S06.03
ASSESSMENT OF CLINICAL SKILLS

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Background: The skills a psychiatrist requires include the ability
i. to take a full, relevant and empathic psychiatric history from a patient
ii. to describe the mental state comprehensively
iii. to carry out a relevant physical examination.
iv. to give a full differential diagnosis
v. to explain a plausible aetiological formulation.
vi. to describe a management plan to treat the disorder.

These skills can be assessed by regular appraisal or by examination.

Assessment Techniques: The following techniques can be used:
i. Log Book. The trainee writes up a number of selected cases.
ii. Presentation of patients to the supervisor and/or to others.
iii. Videotape assessment. This can give feedback to the trainee involved.
iv. Written Examinations

Diagnostic and management skills can be tested by using tests such as the Extended Matching Items Test. Alternatively Problem Boxes can be used that develop a scenario that evaluate diagnostic skills initially and then consider a range of management options.

Examination Methods:
i. Assessment of a real psychiatric patient
ii. Assessment of a simulated psychiatric patient.
iii. Assessment of management skills
iv. Assessment of a series of diagnostic and management options using the Observed Structured Clinical Examination or OSCE.

Conclusions: Regular appraisal is of benefit in improving clinical skills in psychiatric trainees. Examinations require the evaluation of issues posed by real or simulated psychiatric patients.

S06.04
EXAMINATION OF PSYCHIATRISTS IN CANADA

R. Swinson

No abstract was available at the time of printing.

S06.05
THE FUTURE OF PSYCHIATRIC TRAINING: WHAT, WHERE, WHO AND HOW

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In an era of diminishing resources, psychiatry training has faced numerous challenges and has become the forgotten item on the agenda of academic psychiatry. However, it is necessary to challenge the trend of a decreased emphasis on teaching psychiatry. Poor teaching in psychiatry will not only undermine the quality of patent care in the future, but will also threaten the level of excellence within the field of psychiatry. Inadequately prepared psychiatrists will be unable to face external (e.g., from other mental health professions) and internal (e.g., rapidly emerging research findings) pressures. The teaching of psychiatry needs reevaluation on all levels. Certain areas (e.g., where, by whom and how psychiatry is going to be taught; collaboration with other mental health professionals and patients' groups) will require special attention while planning the training of future generations of psychiatrists. This presentation will focus on issues such as; A) What are we going to be teaching (biopsychosocial model? Neuroscience findings?); B) How are we going to teach (computers?, video?) and how will we evaluate our teaching (oral vs. written exams?); C) Who is going to be teaching (faculty vs. voluntary faculty, faculty development); and D) Where are we going to be teaching (inpatient vs. outpatient, special centers?).

S07. Update of genetic research in schizophrenia and affective disorders

Chairs: H. Ewald (DK), W. Maier (D)

S07.01
GENETIC DETERMINANTS OF RESPONSE TO PSYCHOPHARMACOLOGICAL TREATMENT

M.M. Nöthen

No abstract was available at the time of printing.

S07.02
CURRENT STATUS OF THE SEARCH FOR GENES CONTRIBUTING TO BIPOLAR AFFECTIVE DISORDER

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It is hoped that the identification of genes involved in susceptibility to bipolar affective disorder will make further research into the etiology and pathophysiology possible. This may lead to improvement of treatment, treatment choice, diagnostic classification and perhaps even preventive measures.

The first association and linkage studies of bipolar affective disorder were performed around four and three decades ago respectively. Developments in diagnostic instruments and criteria, molecular genetics, computer programs and statistics have helped to identify more than 10 candidate chromosome regions potentially containing genes which increase susceptibility to bipolar affective disorder.

Based on molecular genetic studies it appears less likely that a single specific disease allele is present in all or most cases. Genetic mapping studies have suggested that a combination of susceptibility and perhaps protective alleles at a number of loci determines the genetic risk of developing bipolar affective diaorder in the individual. Some of these are possibly also involved in the etiology of schizophrenia.

Though no DNA sequence variation of relevance has yet been reported the ongoing sequencing of the human genome and recent developments for high-throughput genotypings and other molecular genetic methods will facilitate this.

Considerable efforts are now being aimed at identifying the risk genes in the most promising chromosome regions including chromosome 4p, 12q, 18, 21 and Xq.

S07.03
CURRENT STATUS OF THE SEARCH FOR GENES CONTRIBUTING TO SCHIZOPHRENIA

W. Maier*, M. Rietschel, D. Lichtermann, D. Müller, T. Schulze, S. Schwab, D. Wildenauer. *Department of Psychiatry, University of Bonn, Germany*

Major progress in unraveling the genetic basis of schizophrenia occurred during recent years. A series of genome-wide scans for positional cloning of contributing genes have been completed.

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Although there is no full consistency across all studies, a series of loci on the genome overlap between several studies. Those consensus loci are: 1q, 5q, 6p, 6q, 8p, 13q, 18p and 22q. However, up to now not a single contributing gene has been identified. The multiplicity of these loci demonstrates that schizophrenia is not under the control of a single, causal gene; instead, multiple genes are operating in concert with environmental factors. It remains obscure if the contributing gene mutations are common with a multiplicity of pathogenic mutations for each case or if different subtypes of schizophrenia are each under the control of a subtype-specific major gene.

