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Association of the variant rs17492553 *TAS1R1* gene with taste preferences in childhood obesity

Abstract. ***Background.*** *Genetic variations in taste receptor genes, including taste 1 receptor member 1 (TAS1R1) involved in umami perception, may alter taste sensitivity and contribute to unfavorable dietary patterns. The purpose was to evaluate taste preferences in children with different genotypes of the single nucleotide variant rs17492553 in TAS1R1 and explore potential associations with obesity.* ***Materials and methods.*** *This case-control study included 450 children aged 6–18 years: 350 with obesity (main group) and 100 with normal body weight (controls). Taste preferences were assessed using an adapted Food and Beverage Preference Questionnaire and food diaries. Genotyping of rs17492553 in TAS1R1 was performed via next-generation sequencing in the obesity group at CeGaT GmbH (Tübingen, Germany).* ***Results.*** *Among children with obesity, those homozygous for the CC genotype showed stronger preferences for umami (85 vs 31 %; p < 0.00001) and salty tastes (90 vs 69 %; p = 0.02) compared to CT heterozygotes. CC carriers had higher preference scores for nachos/pizza (p < 0.05) and positive correlations with consumption of steak, broth, and cream. For sweet tastes, CC carriers preferred chocolate, whereas CT carriers favored sugar-sweetened carbonated drinks (p < 0.05). No genotype-dependent differences were observed for sour or bitter tastes overall, though CC genotype correlated with higher consumption of certain bitter foods.* ***Conclusions.*** *The rs17492553 variant in TAS1R1 appears to modulate taste preferences in children with obesity. The CC genotype may be associated with heightened preferences for umami- and salty-tasting foods, while the CT genotype is linked to increased consumption of sugar-sweetened beverages. These differences could contribute to adverse dietary patterns.*

Keywords: *children; obesity; single nucleotide variants; taste 1 receptor member 1; taste preferences*

Introduction

The obesity epidemic is a global problem in both developed and developing countries, especially in children. According to experts from the World Health Organization (WHO), more than 0.39 billion children and adolescents were diagnosed with overweight in 2022. It is known that obesity is associated with the development of insulin resistance, type 2 diabetes mellitus, arterial hypertension, dyslipidemia, metabolically associated steatotic liver disease, polycystic ovary syndrome, sleep disorders, depression. One of the many factors that contribute to the excessive development of adipose tissue is unhealthy eating habits and food preferences that change eating behavior [1–4].

The level of sensitivity of taste perception is a key factor in shaping individual diet, eating behavior, and participating in the regulation of appetite [5, 6]. Taste preferences determine the individual nature of food choices, and changes in the ratio of foods in the dietary spectrum significantly affect the overall metabolism of the body [7]. Preferential choice of consumption of high-calorie foods reliably contributes to the development of overweight and obesity. In particular, it has been demonstrated that a decrease in the level of perception of sweet taste is accompanied by an increase in the consumption of high-calorie foods, associated with the development of obesity and insulin resistance [6, 8]. The choice of food products, determined by taste preference, is predetermined by the peculiarities of the functioning of genes encoding certain taste receptors. The perception of taste is determined by the functioning of taste receptors, the proteins of which are encoded by various genes. In particular, the receptors of sweet taste are encoded by the genes *TAS1R2*, *TAS1R3* and *GNAT3*; the taste of umami — by the

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genes *TAS1R1*, *TAS1R3*, *GNAT3*; bitter taste — by the genes *TAS2R1*, *TAS2R3*, *TAS2R38* [9, 10]. Single nucleotide variants (SNVs) of taste receptor genes cause deviations in taste sensitivity patterns and, as a consequence, contribute to the differentiation of eating behavior [7].

Umami taste is a "pleasant savory, meaty, or brothy" taste [11] caused by the presence of free glutamate, adenosine monophosphate, and guanosine monophosphate. It is known that consumption of foods with umami flavor increases the risk of obesity [12]. We have found that the development of metabolically unhealthy obesity (MUO) is associated with the genotype of CT SNV rs17492553 *TAS1R1* gene (unpublished data) [13]. Probably, the change in the level of perception of umami taste, which is associated with variants of the *TAS1R1* gene, can contribute to the deviation of taste preferences that contribute to the development of MUO.

