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Consumption of ultra-processed foods and dental caries in Brazilian adolescents

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This study aimed to evaluate the association between ultra-processed food (UPF) and dental caries, considering muscle mass (MM), bone mineral density (BMD), and oral hygiene habits (OHH) as mediators. This study has an analytical cross-sectional design with 2,515 adolescents (18–19 years). The main exposure – the UPF intake ratio – was established using the food frequency questionnaire. The outcome was the number of decayed teeth, according to the DMFT index. The model adjustment included socio-economic status (SES), frequency of physical activity, and concurrent risk habits (CRH) as potential confounders. Three latent variables were considered: SES (family income, economic class, household head, and adolescent education), OHH (gingival bleeding on probing index and visible plaque index), and CRH (alcohol and tobacco dependence). The analyses used structural equation modeling, estimating the standardized coefficient (SC) in three models: lumbar BMD(1), femoral BMD(2), and total BMD(3). UPF consumption had a direct (SC_{model1}=0.071, SC_{model2}=0.072, SC_{model3}=0.071; $p < 0.05$) and total (SC_{model1}=0.067, SC_{model2}=0.068, SC_{model3}=0.068; $p < 0.05$) effect on the number of decayed teeth. BMD and MM did not mediate the association between UPF and dental caries, but the indirect association mediated by OHH was significant in all analyses ($p < 0.05$). Dental Caries was explained in other specific pathways: SES→UPF→Dental Caries ($SCmodel_1 = 0.009$, $SCmodel_2 = 0.008$, $SCmodel_3 = 0.009$); SES→OHH→Dental Caries ($SCmodel_1 = 0.033$, $SCmodel_2 = 0.033$, $SCmodel_3 = 0.034$); CRH→UPF→Dental Caries ($SCmodel_1 = 0.009$, $SCmodel_2 = 0.008$, $SCmodel_3 = 0.008$); CRH→OHH→Dental Caries ($SCmodel_1 = 0.029$, $SCmodel_2 = 0.027$, $SCmodel_3 = 0.027$). Dental caries prevention should include encouraging good OHH, healthy eating, and developing equitable public policies in middle and low-income countries like Brazil.

Keywords Adolescent, Bone density, Bone loss, Dental caries, Diet, Ultra-processed foods (UPFs)

According to the Global Burden of Disease, the prevalence of untreated dental caries in permanent teeth in the Brazilian population is 22.99%, making it a public health problem[1]. Dental caries has a multifactorial etiology, explained by risk factors such as socio-economic status (SES)[2], low frequency of brushing and flossing[3], risky habits, such as alcohol consumption[4] and smoking[5], and, especially, a diet rich in added sugars[6]. Therefore, oral hygiene habits should be encouraged to reduce the incidence of dental caries. There must also be investments in public policies that modify social determinants and risk behaviors and promote reducing the amount and frequency of added sugar intake. This additive modifies sensory attributes and is included as an ingredient in ultra-processed foods (UPF).

UPF consists of industrial formulations made from numerous food-derived ingredients, with little or no whole food. They are often added with additives that modify their sensory attributes, such as added sugars, oils, fats, salt, antioxidants, stabilizers, and preservatives[7]. UPF intake was considered a risk factor for periodontal disease[8] and dental caries in children[9] and adolescents[10]. A possible explanation for the relationship between UPF consumption and dental caries is that high added sugar levels are usually present in UPF[11]. In addition, the UPF-rich diet was associated with worse SES[12], increased risk of developing metabolic syndrome[13], obesity[14], decreased lean mass in Brazilian women[15] and muscle mass in adolescents[16].

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Dental caries and periodontal diseases are chronic oral conditions influenced by risky behaviors such as smoking and alcohol consumption. Inadequate oral hygiene habits are also a common cause, resulting in dental biofilm and periodontal inflammation with a consequent increase in gingival bleeding sites[17]. In addition, low bone mineral density was identified as a risk factor for more severely affected periodontal diseases in adolescents[18] and tooth loss in climacteric women[19–21]. Lower muscle strength was associated with a greater chance of developing dental caries[22], while physical activity was associated with reduced periodontal diseases[23]. Consumption of beverages rich in added sugars was associated with lower bone mineral density in adolescents[24]. However, the relationship between UPF consumption and dental caries in younger populations and the potential explanatory pathways mediated by oral hygiene habits, bone mineral density, and muscle mass remain unknown, especially in population-based studies in vulnerable areas, such as a Brazilian capital in the Northeast region.

A high prevalence of dental caries, more frequent UPF consumption, and low bone mineral density and muscle mass may be risk markers for the future development of chronic noncommunicable diseases in younger populations. As dental caries can be considered a complex and multifactorial outcome, a statistical analysis that evaluates total, direct, and indirect effects, such as structural equation modeling (SEM), may be more appropriate for understanding its risk factors than conventional regression models[25].

