

Sex matters – also in psychosis!

S88

Sex differences in emotional reactivity to daily life stress in psychosis

I. Myin-Germeys^{1,*}, G. Merge²¹ Leuven, Belgium² Maastricht University, Department of Psychiatry and Neuropsychology, Maastricht, Netherlands

* Corresponding author.

Background A recent study did not find clear-cut sex differences in psychotic symptoms. Studies investigating altered stress reactivity more consistently report differences between the sexes, although the results are contradicting in suggesting either men or women to be more stress-sensitive. We assessed self-reported experiences in the context of real-life to more fully understand the nature of sex differences in psychosis.

Methods We employed the Experience Sampling Method, a structured diary technique, to investigate in real-life:

- symptoms;
- behavior in context;
- underlying mechanisms in 283 healthy controls, 268 subjects at risk for psychosis and 232 patients with psychotic disorder.

Results Multilevel regression analyses revealed no differences in symptom expression between the sexes. Similarly, men and women did not differ in their level of social interaction and overall activity. However, men at increased risk of psychosis were more often alone and were less involved in goal-directed activities compared to women. Finally, women reported more emotional reactivity to daily life stress than men but women also reported more positive affect when pleasant events had happened.

Discussion The data thus suggest only minor differences between men and women in psychotic symptoms and actual behavior. However, whenever differences were apparent, they consistently pointed towards more severe symptoms and more deficiencies in men compared to women. In contrast, increased environmental reactivity in women (to both negative and positive environments) in addition to more social contacts may constitute a protective factor for the development of more severe psychopathology.

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S89

Sex and gender differences in schizophrenic psychoses

A. Riecher-Rössler

University of Basel, Psychiatric Clinics, Basel, Switzerland

Introduction Sex and gender differences in schizophrenic psychoses have often been described but treatment approaches so far have hardly taken them into account.

Objectives To describe the most important sex and gender differences in schizophrenic psychoses with clinical implications.

Methods Review.

Results Schizophrenic disorders show a later age of onset in women and a slightly better course, especially in young women. As to pathogenesis, there is some evidence that the age difference might be at least partly due to the female sex hormone estradiol being a protective factor. Differences in course might also have to do with this biological factor, but at the same time with the psychosocial advantages of a higher age of onset and other psychosocial factors.

These gender differences have important implications for assessment and therapy. Thus, we have to consider gender differences in coping behaviour as well as psychosocial burdens and needs

deriving from differing roles in partnership, family, household and profession, from dependent relationships, potential abuse and violence. Furthermore, there are specific biological risks such as gonadal dysfunction we have to deal with in both sexes differently. Thus, e.g. women with psychosis can also have very special needs regarding fertility, pregnancy and motherhood. Also, around menopause we have to consider special measures such as replacement of physiological 17- β -estradiol.

Conclusions Women, but also men, with schizophrenic psychoses should get a gender-sensitive assessment and treatment.

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S90

Menopause and psychosis

J. Usall^{1,*}, E. Huerta-Ramos²¹ Parc Sanitari Sant Joan de Déu, GTRDSM, CIBERSAM, Research Unit, Sant Boi de Llobregat, Spain² Parc Sanitari Sant Joan de Déu, Research Unit, Sant Boi de Llobregat, Spain

* Corresponding author.

There has been little research into the effects of menopause on symptoms, social and cognitive functioning in women with schizophrenia, and the results are controversial. The most replicated finding is that late-onset schizophrenia is more prevalent in women than in men and that this fact appears to be related to the diminution of estrogen levels during menopause.

Estrogens have a known protective effect on CNS. Animal research has shown that estrogen has a modulating effect on the dopaminergic, glutamatergic and serotonergic systems.

There are concerns about long-term use of sexual hormone therapy in postmenopausal women with regard to breast cancer risk, and the use of the selective estrogen receptor modulators (SERMS's) can be a better option.

Raloxifene is a SERM that is used in the preventive treatment of postmenopausal osteoporosis and has no effect in the breast and uterus. A number of studies seem to indicate that raloxifene acts on brain dopamine and serotonin systems in a similar way to conjugated estrogens.

In this presentation, I will show the results of some clinical trials that have studied the efficacy of raloxifene as a coadjuvant treatment of patients with schizophrenia. Our team has done two clinical trials that studied the efficacy of 60 mg of raloxifene for the treatment of negative symptoms in postmenopausal women with schizophrenia. Our results showed that raloxifene improved the negative symptoms better than placebo. We concluded that raloxifene seems to be a promising option to treat some patients with schizophrenia.

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Social anxiety disorder – from shyness and blushing to brains and psychotropic drugs

S91

Recent guidelines for evidence-based pharmacological treatment of social anxiety disorder

D. Baldwin

University of Southampton Faculty of Medicine, Clinical and Experimental Sciences, Southampton, United Kingdom

Pharmacological and psychological treatments The findings of meta-analyses and randomized placebo-controlled treatment studies indicate that a range of approaches are efficacious in acute treatment. Pharmacological and psychological treatments, when delivered singly, have broadly similar efficacy in acute treatment. However, acute treatment with cognitive therapy (group or individual) may be associated with a reduced risk of symptomatic relapse at follow-up. Cognitive behaviour therapy is efficacious in adults and children: cognitive therapy appears superior to exposure therapy, but the evidence for the efficacy of social skills training is less strong. It is unlikely that the combination of pharmacological with psychological treatments is associated with greater overall efficacy than with either treatment, when given alone, as only 1 of 4 studies of the relative efficacy of combination treatment found evidence for superior efficacy.