Current work is focussed on narrowing down the candidate regions by finding linkage disequilibrium to either anonymous markers or functional gene variants. Major progress has to be expected in this respect in due course.

S07.04
JOINT EFFECTS OF GENOTYPE AND ENVIRONMENT IN SCHIZOPHRENIA

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To an important degree, genetic effects on behavior come about because they either influence the extent to which the individual is likely to be exposed to individual differences in environmental risk or they affect how susceptible the individual is to environmental adversities. Adoption studies are able to distinguish the effects of environment from the effects of genes. A nationwide Finnish sample of schizophrenics' offspring given up for adoption (N = 186) was compared blindly with matched controls, who were adopted center dot Offspring of nonschizophrenic biological parents (N = 203). The adoptive families were investigated thoroughly using joint and individual interviews and psychological tests. The biological parents were also interviewed and tested. The Finnish adoption study has generated a large sample of adoptees; obtained standardized personal interviews and tests with all subjects whenever possible; used DSM-III-R criteria for all subjects; followed up adoptees who were initially not fully in the age of risk for schizophrenia and re-examined them with standardized diagnostic instruments. Our results support a genetic hypothesis for a schizophrenia spectrum that includes in addition to schizophrenia, nonaffective psychoses and schizotypal personality disorder. However, notable differences between the two groups only emerged in the families which were rated as disturbed. Thus the genetic effect, that is, the propensity for clinically serious psychiatric disorder in the adoptees, was expressed primarily in association with a disturbed adoptive family rearing-environment and was not present in association with a "healthy", possibly protective, adoptive family environment.

S07.05
ETHICAL IMPLICATIONS OF MOLECULAR-GENETIC RESEARCH IN PSYCHIATRY

M. Rietschel. *Department of Psychiatry, Univ. Bonn Sigmund-Freud-Str. 25, 53105 Bonn, Germany*

The etiology of schizophrenia and bipolar disorders is complex with genetic factors accounting for more than 50% of its variance. The aim of molecular genetic research is to identify vulnerability genes in order to gain insight in the pathophysiology. It is hoped that this will lead to better diagnosis, prevention and therapy of the disorders.

Besides those benefits, this progress may have serious ethical implications. Knowledge about vulnerability genes may influence disease concepts and self-awareness, (which may result in increased or decreased stigmatisation), privacy and confidentiality, family and life-planning.

In complex disorder the predictive value of vulnerability genes are limited, they only modify an "a-priori-risk". In monogenic disorder many disease genes have already been identified and a high degree of certainty can be achieved by predictive testing. Problems inherent to predictive testing in monogenic and complex diseases like psychiatric disorders will be discussed.

S08. Pharmacological relapse prevention in alcoholism – from animal models to clinical trials

Chairs: J.A.L. Boning (D), L.G. Schmidt (D)

S08.01
IS THERE A NEUROCHEMICAL BASIS FOR ALCOHOLISM AND RELAPSE? ANIMAL AND HUMAN STUDIES

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Despite numerous neuochemical and molecular biological studies of alcohol abusers and experimental animal models, the pathophysiology and neuochemical basis for alcoholism remains poorly understood. The pharmacokinetics of ethanol clearance from the brain, predominantly by catalase (Ward et al., 2000) will have a profound effect upon the mesolimbic system, ethanol enhancing (Blanchard et al., 1993) and acetaldehyde (Ward et al., 1997) diminishing dopamine release from specific brain regions. In addition an association between a specific allele pattern of the dopamine D2 receptor to the marker hD2G1 in alcoholics differs from that of control subjects and is thought to be involved in the lower dopamine binding affinity to the receptor such that the individual would need to drink more ethanol to obtain the pleasurable effect initiated by dopamine release

It is clear that during chronic alcohol abuse the levels of most neurotransmitters are maintained within their normal concentrations and it is only during detoxification that such equilibrium is drastically disturbed. Excitatory amino acids, particularly glutamic acid, are increased during the initial stages of detoxification which is in part responsible for many of the unpleasant side effects observed in alcohol abusers during withdrawal, (Rossetti et al., 1995). The sulphonated amino acid taurine has been implicated in modulating such changes (Ward et al., 1999) which may be attributable to the alterations in both NMDA and GABA receptors as well as modulation of calcium homeostasis.

Despite the use of different animal models of ethanol sensitivity, tolerance and withdrawal as well as transgenic and knockout animals these have not helped to advance, to any considerable extent, our knowledge of the role of neurotransmitters and their receptors in chronic ethanol intoxication and withdrawal. However the use of agonists and antagonists of specific receptors have yielded a better insight into their role in alcohol intoxication and withdrawal and are the prime target of various pharmaceutical drugs now being developed for the treatment of alcoholism.

(1) Blanchard et al., *Alc Clin Exp Res* **17** 968–973 1993
(2) Rossetti et al., *Eur J Pharmacol* **283** 177–183 1995
(3) Ward et al., *Neuropharmacol* **36** 225–232 1997
(4) Ward et al., *Neurosci Res Comm*, 24 41–49 1999

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