Our central hypothesis is that high UPF consumption may be an independent risk factor for dental caries directly or mediated by low bone mineral density, low muscle mass, and oral hygiene habits. This hypothesis was tested with a sample from the RPS Consortium Cohorts, Brazil — "Obesity, chronic disease precursors, human capital, and determinants of mental health across the life cycle"[26]. RPS consortium cohorts involve three municipalities from different regions of the country: Ribeirão Preto (São Paulo State), in the Southeastern region; Pelotas (Rio Grande do Sul State), in the Southern region; and São Luís (Maranhão State), in the Northeastern region. This consortium aimed to investigate how SES, prenatal and perinatal factors, and unhealthy lifestyle habits can have unfavorable repercussions on health throughout the life cycle[27]. Studies with Consortium data, such as the RPS, can make important contributions to the design of public health policies, especially in low- and middle-income countries, as specific associations can be modified by the context of the countries[27].

This study aims to investigate the association between UPF consumption and dental caries, testing possible pathways mediated by bone mineral density, muscle mass, and oral hygiene habits in Brazilian adolescents, including common risk factors in the model, such as SES, dependence on alcohol, tobacco, and frequency of physical activity.

Methods

Study and sample design

This is a cross-sectional study in the RPS Consortium Cohorts, Brazil. The project referring to the birth cohort of São Luís was approved by the Human Research Ethics Board of the Federal University of Maranhão (CAAE 49096315.2.0000.5086, process number 1.302.489) and was conducted in compliance with the Helsinki Declaration of 1975, as revised in 2013. All participants or their legal representatives signed the informed consent form. We reported this study following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

The initial sample of the cohort was probabilistic with systematic selection in public and private hospitals, comprising 2,541 live births, stillbirths, and single and multiple births of women residing in São Luís, Maranhão, Brazil. Participants were assessed three times: at birth (baseline, in 1997–1998), in childhood (7–9 years old, in 2004–2005), and in adolescence (18–19 years old, in 2016). This study enrolled 2,515 adolescents, followed by the initial cohort ($n=687$) or retrospectively enrolled ($n=1,828$)[26,27]. The strategy of opening the cohort to others born in 1997–1998 by selection from the Brazilian Information System on Live Births (SINASC), with retrospective data collection, allowed increased sample power and prevented future losses.

This sample size had power above 95% to identify associations between the consumption of UPF and the number of decayed teeth, considering the following study parameters: alpha = 0.05, sample size = 1,975, squared partial correlation in the reduced model (only main exposition - UPF - and outcome - dental caries) = 0.0102, squared partial correlation in the full model (including all the mediators and confounding variables) = 0.1143.

Outcome

The dependent variable was the number of decayed teeth. Dental caries was diagnosed according to the DMFT index modified by the World Health Organization (WHO)[28]. An oral mirror, periodontal probe (North Carolina Periodontal Probe, Hu-Friedy), and probe with millimeter markings, recommended by the WHO, were used for measurements. Six previously trained examiners performed all oral examinations in a dental office under artificial light. All teeth were examined on all sides except the third molars. The inter-rater Kappa index was 0.82 for dental caries.

Exposure

UPF consumption was assessed using a food frequency questionnaire that measures consumption in the last 12 months. The food frequency questionnaire was developed[29] and validated[30] for regional eating habits[31]. Photos of each food portion size (large, medium, and small) were made available on the computer to minimize memory bias and improve the accuracy of the portion size information. The average food portion in grams or milliliters was obtained from the Table for Evaluation of Food Consumption in Household Measures and the USDA Nutrient Database for Standard Reference. The consumption frequency reported for each item was converted to annual consumption to estimate food energy consumption. Subsequently, the annual frequency was converted to the daily frequency. In addition, the daily food intake in grams (g) or milliliters (mL) was estimated by multiplying the daily frequency by the size of the recorded portion.

Food items were grouped according to the NOVA classification. This classification groups food according to the nature, purpose, and extent of the processing industry and not in terms of nutrients and types of food[7]. Foods are grouped into four groups: *unprocessed* or minimally processed foods, processed culinary ingredients, processed foods, and ultra-processed. Exposure was estimated in terciles as the ratio of UPF calories by participants in total daily calories.

Mediators

Bone mineral density, muscle mass, and oral hygiene habits were tested as potential mediating variables of the association between UPF consumption and dental caries. Participants' bone mineral density was measured by dual-energy X-ray absorptiometry (DXA) using a Lunar Prodigy enCORE densitometer (GE Healthcare®, Chicago, IL, EUA), calibrated daily. For the examination, participants were barefoot and wore light, tight-fitting clothing with no earrings, rings, dentures, or other metallic materials. Measurements (z-score) were performed in three sites, considering the Brazilian Society for Clinical Densitometry guidelines[32]: lumbar (model 1), femoral (model 2), and entire body (model 3). Bone densitometry measurements were classified as continuous variables. From the DXA, the muscle mass was measured as a continuous variable in kg.

