W11.03
PRACTICAL DEMONSTRATION AND GROUP INDUCTION

U. James

No abstract was available at the time of printing.

W11.04
STRUCTURE AND FUNCTIONS OF BRITISH HYPNOTHERAPY EXAMINATIONS BOARD

L. Mathew

No abstract was available at the time of printing.

FC09. Schizophrenia I

Chairs: N. Lindefors (S), C. Höschl (CZ)

FC09.01
BINOCULAR RIVALRY IS SLOW IN BIPOLAR DISORDER BUT NOT IN SCHIZOPHRENIA OR MAJOR DEPRESSION

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Binocular rivalry refers to the perceptual alternations that occur when different images such as orthogonal gratings, are presented simultaneously, one to each eye. We have demonstrated that the rate of rivalry with drifting, high-spatial frequency (h.s.f.) gratings is slow in bipolar subjects (median = 0.27 Hz) compared with controls (median = 0.60 Hz) (1). Here we used stationary gratings with a low-spatial frequency (l.s.f.) to assess rivalry rates in a different group of bipolars and controls, and in schizophrenia and major depression.

We report that rivalry rate in bipolar subjects (n′, mean = 0.28 Hz) was again significantly slower than in controls (n), mean = 0.40 Hz, $t(54) = -3.72$, $p < 0.001$). Fourteen subjects with schizophrenia (mean = 0.37 Hz) did not differ significantly from controls ($t(41) = 0.85$, $p = 0.40$), and 16 subjects with major depression (mean = 0.37 Hz) also did not differ significantly from controls ($t(43) = 0.72$, $p = 0.48$).

The data replicate our original finding and suggest that drifting, h.s.f. gratings separate bipolar from control groups more effectively than stationary, l.s.f. gratings, and that rivalry rates in schizophrenia and major depression are not slow. In light of our results, sensitivity and specificity data necessary to assess the clinical utility of the slow rivalry marker should be collected using drifting, h.s.f. gratings.

(1) Pettigrew JD, Miller SM: Proc R Soc Lond B 1998, 265: 2141–2148.

FC09.02
THE CALGARY DEPRESSION RATING SCALE FOR SCHIZOPHRENIA (CDSS): RELIABILITY AND VALIDITY DATA OF A GERMAN VERSION

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Background: The CDSS is a semi-standardized interview (9 items, scaled 0–3) for the assessment of depressive symptoms with high sensitivity and specificity in schizophrenia. We recently developed a German CDSS version in collaboration with the author of the source version and we carried out reliability studies. The scale is currently available in 19 languages including Czech, Danish, Dutch, Finish, French, German, Greek, Hungarian, Italian, Polish, Portuguese, Romanian, Russian, Spanish, and Swedish. In an ongoing study we investigate the validity of the CDSS.

Methods: Interrater reliability was assessed by intraclass correlations and Cohen's coefficient kappa. So far, 65 inpatients with a diagnosis of schizophrenia or schizoaffective disorder have been investigated after admission. The CDSS and additional scales (HAMD, BRMES, PANSS, SAS, BARS, AIMS) were used for the present analyses.

Results: The reliability studies revealed ICC > 0.7 for single items, and ICC > 0.9 for the total scale. Preliminary results of the ongoing study show a correlation between CDSS and HAMD or BRMES sum scores of $r > 0.70$ ($P < 0.0005$). No substantial correlation was found between CDSS scores and measures of EPS ($P > 0.05$). CDSS sum scores were moderately ($P < 0.05$) related to PANSS general psychopathology and negative symptoms. No substantial relationship emerged between CDSS and PANSS positive symptoms.

Conclusions: The results suggest that the CDSS (German version) is suitable to assess depressive symptoms in schizophrenia within reliably, rather specifically, and economically. No substantial overlap between CDSS scores and assessments of positive, negative, and EPS symptoms, and a rather high correlation with other depression assessments (HAMD, MADRS) underline the discriminant and converging validity of the CDSS. The careful development of a number of language versions of the CDSS now available on the Interact (http://www.ucalgary.ca/cdss/) make the scale very useful for European and international collaboration.

FC09.03
ESTABILISHING ONSET IN FIRST-EPISODE PSYCHOSIS: THE NOTTINGHAM ONSET SCHEDULE (NOS)

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Background: Most ratings of onset of psychosis use a single global measure. There are few structured instruments to study the phenomenon of onset itself. The Nottingham Onset Schedule is a short guided interviewing and rating schedule to measure onset in psychosis. Onset is defined as the time between the first reported/observed change in mental state/behaviour to the development of psychotic symptoms. Onset is conceptualised as comprising of (i) a prodrome of two parts: a period of 'unease' followed by 'non-diagnostic' symptoms; (ii) appearance of psychotic symptoms; and (iii) a build-up of diagnostic symptoms leading to a definite diagnosis. The schedule was piloted in a sample of first-episode psychosis patients.

Methodology: Consecutive cases of first-episode psychosis were administered the schedule, blind to diagnosis. A consensus ICD-10 diagnosis was made using all available information.

