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Ultra-processed food consumption and its effects on anthropometric and glucose profile: A longitudinal study during childhood

C.S. Costa [a,*], F. Rauber [b], P.S. Leffa [a], C.N. Sangalli [a], P.D.B. Campagnolo [c], M.R. Vitolo [d]

[a] *Graduate Program in Health Sciences, Universidade Federal de Ciências da Saúde de Porto Alegre, RS, Brazil*
[b] *Center for Epidemiological Research in Nutrition and Health, University of Sao Paulo, SP, Brazil*
[c] *School of Health, Universidade do Vale do Rio dos Sinos, Sao Leopoldo, RS, Brazil*
[d] *Graduate Program in Paediatrics, Attention to Children and Adolescent Health, Brazil*

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Abstract *Background and aims:* Obesity and insulin resistance development are related to known risk factors (such as diet) that begin in childhood. Among dietary factors, the consumption of ultra-processed foods has received attention. The present study investigated the association between ultra-processed foods consumption at preschool age and changes in anthropometric measurements from preschool to school age and glucose profile at school age.
Methods and results: The present study was a follow-up of a randomized controlled trial, conducted with 307 children of low socioeconomic status from São Leopoldo, Brazil. At ages 4 and 8 years, children's anthropometric assessments were collected from preschool to school age including body-mass index (BMI) for-age, waist circumference (WC), waist-to-height ratio (WHtR) and skinfold. At the age 8 years, blood tests were performed to measure glucose profile. Dietary data were collected through 24-h recalls and the children's ultra-processed food intake was assessed. Linear regression analysis was used to assess the relationship between ultra-processed food consumption and the outcomes. The percentage of daily energy provided by ultra-processed foods was 41.8 ± 8.7 (753.8 ± 191.0 kcal) at preschool age and 47.8 ± 8.9 (753.8 ± 191.0 kcal) at school age, on average. The adjusted linear regression analyses showed that ultra-processed food consumption at preschool age was a predictor of an increase in delta WC from preschool to school age ($\beta = 0.07$; 95%CI 0.01–0.14; $P = 0.030$), but not for glucose metabolism.
Conclusion: Our data suggest that early ultra-processed food consumption played a role in increasing abdominal obesity in children. These results reinforce the importance of effective strategies to prevent the excessive consumption of ultra-processed foods, especially in early ages.

Introduction

It has been reported that the development and progression of overweight and insulin resistance, which has been accepted as having a pivotal role in the etiopathogenesis of non-communicable diseases [1], are related to a number of risk factors that begin in childhood, including children's diet [2]. There are a number of specific dietary patterns

* Corresponding author.
E-mail address: cintiadossantoscosta@terra.com.br (C.S. Costa).

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that are associated with overweight and insulin resistance, including high-fat and energy-dense food intake [3] and high glycaemic load diets [4].

In this context, in the last decade, special attention has been given to the relationship between the distinctions of foods by type of industrial processing [5]. Analyses of nationally representative dietary surveys conducted in high [6–8] and upper middle-income countries [9] have consistently reported that consumption of ultra-processed foods negatively impacts the nutritional quality of diets, as these products are nutritionally imbalanced compared with no ultra-processed foods. Ultra-processed foods often present relatively high amounts of unhealthy types of fat, free sugar and sodium [7–9], higher glycaemic response and lower satiety potential [10] compared to unprocessed or minimally processed food. Nevertheless, sales of ultra-processed products are rising rapidly worldwide, specifically in Brazil, where the annual growth of ultra-processed foods is 2.1% per year, which is almost twice the annual growth in Canada (1.3% per year) [11].

The impact of ultra-processed food consumption on NCDs global epidemic is a relevant issue. Studies have demonstrated a positive association between the consumption of ultra-processed foods and the risk of overweight and obesity [12–14] and related diseases, such as hypertension [15] and cancer [16] and also higher inflammation-related processes in human microbiota composition [17]. However, only a limited number of studies have investigated the association between ultra-processed food consumption and health's damage during childhood [18,19]. Notably, we previously showed that early ultra-processed food consumption played a role in altering total cholesterol and LDL cholesterol concentrations in school age children [18]. For the present study, our objective was to investigate the association between ultra-processed foods consumption at preschool age and anthropometric measurements from preschool to school age and glucose profile at school age.

