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U.S. obesity as delayed effect of excess sugar

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ABSTRACT

In the last century, U.S. diets were transformed, including the addition of sugars to industrially-processed foods. While excess sugar has often been implicated in the dramatic increase in U.S. adult obesity over the past 30 years, an unexplained question is why the increase in obesity took place many years after the increases in U.S. sugar consumption. To address this, here we explain adult obesity increase as the cumulative effect of increased sugar calories consumed over time. In our model, which uses annual data on U.S. sugar consumption as the input variable, each age cohort inherits the obesity rate in the previous year plus a simple function of the mean excess sugar consumed in the current year. This simple model replicates three aspects of the data: (a) the delayed timing and magnitude of the increase in average U.S. adult obesity (from about 15% in 1970 to almost 40% by 2015); (b) the increase of obesity rates by age group (reaching 47% obesity by age 50) for the year 2015 in a well-documented U.S. state; and (c) the pre-adult increase of obesity rates by several percent from 1988 to the mid-2000s, and subsequent modest decline in obesity rates among younger children since the mid-2000s. Under this model, the sharp rise in adult obesity after 1990 reflects the delayed effects of added sugar calories consumed among children of the 1970s and 1980s.

1. Introduction

In approximately two generations, obesity has become an epidemic across the Developed world (Goryakin et al., 2017). Worldwide, obesity nearly tripled between 1975 and 2016; by 2016, more than 650 million adults were obese, about 13% of the world's adult population (World Health Organization, 2018). Among children and adolescents aged 5–19, the global obesity rate rose from 1% in 1975 to 6% of girls and 8% of boys in 2016 (World Health Organization, 2018).

Although the rise in U.S. obesity dates to the mid 20th century (Sobal and Stunkard, 1989), the most substantial and rapid increase in adult obesity has occurred over the past 30 years (Cook et al., 2017; Dwyer-Lindgren et al., 2013; Flegal et al., 2016; Kranjac and Wagmiller, 2016; Ogden et al., 2014). From 1990 to 2016 the national adult obesity rate almost doubled; in certain U.S. states (WV, MS, AK, LA, AL, KY, SC) it nearly tripled, from about an eighth of the population to more than a third.

There are clearly many contributors to the obesity crisis, many of which have been studied over past decades through randomized control trials and through population-scale statistical analyses (Hruby and Hu, 2015; Rippe, 2013). Among the major factors in this recent increase, one is age. In the U.S. in 2015–16, obesity prevalence was about 36% among adults aged 20–39 years, 43% for 40–59 years, and 41% for 60 and older (Centers for Disease Control & Prevention, 2017a). In 2015–2016, nearly 20% of 6–19 year-olds were obese (Hales et al., 2017). Data from the state of Wisconsin (Wisconsin Health Atlas, 2019), which ranks near the middle of U.S. states in obesity, document the age profiles in more detail for the year 2015, showing the continuous rise in obesity from age two years to middle age, where the obesity rate plateaus near 47%, before falling off among those aged over 75 years (Fig. 1a). The increase in obesity through childhood is a recent phenomenon, however; in the U.S. in 1970s there was an average decrease in obesity rate from ages two to nineteen (Wang and Beydoun, 2002).

Another major factor is socioeconomic status (Hruschka, 2012; Smith, 2017; Sobal and Stunkard, 1989; Tafreschi, 2015; Salmasi and Celidon, 2017; Villar and Quintana-Domeque, 2009). In the U.S., the increase in obesity rate between 1990 and 2015 has correlated inversely with median household income (Bentley et al., 2018). Looking at county-scale obesity rates from 2004 to 2013 (Centers for Disease Control & Prevention, 2017b), the average obesity among the 100 poorest counties in the U.S. increased from about 29.9% to 36.5%, but only from 21.2% to 24.6% among the 100 richest counties. As obesity rates have increased faster in poor communities, a likely driver has been the lowering cost of food calories (Mattson et al., 2014; Mullan et al., 2017), including sugar-sweetened beverages (Basu et al., 2013a; Hu and Malik, 2010;

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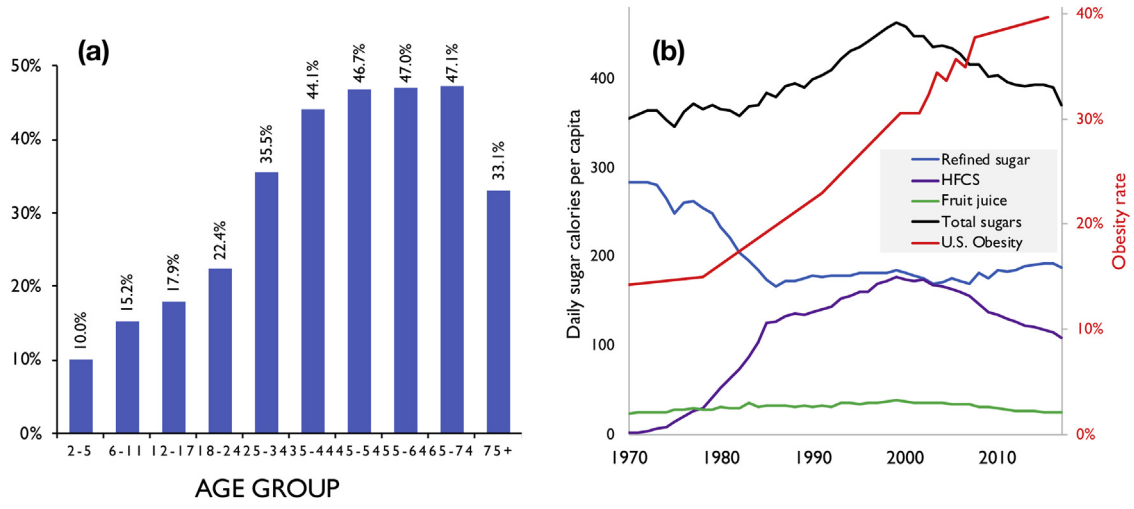

Fig. 1. (a) Obesity rates in Wisconsin, by age group, 2015. Adapted from (Wisconsin Health Atlas, 2019). (b) Daily U.S. sugar consumption per capita, 1970–2017, using data from USDA (U.S. Department of Agriculture, Economic Research Service, 2019), versus U.S. obesity rate (Centers for Disease Control & Prevention, 2017c) using the *y*-axis on the right.

