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The Role of High-Fructose Corn Syrup Imports in Child and Women Obesity

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Abstract: High Fructose Corn Syrup is believed to be responsible for the increase of the obesity epidemic that is affecting both developed and developing countries, yet to date there are no rigorous analyses that quantify this effect. To estimate the impact of consumption of HFCS on obesity, I have paired country-specific data on imports of HFCS from the United Nations International Trade Statistics Database (UN Comtrade) with women and child health data from around 70 countries from 1985 to 2018. The results show that conditional on space and time fixed effects, imports of HFCS with a high concentration of fructose have large and significant positive effects on children's weight-related outcomes. These effects are mainly concentrated in children older than 12 months who live in an urban area. Among women, my key finding is that a one standard deviation increase in exposure to HFCS imports containing at least 50% of fructose is associated with a 1.1 percentage point increase in the change in probability of being obese.

Key Words: *Obesity; Overweight; High Fructose Corn Syrup (HFCS); Trade flows; Imports*

1. Introduction

Since 1975 the prevalence of obesity has nearly tripled and this trend is evident in both adults and children. Most notably, the number of overweight children (aged between 0 and 5 years) skyrocketed from 32 million globally in 1990 to over 41 million in 2016 (WHO, 2019) while 40% of women were overweight in 2016 compared with 6% in 1975 (WHO, 2020). Obesity is linked to coronary heart disease, diabetes, glucose intolerance, asthma, sleep apnea, and psychological effects like low self-esteem and depression. Additionally, children who are obese are more likely to have obesity as adults (Must and Strauss, 1999). In addition, the economic impact from obesity is 2 trillion each year, which is the equivalent to 2.8% of the global GDP. This includes the direct costs related to health service delivery and the indirect costs associated with the reduction of productive years of employees (McKinsey Global Institute, 2014).

Although obesity has traditionally primary affected high-income countries, the prevalence of obesity in low- and middle-income countries has reached alarming levels. Paradoxically, in these countries obesity and undernutrition can coexist at the individual level,[1] at the household level,[2] and at the population level (World Bank, 2017).[3] This phenomenon has been called the "double burden of malnutrition" and imposes a huge burden on government finances. Health systems in developing countries are equipped to treat mainly acute and infectious diseases and they might not be prepared to assist large number of people suffering from chronic diseases (Hoffman, 2001).

It is widely accepted that the immediate cause of obesity is the caloric imbalance between calories consumed and expended, which has been mainly driven by a decrease in physical activity as labor has become more sedentary and changes in dietary patterns, which shifted rapidly towards caloric sweeteners (WHO, 2020). Although sugar has always been the world's most common sweetener, there is a wide range of non-sugar products used today, such as High Fructose Corn Syrup (HFCS).

[1]A child who could be considered stunted in terms of height for age and also obese in terms of weight for height.
[2]When the mother is overweight, and the child is underweight.
[3]When there is both undernutrition and obesity in the same community, region or country.

HFCS is used worldwide as a sweeter, texturizer, and preservative. It is produced from corn starch, which is further refined to produce syrup. Due to corn subsidies in the U.S. it became a cheaper source of sweetener than sucrose which is derived from sugar cane or beet. Its composition is fructose and glucose, but it is classified into different groups depending on the amount of fructose present. HFCS-42 and HFCS-55 are the most commonly used and they contain 42% and 55% of fructose respectively. HFCS-42 is primarily used for baked foods and cereals, whereas HFCS-55 is mostly used for soft drinks. Although U.S. has always been the major producer and user of HFCS in the world, it has gained popularity worldwide in the last few decades.

In 2004, HFCS became one of the most controversial food ingredients when Bray, Nielsen and Popkin (2004) pointed it out as principal contributor to the obesity epidemic in the U.S. While consumption of HFCS increased, obesity rates also rose dramatically. Since then, research has been extensive, but the role of HFCS and, by extension, added sugars on the obesity epidemic is still inconclusive, specifically regarding its long-term effects.

This paper examines whether there is a causal link between consumption of HFCS and the prevalence of obesity. It does so by examining the relationship between children and women obesity and exposure to HFCS imports and quantify the magnitude of this effect. The remainder of this paper first describes relevant existing economic and non-economic research on variables that influence obesity. Secondly, it contains the data sources and methodology I will be using to establish an association between HFCS imports and child and women obesity. Then, I present the data analysis and discussion of my results, and lastly, I offer a conclusion and some policy recommendations.

2. Literature Review

Obesity is a complex disease that is determined by a combination of biological, genetic, social, environmental and behavioral factors. The two main triggers of the epidemic mentioned above (changes in physical activity and dietary patterns) are usually the result of societal and environmental changes associated, in turn, with economic development. In this section, I first review the literature that has explored societal, economic, environmental, and behavioral factors

contributing to obesity. Then, I focus on the evidence revealing the effects of sugar consumption. Finally, I review the theories behind parental investments and family structure.

2.1. Societal and economic factors

Technological progress has contributed in two ways in the rise of obesity, by reducing the cost of food and making physical activity easy to evade. On one hand, as agriculture production transitioned from manual labor to automation, the price of food has consistently declined. On the other hand, as labor has become more sedentary and entails little exercise, people must pay to undertake physical activity (Philipson and Posner, 2003; Lakdawalla and Philipson, 2002).

At the same time, overall obesity prevalence appears to be correlated with wealth. This correlation is due to the dietary shifts propelled by increasing income per capita. At the individual level, Philipson and Posner (2003) consider "closeness" to one's ideal weight a normal good, which implies a non-monotonic relationship between income and weight. For low-income underweight people, an increase in income results in more food consumption to increase weight. However, for wealthy individuals, increasing income increases the demand for thinness since they care more about their appearance. Empirically, this theory helps to explain why in early industrialized countries, obesity primarily affects the rich and newly emerged middle classes, and in wealthy, more modern societies obesity disproportionally affects people with the lowest levels of income. This phenomenon has been called the "poverty-obesity paradox" (Salmasi and Celidon, 2017).

Urbanization is another factor highly associated with several dietary changes and therefore with obesity. In high-income countries obesity is more pervasive in rural areas as a result of farm automation since there are fewer opportunities for leisure-time activities, and junk food is readily available in these countries (Popkin, Adair and Ng, 2012). Historically, in low- and middle-income countries, obesity has been highly correlated with urbanization (Popkin, 1999). However, recently Bixby, Bentham, Zhou et al. (2019) have found that obesity is increasing at faster rate in rural areas, which in turn is reversing this trend and closing the gap between urban and rural areas. The only region in which the difference became larger is sub-Saharan Africa, where agriculture is still mainly manual and for subsistence purposes and the use of cars is limited due

to poor infrastructure, while urban areas have experienced a significant economic growth (Bixby, Bentham, Zhou et al., 2019).

2.2. Environmental factors and new technology in food production

The economic literature has mainly focused their efforts on the environmental influences on the prevalence of obesity in developed countries. It is often argued that fast food is responsible for the rise in obesity. The explanation behind this theory is that "proximity to a fast food restaurant could lower the monetary and non-monetary costs of accessing unhealthy food" (Currie et al., 2010). Currie et al. (2010) find that children whose schools are located within a tenth of a mile of a school of a fast food restaurant and pregnant women whose residence is within half of a mile have significantly higher obesity rates. Also, Chou, Rashad and Grossman (2008) find a strong positive effect of exposure to fast-food restaurant advertising on television on the body mass index for children. In the same direction, some researchers have presented an association between maternal employment and childhood obesity arguing that working mothers "may rely more heavily on higher calorie prepared or fast foods" (Anderson, Butcher and Levine, 2003; Fitzsimons and Pongiglioni, 2019; Cawley and Liu, 2012), while others are less conclusive (Gwozdz et al., 2013; Greve, 2011).

A large body of literature has also focused on the link between neighborhood environment and nutritional equality. In this regard, research has identified the scarce availability and high prices of healthy foods as a cause of unhealthy eating in low-income neighborhoods (Kling, Liebman and Katz, 2007; Bitler and Haider, 2011). However, the difference of supply observed across neighborhoods can only partially explain the eating habits (Allcott et al., 2019).

A growing economics literature has also documented the role of trade liberalization on obesity. On this subject, Giuntella, Rieger and Rotunno (2020) note that "greater openness to trade in foods can affect the nutrition transition and hence obesity prevalence through changes in prices, income, norms, and tastes". Empirically, they establish that "exposure to food imports from the U.S. explains about ten percent of the rise in obesity prevalence among Mexican women between 1988 and 2012".