Methods

Study population

This longitudinal study used data from preschool age (4 years old) and school age (8 years old) children who participated in a randomized trial of dietary counselling on breastfeeding and dietary practices during the first year of life [20]. Five hundred mother–child pairs were recruited between October 2001 and June 2002 at the maternity ward of a hospital in a low-income population setting in the city of São Leopoldo, Brazil. Mothers of healthy, singleton, full-term (>37 weeks) and normal weight (≥2500 g) babies were invited to participate. We excluded HIV-positive mothers, infants with congenital malformations or infants who were admitted to neonatal intensive care units. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and the Ethics Committee of the Universidade Federal de Ciências da Saúde de Porto Alegre, Brazil, approved all procedures involving human subjects. All parents provided written informed consent.

The sample size initially chosen for the trial was based on the goal of detecting a difference in the prevalence of exclusive breast-feeding at 4 months [20]. A power of 80%, CI of 95% and a loss prediction of 25% were used to calculate the sample size, resulting in the inclusion of 200 mother–baby pairs in the intervention group and another 300 in the control group. Among the 500 children initially recruited at birth, 354 underwent assessment at 4 years old and 315 underwent assessment at 8 years old. As the present study had a different aim, we proceeded to estimate if the available sample at 8 years was sufficient to investigate the association between ultra-processed food consumption and outcomes. We calculated that a sample of 254 children would be required to detect a correlation coefficient in the order of 0.3 between ultra-processed food consumption and outcomes (with 99% power and $\alpha = 1\%$).

Data collection

Trained fieldworkers conducted face-to-face structured interviews in home visits with the mothers at 6 months, 4 years, and 8 years following birth. Every month, 10% of the questionnaires were selected randomly and followed up by telephone calls to the mothers to verify the authenticity of the collected data. Data for identification and data required for locating the family in the community were collected at the time of recruitment. Children's sex and birth weight were obtained from hospital records. Pre-pregnancy weight and maternal weight at the end of pregnancy were self-reported and mothers' height was measured during home visits by fieldworkers when the children were six months old. Pre-pregnancy BMI was calculated as pre-pregnancy weight divided by the square of height (kg/m^2). Household income, maternal schooling and duration of exclusive breastfeeding data were assessed when the children reached an age of 6 months by face-to-face interviews with mothers. In order to assess a sedentary lifestyle marker among 8-years-old children, mothers were asked to report the total (hours and minutes) of screen duration on the preceding day of the interview [22].

Anthropometric measurements

Anthropometric data was obtained at the ages of 4 and 8 years using a digital scale (Techline, São Paulo, Brazil) to the nearest 0.1 kg and a stadiometer (SECA, Hamburg, Germany) to the nearest 0.1 cm. BMI-for-age z-scores (BMIz) were estimated based on the World Health Organization standards [23,24]. Waist circumference (WC) was measured at the minimum circumference between the iliac crest and the rib cage using a non-stretchable tape (Sanny, São Paulo, Brazil) to the nearest 0.1 cm. Waist to Height ratio (WHtR) was calculated [25]. Tricipital and subscapular skinfold thickness were measured (Lange, Cambridge, Maryland) to the nearest 0.5 mm in a standardized way and the sum of the two individual skinfolds was computed.

Glucose profile

Venous blood samples were collected from the subjects' right arms after fasting overnight (12 h). At the age of 8 years, we assessed serum glucose and insulin concentration. Serum analyses were performed at the Cardiology Institute of Rio Grande do Sul, Brazil, laboratory by technicians who were blinded to the objectives of this study. Glucose and insulin were estimated using an automatic analyser (Cobas Integra®, Roche, São Paulo, Brazil). At 8 years old, HOMA-IR was calculated as (insulin μU/ml x glucose mmol/l)/22.5 [26].

