

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.

### References

- 1 Waldinger MD, Olivier B. Utility of selective serotonin reuptake inhibitors in premature ejaculation. *Current Opinion in Investigational Drugs* 2004;5:743–7.

- 2 Waldinger MD, Schweitzer DH. Changing paradigms from an historical DSM-III and DSM-IV view towards an evidence based definition of premature ejaculation. Part II: Proposals for DSM-V and ICD-11. *J Sex Med* 2006;3:693–705.
- 3 Waldinger MD. The need for a revival of psychoanalytic investigations into premature ejaculation. *JMHG* 2006;3:390–6.

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

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### 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.

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

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### 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

cannabis use is related to panic attacks, anxiety, depression, and low motivation. It would appear that frequent cannabis use during adolescence is predictive of later depression and anxiety. Cannabis has also been related to amotivational syndrome in chronic users. This disorder is characterized by a personal deterioration with loss of energy and work drive, but the validity of this syndrome remains uncertain. Epidemiological studies have shown a clear association between cannabis use and psychosis, mainly adult schizophreniform disorders. Recent studies have identified a higher frequency of disturbance in sensorium, irritability, affective disturbances, derealization/depersonalization, and visual hallucinations in cannabis induced psychosis than in acute schizophrenia psychosis. Schizophrenia with substance abuse has been associated with poor treatment compliance, increased rates of hospital admissions, suicide, violent behavior and unstable housing and homelessness. Cannabis use specifically has been correlated with the exacerbation of psychotic symptoms and increased tardive dyskinesia. It may be the case that schizophrenic patients are inclined to consume cannabis, either due to lowered impulse control or as a means of reducing negative symptoms. On the other hand, it may be that cannabis use itself either causes or precipitates psychosis. In short, the association between cannabis use, psychosis, depression, behavioral problems, tobacco smoking, excessive drinking and use of illicit drugs shows a severe pattern of comorbidity that may lead to further negative outcomes, and requires further study for the identification of appropriate treatments.

### S06.03

An update on the neurobiology of cannabis addiction

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The endocannabinoid system has been involved in a variety of physiological functions, including the control of nociception, motor behaviour, learning/memory, reward, neuroprotection, food intake and metabolism. This system is mainly activated in response to external stimuli to help establish the steady-state homeostasis of other neurotransmitters and mediators. Recent studies have involved the endocannabinoid system in the common neurobiological substrate underlying drug addictive processes. This system participates in the primary rewarding effects of cannabinoids, nicotine, alcohol and opioids through the release of endocannabinoids in the ventral tegmental area. Endocannabinoids are also involved in the motivation to seek the drug by a dopamine-independent mechanism demonstrated for psychostimulants and opioids. The endocannabinoid system participates as well in relapse to drug-seeking behaviour by mediating the motivational effects of drug-related environmental stimuli and drug re-exposure. In agreement, clinical trials have revealed the effectiveness of the CB1 cannabinoid antagonist rimonabant to obtain smoking cessation. CB1 cannabinoid antagonists could represent a new generation of compounds to treat drug addiction.

### S06.04

In vivo measurement of neuronal dopamine transporter in cannabis dependant subjects, with positron tomography and [11C]-PE2I

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Dopaminergic system within mesocorticolimbic circuit plays a crucial role in addictive behaviors. However, no data to date are available concerning the effect of cannabis addiction on dopaminergic neurotransmission in humans. The neuronal dopamine transporter (DAT) ensures the regulation of dopaminergic neurotransmission by the re-uptake of extracellular dopamine. Observation of DAT density anomalies within the mesocorticolimbic system in cannabis-dependant subjects could provide further evidence for the implication of dopaminergic dysfunction in cannabis addiction. Thus, this work aims at the study of DAT density in control, tobacco-dependents subjects and cannabis-dependants subjects, gender and age-paired with Positron Emission Tomography (PET).

Subjects are scanned on High Resolution Research Tomograph (HRRT) for one hour after injection of a selective DAT radioligand ([11C]-PE2I). The binding potential (BP) in regions of interest previously defined within the mesocorticolimbic circuit was calculated using a simplified reference tissue of Lammertsma in order to measure an index of DAT density. BP-obtained in each group: control, tobacco-dependents and cannabis-dependents subjects, were compared with t-tests. Preliminary results will be presented during the seminar.

### S06.05

An update on state of the art treatment of cannabis dependence

A. Benyamina. *Algeria*

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## S07. Symposium: SOCIAL COGNITION IN SCHIZOPHRENIA: THE KEY FOR SUCCESSFUL CBT INTERVENTIONS (Organised by the AEP Section on Schizophrenia)

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### S07.01

Social cognition and theoretical framework

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In recent years, social cognition became a valuable construct for understanding the nature and disability of schizophrenia (Green et al, 2005), and different studies have pointed out for its potential as a mediator of relations between neurocognition and functional status in schizophrenia (Sergi et al, 2006).

This presentation aims to review the concept of social cognition, describe the key social cognitive domains, discuss the importance of social cognition in schizophrenia by highlighting its functional significance, and finally present a brief overview of the main methodological issues regarding research on some of the presented issues.

### S07.02

Affect recognition in schizophrenia: impairments and treatment

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