HEDG
HEALTH, ECONOMETRICS AND DATA GROUP
THE UNIVERSITY of York

WP 12/08

Average and quantile treatment effects of the American Folic Acid Fortification: an evaluation in a quasi-experimental framework

Elena Fumagalli

May 2012

york.ac.uk/res/herc/hedgwp

Average and quantile treatment effects of the American Folic Acid Fortification: an evaluation in a quasi-experimental framework

Elena Fumagalli[1]

Abstract

The American program of folic acid fortification is generally thought to have increased the average amount of serum folate in the population and, hence, widely considered as a successful public health intervention. We use several waves of the "National Health and Nutrition Examination Survey" (NHANES) to evaluate the causal impact of the fortification of ready-to-eat cereals on serum folate concentration, using a quasi experimental framework. First, we compute the average treatment effect by using matching methods to solve the problem of selection on observables, finding a strong selection into treatment mainly based on race-ethnicity and education. Second, we assess the distributional impact of the fortification by computing quantile treatment effects, under different assumptions on the dependence between the distributions of potential outcomes, and we find significant variation in the impact of fortification across the population, thus rejecting the common effects model. Fortification appears to have had the least (though still modestly beneficial) impact among those that most needed it and the biggest impact among those that needed it least, thus suggesting the presence of folate over-consumption in the latter group, with potential adverse health effects. Third, by controlling our estimates for the concentration of beta-carotene, we find support for the hypothesis that part of the increase in serum folate concentration can be explained by changes in diet, leaving a smaller attributable effect to the fortification itself.

KEYWORDS: Folic acid fortification, quantile treatment effect, matching, policy evaluation.

JEL CODE: C21, I12, I18

[1]Norwich Medical School. University of East Anglia, Norwich. Email: efumagalli@uea.ac.uk.

I am grateful to Jerome Adda, Agar Brugiavini, Eric Brunner, Laura Fumagalli, Andrew Jones, Mario Padula, Nigel Rice, Ernesto Villanueva, Marc Suhrcke and Joachim Winter and the participants to the seminars at the University of East Anglia, University of Cambridge, University of York, IRDES, Paris and the ESPE 2010 conference, ASHE conference 2010, AIES conference 2010 for helpful comments and suggestions.

1) Introduction

The beneficial effects of folic acid fortification are being debated in many countries. Women who have a sufficient intake of folic acid in the first weeks of the pregnancy and before its start have a lower probability to give birth to a baby affected by spina bifida, anencephaly and other neural tube defects (NTDs). In addition, an increase of concentration in folic acid could lower the incidence of cardiovascular diseases and cancer (Hung et al., 2003). In spite of this interest, the fortification of cereal grain products implemented in the US has never obtained a sufficient attention in the economic literature. In this paper we aim at filling this gap, by analyzing the average and distributional impact of the fortification of ready-to-eat cereals on serum folate concentration.

The analysis focuses on the fortification of ready-to-eat (RTE) cereals and disregards the effect of the fortification of flour for two main reasons. First of all, flour is contained in many foods in different percentages, thus making it difficult to separate the treatment from the control group. Second, considering just RTE cereals means focusing on the cases in which an explicit choice of being treated takes place, in order to investigate its correlation with the changes in consumption of fruit and vegetables. In addition, such a choice allows us to have an idea of the percentage of people who self-selected into treatment and their main characteristics.[2] Finally, the analysis of a specific fortified food, which provides a high amount of folic acid, is based on the idea that, from a public health point of view, it is not just interesting whether or not a folic acid fortification is effective, but also which kind of products are the best vehicles for reaching the target population, without increasing overconsumption in the non-deficient groups.

In the 1990ies several ways of tackling the problem of folate deficiency have been proposed. The first option consists in improving the dietary habits[3] through information campaigns warning against the dangers of the diseases associated with low levels of serum folate.[4] Another available option is the fortification with chemical folic acid of some commonly consumed products.[5] A third option involves taking dietary supplements in pills. All the three alternatives have advantages and drawbacks. In particular, the first policy could increase the socioeconomic inequalities in health. In fact, the campaign could have a stronger impact on a particular group, e.g the most educated and wealthiest individuals. On the contrary, the compulsory fortification is the only policy that should in principle ensure the almost total coverage of the population, thus making the unplanned pregnancies less risky. However, the logical drawback of such a policy is its inability to target the high risk groups more than the low risk ones. In addition, if the spread of supplemented foods is very large, the presence of unmetabolized folic acid in serum could be an issue. Compulsory "hidden" fortification may also be seen as an overly paternalistic intervention in that the government decides about what citizens' consume without making them fully

[2] For a formal explanation of self selection into treatment due to a utility maximization process, see Firpo (2007) and Heckman et al. (1997)

[3] Many foods, in particular the green leafy ones do contain a high amount of dietary folate, the natural equivalent of folic acid.

[4] An individual is considered depleted if her serum folate falls below the critical value of 7 nmol/L (see Jacques et al, 1999).

[5] It is worth noting that 1 mg of folic acid is equivalent to 1.7 mg of natural folate, when we consider their capacity to be transformed into serum folate in blood. Hence, fortified foods are able to dramatically increase the concentration of serum folate.

aware of it and without giving them the choice of not consuming the relevant food without folic acid. Finally, the third option, i.e. drug-based supplements, can be in-effective, since compliance is difficult to be achieved.

The USA was the first country that designed a policy to solve the problem of deficiencies in serum folate concentration, immediately followed by Canada and, later, by Chile and Australia. The American effort aiming at increasing the folic acid intake in the target population started in 1992, when the FDA recommended to women a 0.4mg/d of the nutrient (see Daly et al, 1997 for a discussion of the exact amount of folic acid supplementation), in order to limit the cases of NTDs, which affected around 1/1000 live births in the country (Hernandez-Diaz et al 2001). At the same time as the recommendation started, a huge awareness campaign took place. Despite these efforts, only 30% of women followed the advice[6] (see Dunlap et al, 2011). As a consequence, the US health authorities decided in 1996 to recommend folic acid fortification, which became mandatory on 1 January 1998. Since then, the US Food and Drug Administration (FDA) has enforced the fortification of all the enriched (with niacin, thiamin, and riboflavin) cereal grain products with (chemical) folic acid (140 micrograms into 100 grams of grain). On the contrary, none of the European countries has yet implemented the fortification, due to the still limited knowledge of the potential negative effects of unmetabolized folic acid (see Smith et al, 2008; Cuskelly et al, 2008). In many cases, the beginning of the intervention has been delayed due to concerns that arose after the initial enthusiasm that followed the implementation of the policy in the USA.[7]

Although extensive, the literature on the effects of the folic acid fortification has missed some fundamental questions. The evaluations based on randomized experiments rely just on small samples and are only able to measure the capacity of the body to metabolize folic acid and to transform it into serum folate, while they do not take into account the changes in behavior due to the policy and the determinants of the selection into treatment. Since the fortification was preceded by an awareness campaign, the self selection into treatment is likely not to be random, but it could depend on personal tastes (which can be age and ethnicity-specific) and on the probability to get pregnant (which mainly depends on observable characteristics such as age and gender). By contrast, the evaluations conducted using survey data do not capture the exact impact of the policy, since they do not correct for period specific shocks and they do not address the problem of finding a counterfactual. In conclusion, the simple difference in average serum folate before and after 1998 does not capture just the effect of the fortification itself, but also the effect of a possible change in dietary habits.[8]

The paper enriches the literature in a few ways. First, we compare the increase in serum folate concentration to the change in the concentration of other nutrients to separate the effect of the fortification from the one of a change in dietary patterns.

[6] It has been noticed that the women who follow the *"U.S. Dietary Guidelines for Americans"* and the *"U.S. Dietary Pyramid"* are able to reach 0.4 mg of folic acid a day, which is the amount of the nutrient needed to reduce the cases of NTDs of about 50%, according to the *"Centers for Disease Control"* (CDC, 2002, 41(RR-14);001).

[7] In the UK, for example, in May 2007, the Food Standards Agency Board recommended the fortification of bread with folic acid. However, in October of the same year, the Chief Medical Officer (CMO) asked the Scientific Advisory Committee on Nutrition (SACN) to review further medical evidence before implementing the policy. After the review, in October 2009, the SACN did not change its previous recommendation. See: SACN(2009)

[8] As we can see from early evaluations, the evidence of a change in dietary folate is not clear, given the high measurement error present in dietary intake data.

Second we compute the average treatment effect of the fortification of RTE cereals on concentration of serum folate by correcting for selection into treatment. Going beyond average effects, we use e the quantile treatment effect estimator developed by Firpo (2007) to study heterogeneous effect in a health economics context (see Bitler et al, 2006; Bitler et al, 2008; and Volpe Martincus and Caballo, 2010 for other examples in economics). Using this methodology we seek to illustrate the additional information and nuance that can be gained by assessing the heterogeneous effects of public policies, rather than just the average treatment effects. Finally, we measure the change in diet in a new way: by considering examination data rather than by using dietary recall interviews, which are affected by measurement error problems.

Our results suggest that there is a strong selection into treatment mainly based on race-ethnicity and education. We also find significant variation in the impact of fortification across the population, thus rejecting the common effects model. Finally, the larger impacts of the fortification in the serum folate concentration are in the top part of the distribution, suggesting that the program could have increased unmetabolized folic acid in serum. If it was the case, the supplementation could have amplified folic acid overconsumption, which could have harmful effects on health. We also find evidence of a change in dietary patterns and health inequality as second order effect of the fortification.

This paper is organized as follows. In section 2 we describe the previous evaluations of the folic acid fortification program. In section 3 we present the descriptive evidence, the methods we use, and the main results. Section 4 concludes.

2) Background

Many evaluations of the folic acid fortification have been carried out: some performed a cost-benefit and cost-effectiveness analysis (Grosse et al. 2005); some assessed the effect of the governmental action on the incidence of NTDs in the period following the fortification; others measured the effect of the fortification in a clinical trial. Finally, a group of evaluations compared the average level of serum folate in the population before and after 1998, using NHANES data. The focus of the evaluations has been mainly on the change in average serum folate concentration in the population, with some research only recently assessing the potential adverse effects of the policy in the form of folic acid overconsumption and distributional consequences (see Morris, 2007).

There is strong confidence in the positive role that fortification plays in reducing the incidence of NTDs. Hernandez-Diaz et al. (2001) used a controlled trial and found a strong relationship between high folic acid intake and lower incidence of NTDs, given that mothers exposed to folic acid antagonists had a higher risk of having children affected by spina bifida, anencephaly, and encephalocele. However, the exact magnitude of the effect as the result of the US policies is still unclear. First, it is difficult to identify the effect of the policy itself on the incidence of NTDs, since the evaluation only takes the form of a before-and-after comparison. In addition, the incidence of NTDs is measured just on live births without taking into account miscarriages and abortions, which could have been increased after the awareness campaign and as a consequence of better prenatal screening.[9] By using information contained in birth certificates, a decrease of NTDs of about 19% has been estimated

9 All these evaluations rely on the assumption that pregnancy interruption rates did not change as an effect of the treatment

(Honein et al, 2001). On the contrary, according to a study conducted by the Centers for Disease Control and Prevention (CDC),[10] the effect of the fortification was much stronger, i.e. 27%. Finally, Williams et al. (2005) suggest that the decline in spina bifida and anencephaly was significant and high (around 35%) among non-Hispanic white and Mexican-Hispanic, while it was not statistically significant among non-Hispanic blacks.

