related to changes in dopamine activity and therefore respond very efficiently to D2-dopamine antagonists.

S03.03

Why cannabis use is bad for schizophrenia

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Background: Progressive gray matter volume reductions have been found in schizophrenia and greater changes seem to be related to poorer outcome1,2. As patients with schizophrenia who use cannabis have a worse prognosis 3, the progressive gray matter change in these patients might be even greater.

Method: Fifty-one patients with recent-onset schizophrenia (cannabis users n=19; non-users n=32) and thirty-one matched healthy comparison subjects were included in this five year longitudinal MRI study. All subjects were assessed at inclusion and after five years. Total brain, gray and white matter, cerebellar, lateral and third ventricle volumes were measured. Percentages of volume change over time were calculated. Univariate analysis of covariance and pairwise comparisons were performed.

Results: Cannabis using patients, non-using patients and healthy comparison subjects differed significantly in total brain, gray matter, lateral and third ventricles and cerebellum volumes. No change in white matter was observed between the groups.

Cannabis using patients with schizophrenia showed a more rapid decrease in total brain and cerebellar volume and increase in lateral and third ventricle volumes as compared to healthy subject and non-using patients. Gray matter volume decrease occurred in all patients with schizophrenia as compared to healthy subjects, but was significantly greater in patients using cannabis.

Conclusion: In schizophrenia progressive gray matter volume decrease occurs during the first five years of illness. Cannabis use causes an additional decrease of gray matter in patients with schizophrenia and could be explained by either a worse illness outcome or the effects of cannabis.

S03.04

Schizophrenia: A biologist's perspective

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The human-type consciousness (defined as ability of self-recognition) has high evolutionary advantage, but the complexity of its anatomical/neurochemical background makes it prone to disturbances. Schizophrenia is one of cognitive disorders, arising from deficits in neural connectivity and in neurotransmission. The neurochemical hypothesis points to the disturbances in dopaminergic transmission: hyperactivity in mesolimbic and striatal systems (responsible for positive symptoms) with concomitant hypofunction of mesocortical system (hypofrontality and negative symptoms). This poses difficulties for pharmacotherapy: dopaminergic receptor blockade abolishes positive, but may aggravate negative symptoms. Difference in serotonin control of various dopaminergic subsystems permits to overcome some difficulties. The neurodevelopmental hypothesis of schizophrenia underlies the role of prenatal and perinatal stress disturbing neuronal migration leading to disrupted cortical architecture. In neurodevelopmental disorders stress hormones are the wreckers and neurotrophins are protectants. A role for BDNF is suggested as its levels are decreased in schizophrenic brain and its Val66Met polymorphism is associated with earlier onset of the disease. The emergence of schizophrenia in late adolescence may be caused by inadequate BDNF supply at the time of final maturation of prefrontal cortex, and attempts of correction of BDNF level may be worth trying. It may be assumed that schizophrenia is the result of wrong interactions between stress, genetic and developmental factors. Studies on candidate genes for schizophrenia and of endophenotypes lead to better understanding of the disease and prospects for more efficient therapy. The question arises why the genes involved in schizophrenia had not been eliminated in the course of evolution?

S04. Symposium: THE NEW ROLE OF (NEURO) PSYCHIATRY IN SEXUAL MEDICINE

S04.01

Introduction

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From about 1910 till the mid 1950s, medical sexology has mainly been practiced by psychiatrists. Although many psychiatrists used a psychoanalytic approach, particularly the psychiatrists at the " Institut fur Sexual Wissenschaft" in Berlin introduced a biological, e.g. endocrinological, approach to treat sexual dysfunctions. However, this famous Institute was destroyed by the Nazis in 1933 and this marked the end of a very fruitful biological period in medical sexology. After World War II, sexology became more and more investigated and practiced by psychologists who at the time claimed successes of a behavioristic approach. Gradually, psychiatrists lost their interest in sexology. Currently, and internationally, sexology is not any more an important part of psychiatry. Since the 1990s, but particularly after the introduction of Viagra in 1998, sexology has become a major part of urology. The current state of sexology being practiced mainly by psychologists and urologists may be harmful for a balanced development of medical sexology.

Progress in the field of sexual neuropsychopharmacology has shown that sexual functioning is related to brain functioning. This means that there is a new role for (neuro)psychiatry in sexual medicine. Psychiatrists, who more than psychologists and urologists are better equipped to deal with psychopharmacotherapy and psychotherapy, should take part in the new scientific developments, both with regard to drug treatment as in psychotherapy of sexual disorders.