Dietary data

At the ages of 4 and 8 years, two 24-hour dietary recalls for each child were collected on two non-consecutive days, using the multiple-pass method [27]. For preschool children, mothers or other caregivers provided information about all foods and beverages consumed by children during the previous day; recalls of school age children were self-reported with assistance from mothers or other caregivers. Individuals provided information on quantities (using household measures) and preparation methods of each consumed item. Sufficient data were collected during the dietary assessment to make a clear distinction between the four NOVA classification groups (e.g. prepacked frozen dishes versus homemade dishes) and for an accurate quantification of portion size. If necessary, to quantify food portion size, pictures were used to illustrate common household for a variety of foods, such as teaspoons, tablespoons, and cups. Dietary information was entered in a nutritional support program with a database of Brazilians foods (NutWin, version 1.5, Sao Paulo, Brazil) in order to estimate energy and grams of ultra-processed food intake.

To determine the association between ultra-processed food intake and the outcomes, children's diet was assessed using the *NOVA* food classification system proposed by Monteiro and colleagues [5]. *NOVA* classifies all foods according to the nature, extent and purpose of the processing they undergo into four groups: unprocessed and minimally processed foods, including foods that are fresh or processed without the addition of substances such as salt, sugar, oils, or fats and infrequently contain additives (Group 1); processed culinary ingredients, designed to be combined with foods to make meals and dishes (Group 2); processed foods (Group 3), relatively simple products made by adding sugar, oil, salt or other group 2 substances to group 1 foods; and ultra-processed foods and drink products (Group 4), which are formulations manufactured using several ingredients and a series of processes and typically including little or no fresh food (such as soft drinks, sweet or savoury packaged snacks, processed meats and pre-prepared frozen dishes). For the purposes of this study, only Group 4 was analysed.

The usual dietary intake of energy and ultra-processed foods was estimated by the Multiple Source Method (MSM) https://msm.dife.de/ [28]. The MSM calculates dietary intake for individuals and then constructs the population distribution based this data. This method was used to correct dietary data for intra- and inter-personal variability. A probability value of 0.5 (50%) was used to assign habitual ultra-processed foods consumer status, assuming that there is a certain percentage (50%) of real habitual consumers among individuals who did not consume ultra-processed foods (for total and for each ultra-processed food group) during the two dietary recall periods. Therefore, 50% of those who did not consume ultra-processed foods in the 24-hour dietary recall period were randomly assigned to the habitual consumer status group. An intake estimate was calculated for those who were selected in this manner. After the MSM was applied, dietary data were analysed considering the percentage of total energy provided by ultra-processed foods and the percentage of total energy provided by each subgroup of ultra-processed foods, separately.

Statistical analysis

Variables were described using mean and standard deviation (normally distributed data) or median and interquartile range (non-normally distributed data), and percent frequency. Non-normally distributed variables were log-transformed before analysis (insulin concentration and HOMA-IR values in 8 year old children). Mean and standard deviation for the contribution of each subgroup of ultra-processed food to the total energy intake were then calculated (as a percentage of total energy). Because we have anthropometric data at ages 4 and 8 years, these outcomes were expressed as unit changes in measurements from preschool to school age (Δ anthropometric measurements = anthropometric measurement at 8 years old minus anthropometric measurement at 4 years old).

We first analysed if there were differences between intervention and control groups with respect to ultra-processed consumption, and found no differences at the ages of 4 ($p = 0.468$) and 8 ($p = 0.546$) years. In the absence of intervention effect, we grouped control and intervention children to analyse the study hypothesis. Linear regression analysis was used to explore the association between the consumption of ultra-processed foods at age 4 years and delta anthropometric measurements from preschool to school age and the association between the consumption of ultra-processed foods at age 4 years and glucose profile at age 8 years. The model was adjusted for group status in the early phase (intervention and control), pre-pregnancy BMI, sex, birth weight, breastfeeding, family income, maternal schooling and total screen. The BMI z-score at age 8 years was not included in this model as a covariate because it may plausibly mediate the association of ultra-processed foods and anthropometric or glucose outcomes. Additional analyses were performed considering the relation between ultra-processed food consumption at age 4 years and anthropometric measurements at 8 years adjusted for baseline (Supplementary Table S1). However, we made additional analyses to verify the effects of this variable on the results. Data were expressed as standardized regression coefficient β, 95% CI and P values. Collinearity was checked in all models. All statistical analyses were performed using SPSS 16.0 (SPSS IBMInc., Chicago, IL USA) and statistical significance was set at $p < 0.05$, two-sided.