Johnson et al., 2007, 2016; Malik et al., 2006; Shang et al., 2012; Song et al., 2012; Wang et al., 2008).

Since 1970, when high-fructose corn syrup (HCFS) was introduced at commercial scale into processed foods and sugar-sweetened beverages (SSBs), consumption of HFCS increased from virtually zero in 1970 to over 60 pounds per capita annually in 2000 (Gerrior et al., 2004). Overall, the consumption of added sugars increased from about 70 pounds per person annually in 1970 (340 cal/day) to almost 90 pounds per capita (440 cal/day) by the late 1990s (Fig. 1b). By the 2000s, the top 20% of consumers of caloric sweeteners were ingesting over 300 kcal from HFCS per day (Bray et al., 2004). Much of the rise in total added sugar consumption between 1970 and 2000 is attributable to HFCS (Fig. 1b), often in SSBs that contribute additional calories without suppressing appetite for the intake of other foods (Almiron-Roig et al., 2013; Mattes and Campbell, 2009).

The correlation between the increased consumption of added sugars in U.S. foods and the rise of obesity (Fig. 1b) has been recognized (Bray et al., 2004; Basu et al., 2013b; Lustig et al., 2015), even by the sweetener industry (Corn Refiners Association, 2006). Consequently, sugar consumption has been hypothesized to be a primary driver of the recent obesity increase (Basu et al., 2013a,b; Bray et al., 2004; Bray, 2007; Lustig et al., 2015; Nielsen et al., 2002; Nielsen and Popkin, 2004; Popkin and Hawkes, 2016). Time series data from 75 countries, for example, indicate that a 1% rise in soft drink consumption in a country predicts about a 2% increase in obesity (Basu et al., 2013b).

Critics of the sugar-obesity theory, however, point out the substantial time lag between changes in sugar consumption and change in obesity rate. After sugar consumption had begun to decline (Welsh et al., 2011), van Buul et al. (2014, p. 119) noted that "over the last 5 years, the global annual consumption of carbonated soft drinks has remained constant or even has declined "Obesity rates, however, seem to have increased independently of these shifts." In 2014, for example, obesity rates were still rising, despite the 25% decrease in added sugar consumption among U.S. residents between 1999 and 2008 (van Buul et al., 2014; Welsh et al., 2011). By 2017, obesity rates had only just started to level off or decrease, in some but not all U.S. states. The delay is such that annual adult obesity rates from 2004 to 2013 (Centers for Disease Control & Prevention, 2017b) correlate quite well with annual per capita sugar consumption (U.S. Department of Agriculture, Economic Research Service, 2019) twelve years before, i.e. from 1992 to 2001 respectively, with $r^2 = 0.933$ among the 100 poorest counties in the U.S. and $r^2 = 0.908$ among the 100 richest counties.

Although readily observed, this time lag remains unexplained. While most population health studies explore the effect of environmental, nutritional or behavioral factors on obesity levels, few have explicitly explored the temporal delay between cause and effect. A specific study helps illustrate why we would expect a substantial delay between increased sugar consumption and national obesity rates. In a 19-month study controlling for other variables, Ludwig et al. (2001) found that for each additional SSB consumed per day, BMI in children increased by a mean of 0.24 ± 0.14. This suggests an increase on the order of about 1.6 per in BMI decade per extra daily SSB. This "back-of-the-envelope" line of thinking motivates our specific modeling approach.

Here we propose that obesity increase can be explained as the accumulated effect of childhood exposure to excess sugar calories together with continued consumption. Since childhood obesity tends to predict obesity in adulthood, the rising obesity rates of adults the 1990s and 2000s ought to reflect their diets as children of the 1970s and 1980s. In 1990, for example, the 18-year-olds entering the adult CDC dataset would have been born in or about 1972. Current adult obesity rates would have only just begun to register a decline in sugar consumption that began in 1998.

With a model that can capture these different dynamics, with per-capita sugar consumption as the single input variable, we present obesity as the cumulative result of excess sugar consumption since 1970. In formulating a parsimonious, sugar-driven model, our goal is to replicate three different aspects of the obesity trends in the U.S.: (a) the overall increase of U.S. adult obesity since 1970; (b) the profile of obesity by age group for a recent year; and (c) the change in obesity rates by pre-adult age group since 1990.

2. Models and methods

As shown in Fig. 2, we model the obesity rate for each age cohort as its obesity rate in the previous year plus a logit function of the mean excess sugar consumed in the current year. For each new year, obesity for age group y, in year t is modeled as the fraction already obese in the previous year, $O_{t-1,y-1}$ plus an additional fraction of those not yet obese, $(1 - O_{t-1,y-1})$. The current year's age-specific obesity rates are determined by a logit function, as

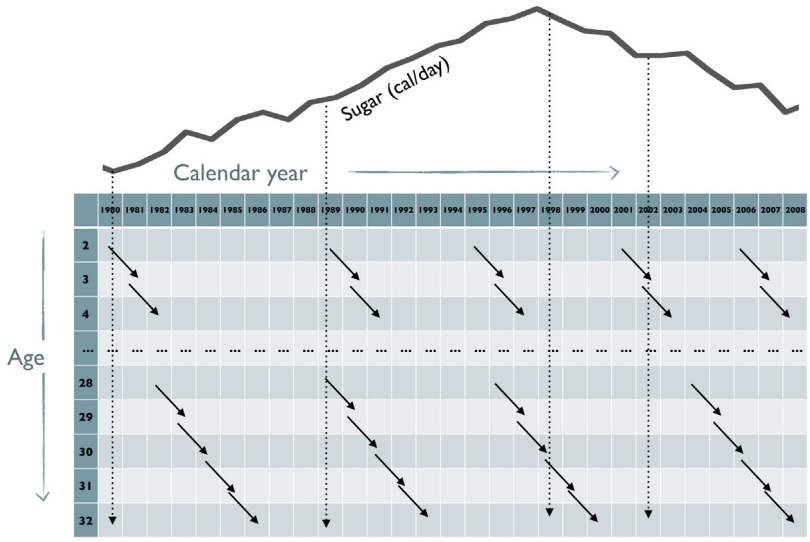

Fig. 2. Schematic of the model. Each box represents a cohort of a certain age in years (vertical axis), for a given calendar year (horizontal axis). The downward arrows represent the transmission process in the model, whereby each age cohort inherits its obesity rate from the previous year plus a simple function of the mean per capita sugar calories (represented by the plotted trend above the matrix) consumed in the current year.

follows:

$$O_{t,y} = O_{t-1,y-1} + (1 - O_{t-1,y-1}) \frac{e^{\alpha+\beta\rho_t}}{1 + e^{\alpha+\beta\rho_t}}. \quad (1)$$

where ρ_t is the excess added sugar consumption in year t, and β and α represent the effect of excess sugar and a baseline, respectively. A logit function is a standard way to model the probability of a binary state (i.e., obese or not obese) using a linear function of predictor variables. It assumes that the logit of the probability of becoming obese, rather than the probability itself, follows a linear model.