Finally, in connection with technological advancement, Cutler, Glaeser and Shapiro (2003) propose a theory based on the division of labor in food preparation to explain the growth in obesity in the United States. In light of this theory, they argue that food innovations have enabled manufactures to cook food and ship to the consumers for rapid consumption. As a result, "the switch from individual to mass preparation lowered the time price of food consumption and led to increased quantity and variety of foods consumed". This theory is consistent with the fact that consumption of mass-produced food has increased the most in the past two decades.

2.3. Behavioral factors

In the last few years, uncontrolled eating and obesity has been associated to addiction. Imperfectly rational addiction models assume that individuals have "inconsistent short and long-run preferences", which it is usually the result of discounting the future very heavily. In other words, in the decision to consume the gains or costs of the future are given little weight (Chaloupka and Warner, 2000; Propper, 2005). Consistent with this theory, Komlos, Smith and Bogin (2004) found a positive relationship between obesity and rates of time preference. Courtemanche, Heutel and McAlvanah (2015) took a step further including in the model food prices. They found that "impatience both increases BMI and strengthens one's response to food prices", which is consistent with the fact that in the US the largest weight gains are concentrated amongst the individuals located in the right tail of the BMI distribution.

Another approach to modelling addiction in the economic literature is the myopic addiction models. In these models the individual "recognizes the dependence of current addictive consumption decisions on past consumption, but then ignores the impact of current and past choices on future consumption decisions when making current choices" (Chaloupka and Warner, 2000). The third approach is the rational addiction models which recognize that "individuals incorporate the interdependence between past, current, and future consumption into their utility maximization process" (Chaloupka and Warner, 2000). Therefore, in terms of the rational addiction model of Becker and Murphy (1988) higher current consumption raises future consumption in order to keep the same level of utility. Some researchers have applied the rational addiction model to food consumption and have provided evidence of addiction to caloric

intake, specifically with respect to certain foods, such as carbohydrates (Richards, Patterson and Tegene, 2007) and soft drinks (Liu and Lopez, 2012).

2.4. Sugar consumption

It is well established that sugar can be a substance of abuse and lead to addiction (DiNicolantonio et al., 2018; Avena et al., 2009). Sugar interferes with the normal operation of both the hormone ghrelin, which signals hunger to the brain, and the hormone leptin, which signals the satiety (Lustig, Schmidt and Brindis, 2012).

On the subject of HFCS, most epidemiological studies and clinical trials have focused on identifying metabolic differences between HFCS and sucrose. While there is some scientific consensus on the equivalence between HFCS and sucrose at normal levels of human consumption (Rippe and Etherton, 2012; Klurfeld et al., 2013), the link between HFCS and obesity and other chronic diseases is still inconclusive, particularly with respect to the long-term impacts (Rippe and Angelopoulos, 2013). On the other hand, recent studies have reported an increase in body weight and fat mass when the dosage of added sugars from either sucrose or HFCS was incremented (Lowndes et al., 2014; Stanhope et al., 2015). This suggests that the dose, rather than the type of sugar, might be playing an important role.

Finally, it is also important to consider the literature that describes how healthy children become healthy adults. In this regard, there is growing evidence that "nutritional deficiencies or an excessive rate of weight gain during gestation, as well as not being exposed to breastfeeding for an adequate period, contribute to a predisposition to overweight and an increased risk of noncommunicable diseases later in life" (Htenas, Tanimichi-Hoberg and Brown, 2017).

2.5. Parental Investments and family structure

Here I explore the literature relative to the decision-making process behind having children. One of the economic theories of fertility assumes that the household demand for children is determined by family preferences for a certain number of surviving children (Schultz, 1973). In this context, fertility is a function of the "preferences of parents for children which are constrained by the parents' resources and the associated alternative economic opportunities in

using their resources" (Schultz, 1973). All fertility theories assume that raising children takes time and wealth, so children are seen as a consumption good (Jones, Schoonbroodt and Tertilt, 2008). This takes us to the inverse relationship between the number of children conceived and the quality of children (Becker & Lewis, 1973; Hanushek, 1992; Schultz, 1973).

According to this theoretical model of quantity-quality trade-off, smaller family sizes may benefit from the fact that the economic resources are allocated to less people and, as a result, it is possible to achieve better human capital outcomes. Although some studies that have tested the quantity-quality theory have observed higher children education outcomes in smaller families (Li, Zhang and Zhu, 2008), the same is not observed when it comes to health outcomes. Zhang, Xu and Liu (2016) affirm that in China, being raised in a one-child family increases the body mass index and probability of being overweight or obese due to the fact that those children eat more high-sugar, high-fat, and high-protein food.

In conclusion, overweight and obesity are complex challenges due to the fact that there are many factors directly and indirectly affecting the energy imbalance and therefore contributing to the causes of this epidemic. The body of literature in this field has made significant progress in establishing considerable evidence of correlation between socioeconomic factors and obesity and in identifying environmental and behavioral influences. However, there is further scope for improvement in establishing causal relationship and recognizing the role that new food technologies, such as HFCS, are playing in the epidemic in the long-term and, with regard to children, the effects of an unhealthy diet in early life. This paper also provides additional evidence on the causes of obesity in developing countries which is a territory that remains quite unexplored.

3. Sample and Data Sources

3.1. Data on Obesity

My data on obesity is taken from the Demographic and Health Survey (DHS) Program. The DHS surveys are conducted at the household level and provide information from around 90 developing countries on health, welfare and nutrition of women of reproductive age and their children. In reference to women, the DHS includes information on date of birth, age, education,

weight, height and BMI (Body Mass Index). Regarding children, it includes data on date of birth, age, age at death if the child died, height, weight, weight-for-age, height-for age, BMI-for age and weight-for-weight normalized into a z-score to indicate the number of standard deviations about or below an average. This standardization allows for clear comparisons across countries and children of different ages and genders across time. The unit of observation is a child/woman measured at time of survey in a given location, allowing me to construct location and year-specific infant/woman-level obesity and overweight dataset.

According to WHO definitions, a woman is defined as obese when her BMI is higher than 30 and overweight when it is between 25 and 29.99. Likewise, following WHO child growth standards, I designated any child under 5 years of age as overweight if his/her weight-for-height is greater than 2 standard deviations above WHO Child Growth Standards median, and as obese if his/her weight-for-height greater than 3 standard deviations above the WHO Child Growth Standards median (de Onis et al., 2007).

3.2. Imports of HFCS Data

Then the infant/maternal obesity data is merged with cross-sectional data on imports of HFCS retrieved from the United Nations International Trade Statistics Database (UN Comtrade). UN Comtrade is the largest depository of international trade flows data which is publicly available on the internet. This large depository of international trade data is extremely useful because it contains information from over 200 countries about their annual international trade flows detailed by commodities/services categories and partners countries since 1962 until the most recent completed year, which in this case is 2018. All commodity trade values are converted into US dollars and quantities are also converted into metric measures (kilograms or liters). Commodities are reported according to the current Standard International Trade Classification (SITC) codes, the Harmonized System (HS), and Broad Economic Categories (BEC) and are converted all the way down to the earliest classification. For this analysis, I used the HS classification from 1992 in which HFCS is classified with the following codes:

- **170240** – Glucose including syrup of 20%-50% dry weight fructose. **Description:** Glucose and glucose syrup, containing in the dry state at least 20 % but less than 50 % by weight of fructose.
- **170250** – Fructose, chemically pure. **Description**: Chemically pure fructose.
- **170260** – Fructose, syrup > 50% fructose, not pure fructose. **Description**: Other fructose and fructose syrup, containing in the dry state more than 50% by weight of fructose.

As shown in Figure 1, the worldwide production of HFCS is highly concentrated among a few countries. For the purpose of this study, none of the countries depicted in the figure is included in either of the two final datasets.

To measure the exposure to HFCS, I created a variable of kilograms of HFCS per capita. To do this, I used data on census and population growth taken from the World Bank Development Indicators. Summary statistics for the trade data indicate that 56 countries imported HFCS in both samples, while 14 and 21 did not in the children's sample and women's sample respectively. On average, the imports of HFCS by country and year were 43 grams per capita in the children dataset with a variation of 133 grams (see Table 1a), and 49 grams per capita with a variation of 140 grams (see Table 8a) in the women dataset.

The final children dataset includes health data with corresponding imports of HFCS for approximately 1,500,000 children ranging from 0 to 5 years old from 70 countries from 1986 to 2018. Summary statistics for the child health data indicate that, on average, our sample is below average for health. The four z-score indicators are below 0 (Table 1b). Also, there are no significant differences in the health indicators between the group of countries with imports of HFCS and the group without them. Among the children of the sample, the prevalence of obesity is around 1% and the prevalence of overweight is around 4%.

