

**Workshop ID: IW03**

## **Basic and clinical aspects of the treatment of major depressive disorder**

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**Educational Objectives:** To offer to clinicians a modern understanding of the ubiquity of depression, and the interactions between unbalanced cerebral monoaminergic neurotransmission and the heterogeneous clinical phenomenology of mood disorders, and the various forms of effective antidepressant treatment regimens.

**Workshop description:** Four interactive lectures on basic and clinical knowledge on depression followed by discussion and opportunities for Q&A.

More than one in every 5 humans suffer from a major depressive disorder at some period in their lives. It is a ubiquitous human experience, and the point prevalence varies from 5 to 10 percent in different countries. Correct diagnosis may be simple to an experienced clinician trained to ask the correct questions to his or her patients, but the clinical settings in which depression occurs may be unsuspected and not directly intuitive. Even though it phenomenologically manifests as a quite heterogeneous disorder, only few subdivisions have been heuristically and therapeutically relevant – e.g. the unipolar/bipolar and typical/atypical dichotomies, the melancholia (or presence of somatic symptoms) concept, and the psychotic dimension.

A depressive psychobiological diathesis that will allow clinical manifestations when challenged. The mechanism has been understood in the past few years as intricate pathological signalling dysfunctions involving monoamine neurotransmitters in the basal ganglia, thalamus, and prefrontal cortex. This knowledge allows predictions on which pharmacological principles will turn clinically beneficial. Genomic dysregulation of synaptic signalling has recently become a target for pharmacotherapeutic attempts. Therapeutic normalization of HPA axis hyper- or hypoactivation has been tried.

All antidepressant drugs in current use enhance noradrenergic and/or serotonergic and/or dopaminergic activity by synaptic reuptake inhibition. Direct induction of receptor up- or down-regulation has proven clinically useful only in special circumstances. In order to augment response clinicians should not turn away from the concurrent use of drugs with different biochemical profiles. Extended vagus nerve stimulation (VNS) is a new treatment modality that might work as a signal-enhancing modality as well.

The efficient choice of first-, second-, and third-line drug strategies is an art which can be taught. The evidence base of this clinical lore varies. Clinical empiricism and repeated trials are clearly important. Tailoring choice of drug to clinical manifestation of the depressive syndrome has a long history, and both successes and disappointments have been recorded. A skilled psychiatrist knows from direct clinical observation when to use SSRI's, TCA's, and dual action drugs, and when to consider dopaminergic or hormonal approaches. Vagus nerve stimulation (VNS) has recently joined the armamentarium but its positioning as a treatment is unsettled.

**Methods:**

Most illustrative material will be shown in PowerPoint and handouts distributed. Key papers will be handed out.

**Target audience:** Psychiatric clinicians in speciality or postgraduate training.

**Workshop level:** Basic academic course in psychiatry is required

**Sponsored by:** Pharmacia Sw. AB