The purpose was to evaluate taste preferences in children with different genotypes of the single nucleotide variant rs17492553 *TAS1R1* and explore potential associations with obesity.

Materials and methods

Ethical approval. The study was carried out in accordance with the ethical standards of the Declaration of Helsinki 2013 and approved by the Human Research Ethics Committee of Dnipro State Medical University (minutes of meeting No. 4 of September 3, 2020). Time of data collection: January 2021 — August 2025.

Informed consent. The parents of all participants provided informed consent.

Study design: case-control genetic association study with cross-sectional elements. The study adhered to the STREGA reporting guidelines [14].

Ethnicity: all children were of Caucasian descent.

Inclusion criteria. Children with polygenic obesity (body mass index (BMI) ≥ 97th percentiles ± 2 SD) 6–18 years old according to the national medical standard "Childhood Obesity" (2022) and WHO recommendations from June 9, 2024 (https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight). Children with monogenic and/or syndromic obesity, pregnancy, acute infections, food allergies or olfactory disorders, and taking medications that affect taste perception *were excluded* [15].

Setting. The study involved 450 children aged 6–18 years. The main group (n = 350) consisted of obese children who were examined in the pediatric endocrinology department of the City Clinical Hospital 6 of the Dnipro City Council. The control group (n = 100) consisted of children with a normal body weight.

Clinical research methods included anthropometric (waist circumference (WC); height; weight; BMI, which was calculated according to the formula: BMI = weight (kg) / height (m)^2 with subsequent analysis of the obtained indicator using the appropriate calculator) [16].

Additional comparison subgroups were formed on the basis of genotyping of the SNV rs17492553 *TAS1R1* gene in children with obesity: the first subgroup — homozygotes for the CC allele and the second subgroup — CT heterozygotes (TT homozygotes had a low frequency of occurrence). To analyze the genotypes of SNV rs17492553 *TAS1R1* gene identified in obese children, the following were used as a control group: RefSeq sequences (NCBI, USA); comparison data with international genomic databases Global Biobank (allele frequency, variant prevalence, clinical associations) [17, 18].

Molecular genetic research

Next-generation sequencing (NGS) was performed in a random sample of 52 obese children in a certified laboratory CeGat (Tübingen, Germany) using Illumina Inc. according to the manufacturer's protocol and in compliance with relevant quality standards. The amount of double stranded DNA was measured using a PicoGreen (Invitrogen Corporation). Allele detection and genotyping were performed in the GenomeStudio genotyping module (Illumina, Inc.). SNVs that were not displayed on autosomal chromosomes were filtered out from the original complete set. SNVs with a minor allele frequency < 0.01 or deviations from the expected Hardy-Weinberg equilibrium ($P < 1.0 \times 10^{-5}$) were removed. Thus, only SNVs that had passed qualitative filtering were left for further analysis.

Bioinformatic analysis — demultiplexing of the sequencing reads was performed with Illumina bcl2fastq (version 2.20). Adapters were trimmed with Skewer, version 0.2.2. DNA-Seq: Trimmed raw reads were aligned to the human reference genome (hg19-cegat) using the Burrows-Wheeler Aligner, BWA — mem version 0.7.17-cegat [19]. ABRA, version 2.18 [20] was used for local restructuring of readings in target regions to improve more accurate detection of indels in the genome.

Assessment of taste preferences

To highlight the prevailing modalities of taste preferences for five most important categories (sweet, sour, umami, salty and bitter), a questionnaire was carried out using an adapted version of IDEFICS (Identification and prevention of Dietary and lifestyle-induced health EFfects In Children and infantS Study) of the Food and Beverage Preference Questionnaire (FBPQ) [21] and the analysis of food diaries. The questionnaire was valid for the surveyed population and consisted of 63 species of individual foods (e.g. banana, spinach), mixed foods (e.g. hot dog, kebab), sauces (e.g. jam, mayonnaise) and drinks (e.g. cola, lemonade). The estimated time to complete the food and drink preference questionnaire was 7 minutes. Each subject assessed their own taste preferences for the corresponding food or drink on a 5-point Likert scale (which was reproduced in the form of "emoticons"), where "1" meant "did not like at all" and "5" meant "very much". Taste preferences were assessed indirectly by having children select familiar foods on a 5-point Likert scale, without the need for verbal explanation of taste categories, which ensured the adaptation of the instrument to the child population and high validity of the methodology.