The final dataset for women also includes around 1,500,000 observations of women aged between 15 to 49 from 77 countries from 1985 to 2014. Summary statistics for the women health data shows a prevalence of obesity of around 6% and a prevalence of overweight of around 14%. Additionally, there are significant differences in the percentage of women in the normal and underweight range between the two groups of countries. In both cases there is a difference of

around 10%, but there is no significant difference corresponding to the obesity's and overweight's rates.

4. Methodology

In order to test whether consumption of HFCS increases obesity in children under five and in women, I use imports of kilograms of HFCS per capita in a given year in a given country to act as my treatment. However, this treatment variable is not a randomly exogenous occurrence. Therefore, the results of this analysis cannot perfectly be interpreted as causal. Likely, as countries industrialize, they also open their markets to international trade and, as a result, they experiment economic growth which, in turn, translates into a higher income which as it has been pointed earlier is highly correlated with obesity. Also, a change in taste preferences might have affected both obesity and HFCS imports at the same time. For this reason, several control variables are entered in the regression. Additionally, with respect to taste preferences, I find improbable that they have shifted radically and systematically every 5 years coinciding with the time in which the surveys were taken.

To reduce bias from economic conditions that might be affecting both imports of HFCS and obesity rates, I use a linear probability model with fixed effects that controls for spatial and spatial-temporal variations. The administrative region fixed effect absorbs the differences in exposure to HFCS between different region and permits to compare children/women from the same location. I then include an interaction of UNICEF region and year fixed effect which accounts for the fact that there are different trends of obesity in different parts of the world. By using this two-way fixed effect model, I can observe what happens to children and women's obesity conditional on exposure to imports of HFCS in a specific administrative region in a specific year.

My regression specification is the following:

$$Y_{iat} = \beta_0 + \beta_1 H_{ct} + \beta_2 X_{it} + \beta_2 Z_{ct} + \tau_t \varphi_r + \mu_a + \mathcal{E}_{iarct} \text{ , (1)}$$

The dependent variable, Y, corresponds to the probability of a binary variable being either zero (probability child/woman is not obese) or one (the probability the child/woman is obese), which

is determined by the explanatory variables. The parameter of interest β_1 indicates the change in the probability that the child/woman is obese due to a one-kilogram per capita increase of the imports of HFCS. H is the average of imports of HFCS per capita in the last three years. This treatment measure allows to smooth the spikes in the imports of HFCS over time. X is a vector of controls for mother's age and education (in the case of children) or age and education (in the case of women), and urban or rural area. Z is a vector of macroeconomic controls that includes the log of total imports and total sugars in country c in year t. τ_t is the year trend interacted with UNICEF region (φ_r), and μ_a is the administrative region fixed effect. ε_{at} represents the clustered standard errors at the administrative region level and it accounts for any serial correlation of the errors within the administrative region. The sub-indexes in the model are: i for child/woman, a for administrative region, c for country and t for year. Finally, I weigh the data using the DHS survey weights at the country level.

In short, the model looks at the likelihood a child/woman becomes obese due to exposure of HFCS, which is measured by imports of HFCS in kilograms per capita, controlling for spatial and time fixed effects and macroeconomic conditions. The impact of exposure of HFCS will vary depending on the age of the children and whether the household of the child/woman is located in an urban area. To test whether the treatment effects are different I estimate versions of equation (1) with interactions of $\beta_1 H_{ct}$ with indicators separately for urban and non-infant (child older than 12 months) and both together in the case of children, and for urban in the case of women.

Finally, both models are run with two different treatment measures. In order to be able to see whether the concentration of fructose has a different impact on obesity, I have created one treatment measure that includes all HFCS types that contain a 20% of fructose or higher, and a more restricted treatment measure that only include HFCS types with a proportion of fructose of 50% or higher.

5. Results

5.1. Children's outcomes

Tables 2 and 3 report estimates of the average treatment effects for the HFCS exposure effect from the linear probability model with fixed effects. First, consumption of HFCS shows no statistically significant average impact on obesity and overweight for the population as a whole (Columns 1 and 2). However, the results show that imports of HFCS are associated with better weight-related nutritional outcomes for children under five years old (Table 3, columns 3, 4 and 6).

When the treatment variable is restricted to imports of HFCS that contain a proportion of 50% of fructose or higher, an increase of 1kg per capita of HFCS increases weight-for-height z-scores by 1.57 standard deviations, weight-for-age z-scores by 1.09 standard deviations; and, BMI-for-age z-scores increase by 1.63 standard deviations (Table 3). However, when I relax the treatment variable and include all the imports of HFCS with any percentage of fructose, there is still some effect on the weight-for-age z-score, but most of the effects disappear. Specifically, in the latter case, when the average of imports of the last three years increases by 1 kg per capita, weight-for-age z-scores increase by 0.246 standard deviations.

To give an idea of the magnitudes of the effects observed consider the effects of one standard deviation difference in weight for various ages[4]. For a girl aged 1 this would be equal to approximately 1.5 kg, while for girls aged 2, 3, 4 and 5 years it would be 2.25 kg, 2.75 kg, 3.75 kg and 4.5kg, respectively. The same amounts for boys are 1.25 kg, 2 kg, 2.5 kg, 3 kg and 3.5 kg, respectively. Hence, the estimated effect would correspond to somewhere between 2.25 kg and 4.5 kg. Generally, the interpretation of weight for age is more complicated given that is influenced by both height and weight[5]. However, my model shows no impact on height-related nutritional outcomes. In this regard, the coefficient on height-for-age is negative and not significant. This result can be explained by the significant presence of wasting in the sample and by the fact that consumption of HFCS have no impact on height. Both explanations are reasonable considering that height for age is a long-term indicator of nutritional deficiencies and that HFCS does not contain any essential nutrient beyond the calories.

[4] See Appendix for charts of the WHO growth standards for girls and boys (Figure 3 and 4).
[5] The discussion regarding the health outcomes is based on de Onis and Blössner (1997).

Additionally, Graphs 1, 2 and 3 show the non-linear relationships between the weight related nutritional outcomes and the exposure of imports of HFCS with high concentration of fructose (50% and above). It is worth mentioning that they clearly depict that the results are not driven by outliers.

As a robustness check, I re-estimate the effect of exposure to HFCS imports on nutritional outcomes for the population as a whole dropping one country at a time to assess whether the results are driven by a particular country. The coefficients are positive and significant across every country in the case of weight-for-age z-scores, verifying that the effect of this indicator is not driven by any particular country. However, in the case of weight-for-height and BMI-for-age z-scores, the coefficients lose significance when Turkey is dropped from the sample, which indicates that the effects on these outcomes might be driven by this country. Also, in all cases, the effect sizes remain similar and statistically significant when I weight the regressions at the child level.

Further, I look at the impact of exposure to HFCS imports in children who are older than 12 months and live in an urban area (urban non-infant). Children older than 12 months are not exclusively breastfeeding anymore and other foods are used as complements to meet their nutritional needs. A substantial amount of literature also identifies urbanization as a major driving force in global obesity. People living in urban areas have greater opportunities for eating processed foods and added sugars thanks to the enormous penetration of super- and megamarket companies and fast food restaurants (Popkin, Adair and Ng, 2012). For these reasons, it is plausible to expect a stronger association between HFCS important and health outcomes in these children.

Tables 4 and 6 show the estimates for the urban, non-infant, and urban and non-infant effects. The results show a large and significant effect on overweight from exposure to imports of HFCS in the urban areas and non-infants. Specifically, when I limit the explanatory variable to HFCS with 50% of fructose or more, an increase of 1 standard deviation of the average of imports of the last three years (0.02, see Table 1a for summary statistics on my main variables) is associated with an increase in the probability of being overweight by almost 0.004 percentage points among

non-infants living in urban areas, while the net effect is 0.002 percentage points (Table 6 – Panel C, column 2).

This result is mainly driven by the urban effect given that the estimate is practically unchanged when I exclusively interact the treatment variable with the urban indicator (Table 6 – Panel A, column 2) and the coefficients are not significant when the treatment variable is solely interacted with the non-infant indicator. In this case, when I expand the interpretation of the treatment variable including all imports of HFCS containing 20% of fructose or above, the results are still positive and significant, but the coefficients are smaller (Table 4). As in the results for the whole population, this model also observes a positive net effect of HFCS imports with weight-for-height, weight-for-age and BMI-for-age indicators.