Results

Among the 500 children initially recruited at birth, 354 underwent assessment at age 4 and 315 underwent assessment at age 8. Loss to follow-up in this cohort was due to refusal to participate, change of address, child or maternal death and genetic disease. No differences were found between children who were lost to follow-up and those who remained at age 8 years in terms of group status ($p = 0.90$), pre-gestational BMI ($p = 0.31$), skin colour ($p = 0.71$), sex ($p = 0.57$), birth weight ($p = 0.28$), birth length ($p = 0.83$), breastfeeding duration ($p = 0.93$), maternal age at child's birth ($p = 0.10$), maternal education level ($p = 0.38$), and annual family income ($p = 0.28$).

The children's and households' characteristics and descriptive information on the children's anthropometric and glucose profile are presented in Table 1. Briefly, the proportion of overweight children (BMI>1 SD) was 20.6% (n = 71) at age 4 years and 27.5% (n = 85) at age 8 years. The proportion of children with waist-to-height ratio (WHtR) higher than 0.5 [21] was 35.3% (n = 119) at age 4 years and 12.0% (n = 37), at age 8 years.

The percentage of energy provided by ultra-processed foods was 41.8 ± 8.7 (753.8 ± 191.0 kcal) at preschool age and 47.8 ± 9.0 (753.8 ± 191.0 kcal) at school age, on average. Table 2 shows the percentage of energy provided by each subgroup of ultra-processed foods at 4 and 8 years. The adjusted linear regression analyses evaluating the associations between ultra-processed food consumption and anthropometric measurements are shown in Table 3. The consumption of ultra-processed foods at preschool age was a significant predictor of an increase in delta WC measurements from preschool to school age. In summary, for every 10% increase in energy intake from ultra-processed foods, delta WC increased by 0.7 cm after adjusting for confounders. After further adjustment for BMI z-score at age 8 years, the magnitude and the statistical significance of the association between the ultra-processed food consumption at 4 years and WC measurement at age 8 years remained fairly the same (Supplementary Table S2). Regarding the analysis considering groups of ultra-processed foods, only after adjustment for BMI z-score at age 8 years, we found a positive association between sweets consumption at age 4 years and delta WC and delta WtH measurement (B = 0.11, p = 0.03, both). No significant association between ultra-processed food consumption and glucose profile was observed (Table 4), even after further adjustment for BMI z-score at age 8 years.

Table 1 Characteristics of 8-year old children and their households (n = 307).

Child	
Group (intervention), n (%)	128 (41.7)
Boys, n (%)	175 (57.0)
Skin colour (non-white), n (%)	162 (57.0)
Birth weight (g), mean (SD)	3374.4 (458.7)
Birth length (cm), mean (SD)	48.8 (2.0)
Exclusive breastfeeding, ≥ 4 months, n (%)	105 (34.8)
Waist circumference (cm), mean (SD)	57.0 (6.7)
Sum of skinfold, mean (SD)	18.0 (9.1)
Glucose (mmol/L), mean (SD)	4.4 (0.4)
Insulin (μU/ml), median (IR)	4.9 (4.8)
HOMA-IR, median (IR)	0.9 (0.9)
Households	
Maternal pre-gestational BMI ≥25 kg/m^2, n (%)	104 (37.3)
Maternal age at child's birth <20 years, n (%)	57 (18.6)
Mother's education <8 years, n (%)	172 (56.2)
Father's education <8 years, n (%)	160 (56.3)
Mother's employment, n (%)	104 (34.9)
Father's employment, n (%)	243 (89.3)
Annual family income in US$ ≤ 3000, n (%)	214 (71.3)

g: grams; cm: centimetres.

Table 2 Contribution (%) of the ultra-processed foods subgroups to total energy intake in children at ages 4 years old and 8 years old children (n = 307).

