

disruption of PPI mediated by 5-HT1A receptor stimulation. The action of haloperidol and raclopride suggests a major involvement of dopamine D2 receptors in this effect, possibly downstream from the initial serotonergic stimulation. The action of aripiprazole could be mediated by its partial agonist properties at 5-HT1A receptors or its dopamine D2 blocking properties.

Longitudinal increases in gamma-phase synchrony contrasts with progressive gray matter atrophy in first-episode schizophrenia

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Background: Our integrative neuroscience model of first-episode schizophrenia (FES) emphasizes a dysfunction in the coordinated neural activity required for selective attention in the disorder. This study investigated the longitudinal changes in neural connectivity (assessed by means of 40-Hz gamma synchrony) and neuroanatomy [assessed by magnetic resonance imaging (MRI)] exhibited by patients with FES.

Method: Twenty-three FES patients underwent an EEG recording in response to an auditory oddball task, both at baseline and 2–3 years subsequently. Gamma-phase synchrony was extracted from the EEG signal for L/R frontal, temporal and posterior brain regions. Thirteen of these patients also underwent an MRI scan at baseline and follow-up, and an automated masking procedure was used to calculate the GM volumes of the analogous cortical regions. A 2 × 6 ('time' × 'region') repeated-measures ANOVA was used for statistical analysis.

Results: An inverse relationship was observed between the longitudinal changes in gamma synchrony and the longitudinal changes in GM volume. While the patients with FES lost significant frontal and parietal GM over the follow-up interval, they also showed a corresponding increase in posterior gamma-phase synchrony.

Conclusions: These results indicate that while gamma-phase synchrony increases over the initial years of illness in patients with FES, GM volume decreases in corresponding cortical regions. Given the role that gamma-phase synchrony has been proposed to play in the integration of discrete perceptual events, these findings support the idea that schizophrenia is caused by a dysfunction in neural connectivity.

Late-onset bipolar disorder: preliminary results from Sydney

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Background: Previous studies have suggested that there may be bipolar disorder subtypes according to age at onset (AAO), including a late-onset (LO) group with onset in the fifth decade of life. LO presentations may be associated with greater cerebrovascular disease and increased neuropsychological deficits. Different AAO may also explain some of the genetic heterogeneity associated with bipolar disorder.

Method: We have commenced recruitment of participants aged 40 years and over, with the aim of assessing early-onset bipolar I, late-onset bipolar I and healthy control groups. Assessment tools included the following: sociodemographic and disability questionnaires, SCID, HDRS, YMRS; cerebral magnetic resonance imaging scan; a neuropsychological battery and venepuncture for genetic testing.

Results: Preliminary results for the first 15 participants with bipolar disorder (mean age 53.9 years, range 46–66 years, 66% women) have shown an average latency of 11 years between the first affective episode and the first episode of mania, and of 17 years before a formal diagnosis of bipolar disorder. There was a high rate of comorbidity with anxiety disorders. Contrary to study hypotheses, the participants tended to be relatively physically healthy with minimal vascular disease burden. Neuropsychological assessment of euthymic participants showed no differences in language and memory, but significant differences in visuospatial organization and self-monitoring tasks.

Conclusions: These preliminary results suggest deficits in frontal executive dysfunction in this sample of older bipolar participants. The recruitment of a relatively young and ambulatory sample may have led to the finding of minimal vascular disease.

SSRI use and bone mineral density in women with a history of depression: Geelong Osteoporosis Study

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Background: Selective serotonin reuptake inhibitors (SSRIs) are a first-line treatment for depression.