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Functional Underpinnings of Emotional Processing

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Major depression is associated with abnormal self-processing (i.e self criticism, rumination) characterized by increased activity in cortical midline structures.

Among these structures the Medial Prefrontal Cortex (MPFC) has focused research attention.

Connected with several cortical and sub-cortical structures,

the MPFC activity may predict clinical outcome of depressed patients treated with antidepressant. We have recently showed

that the level of activity of MPFC before treatment predict clinical remission at 24 weeks in patients receiving antidepressant.

Moreover the level of MPFC activity was not affected by antidepressant, suggesting that this region may constitute a trait marker of major depression.

Recent data emphasize that impaired self processing and MPFC may be observed in subjects with genetic vulnerability for affective disorders.

Thus we will present data showing that subjects with the short allele S of the 5-HTTLPR promoter gene have increased activity of MPFC during self processing. Consistent with gene x environment interaction hypothesis,

the degree of Amygdala activity in S careers was modulated by exposure to life stress events and self processing.

Moreover we will discuss the relationships between self-esteem, social stress and the changes in MPFC activity related to exposure to social threat.

Overall our talk will emphasize the role of MPFC in the pathophysiology of depression

and the multiple determinants (genetic, environmental exposure, social context) of its impairment in affective disorders.