

/n=2/, delirium /n=1/) reoccurred after IFN- α dosing has been replaced. In the medical history of the patients we found 9 cases of psychiatric diseases: depressive disorders (n=5), somatoform disorder (n=1), agoraphobia without panic attack (n=3). In all the cases of depressions the previous mood disorders recurred during the IFN- α treatment. Patients with former agoraphobia developed panic attacks and depressive symptoms. The psychiatric sequelae occurred earlier in patients with positive medical history (mean: 2.9 months) than in other patients (mean: 6.6 months). All of the 37 patients received psychopharmacotherapy: antidepressives (n=31), anxiolytics (n=4), antidepressives plus anxiolytics (n=20), antipsychotics (n=3). In 6 cases (16,2%) the IFN- α were stopped, 7 patients (18,9%) underwent a dose reduction and 21 patients (56,7%) could continue the therapy with primary dose. 3 patients were lost from follow-up. All patients who were dropped out of IFN- α treatment fully recovered from psychiatric syndromes (delirium /n=2/, depression /n=3/, panic /n=1/).

Conclusions: Depressions were found to be the most frequent psychiatric complication, but other syndromes (especially anxiety disorders) also occurred. Our results present that previous mental disorders can hasten the outbreak of the IFN- α induced psychiatric symptoms. Psychopharmacotherapies proved to be effective, therefore dose reduction or drop-out of IFN- α was avoidable in the majority of cases. These results show the necessity of the collaboration between physicians and psychiatrists in the case of all IFN- α treated patients. The examination of CNS effects of the IFN- α can contribute the research of the cytokine network, especially its role in the pathophysiology of mental disorders.

P.1.093 The use of the PHQ as follow-up instrument for MDD in the primary care setting

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Objective: Validation of the PHQ (Patient Health Questionnaire) as follow-up instrument in primary care.

Methods: We included 643 patients with clinical diagnosis of Major Depressive Disorder (MDD). At baseline the PHQ – a self report version of the PRIME-MD with items identical to those of the DSM-IV (ref 1) – was completed (ref 2). Other PHQ-modules were also completed. After inclusion patients were treated with an SSRI and followed during 12 weeks (3 visits). The PHQ was validated using the SCL-90 (commonly used as follow-up scale for depression, anxiety and somatization). Evolution of depression and comorbidities on the PHQ and SCL-90 were studied, together with their impact on work, family and social life.

Results: 643 (71,7% female, average age 49,9 years) patients completed the study. All PHQ and SCL-90 scores (depression, anxiety, somatization) decreased significantly over time ($p < 0.0001$). The more severe the depression, the higher the PHQ scores at baseline and the faster the decrease over time. A GLMM-analysis showed that men had lower PHQ depression scores at baseline than women ($p = 0.002$) but their recovery was slower ($p = 0.006$). The same was seen at the SCL-90: men had lower SCL-90 depression scores ($p = 0.004$) but their scores decreased slower ($p = 0.007$). However, after 12 weeks of treatment similar scores were obtained for both sexes. Older patients had systematically lower depression scores ($p = 0.012$). The PHQ and SCL90 scores

were standardised on a 0–100 scale in order to compare. The evolution over time of the scales was practically the same for the sections “depression” (Table 1) and “anxiety”. For somatization there was a slightly different evolution: SCL-90 had lower somatic end-scores than PHQ. Higher scores on PHQ and SCL-90 for depression, anxiety and somatic disease all resulted in higher Sheehan 4, 5 and 6-scores. The scores decreased over time ($p < 0.0001$). In older patients ($p < 0.0001$) and patients living alone a significant slower decrease was seen on the Sheehan 4 whereas evolution of Sheehan 5 and 6 (impact on social and family life) did not depend on any covariate. The strongest evolution on all scales was seen after 4 weeks of treatment with further reduction of the scores after 8 weeks and remained stable during further treatment.

	Baseline	Week 4	Week 8	Week 12
SCL-90 mean \pm SD	100	79.5 \pm 30.2	67.9 \pm 32.0	65.0 \pm 32.1
PHQ mean \pm SD	100	79.5 \pm 21.8	69.3 \pm 24.9	64.8 \pm 23.9

Conclusion: The PHQ and SCL-90 show a comparable evolution over time, meaning that the PHQ entails comparable monitoring capacity for follow-up on depression and comorbidities as the SCL-90.

References

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P.1.094 Reduction of the brain ¹²³I-ADAM binding is related to the plasma drug level in amitriptyline treated subjects

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Background: The classical antidepressant amitriptyline (At) is a strong inhibitor of serotonin (5HT) uptake in platelets or brain synaptosomes. However, the percentage of serotonin transporter (5HTT) inhibition in the brain of patients during a typical course of At treatment is unknown.

Objective: To test the inhibiting effect of At in brain regions with high or low density of 5HTTs *in vivo* using the new high affinity radioligand ¹²³I-ADAM and SPECT. A second aim was to assess the relationship between ¹²³I-ADAM binding reduction and plasma At levels and 5HT uptake inhibition in platelets.

Material and Methods: Eleven normotensive and non-medicated subjects (5 healthy controls and 6 psychiatric patients according to ICD-10 criteria) were investigated twice, before and after 7 days of At administration (50 mg/day). SPECT images were obtained 4h post injection of ¹²³I-ADAM. Regions of interest (ROI) were drawn manually in areas corresponding to the midbrain, frontal cortex and cerebellum. The measured activities