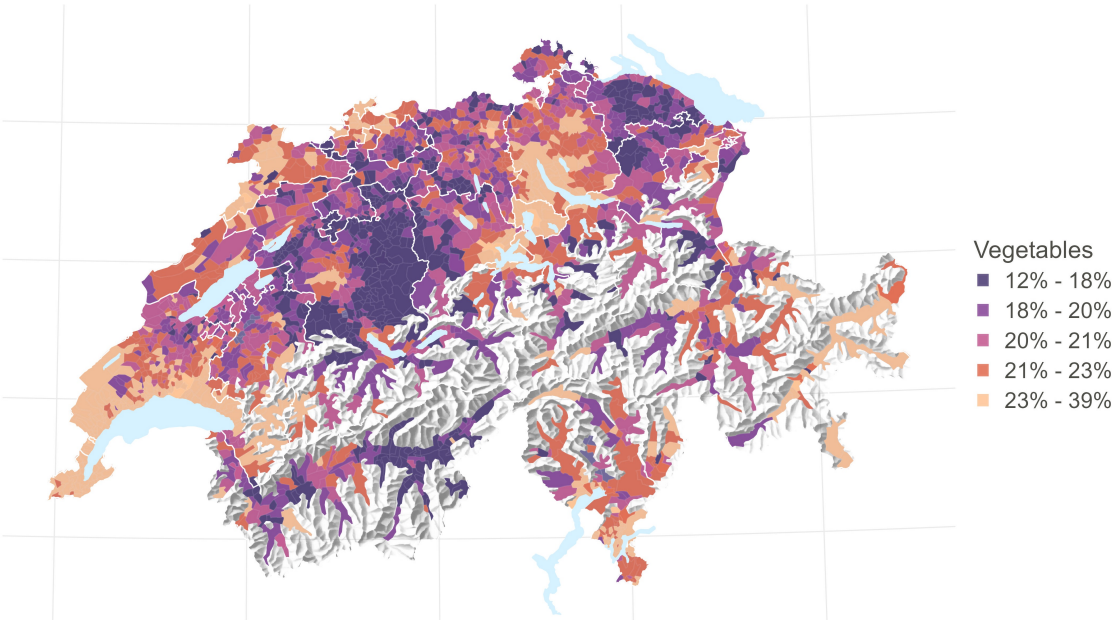
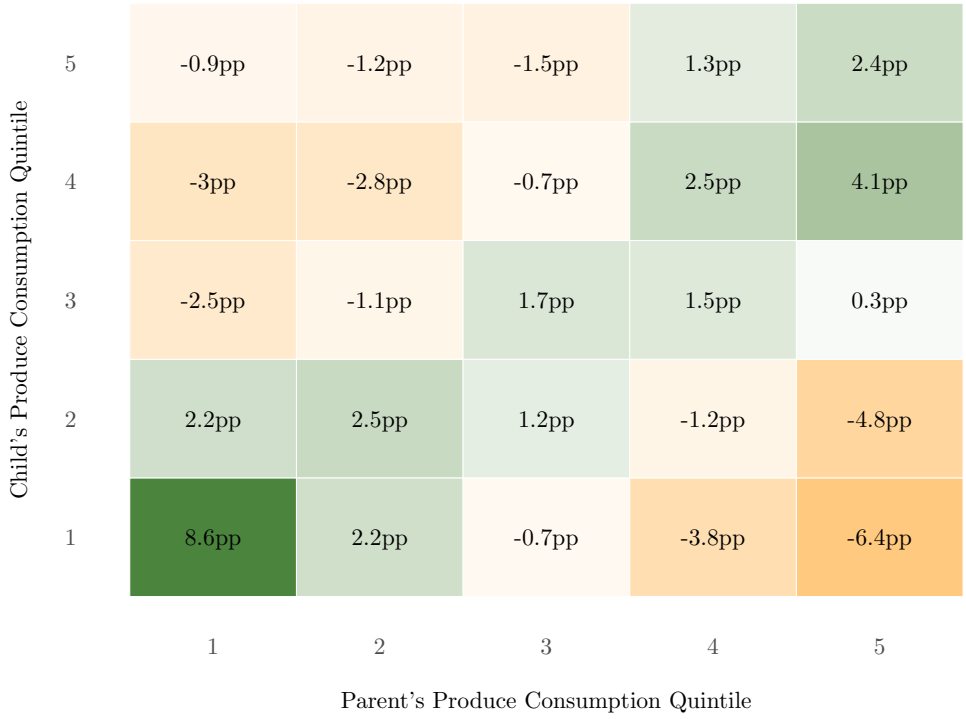
A Appendix

