Effect of reducing ultraprocessed food consumption on obesity among US children and adolescents aged 7–18 years: evidence from a simulation model

Anne Scott Livingston,[1] Frederick Cudhea,[1] Lu Wang,[1] Euridice Martinez Steele,[2,3] Mengxi Du ![ORCID],[1] Y Claire Wang,[4] Jennifer Pomeranz,[5] Dariush Mozaffarian,[1] Fang Fang Zhang,[1,6] on behalf of the Food-PRICE project

ABSTRACT

Background Children and adolescents in the USA consume large amounts of daily calories from ultraprocessed foods (UPFs). Recent evidence links UPF consumption to increased body fat in youth. We aimed to estimate the potential impact of reducing UPF consumption on childhood obesity rate in the USA.
Methods We developed a microsimulation model to project the effect of reducing UPF consumption in children's diet on reducing the prevalence of overweight or obesity among US youth. The model incorporated nationally representative data on body mass index (BMI) percentile and dietary intake of 5804 children and adolescents aged 7–18 years from the National Health and Nutrition Examination Survey 2011–2016, and the effect of reducing UPF consumption on calorie intake from a recent randomised controlled trial. Uncertainties of model inputs were incorporated using probabilistic sensitivity analysis with 1000 simulations.
Results Reducing UPFs in children's diet was estimated to result in a median of −2.09 kg/m² (95% uncertainty interval −3.21 to −0.80) reduction in BMI among children and adolescents aged 7–18 years. The median prevalence of overweight (BMI percentile ≥85th) and obesity (BMI percentile ≥95th percentile) was reduced from 37.0% (35.9%, 38.1%) to 20.9% (15.1%, 29.9%) and from 20.1% (19.2%, 21.0%) to 11.0% (7.86%, 15.8%), respectively. Larger BMI and weight reductions were seen among boys than girls, adolescents than children, non-Hispanic black and Hispanic youth than non-Hispanic white youth, and those with lower levels of parental education and family income.
Conclusions Reducing UPF consumption in children's diet has the potential to substantially reduce childhood obesity rate among children and adolescents in the USA.

What this paper adds

What is already known on this topic

► Children's diets in the USA have shifted to contain high amounts of ultraprocessed foods (UPF), with about two-thirds of daily calorie consumed from UPFs. High-UPF diets have been shown to contribute to high levels of body fat in children.

What this study adds

► Using nationally representative data, we estimated that reducing UPFs in children's diets could reduce the prevalence of overweight from 37% to 21% and obesity from 20% to 11% among US children and adolescents aged 7–18 years.

INTRODUCTION

The obesity rate among children and adolescents (hereafter refered to as children) in the USA has more than tripled since the 1970s.[1] Meanwhile, Americans' diets have shifted to contain a significant amount of foods that are highly processed due to advances in food processing and increased availability of ultraprocessed foods (UPFs).[2] UPFs are foods that have gone through several levels of industrial processing and have few to no whole foods present. UPFs often contain cosmetic additives such as flavour enhancers, colours, emulsifiers, sweeteners and bulking and carbonating agents to make foods highly palatable and ready to eat with long shelf-life.[3] While convenient, UPFs are usually energy dense, high in added sugar and sodium, low in protein, fibre and micronutrients,[4] and offering a suboptimal nutrition profile. A recent randomised controlled cross-over trial provided evidence that consuming a high-UPF diet led to increased ad libitum calorie intake and weight gain among young adults. Over a 2-week period, 20 adults following a high-UPF diet consumed approximately 500 more kcal/day than those on a non-UPF diet and gained approximately 0.9 kg whereas those on the non-UPF diet lost 0.9 kg.[5] Prospective cohort studies in children also suggest that high UPF consumption contributes to high levels of body fat.[6–8] Children and adolescents in the USA consume more than 60% of daily

calories from UPFs.[9] Reducing UPFs in children's diet can have a large impact on reducing childhood obesity in the USA.

In this study, we estimated the effect of reducing UPFs in children's diet on reducing total energy intake and the prevalence of overweight and obesity among US children aged 7–18 years, and further evaluated whether the estimated effects differed among population subgroups by age, sex, race/ethnicity, parental education and family income.

METHODS
Study overview
We developed a microsimulation model to estimate the impact of reducing UPF consumption in children's diet on their body mass index (BMI). The model incorporated nationally representative data on demographic characteristics, weight, height and dietary intakes of 5804 children aged 7–18 years from the National Health and Nutrition Examination Survey (NHANES) 2011–2016.[10] Based on the effect size of reducing UPF on calorie reduction reported from a recent randomised controlled trial (RCT),[5] and the children's weight reduction prediction model developed by Hall et al,[11] we projected children's weight reduction as a result of reducing UPFs in their diet, accounting for potential changes in energy expenditure and appetite. We then estimated change in children's BMI and prevalence of overweight and obesity by comparing the current and postchange BMI distribution and overweight and obesity prevalence among US children aged 7–18 years.

