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)

https://doi.org/10.1016/S0924-9338(00)94239-1 Published online by Cambridge University Press

revealed significant decrease of mRNAs for two splicing variants of the CCK_B receptor in specific layers of the frontal cortex.

This study demonstrates a highly preserved CCK system in brains of human and non-human primates displaying similarities with rodent data. New evidence is added for CCK involvement in schizophrenia. Thus CCKB receptor ligands should be studied more for implications in the treatment of schizophrenia.

ML03. Main Lecture 3

Chair: C.B. Pull (LUX)

ML03.01
MALIGNANT SADNESS – THE EVOLUTIONARY PSYCHOLOGY OF DEPRESSION

L. Wolpert. *University College London, London, UK*

There is a growing consensus that evolutionary theory can throw light on illness. For example sickle cell anaemia has not been eliminated because it can protect against malaria. The same approach can be applied to mental illness by drawing on the insights of evolutionary psychology, which tries to account for the adaptive basis of our various brain functions and behaviours. Can evolutionary theory account for why depression is so common? One theory suggests that it is adaptive in relation to the social hierarchy, its function being to inhibit aggressive behaviour to rivals and superiors when one's status is low. However this theory does not fit with the fact that depression is an illness that has so many negative consequences. An alternative is to consider depression as a pathological form of sadness, a basic emotion whose adaptive function is to maintain attachment and restore loss; grief following bereavement in the cost of that commitment. It is essential to understand grief in order to understand depression. While there may be a continuum of emotional states between normal sadness and severe clinical depression the feelings of those in severe depression bear no relation to feelings in normal life. Severe depression may develop from a pathological interaction between the biological basis of sadness and negative cognition which are mutually reinforcing. How these lead to somatization is not clear.

PS03. Treatment update 2000 – Cognitive disorders

Chair: S. Lovestone (UK)

PS03.01
TREATMENT OF ALZHEIMER'S DISEASE

J.T. O'Brien

No abstract was available at the time of printing.

PS03.02
TREATMENT OF BEHAVIOURAL SYMPTOMS IN DEMENTIA

H. Förstl

No abstract was available at the time of printing.

PS03.03
TREATMENT OF THE DEMENTIA DISEASE PROCESS

S. Lovestone

No abstract was available at the time of printing.

SES13. AEP Section "Child Psychiatry": Developmental perspectives in child and adolescent psychiatry

Chairs: D. Bailly (F), V. Delvenne (B)

SES13.01
POSSIBILITIES AND LIMITATIONS IN UNIFICATION OF STANDARDS – THE CHALLENGE IN CHILD AND ADOLESCENT PSYCHIATRY

M. Dabkowski

No abstract was available at the time of printing.

SES13.02
OUTCOME OF CHILDHOOD SEPARATION ANXIETY DISORDER

D. Bailly

No abstract was available at the time of printing.

SES13.03
GRIEF WORK AND ADOLESCENT DEVELOPMENT

A. Barbosa

No abstract was available at the time of printing.

SES13.04
QUALITY OF ATTACHMENT IN ADOLESCENTS HOSPITALIZED IN CRISIS UNIT

V. Delvenne

No abstract was available at the time of printing.

SES13.05
THE TRAUMA OF SEPARATION FOR ANOREXIC PATIENTS: AN ETIOPATHOGENIC AGENT AND A SUPPORT FOR TREATMENT

J.L. Venisse

No abstract was available at the time of printing.

https://doi.org/10.1016/S0924-9338(00)94239-1 Published online by Cambridge University Press