As shown in Fig. 2, the model generates a matrix of the 46 calendar years, t, from 1971 to 2016, versus life ages, y, from age 2 through age 75. In each cell of this array, obesity in year t for age y is calculated as a function of obesity for the same cohort in the previous year, $t-1$, when the cohort was one year younger, age $y-1$. The model replicates the generational time lag through a simple stochastic process of superfluous sugar calories causing obesity rates to increase over the lifespan of each birth-year cohort. In this multiplicative process, early excesses are compounded over the long-term, such that excess sugar consumption in childhood register years later as rising adult obesity rates.

The two parameters, β and α, are adjusted for the model to fit two aspects of the data: the timeline of increase in average obesity rate, and the profile of age versus obesity rate. The parameter α is the magnitude of the effect, whereas β reflects the log odds of non-obese individuals in a cohort becoming obese in the next year, as a function of their excess daily calories (expressed as fraction of a 2000 calories/day diet). For example, if $\alpha = -8.5$ and $\beta = 20$, then for each age cohort we expect about 0.05% more obesity in the next year for every 100 extra daily calories (5% of a 2000 calorie diet). The logit model is non-linear, so with these same α and β values, but with 300 excess calories per day (15% of a 2000 calorie diet), we would expect an additional 0.4% of the non-obese portion of the cohort to become obese in the next year.

The external input variable, ρ_t, is the caloric equivalent of the average excess sugar consumption per capita in the U.S., expressed for convenience as a fraction of a 2000-calorie diet, for each year since 1970 (U.S. Department of Agriculture, Economic Research Service, 2019). We used historical data (Wang and Beydoun, 2002) to approximate the age-obesity profile in 1970, which serves as the initial year of the model. The sugar data, recorded by the USDA (U.S. Department of Agriculture, Economic Research Service, 2019), are annual estimates of mean sugar consumed (cal/day) per capita in the U.S. since 1970. The data include four categories: refined sugar, HFCS, fruit juices and "other" sugars, and here we use the sum of all four of the categories as our estimate of daily added sugar calories for each year.

To test the model versus change in U.S. obesity prevalence, national level obesity rates for adults adult (20 and over) in the United States were obtained from the CDC, for selected years 1988–1994 through 2015–2016 (Centers for Disease Control & Prevention, 2019). Two additional estimates of U.S. adult obesity from the 1970s were added; these are from the National Health and Nutrition Examination Surveys of 1971–1974 (NHANES I) and 1976–1980 NHANES II (Flegal et al., 2016). For obesity among pre-adult cohorts, we used data reported by Ogden et al. (Ogden et al., 2014) from 40,780 children and adolescents between 1988–1994 and 2013–2014.

3. Results

Using two tunable parameters and the record of mean sugar consumption from 1970 to 2016, the model replicates the average U.S. obesity increase through calendar years and across age profiles. Using $\beta = 19.9$ and $\alpha = -8.5$, the model simultaneously replicates, firstly, the growth in average U.S. adult obesity since 1990 (Fig. 3a). Secondly, using these same parameters, it simultaneously replicates a typical U.S. state's age-obesity curve for 2015 (Fig. 3b). The only aspect of the age profile not replicated is obesity for ages 75 and over in 2015 (Fig. 3b), which is the cohort who grew up before the rapid increase in added sugars in U.S. diets.

The model also replicates aspects of the change in obesity rates by age group. Fig. 4 shows how the same model used in Fig. 3 ($\beta = 19.9$, $\alpha = -8.5$) also yields similar timelines from 1990 to 2014 for ages 2 to 5, 6 to 11, and 12 to 19. The comparison in Fig. 4 shows that the model is predicting a decline in obesity rate first among ages 2 to 5 beginning in the late 1990s, then among ages 6–11 beginning in the early 2000s, and subsequently among teenagers after about 2006. In the model, these decreases in obesity rates are delayed responses to the decline in excess sugars in the late 1990s. The data, though noisier, reflect these declines with about the same timing for the two younger age cohorts, but not the teenagers (Fig. 4). For ages 6 to 11, the actual decline was less than predicted by the model, and for ages 12 to 19, there was no actual decline at all.

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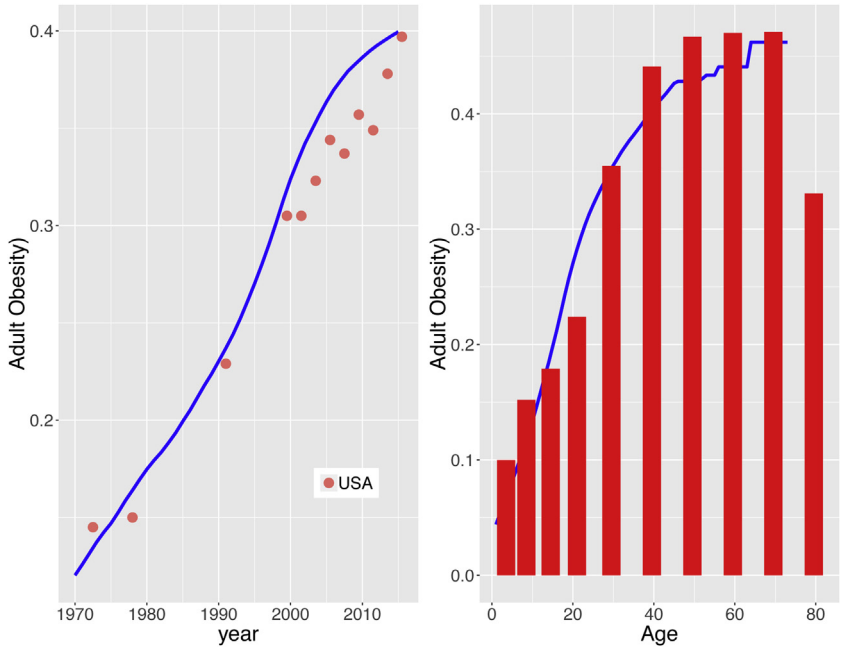