Again, as a robustness check, I re-estimate the effect of exposure to HFCS imports on overweight for the children who live in urban areas dropping one country at a time. The coefficient is positive and significant across every country except when Swaziland is dropped from the sample, suggesting that the result might be driven by this country. On the other hand, in this case, the coefficient is not significant when the regressions are weighted at the child level (Tables 5 and 7).

It is worth mentioning in some occasions (column 1, Table 3 and 6), the sign of the obese coefficient is negative, which I attribute to noise in the model given there are a large number of factors contributing to child obesity.

5.2. Women's outcomes

I document a positive relationship between long-run changes in exposure to HFCS and changes in women's obesity. The corresponding OLS estimates for the women population as a whole are displayed fully in Tables 9 and 10. When the independent variable is defined as imports of HFCS containing at least 50% of fructose, the point estimate of 0.292 (column 1, Table 10) implies that a one standard deviation increase in exposure to HFCS (0.038, see Table 8a for summary statistics on my main variables) is associated with a 1.1 percentage points increase in change in the probability of being obese. In addition, the overweight coefficient is also statistically

significant, indicating a one standard deviation increase in HFCS consumption is correlated with 0.92 percentage points increase in the probability of being overweight. Using the broader definition of HFCS diminishes the effect of one-standard-deviation increase in exposure to HFCS (0.092) to almost 0.4 percentage points increase in the probability of becoming obese, while the overweight's coefficient is not significant.

Again, in Graphs 4 and 5 is plotted the non-linear relationships between the obesity and overweight and the exposure to imports of HFCS with high concentration of fructose (50% and above). Both distinctly show that the results are not driven by outliers either.

On the other hand, I also test the hypothesis whether the effect is mainly concentrated in urban areas. Surprisingly, in this case, the results are not stronger in those regions. When the treatment is limited to the imports of HFCS with 50% of fructose or higher, the coefficient of the urban interaction is not significant for obesity and it is significant and negative for overweight. Concretely, an increase of one standard deviation in exposure to HFCS is associated with 1.14 percentage points decrease in the probability of being overweight among women living in urban areas (Table 12, column 2).

As previously mentioned in the literature review, this unexpected result could indicate that women in urban areas develop a higher awareness for staying healthy and fit as countries experiment economic growth and income increases. As a result, the gap between obesity rates in urban and rural areas should be shrinking (Bixby, H., Bentham, J., Zhou, B. et al., 2019). However, it is difficult to affirm this with great certainty considering the prevalence of obesity in the sample is still much larger in urban settings (Table 8b).

In both analyses, the coefficients remain similar and statistically significant when I weight the regressions at the child level. Also, I re-estimate both effects dropping one country at a time and, again, the coefficient loose significance when Turkey is left out of the sample, suggesting it might be driving the results. Finally, I find similar effects when I estimate the model and its urban version without all the women who had children in the last six months.

6. Discussion and Conclusion

Despite the extensive research, HFCS is still one of the most misunderstood food ingredients, especially with respect to its role in the obesity epidemic and other health effects. In this research, I combine information on HFCS imports from around 70 countries to estimate the effect of HFCS consumption on obesity in children and women. Although I find insignificant effects when I include in the treatment variable all the imports of HFCS with any amount of fructose, I find large and significant positive effects on children's weight-related nutritional outcomes and women's obesity when the treatment variable is defined as imports of HFCS containing at least 50% of fructose. This is consistent with some recent epidemiologic studies that have linked consumption of high doses of fructose to major increases in body weight and cardiovascular mortality (Lowndes et al., 2014; Stanhope et al., 2015). Additionally, I also determine that concerning the children, the effects on weight are concentrated among children older than 12 months who live in urban areas.

The strengths of this study include the large number of countries for which there are trade and health data available, the possibility of distinguish the imports of HFCS based on the amount of fructose, and a very large sample over a long period of time for both women and child health related outcomes, specially, compared to clinical trials which usually include few subjects and are limited to a short period of time (less than a year).

There are also limitations. First, I use only data on imports of HFCS, total sugars and total imports, but I do not have data on other imported processed foods, which could also have an impact on obesity. Second, I am limited to using child and women obesity data and do not have data on other health conditions. Considering consumption of sugar is highly associated with other chronic diseases, I am missing the calculation of effects of exposure of HFCS on diabetes, blood pressure and heart diseases, among other noncommunicable diseases.

As previously stated, obesity is a cause of morbidity and mortality and pose a huge challenge for the sustainability of healthcare systems of all the countries, but specially of developing countries. For this reason, issues related to the consumption of sugars carry nutritional, public health, and public policy implications. Up until now, several scientific organizations, including the WHO, have recommended significant restrictions on added sugars consumption, but have recognized

that it does not exist sufficient nutritional data to properly justify such decisions (WHO, 2015). The results of this study add scientific basis to justify these limitations.

There are a number of effective interventions that have been proven successful to increase the costs of unhealthy food or reduce exposure and access to it and, therefore, reduce its consumption. Regarding added sugars, examples include imposing taxes to sugar-sweetened beverages, which has already been implemented in the U.S (Allcott et al., 2019) and other European countries, or removing unhealthy snacks and other foods from all schools, childcare, healthcare facilities, and other governmental institutions (Gibson-Moore and Valentine, 2009). Other interventions are reducing the density of fast-food restaurants (Li et al., 2009) and limiting the exposure to food advertising on television (Veerman et al., 2009).

Finally, further research is needed to fully understand the mechanisms through which HFCS and, by extension, added sugars contribute to obesity (specifically in the epidemiological field), as well as other research on the impacts of consumption of HFCS on other noncommunicable diseases in order to raise more awareness of the risks of high-sugar diets.

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Appendices

Figure 1: Production of HFCS by country.

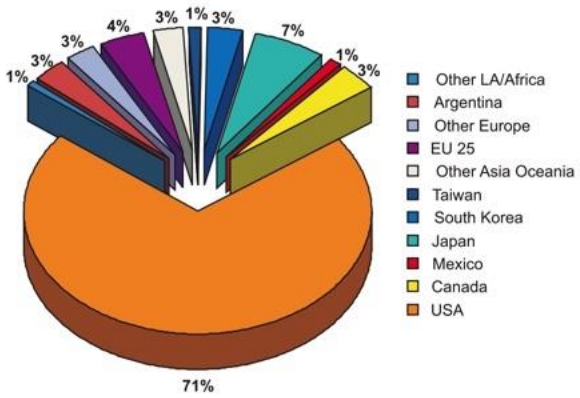


Fig. 1. Country-wise production of high fructose corn syrup.

Source: Singh, R.S. (2011). Enzymatic preparation of high fructose syrup from inulin, in: P.S. Panesar, H.K. Sharma, B.C. Sarkar (Eds.). Bioprocessing of Foods, Asiatech Publishers Inc., New Delhi, 77-98.

Table 1a: Summary Statistics – Trade Data - Children Dataset

Countries with Imports of HFCS					
Trade data Variables:	**N**	**Mean**	**Std. Dev.**	**Min.**	**Max.**
Imports of HFCS (in Kt)	56	0.904	2.476	0	13.99
Imports of HFCS (in Kg per capita)	56	0.043	0.133	0	0.931
Average Imports of HFCS (in Kg per capita)	56	0.035	0.091	0	0.564
Imports of HFCS (>50%) (in Kt)	56	0.353	1.384	0	8.809
Imports of HFCS (>50%) (in Kg per capita)	56	0.007	0.022	0	0.131
Average Imports of HFCS (>50%) (in Kg per capita)	56	0.007	0.021	0	0.115
Imports of HFCS (>20%) (in Kt)	56	0.551	1.365	0	7.579
Imports of HFCS (>20%) (in Kg per capita)	56	0.036	0.119	0	0.843
Imports Total Sugars (in billions of US $)	56	0.091	0.157	0	0.891
Imports Total Sugars (in US $ per capita)	56	5.022	6.795	0	38.29
Imports Total Commodities (in billions of US $)	56	13.57	26.51	0.091	139.96
Imports Total Commodities (in US $ per capita)	56	571.3	676.0	65.16	3,987
Countries with No Imports of HFCS					
Trade data Variables:	**N**	**Mean**	**Std. Dev.**	**Min.**	**Max.**
Imports Total Sugars (in billions of US $)	14	0.007	0.026	0	0.098
Imports Total Sugars (in US $ per capita)	14	1.442	3.914	0	13.595
Imports Total Commodities (in billions of US $)	14	1.043	2.970	0	10.771
Imports Total Commodities (in US $ per capita)	14	177.4	448.7	0	1,614.30