Food diaries (3-day) were used for qualitative verification of questionnaire data and analysis of the frequency of consumption of individual foods (steak, broth, cream, chocolate, carbonated drinks, etc.).

Statistical processing of the results using parametric, nonparametric methods included: analysis of variance with the calculation of the Student's test (t); Spearman correla-

tion analysis with calculation of Spearman's rank correlation coefficient (r), χ^2 (chi-square) test. The critical value of the level of probability (p) for all types of analysis was taken at the level of $p < 0.05$ (5 %). The sample size was determined a priori using G*Power 3.1.9.7 [22] software to achieve at least 80 % power ($1-\beta = 0.80$, $\alpha = 0.05$, two-tailed). For detecting medium effect sizes (Cohen's $d = 0.5$ for t-tests; $w = 0.3$ for χ^2 tests on proportions) with an allocation ratio of approximately 3.5 : 1 (obese : control), the required total sample size was approximately 300–400 participants, which was met by the current study (N = 450). Statistical processing of the results was performed using Microsoft Excel (Office Home Business 2KB4Y-6H9DB-BM47K-749PV-PG3KT), IBM SPSS Statistics 25.0, Python (scipy.stats.ttest_ind_from_stats) was used (IBM Corp., Armonk, NY, USA).

Results

Average BMI z-score in the main group — 2.31 ± 0.42; in the control group — 0.12 ± 0.68 ($p < 0.001$). The main group included 182 boys (52 %) and 168 girls (48 %); the control group included 52 boys (52 %) and 48 girls (48 %). The groups were comparable in terms of gender distribution ($p > 0.05$).

Of the six identified SNV (rs10864628, rs17492553, rs34160967, rs35118458, rs35375392, rs41278020) *TAS1R1*, only SNV rs17492553 was associated with the development of obesity (SIFT; PolyPhen). Genotyping of SNV rs17492553 (1-6576401 C-T (GRCh38)) *TAS1R1* gene in obese children showed the following distribution: the CC genotype was registered in 15 (28.8 %), the CT genotype in 24 (46.2 %) of children. According to gnomAD.broadinstitute.org [23], to assess the significance between the occurrence of the CC genotype frequency, the χ^2 (chi-square) test was used to match the observed genotypes to the expected Hardy-Weinberg weight level in the reference population (or a direct 2 × 3 association test for the three genotypes). Expected frequencies in non-Finnish European (NFE) composition: CC — 23.5 %; CT — 49.9 % ($p = 0.03$). The frequency of the CC genotype in obese children is higher and the CT genotype is lower than expected in the NFE population according to gnomAD v4 data.

Children with obesity (main group) have significantly lower taste preferences for sweet, bitter, salty and fatty/umami tastes compared to the control group (the control group has stronger preferences). No significant difference was found for sour taste (Table 1).

A comparison of the survey results of obese children, carriers of the homozygous CC genotype and heterozygous CT SNV rs17492553 *TAS1R1* gene, made it possible to establish patterns of genetically associated taste preferences that determine its development (Table 2).

It was shown that obese children with the homozygous CC genotype, in contrast to children with the heterozygous CT genotype SNV rs17492553 *TAS1R1* gene, significantly preferred foods with umami flavor and salty foods. Post-hoc power analysis was conducted using G*Power 3.1.9.7 software. The study achieved high statistical power (> 0.99) for detecting genotype-dependent differences in umami and salty taste preferences (χ^2 test) and sufficient power (> 0.80) for key food-specific preference scores (t-test) at $\alpha = 0.05$.