Fig. 3. (a) U.S. obesity rates (red circles) from the CDC (Centers for Disease Control & Prevention, 2017c) versus the model (blue line) of Eq. (1), using $\beta = 19.9$ and $\alpha = -8.5$. (b): Age versus obesity, in same model (blue line) for the modeled year 2016, versus data from representing obesity rates by age in Wisconsin, 2015–16 (Wisconsin Health Atlas, 2019).

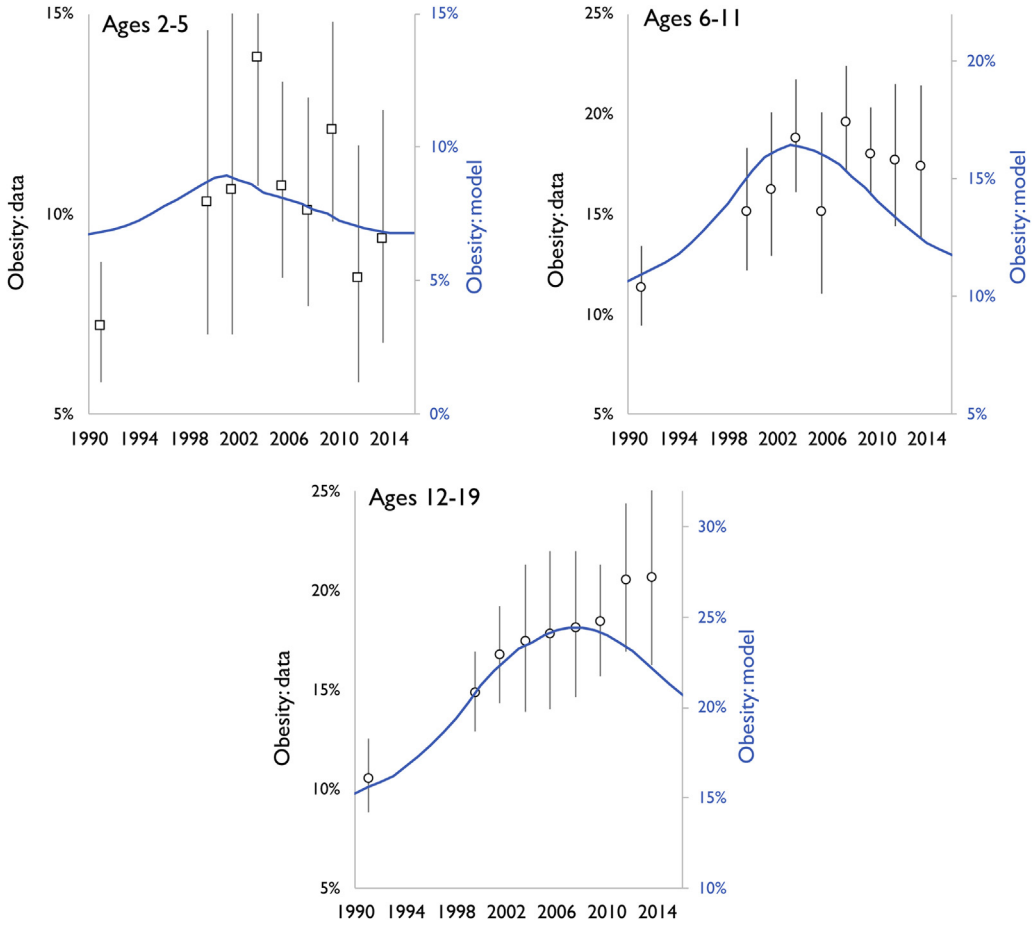

Fig. 4. Age-specific outputs of the specific model in Fig. 3 (blue curves; $\beta = 19.9$, $\alpha = -8.5$), compared with estimates of U.S. obesity prevalence among children and adolescents, 1988–1994 through 2014 (open circles with 95% C.I. bars (Ogden et al., 2014). The year of each data point is plotted midpoint, so the value for 1988–1994 is plotted at 1991, 2013–2014 plotted at 2013.5, etc. Pearson's correlations between model and mean data value: ages 2–5, $r = 0.591$, ages 6–11, $r = 0.502$; ages 12–19, $r = 0.840$. The y-axes on the left and right of each plot correspond to the data and model, respectively; this is due to slightly different definitions of obesity (the model calibrated to data using BMI above 30, the empirical estimates (Ogden et al., 2014) use the 95th percentile on CDC BMI-for-age-growth charts).

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4. Discussion

Here we have modeled the recent increase of U.S. adult obesity as being driven by added sugar calories over the lifespan of each birth-year cohort. With annual USDA sugar consumption figures as the input variable, the two-parameter model simultaneously replicates three different phenomena: the generational lag between sugar and obesity, the magnitude of the national rise in obesity, and a recent age-profile of obesity rates.

Our results indicate that excess U.S. sugar consumption is at least sufficient to explain the timing and magnitude of adult obesity change in the past 30 years, even as other factors (Gentile and Weir, 2018; Muscogiuri et al., 2018; Smits et al., 2017) could be factored into future models. In doing so, the model addresses a key critique of the sugar-obesity hypothesis (van Buul et al., 2014; Soenen and Westerterp-Plantenga, 2007) by introducing the mechanism to explain the years of delay in cause versus effect.

