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REVIEW PAPERS

THE SIGNIFICANCE OF NEUROPEPTIDES IN ALCOHOL PREFERENCE AND CONSUMPTION

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Abstract – Drinking excessive amounts of alcohol regularly for years is toxic to almost every tissue of the body. Several evidences indicate that genetic and environmental factors play significant mean in development of alcohol consumption. One of the environmental factor connected with increased alcohol intake is stress. Stress may enhance alcohol intake through its effects on the activity neuroendocrine system. Neurobiological mechanism that may contribute to increased anxiety-like behavior is a disregulation of the stress-regulatory neuropeptide corticotropin-releasing hormon (CRH). CRH enhances alcohol intake by inducing an anxiogenic effect through its interactions with the central nucleus of the amygdala and other brain regions. Hypothalamic CRH release is altered by ethanol exposure and withdrawal.

Altered secretion of β-endorphine (β-END) has been observed in alcoholism. Acute doses of alcohol stimulate the release of endogenous opioids like ß-END or enkephalins and, thus stimulates opioid receptor. Lowered β-END levels might result directly from chronic alcohol consumption.

Significantly increased neuropeptyd-leptin plasma levels was observed at the onset of alcohol withdrawal and active drinking. The pre-clinical studies have been shown an association of alcohol intake with increased TNF-α and a releasing effect of TNF-α on leptin.

NPY (neuropeptide Y) may be involved in the regulation of ethanol preference. Mutant mice lacking NPY (NPT-/-) were found to consume grater volumes of solutions containing ethanol and were less sensitive to the sedative effects of ethanol, as compared with wild-type (WT NPY +/+) mice.

Key words: neuropeptides, ethanol, consumption, ethanol dependence.