Gingival Bleeding on Probing (GBoP) was recorded as the presence or absence of bleeding after periodontal probing, examined in six sites per tooth, excluding third molars[33]. After drying the teeth, the visible plaque index (VPI) to the naked eye under artificial lighting was evaluated by examining four surfaces of all teeth, except for the third molars[34]. For analytical purposes, the GoP was considered as the proportion of sites with gingival bleeding on probing, and VPI as the proportion of dental faces with visible biofilm. Both were included in the analyses as continuous variables. The inter-examiner Kappa index was 0.84 for GBoP and 0.93 for VPI. This study constructed a proxy for oral hygiene habits from the GBoP and VPI.

Other covariates

Through an interview, the economic class was collected according to the Brazilian Economic Classification Criteria (A/B, C, D/E)[35]; household head education (complete higher education, incomplete higher education; secondary education, primary education, and did not attend school); adolescent education (higher education, secondary education, primary education) and family income in minimum wages (<1; 1 to 3 wages; 3 to <5 wages; $\geqq 5$ wages). The risk of alcohol dependence was assessed using the Alcohol Use Disorders Identification Test. This test consists of 10 questions to evaluate alcohol use, dependence symptoms, and alcohol-related problems[36]. The alcohol dependence risk variable was categorized into low risk (score 0 to 7) and high risk (score >7). Smoking was a dichotomous categorical variable defined as current cigarette smoking. Physical activity was assessed using the Physical Activity Survey, adapted from the Self-Administered Physical Activity Checklist. This variable was categorized into sedentary, low, moderate, and high physical activity levels.

Latent variables

Latent variables reflect the multidimensionality of complex phenomena that are difficult to estimate by observed variables, reducing possible measurement errors[25,37]. This study included three latent variables in the model: (1) SES (from the observed variables, income, ABEP, adolescent and household head education); (2) Concurrent Risk Habit (constructed by the smoking and alcohol dependence variables); and (3) Oral Hygiene Habits (formed by VPI and GBoP, to measure more objectively the oral health care of the adolescent).

Statistical analysis

The analyses initially estimated the absolute and percentage frequencies for categorical variables, means ($\pm$ standard deviations), and medians ($\pm$ interquartile deviations) for numeric variables using the Stata 16.0 software.

A theoretical model was initially proposed to investigate the effects (direct, indirect, and total) of UPF consumption, adjusted for confounding and mediating variables. Worse SES[2], risk behaviors, such as smoking[5] and alcohol dependence[4], less physical activity[38], lower muscle mass, higher amounts of proxy for oral hygiene habits[39,40], and a UPF-rich diet were considered as possible risk factors for the development of dental caries in adolescents[9,10] (Fig. 1). Statistical analyses were conducted with SEM, using Mplus 7.0 software.

SEM is a technique to address multiple dependency relationships simultaneously and can represent concepts not observed in these relationships, reducing the measurement error in the estimation process. This statistical analysis estimates a series of multiple regression equations. The model is an assumed pattern of direct and indirect linear relationships between a set of observed variables and constructs, consisting of two sub-models: the measurement model, which constructs the latent variables through factor analysis, and the structural model, which analyzes the theoretical model as a whole, where associations between variables are estimated by factor loadings (FL). A good latent variable has convergent validity and FL with high values (greater than 0.50). A negative Standardized Coefficient (SC) indicates an inverse association, and a positive SC indicates a direct association[37]. The following fit index values were considered acceptable for the models: Root Mean Square Error of Approximation (RMSEA) <0.05; Confidence Interval of RMSEA <0.08; Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) values >0.90; Weighted Root Mean Square Residual (WRMR) ≈ 1.00. All analyses adopted an alpha of 5%.

Results

Most of the studied population had a family income between 1 and <3 monthly minimum wages ($n=1{,}079$; 42.90%), were high school students ($n=1{,}758$; 69.90%), non-smokers ($n=2{,}410$, 95.83%), low-risk alcohol users ($n=2{,}026$, 80.56%), and sedentary ($n=1{,}052$; 44.92%). The average ratio of UPF caloric intake was 34.12