Simulated population
The model was populated with individuals aged 7–18 years who participated in the three recent NHANES cycles (2011–2012, 2013–2014 and 2015–2016) and provided at least one valid 24-hour recall. Day 1 diet recall or the average of 2-day diet recall whenever available was used to estimate daily calorie intake and energy contribution of UPFs. Those with daily total calorie intake <500 or >5000 kcal/day were excluded, resulting in a total of 5804 children available for the simulation.

UPF consumption
UPF consumption was assessed using dietary intake data collected from 24-hour dietary recalls based on the validated US Department of Agriculture Automated Multiple-Pass Method. Foods were classified into four groups (unprocessed/minimally processed food, processed culinary ingredients, processed foods and UPFs) according to the NOVA food classification (online supplemental text).[12] Briefly, UPFs were defined as ready-to-eat, ready-to-drink or ready-to-heat industrial formulations that are made predominantly or entirely from industrial substances extracted from foods such as oil, fats, sugar, starch and protein and contain little or no whole food and often contain cosmetic additives. Examples of UPFs

include breakfast cereals, biscuits, quick breads, frozen pizza, ready-to-eat or ready-to-heat meals, sweet snacks and sweets, fast-food or reconstituted meat, poultry or fish and sugar-sweetened beverages (SSB). A detailed definition and example food items of UPFs are shown in online supplemental table 1. For mixed dishes judged to be home-made, for example, stew or cake made from home recipe, we used underlying ingredients (SR Codes) to ensure a more accurate classification. Details of the classification process have been published previously.[13 14] The percent of calories (%E) from UPFs was calculated as the percent of calories consumed from UPFs over the total daily calories.

Weight status
Children's weight status was determined using BMI calculated from measured height and weight. Children's age-specific and sex-specific BMI percentile was determined based on the 2000 growth chart using the algorithm provided by the Centers for Disease Control and Prevention.[15] We defined children's weight categories according to the recommended cut-points as follows: normal weight if BMI <85th percentile; overweight if BMI ≥85th percentile; and obese if BMI ≥95th percentile.

Effect of reducing UPF consumption on reducing daily calorie intake
The effect of reducing UPF consumption on total calorie intake was estimated based on a recent RCT conducted among 20 young adults (mean age=31.2 years, mean BMI=27.0 kg/m^2) who were assigned to either a high-UPF diet (81.3% calories from UPFs) or non-UPF diet (0% calories from UPF) for 2 weeks.[5] The two diets were matched for presented calories, energy density, macronutrients, sugar, sodium and fibre. The RCT reported that the ad libitum calorie intake was 509 kcal/day more in participants assigned to the high-UPF diet compared with those assigned to the non-UPF diet (mean calorie intake: 2979 vs 2470 kcal/day), corresponding to a 17.1% increase in total daily calories. Based on this finding, we estimated that reducing UPFs in a child's diet to zero would result in a reduction in total dairy calories proportional to the percent of UPFs in the diet using the following algorithm: total energy intake*17.1%*proportion of UPFs in the diet/81.3%. For example, for a 10-year-old boy with total calories being 2000 kcal/day and 60% energy intake from UPFs, the reduction in his total daily calories is estimated to be 252 kcal/day (=2000*$\frac{60\%}{81.3\%}$*17.1%).

Effect of calorie reduction on children's weight
We estimated the amount of weight reduction (in kilogram) from calorie reduction (in kilocalorie/day) using the weight reduction model by Hall et al.[11] This model estimates the daily calorie reduction required for children aged 7–18 years to reduce 1 kg body weight: 68–2.5 × age for boys and 62–2.2 × age for girls. Thus, the required daily calorie reduction to achieve 1 kg weight reduction for a 10-year-old boy is estimated to be 43 kcal. If he

consumes a daily calorie of 2000 kcal/day with 60% of the daily calories from UPFs, and under our model assumption that zeroing total UPF consumption from his diet leads to 252 kcal reduction in his daily calorie consumption, his estimated weight reduction would be 5.86 kg (= $\frac{252 \ \frac{kcal}{day}}{43 \ \frac{kcal}{day} \ per \ kg}$). Subsequent reduction in BMI was estimated based on weight reduction.

Statistical analysis

Among all eligible US children aged 7–18 years in NHANES 2011–2016, we simulated the effect of reducing UPFs in children's diet on BMI distribution. The BMI distribution and prevalence of overweight and obesity at baseline and postchange were calculated by adjusting for NHANES survey weights to account for the complex sampling design and non-responses to ensure national representativeness. To incorporate uncertainties in effect size estimates,[5] probabilistic sensitivity analysis was used with 1000 Monte Carlo simulations. From the 1000 means, we report the median and 95% uncertainty intervals (UIs) from the resulting distributions. We further estimated the effects among US children subgroups by age (7–11 and 12–18 years), sex (boys and girls), race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic and other), parental education (less than high school, high school or General Educational Development, some college or college graduate) and family income (family income to poverty ratio (FIPR) <1.3, 1.3–3 and ≥3).