The effect of the folic acid fortification in increasing the level of serum folate is not questioned either. Cuskelly et al. (1999) conducted a clinical trial to compare the levels in serum folate of people who were eating fortified cereals to those of individuals who were not, finding a statistically significant (positive) difference. There is also a considerable body of research on folic acid fortification using survey data (mainly NHANES). It has been shown that the incidence of low folate concentrations has been reduced to negligible levels as a result of the fortification (Jacques et al, 1999), and the fortification increased the amount of serum folate in the overall population (Pfeiffer et al, 2005). In particular, Dietrich et al. (2005) find that there was a significant increase in total folate (around 136%) among those that did not take supplements, and this applies in particular to females in the 20-39 age group. On the contrary, dietary folate increased by just 20-30%. Finally, the increment of folic acid intake in the American population after the fortification was not homogeneous by age, gender and race-ethnicity (Benthley et al, 2005) and it has slowed down more recently[11] (Ganji et al, 2006). Data from the Framingham Heart Study (FHS) show that the fortification increased both total folate and folic acid intake, while folate from food remained unchanged (Choumenkovitch et al., 2002). A similar result is suggested by the statistical analysis conducted by Caudill et al. (2001) on a subsample of women in childbearing age living in South California. The authors find that women in childbearing age achieved an adequate level of serum folate after the fortification.[12]

Some authors also address distributional issues. Using NHANES data, Ford et al. (1999) study the importance of ethnicity and educational attainment in predicting the concentration of serum folate. They find that education is not strongly related to serum folate concentration while ethnicity is, since African American and Mexican are characterized by a lower amount of serum folate. This can be explained both from an economic and a biological perspective: first, the fortification has a stronger effect on whites because their diet is richer in fortified foods (mainly cereals and bread). Second, people belonging to different races/ethnic groups absorb folic acid in different ways. Perry et al. (2004) found that after a controlled supply of folic acid, African American women had the lowest amount of serum folate in the population, followed by Mexican American and Caucasian.

If selection into treatment was not random, the folic acid fortification could have also increased health inequality between different socioeconomic groups. Down et al. (2008) assess the effect of the fortification on health inequalities as reflected in socioeconomic inequalities in nutritional outcomes.[13] The comparison between concentration curves before and after the public policy intervention suggests that, in spite of a general increase of red blood folate levels, differences in folate distribution between the low and high income class still persist. In addition, there was an increase of the burden of deficiencies in folate affecting the lower socioeconomic group.

[10] http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5317a3.htm

[11] The authors claim that this can be due to the change in the amount of fortificant contained in bread, which lowered in the years 2002 and 2003.

[12] It is worth noting that the study does not consider a control group before the fortification.

[13] Considering the level of red blood cell folate and using NHANES data.

Finally, with respect to the inequalities between ethnic groups, the prevalence ratio for non Hispanic blacks compared to non Hispanic was found to have increased as a result of the fortification.[14]

There are also concerns about the possible negative effects on health of non-metabolized folic acid, due to an excessive intake of the nutrient and a consequent folate concentration far above the optimal level (see Whittaker, 2001).[15] A high level of folic acid has an ambiguous effect on cancer formation (Choi et al, 2000 and Wien et al, 2011, for a recent systematic review) and could increase neurological deterioration of patients affected by anemia (Dickinson, 1995). In addition, further research is required about the interaction between deficiencies in folate and deficiencies in vitamin B12 (see Smith, 2007). The percentage of people estimated to have passed the upper threshold of optimal serum folate ranges between 67% and 95%, depending on ethnicity, sex and age (Lewis et al, 1999). Thus, it is not clear whether or not it is advisable to expose the entire population to an increased or even excessive amount of folic acid in order to protect only a part of the population that really needs it (Smith et al, 2008). Determinants of overconsumption are not yet clear. Some research has shown that after the fortification, people started consuming significantly more folate than expected because some cereals had been fortified by more than the amount reported on the nutritional labels[16] and given that people often eat more than one serving of cereals (Quinlivan et al, 2003). On the contrary, according to Yang et al. (2009), who use NHANES 2003/2004 and NHANES 2005/2006 data, a concentration of folate exceeding the upper level was found to be present only among supplement users and not among people who consumed fortified grain products without taking supplements.

3) Empirical analysis

3.1) Data and descriptive analysis

We use data from successive waves of the National Health and Nutrition Examination Survey (NHANES III, NHANES 1999/2000, NHANES 2001-2002 and NHANES 2003-2004), a repeated cross section, representative of the US population that contains very detailed information on health status and concentration of nutrients in the blood, including the concentration of serum folate in individuals aged 0 to 90. We also have a dietary recall module to investigate nutritional habits.

We compare the concentration of serum folate with the one of vitamin C[17] at the aggregate level to investigate potential changes in diet as a consequence of the fortification. In fact, the increase in serum folate can be due either to the fortification, or to a change in eating patterns with individuals eating more greens and leafy vegetables over time. If the latter is true, we should observe also an increase in the

[14] In this case, the author did not consider the potential negative effect of folic acid exceeding the upper level threshold.

[15] Plasma concentration of Folate above 59 nmol/L is harmful for elderly according to Smith et al (2007) or 20 ng/mL (around 46 nmol/L) according to Yang (2009)

[16] Whittaker et al (2001) measured the amount of folic acid contained in fortified cereals and they found that, in half of the cereals analyzed, it was by far exceeding the labeled measure (about 150%).

[17] Serum folate is measured in ng/mL, Vitamin C is measured in mg/dL and ferritin in ug/dL in μg/dL

average vitamin C concentrations since most vegetables are a rich source of both nutrients. On the contrary, if a change in nutrition habits took place independently from the government advice, and it was due, for example, to a change in the structure of prices, we should observe also a change in the distribution of nutrients not contained in the recommended vegetables and products. That is why we studied the change of ferritin, which is not contained in folate-rich food.

We first use the changes in concentration of the three nutrients to assess the second order effect of the public policy: the change in inequality of serum folate concentration, which is a proxy of the changes in health inequality as a consequence of the policy.[18] We also compute the average concentration of serum folate a, vitamin C and ferritin to assess the trend in the measures. We also evaluate the inequality by calculating Generalized Lorenz curves, Gini coefficients and Atkinson indices for the concentration of serum folate and vitamin C for years before and after the fortification.

Tables 1-4 provide descriptive statistics for the sample: since in the 1988 wave does not have relevant measures (around 500 individuals interviewed), we consider 9 different waves for serum folate (1989, 1990, 1991, 1992, 1993, 1994, 1999, 2001) and 8 for vitamin C (1989, 1990, 1991, 1992, 1993, 1994, 2003). Given that the governmental action took place in two different points in time, in our descriptive analysis we separately assess the effects of the first phase (after 1992) and of the second one (after 1998).

Figure 1 displays the trend in serum folate and vitamin C. Although both graphs show an increasing trend in the nutrients, the growth of vitamin C is slower and smoother. Moreover, folate concentration in the blood reaches its maximum in 1999 data (right after the start of the fortification) and it decreases a little in the following years. The same pattern cannot be found in the vitamin C data. Figure 2 displays the cumulative distribution of serum folate and vitamin C. We want to assess whether the fortification produced only a shift in the curves, whether or not it was more effective for depleted individuals or it decreased the health inequality in the population. Another interesting issue is whether or not the grain product fortification changed the relative position of some subgroups in the distribution. The first hint the cumulative distributions give us is the following: while there was a shift in both the distribution of serum folate and the distribution of vitamin C, the consumption of products rich in ferritin seems not to have changed, thus suggesting that a folate related dietary change took place. Figure 3 displays the generalized (i.e. scaled up to the mean) Lorenz curves for the three nutrients and shows the effect of the policy on inequality. It confirms that there was a decline in the level of inequality of folate and vitamin C concentration,[19] while no improvements in inequality can be observed when the ferritin distribution is analyzed. Figure 4 shows the change in inequality, measured by means of Gini indices, during the three steps of the fortification. We can easily notice that the first phase of the intervention caused, together with a slight

[18] We can disregard the possible effects of a change in use of dietary supplements, because, according to the centers of disease control and prevention (CDC), data have not indicated a substantial change in supplements use as a consequence of the fortification. In addition, empirical evidence based on NHANES data shows that the consumption of dietary supplements decreases as a consequence of the supplementation (Benthley et al, 2006)

[19] As regards both serum folate and vitamin C, we can notice that the after fortification distributions always Lorenz-dominates the before fortification ones. This implies that it also second order stochastic dominates it. The former statement implies that all the individuals agree on considering the after fortification distribution preferable to the situation before the public health intervention, both in an efficiency (considering the mean) and in an equity (considering the inequality) point of view.

increase in the mean, a greater inequality. From a policy point of view, it would be interesting to assess whether this result is due to a greater focalization of the public policy (e.g. women were more interested in the campaign) or to a different responsiveness of different subgroups of the population due, for example, to different levels of education achieved. Table 5 provides a summary of the inequality before and after fortification for the overall population both by gender and by race-ethnicity. We are interested in inequality between races-ethnicities because the incidence of NTDs prior the fortification was different for different racial/ethnic groups. For example, according to the report 6 of the Council of science and public health, children born from Hispanic mothers were 1.5 times more likely to be affected by spina bifida. Since the products fortified are not very common in the Latin-American diet, we are concerned that the fortification increased once more the gap between these socioeconomic groups. Figure 5 shows the results of the analysis of within-group and between-group inequality, performed using Atkinson indices with parameter equal to 2 (in order to give more importance to changes in the bottom of the distribution).[20] We have considered 3 different partitions of the sample (by race-ethnicity, by gender, by target population), where, as target population, we defined women in childbearing age, i.e. between 15 and 45. We have also split the sample into three different points in time: before 1992 (before the government started to solve the problem of NTDs), between 1992 and 1998 (between the beginning of the campaign and the beginning of the fortification), and after 1998. The graphs suggest that the first stage of the policy intervention (the awareness campaign) increased the differences in serum folate concentration, while subsequent fortification (a less targeted policy) decreased it. Figure 6 shows the increase of serum folate concentration for different ethnic-racial groups. We can notice that before the beginning of the fortification, the "prevalence" of depleted individuals is not equal across ethnic groups, genders and level of education. The descriptive analysis on the entire sample before the fortification (NHANES III), contained in table 7, reveals that men were more depleted than women, and high educated people more than low educated ones. Finally, the number of depleted individuals among black people is the highest followed by the number among Hispanic.

3.2) Econometric specification

We consider as "treated" those people who declare in the dietary recall interview collected by NHANES to have eaten RTE cereals. We recognize that dietary recall data are affected by measurement error due to imperfect recall and due to the less than exact identification of both the recipes of the food consumed and their composition in terms of nutrients. However, we are confident that the individuals interviewed can at least remember whether or not they had eaten RTE cereals. In addition, even if we observe an individual just once, we think that the breakfast habits are pretty stable and that their variation can be considered random.

Since the selection into "treatment" is likely not to be random, to find a counterfactual, two assumptions are needed[21]

[20] We chose to use this index instead of the Gini because the latter is sensible to changes in the median and we are not particularly interested in them

21 Y_1 and Y_0 are the outcomes (the serum folate concentration) of individuals in the treatment and the control group)

Assumption 1: unconfoundedness or **conditional independence** assumption, **CIA**: there is no selection into treatment based on unobservables once controlled for observable characteristics X, formally

$$Y_0 \perp D_i \mid X \quad (1)$$

If assumption (1) holds, we are able to identify the "average treatment effect on the treated" (ATT). However, In order to identify the "average treatment effect on the entire population" (ATE)[22], we need a stronger assumption, i.e. once controlled for the covariates X there is no selection into treatment based on idiosyncratic gains. Namely, we assume that

$$Y_0, Y_1 \perp D_i \mid X \quad (2)$$

We use a propensity score to model the probability of being in the treatment group given the observable characteristics via propensity score,[23] so that (1) and (2) become

$$Y_0 \perp D_i \mid P(X) \quad (3)$$

$$Y_0, Y_1 \perp D_i \mid P(X) \quad (4)$$

Under (3) and (4), it is possible to select a subgroup of the control group which is similar to the treatment group and, therefore, it can serve as a counterfactual.

Assumption 2: Common support assumption: control and treatment group rely on the same support

$$0 < P(D = 1|X) < 1 \quad (5)$$

This means that, for the same value of the propensity score P(X), there is a strictly positive probability to find both an individual belonging to the control group and an individual belonging to the treatment group. If (5) holds, we can estimate the average treatment effects for each value of the propensity score and integrate them over the distribution of the covariates. In the first part of the paper we identify the effect of the fortification of RTE cereals mimicking the counterfactual by matching treated individuals to comparable ones belonging to the control group, using two different matching procedures (nearest neighbour matching and kernel matching).