Peculiarities in preferences for umami-flavored foods

Peculiarities in preferences for umami-flavored foods in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene are presented in Table 3.

Table 1 — Taste preference scores in children with obesity versus controls, M ± m, points

Taste	Main group	Control group	p-value (Student's t-test, Welch)
Sweet	3.36 ± 0.08	3.74 ± 0.07	0.00043
Bitter	2.77 ± 0.15	3.37 ± 0.15	0.00523
Salty	2.95 ± 0.03	3.28 ± 0.04	< 0.00000001
Sour	3.50 ± 0.10	3.80 ± 0.40	0.47
Umami	3.28 ± 0.01	3.60 ± 0.05	< 0.0000001

Table 2 — Basic taste preferences in different genotypes of SNV rs17492553 gene TAS1R1 in children with obesity, %

Taste	Genotype		Probability, p
	CC	CT	
Umami	85	31	0.00001
Sweet	45	54	0.72
Sour	89	92	0.54
Salty	90	69	0.02
Bitter	85	77	0.09

Table 3 — Level of preference for umami-flavored foods in obese children with different genotypes of SNV rs17492553 TAS1R1 gene, points

List of foods with umami taste	Genotypes	
	CC	CT
Broth	3.385 ± 0.213	3.737 ± 0.150
Cream	3.231 ± 0.281	2.368 ± 0.298
Hamburger	2.615 ± 0.368	3.316 ± 0.230
Mashed potatoes	4.077 ± 0.211	4.053 ± 0.179
Kebab	3.615 ± 0.446	3.556 ± 0.345
Sausage (cooked), hot dogs	3.154 ± 0.373	2.947 ± 0.281
Mayonnaise	2.385 ± 0.290	2.050 ± 0.256
Butter	3.000 ± 0.300	2.600 ± 0.255
Milk	4.154 ± 0.274	3.750 ± 0.289
Nachos, pizza	**4.231 ± 0.166***	3.100 ± 0.294
Salami sausage	3.462 ± 0.243	3.263 ± 0.240
Fried potatoes	3.846 ± 0.222	3.500 ± 0.170
Fried chicken	3.538 ± 0.215	3.333 ± 0.256
Steak	4.385 ± 0.290	4.053 ± 0.209
Hot dog	3.462 ± 0.183	3.220 ± 0.222
Chips	2.917 ± 0.452	2.900 ± 0.204
Average preference score for umami flavored foods	3.414 ± 0.165	3.037 ± 0.125

Note (here and in Table 4): * — p < 0.05.

At the same time, according to the data of the point assessment of taste preferences, children of both comparison groups gave equal preference to almost all studied food products with umami taste. The exceptions were nachos and pizza, the level of preference for which was significantly higher in children with the CC genotype SNV rs17492553 *TAS1R1* gene ($p < 0.05$). When conducting a correlation analysis, it was found that preference for consuming such food products with umami taste as steak ($r = 0.44$), broth ($r = 0.39$), cream ($r = 0.34$) is more characteristic of children with the CC genotype SNV rs17492553 *TAS1R1* gene.

Peculiarities in preferences for sweet-tasting food products

The score for the level of preference for sweet-tasting food products in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene is presented in Table 4.

It is interesting that of all the sweet-tasting food products, obese children with the CC genotype SNV rs17492553 *TAS1R1* gene preferred milk chocolate and Nesquik chocolate cones, while those with the CT SNV rs17492553 *TAS1R1* gene preferred sweet carbonated drinks ($p < 0.05$).

According to the data of correlation analysis, carriage of the CC genotype SNV rs17492553 *TAS1R1* gene was associated with a passion for consuming honey ($r = 0.41$), Nesquik chocolate cones ($r = 0.39$), croissants ($r = 0.35$), and cakes ($r = 0.32$).

Peculiarities in preferences for sour-tasting foods

The scores for the level of preference for sour-tasting foods in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene are presented in Table 5.

Taste preferences for sour foods in obese children did not depend on the genotype of SNV rs17492553 *TAS1R1* gene.

