of the MMN deficit in schizophrenia and its neurological basis.
Method: EEG and functional magnetic resonance imaging (fMRI) data were obtained from first-episode and chronic patients with schizophrenia, and matched controls. A duration-deviant MMN paradigm was used in which participants heard an unattended random series of tones consisting of standards (92%, 50 ms) and deviants (8%, 100 ms). High-resolution MMNs were obtained as the difference between deviant and standard ERPs. Cortically constrained LORETA current source density analysis was performed using realistic head models.
Results: A reduction in MMN amplitude was seen in younger patients (40 years). This is consistent with our previous findings. Current source density analysis of the early phase of the MMN suggests that the major cortical generator of the MMN lies in the superior temporal gyrus (STG) as expected. The latter phase of the MMN engages more anterior cortical regions including premotor cortex. Patients show reduced activity in STG but increased activity in right premotor cortex. fMRI analysis showed that patients have greater activation in the insula and premotor cortex, whereas controls show greater activation in middle frontal gyrus.
Conclusions: The results are consistent with a deficit that onsets early in the disorder, that is associated with substantially reduced processing within auditory cortex and that leads to different patterns of activation in frontal cortical regions in patients compared with controls.

Investigation of circadian disruption in bipolar disorder

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Background: There is a growing body of evidence implicating circadian disruption as a possible mechanism underlying the pathogenesis of bipolar disorder. The circadian clock is entrained by changes in our external environment and regulates many of our bodies functions, including our sleep-wake cycle, metabolism, thermoregulation and blood pressure. While the measurement of circadian rhythmicity in mice is relatively straight forward through automated recording of home cage activity, the measurement of circadian function/disruption in humans is more difficult and costly, requiring each patient to undergo actigraphy in a sleep laboratory over a period of several days.
Methods: To collect data on a scale sufficient for genetic studies, a more efficient method of monitoring circadian function in humans is required to determine whether circadian disruption is involved in the primary pathogenesis of bipolar disorder or whether symptomatic sleep disruption is a secondary defect. We are investigating the role of circadian disruption in bipolar disorder, by genetic association, and rhythmic gene expression using immortalized B lymphocytes from a cohort of Australian bipolar families.
Results: Circadian timing in immortalized B lymphocytes is synchronized by serum shock, and circadian gene expression ensues for at least 56 h post synchronization.
Conclusions: Peripheral tissues, including immortalized blood cells, can be used as a cheap and higher throughput method to measure circadian output in humans and will be a useful adjunct to elucidating the molecular mechanisms underlying bipolar disorder.

Silence is golden? Improving cognitive performance in schizophrenia

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Cognitive impairment is a common and disabling symptom of schizophrenia and has adverse effects on functional outcome. Improvement in cognitive function has now been identified as a major target in the treatment of schizophrenia. Cognitive remediation strategies where people with schizophrenia are required to speak aloud while performing learning tasks have shown some success. The present study compared performance on tasks requiring parallel vocalization with task performance when the person did not speak aloud. Results showed selective benefits of verbal strategy on a neuropsychological task requiring multiple executive functions but not on tasks requiring simpler single-component cognitive functions.

Can consent be uninformed? Suggested reform of sexual offences against persons with mental impairment

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In R v Morgan (1970), the Supreme Court of Victoria stated that for incapacity to consent to be proved, it must be shown that a person 'has not sufficient knowledge or understanding to comprehend (a) that what is

https://doi.org/10.1017/S0924270800030301 Published online by Cambridge University Press

proposed to be done is the physical fact of penetration of her body by the male organ or, if that is not proved, (b) that the act of penetration proposed is one of sexual connexion as distinct from one of totally different character.' It is my contention that this standard of knowledge is insufficient to allow a person to protect him/herself against the commonly recognized consequences of sexual acts, namely pregnancy and sexually transmitted diseases. Although the literature suggests that increasing the required standard of knowledge to encompass these facts would mean that many persons with mental impairment would be deemed incapable of consent, I argue that consent that is not based on a standard of knowledge sufficient to allow an individual to safeguard their own interests cannot be considered true consent. Law reform is required so that consent to sexual acts more closely resembles the informed consent required for medical treatment.

Cognition and volition: a melancholic condition

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Background: Melancholia, a subtype of depression described as a specifier in DSM-IV, also forms a component of bipolar disorder (BD) and is distinguished from nonmelancholic depression on the basis of additional psychomotor disturbance (PMD). The latter comprises both retardation and agitation; concomitant cognitive disturbances are clinically observable and identified by patients as 'a difficulty in thinking', with lapses in concentration and decision-making impairments.
Methods: A literature review of experimental studies of cognition in melancholia was undertaken alongside a review of literature on bradyphrenia (slowed cognition) within various neuropsychiatric conditions. Particular emphasis was assigned to studies that attempted to distinguish PMD from processing speed on cognitive tasks, in terms of both functional neuroanatomy and relationships with impaired volition. The implications for future studies of cognition in melancholia are carefully critiqued.
Results: There has been relatively little empirical investigation of cognition and volition in melancholia. Studies thus far report partitioning of the neuropsychological profile of depression across melancholic and nonmelancholic subtypes implicating distinct neural dysfunction within dorsal and ventral prefrontal networks. In comparison, research examining psychomotor retardation and impaired volition has gravitated toward abnormal functioning within basal ganglia and related networks, with the archetypal example of this being Parkinson's disease.
Conclusions: Further empirical examination of cognition in depressive subtypes is required to evaluate the potential contribution of impaired volition to psychomotor retardation and distinct neuropsychological deficits in melancholia. Evidence for a cognitive marker of melancholia may prove useful for refining diagnostic criteria and elucidating functional brain networks that subserve endogeneity.

Childhood risk for obsessive compulsive disorder

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Background: Identifying childhood risk factors for obsessive compulsive disorder (OCD) is essential for improved understanding of its etiology and pathogenesis. Previous research has been largely reliant on retrospective self-report measurement of risk factors. The purpose of the current study was to link early appearing temperamental traits, behaviour, psychiatric symptoms and environmental stressors to the later emergence of OCD.
Methods: Longitudinal data were collected from participants of the Dunedin Multidisciplinary Health and Developmental Study. Study members were assessed from birth to adulthood on various measures of health and behaviour. Childhood temperament, behaviour, psychiatric symptoms and psychosocial stressors were linked with adult diagnostic outcomes.
Results: Preliminary findings suggest that certain types of childhood temperament and behaviour styles were associated with increased risk of developing OCD in adulthood. Childhood symptoms of OCD and childhood abuse were also risk factors for adult OCD diagnosis. Associations between childhood factors and OCD were largely independent of gender or socioeconomic status.
Conclusions: Results of the current study suggests that within-child characteristics and adverse life events play a role in the development of OCD. Findings also underline the need for targeted interventions for children with high-risk temperament or behavioural styles or childhood OCD symptoms.

https://doi.org/10.1017/S0924270800030301 Published online by Cambridge University Press