

S33-4**THE OCD SCHOOL: COGNITIVE BEHAVIOUR THERAPY**

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While cognitive behavioral therapy is the psychological treatment of choice for Obsessive Compulsive Disorder (OCD), there are far too few therapists for increasing numbers of patients who request this form of therapy. The OCD School was started to provide primary care givers with the necessary skills to provide more adequate, qualified treatment.

During the 5 day course, participants were given approximately 14 hours of lectures, demonstrations and group discussions in addition to reading 200 pages of literature about the treatment of OCD with cognitive behavior therapy. Individual cases were presented by each participant. Analysis of relevant factors and discussion of considerations for designing a treatment program were then conducted using these cases. During the time interval between course days, the participants had specified exercises to conduct with their patients in order to practice the skills learned during the course.

Our experiences with 3 OCD schools (with approximately 100 participants: 50 psychiatrists and 50 of their co-workers) show that this form of training can increase all participants knowledge about effective treatment for OCD and enable many mental health workers to use behavioral interventions to help OCD patients.

S33-5**THE OCD SCHOOL: DRUG TREATMENT**

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One important component of the OCD School is to instruct members of psychiatric teams how to carry out a high standard drug treatment of Obsessive Compulsive Disorder (OCD). The goal is that both members of the team, i.e. both the physician and the non-physician should have a fundamental knowledge of treatment principles. Using the well known "Problem-Based Learning" approach, groups of approximately 8 people work with diagnostic and treatment issues for presented cases. The cases pose various problems, including diagnostic problems, choice of first and second line treatments, as well as more complicated issues such as drug-drug interaction and comorbidity problems. Patients own preferences concerning length of treatment are also considered when finding solutions for individual cases.

S34. Impulsivity and aggression

Chairs: C Pull (LUX), SP Tyrer (UK)

S34-1

No abstract received

S34-2**ANIMAL MODELS OF IMPULSIVITY AND AGGRESSION**

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Animal models of impulsivity are not developed extensively. Because of the connection between aggression and impulsivity, one

way of studying animal models of impulsivity, is looking carefully into animal models of aggression. Because almost all research into the CNS and aggression has been performed in rodents, the animal models used are mainly based on rats and mice. The aggressive behaviour of rodents is often divided into offensive and defensive aggression. A much used offensive/defensive model is the "Resident Intruder" paradigm, in which the resident (a male rat in a territory) shows offensive behaviour, whereas the intruder (a strange male rat) models defensive behaviour. Evidence exists that a number of neural systems in the CNS is prominently involved in offensive behaviour, notably the serotonergic and the GABA_A-BDZ receptor systems. There is strong evidence that stimulation of one type of serotonin-receptor (the 5-HT_{1B} receptor) inhibits offensive aggression, although the 5-HT_{1A} receptor also plays a role. Remaining 5-HT receptors are not clearly involved in offensive aggression. Defensive aggression on the contrary is mainly influenced by stimulation of 5-HT_{2(A,C)} receptors and not by 5-HT₁ receptors. Stimulation of the GABA_A-BDZ receptor complex (e.g. with benzodiazepines or alcohol) may lead to disinhibited (paradoxal) aggression. Recently, the gene (DNA) coding (via mRNA) for the 5-HT_{1B} receptor has been functionally removed from the genome of a mouse. This so-called 5-HT_{1B} knock-out mouse gives us the opportunity to study the role of this receptor in aggressive and impulsive behaviour. This mouse (both male and female) was more (offensively) aggressive than the normal mouse (wildtype). Apparently, the 5-HT_{1B} receptor has under normal conditions an inhibitory function in offensive behaviour. Further research shows a more general inhibition, suggesting that the 5-HT_{1B} receptor KO-mouse is "impulsive". We hypothesize that the 5-HT_{1B} KO-mouse is an animal model for impulsivity.

S34-3**THE USE OF SEROTONERGIC DRUGS IN THE TREATMENT OF IMPULSE CONTROL DISORDERS**

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The basis for treating impulse control disorders (ICD) with serotonergic drugs comes from some evidence indicating that excessive impulsiveness is associated to a low cerebral serotonin function. Violent aggressive patients, gamblers and fire-setters have shown decreased 5-HT indexes in several studies, but these results are not conclusive and cannot be extended to all the impulse control disorders.

The treatment of ICD with serotonergic drugs is, therefore, empirically oriented. Antidepressants have been used in these patients for many years before the hiposerotonergic hypothesis of impulsivity with promising results. Clomipramine, but also amitriptyline and nortriptyline, have demonstrated some efficacy in kleptomaniac patients. Pathological gamblers, compulsive buyers, trichotillomaniac patients and binge eating patients have also demonstrated a satisfactory response to antidepressants. Lithium carbonate has also demonstrated some efficacy in trichotillomaniacs, gamblers and violent behaviours.

There are only a few reports suggesting that, like in OCD, serotonergic drugs can be more effective than noradrenergic ones in the treatment of ICD patients. Globally, it seems to be a positive response to SSRIs and a good tolerance to side effects. Some patients improve at doses similar to that of depression, but in others, higher doses, like in OCD, are needed. Also, the response is not homogeneous for all the ICDs. While binge eating and trichotillomania show a better response, others like gambling and fire setting seem to have a poorer response than other ICD.