Features in preferences for salty foods

The score assessment of the level of preference for salty foods in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene is presented in Table 6.

As with sour-tasting foods, taste preferences for salty-tasting foods in obese children were not associated with SNV rs17492553 *TAS1R1* gene. At the same time, consumption of salted olives and feta cheese was more characteristic of carriers of the CC genotype of SNV rs17492553 *TAS1R1* gene ($r = 0.44$; $r = 0.31$, respectively).

Peculiarities in preferences for bitter-tasting food products

The scores for the level of preference for bitter-tasting food products in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene are presented in Table 7.

According to the obtained data, the spectrum and level of taste preferences for bitter-tasting food products in obese children with different genotypes of SNV rs17492553 *TAS1R1* gene did not differ from each other. At the same time, broccoli ($r = 0.47$), sprouts ($r = 0.39$), spinach ($r = 0.33$), olives ($r = 0.36$) were predominantly chosen as food products by children with the CC genotype of SNV rs17492553 *TAS1R1* gene.

Table 4 — Level of preference for sweet-tasting foods in obese children with different genotypes of SNV rs17492553 TAS1R1 gene, points

Products with sweet taste	Genotypes	
	CC	CT
Jam	3.154 ± 0.274	3.200 ± 0.172
Grapes	3.538 ± 0.215	3.800 ± 0.236
Jelly candies (marmalade)	3.231 ± 0.323	3.500 ± 0.170
Sweet carbonated drinks	2.538 ± 0.268	**3.263 ± 0.200***
Carbonated drinks with sweeteners	2.182 ± 0.352	2.944 ± 0.262
Croissant without filling	3.385 ± 0.350	2.750 ± 0.216
Honey	3.231 ± 0.257	3.350 ± 0.244
Milk chocolate	4.077 ± 0.211	3.850 ± 0.167
Ice cream	4.00 ± 0.160	4.200 ± 0.156
Chocolate bar	**3.846 ± 0.191***	2.950 ± 0.135
Donut	3.375 ± 0.375	3.235 ± 0.219
Beetroot dishes	3.000 ± 0.300	3.000 ± 0.251
Cake	3.538 ± 0.243	3.650 ± 0.131
Chocolate paste	3.545 ± 0.157	3.278 ± 0.240
Chocolate croissant	3.083 ± 0.260	3.300 ± 0.206
Chocolate pudding	3.000 ± 0.309	3.133 ± 0.215
Chocolate balls (Nesquik)	**3.692 ± 0.175***	3.100 ± 0.116
Average score of preferences for sweet-tasting foods	3.394 ± 0.067	3.510 ± 0.176

Table 5 — Level of preference for sour-tasting foods in obese children with different genotypes of SNV rs17492553 TAS1R1 gene, points

Products with sour taste	Genotypes	
	CC	CT
Natural yogurt	3.923 ± 0.239	3.500 ± 0.202
Juice	3.385 ± 0.266	3.950 ± 0.170
Average preference score for sour-tasting foods	3.562 ± 0.194	3.514 ± 0.163

Table 6 — Level of preference for salty foods in obese children with different genotypes of SNV rs17492553 TAS1R1 gene, points

Products with salty taste	Genotypes	
	CC	CT
Salt	2.077 ± 0.265	2.850 ± 0.274
Salted olives	3.462 ± 0.332	2.944 ± 0.347
Salty cookies	3.231 ± 0.281	3.300 ± 0.164
Salted nuts	2.923 ± 0.383	2.700 ± 0.242
Salty sticks	2.538 ± 0.369	2.737 ± 0.214
Salted pistachios	2.583 ± 0.417	3.105 ± 0.275
Feta	3.769 ± 0.257	3.533 ± 0.307
Average preference score for salty-tasting foods	2.949 ± 0.235	2.856 ± 0.144

Table 7 — Level of preference for bitter-tasting food products in obese children with different genotypes of SNV rs17492553 TAS1R1 gene, points