Since it is not possible to directly check the validity of the CIA assumption, we will support it by considering the descriptive evidence presented in figures 1 and 2 and, in general, in the previous section. Figure 1, shows a peek in serum folate and vitamin C concentration in 1992, the year in which the awareness campaign started. It means that, at least initially, the governmental intervention raised the awareness of the risks and the determinants of NTDs, thus inducing a change in people's nutritional behaviors. Therefore, we can assume that people had sufficient information to consider folate deficiencies as an issue and that the selection into treatment was not a random process, but it can be seen as a selection based on observables characteristics such as gender, age and race-ethnicity and level of education. Recall that the main benefits in assuming folic acid are achieved by women in fertile age, since folic acid decreases the probability to give birth to children affected by NTDs. We thus expect that young women in a stable relationship are more willing to increase their amount of serum folate, while the campaign does not significantly affect old men. Finally, we can speculate that more educated people are more able to understand the benefits of the treatment and so they select into the treatment with a higher probability. On the

[22] ATE is the outcome we have if individuals were randomly assigned to the treatment
[23] See Rosenbaum and Rubin (1983)

contrary, we can rule out self-selection based on unobserved idiosyncratic gains,[24] given people are not aware of their capacity of metabolizing folic acid. Personal taste in food is another important factor affecting the selection into treatment. Given that the nutritional habits are likely to be ethnicity specific, we believe we can partially control for it by including a dummy variable indicating the respondents' racial-ethnic group. Another part of the effect is random and due to personal tastes, but it is not correlated to the outcome, so it does not bias our estimates. In conclusion, we decided to regress the dummy variable indicating the selection into treatment on a set of covariates such as age[25] (and its higher order terms), ethnic-racial group, gender, educational level and their interactions. At this stage, we do not address the issues of possible changes in nutritional habits induced by the fortification, which will be discussed in one of the next sessions. The common support assumption does not create any problem, since almost all the observations are on support, so we can potentially match all the individuals in the control group and we do not miss any important piece of information by excluding observations from the matching procedure.

Additionally, we measure the effect of the treatment on different quantiles of the distribution. We are interested in the quantile effect of the fortification because a well balanced policy, aiming at decreasing the prevalence of folate deficiencies, would mainly affect the lower quantiles of the distribution (in which the folic acid depleted individuals are contained) and it would leave the higher quantiles almost unchanged, thus avoiding overconsumption. To assess the distributional effect of the policy, we first test whether our model can be seen as a *"common effect model"*,[26] i.e. whether the program has the same effect on everybody. Previous evaluations suggest that this is not the case, given that different racial groups and age categories have different capacities of metabolizing folic acid. In addition, the amount of the treatment (in our case the amount of RTE cereals eaten) varies between individuals. Testing the hypothesis of equal treatment is crucial for two reasons. First of all, the fortification and, more specifically, the amount of folic acid added to the RTE cereals rely on the underlying assumption that individuals eat a serving of cereals a day, which implies that the actual effects in the case of a failure of the equal treatment hypothesis could be very different from the predicted ones. In addition, if the common effect assumption holds, the average effect is equivalent to the quantile treatment effect for each quantile of the serum folate distribution, implying that the only parameter of interest is the average treatment effect itself. One of the possible ways of testing the validity of the common model assumption consists in computing the variance of the treatment, where a variance equal to zero confirms the common effect hypothesis. In principle, our test would not be able to rule out the common effect hypothesis, since we do not know the structure of the dependence between the potential outcomes. However, we are able to identify the values of the correlation between the potential outcomes Y_0 and Y_1 for which it is possible to reject the common effect model assumption.

We can define the variance of the our treatment ($\Delta = Y_1 - Y_0$) as

$$Var(\Delta) = Var(Y_1 - Y_0) \qquad (6)$$

[24] In this case the output of the treatment is the serum folate concentration.

[25] We use education as a proxy of SES, since the income is poorly measured and the variables has a large number of missing values.

[26] See Heckman et al. (1997)

We can write (6) as:

$$Var(\Delta) = Var(Y_1 - Y_0) = Var(Y_1) + Var(Y_0) - 2Cov(Y_1 Y_0) \quad (7)$$

$$Var(\Delta) = Var(Y_1 - Y_0) = Var(Y_1) + Var(Y_0) - 2\rho\sqrt{Var(Y_1)Var(Y_0)} \quad (8)$$

We can thus derive the values of ρ under which the variance of the treatment is equal to zero. Notice that $Var(Y_1)$ and $Var(Y_0)$ are observable, since they can be derived by the moments of the marginal distributions, while some other elements, such as $Cov(Y_1 Y_0)$, are unobservable.

Further econometric problems arise when one wants to estimate the treatment effect considering characteristics of the distribution other than the mean, e.g. when one wants to estimate the treatment effect on different quantiles of the distribution. The first conceptual difficulty arises in the definition of quantile treatment effect itself. We can define it in two ways. The first is *"the difference between the treated and the control group in quantiles of the marginal distribution"* (Firpo, 2007*)*, which considers the effects of the policy at the aggregate level. The second is the effect of the policy on people belonging to a specific quantile of the not treated distribution, ie. the quantiles of the distribution of treatments. The two definitions are equivalent if *"rank invariance"* holds, i.e. if individuals maintain the same position (rank) in both distributions of potential outcomes. In this case, the QTE is just the horizontal distance between the quantiles of the two marginal distributions.

Unfortunately, rank invariance is a very strong assumption. Heckman et al. (1997) makes assumptions about the unobserved conditional distribution of the potential outcomes Y_0 and Y_1 while Firpo(2007) focuses on the study of the effect of the policy on quantiles of the marginals, which is still very relevant from a point of view of policy.

Following Firpo (2007), the univariate marginal distributions of the treatment and the control groups can be expressed as $Y_1 = (q_{1,\frac{1}{p}}, q_{1,\frac{2}{p}}, ...q_{1,1})$ and $Y_0 = (q_{0,\frac{1}{p}}, q_{0,\frac{2}{p}}, ...q_{0,1})$ and their difference can be weighted using parameters $a_{\frac{j}{p}}$, which reflect the importance the policy maker attaches to different quantiles.

Therefore, the policy maker can decide to implement the treatment if

$$Y_1 - Y_0 = \sum_{j=1}^{P} a_{\frac{j}{p}} (q_{1,\frac{1}{p}} - q_{0,\frac{1}{p}}) \geq 0 \quad (9)$$

For given values of the weight $a_{\frac{j}{p}}$.

In this framework, the policy maker is not interested in which individuals benefit from the reform, but just in the number of people who actually do. In our case, Firpo's QTE identifies whether or not the effect of the policy was stronger in the lowest quantile of the distribution, but it does not identify the specific individuals who benefit from it.

Following Firpo (2007), we define the quantile treatment effect and the quantile treatment effect on the treated as:

Quantile treatment effect:

$$\Delta_\tau = q_{1\tau} - q_{0\tau} \quad (10)$$

$j = 0,1$ Where $q_{j\tau}$ is $\Pr[Y(j) \leq q] = \tau$ and where j = 1 indicates the distribution of

the treated population)

Quantile treatment effect on the treated

$$\Delta_{\tau|T=1} = q_{1\tau|T=1} - q_{0\tau|T=1} \quad (11)$$

Where $q_{j\tau|T=1}$ is $\Pr[Y(j) \le q \mid T = 1] = \tau$ and $j = 0,1$

For our estimation we use the semi-parametric two steps procedure proposed by Firpo (2007): in the first step we compute a propensity score estimating the probability of being treated based on observable characteristics, while in the second step we perform a quantile treatment effect estimator, which uses the results of the propensity score to correct for the selection into treatment.

Since we do not know the joint distribution of the control and the treatment, but just their marginal Y_0 and Y_1, in the first step we compute the propensity score non-parametrically, by using the following kernel function.

$$K_{h,\lambda}(X_i - x) = \prod_{q=1}^{q1} k\left(\frac{X_{q,i} - x_q}{h}\right) \prod_{q=q1+1}^{Q} \lambda^{1\left(X_{q,i} \neq x_q\right)} \quad (12)$$

Where the q_1 regressors are continuous (in our case age, age squared and the beta-carotene concentration) and $Q - q_1$ are discrete (ethnic dummies, education and gender). The term $\prod_{q=1}^{q1} k\left(\frac{X_{q,i} - x_q}{h}\right)$ is a standard product kernel for the continuous variables, while the term $\prod_{q=q1+1}^{Q} \lambda^{1\left(X_{q,i} \neq x_q\right)}$ measures the mismatch between the discrete ones. If h=∞ and $\lambda = 1$ the model is estimated parametrically, since the bandwidth h consists in the entire interval. The optimal values of the smoothing parameters h and λ are chosen by cross validation.

In the second step we need to minimize the following function, for j=0,1

$$\hat{q}_{J.\tau} = \arg\min_{q} \sum_{i=1}^{N} \hat{\omega}_{j,i} \rho_\tau (Y_i - q) \quad (13)$$

Where the check function $\rho_\tau(.)$ is

$$\rho_\tau (Y_i - q) = (Y_i - q)(\tau - I\{Y_i - q \le 0\})$$

And the weights are defined as $\hat{\omega}_{1,i} = \frac{1 - T_i}{N(1 - \hat{p}(X_i))}$ and $\hat{\omega}_{1,i} = \frac{T_i}{N\,\hat{p}(X_i)}$ and N is the sample size

The function (13) is similar to the function used in the standard quantile regression framework, but its arguments are weighted by the inverse of the propensity score computed in the first stage.[27] Given that we use the propensity score to weight the observations, our quantile treatment effect estimator identifies unconditional quantile treatment effects under the assumption of selection into treatment based on observable characteristics X, i.e. the treatment is considered exogenous conditioning on X. In particular, the quantile treatment effects are identified under strong

[27] The effect is estimated by using the command developed by Frolich and Melly (2008).

ignorability (Unconfoundness and common support) and under the additional assumptions of existence and uniqueness of quantiles (see Firpo, 2007).

The other way to deal with the lack of information due to the not observability of the conditional distribution of the potential outcomes, consists in computing the quantile treatment effects under alternative assumptions about the joint distribution of the potential outcomes, i.e. about the dependence of the two marginals. One of those assumptions, the strongest possible is precisely *rank invariance*, i.e. the perfect positive correlation between Y_0 and Y_1. Following Heckman et al. (1997), we proceed as follows. We compute the propensity score and we divide the sample into quintiles according to the value of the propensity score. Then we compute 50 quantiles of the two distributions and, within each propensity score stratus, we collapse[28] the observations into quantiles in order to have the same number of observations in the treatment and the control group. We then derive the treatments effects under perfect positive correlation and perfect negative correlation of the marginal distributions computing the horizontal distance between the two values and integrating it over the distribution of the propensity score. The cases of perfect positive and perfect negative dependence are just two possible ways the two distributions can be related. To estimate the full distribution of the treatment, given all the possible rearrangements of the individuals within the groups, one should consider all the n! permutations (in our case 50!) of the treatment group and then compute the effect of the fortification considering all the horizontal differences in the n! random permutations. However, in order to decrease the computational burden, we consider just 1000 random permutations of the quantiles of the distribution Y_0 (within each propensity score stratus).

3.3) Results[29]

We first analyze the effect of the fortification by simply comparing the mean value of serum folate among the RTE cereal eaters (around 30% of the sample) to the average concentration of serum folate among the non RTE cereal eaters in the full sample[30]. We found a huge and significant difference between the two groups of about 9,8 nmol/L (see table 8), suggesting a clear and strong effect of the fortification. However, such a naïve estimator does not measure the correct causal effect of the policy, given that the treatment and the control groups are not similar with respect to several dimensions, namely race-ethnicity, age and education (see table 15, when the unmatched sample is considered). In the "treated" group we cannot find any depleted individual, hence implying the supplementation have almost eliminated the cases of individuals at the high risk of giving birth to a baby affected by NTDs.

As expected selection into treatment is an issue (results are shown in table 9) and people belonging to ethnic minorities (Black, Mexican-Hispanic and other ethnic groups) are more likely to be included in the control group, while, even controlling for the race-ethnicity variable, more educated people eat more fortified cereals. Comparing these results with the descriptive evidence contained in table 7 could help understand how the choice of RTE cereals affected inequality in health. Selection into

[28] We considered the mean value within each stratus.

[29] All the analyses are conducted using SI units (nmol/L) in order to compare the results with the findings of the literature.

