

period may imply a more rapid blockage of central sympathetic outflow, possibly an anti-noradrenergic effect of St. John's wort.

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### Tues-P15

#### PHARMACOTHERAPY OF DEPRESSION AND CHRONIC PAIN

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Depression and chronic pain syndrome often go together. This is confirmed by numerous clinical experiences; their mechanism of onset is often similar and the analysis of biological markers also shows that pain and depression are interrelated. Clinical similarity is often very marked, especially in the case of masked depression and various forms of chronic pain (idiopathic pain, chronic abdominal pain, tension headaches, some forms of migrainous syndrome, etc.). Prevalence of chronic pain syndrome is about 10% of general population. In this study, the sample consisted of 60 patients aged 20–74 years ( $x = 43.2$ ,  $SD \pm 12.4$ ) of both sexes. The sample is divided into two subgroups with 30 patients in each. These subgroups are homogenous in relation to clinical and sociodemographic characteristics. Among diagnostic instruments, structured interviews according to DSM-IV and ICD-10 criteria were used. If the pain lasted more than six months it was defined as "chronic". HAMD-11 was used for evaluation of results of depressive syndrome treatment, and WHO criteria for pain syndrome. The first group was treated with combination of antidepressants from SSRI's group (fluoxamin) 50–100 mgr/day and the newest generation of antiepileptics (gabapeptin) 600 mgr/day in the period of six weeks. The second group was treated with classic antidepressants (maprotiline) in the doses of 250/125 mgr/day with usual doses of nonsteroid analgesics in the same period of 6 weeks. The evaluation was done at the beginning of the treatment, and at intervals of seven days. The final evaluation showed that the effectiveness of applied therapy in the first group (fluoxamin + gabapeptin) is 65% better than in the control group (maprotiline + NAIS analgesics),  $p = 0.001$ . The improvement in the first group was observed already after two weeks of treatment, it was 41%, while the improvement in the second group was 19.2% ( $p = 0.05$ ). Possible explanation of the difference is in the fact that both syndromes are conditioned by similar neurochemical changes primarily by the change in serotonergic neurotransmitter system and partially in the gabaergic system.

### Tues-P16

#### EFFICACY AND TOLERANCE OF VENLAFAXINE IN HOSPITALIZED AND AMBULATORY PATIENTS

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Venlafaxine is a newly introduced noradrenaline/serotonin reuptake inhibitor (SNRI) antidepressant. Its efficacy and safety has been studied in various subsets of patients. This open, prospective study attempted to further investigate the safety and efficacy of venlafaxine in a large cohort of patients. One hundred and twenty six patients [30 males/96 females, mean age 49 years, hospitalised ( $n = 26$ ), ambulatory ( $n = 100$ ), newly diagnosed ( $n = 83$ ), refractory to previous antidepressants ( $n = 43$ )], with mild to moderate

(MADRS score 20–25) or severe (MADRS score > 25) major depression (DSM-IV), were enrolled in six psychiatric centers. The initial mean MADRS and 21-item HAM-D scores were 30.6 and 29.9 respectively. Repeated episodes were diagnosed in 30.6% of the patients and 91.6% had melancholic features. Patients were evaluated before and at 1, 2, 4 and 6 weeks. The initial venlafaxine dose was either 75 mg/d (mild/moderate disease) or 150 mg/d (severe) and was further titrated according to the response up to 375 mg/d.

Full response (MADRS and HAM-D scores reduced by at least 50% and CGI rating 1 or 2) was achieved by 102 (81%) patients. Seventy of them (55.6%) achieved full response by week 4. Improvement in all rating scales was statistically significant ( $p < 0.001$ ). The CGI rating at last visit was very much better in 68.1% and much better. Six patients (4.7%) discontinued therapy due to nausea (3), vomiting, sweating or vertigo in 28.3% of the patients.

The present study reconfirms the high efficacy and safety of venlafaxine in major depression.

### Tues-P17

#### PREDICTORS OF RESPONSE TO ANTIDEPRESSANT DRUG THERAPY AND THEIR SIGNIFICANCE FOR THE DURATION OF TREATMENT

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In this retrospective investigation on 659 depressive outpatients and 157 depressive inpatients, treated with antidepressants (amitriptyline, fluoxetine), benzodiazepines (alprazolam, lorazepam, or diazepam) or placebo, predictors of antidepressant drug response were evaluated. A therapeutic response was defined as a reduction of at least 50% in the Hamilton Psychiatric Rating Scale for Depression (HAM-D) at the end of the study. Neither demographic variables nor duration of the current episode, severity of depressive symptoms or baseline scores of psychiatric rating scales showed any clinically relevant relation to therapeutic outcome. It was shown that the clinical improvement according to HAM-D scale within the first two weeks of treatment was of high predictive value with respect to the final therapeutic outcome. In all, 76% of patients without a significant reduction (less than 20%) of the score in the Hamilton Psychiatric Rating Scale for Depression after 2 weeks of treatment did not fulfill response criteria after 6 weeks. The predictive value of the improvement criterion after two weeks did not depend on the treatment procedure (antidepressants, benzodiazepines, placebo). For clinical practice these results imply the necessity of reviewing the applied drug regimen early, i.e. about 2 weeks after beginning, to check for compliance with treatment or an individual problem of resorption or metabolism in order to amend the regimen by increasing dosage and/or adding sleep deprivation, lithium, thyroxine or psychotherapy. After 4 weeks of treatment without any recognizable success the therapeutic procedure should be changed and the usage of an antidepressant with different biochemical properties should be taken into account.