	4 years old	8 years old
Ultra-processed foods, mean (SD)		
Biscuits[a]	6.51 (4.73)	7.79 (5.66)
Breads	8.22 (4.27)	13.57 (5.76)
Breakfast cereal	0.67 (0.25)	0.60 (1.19)
Powdered chocolate	3.31 (3.14)	2.26 (2.37)
Processed meat	2.40 (2.66)	3.77 (2.30)
Savoury[b]	2.77 (3.55)	3.86 (3.24)
Soft drink[c]	5.01 (2.63)	5.21 (2.90)
Sugary milk beverages	2.12 (2.90)	2.00 (2.80)
Sweets[d]	9.27 (4.92)	7.36 (4.49)
Others[e]	1.09 (1.93)	1.37 (2.69)
Total ultra-processed foods	41.82 (8.78)	47.80 (8.99)

[a] Crackers and cookies.
[b] Chips and others salty snacks.
[c] Soda, sweetened juice and sport drinks.
[d] Candy, chocolate and ice cream.
[e] Instant noodle, dehydrated soup, mayonnaise, dressing and sauces.

Discussion

Consumption of ultra-processed foods has been pointed out as a risk factor for increasing overall obesity among both adolescents and adults [12–14]. Using data of a longitudinal study, we found that ultra-processed foods consumption was associated with increased WC from preschool to school-age. WC is a very sensitive measurement of abdominal obesity [25] and is valuable for identifying subjects at risk of developing metabolic complications [29]. Changes in WC have been also positively associated with all-cause mortality in healthy middle-aged men and women after accounting for concurrent changes in BMI [30]. The main finding of the present study is that a 10% increase in energy consumption from ultra-processed foods at preschool age, increased delta WC from preschool to school age by up to 0.7 cm, even when adjusting for potential confounding factors. This result is consistent with those of a previous cross-

Table 3 Linear regression of ultra-processed foods subgroups consumption at age 4 years on anthropometric measurements at age 8 years[a].

	ΔBMI (kg/m^2)			ΔWaist circumference (cm)			ΔWaist to height ratio (cm)			ΔSkinfold sum[b] (mm)		
	B	(95% IC)	p	B	(95% IC	p	B	(95% IC)	p	B	(95% IC)	p
Ultra-processed foods												
Biscuits[c]	−0.02	(−0.05 to 0.01)	0.131	−0.05	(−0.17 to 0.07)	0.404	0.00	(−0.01 to 0.00)	0.239	−0.07	(−0.25 to 0.11)	0.449
Bread	0.01	(−0.02 to 0.04)	0.458	0.06	(−0.05 to 0.17)	0.304	0.00	(−0.01 to 0.01)	0.885	0.08	(−0.13 to 0.28)	0.458
Breakfast cereal	−0.03	(−0.63 to 0.58)	0.932	1.48	(−1.25 to 4.22)	0.287	0.01	(−0.01 to 0.03)	0.318	−0.36	(−4.48 to 3.87)	0.862
Powdered chocolate	0.02	(−0.02 to 0.07)	0.270	0.01	(−0.19 to 0.19)	0.977	0.00	(−0.00 to 0.00)	0.500	0.01	(−0.30 to 0.27)	0.916
Processed meat	0.02	(−0.02 to 0.07)	0.279	0.11	(−0.10 to 0.32)	0.291	0.00	(0.00–0.00)	0.324	0.24	(−0.07 to 0.56)	0.130
Savoury[d]	0.02	(−0.02 to 0.05)	0.320	0.10	(−0.06 to 0.26)	0.212	0.01	(0.00–0.02)	0.215	0.03	(−0.22 to 0.28)	0.832
Soft drink[e]	0.03	(−0.02 to 0.07)	0.284	−0.01	(−0.23 to 0.21)	0.915	0.00	(0.00–0.00)	0.417	−0.13	(−0.47 to 0.20)	0.434
Sugary milk beverages	−0.01	(−0.05 to 0.04)	0.852	−0.01	(−0.22 to 0.19)	0.892	0.00	(−0.00 to 0.00)	0.824	−0.04	(−0.34 to 0.27)	0.814
Sweets[f]	0.01	(−0.02 to 0.03)	0.541	0.10	(−0.02 to 0.22)	0.094	0.01	(0.00–0.01)	0.088	0.05	(−0.12 to 0.23)	0.539
Others[g]	−0.03	(−0.10 to 0.04)	0.434	−0.19	(−0.50 to 0.13)	0.235	0.00	(−0.00 to 0.00)	0.593	−0.12	(−0.60 to 0.35)	0.613
Total ultra-processed foods	0.00	(−0.02 to 0.01)	0.569	0.07	(0.01–0.13)	0.035	0.00	(0.00–0.00)	0.108	0.05	(−0.04 to 0.15)	0.282