Figure 2: Imports of HFCS by country (1986-2018) – Children's Dataset

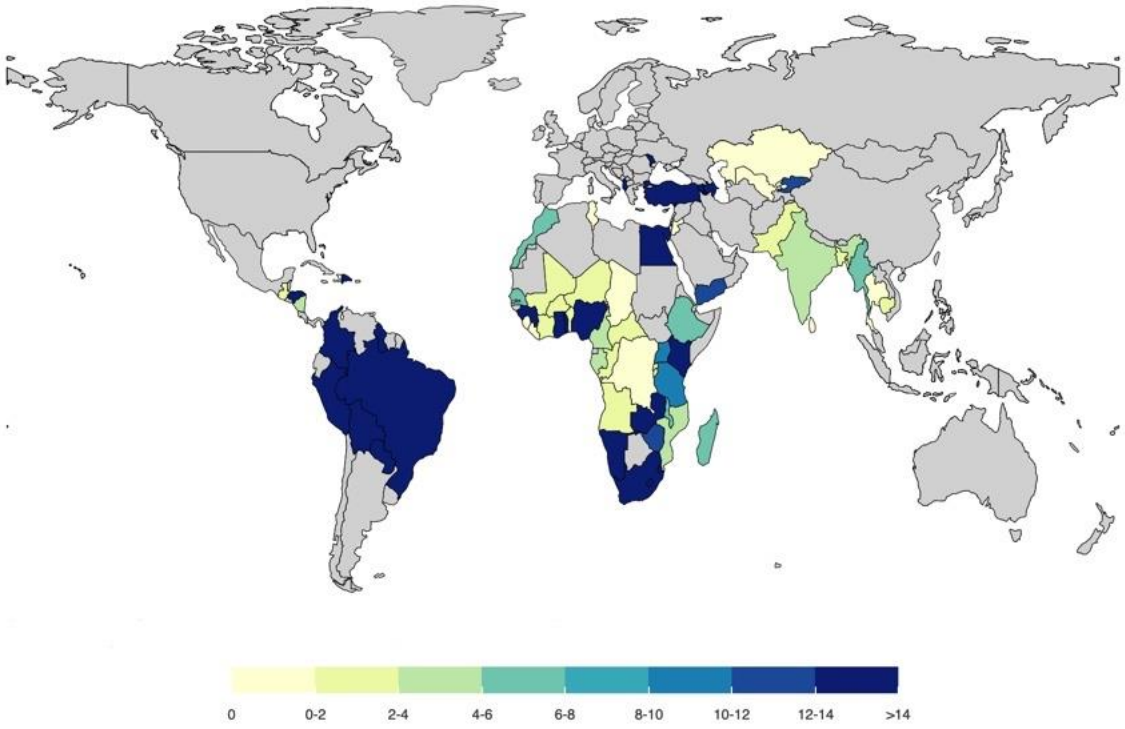


Table 1b: Summary Statistics – Children Health

Means with Standard Deviations in Parenthesis

	Countries with Imports of HFCS	Countries with No Imports of HFCS	World DHS
Num. of Subjects	1,392,788	115,024	1,507,812
Health Variables:			
Obese (%)	1.1	1.5	1.1
	(10.4)	(12.1)	(10.5)
Overweight (%)	3.7	4.2	3.7
	(18.8)	(20.0)	(18.9)
Weight-for-length/height z-score	-0.573	-0.389	-0.558
	(1.448)	(1.351)	(1.440)
Weight-for-age z-score	-1.275	-0.91	-1.245
	(1.418)	(1.279)	(1.411)
Length/height-for-age z-score	-1.481	-1.136	-1.452
	(1.803)	(1.675)	(1.795)
BMI-for-age z-score	-0.424	-0.265	-0.41
	(1.479)	(1.391)	(1.472)

Table 2

Panel A: Overall Effect (HFCS >20%) - Weighted Avg. Country

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>20%) last three years	0.00326	0.0257	0.196	0.246**	0.0779	0.198
	(0.00980)	(0.0171)	(0.162)	(0.117)	(0.167)	(0.169)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country	×	×	×	×	×	×
Avg. Child						
Constant	0.133***	0.284***	0.0981	-1.630***	-2.976***	0.231
	(0.0154)	(0.0371)	(0.378)	(0.298)	(0.305)	(0.333)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.017	0.029	0.110	0.180	0.101	0.093

Panel B: Overall Effect (HFCS >20%) - Weighted Avg. Child

Avg. Imp. HFCS pc (>20%) last three years	-0.00465	0.0143	0.556***	0.779***	0.277**	0.573***
	(0.00733)	(0.0235)	(0.197)	(0.159)	(0.129)	(0.198)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country						
Avg. Child	×	×	×	×	×	×
Constant	0.124**	0.227**	0.104	-1.630**	-3.718***	0.255
	(0.0610)	(0.110)	(0.741)	(0.646)	(0.549)	(0.705)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.015	0.034	0.159	0.233	0.104	0.133

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 3

Panel A: Overall Effect (HFCS >50%) - Weighted Avg. Country

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>50%) last three years	-0.0334	0.00762	1.566***	1.094***	-0.154	1.627***
	(0.0448)	(0.0906)	(0.399)	(0.323)	(0.456)	(0.401)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country	×	×	×	×	×	×
Avg. Child						
Constant	0.126***	0.279***	0.345	-1.486***	-3.028***	0.486
	(0.0148)	(0.0376)	(0.371)	(0.297)	(0.304)	(0.346)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.017	0.029	0.111	0.180	0.101	0.093

Panel B: Overall Effect (HFCS >50%) - Weighted Avg. Child

Avg. Imp. HFCS pc (>50%) last three years	-0.0144	0.0204	1.291***	1.520***	0.422**	1.331***
	(0.0146)	(0.0484)	(0.298)	(0.205)	(0.210)	(0.300)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country						
Avg. Child	×	×	×	×	×	×
Constant	0.123**	0.225**	0.150	-1.630**	-3.748***	0.301
	(0.0597)	(0.107)	(0.742)	(0.666)	(0.563)	(0.707)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.015	0.034	0.159	0.233	0.104	0.133

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 4

Panel A: Urban Effect - (>20% HFCS) - Weighted Avg. Country

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>20%) last three years	0.000102	-0.00234	0.139	0.226*	0.0599	0.139
	(0.0136)	(0.0232)	(0.162)	(0.120)	(0.191)	(0.168)
Avg. Imp. HFCS pc (>20%) last three years x Urban	0.00653	0.0657**	0.167	-0.00824	-0.108	0.202*
	(0.0179)	(0.0283)	(0.110)	(0.0864)	(0.137)	(0.113)
Constant	0.141***	0.301***	-0.128	-1.337***	-2.037***	-0.210
	(0.0158)	(0.0372)	(0.372)	(0.296)	(0.303)	(0.327)
Non-infant Effect - (>20% HFCS) - Weighted Avg. Country						
Avg. Imp. HFCS pc (>20%) last three years	0.0174	0.0652**	0.254	0.247*	-0.0617	0.325**
	(0.0137)	(0.0283)	(0.165)	(0.131)	(0.173)	(0.163)
Avg. Imp. HFCS pc (>20%) last three years x Non-infant	-0.0184	-0.0509*	-0.0580	-0.0317	0.0976	-0.129
	(0.0126)	(0.0288)	(0.112)	(0.0961)	(0.156)	(0.103)
Constant	0.140***	0.294***	-0.141	-1.339***	-2.026***	-0.229
	(0.0161)	(0.0373)	(0.373)	(0.295)	(0.304)	(0.328)
Urban Non-infant Effect - (>20% HFCS) - Weighted Avg. Country						
Avg. Imp. HFCS pc (>20%) last three years	0.00181	0.00571	0.113	0.227*	0.0736	0.121
	(0.0134)	(0.0220)	(0.156)	(0.120)	(0.181)	(0.162)
Avg. Imp. HFCS pc (>20%) last three years x Urban Non-infant	0.00310	0.0591*	0.293**	-0.0139	-0.180	0.314**
	(0.0233)	(0.0345)	(0.122)	(0.108)	(0.149)	(0.121)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country	×	×	×	×	×	×
Avg. Child						
Constant	0.141***	0.300***	-0.121	-1.338***	-2.041***	-0.204
	(0.0159)	(0.0374)	(0.374)	(0.296)	(0.304)	(0.328)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.018	0.030	0.118	0.192	0.179	0.118