[30] We performed the analysis also on the sub sample of women above 10 years old and the results are qualitatively similar.

treatment seems to make race-based health inequality in health even stronger. In fact, Black and Hispanic people were the most affected by low concentrations of serum folate before the intervention and the ones that consume RTE cereals the least. If selection into treatment is correlated to the probability of becoming pregnant, the fortification of RTE cereals does not help lowering the prevalence of NTDs in unwanted pregnancies. In addition, our estimates show that selection into treatment is not a gender driven process. The result can be due to the fact that nutritional habits are similar within households, which are likely to be homogeneous with respect to age, ethnic/racial origin and level of education, but not with respect to gender. Finally, none of the interaction terms is significant, but this can be due to the not linearity of the parametric form chosen for the estimation of the propensity score in the first part of the analysis (probit specification).

We computed three different effects: ATT, ATE and ATU. The potential effect on the untreated is the greater in magnitude, ie. the policy would potentially benefit the individuals in the control group more than the ones in the treatment group. It means that there is no selection based on idiosyncratic gains. This can be due to the fact that people who are concerned about their level of folate (or who have a better diet) have a higher probability to be selected in the treatment group. We thus expect that people who get folic acid from cereals get more serum folate from diet as well. To check the robustness of our estimates, and given the higher number of people included in the control group, we use two types of matching: a nearest neighborhood matching (one-to-one) and a kernel one. As expected, the effect obtained using the kernel matching is lower than the one obtained with the nearest neighborhood one. In fact, the kernel matching matches each treated individual with more than one person in the control group, so also observations different from the treated individuals are considered (although with a lower weight). We propose different tests to assess the quality of the two procedures. We perform a set of t-tests to compare the means of the variables in the two groups (see table 15 for the kernel matching) and in none of them we are able to reject the null hypothesis of equal means after the matching, thus indicating that the treatment and the control group are well balanced. In addition, the R squared of the regression on matched covariates is lower than the one on the unmatched sample (and very low in absolute terms) suggesting that, after controlling for observables, the selection into treatment is mainly random and, therefore, it should not bias our results. Both matching procedures give us comparable results. The results are presented in tables 11 and 12. Conditioning on the covariates reduces the estimated effect of the fortification, which is lower than the one found when we naively compare the means of the two groups (recall that the difference in average serum folate concentration between the two groups was 9.71).

Table 15 shows that the variance of the treatment is above zero for every value of the correlation between the two distributions, even when the perfect negative dependence (lower bound of the variance of the treatment) is assumed. Unambiguously, this rules out the hypothesis of equal treatment, because both the minimum and the maximum possible variances are well above zero.

The quantile treatment effects estimated using Firpo's (2007) estimator (see table 18[31] and figure 8) confirm the impact of the treatment is not homogeneous across the serum folate distribution. For all the quantiles considered the effects are

[31] In order to have a more parsimonious description of the effect, we present also the estimates of the QTE when only three main percentiles (25th, 50th and 75th) are taken into account. Results are shown in table 19.

estimated with precision, given they are all significant at the 1% level. The part of the distribution less affected by the policy is the bottom one, where the treatment effect is just between 2 and 5. The estimated quantile effect is growing in the left part of the distribution and it is quite flat near the median, around which the increase seems to stop. On the contrary, it increases sharply around the 75th-80th percentile, thus suggesting that the policy could have provoked overconsumption without consistently increasing the serum folate concentration of the depleted individuals. The not complete linearity of the effect could be due to two different forces in act. People at the top part of the distribution are those who are more likely to eat foods rich in folate/folic acid. As a consequence, if they do not adjust their dietary habits when the fortification takes place, they are likely to incur in the risk of overconsumption. On the contrary, people in the center of the distribution are probably used to a lower consumption of such foods and they could decrease their intake of dietary folate as a consequence of the greater availability of chemical folic acid at a lower price.

Table 21 presents the principal moments of the distribution of the impacts under different assumptions on the dependency between the marginal distributions. The analysis of the table corresponding to the case of perfect positive correlation confirms the results obtained by using Firpo's estimator,[32] with the effect of the program increasing with quantiles. However, when we impose a negative correlation, some of the effects change their sign and the policy turns out to be more effective for the depleted individuals. It is not surprising given that there is perfect negative dependence between Y_0 and Y_1, and given that high values of Y_0 are associated to low quantiles of Y_1. This means that the policy was increasing the amount of serum folate of those at the bottom of the distribution and it was decreasing the serum folate concentration of those at the top of it. This result does not contradict our previous findings obtained using Firpo's estimator, since it can happen that the fortification increased overconsumption overall, but the individuals swap their places in the serum folate distribution. A somehow intermediate situation is found when the quantiles of the two distributions are randomly matched.[33] In this case the distributions of the effect are similar for different quantiles, as shown in figure 9 and in table 22.[34]

Let us now consider the assumption of rank invariance. If we assume negative dependence between the potential outcomes it means that the individuals with a low folate intake before the beginning the fortification realize that they have to increase their serum folate concentration and they try to consume folate/folic-acid rich foods. On the contrary, people who were not depleted, lower their previous intake. Assuming positive dependence means that, those who were eating dietary folate rich foods before the beginning of the governmental intervention do so also after its beginning.

3.4) Tackling the problem of changes in dietary habits

In this section we correct our estimates taking this effect into consideration. Data on nutrients consumption are both included in the dietary recall file and they can be indirectly derived by the analysis of their concentration in blood. Given the low

[32] They are not perfectly equivalent because, in Firpo's case we used the propensity score to weight the observations, here they are used to stratify the sample (and the effect is an average of the effects in the different samples).

[33] Recall that it takes place within the propensity score cells.

[34] Note that, for sake of simplicity, the graph consider quantiles of the distribution of the control group, so that the effect can be interpreted as the expected effect on a particular quantile of the distribution under study before the implementation of the policy.

reliability of data on food intake due to measurement error, we decided to use the information contained in the examination files of the survey.

Our identification strategy relies on the (partial) co-movement of vitamin C and folate due to their being both contained in green leafy vegetables. In section 3.2 we showed that the average concentration of both vitamin C and folate in blood of NHANES members increased after the awareness campaign started in 1992. Unfortunately, in the NHANES 99/00 dataset the information on the concentration of vitamin C in blood is missing so that another approximation must be found. There are three possible "candidates" as proxies of consumption of food rich in folate. These are vitamin A, vitamin E and beta-carotene, which all turn out to be correlated with the serum folate concentration, even before the fortification. We chose to control for beta-carotene concentration because fortified cereals are added with both vitamin A and vitamin E, thus making them bad proxies for changes in nutrition independent from the consumption of RTE cereals. We control for dietary folate intake in two different ways: parametrically, using beta-carotene as an explanatory variable in the matching procedure, and not parametrically, stratifying our sample by quintiles of beta-carotene distribution. Although the beta-carotene variable is not highly significant in the first stage of the matching (see table 10), it is as expected positively correlated to the selection into treatment and its inclusion decreases the size of the estimated average treatment effect of the folic acid supplementation (see tables 13 and 14). Notice that the inclusion of beta-carotene as an explanatory variable has two different aims: the first one is studying the changes in diet correlated with the selection into treatment; the second one is simply washing out the amount of serum folate due to the intake of dietary folate. Even if the evidence of the impact of beta-carotene on the selection procedure is not striking, the second effect is still important. The results confirm our previous findings, i.e. that part of the average increase in serum folate concentration is due to a change in the consumption of vegetables containing dietary folate. The same results are obtained when we compute the quantile treatment effects *a' la Firpo* (2007). Also in this case, the estimated impact of the fortification is reduced,[35] in particular in the top part and the bottom part of the serum folate distribution, while no effect is found around the median (see table 20). This, again, supports our idea of a possible substitution between the two sources of folate.

4) Conclusions

Early analyses have found the American folic acid fortification to be successful, with a large increase in serum folate in the population. However, further investigation pointed to the possible negative effects of the policy, leading to overconsumption of folic acid for given groups of the population. We first conducted a descriptive analysis using different waves of the National Health and Nutrition Examination Survey (NHANES III, NHANES 1999/2000, NHANES 2001/2002 and NHANES 2003/2004) covering the time span between 1988 and 2003, to understand the impact of different phases of the policy intervention. We found an increase in serum folate concentration right after the start of the awareness campaign on the importance of a high folic acid intake. In the same time period, we also found an increase of vitamin C. These stylized facts suggest that the change in total folate in blood can be decomposed into a change in serum folate concentration caused by the folic acid fortification and a change in dietary folate intake, due to modified dietary

[35] Once compared to the one estimated when dietary habits have not be taken into consideration

habits, proxied at the aggregate level, by the concentration of vitamin C. In addition, the different impact of the fortification on different ethnic groups raised the problem of a possible endogenous selection into treatment and a possible heterogeneity in the effects.

We focused on the fortification of RTE cereals, which are one of the main sources of folic acid after the compulsory fortification in1998, and we considered the time span 1999-2000. We exploit the rich source of information contained in the NHANES data and we computed the average treatment effects controlling for selection on observables and for different dietary patterns. We included the effect of diet by using the measured concentration of beta-carotene in blood. We found a strong evidence of ethnic-race and education based selection into treatment, confirming the stylized evidence presented in the first part of the paper and implying a possible increase in health inequalities. Consequently, controlling for endogenous selection into treatment reduced the estimated average treatment effect of the folic acid fortification that we found when we just naively compared the serum folate concentration in the treatment and in the control group. This finding, together with the analysis of the trends by subgroups, implies that the intervention did not reach all the strata of the population in the same way. We also found that part of the average increase in serum folate concentration can be explained by changes in diet, as suggested by our descriptive analysis and as pointed out by part of the literature. This confirms our idea that behavioral effects played a role in determining the difference in serum folate concentration, before and after 1998.

The core of the paper focuses on the estimation of heterogeneous treatment effects. After rejecting the hypothesis of common effect model, we moved to the distributional effect of the fortification, investigating the impact of the policy on different quantiles of serum folate concentration by computing the semi-parametric quantile treatment effect developed by Firpo (2007). We observed a stronger impact of the policy on the right tail of the distribution, meaning the decrease in the prevalence of serum folate deficiencies was accompanied by an increase of overconsumption. This result holds even when dietary habits are taken into account. Finally, relaxing the rank invariance assumption on which Firpo's estimator relies, we were able to link the effects of the policy to the change in individual behaviors, as a response to the change in folic acid availability.

References

Bitler M. Gelbach J and Hoynes H, (2006). "What Mean Impacts Miss: Distributional Effects of Welfare Reform Experiments," American Economic Review, American Economic Association, vol. 96(4), pages 988-1012, September

Bitler M. Gelbach J and Hoynes H, (2008). "Distributional impact of the Self-Sufficient Project" Journal of Public Economics 92, 748-765

Benthley TCK, Willet WC, Weinstein MC and Kuntz KM (2006) "Population-level changes in folate intake by age, gender and race/ethnicity after folic acid Fortification". *American Journal of Public health;* 96(11):2040-7.

Caudill MA, Le T, Moonie S, Esfahani S, Cogger EA (2001) "Folate status in women of childbearing age residing in southern California after folic acid fortification". *Journal of the American College of nutrition.* Vol. 20. No2, 129-134

Centers for Disease Control and Prevention: "Recommendations for the use of folic acid to reduce the number of cases of spina bifida and other neural tube defects". MMWR Morb Mortal Wkly Rep 1992; 41: 1-7

Centers for Disease Control and Prevention: "Use of dietary supplements containing folic acid among women of childbearing age. United States 2005". MMWR 1992; 51: 808—10

Centers for Disease Control and Prevention: "Spina bifida and anencephaly before and after folic acid mandate—United States, 1995–1996 and 1999–2000". 362–365. 2004. MMWR. 5-7-2004.

