Abstract

The exact mechanism by which ethanol exerts its effects on the brain is still unknown. However, nowadays it is well known that ethanol interacts with specific neuronal membrane proteins involved in signal transmission, resulting in changes in neural activity. In this review different neurochemical alterations produced by ethanol are described. Primarily, ethanol interacts with two membrane receptors: GABAA and NMDA ion channel receptors. Ethanol enhances the GABA action and antagonizes glutamate action, therefore acting as a CNS depressant. In addition, ethanol affects most other neurochemical and endocrine systems. In regard to the brain reward system, both dopaminergic and opioid system are affected by this drug. Furthermore, the serotonergic, noradrenergic, corticotropin-releasing factor and cannabinoid systems seem to play an important role in the neurobiology of alcoholism. At last but not least, ethanol can also modulate cytoplasmic components, including the second messengers. We also review briefly the different actual and putative pharmacological treatments for alcoholism, based on the alterations produced by this drug.

Keywords

alcohol dependence, neurobiology, neurotransmitters, GABA, glutamate.