Figure A1: Vegetable Consumption Across Space



Notes: The map shows the share of fruit and vegetables of food consumption at Migros across Switzerland. Monthly household expenditures are aggregated and averaged onto a municipality level.

Figure A2: Dietary vs. Income Mobility Premium



Notes: The figure shows the absolute difference between the transition probabilities in diet in Figure 2 and the transition in income in Chuard and Grassi (2020). Quintiles are then plotted against each other for children and parents.

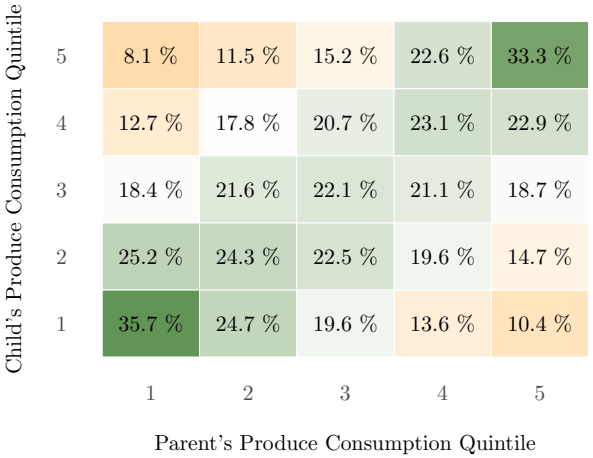
Table A 1: Rank-Rank Regressions

Dependent Variables:	Child's Rank				
Model:	(1)	(2)	(3)	(4)	(5)
Variables 2nd stage					
(Intercept)	39.0***	-11.1***	-10.9***	-10.9***	-10.8***
	(0.174)	(0.173)	(0.364)	(0.173)	(0.173)
Parent's Rank	0.228***	0.220***	0.220***	0.215***	0.213***
	(0.003)	(0.003)	(0.006)	(0.003)	(0.003)
First Stage					
Income Child		✓	✓		
Income Parent			✓		
SES Child				✓	✓
SES Parent					✓
Fit statistics					
Observations	106,536	106,536	22,822	106,536	106,536
Adjusted R^2	0.052	0.049	0.050	0.047	0.046

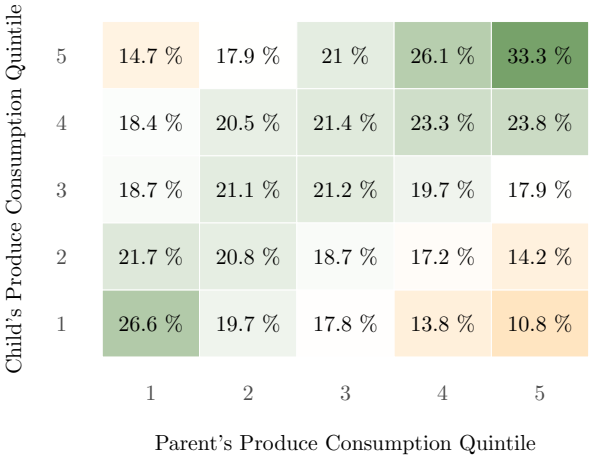
Clustered (id) standard-errors in parentheses

Notes: The table shows regression results for the rank-rank models in Figure 3. Model 1 regresses directly the child's rank on the parent's rank. Model 2 first regresses the child's rank on her income (and her parent's income in model 3), and then the residuals on the parents' rank. Model 4 first regresses the child's rank on the average adjusted household income within 500 meters of her residence (and for her parents in model 5).