IC: confidence interval.
[a] Ultra-processed foods consumption expressed as percentage of total intake and adjusted to sex, group status in the early phase (intervention and control), pre-pregnancy BMI, birth weight, breastfeeding, family income, maternal schooling and total screen duration.
[b] Skinfold sum: triceps and subscapular.
[c] Crackers and cookies.
[d] Chips and others salty snacks.
[e] Soda, sweetened juice and sport drinks.
[f] Candy, chocolate and ice cream.
[g] Instant noodle, dehydrated soup, mayonnaise, dressing and sauces.

sectional study where higher consumption of ultra-processed foods was linked to the prevalence of metabolic syndrome (MS) in adolescents, using increased WC as criteria for MS definition [19]. Investigation on consumption of ultra-processed foods during childhood is important because the dietary habits acquired over this period tend to track throughout life [31]. Our results suggest that the dysmetabolic effects of ultra-processed foods may even start in childhood, placing the paediatric population at potential risk for non-communicable diseases.

Several mechanisms may explain the relationship between higher consumption of ultra-processed foods and

Table 4 Linear regression of ultra-processed foods subgroups consumption at age 4 years on glucose profile and insulin resistance values at age 8 years[a].

	Glucose (mmol/l)			Insulin (uU/ml)			HOMA-IR[b]		
	B	(95% IC)	p	B	(95% IC)	p	B	(95% IC)	p
Ultra-processed foods									
Biscuits[c]	−0.00	(−0.01 to 0.01)	0.987	0.00	(−0.01 to 0.01)	0.530	−0.01	(−0.01 to 0.01)	0.549
Breads	0.00	(−0.01 to 0.01)	0.971	−0.01	(−0.01 to 0.01)	0.566	0.00	(−0.01 to 0.01)	0.577
Breakfast cereal	0.13	(−0.12 to 0.38)	0.307	0.11	(−0.12 to 0.35)	0.350	0.13	(−0.12 to 0.37)	0.315
Powdered chocolate	0.01	(−0.01 to 0.03)	0.210	0.00	(−0.01 to 0.02)	0.790	0.00	(−0.01 to 0.02)	0.701
Processed meat	−0.01	(−0.02 to 0.01)	0.700	0.00	(−0.02 to 0.02)	0.780	0.00	(−0.02 to 0.02)	0.795
Savoury[d]	−0.01	(−0.02 to 0.00)	0.163	−0.00	(−0.02 to 0.01)	0.702	−0.00	(−0.02 to 0.01)	0.614
Soft drink[e]	0.01	(−0.01 to 0.03)	0.365	0.01	(−0.01 to 0.02)	0.465	0.01	(−0.01 to 0.03)	0.435
Sugary milk beverages	−0.00	(−0.02 to 0.02)	0.790	−0.01	(−0.02 to 0.01)	0.437	−0.01	(−0.02 to 0.01)	0.467
Sweets[f]	−0.01	(−0.02 to 0.01)	0.301	0.00	(−0.01 to 0.01)	0.996	0.00	(−0.01 to 0.01)	0.927
Others[g]	0.00	(−0.03 to 0.02)	0.735	0.00	(−0.02 to 0.03)	0.919	0.00	(−0.03 to 0.03)	0.951
Total ultra-processed foods	0.00	(−0.01 to 0.00)	0.669	0.00	(−0.00 to 0.01)	0.971	0.00	(−0.01 to 0.01)	0.995

IC: confidence interval.
[a] Ultra-processed foods consumption expressed as percentage of total intake and adjusted to sex, group status in the early phase (intervention and control), pre-pregnancy BMI, birth weight, breastfeeding, family income, maternal schooling and total screen duration.
[b] HOMA-IR: homeostatic model assessment of insulin resistance (insulin μU/ml x glucose mmol/l)/22.5).
[c] Crackers and cookies.
[d] Chips and others salty snacks.
[e] Soda, sweetened juice and sport drinks.
[f] Candy, chocolate and ice cream.
[g] Instant noodle, dehydrated soup, mayonnaise, dressing and sauces.