Products with bitter taste	Genotypes	
	CC	CT
Broccoli	3.000 ± 0.408	3.158 ± 0.279
Grapefruit	3.000 ± 0.358	3.350 ± 0.254
Coffee	3.077 ± 0.400	3.474 ± 0.290
Lettuce	3.091 ± 0.476	3.526 ± 0.193
Olives	3.385 ± 0.350	3.000 ± 0.352
Sprouted sprouts	3.300 ± 0.473	2.364 ± 0.338
Asparagus	3.167 ± 0.490	3.077 ± 0.348
Red cabbage	2.846 ± 0.317	3.118 ± 0.256
Spinach	2.846 ± 0.390	3.000 ± 0.214
Average preference score for bitter-tasting foods	2.846 ± 0.213	2.761 ± 0.191

Discussion

The *TAS1R1* gene is located on the short arm p36.23 of chromosome 1 and encodes the T1R1 protein, which is involved in umami taste recognition [24]. Single nucleotide variants rs17492553 *TAS1R1* gene are associated with changes in the level of perception of umami-tasting foods. Homozygotes of TT SNV rs17492553 *TAS1R1* gene are characterized by a lower level of sensitivity of T1R1/T1R3 receptors to monosodium glutamate [25]. We have shown that the CT SNV rs17492553 genotype *TAS1R1* gene is associated with the development of MUO, which is probably due to changes in taste preferences [13].

It was found that obese children with the homozygous genotype CC SNV rs17492553 *TAS1R1* gene prefer food products with umami and salty taste to a greater extent, in contrast to heterozygous carriers of the genotype CT SNV rs17492553 of the *TAS1R1* gene. The three basic tastes of sweet, salty, and umami are thought to stimulate food intake and contribute to obesity in the context of excess food intake. Glutamate-mediated activation of adenosine monophosphate deaminase 2 (AMPD2) in brain and liver cells via AMP-activated protein kinase has been shown to cause both increased lipogenesis and insulin resistance. At a metabolic level, increased umami intake — particularly from free glutamate — may engage deleterious pathways linking taste preference to adiposity. Glutamate uptake in hepatocytes can fuel nucleotide synthesis, followed by degradation via enzymes such as AMPD2, culminating in elevated uric acid production. Hyperuricemia, in turn, promotes *de novo* lipogenesis, mitochondrial oxidative stress, and insulin resistance, mechanisms implicated in obesity and metabolic syndrome. These pathways parallel those activated by fructose and high salt intake, suggesting convergent metabolic risks from taste buds. Although direct evidence tying rs17492553 to AMPD2 dysregulation is lacking, the genotype-dependent umami preference observed here provides a plausible upstream trigger, warranting functional studies (e.g., eQTL analysis or receptor binding assays) to elucidate causality [26]. We believe that the maintenance of the C allele of SNV rs17492553 *TAS1R1* gene is associated with an increased risk of obesity, and the heterozygous CT genotype contributes to the development of metabolic disorders. It cannot be ruled out that the CT genotype of SNV rs17492553 *TAS1R1* gene is accompanied by a change in the activity of some signaling pathways involved in the development of insulin resistance and/or other metabolic complications.

Carriers of the CC genotype SNV rs17492553 *TAS1R1* gene were characterized by a passion for consuming honey and chocolate products. However, the consumption of these food products, which may have been influenced by parental control of sweet food intake. Whereas carriers of the CT genotype SNV rs17492553 *TAS1R1* gene preferred sweet carbonated drinks. It is known that sweet carbonated drinks are an unfavorable dietary factor that contribute to the development of metabolic disorders. According to the National Health and Nutrition Examination Survey, almost 61 % of children consume sweet carbonated drinks daily, which carry a risk of developing metabolic disorders. The main mechanism of action of sweet carbonated drinks that cause obesity and metabolic disorders is considered to be a decrease in the feeling of satiety and compensatory suppression of energy consumption activity [27–30].

Of interest is the preference for bitter-tasting foods such as broccoli, sprouts, spinach, and olives by children with the CC genotype SNV rs17492553 *TAS1R1* gene. At the same time, it is known that consumption of cruciferous vegetables is associated with a low probability of developing obesity and metabolic disorders [31]. Excitation of bitter taste receptors (TAS2R) inhibits appetite, induces the release of antimicrobial peptides such as α-defensin 5 and islet-derived protein 3α, and indirectly affects the gut microbiome [32–34].