RESULTS

The mean (±SE) age of the US children aged 7–18 years was 12.7 (±0.08) years. About 51% were boys, 55% were non-Hispanic white children, 41% had parental education at high school or less and 36% were from low-income families (FIPR <1.3) (table 1).

US children aged 7–18 years consumed an average of 66.4% (±0.41%) daily calories from UPFs. About 37.0% of the children were overweight and 20.1% were obese.

Reducing UPFs in children's diet was estimated to result in a decrease in total daily calorie intake of 276 (95% UI −131 to −404) kcal/day, from 1967 (95% CI 1934 to 1999) kcal/day to 1690 (95% UI 1563 to 1836) kcal/day (online supplemental table 2). Subsequently, the estimated mean reduction in children's weight was 5.12 (95% CI −7.87 to −1.97) kg, from 54.4 (95% CI 53.9 to 54.9) kg to 49.3 (95% CI 46.4 to 52.6) kg; the mean BMI reduction was 2.09 (95% CI −3.21 to −0.80) kg/m², from 22.0 (95% CI 21.8 to 22.1) kg/m² to 19.9 (95% CI 18.7 to 21.2) kg/m² (table 2). These led to a reduction in the prevalence of overweight in absolute per cent points by 16.1% (95% CI −22.4% to −6.85%), from the current prevalence of 37.0% (95% CI 35.9% to 38.1%) to 20.9% (95% CI 15.1% to 29.9%), and a reduction in the prevalence of obesity absolute per cent points by 9.11% (95% CI −12.8% to −4.0%), from the current prevalence of 20.1% (95% CI

Table 1 Sociodemographic characteristics of US children aged 7–18 years, NHANES 2011–2016

	n (Weighted %)*
Age (years)	
7–11	2628 (38.3)
12–18	3176 (61.7)
Sex	
Boys	2943 (50.5)
Girls	2861 (49.5)
Race/ethnicity	
Non-Hispanic white	1529 (54.8)
Non-Hispanic black	1467 (13.6)
Hispanic†	1908 (22.8)
Other	900 (8.8)
Parental education‡	
Less than high school	1453 (20.5)
High school or GED	1243 (20.9)
Some college	1669 (30.5)
College graduate	1284 (28.1)
Family income to poverty ratio§	
<1.3	2791 (35.9)
1.3–3	1613 (29.6)
≥3	1400 (34.5)

*Percentages were adjusted for NHANES survey weights.
†Hispanic includes respondents self-identified as 'American Mexican' or as 'Hispanic' ethnicity. 'Other' includes race/ethnicity other than non-Hispanic white, non-Hispanic black and Hispanic, including multiracial.
‡Parental education level represents the educational level of the household reference person.
§Ratio of family income to poverty level represents the ratio of family income to the federal poverty threshold. A higher ratio indicates a higher level of income.
GED, General Educational Development; NHANES, National Health and Nutrition Examination Survey.

19.2% to 21.0%) to 11.0% (95% CI 7.86% to 15.8%) (table 3).

By population subgroups, boys were predicted to have higher levels of reduction in weight, BMI and prevalence of overweight and obesity than girls. Adolescents aged 12–18 years were predicted to have a higher level of reduction in weight and BMI than children aged 7–11 years, whereas children aged 7–11 years had a higher level of reduction in the prevalence of overweight and obesity than adolescents aged 12–18 years. Non-Hispanic black and Hispanic children were predicted to have higher levels of reduction in weight and BMI than non-Hispanic white children. The reduction in overweight and obesity prevalence was higher for Hispanic children than non-Hispanic children (non-Hispanic white and black). Children with lower levels of parental education (high school or less than high school) or those from low-income families (FIPR <3) were predicted to have

Table 2 Estimated reduction in BMI and weight among US children aged 7–18 years after replacing all UPFs with non-UPFs in their diet

