Dear Editor,

According to the Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition), obsessive-compulsive disorder (OCD) is a debilitating neuropsychiatric condition. Although there is strong evidence of genetic contribution, susceptibility locus has not yet been identified. The serotonin system plays an important role in the pathogenesis of OCD as demonstrated mainly by the efficacy of serotonin-reuptake inhibitors. Genetic variations in the serotonin transporter gene (SLC6A4) may be involved in the vulnerability to psychiatric disorders, including OCD. A common polymorphism located in the promoter region (5-HTTLPR) and consisting of either the insertion or deletion of 44 base pairs has been suggested to be associated to OCD. The 5-HTTLPR has two variants: the long variant (L), which has been reported to generate more gene expression than the short one (S). Moreover, the S allele is less efficient in the uptake of serotonin. More recently, it has been suggested that 5-HTTLPR could be functionally triallelic (La, Lg, and S). The L allele with a common G substitution (Lg) has shown to have lower expression than the La and nearly equivalent expression compared to the S allele.

Although several researchers have studied OCD and 5-HTTLPR, their findings are controversial and inconclusive. We sought to investigate this association in a sample of 92 Brazilian-Caucasian OCD patients and 115 matched healthy controls. The study was approved by the Ethics Committee of the Hospital das Clínicas of the Universidade Federal de Minas Gerais (UFMG) and all participants were required to sign informed consent forms.

Genomic DNA was extracted from venous blood samples and the polymorphism was analyzed as described previously. We used a chi-square test for the analysis of the differences between OCD patients and controls with regards to allele and genotype frequencies by grouping S- and/or Lg-carriers in view of the fact that they act in a nearly dominant way and have equivalent expression.

The genotypic distributions were in Hardy-Weinberg equilibrium. The La allele was associated to OCD (OR = 1.36; 1.15 < OR < 1.87; p = 0.002) but we did not find differences in genotypic distribution (p = 0.54).

Our results are similar to those found in an important recent study that associated the La allele to OCD. Although several previous results had been inconsistent, this association may be significant only in certain subgroups, such as that of Caucasian patients. Moreover, earlier biallelic studies have classified the Lg variant as the long-form of the allele, a fact which may partially account for these contradictory results.

The fact that our sample consisted of Caucasian patients may account for the results achieved. Although our sample was comprised only of self-defined Caucasian-Brazilian individuals, race (as determined by self and/or clinical evaluation) is a poor predictor of ancestry in Brazil and, therefore, it cannot be ruled out that a certain bias towards ethnic stratification did exist.

More studies with triallelic model and more homogeneous groups are required to clarify the contribution of genetics to OCD.

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Dear Editor,

Psychiatric disorders may be secondary to endocrinological diseases what may be represented by the association between thyroid gland function and behavior manifestations. Thyroid hormones are widely arranged in brain and may influence some psychic symptoms, mainly by changing neurotransmitters’ release.

We would like to report a case of a high school student, 17 years old, who presented with agitation, insomnia, tremors, disinhibited behavior, exalted mood, grandiose delusion and auditory hallucinations for three months. He had never been in a psychiatry service before. His relatives reported no family history of psychiatric disorder. In admission, the patient exhibited an expansive attitude, dysphoria and accelerated thoughts. He reported diary cannabis abuse for the last year. After one month using risperidone 2mg every day the patient returned home with remission of all psychotic symptoms, but still presented exalted mood. His follow-up in the outpatient clinic evidenced tremors, hypertension (AP 140x100mmHg) and tachycardia (CF 110bpm) for two months. Laboratory tests showed elevated thyroxin and reduced TSH. The patient received propiltiouracil and propranolol and after 45 days he presented remission of physical and psychic symptoms, besides stopping cannabis use.

There have been reports showing association between hyperthyroidism and cannabis abuse as well as with psychiatric disorders. Yet, there has been no association between cannabis use and hyperthyroidism. Animal models, however, demonstrated that acute cannabinoid administration disrupts many hormone systems, including suppression of gonadal steroids, growth hormone, prolactin and thyroid hormones, besides the hypothalamic-pituitary-adrenal axis. These effects are caused by endogenous cannabinoid receptors located close to and inside the hypothalamus.

Although all the consequences of chronic cannabis use in human endocrine system are still unclear, there is evidence that in thyrotoxicosis the number of binding sites for catecholamines increases. This report shows the importance of laboratory tests for thyroid diseases in patients with mood disorders. Establishing a differential diagnosis is essential to conduct the best psychopharmacological treatment aiming at the best treatment.

References