The sugar-obesity hypothesis is also supported by physiological, historical and economic evidence. Physiologically, sugar consumption elevates lipid levels in the blood (DiNicolantonio et al., 2015; Lustig et al., 2015; Grande, 1967; Grande et al., 1965; Kaufmann et al., 1966; Kuo and Bassett, 1965; Macdonald and Braithwaite, 1964; Teff et al., 2004). Although its specific role in the obesity epidemic is debated (Rippe, 2013; van Buul et al., 2014), fructose specifically does not stimulate insulin secretion or the production of the hormone (leptin) regulating long-term food energy balance, such that this sugar tends not to satisfy appetite (Bantle et al., 2000; Curry, 1989; Havel, 2005; Luo et al., 2015; Figlewicz and Benoit, 2009; Stanhope et al., 2009) and affects glucose metabolism, lipid profile and insulin resistance (Beyer et al., 2005; Bocarsly et al., 2010; Bray et al., 2004; Johnson et al., 2016; Jürgens et al., 2005; Pereira et al., 2017).

Consumed from a young age, sugar consumption appears to have long-lasting effects, not just habitually but also physiologically, in ways that could explain a generational delay between U.S. sugar consumption and subsequent obesity rates. Infant and toddler obesity is correlated with high-sugar infant foods (Koo et al., 2018) and even fructose in breast milk (Goran et al., 2017). Sugar consumption during pregnancy leads to increases in recruitment of pre-adipocytes to adipocytes in utero, such that children are born with an increase in fat cells that will accumulate more fat during life (Goran et al., 2013).

By the mid 1970s, children 0–2 years old were consuming about 6 grams of added sugar per kg of body weight, about three times that of adults at the time (Fomon, 1975; Life Sciences Research Office, 1976). From the 1970s to the 1990s, soft drinks were increasingly sweetened with HFCS, an inexpensive, domestically produced liquid sweetener (Corn Refiners Association, 2002). Between 1977 and 2001, sweetened beverage consumption increased by 135% across all age groups, equivalent to about 278 additional calories per day (Nielsen et al., 2002; Nielsen and Popkin, 2004). In the 2000s and 2010s, total added sugar intake in the US declined (Popkin and Hawkes, 2016), and by 2016, obesity rates in some U.S. states were leveling off.

Because 75-year-olds experienced childhood before the large-scale increase of sugar in processed foods, they may have developed less lifelong preference for added sugars in foods, but also it may be that they never laid down excess adipose tissue in during gestation. These explanations, potentially complementary to each other, may be addressed by detailed research on this elderly age group.

The age-stratified obesity data we used run through 2013–14, and in the future it will be revealing to see whether teenage obesity has declined since 2014 as the sugar-driven model predicts. It may be that for high school ages in particular, the effect socioeconomic status on food choice (Campbell et al., 2019) is obscured by aggregated obesity statistics. Data from the Centers for Disease Control & Prevention (2018) from 1999 to 2017, for example, suggest that obesity among high schoolers may have peaked in 2011 and 2013 among Asians and whites, respectively, but that obesity had continued to increase among blacks and Hispanics.

We speculate that poverty is a driver of sugar consumption (Alvarado, 2016; Hernandez, 2015; Shrewsbury and Wardle, 2012; Anderson, 2012; Datar, 2017; Hughes et al., 2010; Kowaleski-Jones et al., 2017; Mata et al., 2017; Moreno et al., 2016; Rosinger et al., 2017; Bentley et al., 2018). Economically, sugar is an inexpensive source of calories, and sweetened beverages have been a substantial portion of expenditures for low-income households (Garasky et al., 2016). Childhood obesity decreased after the 2009 changes in the US Special Supplemental Nutrition Program for Women, Infants, and Children (Daepp et al., 2019; Pan et al., 2019); we believe cutting the juice allowance by half helps explain why. If our model is correct, the effect of this 2009 change will follow these children into adulthood.

In the future, our model should be tested against different socioeconomic and demographic subpopulations, as well as for nations where there exist historical data on sugar consumption and obesity rates. For example, our delayed-effect model could offer a plausible explanation for the "Australian Paradox" (Barclay and Brand-Miller, 2011), that sugar consumption decreased in Australia at the same time that obesity increased.

5. Conclusion

In summary, we have modeled the recent increase of U.S. adult obesity rates since the 1990s as a legacy of increased consumption of excess sugars among children of the 1970s and 1980s. Our model proposes, for each age cohort, that the current obesity rate will be the obesity rate in the previous year plus a simple function of the mean excess sugar consumed in the current year. With just these inputs, the model can replicate the timing and magnitude of the national rise in obesity, as well as the profile of obesity rates by age group, and the different patterns of change in obesity among children and adolescent age group, where reduction in obesity registered first among young children in the late 1990s. This supports the perspective that the rise in U.S. adult obesity after 1990 was a generation-delayed effect of the increase in excess sugar calories consumed among children of the 1970s and 1980s.

References

Almiron-Roig, E., Palla, L., Guest, K., Ricchiuti, C., Vint, N., Jebb, S.A., Drewnowski, A., 2013. Factors that determine energy compensation: a systematic review of preload studies. Nutr. Rev. 71 (7), 458–473.
Alvarado, S.E., 2016. Neighborhood disadvantage and obesity across childhood and adolescence: evidence from the NLSY children and young adults cohort (1986–2010). Soc. Sci. Res. 57, 80–98.
Anderson, P.M., 2012. Parental employment, family routines and childhood obesity. Econ. Hum. Biol. 10 (4), 340–351.
Bantle, J.P., Raatz, S.K., Thomas, W., Georgopoulos, A., 2000. Effects of dietary fructose on plasma lipids in healthy subjects. Am. J. Clin. Nutr. 72 (5), 1128–1134.
Barclay, A.W., Brand-Miller, J., 2011. The Australian paradox: a substantial decline in sugars intake over the same timeframe that overweight and obesity have increased. Nutrients 3 (4), 491–504.
Basu, S., Yoffe, P., Hills, N., Lustig, R.H., 2013a. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. PLoS ONE 8 (2), e57873.
Basu, S., McKee, M., Galea, G., Stuckler, D., 2013b. Relationship of soft drink consumption to global overweight, obesity, and diabetes: a cross-national analysis of 75 countries. Am. J. Public Health 103 (11), 2071–2077.
Bentley, R.A., Ormerod, P., Ruck, D.J., 2018. Recent origin and evolution of obesity-income correlation across the United States. Palgrave Commun. 4 (1), 146.
Beyer, P.L., Caviar, E.M., McCallum, R.W., 2005. Fructose intake at current levels in the United States may cause gastrointestinal distress in normal adults. J. Am. Diet. Assoc. 105 (10), 1559–1566.
Bocarsly, M.E., Powell, E.S., Avena, N.M., Hoebel, B.G., 2010. High-fructose corn syrup causes characteristics of obesity in rats: increased body weight, body fat and triglyceride levels. Pharmacol. Biochem. Behav. 97 (1), 101–106.

