A common microdeletion affecting a hippocampus- and amygdala-specific isoform of tryptophan hydroxylase 2 is not associated with affective disorders

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OBJECTIVES: Copy number variants (CNVs) have been shown to affect susceptibility for neuropsychiatric disorders. To date, studies implicating the serotonergic system in complex conditions have just focused on single nucleotide polymorphisms (SNPs). We therefore sought to identify novel common genetic copy number polymorphisms affecting genes of the serotonergic system, and to assess their putative role in bipolar affective disorder (BPAD) and major depressive disorder (MDD). METHODS: A selection of 41 genes of the serotonergic system encoding receptors, the serotonin transporter, metabolic enzymes and chaperones were investigated using a paired-end mapping (PEM) approach on next-generation sequencing data from the pilot project of the 1000 Genomes Project. For association testing, 593 patients with MDD, 1,145 patients with BPAD, and 1,738 healthy controls were included in the study. RESULTS: PEM led to the identification of a microdeletion in the gene encoding tryptophan hydroxylase 2 (TPH2), affecting an amygdala- and hippocampus-specific isoform. It was not associated with BPAD or MDD using a case-control association approach. CONCLUSIONS: We did not find evidence for a role of the TPH2 microdeletion in the pathoetiology of affective disorders. Further studies examining its putative role in behavioral traits regulated by the limbic system are warranted.
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