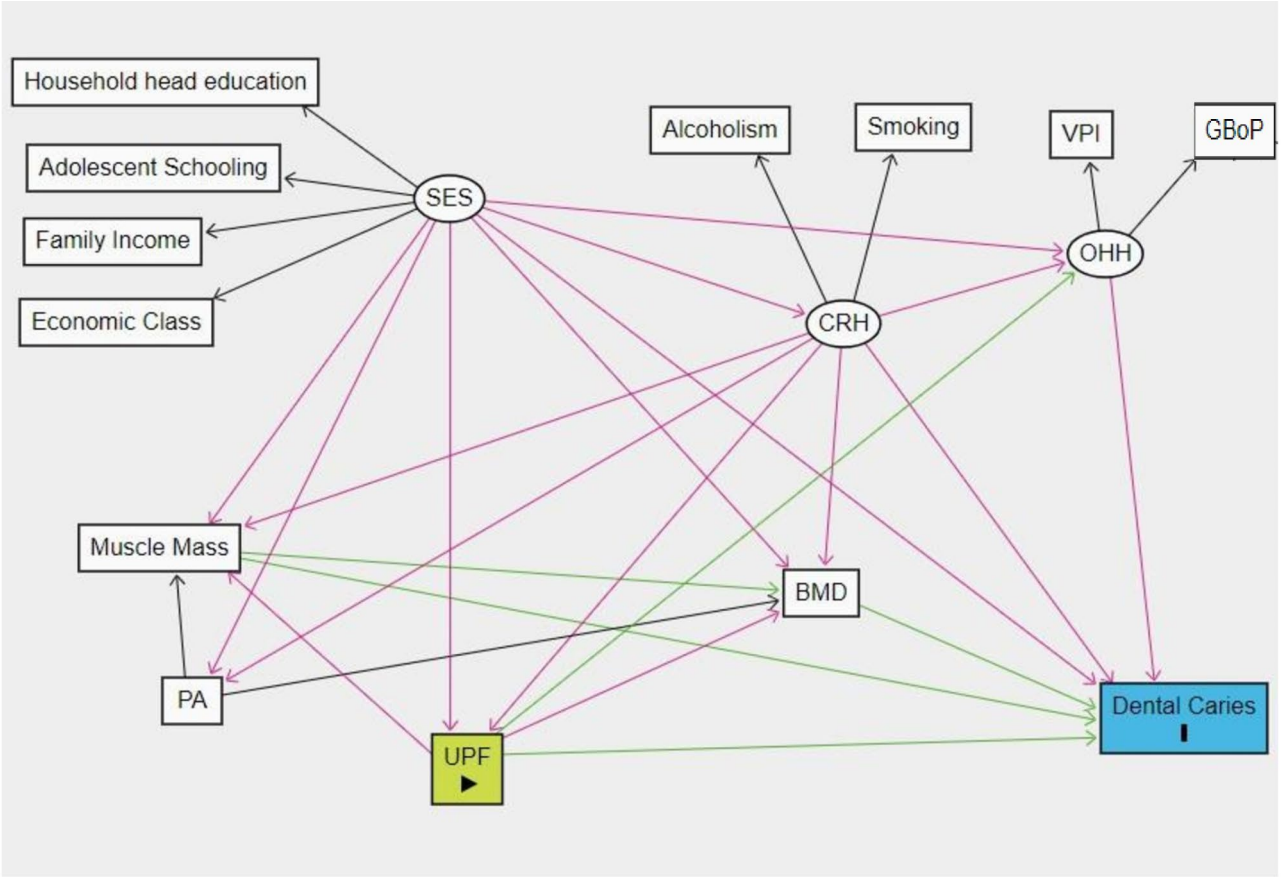

Fig. 1. A theoretical model for the association between UPF consumption and dental caries in adolescents aged 18–19. São Luís, Maranhão, Brazil, 2016. SES: Socio-economic Status; PA: Physical Activity; UPF: Ultra-Processed Foods; CRH: Concurrent Risk Habits; BMD: Bone Mineral Density; OHH: Oral Hygiene Habits; VPI: Visible plaque Index; GBoP: Gingival Bleeding on Probing. ▸: Main Exposure; ▮: Outcome; ▭: Other observed variables; ◯: Unobserved variables (latent); ▬: Main direct and indirect (mediated) paths tested in the model.

(±1.29); mean total bone mineral density was 0.317 (±1.13); femoral bone mineral density was 0.475 (±1.18), and lumbar bone mineral density was 0.280 (±0.99). The prevalence of dental caries in the sample was 53.16%, and the mean number of decayed teeth was 1.58 (±2.13) (Table 1).

All model fit indicators were satisfactory. The RMSEA was less than 0.05. The upper limit of the 90% CI was less than 0.08. The TLI and CFI indexes showed values greater than 0.90, and the WRMR was close to 1.00 (Table 2). The latent variables, including SES, concurrent risk habits, and oral hygiene habits, had standardized FL greater than 0.5, indicating good convergent validity (Table 3).