Efficacy and length of acute pharmacological treatment Antidepressant drugs with proven efficacy include most SSRIs, the SNRI venlafaxine, the MAOI phenelzine and the RIMA moclobemide: the potential efficacy of tricyclic antidepressants is unknown. Some benzodiazepines and anticonvulsants and the antipsychotic olanzapine also appear efficacious in acute treatment. A number of small single-dose placebo-controlled crossover studies together suggest that beta-blockers can be beneficial in reducing anxiety symptoms in individuals with 'performance anxiety' (for example, when speaking in public), which overlaps with mild non-generalized social anxiety disorder. Acute treatment studies indicate that the proportion of responding patients increases steadily over time. A post-hoc analysis of the clinical trial database with paroxetine indicates that many non-responders to treatment at 8 weeks become responders with a further 4 weeks of double-blind treatment: however a post-hoc analysis of the clinical trial database for escitalopram indicates that response is unlikely if there is no onset of clinical effect within the first 4 weeks of treatment.

Longer-term treatment and further treatment after non-response The findings of randomized placebo-controlled relapse prevention studies in patients who have responded to previous acute treatment reveal a significant advantage for staying on active medication (clonazepam, escitalopram, paroxetine, pregabalin, sertraline) for up to six months. Fixed-dose randomized controlled trials do not provide consistent evidence of a dose-response relationship with antidepressant drugs: but a fixed-dose study of pregabalin found that only the higher daily dosage was efficacious. A double-blind randomized controlled dosage escalation trial found no advantage for increasing to a higher daily dosage (of duloxetine), when compared to continuing treatment with a lower dosage. Switching between treatments with proven efficacy may be helpful. An uncontrolled study of augmentation of SSRI treatment with buspirone found some evidence of beneficial effects; but a placebo-controlled crossover-study of the augmentation of paroxetine with pindolol found no evidence of efficacy. A small placebo-controlled study of the augmentation of paroxetine with clonazepam found the combination was marginally short of superiority, when compared to paroxetine alone.

Disclosure of interest The author has not supplied his declaration of competing interest.

Further reading

Baldwin DS, et al. Benzodiazepines: risks and benefits. A reconsideration. *J Psychopharmacol* 2013;11:967–71.

Baldwin DS, et al. Evidence-based pharmacological treatment of anxiety disorders, posttraumatic stress disorder and obsessive-compulsive disorder: a revision of the 2005 guidelines from the British Association for Psychopharmacology. *J Psychopharmacol* 2014;28:403–39.

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S92

Oxytocin in social anxiety: An overview

I. Iancu

Tel Aviv University, Tel Aviv, Israel

Oxytocin is a neuropeptide that is synthesized in the hypothalamus. It acts as a central neurotransmitter, as well as a peripheral hormone. It is called also trust hormone or love hormone. Because of its anxiolytic, pro-social and social cognitive enhancing effects, oxytocin has been suggested as a promising novel treatment for patients with social anxiety disorder. However, controlled research is small and the studies' results are inconclusive. I will present the results of several studies with several recommendations about the role of oxytocin in social anxiety disorder. Whereas oxytocin shows some promising effects in resistant cases, of course the preferred agents are SSRIs, SNRIs and CBT.

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S93

The relationship between social anxiety, shyness and blushing

A. Pelissolo*, A. Moukheiber

AP-HP, Hôpitaux Universitaires Henri-Mondor, Department of Psychiatry, UPEC, Créteil, France

* Corresponding author.

The diagnosis of social anxiety disorder (SAD) has seen substantial changes in the last 35 years from its first appearance in the DSM-III in 1980 up to the most recent ones in the DSM-5. Throughout all these changes, this disorder, previously called social phobia, is still considered one homogenous entity with only one specifier ("performance only") introduced in the DSM-5 revision with specific fears or associated personality profiles not being considered relevant clinical markers to define SAD subtypes. However, our therapeutic experience suggested substantial particularities associated with the fear of blushing in patients with SAD. Some patients presenting this profile, historically called "erythrophobia", seem to have a very specific type of social anxiety that does not include shyness and other characteristics of classical SAD. In a study conducted in a sample of 450 new consecutive outpatients seeking treatment for SAD, we compared 142 subjects with fear of blushing without other social fears, 97 subjects with fear of blushing with other associated social fears and 190 SAD subjects without fear of blushing. The group with pure fear of blushing presented a different profile when compared with the two other groups: later age of onset, less comorbidity, lower behavioral and temperamental inhibition, i.e. less shyness, and higher self-esteem. Furthermore, from a therapeutic point of view, some specific strategies such as the Task Concentration Training have shown to be particularly effective in fear of blushing. We will further argue the validity of a possible "fear of blushing" subtype of SAD.

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