abdominal obesity. Such products are nutritionally unbalanced and contribute to a significant increase in the daily energy intake because of its excessive content of free sugars, trans fat and saturated fat [8–10] and lower thermogenesis [32] when comparing to non ultra-processed food. In addition, ultra-processed foods are highly palatable and can lead to the disruption of hunger and satiety signals [10]. Finally, the omnipresence, convenience and aggressively marketed associated with eating these products, that are often commercialized in large portion sizes, encourage consumption behaviours such as snacking and eating while watching television [5,11]. All these factors are associated with overconsumption and subsequent obesity. Another important concern is the increased cumulative intake of artificial substances added to ultra-processed food products by industry. Research indicates that artificial fructose may contribute to obesity by modulating the gut microbiota [33], artificial sweetener may contribute to weight gain by stimulating basal insulin secretion [34], and sodium sulphite, sodium benzoate and curcumin may promote obesity by decreasing leptin secretion [35].

We did not observe an association between ultra-processed food consumption at age 4 years and delta BMI from ages 4 to 8 years. There is evidence that BMI has a low sensitivity and a high specificity in detecting adiposity in children [36]. Then, it is possible that the impact of ultra-processed food consumption is smaller on BMI (which represents the sum of fat-free mass and fat mass) compared to WC (a best external indicator of the fat compartments related to metabolic complications) in our sample. The lack of association between consumption of these products and glucose profile in the present study may be explained by the young age of our sample who may have not been exposed to the effects of ultra-processed foods diet long enough to cause damage in glucose metabolism. Nevertheless, the negative effect of ultra-processed foods consumption on the abdominal obesity at age 8 years may be considered a risk factor for an impaired glucose metabolism at later stages.

Ultra-processed food production and consumption are gradually increasing globally, particularly in lower-income countries [37]. In order to combat this, strategies of public health are needed. A recent study showed that a modest reduction of ultra-processed foods intake could prevent or postpone approximately 5.5% of cardiovascular deaths by 2030 in Brazil [38]. Unless ultra-processed foods overconsumption, specially during childhood, is curtailed, other interventions will have limited impact on controlling obesity and its related NCDs, since food preferences are formed early in life and predict food consumption patterns throughout life [39].

There are some specific limitations of this study that warrant discussion. First, its longitudinal design allows subjects to be lost during follow-up. However, we found no differences between the baseline characteristics of children who remained in the study and those who were lost to follow-up. Second, the study included a sample of children of low socioeconomic status and this could limit the ability to generalize the results to populations with different economic status. It is noteworthy that socioeconomic vulnerability predisposes these children to worse health outcomes, highlighting the importance of studies in populations of low socioeconomic status. In addition, the consumption of ultra-processed foods in Brazil rises with income [40], therefore, the observed relationship could be even more important for middle and upper socioeconomic groups. Third, the use of two 24- hour dietary recalls is not ideal, as foods consumed on the assessment day may not be representative of usual diet. However, it was not practically possible to include more days. Therefore, the dietary intake data was corrected for intra- and inter-personal variability using the MSM method [28] to partially counteract reporting error. Finally, we were not able to measure insulin resistance before age 8 years, and other studies will be needed to fill this gap. Despite these limitations, this study has a number of strengths. Most importantly, because we used a longitudinal design, it is possible to assess cause–effect relationships between variables. In addition, the statistical analyses controlled for several confounding variables, including socioeconomic, anthropometric and dietary factors, and used NOVA system has been recognized as a tool in nutrition research with potential for wider application in food policy [41].

Conclusion

Our results add new evidence on the relationship between the early consumption of ultra-processed foods and increasing waist circumference in children, and highlight the need for a comprehensive assessment of the effect of ultra-processed foods on health. These results may provide a substantial basis for intervention studies and are relevant for formulating public health strategies aimed at preventing potential risks for NCDs and reducing consumption of ultra-processed foods.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.numecd.2018.11.003.

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