UPF consumption had a direct (SC_{model1}=0.071, SC_{model2}=0.072, SC_{model3}=0.071) and total (SC_{model1}=0.067, SC_{model2}=0.068, SC_{model3}=0.068) effect on the number of decayed teeth in three models. The indirect association mediated by oral hygiene habits (SC_{model1}=0.041, SC_{model2}=0.040, SC_{model3}=0.039; $P<0.05$) was also significant in all analyses. However, indirect pathways mediated by muscle mass and bone mineral density were insignificant ($P>0.05$). SES (SC_{model1}=0.270, SC_{model2}=0.269, SC_{model3}=0.269) and oral hygiene habits (SC_{model1}=0.200, SC_{model2}=0.200, SC_{model3}=0.200) had a direct effect on the study outcome. The higher number of decayed teeth was explained by the following specific pathways: SES→UPF→Dental Caries (SC_{model1}=0.009, SC_{model2}=0.008, SC_{model3}=0.009); SES→Oral Hygiene Habits→Dental Caries (SC_{model1}=0.033, SC_{model2}=0.033, SC_{model3}=0.034); Concurrent Risk Habit→UPF→Dental Caries (SC_{model1}=0.009, SC_{model2}=0.008, SC_{model3}=0.008) and Concurrent Risk Habit→Oral Hygiene Habits→Dental Caries (SC_{model1}=0.029, SC_{model2}=0.027, SC_{model3}=0.027). The models considering lumbar, femoral, and total bone mineral density did not differ (Table 4).

DISCUSSION

Lower bone mineral density and lower muscle mass had no total, direct, or indirect effects on the number of decayed teeth in adolescents. SES, oral hygiene habits, and UPF consumption explained the study outcome.

We identified a direct association between UPF consumption and a more significant number of decayed teeth. However, lower muscle mass did not mediate this association. This result matches the results obtained in a study with 1,442 Chinese (50–85 years), in which dental caries were positively associated with low muscle strength and sarcopenia but not with low muscle mass[22]. The decrease in muscle mass can occur later and shows

Variables	Total		
	n	%	Mean (± SD)
ABEP			
A/B	659	26.20	–
C	1116	44.37	–
D/E	450	17.90	–
Missing	290	11.53	–
Family Income in Minimum Wages			
> 1 wage	797	31.69	–
1 to < 3 wages	1079	42.90	–
3 to < 5 wages	338	13.44	–
≧ 5 wages	285	11.33	–
Missing	16	0.64	–
Household Head Education			
Did Not Attend School	286	11.37	–
Primary Education	563	22.39	–
Secondary Education	1260	50.10	–
Incomplete Higher Education	81	3.22	–
Complete Higher Education	3.25	12.92	–
Adolescent Education			
Primary Education	83	3.30	–
Secondary Education	1758	69.90	–
Higher Education	672	26.72	–
Missing	2	0.08	–
Degree of Alcohol Dependence			
Low Risk	2026	80.56	–
High Risk	489	19.44	–
Smoking			
Non-smoking	2410	95.83	–
Smoker	89	3.54	–
Missing	16	0.64	–
Physical Activity			
High	459	19.60	–
Moderate	566	24.17	–
Low	265	11.32	–
Sedentary	1052	44.92	–
UPF Intake Ratio			34.12 (1.29)
UPF Consumption			
First Tercile	824	33.36	–
Second tercile	823	33.32	–
Third Tercile	823	33.32	–
Muscle Mass			41.57 (9.6)
Total bone mineral density			0.317 (1.13)
Femoral bone mineral density			0.475 (1.18)
Lumbar bone mineral density			0.280 (0.99)
VPI			0.217 (0.15)
GBoP			0.159 (0.13)
Number of Decayed Teeth			1.58 (2.13)

Table 1. Characteristics of the study population of adolescents. São Luís, Brazil (São Luís, Brazil, 2016). Bone Mineral Density; VPI: Visible plaque Index; GBoP: Gingival Bleeding on Probing; Economic class according to the Brazilian Association of Research Companies criteria (ABEP, 2015).

a different behavior concerning other body composition parameters, such as fat mass. Furthermore, the study's cross-sectional design did not allow inferring that there is no longitudinal relationship between dental caries and muscle mass. Bone mineral density had no total, direct, or indirect effect on dental caries in this population, unlike the hypothesis of our study. In addition, the metabolism of bone mass is intense, and the femur is still in

Adjustment parameters	Model 1. Bone Mineral Density-Lumbar	Model 2. Bone Mineral Density-Femoral	Model 3. Bone Mineral Density-Total
Nº of free parameters	60	60	60
Degrees of freedom	48	48	48
χ^2	205.990	212.321	210.833
RMSEA[a]	0.036	0.037	0.036
90% CI of the RMSEA[b]	0.031–0.041	0.032–0.042	0.031–0.041
CFI[c]	0.952	0.953	0.952
TLI[d]	0.922	0.924	0.922
WRMR[e]	1.264	1.290	1.264

Table 2. Adjustment measures of the structural equation model to analyze the association between Ultra-processed Foods and Dental Caries in adolescents (São Luís, Brazil, 2016). [a]Root Mean Square Error of Approximation - RMSEA (reference: less than 0.05). [c]Upper limit of the 90% Confidence Interval (reference: upper limit of the 90% CI close to 0.08). [c]Comparative Fit Index – CFI (reference: greater than 0.95). [d]Tucker-Lewis Index – TLI (reference: greater than 0.95). [e]Weighted Root Mean Square Residual – WRMR (reference: close to 1.0).