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Results: A preliminary analysis is reported for this abstract. Complete data were available for 53 consecutive cases (33 males, 20 females, mean age 32.2 year; 20 affective psychosis: F 30–33; 27 schizophrenia group: F20–29; and 6 Substance-related psychosis: F10–19). All three stages were identified in 46 (86.7%) cases. The NOS estimated mean and median prodrome lengths of 624 and 280 days respectively for schizophrenia group; and 106 and 49 days for affective psychosis (mean difference 517; 95%-CI 115, 919; $p = 0.013$). This difference was due to a significantly longer period of unease in the schizophrenia group.

Conclusions: The NOS helps identify individual components of onset in psychosis. Onset is significantly longer in schizophrenic disorders than in affective psychosis.

FC09.04

THE PRODROME OF FIRST ONSET PSYCHOSES: ARE AFFECTIVE SYMPTOMS SPECIFIC TO DIAGNOSIS?

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Background: Insidious onset and longer duration of untreated psychosis predict a poorer outcome in psychotic disorders. Characterising the symptoms of psychotic prodromes underlies prediction, early intervention and may illuminate mechanisms of onset. Affective symptoms are common in the prodrome of schizophrenia, but their specificity to a particular diagnostic group has not been established.

Aim: To quantify prodromal affective symptoms of first onset psychoses and determine their specificity to diagnosis.

Method: Subjects were drawn from a first onset inception cohort collected over 2 years in Nottingham. Each was assessed blind to diagnosis using the Nottingham Onset Schedule (NOS). Consensus diagnoses were made according to ICD 10 criteria using data from SCAN version 2 and case notes. Prodromal affective and non specific neurotic symptoms were quantified and diagnostic groups compared using Fisher's Exact Test.

Results: A preliminary sample is reported for this abstract. Complete data from 53 of 63 consecutive cases was initially available - 26 with schizophrenia and other psychoses (F20–29), 21 with affective psychoses (F30–33). 6 with drug induced psychoses were excluded. Depressive symptoms of low mood ($p = 0.7$), anhedonia ($p = 1.0$), fatigue ($p = 0.6$) and anxiety ($p = 1.0$) were all relatively common in both groups and not specific to diagnosis. Sleep and appetite disturbance and impairment of concentration are more common in affective psychoses as was a manic "triad" of elated mood, overtalkativeness and overactivity.

Discussion: These results suggest that core depressive symptoms do not have a diagnostic specificity and are common in the prodrome of both schizophrenia and affective psychoses. Biological depressive symptoms are more common in affective psychoses. Work is ongoing with the rest of the cohort. This will allow us to investigate these findings in a larger sample and in more detail.

FC09.05

CLOZAPINE, OLANZAPINE, RISPERIDONE, AND HALOPERIDOL IN REFRACTORY SCHIZOPHRENIA

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This was a prospective, double-blind, randomized 14-week trial in which patients were assigned to either clozapine (CLO), olanzapine (OLZ), risperidone (RIS), or haloperidol (HAL). The subjects were 157 treatment-resistant inpatients diagnosed (DSM-IV) with chronic schizophrenia or schizoaffective disorder. The trial consisted of Period 1 (8 weeks, escalation and fixed dose) and Period 2 (6 weeks, variable dose). The doses were escalated to their target levels: CLO 500, OLZ 20, RIS 8, and HAL 20 mg/day, and remained fixed until the end of Period 1. In Period 2, the doses were titrated within dose ranges: CLO 200–800; OLZ 10–40; RIS 4–16; HAL 10–30. CLO, OLZ and RIS (but not HAL) resulted in statistically significant ($p < 0.05$) improvements on total PANSS score in Period 1. In Period 2, CLO and OLZ were more effective ($p < 0.002$) against negative symptoms (PANSS subscale) than HAL; these differences were not mediated by extrapyramidal side effects. OLZ was also superior to HAL on total PANSS and General Psychopathology PANSS subscale ($p < 0.05$). CLO, OLZ, and RIS had less extrapyramidal side effects than HAL. Further research is required to determine whether these results generalize to other populations and dosage regimens.

FC09.06

CHOLECYSTOKININ CCK_B RECEPTOR mRNA ISOFORMS: EXPRESSION IN POST-MORTEM MONKEY AND HUMAN BRAIN – ALTERATIONS FOLLOWING SCHIZOPHRENIA

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CCK_B receptors are implicated in various psychiatric disorders. Their brain distribution has been studied with e.g. ligand binding techniques. This study focuses on the CCK_B receptor mRNA expression in cynomolgus monkey and human brain revealing implications for schizophrenia. We examined the monkey and human brain distribution of mRNAs encoding CCK_B receptors compared with mRNA encoding CCK peptide using in situ hybridisation histochemistry.

Monkey and human brain expression of CCK_B receptor mRNA show preferentially cortical distribution, with laminar expression of CCK_B receptor mRNA in the neocortex, hippocampus and cerebellar cortex. Low CCK_B receptor mRNA levels are seen in subcortical structures such as the striatum, amygdala and claustrum. CCK peptide mRNA in monkey is marc specifically distributed to neocortex and hippocampus, displaying laminar distribution. Lower levels are scan in the amygdala, claustrum and substantia nigra. The human brain distribution of mRNAs for CCK_B receptors and CCK peptide, respectively, is similar to that of the cynomolgus monkey brain. Hybridisation to tissue sections of post-mortem frontal cortex of schizophrenics and matched controls (B.A. 10)

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