	BMI (kg/m²), mean (95% CI)			Weight (kg), mean (95% CI)*		
	Current	Postchange	Difference	Current	Postchange	Difference
All US children (7–18 years)	22.0 (21.8 to 22.1)	19.9 (18.7 to 21.2)	−2.09 (−3.21 to 0.80)	54.4 (53.9 to 54.9)	49.3 (46.4 to 52.6)	−5.12 (−7.87 to −1.97)
Sex						
Boys	21.7 (21.5 to 21.9)	19.5 (18.3 to 20.9)	−2.17 (−3.34 to 0.83)	55.8 (55.0 to 56.5)	50.3 (47.0 to 54.0)	−5.52 (−8.50 to −2.12)
Girls	22.2 (22.0 to 22.4)	20.2 (19.1 to 21.5)	−2.01 (−3.09 to 0.77)	53.0 (52.4 to 53.6)	48.3 (45.5 to 51.4)	−4.71 (−7.25 to −1.81)
Age (years)						
Children (7–11)	18.9 (18.8 to 19.1)	16.9 (15.9 to 18.2)	−2.00 (−3.07 to 0.76)	36.9 (36.5 to 37.3)	33.0 (30.8 to 35.5)	−3.86 (−5.94 to −1.48)
Adolescents (12–18)	23.8 (23.6 to 24.0)	21.7 (20.5 to 23.1)	−2.15 (−3.31 to 0.82)	65.3 (64.6 to 65.9)	59.4 (56.0 to 63.2)	−5.90 (−9.08 to −2.26)
Race/ethnicity						
Non-Hispanic white	21.5 (21.3 to 21.7)	19.4 (18.3 to 20.8)	−2.07 (−3.19 to 0.79)	53.7 (53.0 to 54.4)	48.6 (45.6 to 52.1)	−5.10 (−7.86 to −1.96)
Non-Hispanic black	22.8 (22.6 to 23.1)	20.6 (19.4 to 22.1)	−2.20 (−3.39 to 0.84)	57.7 (56.9 to 58.5)	52.3 (48.9 to 56.1)	−5.48 (−8.44 to −2.10)
Hispanic†	22.7 (22.5 to 22.8)	20.5 (19.3 to 21.9)	−2.16 (−3.32 to 0.83)	54.3 (53.6 to 55.0)	49.2 (46.2 to 52.7)	−5.12 (−7.89 to −1.96)
Other	21.5 (21.2 to 21.9)	19.6 (18.4 to 21.0)	−1.89 (−2.93 to 0.72)	53.6 (52.2 to 55.1)	48.9 (45.4 to 52.9)	−4.67 (−7.27 to −1.77)
Parental education‡						
Less than high school	22.5 (22.2 to 22.8)	20.4 (19.1 to 21.8)	−2.15 (−3.32 to 0.82)	54.7 (53.7 to 55.6)	49.5 (46.3 to 53.2)	−5.15 (−7.96 to −1.97)
High school or GED	22.6 (22.4 to 22.9)	20.5 (19.2 to 21.9)	−2.20 (−3.40 to 0.84)	56.3 (55.5 to 57.1)	50.9 (47.7 to 54.6)	−5.38 (−8.32 to −2.06)
Some college	22.4 (22.1 to 22.6)	20.2 (19.0 to 21.6)	−2.15 (−3.30 to 0.82)	55.9 (54.9 to 56.8)	50.6 (47.2 to 54.3)	−5.32 (−8.19 to −2.04)
College graduate	20.6 (20.4 to 20.8)	18.7 (17.6 to 20.0)	−1.90 (−2.93 to 0.72)	51.2 (50.4 to 52.0)	46.6 (43.7 to 49.8)	−4.65 (−7.20 to −1.77)
Family income§						
<1.3	22.4 (22.3 to 22.6)	20.3 (19.1 to 21.7)	−2.16 (−3.32 to 0.83)	54.5 (53.9 to 55.1)	49.3 (46.4 to 52.7)	−5.19 (−7.98 to −1.99)
1.3–3	22.3 (22.0 to 22.5)	20.1 (18.9 to 21.6)	−2.15 (−3.31 to 0.83)	55.1 (54.3 to 56.0)	49.9 (46.6 to 53.6)	−5.27 (−8.10 to −2.02)
≥3	21.2 (20.9 to 21.4)	19.2 (18.1 to 20.5)	−1.97 (−3.04 to 0.75)	53.6 (52.7 to 54.5)	48.7 (45.6 to 52.2)	−4.93 (−7.61 to −1.88)

*Mean and SE of current BMI and weight were adjusted for survey weights; mean of the BMI and weight after replacing UPFs with non-UPFs (postchange) and differences between current and postchange were estimated as the median of the simulated distribution of 1000 means; mean of the BMI and weight after replacing UPFs with non-UPFs (postchange) and differences between current and postchange were estimated as the median of the simulated distribution of 1000 means; upper and lower bounds were estimated as corresponding to the 2.5th and 97.5th percentiles of the simulated distribution of the 1000 means.

†Hispanic includes respondents self-identified as 'American Mexican' or as 'Hispanic' ethnicity. 'Other' includes race/ethnicity other than non-Hispanic white, non-Hispanic black and Hispanic, including multiracial.

‡Parental education represents the educational level of the household reference person.

§Family income corresponds to the ratio of family income to the federal poverty threshold. A higher ratio indicates a higher level of income.

BMI, body mass index; GED, General Educational Development; UPF, ultraprocessed food.

Table 3 Estimated reduction in prevalence of overweight or obesity among US children aged 7–18 years after replacing all UPFs with non-UPFs in their diet

