with use of the new standardized diagnostic instruments. This is now called comorbidity or sometimes "dual diagnosis" in relation to substance use disorders. This rediscovery is important for the treatment and prognosis of alcoholism and other substance use disorders, which have been segregated from other mental disorders for various reasons.

The magnitude of the comorbidity varies with the sampling frames used. In substance use disorders it increases with increasing severity of the disorders as manifested by treatment seeking. Also, the comorbidity is more prevalent with multiple substance abuse than with alcohol abuse only.

Recent estimates of the prevalence of comorbidity disorders with substance use in the general population indicate that up to 40% of substance abusers have another psychiatric disorder. Most prevalent are anxiety, affective, and personality disorders. The prevalence of comorbid disorders is higher among substance abusers seeking treatment reaching 80% in specialized abuse treatment settings. Again anxiety, affective, and personality disorders are most frequent as well as multiple substance abuse.

Another important aspect is substance use disorder among other psychiatric patients. Studies of psychiatric inpatients have, in addition to the mentioned diagnostic groups, identified comorbid substance abuse as significant problem among patients with psychotic disorders, especially those with schizophrenia.

The data about comorbidity indicates the importance of considering other mental disorders in the treatment and prevention of substance use disorders.

ANXIETY DISORDERS AND ADDICTIONS

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Alcohol abuse/dependence has been found the most common psychiatric diagnosis in the Epidemiologic Catchment Area (ECA) study, the largest ever personal interview general population survey in the United States. Therefore, it is not surprising that alcoholism and anxiety disorders overlap, although with little compelling evidence of a higher rate of alcoholism in groups of anxious patients. According to different studies, a great variety exists in the prevalence of the occurrence of both pathological states. Several reasons are responsible for this. First of all, denial is typical of alcoholics and they may find more acceptable (especially if female confronting male doctors) to attribute heavy drinking to a cause (like anxiety) that may elicit sympathy. Another reason is the loose definition of anxiety which has been used in the past (comprehensive of fear, irritability, restlessness and any kind of unpleasant mood state). Still more important is the timing of interview. Most alcoholics undergo a mild withdrawal syndrome including feeling of tension and anxiety, which is over in one week. The first two weeks after entering treatment, alcoholics are also realistically anxious and worried about their personal situation. For this reason, attention should be paid only to symptoms occurring after two weeks of abstinence. An accurate history to document the chronology of development of both alcoholism and anxiety states is also negatively affected by the gradual onset of alcohol problems (5–20 years), and cognitive disturbances. Specialists under-report alcoholism also for their own attitudes and drinking patterns. A limited exposure to a formal substance-abuse curriculum make them reluctant to treat alcohol withdrawal. They may be discouraged also by the time demands to their busy practice to maintain abstinence, and by the family pressure to identify a psychological cause to alcohol problems. As a result psychiatrists over-prescribe benzodiazepines or antidepressants instead of using drugs to inhibit drinking behavior. Abstinent subjects with an anxiety disorder are actually less than 10%.

S3. The neuroendocrinology of depression

Chairmen: S Checkley, W Hoogendijk

HPA REGULATION IN DEPRESSION

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In relation to the hypothalamic pituitary adrenal (HPA) axis the neuroendocrinology of depression is very similar to the neuroendocrine consequences of chronic stress in experimental animals. Both are characterized by an increased central drive of the HPA axis, impairment of its delayed negative feedback control (as tested in man in the dexamethasone suppression test), and hypertrophy of the adrenal glands. Impairment of the fast feedback of the ACTH by rapidly rising plasma corticosteroid concentration is also seen in animals exposed to chronic stress. We now report the same change in patients with depression: a progressively faster rate of hydrocortisone inhibited the secretion of ACTH in 10 healthy controls but not in 10 depressed patients of the same age and sex.

All these changes contribute to the increased secretion of cortisol which is seen in major depression. The measurement of cortisol in saliva provides a convenient way to monitor the effects of environmental stress on cortisol in depressed patients. Data will be presented on the effects of meals, arguments and intrusive negative thoughts. The importance of hypercortisolaemia in depression is emphasized by our recent finding that depression can be detected by inhibiting the synthesis of cortisol using metyrapone.

THE ROLE OF ADRENAL STEROIDS IN BRAIN FUNCTION AND DYSFUNCTION WITH PARTICULAR REFERENCE TO CORTISOL AND DHEA

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Dysregulation of peripheral cortisol is a well-known feature of a proportion of patients with major depressive disorder, though its significance is still debated. It is important to distinguish feedback (dexamethasone) insensitivity from hypercortisolaemia. There is now evidence that cortisol may have direct actions both on cerebral function and structure, including the induction of hippocampal damage. Administration of corticoids alter both cognitive ability and affective states, though whether the hypercortisolaemia of depression contributes either to symptoms or to onset is still unclear. A recent study in adolescents showed that about 30% had evening hypercortisolaemia and that this was significantly associated with the presence of co-morbid dysthymia, suggesting a distinct pathophysiological state. Abnormalities of DHEA in depression are also becoming evident. About 30% of depressed adolescents had low morning DHEA, though less than half overlapped with those showing evening hypercortisolaemia. The differences between cortisol and DHEA in both incidence and diurnal pattern suggests distinct psychopathological mechanisms. Experimental evidence suggests that DHEA might have a powerful anti-glucocorticoid action, so that low levels (from whatever cause) might accentuate cortisol-induced brain dysfunction or damage. DHEA also acts directly on GABA receptors, and this mechanism may also contribute to depressive states. These findings open up new prospects for understanding more about the contribution of disordered steroids to depression.

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