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 5

Panel A: Urban Effect - (>20% HFCS) - Weighted Avg. Child

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>20%) last three years	-0.00146	0.00321	0.643***	0.700***	0.0193	0.665***
	(0.0113)	(0.0275)	(0.212)	(0.171)	(0.236)	(0.218)
Avg. Imp. HFCS pc (>20%) last three years x Urban	-0.00642	0.0195	-0.150	0.100	0.328	-0.144
	(0.0161)	(0.0315)	(0.159)	(0.169)	(0.348)	(0.169)
Constant	0.128**	0.234**	0.0256	-1.474**	-3.347***	0.0529
	(0.0621)	(0.111)	(0.793)	(0.577)	(0.449)	(0.816)
Non-infant Effect - (>20% HFCS) - Weighted Avg. Child						
Avg. Imp. HFCS pc (>20%) last three years	0.0342	0.0868*	0.539**	0.603***	-0.573*	0.843***
	(0.0258)	(0.0473)	(0.221)	(0.231)	(0.341)	(0.221)
Avg. Imp. HFCS pc (>20%) last three years x Non-infant	-0.0484	-0.0903*	0.0287	0.186	0.951**	-0.317
	(0.0308)	(0.0529)	(0.190)	(0.191)	(0.404)	(0.195)
Constant	0.127**	0.233**	0.0212	-1.470**	-3.327***	0.0445
	(0.0620)	(0.111)	(0.794)	(0.576)	(0.444)	(0.817)
Urban Non-infant Effect - (>20% HFCS) - Weighted Avg. Child						
Avg. Imp. HFCS pc (>20%) last three years	0.00783	0.00748	0.547***	0.728***	0.0140	0.619***
	(0.0150)	(0.0289)	(0.197)	(0.181)	(0.192)	(0.202)
Avg. Imp. HFCS pc (>20%) last three years x Urban Non-infant	-0.0291	0.0143	0.0348	0.0582	0.415	-0.0732
	(0.0310)	(0.0436)	(0.112)	(0.198)	(0.324)	(0.116)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country						
Avg. Child	×	×	×	×	×	×
Constant	0.129**	0.234**	0.0199	-1.473**	-3.348***	0.0503
	(0.0622)	(0.111)	(0.794)	(0.577)	(0.450)	(0.816)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.017	0.035	0.166	0.246	0.195	0.162

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 6

Panel A: Urban Effect - (>50% HFCS) - Weighted Avg. Country

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>50%) last three years	-0.0770	-0.144	1.255***	1.022***	-0.0781	1.243**
	(0.0586)	(0.111)	(0.475)	(0.347)	(0.613)	(0.498)
Avg. Imp. HFCS pc (>50%) last three years x Urban	0.0709	0.245**	0.480	0.104	-0.133	0.583*
	(0.0598)	(0.102)	(0.326)	(0.221)	(0.420)	(0.342)
Constant	0.134***	0.293***	0.107	-1.187***	-2.068***	0.0256
	(0.0149)	(0.0373)	(0.366)	(0.298)	(0.303)	(0.339)
Non-infant Effect - (>50% HFCS) - Weighted Avg. Country						
Avg. Imp. HFCS pc (>50%) last three years	-0.0171	0.0794	1.412**	1.107***	-0.120	1.715***
	(0.0598)	(0.123)	(0.568)	(0.364)	(0.503)	(0.480)
Avg. Imp. HFCS pc (>50%) last three years x Non-infant	-0.0198	-0.0883	0.171	-0.0262	-0.0493	-0.137
	(0.0341)	(0.0893)	(0.359)	(0.249)	(0.505)	(0.283)
Constant	0.134***	0.291***	0.105	-1.188***	-2.067***	0.0202
	(0.0150)	(0.0374)	(0.365)	(0.298)	(0.304)	(0.340)
Urban Non-infant Effect - (>50% HFCS) - Weighted Avg. Country						
Avg. Imp. HFCS pc (>50%) last three years	-0.0602	-0.0921	1.237***	1.119***	0.0221	1.292***
	(0.0563)	(0.107)	(0.464)	(0.347)	(0.539)	(0.462)
Avg. Imp. HFCS pc (>50%) last three years x Urban Non-infant	0.0534	0.198**	0.623**	-0.0645	-0.362	0.617**
	(0.0624)	(0.0927)	(0.254)	(0.191)	(0.332)	(0.298)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country	×	×	×	×	×	×
Avg. Child						
Constant	0.134***	0.293***	0.108	-1.189***	-2.070***	0.0258
	(0.0149)	(0.0374)	(0.366)	(0.298)	(0.303)	(0.340)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.018	0.030	0.118	0.192	0.179	0.118

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 7

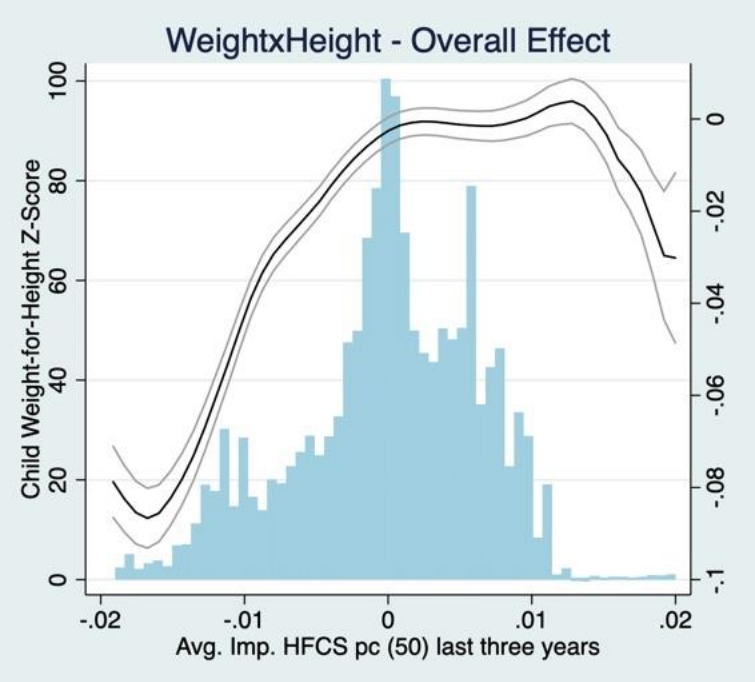
Panel A: Urban Effect - (>50% HFCS) - Weighted Avg. Child

VARIABLES	(1) Obese	(2) Overweight	(3) Weight for Height	(4) Weight for Age	(5) Height for Age	(6) BMI for Age
Avg. Imp. HFCS pc (>50%) last three years	-0.0206	-0.00881	1.490***	1.584***	0.113	1.514***
	(0.0210)	(0.0534)	(0.348)	(0.210)	(0.330)	(0.354)
Avg. Imp. HFCS pc (>50%) last three years x Urban	0.00945	0.0448	-0.310	-0.143	0.327	-0.291
	(0.0210)	(0.0326)	(0.202)	(0.227)	(0.360)	(0.202)
Constant	0.127**	0.232**	0.0689	-1.464**	-3.358***	0.0916
	(0.0608)	(0.109)	(0.796)	(0.595)	(0.456)	(0.820)
Non-infant Effect - (>50% HFCS) - Weighted Avg. Child						
Avg. Imp. HFCS pc (>50%) last three years	0.0207	0.0672	0.982***	1.218***	-0.433	1.674***
	(0.0339)	(0.0724)	(0.376)	(0.400)	(0.634)	(0.328)
Avg. Imp. HFCS pc (>50%) last three years x Non-infant	-0.0433	-0.0579	0.377	0.335	0.935	-0.430
	(0.0387)	(0.0709)	(0.274)	(0.357)	(0.717)	(0.298)
Constant	0.127**	0.233**	0.0646	-1.466**	-3.348***	0.0844
	(0.0608)	(0.108)	(0.795)	(0.595)	(0.455)	(0.820)
Urban Non-infant Effect - (>50% HFCS) - Weighted Avg. Child						
Avg. Imp. HFCS pc (>50%) last three years	-0.0102	-0.00113	1.314***	1.600***	0.172	1.478***
	(0.0194)	(0.0513)	(0.311)	(0.219)	(0.289)	(0.296)
Avg. Imp. HFCS pc (>50%) last three years x Urban Non-infant	-0.00794	0.0404	-0.0481	-0.205	0.292	-0.288*
	(0.0290)	(0.0389)	(0.130)	(0.170)	(0.327)	(0.148)
Demographic Controls	×	×	×	×	×	×
Trade Controls	×	×	×	×	×	×
Avg. Country						
Avg. Child	×	×	×	×	×	×
Constant	0.127**	0.232**	0.0638	-1.464**	-3.356***	0.0906
	(0.0608)	(0.109)	(0.795)	(0.595)	(0.456)	(0.820)
Observations	980,701	980,701	980,701	1,013,539	993,860	981,860
R-squared	0.017	0.035	0.166	0.246	0.195	0.162

