The spread of obesity has become pandemic, and it is expected that during 2021 the number of obese children and adolescents (5 to 19 years) will be 158 million [World Obesity Federation, 2020]. There is an alarming trend towards an increase in the incidence of metabolically unhealthy obesity (MUO), especially among children [Williamson K. et al., 2020]. Genetically determined violation of taste preferences leads to an inversion of the perception of tastes and overeating, distorting the homeostatic feedback of the peripheral energy status with hedonic centers, causing obesity.

In our work, the importance of genetic variants of the taste 2 receptor member 38 (TAS2R38) is presented as the most significant predictor of metabolically unhealthy obesity among genes responsible for taste formation, according to GWAS data.

Materials and methods

90 obese children aged 6-18 years were examined. The main group (n = 52) was represented by children according to the criteria of the consensus of the International Diabetes Federation (IDF) with MUO. The control group (n = 38) was formed by patients with MHO. To identify the prevailing modalities of taste preferences in the 5 most important categories (sweet, sour, fatty/umami, salty, and bitter), a survey was conducted using an adapted version of the approved and used in the study IDEFICS (Identification and prevention of Dietary and lifestyle-induced health Effects In Children and infantS Study) of the Food and Beverage Preference Questionnaire (FBPQ) and an analysis of food diaries. To determine the SNVs of the TAS2R38 gene in the formation of metabolically unhealthy obesity, we used the method of complete genomic sequencing on the Illumina platform in a certified laboratory CGCat (Tübingen, Germany), followed by bioinformatics analysis.

For statistical processing of the obtained results the variational analysis with definition of the reliability of Student, the relation of chances, the correlation analysis of Spearman, calculation of the prognostic factor is used.

Table 1. SNVs of the TAS2R38 gene (missense mutation) and CADD

<table>
<thead>
<tr>
<th>SNP</th>
<th>Allele 1</th>
<th>Allele 2</th>
<th>CADD Score</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>TAS2R38 C05374174 T/C</td>
<td>0/0000</td>
<td>0/10000</td>
<td>9.9999</td>
<td>OR 1.019; 95%DI 0.958-10.90</td>
<td></td>
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</table>

To study the role of SNV gene TAS2R38 in the formation of MUO in children 6-18 years old.

The level of the average value of preference for bitter food in the main group was 2.75 ± 0.15 points, while in the control group 3.24 ± 0.14 points and the Student’s test in the compared populations was 2.39, with p < 0.02. There were no statistically significant differences in preferences for sour, salty and fatty/umami flavors in the comparison groups, p > 0.05. Analysis of food diaries in children showed a positive correlation between daily non-consumption of fresh vegetables and the formation of MUO (r = -0.32) with a prognostic factor of 2.7; p < 0.05.

SNVs of the TAS2R38 gene (missense mutation) were diagnosed by complete genomic sequencing (Table 1). The probability of detecting a geronyzotic variant of the C/G genotype rs713598 of the TAS2R38 gene in the main group was 1.75 times higher than in the control group of obese children, p = 0.05 (Table 2, Fig. 1).

Conclusion

Decreased taste preferences for bitter foods increase the risk of developing MUO in children.

The C/G genotype rs713598 has the greatest association among the SNV of the TAS2R38 gene detected by us with the formation of metabolically unhealthy obesity.

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Authors’ contributions

AA was responsible for the idea and study design, looked over the articles, extracted the data, and interpreted bioinformatics analysis data. DR provided the collection of biological material using dried blood spot shipping kit, AN analyzed the data and interpreted it. Both authors reviewed the paper and approved the final manuscript.

Conflict of Interest: The authors declare that they have no conflict of interest.