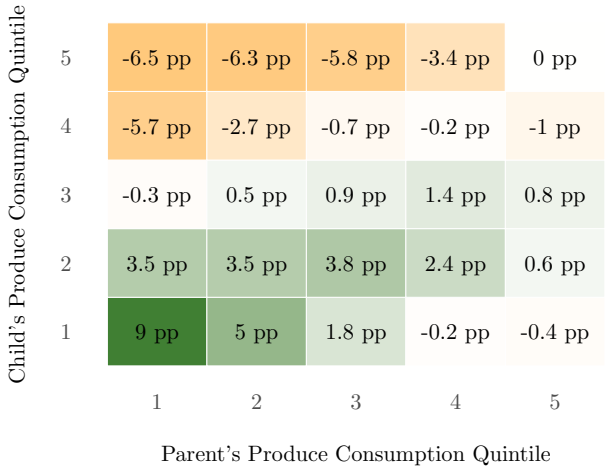
Figure A3: Intergenerational Diet: The role of distance to parents



(a) Living close to parents



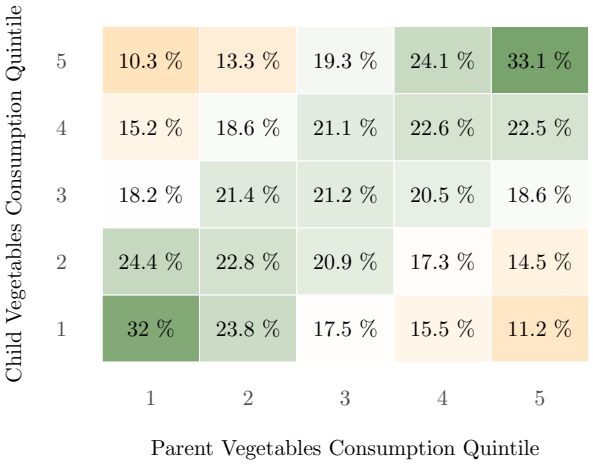
(b) Living far from parents



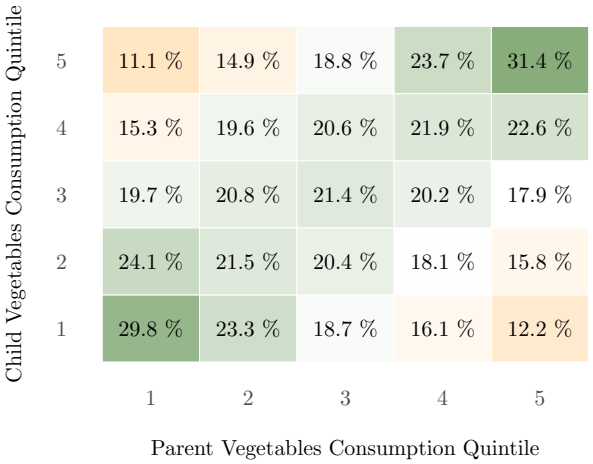
(c) Living-close premium

Notes: The figure shows the transition matrix for all households that could uniquely be matched to their parents in both data sets for the lowest (Figure A3a) and highest distance-to-parents quartile (Figure A3b). Quintiles are then plotted against each other for children and parents. Figure A3c takes the absolute differences between each quintile-quintile combination in the previous two plots.