	Prevalence (%) of overweight or obesity (BMI ≥85th percentile)			Prevalence (%) of obesity (BMI ≥95th percentile)		
	Mean (95% CI)			Mean (95% CI)		
	Current*	Postchange*	Difference*	Current*	Postchange*	Difference*
All US children (7–18 years)	37.0 (35.9 to 38.1)	20.9 (15.1 to 29.9)	−16.1 (−22.4 to 6.85)	20.1 (19.2 to 21.0)	11.0 (7.86 to 15.8)	−9.11 (−12.8 to −4.00)
Sex						
Boys	37.4 (36.1 to 38.7)	20.0 (13.9 to 29.5)	−17.4 (−24.1 to 7.52)	20.6 (19.4 to 21.9)	10.9 (7.65 to 16.2)	−9.71 (−13.8 to −4.00)
Girls	36.7 (35.2 to 38.1)	21.8 (16.0 to 30.5)	−14.8 (−21.3 to 5.68)	19.6 (18.6 to 20.6)	11.1 (7.71 to 15.7)	−8.43 (−12.4 to −3.43)
Age (years)						
Children (7–11)	35.7 (34.2 to 37.2)	17.6 (12.0 to 27.8)	−18.0 (−24.5 to 7.42)	19.1 (17.9 to 20.3)	9.16 (5.86 to 14.7)	−9.90 (−14.1 to −3.92)
Adolescents (12–18)	37.9 (36.4 to 39.3)	22.9 (16.8 to 31.4)	−14.9 (−21.7 to 6.00)	20.8 (19.6 to 21.9)	12.1 (8.92 to 16.7)	−8.57 (−12.5 to −3.55)
Race/ethnicity						
Non-Hispanic white	33.1 (31.8 to 34.5)	17.7 (11.7 to 26.6)	−15.4 (−22.0 to 6.16)	17.1 (16.0 to 18.2)	8.73 (5.78 to 13.3)	−8.31 (−12.1 to −3.33)
Non-Hispanic black	40.8 (39.1 to 42.5)	24.7 (18.9 to 33.7)	−15.9 (−22.8 to 6.49)	23.9 (22.5 to 25.4)	15.2 (11.1 to 20.7)	−8.72 (−13.8 to −2.64)
Hispanic†	45.3 (43.9 to 46.6)	27.1 (20.4 to 37.1)	−18.2 (−25.4 to 7.80)	25.5 (24.2 to 26.7)	14.8 (10.9 to 20.7)	−10.5 (−15.3 to −4.25)
Other	34.2 (31.8 to 36.6)	18.8 (12.2 to 29.6)	−15.2 (−23.6 to 3.70)	19.3 (16.9 to 21.6)	8.57 (4.50 to 14.8)	−10.6 (−16.9 to −3.11)
Parental education‡						
Less than high school	41.9 (40.2 to 43.5)	24.7 (18.0 to 34.7)	−17.1 (−24.7 to 6.56)	23.7 (22.0 to 25.4)	13.6 (9.17 to 19.6)	−10.1 (−15.7 to −3.25)
High school or GED	42.9 (41.3 to 44.6)	24.9 (18.1 to 35.9)	−17.9 (−25.7 to 6.46)	24.4 (22.9 to 25.8)	13.7 (9.50 to 19.7)	−10.6 (−15.7 to −4.02)
Some college	37.8 (35.8 to 39.7)	23.1 (16.8 to 31.5)	−14.7 (−22.1 to 5.56)	22.4 (20.8 to 23.9)	13.0 (9.23 to 18.3)	−9.35 (−14.3 to −3.32)
College graduate	28.5 (26.7 to 30.3)	13.3 (7.92 to 22.3)	−15.1 (−21.8 to 5.62)	12.7 (11.5 to 13.9)	5.40 (3.14 to 9.43)	−7.21 (−10.6 to −2.65)
Family income§						
<1.3	42.0 (40.8 to 43.2)	25.0 (18.6 to 34.1)	−17.0 (−23.9 to 7.57)	23.4 (22.3 to 24.4)	14.0 (9.87 to 18.8)	−9.43 (−14.0 to −4.10)
1.3–3	39.3 (37.7 to 40.9)	22.2 (15.9 to 32.4)	−17.0 (−24.2 to 6.47)	22.2 (20.5 to 23.9)	12.1 (8.02 to 18.0)	−10.1 (−15.4 to −3.40)
≥3	29.9 (28.4 to 31.5)	15.6 (9.95 to 24.0)	−14.3 (−20.8 to 5.35)	15.0 (13.8 to 16.2)	7.09 (4.65 to 11.6)	−7.78 (−11.3 to −2.83)

*Prevalence of overweight and obesity was adjusted for survey weights.
†Hispanic includes respondents self-identified as 'American Mexican' or as 'Hispanic' ethnicity. 'Other' includes race/ethnicity other than non-Hispanic white, non-Hispanic black and Hispanic, including multiracial.
‡Parental education represents the educational level of the household reference person.
§Family income corresponds to the ratio of family income to the federal poverty threshold. A higher ratio indicates a higher level of income.
BMI, body mass index; GED, General Educational Development; UPF, ultraprocessed food.

higher levels of reduction in weight, BMI and prevalence of overweight and obesity than children with high levels of parental education (college graduates) or those from higher income families (FIPR ≥3) (table 3 and online supplemental eFigure 1).

DISCUSSION

US children and adolescents aged 7–18 years consume two-thirds of daily calories from UPFs. Our results from a microsimulation study suggested that reducing UPFs in children's diet could reduce the prevalence of overweight and obesity in per cent points by 16% and 9%, respectively, among this population. US food and dietary policy generally focuses on nutrients to avoid or food groups to consume but rarely identifies UPF as a specific target for intervention.[16 17] Future public health initiatives should consider the reduction of UPFs a primary policy goal to prevent child and adolescent obesity and obesity-related chronic diseases.