Choi SW, Mason JB. "Folate and carcinogenesis: an integrated scheme" (2000). *Journal of Nutrition.;130:129–32.*

Choumenkovitch SF, Selhub J, Wilson PWF, Rader JI, Rosenberg IH, Jacques PF. (2002) "Folic acid intake from Fortification in united states exceeds predictions" *Journal of Nutrition 132: 2792-2798*

Council of science and public health "Report 6: Folic acid fortification of grain products"

Cuskelly G, McNulty, Scott, J (1999) "Fortification with Low amount of folic acid makes a significant difference in folate status in young women: implications for the prevention of neural tube defects". *American Journal of Clinical Nutrition.* 70; 234-9

Cuskelly G, Mooney K, Young I (2007) "Folate and vitamin B12: friendly or enemy nutrients for the elderly". *Proc Nutri Soc;* 66: 548-58

Dickinson CJ. (1995) "Does folic acid harm people with vitamin B12 deficiency?" *Q. J. Med. 311: 257*

Dietrich, M., Brown, C. J.P., Block, G. (2005). "The Effect of Folate Fortification of Cereal-Grain Products on Blood Folate Status, Dietary Folate Intake, and Dietary Folate Sources among Adult Non-Supplement Users in the United States". *Journal of the American College of nutrition.* 24: 266-274

Daly s, Mills J, Molloy A, Conley M, Lee Y Kirke P et al. (1997) "Minimum effective dose of folic acid for food fortification to prevent neural-tube defects". *Lancet* 350(9092): 1666-1669

Dowd JB and Aiello AE (2008) "Did national folic acid fortification reduce socioeconomic and racial disparities in folate status in the US" *International Journal of Epidemiology. 37, 1059-1066*

Dunlap B, Shelke K, Salem S and Keith L (2011) "Folic acid and human reproduction-ten important issueas for clinicians" *Journal of Experimental and Clinical Assisted reproduction 8: 2*

Firpo, S. (2007): "Efficient Semiparametric Estimation of Quantile Treatment effects". *Econometrica, 75, 259.276.*

Ford, ES, Bowman, B. (1999): "Serum and red blood cell folate concentrations, race, and education: findings from The third National Health and Nutrition Examination Survey". *American Journal of Clinical Nutrition.* 69; 47681

Froelich, M and Melly, B: (2008). "Estimation of quantile treatment effects with STATA". Mimeo.

Ganji, V., Kafai, M. R. (2006). "Trends in Serum Folate, RBC Folate, and Circulating Total Homocysteine Concentrations in the United States: Analysis of Data from National Health and Nutrition Examination Surveys, 1988-1994, 1999-2000, and 2001-2002". *Journal of Nutrition.* 136: 153-158

Grosse D. Scott, N.J Waitzman, P.S. Romano. J.Mulinare (2005) "Reevaluating the Benefits of folic acid fortification in the United States: economic analysis, regulation and public health". *American Journal of public health Vol 95. No 11*.

Heckman J, Smith J, Clemens, N (1997) "Making the most out of programme evaluations and social experiments: accounting for heterogeneity in programme impacts" *Review of economic studies 64, 487-535.*

Hernandez-Diaz S, Werler M, Walker AM Mitchell A (2001) "Neural tube defects in relation to use of folic acid antagonists during pregnancy" *American Journal of Epidemiology. Vol 153. No 10*

Honein MA, Paulozzi LJ, Mathews TJ, Erickson JD and Wong LC (2001) "Impact of folic acid fortification of the US food supply on the occurrence of Neural Tube Defects" *JAMA 285 (23) 2981-2986*

Jacques, P. F. Jacob Selhub, Andrew G. Bostom, Peter W.F. Wilson, and Irwin H. Rosenberg. (1999) "The Effect of Folic Acid Fortification on Plasma Folate and Total

Homocysteine Concentrations". *The New England Journal of Medicine, 340:1449-1454*

James WP, Nelson M, Ralph A, Leather S. (2007) "Socioeconomic determinants of health: the contribution of nutrition to inequalities in health." *British Medical Journal 1997; 314: 1545*

Lewis CJ. Crane NT, Wilson DB. Yatley EA. (1999) "Estimated folate intake: data updated to reflect food fortification, increased bioavailability, and dietary supplement use." *American Journal of Clinical Nutrition. 70:198-207*

Morris MS, Jacques PF, Rosemberg IH, Selhub J. (2007) "Folate and Vitamin B-12 status in relation to anemia, microcystosis, and cognitive impairment in older Americans in the age of folic acid fortification". *American Journal of Clinical Nutrition. 85. 193-200*

Perry CA, Renna SA, Khitun E, Ortiz M, Moriarty D, Caudill M (2004) "Ethnicity and race influence the folate status response to controlled folate intakes in young woman". *The Journal of nutrition. 134(7):1786-92.*

Pfeiffer, C. M, Caudill, S. P, Gunter, E. W, Osterloh, J., Sampson, E. J (2005). "Biochemical indicators of B vitamin status in the US population after folic acid fortification: results from the National Health and Nutrition Examination Survey 1999-2000". *American Journal of Clinical Nutrition.* 82: 442-450.

Quinlivan EP Gregory JF (2003) "Effect of food fortification on folic acid intake in the United States". *American Journal of Clinical Nutrition. 77:221-225*

Rosenbaum, P. R., and Rubin, D. B. (1983), "The Central Role of the Propensity Score in Observational Studies for Causal Effects," *Biometrika* 70, 41–55.

SACN. "Summary of Report to CMO on folic acid and colorectal cancer risk" - October 2009

Smith D. (2007) "Folic acid supplementation: the good, the bad and the puzzle of vitamin B12" *American Journal of Clinical Nutrition. 85:3-5*

Smith D, Kim YI, Refsum, H (2008) "Is folic acid good for everyone?" *American Journal of Clinical Nutrition. 85:3-5*

Volpe Martincus C and Carballo J (2010) "beyond the average effects: the distributional impact of export promotion programs in developing countries" *Journal of Development Economics 92, 201-214*

Yang Q, Cogswell , Hammer, H Carriquiry A, Bailey L, Pfeiffer C Berry, R. (2009) "Folic acid source, usual intake, and folate and vitamin B-12 status in US adults. National health and Nutrition Examination Survey (NHANES) 2003-2006". doi:10.3945/ajcn.2009.28401

Whittaker P, Tufaro P, Rader J (2001). "Iron and folate in fortified cereals". *Journal*

of the American college of nutrition, Vol 20. No 3, 247-254

Wien T, Pike E, Wisloff T, Staff A, Smeland S and Klemp M (2012) "Cancer risk with folic acid supplements: a systematic review and meta-analysis" BMJ Open 2012

Williams LJ, Rasmussen SA, Flores A. Kirby RS, Edmond LD. (2005) "Decline in the prevalence of spina bifida and anencephaly by race/ethnicity: 1995—2002". *Pediatrics 2005; 116: 580—6*

Appendix 1: graphs and tables

NUTRIENT	TOTAL		FEMALES		MALES	
	Mean	obs.	mean	obs.	mean	obs.
Serum folate	11.56	38,881	12.23	20,186	10.85	18,695
Vitamin C	.89	27,572	0.95	14,372	.83	13,200
Ferritin	105.46	42,272	68.32	21,869	144.16	20,403

Table 1: Descriptive analysis of the data by gender

NUTRIENT	TOTAL		WHITE		BLACK		HISPANIC MEXICAN		OTHER	
	mean	obs.	Mean	obs.	mean	obs.	mean	obs.	Mean	obs.
Serum folate	11.56	38,881	11.94	14,637	9.55	10,418	10.86	11,482	11.57	2,344
Vitamin C	0.89	27,572	0.90	10,939	0.82	7,793	0.90	7,536	0.89	1,304
Ferritin	105.46	42,272	106.63	15,640	113.07	11,420	85.92	12,630	102.73	2,582

Table 2: Descriptive analysis of the data by ethic group

NUTRIENT	TOTAL		LOW EDUCATION		HIGH EDUCATION	
	mean	obs.	mean	Obs.	mean	obs.
Serum folate	11.41	38,058	11.43	21,958	11.40	16,100
Vitamin C	0.89	27,563	0.90	14,770	0.88	12,793
Ferritin	107.53	40,72	86.38	24,617	121.23	16,108

Table 3: Descriptive analysis of the data by level of education

NUTRIENT	TOTAL		PHASE 1		PHASE 2		PHASE 3	
	mean	obs.	Mean	obs.	mean	obs.	mean	obs.
Serum folate	11.56	38,881	6.48	11,776	7.99	11,193	15.42	15,912
Vitamin C	0.89	27,572	0.76	10,450	0.81	9,845	0.98	7,277
Ferritin	105.46	42,272	104.12	13,164	105.67	12,452	106.00	16,656

Table 4: Descriptive analysis of the data by steps of the fortification

NUTRIENT	TOTAL	GENDER		RACE			
		MALE	FEMALE	WHITE	BLACK	MX_HISPANIC	OTHER
Serum folate	0.37	0.36	0.37				
Vitamin C	0.37	0.32	0.30	0.32	0.30	0.26	0.26
Ferritin	.053	0.47	0.52	0.51	0.56	0.55	0.56

Table 5: Descriptive analysis of the inequality in serum folate, vitamin C and ferritin concentration (Gini indices)

variable	before the supplementation	after the supplementation	t-test	significance
Folate	7.039609	15.48512	-9.0420	***
Ferritin	102.3719	106.1037	-1.2142	
* p < .10 ** p < .05 *** p < .01				