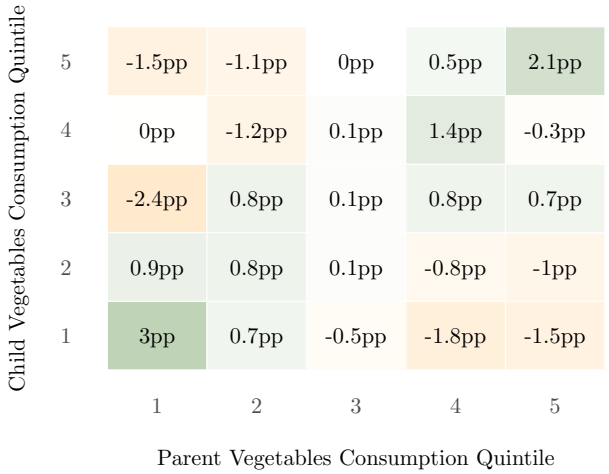
Figure A4: Within a couple: Which earning status dominates?



(a) Parents of the Second-Earner Partner



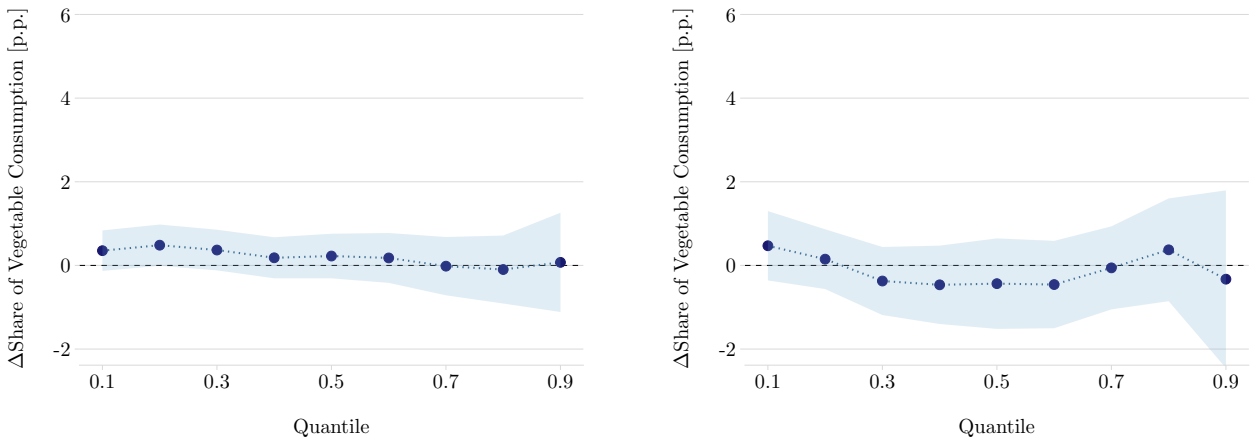
(b) Parents of the First-Earner Partner



(c) Second-earner premium

Notes: The figure shows the transition matrix for all couples that could uniquely be matched to their parents in both data sets for the second earner's parents (Figure A4a) and the first earner's parents (Figure A4b). Quintiles are then plotted against each other for children and parents. Figure A4c takes the absolute differences between each quintile-quintile combination in the previous two plots.

Figure A5: Changes-in-Changes Estimates: Death due to a non-lifestyle-related disease