The estimated effect (-2.09 kg/m^2) was greater than that of several other childhood obesity prevention strategies, such as a penny-per-ounce SSB excise tax (-0.16 kg/m^2),[18] setting nutrition standards for school meals (-0.83 kg/m^2) and other foods and beverages sold in schools (-0.24 kg/m^2),[19] eliminating the tax deductibility of costs for advertising unhealthy foods and beverages to children (-0.34 kg/m^2)[19] and requiring school-based physical education (-0.02 kg/m^2).[20] As high UPF consumption in childhood may persist into adulthood,[21–23] reducing UPF consumption in children's diet could also have great impacts on reducing obesity and obesity-associated chronic diseases in adults.[6 24–28]

Recent prospective cohort studies provide compelling evidence that high UPF consumption is associated with increases in percentage of body fat,[7 29] waist circumference,[8] and levels of total cholesterol, low-density lipoprotein (LDL) and triglycerides among children.[30 31] For example, a longitudinal study among 307 children aged 4 years in Brazil found that every 10% increase in calories consumed from UPFs was associated with a 0.7 cm increase in waist circumference over 4 years.[8] From the same cohort of children, a high UPF consumption was also associated with increases in levels of total cholesterol and LDL cholesterol over 4 years.[30] Similar findings were reported by another prospective cohort study in Brazil that a high UPF consumption was associated with elevated levels of cholesterol and triglycerides among children who were followed from ages 3 to 6 years.[31] Our results corroborated these findings, suggesting that replacing UPFs with unprocessed or minimally processed foods in children's diet could have a substantial impact on reducing obesity rates among US children and adolescents.

These findings call for intervention strategies to reduce UPF consumption among US children. Policy options may include front-of-package labelling, taxes and school nutrition standards focused specifically on UPFs. Front-of-package labels such as the traffic lights have been shown to improve the nutrition quality of snack food choices, both when children were choosing snacks for themselves and when parents were selecting snacks for their children.[32 33] SSB taxes decrease sale of these drinks and increase the consumption of healthier alternatives such as water.[34] Expanding taxes to other unhealthful UPFs could also reduce the consumption of those UPFs.[35 36] Setting standards for school food environments is also important in shaping the quality of children's diet. Previous studies showed that the quality for school meals significantly improved following the updated nutrition standards mandated by the Healthy, Hunger-Free Kids Act of 2010.[37–39] Of note, Chile's unhealthy food marketing regulation significantly reduced children's exposure to food advertising of unhealthy foods and beverages, but the availability of this option in the USA is unclear from a legal perspective.[40] Implementation of similar policy strategies that specifically focus on UPF consumption has great potential to reduce childhood obesity in the USA.

The current study estimated that children from low-income families would have a greater reduction in overweight and obesity rates if they reduced consumption of UPFs than those from higher income families. This reflects a higher baseline level of consumption of UPFs and a higher rate of overweight and obesity among children from low-income families. Similarly, reducing UPF consumption would have a larger impact on reducing obesity in children with a lower level of parental education than those with a higher level of parental education. Low socioeconomic status has been well recognised as a risk factor for childhood obesity in the USA.[41 42] These findings highlight the need for targeted policy strategies to simultaneously increase the availability, affordability and accessibility of unprocessed healthful foods to this group; for example, by providing financial incentives for unprocessed healthful foods through the Supplemental Nutrition Assistance Program and the Women, Infants and Children Nutrition Program.

The strengths of our study included the use of nationally representative data on dietary intake, BMI and demographics, increasing generalisability to US children. Children's weight was measured according to standard protocols by trained personnel, reducing measurement errors. Uncertainty in dietary intakes, child BMI and effect size estimates was accounted for by using 1000 simulations. However, this study also has limitations. First, we estimated UPF consumption of US children using self-reported 24-hour dietary recall data from NHANES, which is subject to measurement error. However, NHANES incorporated one or two standardised, 24-hour diet recalls per person which were energy adjusted and averaged whenever possible, reducing measurement error. Second, NHANES collects some information indicative of food processing (eg, place of meals, product brands), but these data are not consistently determined for all food items, which could lead to underestimation or overestimation in UPF intakes. Third, we estimated the effect of reducing UPFs in children's calorie intake

based on findings from an RCT conducted among young adults, as no direct evidence is available from children. To account for lower levels of daily calories consumed by US children than adults, we estimated the per cent of calorie intake reduction from UPFs instead of the absolute change as the effect size, which avoids overestimation. However, the observed difference in ad libitum daily calorie consumption among participants of the RCT was based on two diets (high UPF vs non UPF) matched by calorie and nutrient content. Because the energy density of UPFs is usually higher than that of non-UPFs, the actual calorie reduction in response to reducing UPF consumption in children's diet and its subsequent impact on reducing childhood obesity could be higher than what estimated in this study. Fourth, we did not estimate the effect among children younger than 7 years due to the lack of reliable estimation of weight reduction per calorie reduction in younger children. In addition, we used the weight reduction model derived for children aged 7–18 years as a whole, which did not incorporate potentially heterogeneous estimates among population subgroups of US children.[11] Fifth, our study estimated the impact of reducing UPF consumption from children's diet on obesity prevention under counterfactual scenarios; the actual impact of prevention strategies to reduce UPF consumption on reducing childhood obesity depends on how effectively these strategies reduce UPF consumption in US children.