Table 6: T-test for mean differences

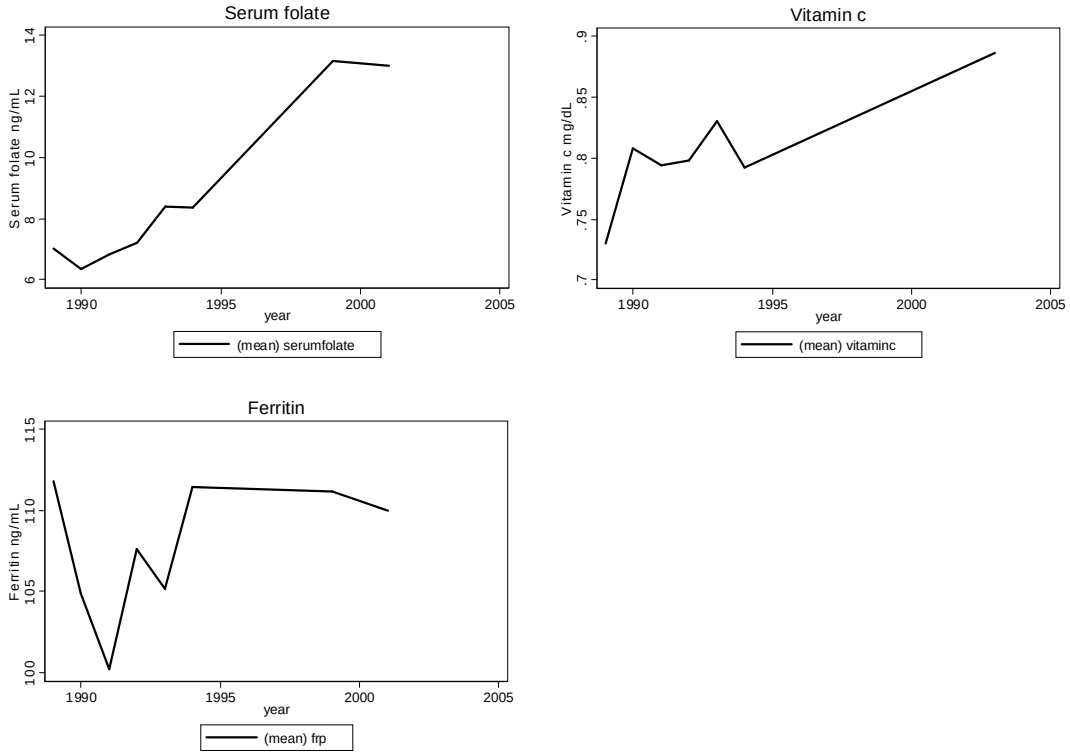


Figure 1: Trends in Average Serum Folate, Vitamin C and Ferritin

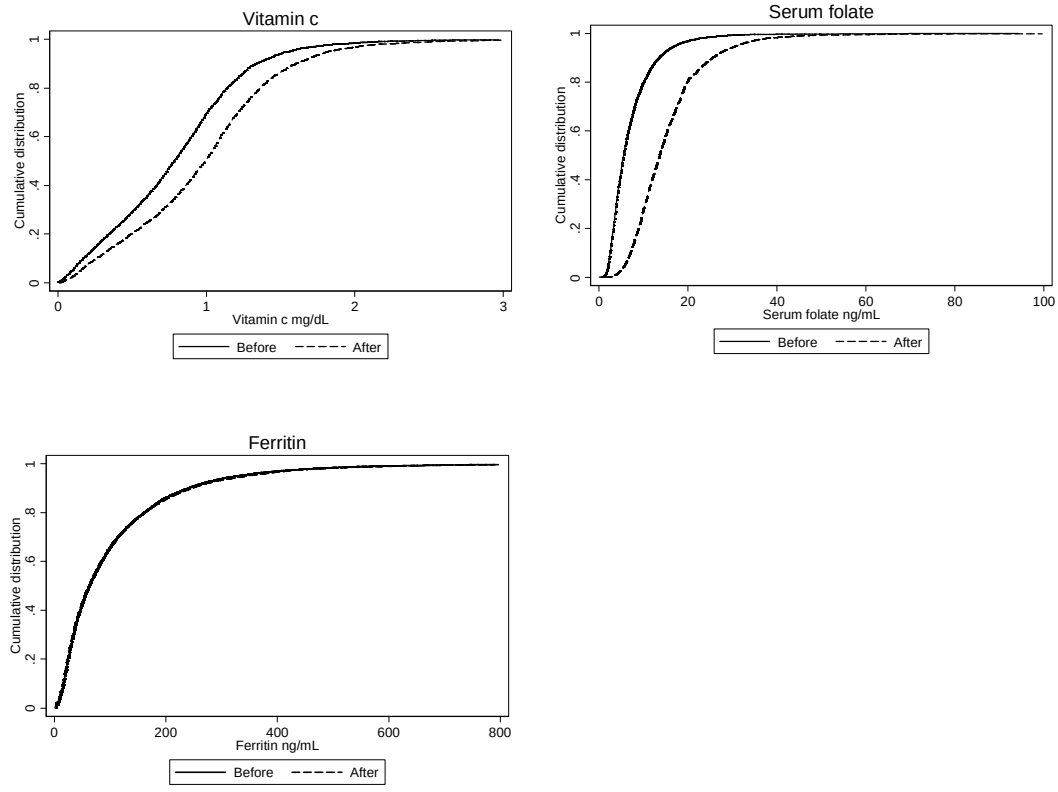
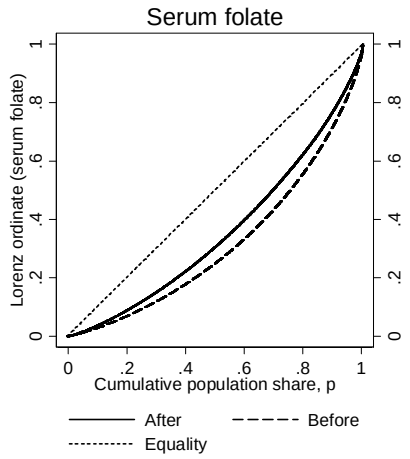
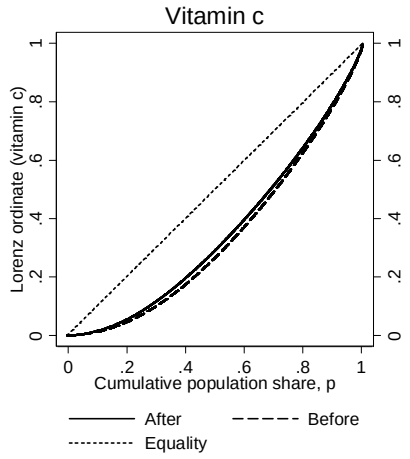


Figure 2: Cumulative Distribution of Serum Folate, Vitamin C and Ferritin Concentration





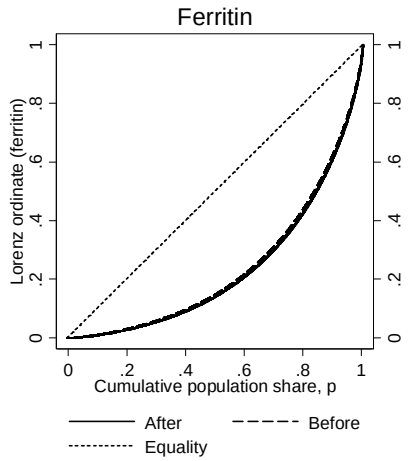


Figure 3: Lorenz Curves for Serum Folate, Vitamin C and Ferritin

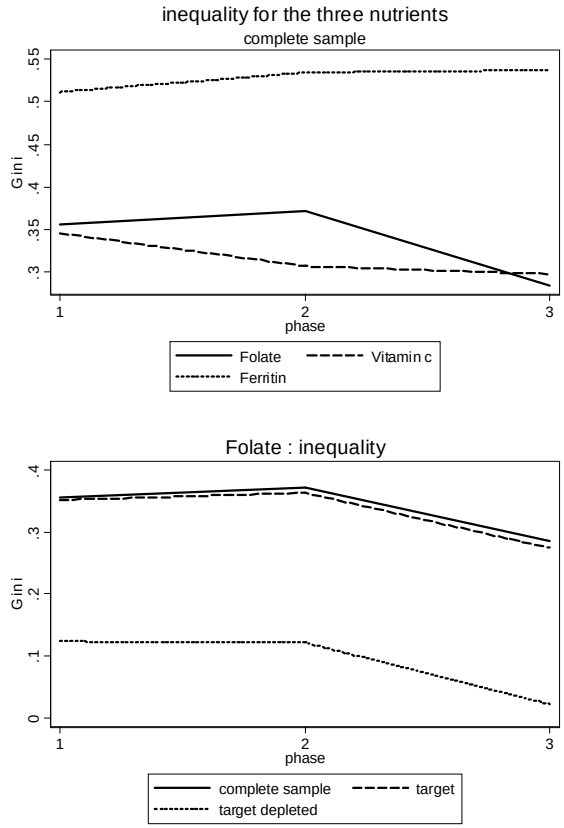


Figure 4: Gini indices dynamics

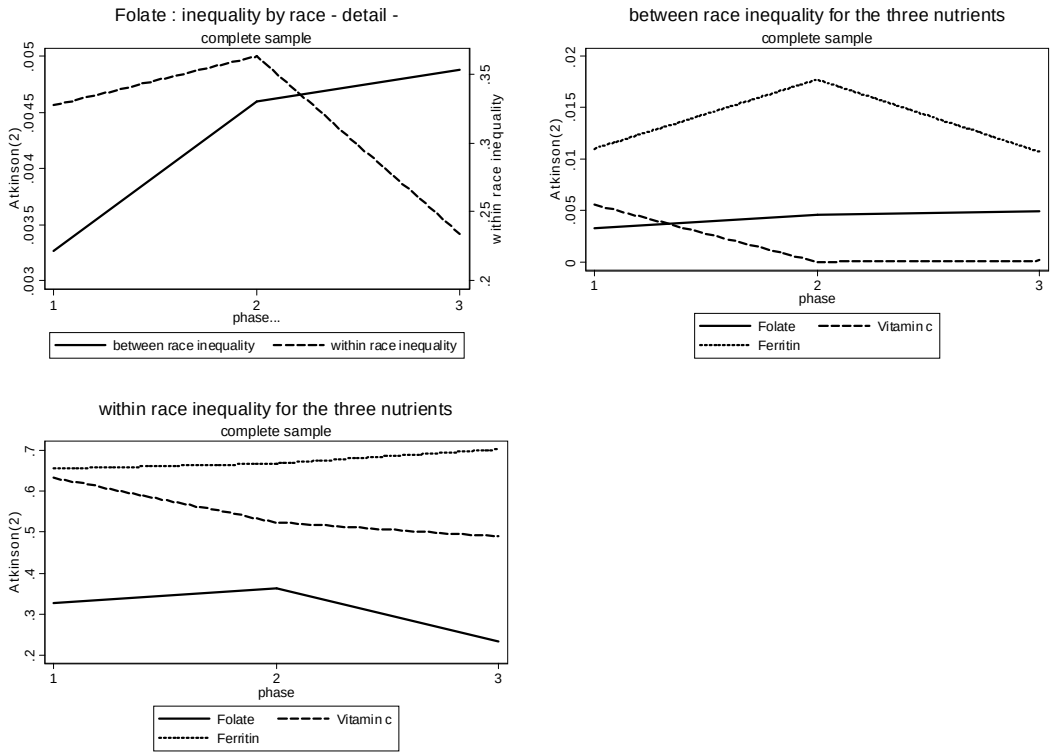


Figure 5: Within and between inequality (Atkinson indices)

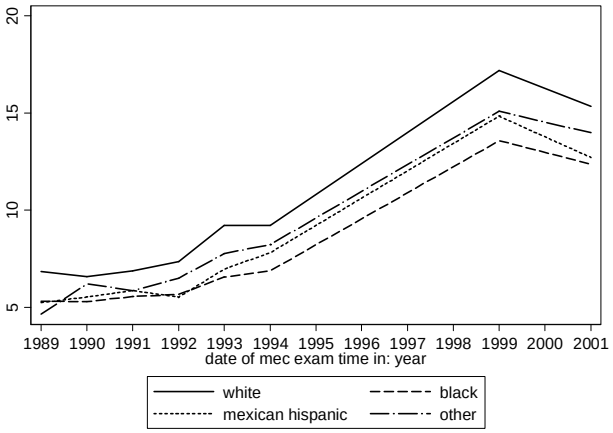


Figure 6: trends for different races

variable	Percentage of depleted individuals
Men	0.176
Women	0.156
High Education	0.181
Low education	0.143
Black	0.233
White	0.158
Mexican-Hispanic	0.179
Other	0.123

Table 7: Prevalence of the depleted individuals in the population (NHANES III)

variable	Control group	Treatment group	t-test	significance
Folate	33.35201	43.18042	-9.828402	***
* p < .10 ** p < .05 *** p < .01				

Table 8: difference in serum folate concentration between between RTE cereal eaters and not RTE cereal eaters (NHANES 1999/2000)

Variable	Coefficient	Standard errors	Significance
Male	-.0354655	.0727737	
Black	-.3572016	.0664819	***
Black_Male	.136818	.0947454	
Mexican-Hispanic	-.3121821	.0608138	***
Male_Mexican-Hispanic	.0652721	.086865	
Other	-.5028332	.0913785	***
Other_male	-.0290933	.1369034	
High education	.1561664	.0563013	***
High education_male	-.0080338	.0759514	
Age	-.0656697	.0037788	***
Age2	.0006857	.0000423	***
constant	.5583536	.0713233	***
Observations			6879
Pseudo R2			0.0569

Table 9: Estimation of the propensity score. Probit regression (not including beta carotene concentration)

Variable	Coefficient	Standard errors	Significance
Male	-0.01815	0.073952	
Black	-0.35257	0.067338	***
Black_Male	0.108445	0.096266	
Mexican-Hispanic	-0.29717	0.061364	***
Male_MexicanHispanic	0.043939	0.08781	
Other	-0.48295	0.091986	***
Other_male	-0.0608	0.138158	
High education	0.155543	0.056912	***
High education_male	-0.01076	0.076891	
Age	-0.06576	0.003833	***
Age2	0.000682	.0000431	***
Beta-carotene concentration	0.001909	0.001156	*
constant	0.532494	0.07407	***
Observations			6733
Pseudo R2			0.0574

Table 10: Estimation of the propensity score. Probit regression (including beta carotene concentration as a covariate)

Sample	Treated	Controls	Difference	St.errors	T-stat
Unmatched	43.180416	33.352014	9.82840201	.562338412	17.48
ATT	43.180416	37.3359978	5.8444182	1.52418222	3.83
ATE			6.8939049		
ATU	33.3709189	40.6308134			

Table 11: ATT, ATE and ATU after the one-to-one matching (without including concentration of beta carotene as a control)

Sample	Difference	St.errors	T-stat
ATT	7.53766	.5400747	13.96
ATE	7.934504	.812564	9.76
ATU	8.07308889		

Table 12: ATT , ATE and ATU after the kernel matching (not including concentration of beta carotene as a control –parametrically-). Bootstrapped

Sample	Difference	St.errors	T-stat
ATT	7.365961	.6458979	11.40
ATE	7.725742	.584059	13.23
ATU	7.85119134		