S04.02

Diagnostic criteria of sexual dysfunctions: Need for a change

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The DSM diagnostic criteria of sexual dysfunctions have been widely used by clinicians, researchers and in pharmaceutical trials. However, these diagnostic criteria do not reflect the developments in the field of sexual medicine. These criteria are vague and do not include many of the criteria used in other mental disorders classified in the DSM. Better defined operational criteria are needed to define more homogenous population samples and to help answer some basic research questions.

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Some of the issues that need to be addressed in the new revision of the sexual dysfunctions diagnostic criteria include the duration of sexual dysfunction, intensity and frequency of sexual dysfunction, the use of distress as a diagnostic criterion, whether there are specific differences in diagnosing female and male sexual dysfunction, validity of some diagnostic entities (e.g., sexual aversion disorder), reclassifying some sexual dysfunctions (e.g., dyspareunia as a pain disorder), and the overlap of diagnoses.

Further deliberation of sexual dysfunction classification should also include two core questions: a) when does a sexual problem become a sexual dysfunction, and related to that b) what do we consider ''normal'' and/or what is a biological variation of sexual functioning (e.g., are rapid ejaculation and extremely delayed ejaculation dysfunctions or normal variants of sexual performance at the very ends of the spectrum?).

This presentation will review in detail the deficiencies of the standing diagnostic criteria and will provide suggestions for improvement of these criteria based on evidence from the literature and on recommendations of expert panels.

S04.03

Female sexual dysfunction

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There has been considerable research concerning the epidemiology and treatment of female sexual dysfunction. Research has indicated a high prevalence of female sexual problems in most cultures. Clinical trials have tested the efficacy of a variety of pharmaceutical agents for the treatment of female sexual dysfunction. This research can be grouped into three major areas: the use of hormonal agents, the use of centrally acting compounds and the use of agents promoting peripheral vasodilation. This presentation will review current research, treatment options, and gaps in our knowledge.

S04.04

Drug treatment and psychotherapy of premature ejaculation

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Drug treatment of lifelong premature ejaculation (PE) consists of daily use of SSRIs, particularly paroxetine 20mg and sertraline 50-100mg, on-demand use of clomipramine 20-50mg (3-6 hour prior to coitus) and/or topical anesthetics, such as lidocaine and prilocaine [1].

PE is a common male sexual complaint in approximately 20-40% of men. However, not all these men require treatment. PE has been distinguished in Lifelong and Acquired PE. Recently, two other PE syndromes have been classified [2,3]. In ''Normal Variable PE'' the occurrence of early ejaculation is rather inconsistent and should be regarded as a normal pattern of ejaculatory performance [2]. In ''Premature-like Ejaculatory Dysfunction'' men complain of an early ejaculation while the duration of the IELT is in the normal range (about 5 minutes) or even longer (5-10 min) [3]. The four PE syndromes require different forms of treatment. Lifelong PE should be treated with medication. Acquired PE needs medication and/or psychotherapy. Normal Variable PE requires psycho-education and Premature-like PE requires either psychotherapy, psycho-education or counselling.

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S05. Symposium: THE CLINICAL SIGNIFICANCE OF AT-RISK HAPLOTYPES IN SCHIZOPHRENIA

S05

The correlation of the endophenotypes to the at-risk haplotypes

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Although a series of DNA-sequence variants in proposed disposition genes for schizophrenia have been identified, the mechanisms to translate the genetic vulnerability for schizophrenia to the manifestation of the disease remain obscure. The analysis of the relationship of disease-associated alleles and combinations of alleles (haplotypes) to the clinical features and associated neurobiological correlates offer a tool to increase our understanding of the aetiology of schizophrenia.

We will explore this relationship in a series of case-control samples by (1) extracting schizophrenia-associated alleles and haplotypes of postulated susceptibility and modifying genes, (2) testing these identified genetic markers for association with neuropsychological and neuroimaging features of schizophrenia.

S06. Symposium: CANNABIS DEPENDENCE AND ABUSE: FROM NEUROBIOLOGICAL UNDERSTANDING TO TREATMENT

S06.01

How to screen adolescents for cannabis dependence

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Abstract not available at the time of printing.

S06.02

Cannabis abuse comorbidity with psychiatric disorders

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Cannabis use has been related to many psychiatric problems, particularly psychotic disorders, affective disorders, and anxiety. Chronic

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