of parents, health problems of one of the parents, reason of referral, behavioral problems during childhood, school functioning, options offered to the adolescents before referral by the community agents and the therapeutic approaches proposed by the clinicians.

It is as yet unclear whether these findings relate to differences in the clinical characteristics of the patients or to differences in environmental and cultural approaches.

S63. Generalised anxiety disorder: facts and controversies

Chairs: HGM Westenberg (NL), J-P Lépine (F)

S63-1
No abstract received

S63-2
No abstract received

S63-3
THE NEUROBIOLOGY OF ANXIETY DISORDERS

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Pathological anxiety can be defined as exaggerated normal fear characterized primarily by hypervigilance biased to aversive stimuli and emotions related to a sense of uncontrollability and uncertainty. Clinically, a number of separate anxiety disorders can be discerned. The distinctions between these conditions, presented in the DSM-IV and ICD-10, are primarily based on clinical features.

From a neurobiological perpective, the distinctions between these diagnostic entities are less clear. Thus, patients with anxiety disorders are, regardless of their diagnosis, more sensitive to the panicogenic properties of pentagastrin and sodium lactate than healthy controls. mCPP, a 5-HT_{2C} receptor agonist, elicites anxiety in patients with panic disorder (PD) and generalized anxiety disorders (GAD) to a higher degree than in controls. The growth hormone response to clonidine is blunted in all anxiety disorder patients and SSRI's have shown to be efficacious in patients with an anxiety disorder, irrespective of the diagnostic category. On the other hand, 5-HT_{1A} receptor agonists, such as buspirone and flesinoxan, but not in subjects who qualify for PD or social anxiety disorders. Preclinical data have revealed the amygdala and its connection to play a central role in normal and pathogical anxiety. This fear circuitry evaluates the degree of threat posed by internal and external cues and it adds to the emotional coloring of interoceptive, exteroceptive and proprioceptive cues. Therefore, hyperexcitability of the amygdala could be central in the development and maintenance of pathological anxiety. Serotonergic pathways to these structures play a role in the excitability of this circuitry. The global anxiolytic effects of SSRIs could be accounted for by an amplified inhibition of this fear circuitry. The differential effects of 5-HT selective compounds are more difficult to explain, but the fact that different 5-HT receptors with opposite effects on this fear-circuitry are implicated, could explain these paradoxical findings.

S63-4
THE TREATMENT OF GENERALIZED ANXIETY DISORDER

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Generalized Anxiety Disorder (GAD) was first delineated as a distinct syndrome in the DSM-III in 1980. The logic for the break up of the prior parent disorder anxiety neurosis into 2 entities-panic disorder and GAD-was fundamentally driven by a concept of pharmacological dissection. GAD was believed to be the benzodiazepine sensitive syndrome, and was probably not sensitive to antidepressants. Panic disorder was the antidepressant responsive syndrome and not thought at the time to be sensitive to benzodiazepines. A series of international studies in the 1980s demonstrated that panic disorder was responsive to benzodiazepines as well as tricyclic antidepressants and MAO inhibitors. GAD continued to be treated with benzodiazepines and 5HT1a agonists became the other widely adopted treatment.

Recently, as the range of indications for SSRIs and SNRIs grows, several large European and US studies have been conducted on their use in GAD. The most thoroughly studied of the newer medications is Venlafaxine-ER. Two double-blind, placebo controlled, 8 week outpatient studies using Venlafaxine-XR will be presented. In the first study 377 GAD patients were randomly assigned to either placebo, 75 mg, 150 mg or 225 mg per day in a fixed dose study design. The 225 mg/day dose was significantly superior to to placebo on all outcome measures, while the 150 mg/day dose was superior to placebo on several outcome measures. The 75 mg/day dose was not significantly superior to placebo. In the second study, 405 patients with GAD who did not have comorbid major depression were randomly assigned to either placebo, buspirone 10 mgs t.i.d., Venlafaxine-XR 75 mgs/day or Venlafaxine-XR 150 mgs/day. Both Venlafaxine-XR doses separated significantly from placebo on several outcome measures, while buspirone failed to separate from placebo on any outcome measure. The results suggest that the newer antidepressants like Venlafaxine-XR are effective in the treatment of GAD.

S63-5
No abstract received

S64. Consultation-liaison psychiatry

Chairs: P Fink (DK), R Mayou (UK)

S64-1
No abstract received

S64-2
PREVALENCE OF, SCREENIG FOR AND GPS' RECOGNITION OF SOMATOFORM DISORDER IN PRIMARY CARE

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The purposes of the study were to investigate the prevalence and nature of somatization illness in primary care, to assess the general practitioner's ability to recognize somatization, and to evaluate the Whiteley Index for Hypochondriasis as a screening tool.

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