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Bray, G.A., Nielsen, S.J., Popkin, B.M., 2004. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. Am. J. Clin. Nutr. 79 (4), 537–543.
Bray, G.A., 2007. How bad is fructose? Am. J. Clin. Nutr. 86 (4), 895–896.
Campbell, E.T., Franks, A.T., Joseph, P.V., 2019. Adolescent obesity in the past decade: a systematic review of genetics and determinants of food choice. J. Am. Assoc. Nurse Pract. 31 (6), 344–351.
Centers for Disease Control & Prevention, 2017a. Prevalence of Obesity Among Adults and Youth: United States, 2015–2016 NCHS Data Brief No. 288 https://www.cdc.gov/nchs/data/databriefs/db288.pdf.
Centers for Disease Control & Prevention, 2017b. Behavioral Risk Factor Surveillance System: Methods and References for County-Level Estimates and Ranks and State-Level Modeled Estimates. http://www.cdc.gov/brfss.
Centers for Disease Control & Prevention, 2017c. Data and Statistics, Overweight and Obesity. http://www.cdc.gov/obesity.
Centers for Disease Control & Prevention, 2018. Youth Risk Behavior Surveillance System. https://www.cdc.gov/healthyyouth/data/yrbs.
Centers for Disease Control & Prevention, 2019. Selected Health Conditions and Risk Factors, by Age: United States, Selected Years 1988–1994 Through 2015–2016. National Center for Health Statistics, Table 53. https://www.cdc.gov/nchs/hus/contents2017.htm#053.
Cook, W.K., Tseng, W., Tam, T.C., John, I., Lui, C., 2017. Ethnic-group socioeconomic status as an indicator of community-level disadvantage: a study of overweight/obesity in Asian American adolescents. Soc. Sci. Med. 184, 15–22.
Corn Refiners Association, 2002. Corn Syrups. https://www.industrydocumentslibrary.ucsf.edu/food/docs/fjpj0226.
Corn Refiners Association, 2006. The Many Uses of High Fructose Corn Syrup. https://www.industrydocumentslibrary.ucsf.edu/food/docs/txpj0226.
Curry, D.L., 1989. Effects of mannose and fructose on the synthesis and secretion of insulin. Pancreas 4 (1), 2–9.
Daepp, M.I.G., Gortmaker, S.L., Wang, Y.C., Long, M.W., Kenney, E.L., 2019. WIC food package changes: trends in childhood obesity prevalence. Pediatrics 143 (5), e20182841.
Datar, A., 2017. The more the heavier? Family size and childhood obesity in the U.S. Soc. Sci. Med. 180, 143–151.
DiNicolantonio, J.J., O'Keefe, J.H., Lucan, S.C., 2015. Added fructose: a principal driver of type 2 diabetes mellitus and its consequences. Mayo Clin. Proc. 90 (3), 372–381.
Dwyer-Lindgren, L., Freedman, G., Engell, R.E., Fleming, T.D., Lim, S.S., Murray, C.J.L., Mokdad, A.H., 2013. Prevalence of physical activity and obesity in U.S. counties, 2001–2011: a road map for action. Popul. Health Metr. 11, 7.
Figlewicz, D.P., Benoit, S.C., 2009. Insulin, leptin, and food reward: update 2008. Am. J. Physiol. Regul. Integr. Comp. Physiol. 296 (1), R9–R19.
Flegal, K.M., Kruszon-Moran, M.S., Caroll, M.D., Fryar, C.L., Ogden, C.L., 2016. Trends in obesity among adults in the United States, 2005 to 2014. JAMA 315 (21), 2284–2291.
Fomon, S.J., 1975. What are infants fed in the United States? Pediatrics 56 (3), 350–354.
Garasky, S., Mbwana, K., Romualdo, A., Tenaglio, A., Roy, M., 2016. Foods typically purchased by SNAP Households. United States Department of Agriculture. Food and Nutrition Service . http://www.fns.usda.gov/research-and-analysis.
Gentile, C.L., Weir, T.L., 2018. The gut microbiota at the intersection of diet and human health. Science 362 (6416), 776–780.
Gerrior, S., Bente, L., Hiza, H., 2004. Nutrient Content of the U.S. Food Supply, 1909–2000. United States Department of Agriculture.
Goran, M.I., Dumke, K., Bouret, S.G., Kayser, B., Walker, R.W., Blumberg, B., 2013. The obesogenic effect of high fructose exposure during early development. Nat. Rev. Endocrinol. 9 (8), 494–500.
Goran, M.I., Martin, A.M., Alderete, T.L., Fujiwara, H., Fields, D.A., 2017. Fructose in breast milk is positively associated with infant body composition at 6 months of age. Nutrients 9 (2), 146.
Goryakin, Y., Rocco, L., Suhrcke, M., 2017. The contribution of urbanization to non-communicable diseases: evidence from 173 countries from 1980 to 2008. Econ. Hum. Biol. 26, 151–163.
Grande, F., Anderson, J.T., Keys, A., 1965. Effect of carbohydrates of leguminous seeds, wheat and potatoes on serum cholesterol concentrations in man. J. Nutr. 86, 313–317.
Grande, F., 1967. Dietary carbohydrates and serum cholesterol. Am. J. Clin. Nutr. 20 (2), 176–184.
Hales, C.M., Carroll, M.D., Fryar, C.D., Ogden, C.L., 2017. Prevalence of obesity among adults and youth: United States, 2015–2016. NCHS Data Brief 288, 1–8.
Havel, P.J., 2005. Dietary fructose: implications for dysregulation of energy homeostasis and lipid/carbohydrate metabolism. Nutr. Rev. 63 (5), 133–157.
Hernandez, D.C., 2015. The impact of cumulative family risks on various levels of food insecurity. Soc. Sci. Res. 50, 292–302.
Hruby, A., Hu, F.B., 2015. The epidemiology of obesity: a big picture. Pharmacoeconomics 33 (7), 673–689.
Hruschka, D.J., 2012. Do economic constraints on food choice make people fat? A critical review of two hypotheses for the poverty-obesity paradox. Am. J. Hum. Biol. 24 (3), 277–285.
Hu, F.B., Malik, V.S., 2010. Sugar-sweetened beverages and risk of obesity and type 2 diabetes: epidemiologic evidence. Physiol. Behav. 100 (1), 47–54.
Hughes, C.C., Sherman, S.N., Whitaker, R.C., 2010. How low-income mothers with overweight preschool children make sense of obesity. Qual. Health Res. 20 (4), 465–478.
Johnson, R.J., Segal, M.S., Sautin, Y., Nakagawa, T., Feig, D.I., Kang, D.H., Gersch, M.S., Benner, S., Sánchez-Lozada, L., 2007. Potential role of sugar (fructose) in the epidemic of hypertension, obesity and the metabolic syndrome, diabetes, kidney disease, and cardiovascular disease. Am. J. Clin. Nutr. 86 (4), 899–906.