Table 13: ATT , ATE and ATU after the kernel matching (including concentration of beta carotene as a control –parametrically-). Bootstrapped

Sample	Difference	St.errors
ATT	7.05878	.4708386
ATU	7.750677	.9349017

T**able 14: ATT and ATU after the kernel matching for each quintile of the propensity score (including concentration of beta carotene as a control –non parametrically-)**

variable	sample	treated	Control	%bias	%reduct bias	t	Pvalue
Black	Unmatched	.20857	.22742	-4.6		-1.64	0.100
	Matched	.20857	.20666	0.5	89.9	0.14	0889
Male_Black	Unmatched	.10767	.10676	0.3		0.11	0.915
	Matched	.10767	.10382	1.2	-323.6	0.37	0.710
Mexican-Hispanic	Unmatched	.31849	.34711	-6.1		-2.19	0.028
	Matched	.31849	.31743	0.2	96.3	0.07	0.946
Male_MexicanHispanic	Unmatched	.16234	.1669	-1.2		-0.44	0.657
	Matched	.16234	.15779	1.2	-0.1	0.37	0.712
Other	Unmatched	.05806	.10186	-16.2		-5.55	0.000
	Matched	.05806	. 05837	-0.1	99.3	-0.04	0. 969
Male_other	Unmatched	.02537	.04603	-11.2		-3.80	0.000
	Matched	.02537	.0248	0.3	97.3	0.11	0.915
Male	Unmatched	.49775	.4811	3.3		1.21	0.227
	Matched	.49775	. 48834	1.9	43.5	0.56	0.576
High education	Unmatched	.35738	.40842	-10.5		-3.79	0.000
	Matched	.35738	.36377	-1.3	87.5	-0.40	0.692
Male_high education	Unmatched	.16291	.18492	-5.8		-2.08	0.037
	Matched	.16291	.16342	-0.1	97.7	-0.04	0.967
Age	Unmatched	31.06	35.762	-19.9		-7.43	0.000
	Matched	31.06	31 425	-1.5	92.2	-0.44	0.661\
Age2	Unmatched	1582.1	1775.2	-9.5		-3.55	0.356
	Matched	1582.1	1600.1	-0.9	90.7	-0.25	0.804

Distribution of absolute bias		
statistics	Unmatched	matched
5th percentile	.2935	.1129687
25th percentile	3.330224	.2238093
median	6.075608	.6970244
75th percentile	11.15422	1.315385
95th percentile	19.92101	1.880633

Sample	Pseudo R2	LR chi2	p>chi2
Unmatched	0.057	446.95	0.000
Matched	0.000	1.42	1.000

Table 15: Assessing the quality of the matching procedure (kernel matching without including concentration of beta carotene as a control)

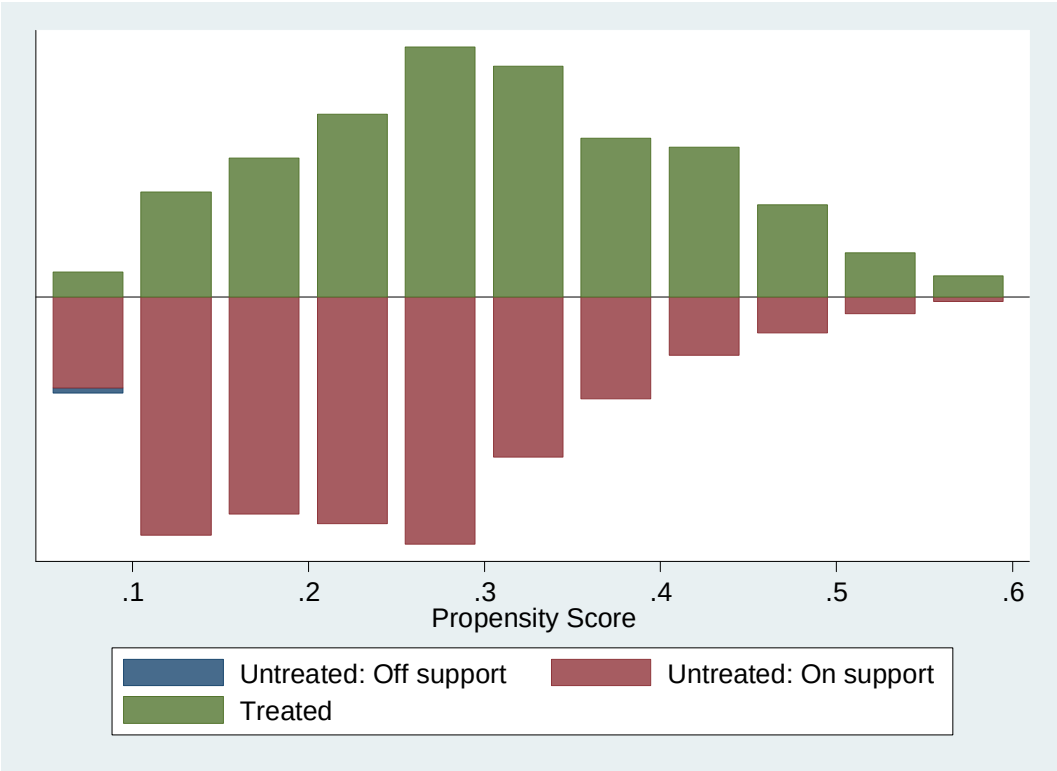


Figure 7: balance of the covariates

variable	sample	treated	Control	%bias	%reduct bias	t	Pvalue
Black	Unmatched	.20624	.22731	-5.1		-1.82	0.069
	Matched	.2066	.20582	0.2	96.3	0.06	0.955
Male_Black	Unmatched	.10514	.10696	-0.6		-0.21	0.833
	Matched	.10532	.10247	0.9	-57.2	0.27	0.783
Mexican-Hispanic	Unmatched	.32467	.35146	-5.7		-2.02	0.043
	Matched	.32523	.32424	0.2	96.3	0.06	0.950
Male_MexicanHispanic	Unmatched	.16464	.16913	-1.2		-0.43	0.667
	Matched	.16464	.16023	1.3	-4.7	0.37	0.708
Other	Unmatched	.05893	.10216	-15.9		-5.40	0.000
	Matched	.05903	.0594	-0.1	99.1	-0.05	0.963
Male_other	Unmatched	.02542	.04638	-11.3		-3.79	0.000
	Matched	.02546	.02404	0.8	93.2	0.27	0.788
Male	Unmatched	.49509	.48001	3.0		1.08	0.279
	Matched	.49479	.48549	1.9	38.4	0.55	0.585
High education	Unmatched	.35586	.40664	-10.5		-3.73	0.000
	Matched	.3559	.36041	-0.9	91.1	-0.28	0.782
Male_high education	Unmatched	.16118	.18293	-5.8		-2.04	0.041
	Matched	.16146	.16144	0.0	99.9	0.00	0.999
Age	Unmatched	30.927	35.741	-20.5		-7.53	0.000
	Matched	30.927	31.154	-1.0	95.2	-0.08	0.939
Age2	Unmatched	1568.8	1772.4	-10.1		-3.71	0.000
	Matched	1567.3	1573.6	-0.3	96.9	0.09	0.931
Betacarotene	Unmatched	14.515	14.003	3.5		1.21	0.226
	Matched	14.36	14.506	-1.0	71.4	-0.28	0.779

	Distribution of absolute bias	
statistics	Unmatched	matched
5th percentile	.5895849	.0039087
25th percentile	3.237787	.1991881
median	5.714166	.8461977
75th percentile	10.87495	.9875404
95th percentile	20.45502	1.859767

Sample	Pseudo R2	LR chi2	p>chi2
Unmatched	0.057	440.89	0.000
Matched	0.000	1.41	1.000

Table 16: Assessing the quality of the matching procedure (kernel matching including concentration of beta carotene as a control)

Value of the correlation	Variance of the treatment = $V(Y_1 - Y_0)$
-1	1367.225
-0.9	1322.23
-0.8	1277.234
-0.7	1232.239
-0.6	1187.244
-0.5	1142.249
-0.4	1097.254
-0.3	1052.259
-0.2	1007.263
-0.1	962.2682
0	917.273
0.1	872.2778
0.2	827.2826
0.3	782.2875
0.4	737.2923
0.5	692.2971
0.6	647.3019
0.7	602.3067
0.8	557.3116
0.9	512.3164
1	467.3212

Variance of Y_0 =369.8062 and variance of Y_1 =547.4668

Table 17: Variance of the treatment

Quantile	Treatment	Standard errors	Z	P-value	95% confidence interval	
Quantile1	2.497001	0.705631	3.54	0.000	1.113989	3.880013
Quantile2	2.723999	0.709852	3.84	0.000	1.332715	4.115283
Quantile3	3.178	0.643023	4.94	0.000	1.917698	4.438303
Quantile4	3.178	0.672025	4.73	0.000	1.860856	4.495145
Quantile5	2.951	0.700423	4.21	0.000	1.578197	4.323804
Quantile6	2.951	0.682467	4.32	0.000	1.61339	4.28861
Quantile7	3.632	0.618276	5.87	0.000	2.420202	4.843798
Quantile8	3.405002	0.62718	5.43	0.000	2.175752	4.634252
Quantile9	3.858999	0.600139	6.43	0.000	2.682749	5.03525
Quantile10	4.085999	0.591308	6.91	0.000	2.927056	5.244943
Quantile11	4.539999	0.570006	7.96	0.000	3.422807	5.657191
Quantile12	4.539999	0.584594	7.77	0.000	3.394216	5.685782
Quantile13	4.540001	0.592422	7.66	0.000	3.378875	5.701126
Quantile14	4.312998	0.590543	7.3	0.000	3.155555	5.47044
Quantile15	4.539999	0.577706	7.86	0.000	3.407715	5.672283
Quantile16	4.766998	0.577722	8.25	0.000	3.634685	5.899312
Quantile17	4.767	0.578627	8.24	0.000	3.632912	5.901089
Quantile18	4.767	0.584661	8.15	0.000	3.621085	5.912915
Quantile19	4.766998	0.581031	8.2	0.000	3.628198	5.905799
Quantile20	4.767	0.577337	8.26	0.000	3.635441	5.89856
Quantile21	4.994001	0.578122	8.64	0.000	3.860903	6.1271
Quantile22	4.766998	0.581661	8.2	0.000	3.626964	5.907032

Quantile23	4.993999	0.577977	8.64	0.000	3.861185	6.126814
Quantile24	4.767	0.588633	8.1	0.000	3.6133	5.9207
Quantile25	4.993999	0.590337	8.46	0.000	3.836961	6.151038
Quantile26	5.447998	0.595021	9.16	0.000	4.281778	6.614218
Quantile27	5.675001	0.600293	9.45	0.000	4.498448	6.851554
Quantile28	5.448	0.602115	9.05	0.000	4.267877	6.628123
Quantile29	5.674999	0.601521	9.43	0.000	4.49604	6.853959
Quantile30	5.675001	0.609405	9.31	0.000	4.480589	6.869413
Quantile31	5.674999	0.613583	9.25	0.000	4.472399	6.8776
Quantile32	5.902002	0.621361	9.5	0.000	4.684157	7.119848
Quantile33	5.902	0.620713	9.51	0.000	4.685426	7.118575
Quantile34	5.901999	0.627044	9.41	0.000	4.673014	7.130983
Quantile35	6.129002	0.632357	9.69	0.000	4.889605	7.368398
Quantile36	6.356001	0.638977	9.95	0.000	5.10363	7.608372
Quantile37	6.129	0.642187	9.54	0.000	4.870336	7.387664
Quantile38	6.355999	0.644017	9.87	0.000	5.093748	7.61825
Quantile39	6.355999	0.643639	9.88	0.000	5.09449	7.617508
Quantile40	6.356001	0.643053	9.88	0.000	5.095641	7.616361
Quantile41	6.356001	0.644945	9.86	0.000	5.091932	7.62007
Quantile42	6.355997	0.643976	9.87	0.000	5.093828	7.618166
Quantile43	6.582998	0.644801	10.21	0.000	5.319212	7.846784
Quantile44	6.810001	0.645863	10.54	0.000	5.544134	8.075869
Quantile45	6.810001	0.645514	10.55	0.000	5.544818	8.075185
Quantile46	6.810001	0.645673	10.55	0.000	5.544506	8.075496
Quantile47	7.037001	0.644997	10.91	0.000	5.77283	8.301171
Quantile48	7.037001	0.648055	10.86	0.000	5.766837	8.307164
Quantile49	7.037001	0.648055	10.86	0.000	5.766837	8.307164
Quantile50	6.583002	0.646656	10.18	0.000	5.31558	7.850424
Quantile51	6.810001	0.648753	10.5	0.000	5.53847	8.081533
Quantile52	6.583	0.648823	10.15	0.000	5.31133	7.85467
Quantile53	6.583	0.649845	10.13	0.000	5.309328	7.856672
Quantile54	6.355997	0.649306	9.79	0.000	5.08338	7.628614
Quantile55	6.355999	0.649172	9.79	0.000	5.083644	7.628354
Quantile56	6.356003	0.650546	9.77	0.000	5.080956	7.63105
Quantile57	6.582996	0.648107	10.16	0.000	5.31273	7.853262
Quantile58	6.583	0.650534	10.12	0.000	5.307977	7.858023
Quantile59	6.583	0.652068	10.1	0.000	5.30497	7.861031
Quantile60	6.128998	0.652558	9.39	0.000	4.850007	7.407989
Quantile61	6.129002	0.657959	9.32	0.000	4.839425	7.418578
Quantile62	6.356003	0.664889	9.56	0.000	5.052844	7.659161
Quantile63	6.128998	0.677773	9.04	0.000	4.800588	7.457407
Quantile64	6.129002	0.694612	8.82	0.000	4.767588	7.490415
Quantile65	5.902	0.705758	8.36	0.000	4.518741	7.28526
Quantile66	5.902	0.726557	8.12	0.000	4.477975	7.326026
Quantile67	5.902	0.75104	7.86	0.000	4.429989	7.374012
Quantile68	5.901997	0.773482	7.63	0.000	4.386001	7.417993

