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
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http://dx.doi.org/10.1016/j.eurpsy.2016.01.907

S92

Oxytocin in social anxiety: An overview

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

http://dx.doi.org/10.1016/j.eurpsy.2016.01.908

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The relationship between social anxiety, shyness and blushing

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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.
Disclosure of interest The authors have not supplied their declaration of competing interest.

http://dx.doi.org/10.1016/j.eurpsy.2016.01.909