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ORBITOFRONTAL CORTEX AS A BRAIN BIOMARKER OF SUICIDE VULNERABILITY IN MOOD DISORDERS: ROLE OF VALUATION PROCESSES AND SEROTONERGIC GENES

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Background: The epidemiology of suicidal behaviours (SB) reflects our lack of understanding of these complex behaviours, as well as our difficulty to predict them. It is now admitted that vulnerability to SB relies on the interaction between genetic predisposition and environmental factors. Serotoninergic genes and orbitofrontal cortex (OFC) dysfunction are potential biomarkers for SB.

Methods: Three groups of euthymic participants were included: 1) suicide attempters—individuals with a past history of both major depressive episode and suicidal behaviour; 2) affective comparison subjects—individuals with a past history of major depressive episode but no history of suicidal acts; 3) healthy comparison subjects—individuals with no past history of any DSM-IV axis I diagnosis. We compared neural activity using functional MRI while performing face processing (happiness, anger, sadness visualisation) and decision-making tasks (Iowa Gambling Task). We characterized of our sample for five genotypes: 5HTTLPR, TPH-1 rs1800532 , TPH-1 rs7933505, MAOA u-VNTR, and TPH2 rs7305115. We performed *a priori* ROI analysis focused on OFC.

Results: We found decreased activation during risky vs. safe choices and increased activation during wins vs. losses in left OFC in suicide attempters in comparison to controls. Left lateral OFC activation was correlated with IGT net score in suicide attempters. We found an increased activation of right lateral OFC cortex in suicide attempters in comparison to affective controls when viewing angry but not happy or sad faces vs. neutral faces. Lateral OFC activation was modulated by 5HTTLPR polymorphisms.

Conclusion: Based on OFC dysfunction, suicide attempters may overevaluate social negative signs and underevaluate deleterious consequences of short-term rewarded choices.

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