professionals (Spitzer et al 1994). At present an ICD-10 version of PRIME-MD is being developed and tested.

(1) Spitzer, R.L., Williams, J.B.W., Kroenke, K. et al.: Utility of a new procedure for diagnosing mental disorders in primary care, JAMA 272 (1994) 1749–1756

S56. The treatment of panic disorder: rationale and empirical findings

Chairs: R Rosenberg (DK), JA den Boer (NL)

S56-1
EPIDEMIOLOGY AND COURSE OF PANIC DISORDER

H. Katschnig. *Department of Psychiatry, University of Vienna, Austria*

Approximately one in sixty to one in thirty persons in the general population suffers from panic disorder at one or the other time of their life. At least, this is what the two large epidemiological studies, the ECA (1.6% lifetime prevalence) and the NCS (3.6% lifetime prevalence) carried out in the USA over the last fifteen years, suggest. In countries as diverse as Korea and Italy, Germany and Puerto Rico, or Canada and Lebanon, the rates are in the same order. Female rates are usually at least twice as high as male rates. However, lifetime prevalence is a clinically not very useful concept, since it does not tell us anything about the severity, let alone about the chronicity of a disorder. Clinically panic disorder is regarded as a chronic and even progressing condition, starting with spontaneous panic attacks which are followed by situational attacks and phobic avoidance, leading, finally, to agoraphobia and a number of comorbid conditions, such as depression and substance abuse. But how frequent is this pattern? Is panic disorder really a chronic and progressing condition? And how many patients go on to develop agoraphobia? In the light of new research some of these questions can be answered in a differentiated way. First, the 12 months and one month prevalence rates of the ECA and the NCS study reveal that a large proportion of patients must have experienced a remission of panic disorder, since 12 months prevalence rates are around two thirds, and one month prevalence rates only around one third of the lifetime prevalence rates. Second, while in clinical populations up to 80% of all panic disorder patients suffer from agoraphobia, this proportion is much smaller in epidemiological studies, where it is less than one third. Finally, while, based on ECA data, around one in ten persons experiences at least one panic attack over their lifetime and 1.6% (i.e. one in six of these persons) develops panic disorder, among those 8.6% who *do not* meet criteria for panic disorder, around one in seven develops phobic avoidance, which is usually a disabling condition. While these epidemiological data already hint at the diversity of patterns of course, prospective follow-up study demonstrate that around one third of those who have developed full panic disorder remit, 50% show a waxing and waning course, and 20% experience a severe unremitting chronic course. Duration of illness and phobic avoidance seem to be the most relevant predictors for developing a chronic course. Clinically, it is therefore important to identify those patients who will have a severe and chronic course, at an early stage in order to concentrate therapeutic resources more vigorously on them.

S56-2
COMPONENTS IN 5-HT NEUROTRANSMISSION IN DEPRESSION AND ANXIETY

J.F.W. Deakin*, C. Mortimore, P.L. Strickland, I.M. Anderson. *Neuroscience and Psychiatry Unit, Room G.907, Stopford Building, Oxford Road, Manchester, UK*

We have suggested that anticipatory anxiety involves increased 5-HT release onto 5-HT_2 receptors. This also reduces the intensity and the probability of a panic attack. We have attempted to demonstrate these effects in studies using 5% CO_2 challenge in patients with panic disorder and normal volunteers.

We have previously reported that tryptophan depletion increases ratings of anxiety in patients with panic disorder when they breathe CO_2. Now, we report that increasing 5-HT_2 function using fenfluramine causes significant increases in anticipatory anxiety but on some measures attenuates the severity of anxiety during CO_2 challenge.

Recently we have investigated whether allelic variations in 5-HT-related genes predisposed to anxiety diagnoses in 100 samples from stressed women in the community. There are no clear cut associations with variants in the 5-HT transporter or in 5-HT_{2c} or 2a receptors. However, there are some intriguing hints. It is clear that 5-HT has different functions in different components of anxiety.

S56-3
NEUROBIOLOGY OF PANIC DISORDER

J.A. den Boer[1]*, I.M. van Vliet[2], H.J.G.M. van Megen[2], H.G.M. Westenberg[2]. [1]*Academic Hospital Groningen;* [2]*Academic Hospital Utrecht, The Netherlands*

Many different provocative agents, including metachlorphenypiperazine (mCPP), CO_2, lactate, yohimbine and cholecystokinin (CCK) have been used as panicogenic agents. Most of these challenge agents lack specificity which limits their use in identifying neurotransmitter systems or receptor dysfunctions involved in panic disorder (PD). CCK clearly has advantages over other provocative agents because it induces panic attacks dose-dependently and reliably in PD. A disadvantage, however, is that it lacks diagnostic specificity as it also induces panic attacks in social phobia and obsessive compulsive disorder. In a series of studies we have shown that CCK_B receptor antagonists like L-365,260 are able to block CCK_4 induced panic attacks, mitigates lactate induced anxiety, but is without anxiolytic effects in patients suffering from anxiety disorders.

After successful treatment with the SSRI fluvoxamine, CCK_4 was unable to induce panic attacks, which suggests a relationship between CCK and 5-HT neuronal systems.

A large number of studies has shown that several SSRI's are effective in reducing panic attacks and agoraphobia in PD. A number of patients, however, appears to be resistant to treatment. In depression, preliminary data have shown that the β-adrenoceptor/5-HT_A agonist pindolol reduced the latency and potentiated the response of several SSRI's. In view of the fact that there may be a shared biological diathesis between depression and PD we conducted a study in which we treated patients with PD with either fluvoxamine or a combination of fluvoxamine plus pindolol. In this pilot study no evidence was found for an accelerating or anxiolytic response of pindolol augmentation in PD. Possible reasons for this lack of efficacy will be discussed.

https://doi.org/10.1016/S0924-9338(99)80246-6 Published online by Cambridge University Press