GENDER AND PUBERTAL TENDENCIES OF PLASMA LEPTIN AND DOPAMINE LEVELS DEPENDING ON TAQIA DRD2 GENE POLYMORPHISM IN THE DIFFERENT PEDIATRIC OBESITY CLASSES

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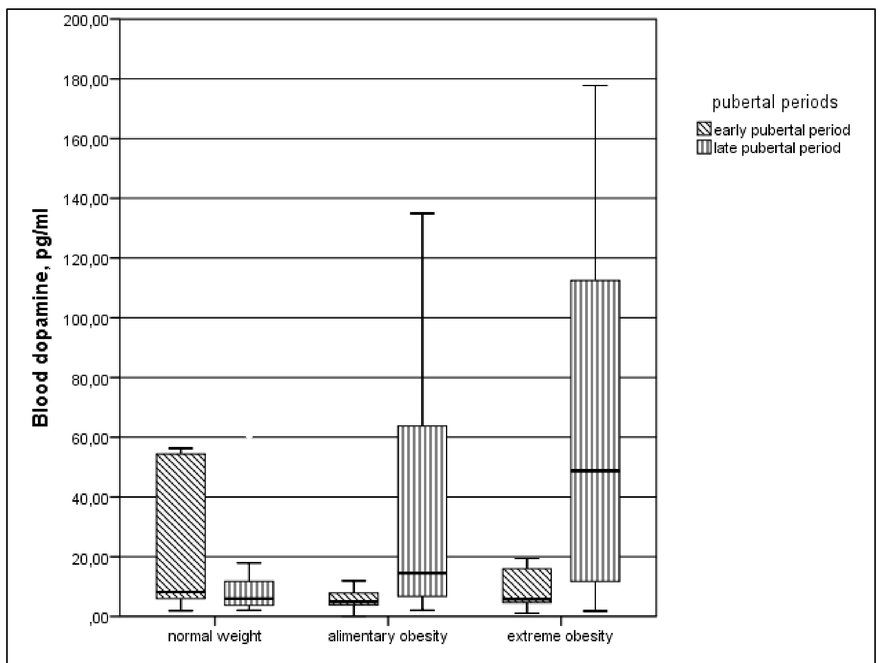
OBJECTIVES

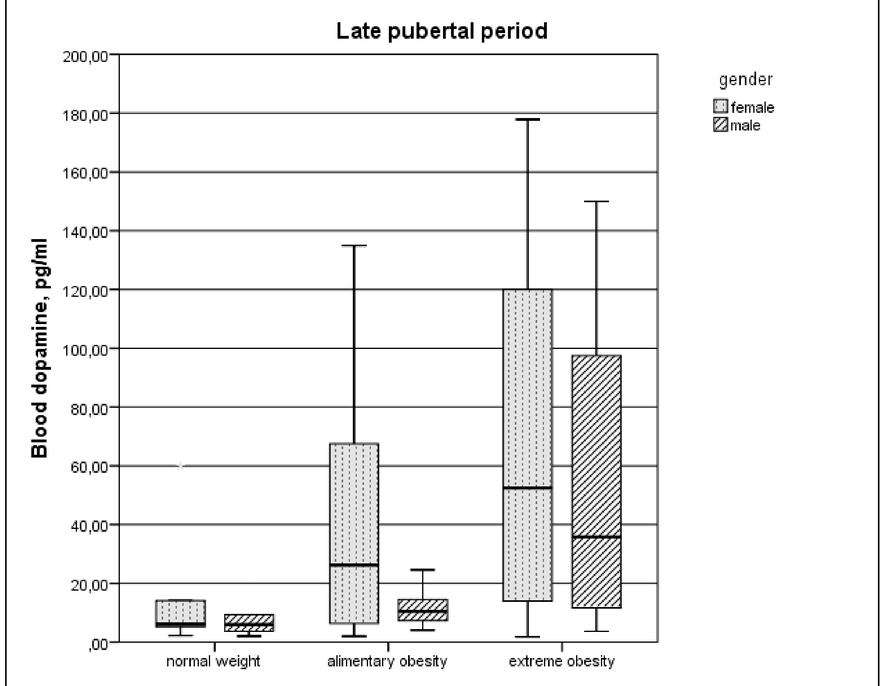
Background: The TaqI (rs1800497) polymorphism of the Dopamine Receptor type 2 (DRD2) gene allele has been commonly related to increased ad lib food intake, weight gain, high risk for overeating and extreme obesity. [1,2,3].
In term of this we supposed evaluate the influence of DRD2 gene TaqIA polymorphism, plasma
leptin and dopamine concentrations to obesity development in children with alimentary, extreme obesity and normal weight, depending on gender and pubertal development stage.

