COMT Gene Polymorphisms and Fibromyalgia

To the Director: We have read with interest the article by García Fructuoso et al. about the possible implication of the COMT gene in the susceptibility and the severity of fibromyalgia (FM). We do not share some aspects of the employed methodology and the relationship between these findings as well as the interpretations of the authors, and we wish to do the following commentary. The supposed relationship of the COMT gene polymorphism in multiple processes is a fact so thoroughly studied as discussed. In part, the origin of the absence of objective findings is simple: to establish an unequivocal relationship between a determined genotype and a phenotype, and the latter has to be concrete, well established and with the least possible amount of environmental interference. Obviously, FM and a series of psychiatric illnesses, in which a possible relationship with the Val158Met polymorphisms have been observed, are not characterized as being concrete, objective and free of environmental interference. These polymorphisms have even been observed in chronic fatigue syndrome (with a percentage superior to 70% of patients that have generalized pain and more than 11 positive ACR trigger points) and in “healthy” people with an abnormal pain process, smoking, and alcoholism as well. These are some of the reasons due to which, before investigating possible genetic associations with certain processes, we must delve deep into establishing with security that we are confronted with a unique illness and not with a variable group of symptoms that can have different causes. In this sense, the analysis of the comorbidities that the studied patients presented (depression or anxiety, stressful events, drug or alcohol consumption) could be explained as part of the observed findings. FM is one of the main causes of chronic generalized pain and, in spite of a recent study, the observed findings. FM is one of the main causes or alcohol consumption could be explained as part of the analysis of the comorbidities that the studied patients presented (depression or anxiety, stressful events, drug) implying an evident selection bias. Regarding the establishment of the severity of FM through FIQ, whose objective seems to be only judicial, as commented in the article, we consider that it could induce error because it employs subjective variables and, logically, the sum of subjectivities does not become an objective and annulls its value to establish or demonstrate a determined degree of limitation. In summary, even though the studies of genetic association are an invaluable tool for gaining knowledge into the pathogenesis of certain illnesses with clearly established phenotypes, in processes as complex as FM it is fundamental that the study of large samples that permit a subgroup analysis in which multiple co-morbidities that, with high frequency, are present in these patients, as well as an exhaustive examination of their history in which documentation of existing past or present stressful episodes, drug consumption, addictive behavior, work situation, etc. Otherwise we cannot conclude we are faced with genetic association that, if true, could open new and interesting doors in research.

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References
