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The revised dopamine hypothesis: implications on our understanding of psychotic symptomatology

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A complex dopaminergic dysregulation may underlie the neuro-chemical pathogenesis of schizophrenia. How this dopaminergic dysregulation arises in vulnerable individuals is unknown. An indirect insight into dopaminergic regulation can be achieved by examination of COMT (catechol-O-methyltransferase) genotyping, variants of which, have differential impact on prefrontally mediated dopaminergic neurotransmission. As part of the ASPIS project (Athens Study of Psychosis Proneness and Incidence of Schizophrenia), we have genotyped 547 apparently healthy young males who also completed a computerised neurocognitive battery and dimensional self rated schizotypy questionnaires. We hypothesized that negative schizotypal personality traits, which often in retrospect proceed the onset of overt positive psychotic symptoms, will be associated with the high activity COMT allele, indicative of a relative hypodopaminergia in the PFC. We find a clear effect of COMT genotyping on schizotypy self rated measures and in particular a strong association between the high activity COMT allele and the negative schizotypy factor derived from Confirmatory Factor Analysis of the schizoypy scales. We propose, that the negative-social deficits in the population are associated with a trait prefrontal dopaminergic hypoactivity, on which later on in the process of the disease, a subcortical hyperdopaminergic state is superimposed, coinciding with the development of positive psychotic symptoms.