S36.04
FIRST-RANK SYMPTOMS IN SCHIZOPHRENIA: NO BASIS IN EVIDENCE

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Background: One of the major assumptions of the categorical approach to schizophrenia is that there are symptoms wich are relatively characteristics of the disorder. The paradigm of such as symptoms are the so called first-rank symptoms (FRS). The present study was aimed to examine the diagnostic relevance of these symptoms for schizophrenia.

Methods: Six-hundred sixty psychotic inpatients were assessed for FRS and their diagnostic value for schizophrenia (Feighner criteria) was tested against nonschizophrenic psychoses by means of the likelihood ratio (LR). Logistic regression analysis was used to determine the influence of confounding variables (clinical and demographic) and broad vs narrow FRS definitions on the association between FRS and schizophrenia. ROC analysis was used to determine the influence of the number of SPR in their diagnostic value.

Results: FRS were highly prevalent in both, schizophrenic and nonschizophrenic psychoses. The LR of at least one FRS for schizophrenia was 1.06 (95% CI = 0.94–1.20). After adjusting for confounding variables the association between FRS and schizophrenia remanined nonsignificant (OR = 1.25, 95% CI = 0.88–1.80). Neither broad nor narrow definitions of FRS were associated with schizophrenia, respectively (OR and 95% CI): 1.35 (0.98–1.87) and 1.29 (0.94–1.77). The number of FRS present did not influence their (lack of) diagnostic value (AUC = 0.54, 95% CI = 0.49–0.58).

Conclusions: FRS are not useful in differentiating schizophrenia for nonschizophrenic psychoses, and as a consequence they should not be given diagnostic prominence in future diagnostic criteria of schizophrenia. These data are in agreement with a dimensional view of the psychopathology of the psychoses and with the psychotic continuum hypothesis.

S36.05
ARE SCHIZOPHRENICS COGNITIVELY IMPAIRED MANICS?

R.M. Murray

No abstract was available at the time of printing.

S37. Suicide Part II. Clinical evaluation of suicidal behaviors

Chairs: J. Angst (CH), Y. Lecrubier (F)

S37.01
PSYCHOLOGICAL AUTOPSY STUDIES

E.T. Isometsä. *Research on Mood Disorders and Self-Destructive Behaviour, Department of Mental Health and Alcohol Research, National Public Health Institute, Helsinki, Finland*

Psychological autopsy is one of the most valuable tools of research on completed suicide. The method was developed in the USA, and the first psychological autopsy study of suicides in St. Louis was published in 1959. Since that there have been over twenty major psychological autopsy study projects in North America, Europe, Australia, New Zealand, Israel, Taiwan and India. The method involves collecting all available information on the deceased via structured interviews of family members, relatives or friends as well as attending health care personnel. In addition, information is collected from available health care and psychiatric records, other documents, and forensic examination. Thus a psychological autopsy synthetizes the information from multiple informants and records.

The early generation of psychological autopsies provided descriptive information on suicides, and has established the view that irrespective of setting, more than 90% of completed suicides have suffered from usually comorbid mental disorders, most of them mood disorders and/or substance use disorders. Furthermore, they revealed the remarkable undertreatment of these mental disorders, often despite contact with psychiatric or other health care services. More recent psychological autopsy studies have mostly used case-control designs, thus having been able to find and estimate the risk factors for suicide. The questions to be investigated in the future psychological autopsy studies may be somewhat different from those of the past. In particular, they may be more focused on interactions between risk factors or risk factor domains; focused on some specific suicide populations of major interest for suicide prevention, or combine psychological autopsy methodology with post mortem brain imaging, molecular genetic methods, or other biological measurements.

S37.02
THE INFLUENCE OF COMORBIDITY ON THE PREVALENCE OF SUICIDAL BEHAVIOUR

Y. Lecrubier

No abstract was available at the time of printing.

S37.03
SUICIDE ATTEMPTS IN PLACEBO CONTROLLED TRIALS

T. Laughren

No abstract was available at the time of printing.

S37.04
THE EFFICACY OF PSYCHOSOCIAL AND PHARMACOLOGICAL TREATMENT FOLLOWING DELIBERATE SELF-HARM: A SYSTEMATIC REVIEW

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Deliberate self-harm is an important health problem, associated with considerable risk of subsequent self-harm, including completed suicide. In a systematic review of the world literature we have examined the effectiveness of various treatments for deliberate selfharm patients in terms of prevention of suicidal behaviour. Promising results were found for problem-solving therapy, depot flupenthixol for recurrent repeaters of self-harm and long-term psychological therapy (dialectical therapy) for female patients with borderline personality disorder and recurrent self-harm. Interesting but non-conclusive results were found following sub-group analyses in trials of antidepressants for recurrent self-harm patients and the provision of a card to allow emergency contact with services. However, insufficient numbers of subjects in nearly all the trials limit the conclusions that can currently be reached about the most

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effective treatments for deliberate self-harm patients. Larger trials are badly needed.

