need to be confirmed in enlarged samples and further analyses are requested in order to explore specific subgroups in each diagnostic category.

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P.2.h.011 Somatic comorbidity in psychiatric outpatients with affective and anxiety disorders

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Introduction: Psychiatric patients are a vulnerable population group to somatic diseases, and probably to greater use of medical services than the general population. Previous studies inform about association between anxiety disorders and several organic illnesses, as certain heart conditions, hypertension, gastrointestinal, genitourinary, and musculoskeletal and migraine. Also, patients with depression and anxiety have increased somatic symptoms than patients with other chronic mental illnesses [1].

Objectives: (1) To analyze the presence of somatic health problems in a sample of patients with affective disorders and anxiety. (2) To study differences in relation to sociodemographic variables.

Methods: Retrospective descriptive study of psychiatric outpatients with Anxiety or Depressive Disorder, treated in a Mental Health Unit in the Clinical University Hospital in A Coruña (Spain). Information was obtained from clinical records.

The sample is composed of all patients attended in a mental health clinic in a period of 6 months (from January to June 2012) and diagnosed by a psychiatrist of Anxiety or Depressive Disorders. N = 284.

1 – A protocol configured for sociodemographic variables, somatic comorbidity and psychiatric variables (DSM-IV-TR diagnoses) was developed.

Data were collected from medical records of patients and grouped into two categories: Affective and anxiety disorders.

2 – We performed a descriptive analysis of the sample and a cross between quantitative variables (age) and qualitative (comorbidity). As statistical significance was used analysis of variance. For the intersection between qualitative variables (comorbidity, sex and condition group) Chi square was used as statistical significance. Data were analyzed with SPSS for windows v. 17.0.

Results: The sample is composed of 284 patients (185 women and 99 men) with an average age of 31.94 (SD 10.46). The median age for men was 34.2 and for women is 30.9 years.

Somatic comorbidity was found in 29% of the sample.

Comorbidity among patients with affective disorders was 33.5%, higher than comorbidity in patients with anxiety disorders that was found in 23.4%, with statistical significance ($p < 0.05$).

In the whole sample, all somatic diagnoses were reported as more prevalent among women than among men, except myocardial infarction and hypertension. Nevertheless men with affective disorder were the group that showed greater comorbidity (38%) followed by women with the same group of disorders, without statistical significance.

Age was associated to comorbidity that was found more frequently in older patients.

The most frequent comorbidity were digestive disorders (25%), followed by respiratory (24%), cardiovascular (18%) and endocrine disorders (9%).

Conclusions: Psychiatric outpatients with affective and anxiety disorders present frequent somatic comorbidity.

Clinicians should pay attention to somatic health problems in patients with depression and anxiety as about one in three patients consulting a psychiatrist for depressive or anxiety disorders also has somatic health problems.

Active identification and treatment of these co-occurring mental disorders are of practical importance.

The relationship between specific somatic health problems, as cause of anxiety or depression must be studied in future investigations that should analyze specific depressive and anxiety disorders as risk factors for the development of somatic disorders.

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P.3.a. Psychotic disorders and treatment – Psychotic disorders (basic)

P.3.a.001 Familial liability to psychosis is associated with attenuated dopamine stress signaling in ventromedial prefrontal cortex

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Objective: Patients diagnosed with a psychotic disorder and their first-degree relatives display increased reactivity to stress [1].

Theory predicts that experience of psychosocial stress is associated both with ventromedial prefrontal and mesolimbic dopamine neurotransmission.

However, while there is evidence of aberrant striatal dopamine processing in psychotic disorder, the role of the prefrontal cortex

remains under-researched. The current study aimed at investigating stress-induced in vivo dopamine release in ventromedial prefrontal cortex (vmPFC) of individuals at familial risk for psychosis.

It was hypothesized that first-degree relatives would display impaired prefrontal regulation of stress-induced mesolimbic dopamine release, reflected by attenuated stress-induced vmPFC dopamine signaling associated with psychotic reactivity to the laboratory stressor.

Method: 14 healthy first-degree relatives (7 siblings, 6 parents, and 1 child) of patients with a diagnosis of psychotic disorder and 10 control subjects underwent a single dynamic PET scanning session after intravenous administration of 183.2 (SD = 7.6) MBq [^{18}F]fallypride, a high affinity dopamine $D_{2/3}$ radioligand.

Psychosocial stress was initiated at 100 minutes postinjection using the Montreal Imaging Stress Task [2] – a computerized mental arithmetic task with social evaluative threat components.

PET data were analyzed using the linearized simplified reference region model.

