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ALCOHOLISM AND DEPRESSION: RECENT RESULTS ON THE MUTUAL INTERACTION BETWEEN ALCOHOL USE, STRESS-AXIS DYSFUNCTION, AND AFFECTIVE DISORDERS

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Alcohol intake is known to modulate plasma concentrations of neuroendocrine peptides. However, recent results suggest that the endocrine system may not only respond passively to alcohol intake, but that -vice versa- it also actively modulates alcohol intake behaviour. The most coherent body of data concerns the hypothalamo-pituitary-adrenocortical (HPA) axis, with low corticotropin releasing hormone (CRH) being associated with more intense craving and increased probability of relapse after acute detoxification. It is important to bear in mind that dysregulation of the HPA system, as observed in alcohol dependence, is also a feature of anxiety and depression, two conditions which are frequently linked with alcohol dependence and have been reported to be associated with a poor prognosis. In depression, increased secretion of CRH seems to be one crucial mechanism. It has been found as a marker of depressive symptoms, which normalises when depression is successfully treated. Hypersecretion of CRH is associated with a general hyperactivity of the HPA system, notably elevated plasma levels of ACTH and cortisol, a blunted cortisol stress response and a blunted dexamethasone suppression test. In any case, HPA dysregulation, alcohol dependence, and depression are closely interrelated. Exactly which component of this triad is the driving force behind the various neuroendocrine correlates of drinking behaviour is currently unclear, and will need to be elucidated by future research. This will allow for an enlightened choice of potentially therapeutic agents for the treatment of co-morbid anxiety, depression, and alcohol dependence, acting primarily on the HPA system.

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