SES14. AEP Section "Psychopharmacology": From receptor pharmacology to clinical prescription

Chair: M. Ackenheil (D)

SES14.01

FROM RECEPTOR PHARMACOLOGY TO CLINICAL PRESCRIPTION: ANTIDEPRESSANTS

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In the advent of modern antidepressant drug therapy we have moved from early empirical serendipity of tentative compounds in the middle of the past century to understanding of details in the pharmacodynamic actions of several CNS-monoamine enhancing such drugs as of today. The result of this progress is that a number of very effective clinical antidepressants are now available, but still there is need for improvements in this important psychiatric treatment armamentarium. However, should this be achieved primarily by introduction of yet other novel potential antidepressant compounds or is there now room for development of another conceptual approach towards drug development in counteracting major depressive disorders? The latter statement is supported by important factors to consider in the naturalistic clinical setting like problems with patient compliance with prescription of antidepressant agents and multiple possibilities for drug-drug interactions to occur due to common polypharmacy in this scenario, factors that would not change only if newer compounds are to be the future solution for optimising antidepressant drug therapy. By preference, a new conceptual scientific effort should be guided by a strive towards defining better strategies for individual dose optimisation procedures in the future.

Accordingly, in parallel with pursuing traditional pharmacological receptor research it is today necessary to focus also on delineating neglected clinical psychopharmacological issues of the established antidepressant drugs. To these issues belong a better definition of e.g. the many pharmacokinetic changes that may occur in clinical reality, as well as to improve our understanding of why these changes are brought about and when they may even be hazardous to a certain group of patients or the single individual prescribed such drug therapy. The rapidly escalating knowledge of details in drug metabolic features for antidepressant drugs and clinical application of techniques such as Therapeutic Drug Monitoring (TDM) and cytochrome P-450 (CYP) genotyping procedures based on these scientific fundaments therefore bears a great promise for the future development of advancing clinical antidepressant pharmacology into a more closely prescription-related research.

In recent years, therefore, the situation in this scientific field has come to change focusing more and more on antidepressant posology in a clinical context. To this end, introducing "clinical outcome" studies recording concentration- rather than doserelated drug effects in both in vivo in animal models as well as in phase III-IV human trials have become essential tools in this process. Underlying motives and state-of-theart for this antidepressant drug development that is complementary to traditional receptor pharmacology giving rise to an approach of "psychopharmacological holism" will be conveyed in the present lecture. This include a brief survey of the utility for TDM and CYP-genotyping methodology in clinical practice when treating affective disorders.

SES14.02

FROM RECEPTOR PHARMACOLOGY TO CLINICAL PRESCRIPTION: ANTIPSYCHOTICS

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The receptor binding properties of the new antipsychotics (risperidone, olanzapine, sertindole, quetiapine and ziprasidone) plays a critical role to explain the clinical profile of these drugs. All of them share 5HTA2/D2 antagonism, but they bind also to other multiple different receptors. D2 antagonism is related to both clinical efficacy on positive symptoms and extrapyramidal side effects and the antagonism of 5HTA2 receptors may be responsible of clinical efficacy on negative signs and symptoms of schizophrenia and reduced extrapyrarddal side effects The different binding profile on other receptors can be related to specific clinical and unwanted effects and can lead to a better clinical prescription if we consider the overall consequences but not the effect of the antagonism of one specific receptor. The binding affinities d the new drugs are:

Olanzapine: high for D4, 5-HT2A, 5-HT2C (high-moderate), 5-HT6, M1, M4 and alpha 1; moderate for D3; low for D2, 5-HT7 and H1.

Risperidone: high for D2, D3, D4, 5-HT2A, 5-HT7 and alpha 1; low for 5-HT2C, 5-HT6, M4 and H1.

Quetiapine: high for H1, 5-HT2C, 5-HT6 and 5-HT7; moderate for 5-HT2A and alpha 1; low for D2, D3, D4 and M4.

Sertindole; high for D3, D4, 5-HT2A, 5-HT2C, 5-HT6, 5-HT7 and alpha

1; moderate for D2; low for M4 and H1.

Ziprasidone. High for D2, D3, 5-HT2A, 5-HT6, 5-HT7 and alpha 1; moderate for D4, 5-HT2C and H1; low for M4.

The overall effect v.s. single binding consequences can be represented by t he sedating effect of quetiapine. There is general agreement that H1 antagonism induces a high degree of sedation but quetiapine increase the release of tele-methyl-histamine via 5-HT2 antagonism and the clinical consequence is a low sedating effect.

SES14.03

PRACTICAL PROBLEMS WITH CO-PRESCRIPTION: PROBLEMS WITH EFFICACY

M. Ackenheil. *Psychiatric Clinic, Ludwig-Maximilians-University Munich, Germany*

Polypharmacy, the simultaneous prescription of several drugs is the usual treatment for most of the psychiatric patients. In the mean each patient is treated with 3 psychotropic drugs, additionally with medicaments for somatic diseases like e.g. hypertension (betablockers, AT_1-Inhibitors, α-Blockers). Many of these drugs show interactions, both on the level of pharmacokinetic and pharmacodynamic. Such combinations through different mechanismes can diminish the therapeutic efficacy. Augmentation strategies utilize such interactions to shorten the response delay or to overcome non response. However, most of the co-medication lack any scientific evidence and even are contradictory.

The availability of new specific drugs leads to frequent combination. For depression, new SSRIs are prescribed togethter with classical antidepressants, lithium and mood stabilizers. Non response due to too high plasma labels or to pharmacodynamic interactions are resulting. Schizophrenic and manic patients are treated with

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