It is likely that avoidance of bitter-tasting foods by children with the CT genotype SNV rs17492553 *TAS1R1* gene may be one of the factors that contributes to the development of metabolic disorders.

The identified genotype-specific features can be used for individualization of diet therapy (replacement of umami products in CC patients, replacement of licorice drinks in CT patients) and development of personalization preventive programs.

Limitations. Children with polygenic obesity exhibit lower overall taste preferences compared with normal-weight peers. The rs17492553 variant in *TAS1R1* appears to modulate preferences in obese children, with CC associated with umami/salty foods and CT with sugar-sweetened beverages. These genotype-dependent patterns may influence dietary behavior and obesity maintenance. At the same time, our study has certain limitations: case-control genetic association study with cross-sectional elements, lack of data on objective sensory testing, possibility of parental influence, limited extrapolation to other populations. Further, population-wide longitudinal studies are needed to establish a causal relationship in more detail.

Conclusions

Children with polygenic obesity exhibit lower overall taste preferences compared to normal-weight peers.

The rs17492553 variant in the *TAS1R1* gene likely modulates preferences in obese children, could contribute to adverse dietary patterns, particularly increased consumption of umami- and salty-tasting foods (CC genotype) or sugar-sweetened beverages (CT genotype).

These genotype-dependent patterns may influence eating behavior and the maintenance of obesity.

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Асоціація варіанта гена *TAS1R1* rs17492553 зі смаковими уподобаннями при дитячому ожирінні

Резюме. ***Актуальність.*** Генетичні варіації генів смакових рецепторів, включно з представником 1 смакового рецептора 1 (taste 1 receptor member 1 — *TAS1R1*), що бере участь у сприйнятті умамі, можуть змінювати смакову чутливість та обумовлювати формування несприятливих харчових моделей. ***Мета:*** оцінити смакові вподобання дітей із різними генотипами однонуклеотидного варіанту rs17492553 *TAS1R1* та вивчити потенційні зв'язки з ожирінням. ***Матеріали та методи.*** Дослідження типу «випадок — контроль» включало 450 дітей віком від 6 до 18 років: 350 з ожирінням (основна група) та 100 з фізіологічною масою тіла (контрольна група). Смакові вподобання оцінювали за допомогою адаптованої Анкети щодо вподобань у їжі та напоях і харчових щоденників. Генотипування однонуклеотидного варіанту rs17492553 *TAS1R1* проводили за допомогою секвенування наступного покоління в групі з ожирінням (CeGaT GmbH, Тюбінген, Німеччина). ***Результати.*** Серед дітей з ожирінням гомозиготні за генотипом CC демонстрували сильніші вподобання до умамі (85 проти 31 %; $p < 0,00001$) та солоного смаків (90 проти 69 %; $p = 0,02$) порівняно з гетерозиготами CT. Носії генотипу CC мали вищі показники переваги до начос/піци ($p < 0,05$) і позитивну кореляцію зі споживанням стейків, бульйону й вершків. Щодо солодкого, носії генотипу CC віддавали перевагу шоколаду, тоді як носії генотипу CT — підсолодженим цукром газованим напоям ($p < 0,05$). Загалом не спостерігалося генотипзалежних відмінностей для кислого або гіркого смаків, хоча генотип CC корелював із більшим споживанням деяких гірких продуктів. ***Висновки.*** Варіант rs17492553 *TAS1R1*, ймовірно, модулює смакові уподобання в дітей з ожирінням. Генотип CC може бути пов'язаний із підвищеною схильністю до умамі та солоних продуктів, тоді як генотип CT — зі збільшенням споживання підсолоджених цукром напоїв. Ці відмінності обумовлюють формування несприятливих харчових моделей.

Ключові слова: діти; ожиріння; однонуклеотидні варіанти; представник 1 смакового рецептора 1; смакові вподобання