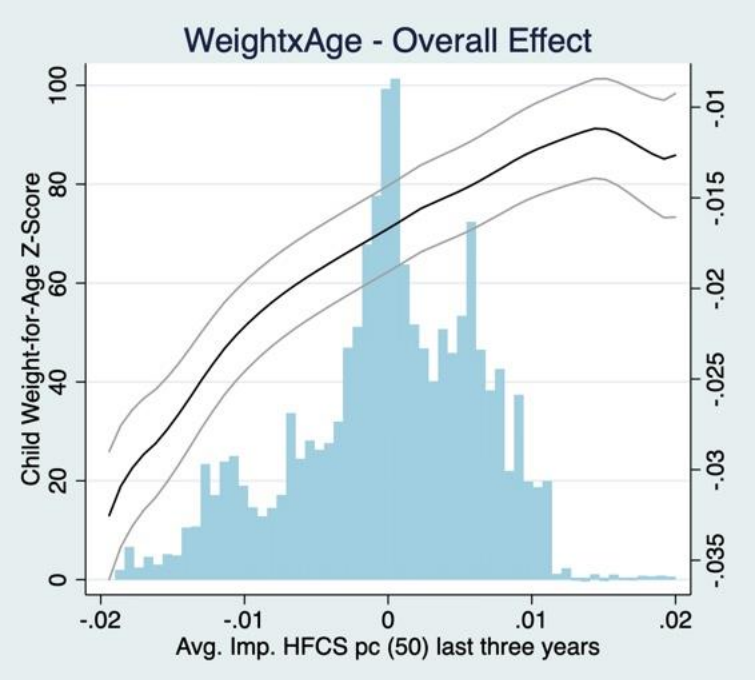
Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Graph 1: Non-linear relationship between weight-for-height and Imports of HFCS (50%)



Graph 2: Non-linear relationship between weight-for-age and Imports of HFCS (50%)



Graph 3: Non-linear relationship between BMI-for-age and Imports of HFCS (50%)

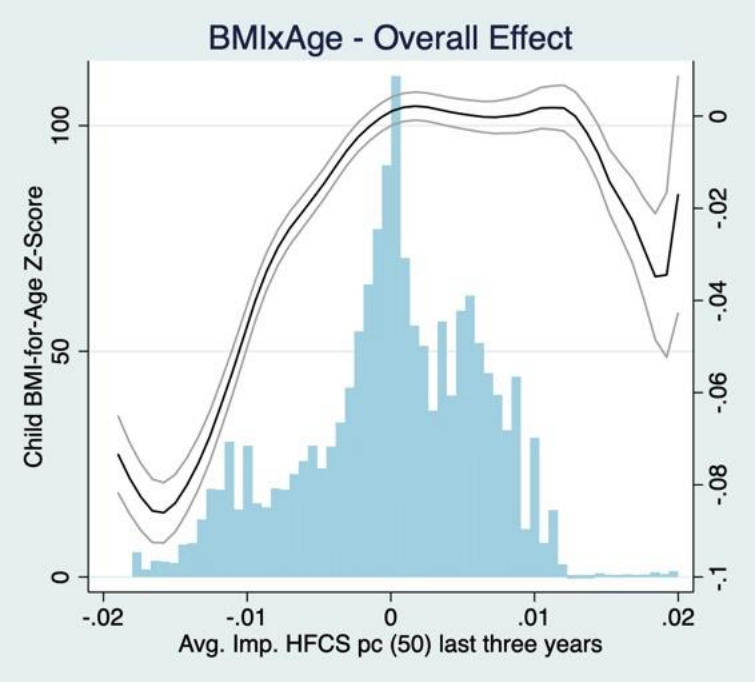


Figure 3: Child Growth Standards (WHO)

Figure 4: Child Growth Standards (WHO)

Table 8a: Summary Statistics – Trade Data - Women

Countries with Imports of HFCS					
Trade data Variables:	**N**	**Mean**	**Std. Dev.**	**Min.**	**Max.**
Imports of HFCS (in Kt)	56	1.420	5.181	0	34.64
Imports of HFCS (in Kg) per capita	56	0.049	0.140	0	0.931
Average Imports of HFCS (in Kg) per capita	56	0.035	0.092	0	0.564
Imports of HFCS (>50%) (in Kt)	56	0.886	4.259	0	30.17
Imports of HFCS (>50%) (in Kg per capita)	56	0.012	0.046	0	0.308
Average Imports of HFCS (>50%) (in Kg) per capita	56	0.011	0.038	0	0.236
Imports of HFCS (>20%) (in Kt)	56	0.534	1.291	0	7.762
Imports of HFCS (>20%) (in Kg per capita)	56	0.036	0.119	0	0.843
Imports Total Sugars (in billions of US $)	56	0.082	0.140	0	0.816
Imports Total Sugars (in US $) per capita	56	4.254	6.165	0.064	38.29
Imports Total Commodities (in billions of US $)	56	11.86	17.96	0.091	73.17
Imports Total Commodities (in US $) per capita	56	467.9	464.5	27.76	1902.0
Countries with No Imports of HFCS					
Trade data Variables:	**N**	**Mean**	**Std. Dev.**	**Min.**	**Max.**
Imports Total Sugars (in billions of US $)	21	0.005	0.021	0	0.098
Imports Total Sugars (in US $) per capita	21	2.053	5.766	0	22.92
Imports Total Commodities (in billions of US $)	21	0.898	2.497	0	10.771
Imports Total Commodities (in US $) per capita	21	263.5	677.3	0	2733.8