Latent variables		Model 1. Bone Mineral Density-Lumbar		Model 2. Bone Mineral Density-Femoral		Model 3. Bone Mineral Density- Total	
		FL	P-value	FL	P-value	FL	P-value
SES	Income	**0.575**	**<0.001**	**0.570**	**<0.001**	**0.576**	**<0.001**
	Adolescent Education	**0.617**	**<0.001**	**0.622**	**<0.001**	**0.617**	**<0.001**
	Household Head Education	**0.608**	**<0.001**	**0.609**	**<0.001**	**0.608**	**<0.001**
	ABEP Economic Class	**0.728**	**<0.001**	**0.727**	**<0.001**	**0.726**	**<0.001**
CRH	Smoking	**0.833**	**<0.001**	**0.806**	**<0.001**	**0.820**	**<0.001**
	Alcohol	**0.754**	**<0.001**	**0.783**	**<0.001**	**0.770**	**<0.001**
OHH	Visible plaque Index	**0.952**	**<0.001**	**0.957**	**<0.001**	**0.949**	**<0.001**
	Gingival Bleeding on Probing Index	**0.530**	**<0.001**	**0.527**	**<0.001**	**0.531**	**<0.001**

Table 3. Factor loading, standard error, and *p*-value for the effect indicators of latent variables (São Luís, Brazil, 2016). FL: Factor Loading. CRH: Concurrent Risk Habits; OHH: Oral Hygiene Habits; SES: Socio-economic Status. ABEP: Brazilian Association of Research Companies.

the growth process until the age of 20[32]. This particularity may have contributed to the lack of identification of this association among adolescents (18–19 years). However, bone mineral density was measured using DXA, a method considered the gold standard for evaluating bone composition[32]. Additionally, we evaluated three models in our study - total, lumbar, and femur bone mineral density - considering the Brazilian Society for Clinical Densitometry guidelines, and no differences were observed between the results of the models.

The participation of UPF in the diet increased, and the consumption of *in natura* or minimally processed foods and culinary ingredients decreased in Brazil[41]. Furthermore, the high caloric participation of UPF in the diet has already been associated with lower muscle mass[16]. At the same time, the consumption of sugary drinks has been identified as a risk factor for the decrease in lumbar bone mineral density in adolescents[24]. Relevant factors for the onset of osteopenia and sarcopenia, such as SES, high UPF consumption, smoking, and alcohol consumption[15,16,24], contributed to the higher risk of developing dental caries in our study, suggesting that dental caries was an unfavorable outcome earlier. Likewise, low bone mineral density and decreased muscle mass can be considered later metabolic registers[15,16], resulting from these common risk factors shared with dental caries. The association between these phenomena and dental caries can occur at other times in the life cycle.

Oral hygiene habits mediated the association between UPF consumption and dental caries. Previous studies have shown a consistent association between added sugars[42], carbohydrate consumption[43], and dental caries. Furthermore, an unhealthy diet with higher fast food consumption and industrialized products was associated with early and moderate periodontitis in Brazilian adolescents[44]. A UPF-rich diet can be a risk factor for the balance of the oral microbiota since the higher intake of added sugars, an essential element in the compositions of several UPF, was associated with a reduction in the diversity of the oral microbiome and the predominance of cariogenic bacterial species[45].

Worse SES mediated by oral hygiene habits contributed to increased decayed teeth in adolescents, in line with previous studies[39,46]. This result matches a study conducted with data from the National Health Survey, in which inadequate or partially adequate oral self-care was associated with illiteracy, low education, lack of natural teeth, and a more significant number of lost upper teeth[47]. This explanatory path reflects that access barriers to health services and difficulties in self-care in oral health contribute to problems in performing adequate mechanical