CONCLUSIONS

Reducing UPFs in children's diet has the potential to reduce the obesity rate to a great extent among US children and adolescents aged 7–18 years, especially among boys, children aged 7–11 years, Hispanic children and adolescents and those with low parental education and family income. Future public health initiatives should focus on reducing UPF consumption among US children and adolescents to prevent obesity and obesity-related chronic diseases.

Author affiliations
[1]Friedman School of Nutrition Science and Policy, Tufts University, Boston, Massachusetts, USA
[2]Department of Nutrition, School of Public Health, University of São Paulo, São Paulo, Brazil
[3]Center for Epidemiological Studies in Health and Nutrition, University of São Paulo, São Paulo, Brazil
[4]Department of Health Policy and Management, Columbia University Mailman School of Public Health, New York, New York, USA
[5]Department of Public Health Policy and Management, School of Global Public Health, New York University, New York, New York, USA
[6]Tufts Institute for Global Obesity Research, Boston, Massachusetts, USA

ORCID iD
Mengxi Du http://orcid.org/0000-0002-6406-7250

REFERENCES

1. Ng M, Fleming T, Robinson M, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980-2013: a systematic analysis for the global burden of disease study 2013. *Lancet* 2014;384:766–81.
2. Baraldi LG, Martinez Steele E, Canella DS, et al. Consumption of ultra-processed foods and associated sociodemographic factors in the USA between 2007 and 2012: evidence from a nationally representative cross-sectional study. *BMJ Open* 2018;8:e020574.
3. Monteiro CA, Cannon G, Levy RB, et al. Ultra-processed foods: what they are and how to identify them. *Public Health Nutr* 2019;22:936–41.
4. Moodie R, Stuckler D, Monteiro C, et al. Profits and pandemics: prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. *The Lancet* 2013;381:670–9.
5. Hall KD, Ayuketah A, Brychta R, et al. Ultra-Processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of AD libitum food intake. *Cell Metab* 2019;30:67–77.
6. Louzada MLdaC, Baraldi LG, Steele EM, et al. Consumption of ultra-processed foods and obesity in Brazilian adolescents and adults. *Prev Med* 2015;81:9–15.
7. Costa CS, Del-Ponte B, Assunção MCF, et al. Consumption of ultra-processed foods and body fat during childhood and adolescence: a systematic review. *Public Health Nutr* 2018;21:148–59.
8. Costa CS, Rauber F, Leffa PS, et al. Ultra-processed food consumption and its effects on anthropometric and glucose profile: a longitudinal study during childhood. *Nutr Metab Cardiovasc Dis* 2019;29:177–84.

9 Neri D, Martinez-Steele E, Monteiro CA, *et al*. Consumption of ultra-processed foods and its association with added sugar content in the diets of US children, NHANES 2009-2014. *Pediatr Obes* 2019;14:e12563.

10 CDC. *NHANES questionnaires, datasets, and related documentation*, 2020.

11 Hall KD, Butte NF, Swinburn BA, *et al*. Dynamics of childhood growth and obesity: development and validation of a quantitative mathematical model. *Lancet Diabetes Endocrinol* 2013;1:97–105.

12 Monteiro CA, Cannon G, Moubarac J-C, *et al*. The un decade of nutrition, the nova food classification and the trouble with ultra-processing. *Public Health Nutr* 2018;21:5–17.

13 Martínez Steele E, Baraldi LG, Louzada MLdaC, *et al*. Ultra-processed foods and added sugars in the US diet: evidence from a nationally representative cross-sectional study. *BMJ Open* 2016;6:e009892.

14 Juul F, Martinez-Steele E, Parekh N, *et al*. Ultra-processed food consumption and excess weight among US adults. *Br J Nutr* 2018;120:90–100.

15 Papadimitriou N, Muller D, van den Brandt PA, *et al*. A nutrient-wide association study for risk of prostate cancer in the European prospective investigation into cancer and nutrition and the Netherlands cohort study. *Eur J Nutr* 2020;59:2929–37.

16 Pomeranz JL, Wilde P, Huang Y, *et al*. Legal and administrative feasibility of a federal junk food and sugar-sweetened beverage Tax to improve diet. *Am J Public Health* 2018;108:203–9.

17 United States. Department of Health and Human Services., United States. Department of Agriculture., and United States. Dietary Guidelines Advisory Committee. *Dietary guidelines for Americans, 2015-2020*. Eighth edition. Washington, D.C: U.S. Department of Health and Human Services and U.S. Department of Agriculture, 2015: 122.