Figure 5: Imports of HFCS by country (1985-2014) – Women's Dataset

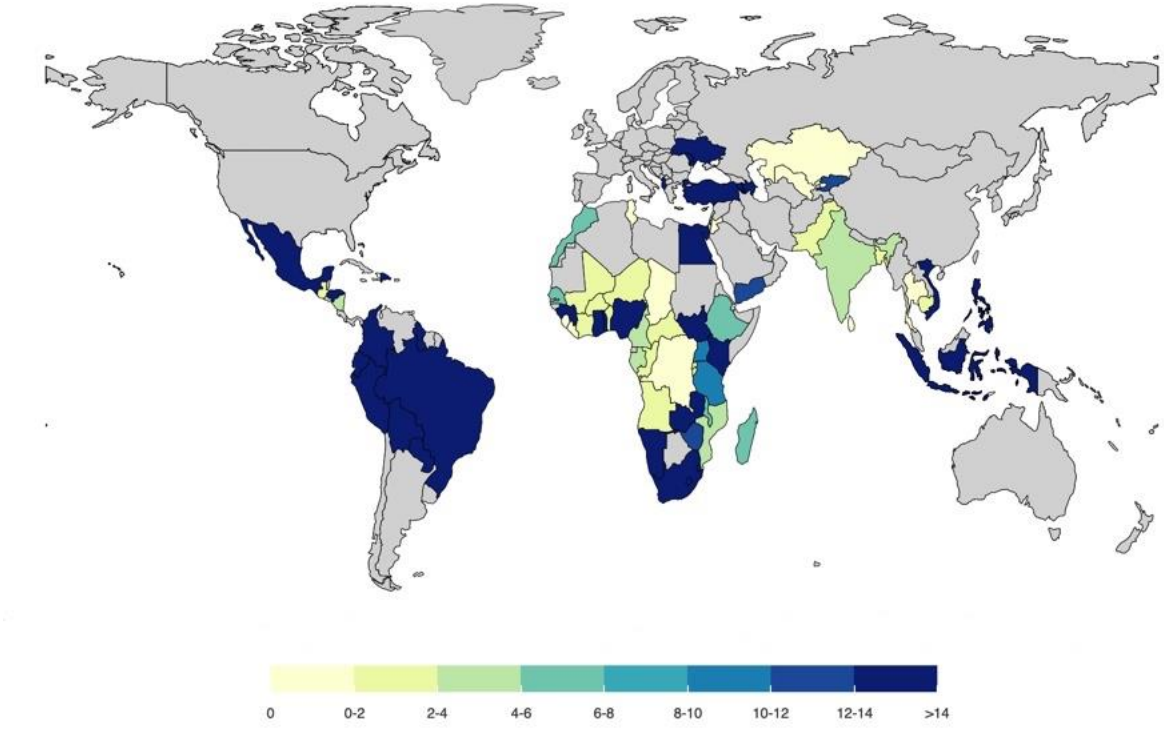


Table 8b: Summary Statistics – Women Health

Means with Standard Deviations in Parenthesis

	Countries with Imports of HFCS	Countries with No Imports of HFCS	World DHS
Num. of Subjects	1,412,370	121,344	1,533,714
Health Variables:			
Obese (%)	5.8	6.6	5.9
	(23.4)	(24.9)	(23.5)
Obese (%) if urban	10.8	10.0	10.7
	(31.0)	(30.0)	(30.9)
Overweight (%)	13.3	15.7	13.5
	(34.0)	(36.3)	(34.1)
Overweight (%) if urban	21.8	20.2	21.6
	(41.3)	(40.1)	(41.2)
Normal range (%)	56.1	66.1	56.7
	(49.6)	(47.3)	(49.6)
Underweight (%)	24.8	11.6	24.0
	(43.2)	(32.0)	(42.7)
Body Mass Index (BMI)	21.7	22.7	21.8
	(4.6)	(4.4)	(4.6)
Weight (in Kg)	51.5	56.9	51.8
	(12.5)	(11.8)	(12.6)
Height (in m)	1.5	1.6	1.5
	(6.8)	(7.3)	(6.9)

Table 9

Panel A: Overall Effect (HFCS >20%) - Weighted Avg. Country

VARIABLES	(1) **Obese**	(2) **Overweight**	(3) **Normal**	(4) **Underweight**
Avg. Imp. HFCS pc (>20%) last three years	0.0511**	0.0165	-0.0495	-0.0180
	(0.0255)	(0.0308)	(0.0428)	(0.0251)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country	×	×	×	×
Avg. Child				
Constant	-0.0531	-0.190**	0.997***	0.246***
	(0.0493)	(0.0845)	(0.117)	(0.0581)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.124	0.076	0.111	0.083

Panel B: Overall Effect (HFCS >20%) - Weighted Avg. Child

Avg. Imp. HFCS pc (>20%) last three years	0.153***	0.0681	-0.150*	-0.0709**
	(0.0494)	(0.0443)	(0.0828)	(0.0314)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country				
Avg. Child	×	×	×	×
Constant	0.104	-0.412*	0.712	0.596***
	(0.253)	(0.228)	(0.457)	(0.134)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.135	0.108	0.044	0.124

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 10

Panel C: Overall Effect (HFCS >50%) - Weighted Avg. Country

VARIABLES	(1) **Obese**	(2) **Overweight**	(3) **Normal**	(4) **Underweight**
Avg. Imp. HFCS pc (>50%) last three years	0.292***	0.243***	-0.387***	-0.148***
	(0.0817)	(0.0765)	(0.0958)	(0.0534)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country	×	×	×	×
Avg. Child				
Constant	0.00579	-0.127	0.909***	0.212***
	(0.0495)	(0.0847)	(0.113)	(0.0639)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.125	0.076	0.111	0.083

Panel D: Overall Effect (HFCS >50%) - Weighted Avg. Child

Avg. Imp. HFCS pc (>50%) last three years	0.354***	0.200**	-0.380**	-0.174***
	(0.103)	(0.0976)	(0.163)	(0.0463)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country				
Avg. Child	×	×	×	×
Constant	0.190	-0.343	0.604	0.549***
	(0.238)	(0.222)	(0.446)	(0.124)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.135	0.108	0.044	0.124

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Table 11

Panel A: Urban Effect - (>20% HFCS) - Weighted Avg. Country				
	(1)	(2)	(3)	(4)
VARIABLES	**Obese**	**Overweight**	**Normal**	**Underweight**
Avg. Imp. HFCS pc (>20%) last three years	0.0352	0.0497*	-0.0464	-0.0385
	(0.0234)	(0.0293)	(0.0423)	(0.0302)
Avg. Imp. HFCS pc (>20%) last three years x Urban	0.0293*	-0.0614***	-0.00573	0.0378**
	(0.0171)	(0.0226)	(0.0385)	(0.0189)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country	×	×	×	×
Avg. Child				
Constant	-0.0515	-0.194**	0.997***	0.248***
	(0.0496)	(0.0853)	(0.117)	(0.0585)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.124	0.076	0.111	0.083
Panel B: Urban Effect - (>20% HFCS) - Weighted Avg. Child				
Avg. Imp. HFCS pc (>20%) last three years	0.140***	0.160***	-0.0502	0.160***
	(0.0496)	(0.0478)	(0.0845)	(0.0478)
Avg. Imp. HFCS pc (>20%) last three years x Urban	0.0243	-0.174***	-0.189**	-0.174***
	(0.0409)	(0.0337)	(0.0738)	(0.0337)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country				
Avg. Child	×	×	×	×
Constant	0.107	-0.433*	0.690	-0.433*
	(0.252)	(0.235)	(0.451)	(0.235)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.135	0.108	0.044	0.108

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

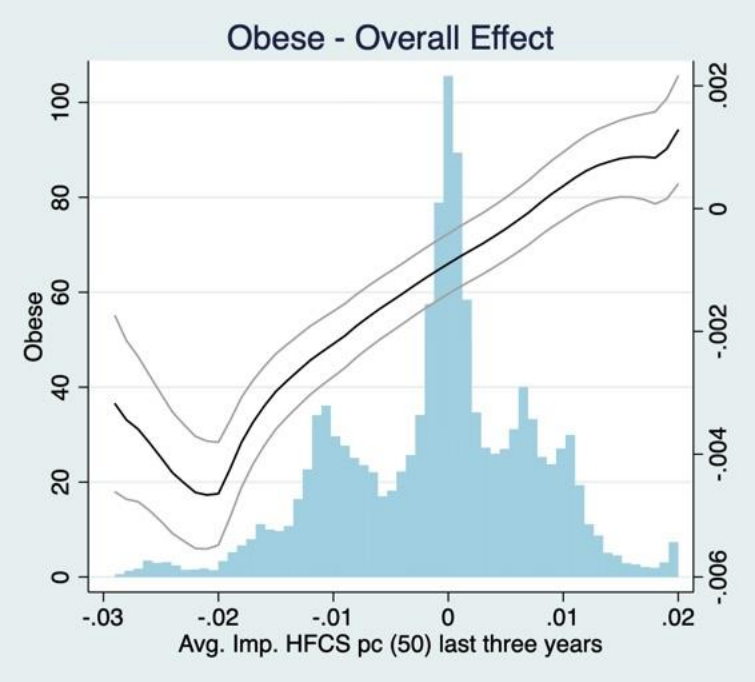
Table 12

Panel C: Urban Effect - (>50% HFCS) - Weighted Avg. Country				
VARIABLES	(1) Obese	(2) Overweight	(3) Normal	(4) Underweight
Avg. Imp. HFCS pc (>50%) last three years	0.276***	0.430***	-0.456***	-0.249***
	(0.0640)	(0.0820)	(0.0952)	(0.0660)
Avg. Imp. HFCS pc (>50%) last three years x Urban	0.0261	-0.300***	0.111	0.163***
	(0.0715)	(0.0478)	(0.0915)	(0.0470)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country	×	×	×	×
Avg. Child				
Constant	0.00622	-0.132	0.911***	0.215***
	(0.0501)	(0.0841)	(0.113)	(0.0642)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.125	0.077	0.111	0.083
Panel D: Urban Effect - (>50% HFCS) - Weighted Avg. Child				
Avg. Imp. HFCS pc (>50%) last three years	0.421***	0.449***	-0.365**	-0.505***
	(0.0923)	(0.102)	(0.160)	(0.0755)
Avg. Imp. HFCS pc (>50%) last three years x Urban	-0.0985	-0.371***	-0.0231	0.493***
	(0.0858)	(0.0629)	(0.0926)	(0.0954)
Demographic Controls	×	×	×	×
Trade Controls	×	×	×	×
Avg. Country				
Avg. Child	×	×	×	×
Constant	0.191	-0.338	0.604	0.543***
	(0.238)	(0.220)	(0.446)	(0.123)
Observations	1,159,174	1,159,174	1,159,174	1,159,174
R-squared	0.135	0.108	0.044	0.124

Standard errors in parenthesis

* p≤.1; ** p≤.05; *** p≤.01

Graph 4: Non-linear relationship between obesity and Imports of HFCS (50%)



Graph 5: Non-linear relationship between overweight and Imports of HFCS (50%)