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EXPRESSION OF OBESITY RELATED GENES AND LEPTIN SERUM LEVELS ARE DECREASED IN TURKISH SCHIZOPHRENIA PATIENTS, CASE-CONTROL STUDY

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Antipsychotic medications can induce metabolic abnormalities (weight gain) in schizophrenia (SCH). Leptin receptor (LEPR), leptin (LEP) and peroxysome proliferator-activated receptor g2 (PPARg2) are the potential genetic determinants which might be liable of the metabolic dysregulation. The aim of this study was to evaluate the effect of LEP c.-2548G>A, LEPR p.Q223R polymorphisms and the impact of mRNA levels of LEP, LEPR, and PPARg2 along with the serum leptin levels on metabolic adversities in SCH patients (n=132) and controls (n=114).

Methods: Metabolic profiles, LEP, LEPR gene polymorphisms and the gene expressions of LEP, LEPR and PPARg2 were studied in SCH patients and controls.

Results: BMI, cholesterol and fasting glucose levels were higher in SCH patients compared to controls. LEP c.-2548 (GA+AA) genotypes were two fold lower in SCH patients versus controls ($p < 0.05$), and no significant difference was observed in LEPR p.Q223R genotypes. Interestingly, leptin serum levels were lower in SCH patients compared to controls ($p < 0.05$). Leptin, leptin receptor and PPARg2 gene expressions were found to be decreased in SCH patients compared to controls ($p < 0.001$, $p < 0.001$ and $p \leq 0.05$, respectively).

Conclusion: Leptin receptor, leptin and PPARg2 genes could be potential risk factors in developing metabolic adversities in SCH.

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