Johnson, M.L., Distelmaier, K., Lanza, I.R., Irving, B.A., Robinson, M.M., Konopka, A.R., Shulman, G.I., Nair, K.S., 2016. Mechanism by which caloric restriction improves insulin sensitivity in sedentary obese adults. Diabetes 65 (1), 74–84.
Jürgens, H., Haass, W., Castañeda, T.R., Schürmann, A., Koebnick, C., Dombrowski, F., Otto, B., Nawrocki, A.R., Scherer, P.E., Spranger, J., Ristow, M., Joost, H.G., Havel, P.J., Tschöp, M.H., 2005. Consuming fructose-sweetened beverages increases body adiposity in mice. Obes. Res. 13 (7), 1146–1156.
Kaufmann, N.A., Poznanski, R., Blondheim, S.H., Stein, Y., 1966. Changes in serum lipid levels of hyperlipemic patients following the feeding of starch, sucrose and glucose. Am. J. Clin. Nutr. 18 (4), 261–269.
Kowaleski-Jones, L., Brown, B.B., Fan, J.X., Hanson, H.A., Smith, K.R., Zick, C.D., 2017. The joint effects of family risk of obesity and neighborhood environment on obesity among women. Soc. Sci. Med. 195, 17–24.
Kranjac, A.W., Wagmiller, R.L., 2016. Decomposing trends in adult body mass index, obesity, and morbid obesity, 1971–2012. Soc. Sci. Med. 167, 37–44.
Kuo, P.T., Bassett, D.R., 1965. Dietary sugar in the production of hyperglyceridemia. Ann. Intern. Med. 62, 1199–1212.
Koo, Y.-C., Chang, J.-S., Chen, Y.C., 2018. Food claims and nutrition facts of commercial infant foods. PLoS ONE 13 (2), e0191982.
Life Sciences Research Office, 1976. Evaluation of the Health Aspects of Sucrose as a Food Ingredient. Center for Science in the Public Interest. https://www.industrydocumentslibrary.ucsf.edu/food/docs/qymk0226.
Ludwig, D.S., Peterson, K.E., Gortmaker, S.L., 2001. Relation between consumption of sugar-sweetened drinks and childhood obesity: a prospective, observational analysis. Lancet 357 (9255), 505–508.
Luo, S., Monterosso, J.R., Sarpelleh, K., Page, K.A., 2015. Differential effects of fructose versus glucose on brain and appetitive responses to food cues and decisions for food rewards. Proc. Natl. Acad. Sci. U. S. A. 112 (20), 6509–6514.
Lustig, R.H., Mulligan, K., Noworolski, S.M., Tai, V.W., Wen, M.J., Erkin-Cakmak, A., Gugliucci, A., Schwarz, J.-M., 2015. Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. Obesity 24 (2), 453–460.
Macdonald, I., Braithwaite, D.M., 1964. The influence of dietary carbohydrates on the lipid pattern in serum and in adipose tissue. Clin. Sci. 27, 23–30.
Malik, V.S., Schuize, M.B., Hu, F.B., 2006. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am. J. Clin. Nutr. 84 (2), 274–288.
Mata, J., Dallacker, M., Hertwig, R., 2017. Social nature of eating could explain missing link between food insecurity and childhood obesity. Behav. Brain Sci. 40, e122.
Mattes, R.D., Campbell, W.W., 2009. Effects of food form and timing of ingestion on appetite and energy intake in lean young adults and in young adults with obesity. J. Am. Diet Assoc. 109 (3), 430–437.
Mattson, M.P., Allison, D.B., Fontana, L., Harvie, M., Longo, V.D., Malaisse, W.J., Mosley, M., Notterpek, L., Ravussin, E., Scheer, F.A., Seyfried, T.N., Varady, K.A., Panda, S., 2014. Meal frequency and timing in health and disease. Proc. Natl. Acad. Sci. U. S. A. 111 (47), 16647–16653.
Moreno, J.P., Vaughan, E., Hernandez, D.C., Cameron, R.T., Foreyt, J.P., Johnston, C.A., 2016. The impact of acculturation level on weight status and weight outcomes in Hispanic children. J. Racial Ethn. Health Disparities 3 (4), 582–589.
Mullan, B., Ntoumanis, N., Thøgersen-Ntoumani, C., Lipp, O.V., 2017. It's a bit more complicated than that: a broader perspective on determinants of obesity. Behav. Brain Sci. 40, e124.
Muscogiuri, G., Balercia, G., Barrea, L., Cignarelli, A., Giorgino, F., Holst, J.J., Laudisio, D., Orio, F., Tirabassi, G., Colao, A., 2018. Gut: a key player in the pathogenesis of type 2 diabetes? Crit. Rev. Food Sci. Nutr. 58 (8), 1294–1309.
Nielsen, S.J., Popkin, B.M., 2004. Changes in beverage intake between 1977 and 2001. Am. J. Prev. Med. 27 (3), 205–210.
Nielsen, S.J., Siega-Riz, A.M., Popkin, B.M., 2002. Trends in energy intake in U.S. between 1977 and 1996: Similar shifts seen across age groups. Obes. Res. 10 (5), 370–378.
Ogden, C.L., Carroll, M.D., Lawman, H.G., Fryar, C.D., Kruszon-Moran, D., Kit, B.K., Flegal, K.M., 2014. Trends in obesity prevalence among children and adolescents in the United States, 1988–1994 through 2013–2014. JAMA 315 (21), 2292–2299.
Pan, L., Freedman, D.S., Park, S., Galuska, D.A., Potter, A., Blanck, H.M., 2019. Changes in obesity among U.S. children aged 2 through 4 years enrolled in WIC during 2010–2016. JAMA 321 (23), 2364–2366.
Pereira, R.M., Botezelli, J.D., da Cruz Rodrigues, K.C., Mekary, R.A., Cintra, D.E., Pauli, J.R., da Silva, A.S.R., Ropelle, E.R., de Moura, L.P., 2017. Fructose consumption in the development of obesity and the effects of different protocols of physical exercise on the hepatic metabolism. Nutrients 9 (4), 405.
Popkin, B.M., Hawkes, C., 2016. The sweetening of the global diet, particularly beverages: patterns, trends, and policy responses for diabetes prevention. Lancet Diab. Endocrinol. 4 (2), 174–186.
Rippe, J.M., 2013. The metabolic and endocrine response and health implications of consuming sugar-sweetened beverages: findings from recent randomized controlled trials. Adv. Nutr. 4 (6), 677–686.
Rosinger, A., Herrick, K., Gahche, J., Park, S., 2017. Sugar-Sweetened Beverage Consumption Among U.S. Youth 2011–2014. NCHS Data Brief. No 271. National Center for Health Statistics., Hyattsville, MD, pp. 2017.
Salmasi, L., Celidon, M., 2017. Investigating the poverty-obesity paradox in Europe. Econ. Hum. Biol. 26, 70–85.