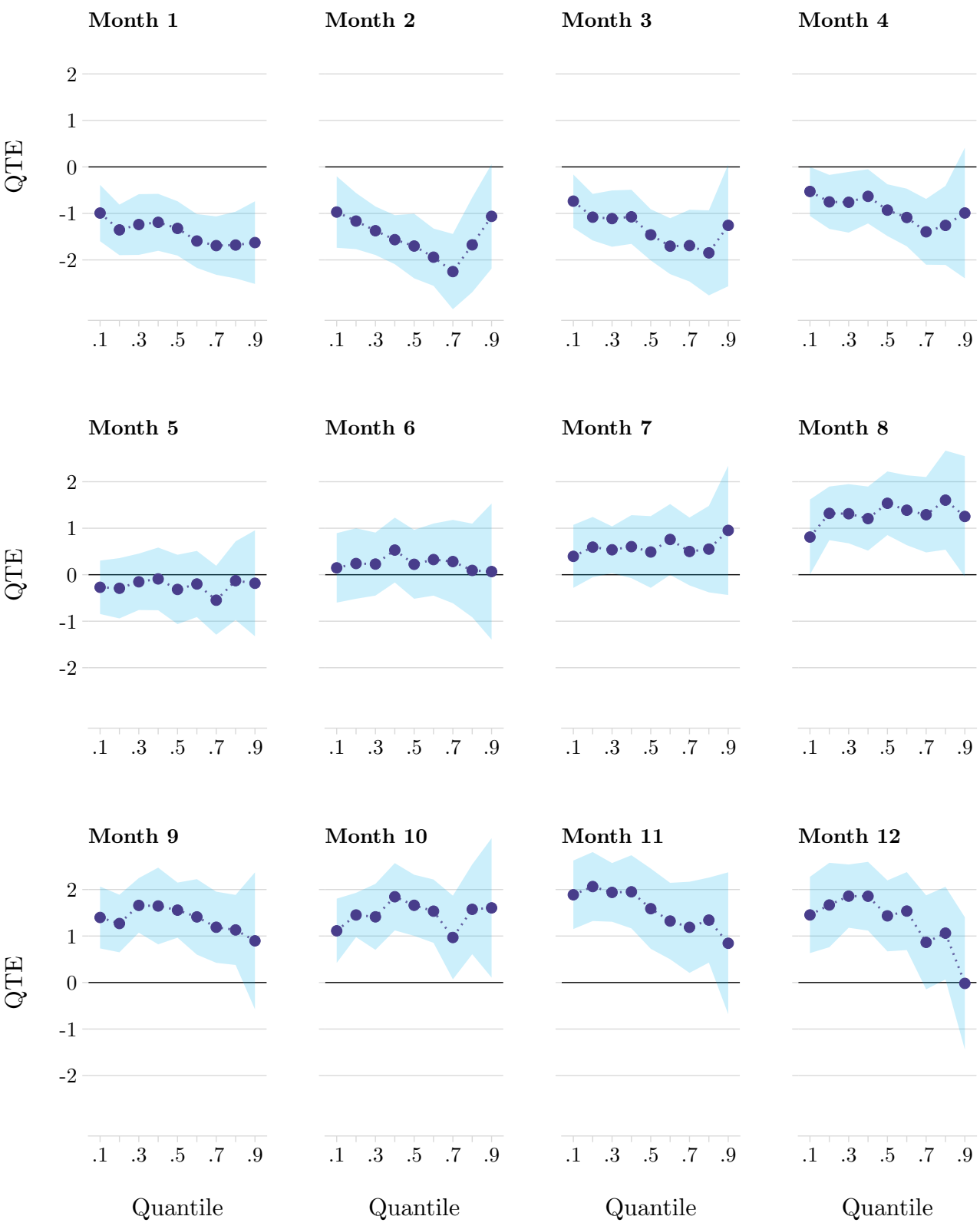


(a) Pneumonia, Influenza, Bronchitis

(b) Infectious Diseases

Notes: The figure shows the average quantile treatment effect of the non-lifestyle-related death of a parent on the share of vegetables of total food consumption. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications. Figure A5a shows the results for children whose parents died from pneumonia, influenza, or bronchitis. Figure A5b shows the same results for infectious diseases.

Figure A6: Dynamic CIC: Birth of the firstborn child for high-income households



Notes: The figure shows the dynamic average quantile treatment effect of the birth of your first child on the share of vegetables of total food consumption for the first 12 months after the treatment. I consider only households with an adjusted household income above the median. Estimates are for the changes-in-changes model with multiple periods using 1'000 bootstrap replications.