METHODS

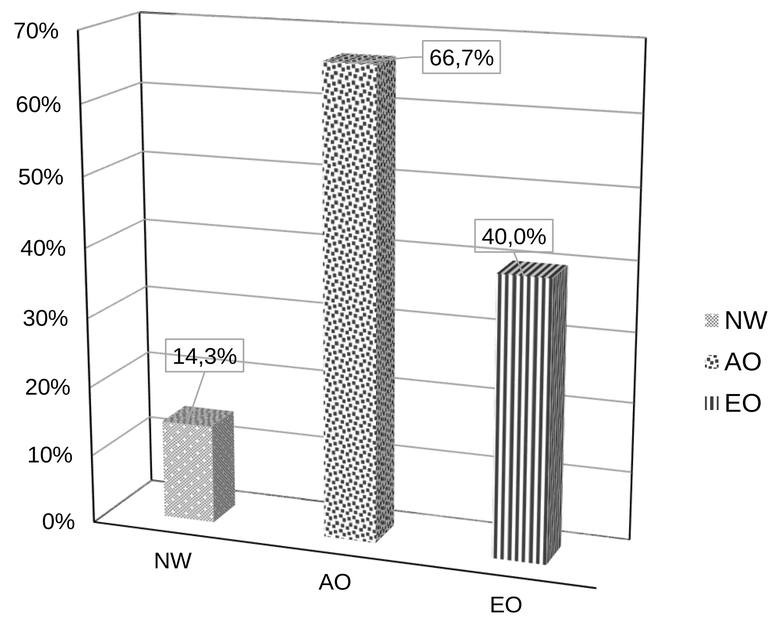
223 children aged from 11 to 17.9 years were involved in the retrospective cross-sectional study, 179 of them were randomly genotyped in the TaqIA of DRD2 gene and blood dopamine (BD) and leptin (BL) concentrations were detected. Children were split up in terms of BMI SDS into three groups: the first — normal weight (NW) (±1, n=30), the second — alimentary obesity (AO) (BMI≥+2, <+4, n=86), the third — extreme obesity (EO) (≥+4, n=107). These groups were split off by gender (f/m, n=112/111) and pubertal periods (preprubertal – the 1st Tanner stage; early pubertal – the 2nd + 3rd Tanner stage; late pubertal – the 4th+5th Tanner stage) Statistical analysis were performed by means of SPSS 21.0 ($p < 0.05$) with data shown as a median and interquartile range (25 – 75).



Picture 1. Dopamine levels boys with normal weight, alimentary and extreme obesity depends on pubertal periods



Picture 2. Blood dopamine levels in boys and girls with normal weight, alimentary and extreme obesity



A1A1 genotype rate of the DRD2 gene rs1800497 polymorphism

Picture 3. A1A1 genotype rate of the DRD2 gene rs1800497 polymorphism in boys with obesity and normal weight

RESULTS

Children with extreme obesity had increased BD levels 31.88 (7.82 – 99.38) pg/ml in comparison with normal weight kids (6.28 (4.57 – 26.54) pg/ml), p=0.002; and patients with AO (10.44 (4.80 – 46.88) pg/ml), p=0.008. The same pattern of BD had boys with EO (31.88 (9.69 – 93.75) pg/ml) compared to NW kids (6.46 (3.73 – 26.54) pg/ml), p=0.002; and patients with AO (7.83 (4.47 – 13.04) pg/ml), p=0.008.
Early pubertal male patients with AO had the lowest BD 4.84 (3.54 – 9.32), that were significantly differ than NW children (8.20 (5.96 – 56.25) pg/ml); p=0.046; and EO ones (5.78 (4.62 – 19.38) pg/m); p=0.002 (picture 1). NW late pubertal boys had minimal BD levels (5.96 (3.73 – 14.16)) pg/ml with gradual BD raising in AO (14.53 (6.55 – 65.63) pg/ml), p=0.01; and EO groups (48.75 (11.68 – 114.25) pg/ml), p=0.0001 (picture 2).
Early pubertal NW children had diminished L levels (4.84 (3.54 – 9.32) ng/ml) relatively to AO (24.1 (14.75 – 31.32) ng/ml), p=0.0001; and EO patients (48.75 (21.31 – 69.6) ng/ml), p=0.0003. NW late pubertal children were characterized by minimal L levels (4.77 (2.43 – 11.06) ng/ml) with the next gradual raising in the AO (22.34 (12.17 – 40.48) ng/ml, p=0.0002) and EO groups (27.8 (17.52 – 45.99) ng/ml, p=0.0001) and significant differences between these children groups (p=0.03).
Children with AO and EO had raised A1 TaqI DRD2 allele frequencies: in 50% and 40% equally in contrast with NW patients (10%) (p=0.015). AO boys had more frequent TaqI A1 polymorphism of DRD2-gene (66.7%) compared to EO (23.8%) and NW boys (14.3%), p=0.05.

CONCLUSIONS

Patients with EO had enlarged prevalence of the high BD concentrations related to AO and NW children, who had decreased and low these neuropeptid levels (p = 0.002; p = 0.008). A1/A1 genotype was more prevalent in AO as opposed to and EO patients and NW children (p = 0.05).

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