18 Long MW, Gortmaker SL, Ward ZJ, *et al*. Cost effectiveness of a sugar-sweetened beverage excise Tax in the U.S. *Am J Prev Med* 2015;49:112–23.

19 Gortmaker SL, Wang YC, Long MW, *et al*. Three interventions that reduce childhood obesity are projected to save more than they cost to implement. *Health Aff* 2015;34:1932–9.

20 Barrett JL, Gortmaker SL, Long MW, *et al*. Cost effectiveness of an elementary school active physical education policy. *Am J Prev Med* 2015;49:148–59.

21 Ventura AK, Worobey J. Early influences on the development of food preferences. *Current Biology* 2013;23:R401–8.

22 Cooke L. The importance of exposure for healthy eating in childhood: a review. *J Hum Nutr Diet* 2007;20:294–301.

23 Lake AA, Adamson AJ, Craigie AM, *et al*. Tracking of dietary intake and factors associated with dietary change from early adolescence to adulthood: the ASH30 study. *Obes Facts* 2009;2:157–65.

24 Srour B, Fezeu LK, Kesse-Guyot E, *et al*. Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *BMJ* 2019;365:l1451.

25 Rico-Campà A, Martínez-González MA, Alvarez-Alvarez I, *et al*. Association between consumption of ultra-processed foods and all cause mortality: *SUN prospective cohort study*. *BMJ* 2019;7:l1949.

26 Askari M, Heshmati J, Shahinfar H, *et al*. Ultra-processed food and the risk of overweight and obesity: a systematic review and meta-analysis of observational studies. *Int J Obes* 2020;44:2080–91.

27 Chen X, Zhang Z, Yang H, *et al*. Consumption of ultra-processed foods and health outcomes: a systematic review of epidemiological studies. *Nutr J* 2020;19:86.

28 Pagliai G, Dinu M, Madarena MP, *et al*. Consumption of ultra-processed foods and health status: a systematic review and meta-analysis. *Br J Nutr* 2021;125:1–11.

29 Costa CDS, Assunção MCF, Loret de Mola C, *et al*. Role of ultra-processed food in fat mass index between 6 and 11 years of age: a cohort study. *Int J Epidemiol* 2021;50:256–65.

30 Rauber F, Campagnolo PDB, Hoffman DJ, *et al*. Consumption of ultra-processed food products and its effects on children's lipid profiles: a longitudinal study. *Nutr Metab Cardiovasc Dis* 2015;25:116–22.

31 Leffa PS, Hoffman DJ, Rauber F, *et al*. Longitudinal associations between ultra-processed foods and blood lipids in childhood. *Br J Nutr* 2020;124:341–8.

32 Arrúa A, Curutchet MR, Rey N, *et al*. Impact of front-of-pack nutrition information and label design on children's choice of two snack foods: Comparison of warnings and the traffic-light system. *Appetite* 2017;116:139–46.

33 Poquet D, Ginon E, Goubel B, *et al*. Impact of a front-of-pack nutritional traffic-light label on the nutritional quality and the hedonic value of mid-afternoon snacks chosen by mother-child dyads. *Appetite* 2019;143:104425.

34 Falbe J, Thompson HR, Becker CM, *et al*. Impact of the Berkeley excise tax on sugar-sweetened beverage consumption. *Am J Public Health* 2016;106:1865–71.

35 Taillie LS, Rivera JA, Popkin BM, *et al*. Do high vs. low purchasers respond differently to a nonessential energy-dense food tax? Two-year evaluation of Mexico's 8% nonessential food tax. *Prev Med* 2017;105S:S37–42.

36 Assessment of the impact of a public health product Tax. P. 30.

37 Azeredo CM, de Rezende LFM, Canella DS, *et al*. Food environments in schools and in the immediate vicinity are associated with unhealthy food consumption among Brazilian adolescents. *Prev Med* 2016;88:73–9.

38 Rosettie KL, Micha R, Cudhea F, *et al*. Comparative risk assessment of school food environment policies and childhood diets, childhood obesity, and future cardiometabolic mortality in the United States. *PLoS One* 2018;13:e0200378.

39 Gearan EC, Fox MK. Updated nutrition standards have significantly improved the nutritional quality of school Lunches and Breakfasts. *J Acad Nutr Diet* 2020;120:363–70.

40 Dillman Carpentier FR, Correa T, Reyes M, *et al*. Evaluating the impact of Chile's marketing regulation of unhealthy foods and beverages: pre-school and adolescent children's changes in exposure to food advertising on television. *Public Health Nutr* 2020;23:747–55.

41 Ogden CL, Lamb MM, Carroll MD, *et al*. Obesity and socioeconomic status in children and adolescents: United States, 2005-2008. *NCHS Data Brief* 2010:1–8.

42 Rogers R, Eagle TF, Sheetz A, *et al*. The relationship between childhood obesity, low socioeconomic status, and Race/Ethnicity: lessons from Massachusetts. *Child Obes* 2015;11:691–5.