Quantile69	5.902	0.795923	7.42	0.000	4.34202	7.461981
Quantile70	5.902	0.822221	7.18	0.000	4.290477	7.513524
Quantile71	5.902	0.853538	6.91	0.000	4.229097	7.574904
Quantile72	5.675003	0.886608	6.4	0.000	3.937283	7.412724
Quantile73	5.902	0.947493	6.23	0.000	4.044948	7.759053
Quantile74	6.582996	1.092321	6.03	0.000	4.442086	8.723907
Quantile75	7.264004	1.243002	5.84	0.000	4.827765	9.700242
Quantile76	7.491001	1.313621	5.7	0.000	4.916351	10.06565
Quantile77	7.945	1.395362	5.69	0.000	5.210141	10.67986
Quantile78	8.399002	1.437774	5.84	0.000	5.581018	11.21699
Quantile79	9.306995	1.422053	6.54	0.000	6.519822	12.09417
Quantile80	10.442	1.428172	7.31	0.000	7.642836	13.24117
Quantile81	10.442	1.438655	7.26	0.000	7.622285	13.26171
Quantile82	10.669	1.443927	7.39	0.000	7.838955	13.49904
Quantile83	10.896	1.460212	7.46	0.000	8.034038	13.75796
Quantile84	9.987999	1.508967	6.62	0.000	7.030478	12.94552
Quantile85	9.988003	1.578506	6.33	0.000	6.894189	13.08182
Quantile86	10.215	1.625458	6.28	0.000	7.029162	13.40084
Quantile87	9.987999	1.690778	5.91	0.000	6.674135	13.30186
Quantile88	9.760998	1.754369	5.56	0.000	6.322497	13.1995
Quantile89	9.760998	1.783106	5.47	0.000	6.266173	13.25582
Quantile90	9.080002	1.890579	4.8	0.000	5.374535	12.78547
Quantile91	8.852997	2.099246	4.22	0.000	4.738549	12.96744
Quantile92	8.625999	2.224078	3.88	0.000	4.266886	12.98511
Quantile93	8.852997	2.692174	3.29	0.001	3.576433	14.12956
Quantile94	9.761002	3.0876	3.16	0.002	3.709416	15.81259
Quantile95	9.987999	3.199317	3.12	0.002	3.717453	16.25854
Quantile96	10.66901	3.280624	3.25	0.001	4.239101	17.09891
Quantile97	9.987991	4.442543	2.25	0.025	1.280768	18.69521
Quantile98	11.35	6.489658	1.75	0.080	-1.3695	24.06949
Quantile99	12.712	8.673194	1.47	0.143	-4.28715	29.71115

Table 18 Estimated quantile treatment effects assuming rank invariance and controlling for concentration of betacarotene

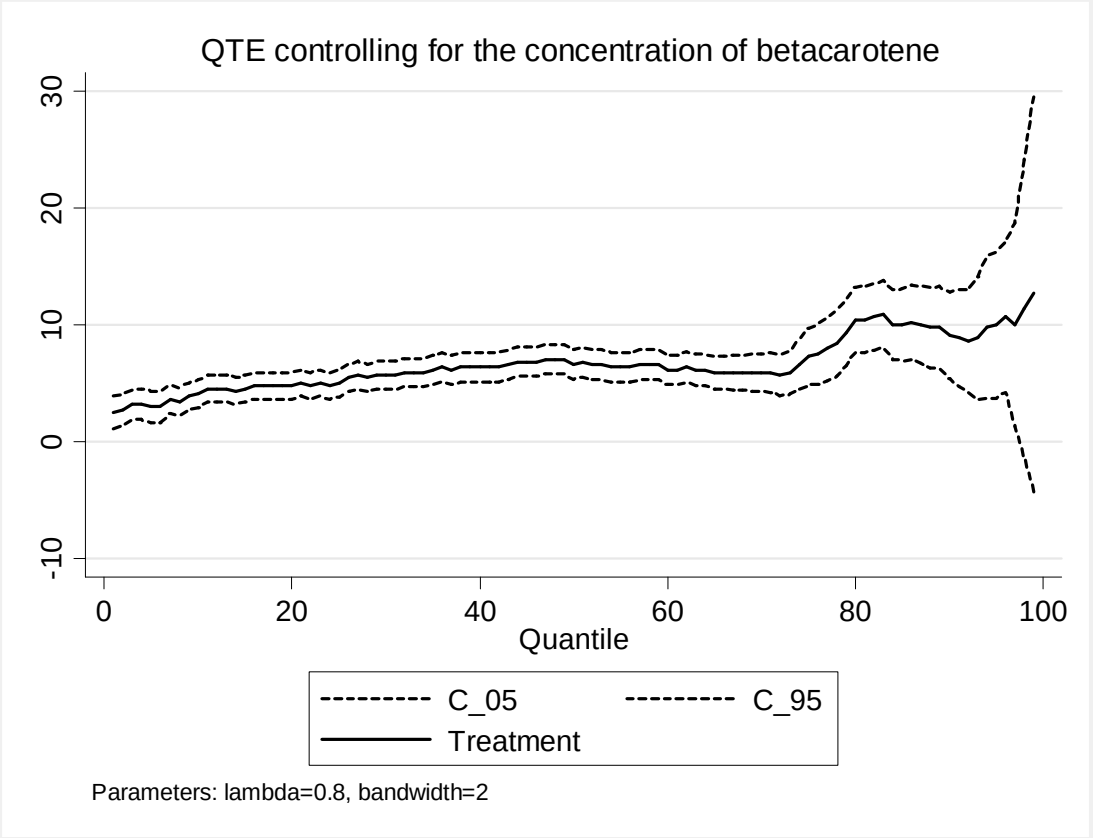


Figure 8: Quantile treatment effects (with confidence intervals) estimated using the quantile treatment effect estimator proposed by Firpo (2007)

Quantile	Coef.	Std. Err. z	P>z	[95% Conf.	Interval]
0.25	5.447998	.5711376	9.54	0.000	4.328589	6.567407
0.5	7.037001	.6035824	11.66	0.000	5.854001	8.22
0.75	7.945004	1.156583	6.87	0.000	5.678143	10.21186

Table 19: Quantile treatment effects estimated using Firpos's estimator without controlling for dietary habits

Quantile	Coef.	Std. Err. z	P>z	[95% Conf.	Interval]
0.25	4.993999	.5903368	8.46	0.000	3.836961	6.151038
0.5	6.583002	.6466559	10.18	0.000	5.31558	7.850424
0.75	7.264004	1.243002	5.84	0.000	4.827765	9.700242

Table 20: Quantile treatment effects estimated using Firpos's estimator controlling for dietary habits (parametrically)

	without controlling for dietary habits		without controlling for dietary habits	
Statistics	perfect positive dependence	perfect negative dependence	perfect positive dependence	perfect negative dependence
5th percentile	4.1768	-51.0296	4.0406	-50.8934
25th percentile	5.947399	-12.8482	5.6296	-34.7537
Median	7.5364	7.5364	7.4683	7.5818
75th percentile	8.535199	27.7848	8.489799	26.8314
95th percentile	12.712	66.6018	13.5292	69.0534

Table 21: Percentiles of parameters of the impact distributions (perfect positive and perfect negative dependency)

.

Statistics	distribution of the 25th percentile	distribution of the median	distribution of the 75th percentile
minimum	-62.9698	-32.7334	-20.4754
25^{th}	-36.6832	-1.770601	9.4205
50^{th}	-4.8351	4.0406	15.1636
75^{th}	-0.4085999	8.4898	19.6128
maximum	12.3942	24.5614	30.0094

Table 22: Percentiles of parameters of the impact distributions with a random sample of 1000 quantile permutations

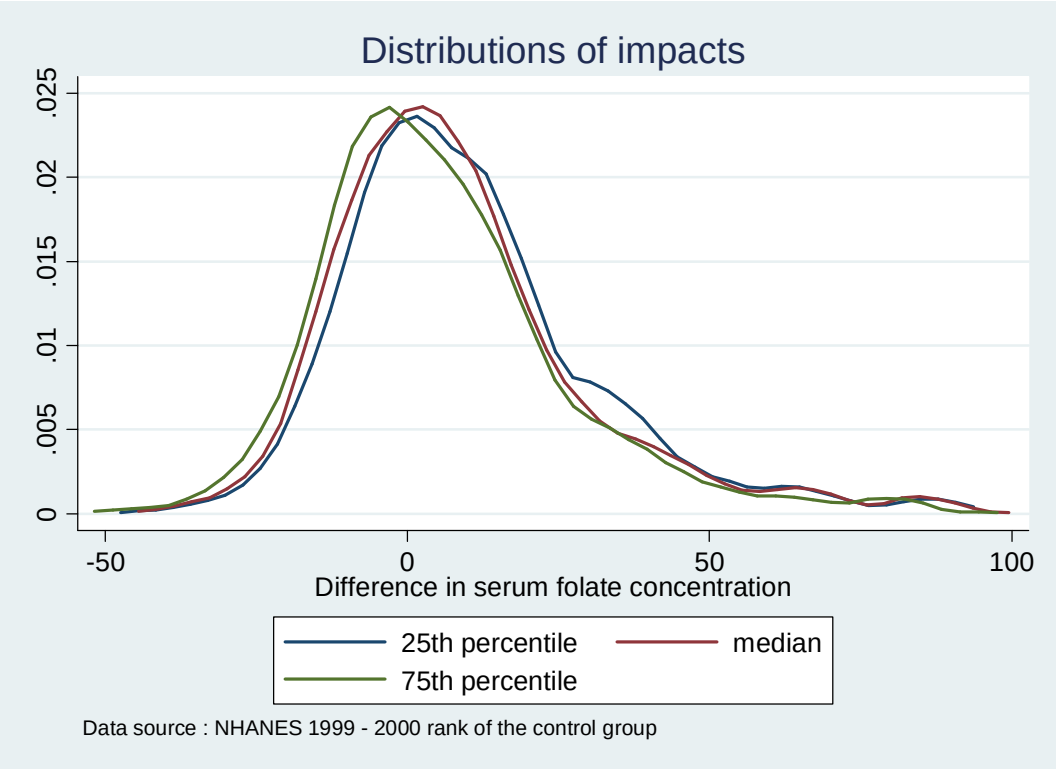


Figure 9: Distribution of the impacts assuming no correlation between the potential outcomes