Direct, indirect, and total pathways		Model 1. Bone Mineral Density-Lumbar		Model 2. Bone Mineral Density-Femoral		Model 3. Bone Mineral Density-Total	
		SC	*P*-value	SC	*P*-value	SC	*P*-value
Direct							
	UPF → DC	**0.071**	**0.003**	0.072	0.003	**0.071**	**0.003**
	MM→ DC	0.030	0.193	0.025	0.314	0.035	0.136
	BMD→ DC	−0.001	0.947	0.019	0.655	−0.017	0.405
	OHH → DC	**0.200**	**< 0.001**	**0.200**	**< 0.001**	**0.200**	**< 0.001**
	CRH → DC	−0.041	0.314	−0.055	0.318	−0.042	0.304
	SES → DC	**0.270**	**< 0.001**	**0.269**	**< 0.001**	**0.269**	**< 0.001**
Indirect							
UPF → Dental Caries	Total	**0.067**	**0.004**	**0.068**	**0.004**	0.068	**0.004**
	Total indirect	−0.004	0.211	−0.004	0.202	−0.003	0.312
	Indirect specific						
	UPF→BMD→DC	0.000	0.947	0.000	0.668	0.001	0.444
	UPF→MM→DC	−0.004	0.206	-0.003	0.323	−0.005	0.151
	UPF→MM→BMD→DC	0.000	0.947	0.000	0.655	0.001	0.415
	UPF→OHH→DC	0.041	0.002	0.040	0.002	0.039	0.003
MM→ Dental Caries	Total	0.030	0.190	0.028	0.226	0.031	0.177
	Total indirect	0.000	0.947	0.003	0.655	−0.004	0.409
	Indirect specific						
	MM→BMD→DC	0.000	0.947	0.003	0.655	0.035	0.136
CRH → Dental Caries	Total	**0.042**	**< 0.001**	**0.039**	**< 0.001**	**0.040**	**< 0.001**
	Total indirect	**0.042**	**< 0.001**	**0.039**	**< 0.001**	**0.040**	**< 0.001**
	Indirect specific						
	CRH→BMD→DC	0.001	0.863	0.000	0.684	0.000	0.951
	CRH→UPF→DC	**0.009**	**0.038**	**0.008**	**0.040**	**0.008**	**0.041**
	CRH→MM→DC	0.005	0.210	0.004	0.324	0.006	0.156
	CRH→OHH→DC	**0.029**	**< 0.001**	**0.027**	**0.001**	**0.027**	**0.001**
	CRH→UPF→BMD→DC	0.000	0.947	0.000	0.672	0.000	0.461
	CRH→MM→BMD→DC	0.000	0.255	0.000	0.657	−0.001	0.416
	CRH→UPF→MM→BMD→DC	0.000	0.947	0.000	0.358	−0.001	0.210
PA→ Dental Caries	Total	−0.012	0.198	-0.013	0.186	−0.012	0.193
	Total indirect	−0.012	0.198	-0.013	0.186	−0.012	0.193
	Indirect specific						
	PA→BMD→DC	0.000	0.946	-0.002	0.656	0.000	0.533
	PA→MM→DC	−0.012	0.196	-0.010	0.316	−0.014	0.139
	PA→MM→BMD→DC	0.000	0.947	-0.001	0.655	0.002	0.410
SES→ Dental Caries	Total	**0.311**	**< 0.001**	**0.310**	**< 0.001**	**0.311**	**< 0.001**
	Total indirect	**0.041**	**< 0.001**	**0.042**	**< 0.001**	**0.042**	**< 0.001**
	Indirect specific						
	SES →BMD→ DC	0.000	0.947	0.001	0.653	0.001	0.407
	SES → UPF→ DC	**0.009**	**0.008**	0.009	**0.008**	**0.009**	**0.009**
	SES →MM→ DC	−0.002	0.277	-0.001	0.372	−0.002	0.231
	SES →OHH→ DC	**0.033**	**< 0.001**	**0.033**	**< 0.001**	**0.034**	**< 0.001**
	SES → UPF→BMD→DC	0.000	0.947	0.000	0.670	0.000	0.451
	SES → UPF→MM→DC	0.000	0.947	0.000	0.335	-0.001	0.171
	SES → CRH →BMD→ DC	0.000	0.978	0.000	0.992	0.000	0.971
	SES → CRH→ UPF →DC	0.000	0.976	0.000	0.992	0.000	0.966
	SES → CRH→MM→DC	−0.001	0.277	0.000	0.992	0.000	0.966
	SES →CRH→OHH → DC	0.000	0.976	0.000	0.992	0.000	0.966
	SES → CRH→UPF→BMD→DC	0.000	0.978	0.000	0.992	0.000	0.966
	SES → UPF →MM →BMD→DC	0.000	0.947	0.000	0.657	0.000	0.423
	SES → CRH→MM→BMD→ DC	0.000	0.978	0.000	0.992	0.000	0.966
	SES →CRH →UPF →MM→ DC	0.000	0.976	0.000	0.992	0.000	0.966
	SES→CRH→UPF→MM→ BMD→ DC	0.000	0.978	0.000	0.992	0.000	0.966