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Shang, X.W., Liu, A.L., Zhang, Q., Hu, X.Q., Du, S.M., Ma, J., Xu, G.F., Li, Y., Guy, H.W., Du, L., Li, T.Y., Ma, G.S., 2012. Report on childhood obesity in China (9): sugar-sweetened beverages consumption and obesity. Biomed. Environ. Sci. 25 (2), 125–132.
Song, W.O., Wang, Y., Chung, C.E., Song, B., Lee, W., Chun, O.K., 2012. Is obesity development associated with dietary sugar intake in the U.S.? Nutrition 28 (11–12), 1137–1141.
Shrewsbury, V., Wardle, J., 2012. Socioeconomic status and adiposity in childhood: a systematic review of cross-sectional studies 1990–2005. Obesity 16 (2), 275–284.
Smith, T.G., 2017. Household-level financial uncertainty could be the primary driver of the global obesity epidemic. Behav. Brain Sci. 40, e128.
Smits, S.A., Leach, J., Sonnenburg, E.D., Gonzalez, C.G., Lichtman, J.S., Reid, G., Knight, R., Manjurano, A., Changalucha, J., Elias, J.E., Gloria, M.G., Dominguez-Bello, Sonnenburg, J.L., 2017. Seasonal cycling in the gut microbiome of the Hadza hunter-gatherers of Tanzania. Science 357 (6353), 802–806.
Sobal, J., Stunkard, A.J., 1989. Socioeconomic status and obesity: a review of the literature. Psychol. Bull. 105 (2), 260–275.
Soenen, S., Westerterp-Plantenga, M.S., 2007. No differences in satiety or energy intake after high-fructose corn syrup, sucrose, or milk preloads. Am. J. Clin. Nutr. 86 (6), 1586–1594.
Stanhope, K.L., Schwarz, J.M., Keim, N.L., Griffen, S.C., Bremer, A.A., Graham, J.L., Hatcher, B., Cox, C.L., Dyachenko, A., Zhang, W., McGahan, J.P., Seibert, A., Krauss, R.M., Chiu, S., Schaefer, E.J., Ai, M., Otokozawa, S., Nakajima, K., Nakano, T., Beysen, C., Hellerstein, M.K., Berglund, L., Havel, P.J., 2009. Consuming fructose-sweetened, not glucose-sweetened, beverages increases visceral adiposity and lipids and decreases insulin sensitivity in overweight/obese humans. J. Clin. Invest. 119 (5), 1322–1334.
Tafreschi, D., 2015. The income body weight gradients in the developing economy of China. Econ. Hum. Biol. 16, 115–134.
Teff, K.L., Elliott, S.S., Tschöp, M., Kieffer, T.J., Rader, D., Heiman, M., Townsend, R.R., Keim, N.L., D'Alessio, D., Havel, P.J., 2004. Dietary fructose reduces circulating insulin and leptin, attenuates postprandial suppression of ghrelin, and increases triglycerides in women. J. Clin. Endocrinol. Metab. 89 (6), 2963–2972.
U.S. Department of Agriculture, Economic Research Service, 2019. Estimated Number of Per Capita Calories Consumed Daily, by Calendar Year: Refined Cane and Beet Sugar (Table 51). High Fructose Corn Syrup (Table 52), Other Sweeteners (Table 53). Sugar and Sweeteners Yearbook 2019.
van Buul, V.J., Tappy, L., Brouns, F.J.P.H., 2014. Misconceptions about fructose-containing sugars and their role in the obesity epidemic. Nutr. Res. Rev. 27 (1), 119–130.
Welsh, J.A., Sharma, A.J., Grellinger, L., Vos, M.B., 2011. Consumption of added sugars is decreasing in the United States. Am. J. Clin. Nutr. 94 (3), 726–734.
Villar, J.G., Quintana-Domeque, C., 2009. Income and body mass index in Europe. Econ. Hum. Biol. 7, 73–83.
Wang, Y., Beydoun, M.A., 2002. The obesity epidemic in the United States: gender, age, socioeconomic, racial/ethnic, and geographic characteristics. Epidemiol. Rev. 29, 6–28.
Wang, Y.C., Bleich, S.N., Gortmaker, S.L., 2008. Pediatrics 121 (6), e1604–1614.
World Health Organization, 2018. Obesity and Overweight, Fact Sheet 311. World Health Organization, Geneva.
Wisconsin Health Atlas, 2019. University of Wisconsin School of Medicine and Public Health. . https://www.wihealthatlas.org/obesity/age.

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