Regression analyses were performed to compare the spatial extent of task-related ligand displacement between control subjects and relatives, and how it related to self-rated experiences of psychosocial stress and psychosis.

Results: No large or significant sociodemographic differences, nor differences in subclinical psychopathology were observed between groups. Mean levels of vmPFC baseline nondisplaceable binding potential (BP_{ND}), task induced stress, psychosis, and spatial extent of [^{18}F]fallypride ligand displacement in vmPFC were similar between groups. The stress condition was experienced as significantly more stressful compared to the control condition, reflected by higher scores on the subjective stress scale.

Individuals at familial risk for psychosis displayed attenuated dopamine stress processing in ventromedial prefrontal cortex (vmPFC), a brain region previously identified to play a key role in human dopaminergic stress regulation [3]. Furthermore, increased levels of subjectively rated stress were associated with increased intensity of psychotic experiences. This effect was particularly pronounced in first-degree relatives, fitting previously reported associations between abnormal dopamine reactivity and increased stress reactivity in the daily life of subjects at familial risk of developing psychosis.

Conclusion: Although previous studies have hypothesized a role for prefrontal dopamine dysfunction in psychosis, this study, to our knowledge, is the first in vivo human imaging study showing attenuated (i.e., hyporeactive) dopamine stress neuromodulation in vmPFC of individuals at familial risk for psychosis. Development of high affinity $D_{2/3}$ radioligands, among which [^{18}F]fallypride, and advances in PET-methodology offer possibilities for further exploration of extrastriatal dopamine sites suggested to be involved in the pathophysiology of psychosis. The work presented in the current study is at an early stage, and there is an urgent need for further human in vivo studies aimed at clarifying mechanisms of pathophysiological dopamine communication between cortical and subcortical brain regions in the context of psychosis.

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P.3.a.002 Antidepressant-like effect of 1-methyl-1,2,3,4-tetrahydroisoquinoline in the rat forced swimming test: comparison with citalopram

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In the etiopathology of depression an important role plays unbalance of monoaminergic transmission in the brain. In treatment of depression are used drugs which selectively inhibit the neuronal reuptake of serotonin and noradrenaline. Unfortunately, medicines are till now have shown poor therapeutic efficiency. Thus there is an urgent medical need for a better, more effective pharmacotherapy. Citalopram is in the class of drugs called selective serotonin reuptake inhibitors (SSRI). Since uptake is an important mechanism for removing released neurotransmitters and terminating their actions on adjacent nerves, the reduced uptake caused by citalopram results in more free serotonin in the brain to stimulate nerve cells. 1-Methyl-1,2,3,4-tetrahydroisoquinoline (1MeTIQ) is an endogenous substance which is present in the human and mammalian brain. As we have shown previously, this compound exhibits neuroprotective, antiaddictive and MAO-inhibiting properties [1,3]. 1MeTIQ strongly affects on the dopamine metabolism, blocks the oxidation pathway and shifts the dopamine catabolism toward the O-methylation route. Due to this properties, 1MeTIQ was proposed as an endogenous regulator of dopaminergic and serotoninergic activity. In the present study we tested antidepressant-like effects of 1MeTIQ, Citalopram and co-administration of these drugs in the modified forced swimming test (FST) [2] in rat. Additionally, in the neurochemical studies the rate of dopamine (DA), serotonin (5-HT) and noradrenaline (NA) metabolism were established after behavioral experiments in different brain structures (hypothalamus, striatum, frontal cortex) by HPLC methodology. The data were analyzed by means of a two-way ANOVA followed by Duncan's test.

Results: The FST data has shown that 1MeTIQ in a low dose 15 mg/kg i.p evoked antidepressant-like effects, and significantly reduced the immobility time ($P < 0.01$) and concurrently produced an increase of a climbing time in rat ($P < 0.05$). Acute administration of citalopram, (20 mg/kg i.p.) also induced significant reduction of immobility time ($P < 0.01$) in FST to comparison of saline group with simultaneously elevation of swimming time ($P < 0.01$). The combined administration of citalopram and 1MeTIQ enhanced the antidepressant activity and significantly reduced immobility time in the FST. The neurochemical data demonstrated that 1MeTIQ produced a clear and significant elevation the rate of noradrenaline and dopamine metabolism in frontal cortex, striatum and hypothalamus ($P < 0.01$). In the same time, the rate of serotonin metabolism was significantly decrease by acute dose of 1MeTIQ as well as citalopram ($P < 0.01$). Co-administration of these compounds intensified the effects of 1MeTIQ and citalopram and results in a significant increase in the rate of dopamine and noradrenaline metabolism ($P < 0.01$) while a significant decrease in the rate of serotonin metabolism ($P < 0.01$).