Table 4. Direct, total, and total Indirect effects of Ultra-processed Foods on Dental Caries in adolescents (São Luís, Brazil 2016). Significant values ($P < 0.05$) are in bold. SC: Standardized Coefficient. SES: Socioeconomic Status; PA: Physical Activity; UPF: Ultra-Processed Foods; CRH: Concurrent Risk Habits; BMD: Bone Mineral Density; OHH: Oral Hygiene Habits; VPI: Visible plaque Index; DC: Dental Caries.

control of oral biofilm, exposing the individual to a greater risk of developing dental caries. Adolescents may be more prone to excessive UPF consumption due to its practicality, low cost, easy access provided by the environment around homes and schools, and intense commercialization of industrial foods and beverages[48,49]. Therefore, more vulnerable populations may have a less favorable food environment for establishing a healthier diet[50], predisposing them to dental caries.

Habits such as smoking, alcohol consumption, and UPF consumption have been associated with tooth decay. The results with data from the National School Health Survey (2019) showed that Brazilian adolescents with higher rates of UPF consumption are more likely to report psychoactive substance use, such as frequent consumption of alcoholic beverages and occasional smoking, in crude and adjusted regression models for socio-economic and mental health-related variables[51]. Therefore, simultaneous risk habits may contribute to establishing unhealthy eating behavior. It also contributes to changes in the oral microbiota and difficulties in establishing adequate oral health care, resulting in a higher occurrence of dental caries in adolescents.

Harmful habits such as smoking[51,52] and alcohol dependence[52,53] can increase the risk, pathogenesis, and exacerbation of periodontal diseases by a combination of several mechanisms: the decrease in gingival perfusion that restricts the supply of nutrients and oxygen and the removal of waste from cellular metabolism; suppression of the immune response, exacerbating inflammation; the suppression of the morphological and functional recovery of the periodontium; and the dysbiosis and increase of the periodontopathogenic activity of the oral microbiota[51,52]. Combining these harmful habits may accelerate and modify the periodontal disease course. Caries indicators have already been strongly correlated with periodontal parameters, forming a construct called Burden of Chronic Oral Disease in Brazilian adolescents[39] and in different age groups with 14,421 Americans from the NHANES III study[40].

Limitations of this study include the cross-sectional design, which fails to make causal inferences, and the use of the food frequency questionnaires, an instrument subject to memory and measurement bias, which may overestimate food consumption. We confirm, however, that the food frequency questionnaire is a method widely used in epidemiological studies since it can assess the usual diet pattern in a practical and low-cost way, with reliability and data accuracy when its methodological aspects are respected[54]. In addition, the instrument used in the research was validated, and regional food factors were considered. On the other hand, the study strengths are the diagnosis of dental caries and periodontal parameters measured by clinical dental examination performed by previously trained examiners and the bone mineral density and muscle mass measured by DXA, a standard device for diagnosing osteoporosis, osteopenia, and measuring lean mass. Also, constructing latent variables such as SES, concurrent risk habits, and oral hygiene habits reduced possible measurement biases, and the SEM analysis considered multiple paths simultaneously.

CONCLUSION

UPF consumption directly and widely explains dental caries. Worse SES, UPF-rich diet, oral hygiene habits, and concurrent risk habits contributed to the higher rates of decayed teeth in Brazilian adolescents. However, bone mineral density and muscle mass were not associated with this outcome. The fight against dental caries must align with Brazil's Sustainable Development Goals and Strategic Action Plan (2021–2030). The prevention of dental caries in adolescents in low- and middle-income countries is related to the mitigation of social inequalities, control of smoking and alcohol consumption, and access to healthy food.

Data availability

Due to the sensitive nature of the questions asked in this study, respondents were assured that the raw data would remain confidential and would not be shared. While the data is confidential, further information can be requested by contacting Professor Erika Bárbara Abreu Fonseca Thomaz (Department of Public Health, Federal University of Maranhão, Rua Barão de Itapari, 155, Centro, 65020-070, São Luís, Maranhão, Brazil. E-mail: erika.barbara@ufma.br).

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Author contributions
EMC contributed to the conception and design study, data acquisition, analysis, and interpretation, drafted and critical revision of the manuscript; MTSSB contributed to the conception and design study, data acquisition, and critical revision of the manuscript; LCSR contributed to data analysis and interpretation, drafted and critical revision of the manuscript; NPS contributed to the conception and design study and critical revision of the manuscript; MMPF contributed to the conception and design study and contributed to the interpretation and critical revision of the manuscript; CCCR contributed to the conception and design study and critical revision of the manuscript; CMCA contributed to the conception and design study and critical revision of the manuscript; EBAFT contributed to the conception and design study, data acquisition, analysis, and interpretation, drafted and critical revision of the manuscript. All authors approved the final version.

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Declarations

Competing interests
The authors declare no competing interests.

Additional information
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