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Alcoholism and the Brain Architecture

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The progress and research in alcoholism from a neuroscience perspective over the past few years has revealed the concepts of neuroplasticity in the human brain. The original design of brain structure modification was a unidirectional approach-that is, without the chance of neuronal regeneration, degradation occurred with age or disease exemplified by our understanding of neural degradation with chronic alcoholism and repair with sobriety. Now, there is supporting evidence for the possibility of neurogenesis as part of a repair process [1]. Replacement of white matter constituents, including myelin, has recently emerged.

Observable brain pathology is another broad area of alcohol research. Use of various sophisticated imaging technologies-such as computed tomography(CT), magnetic resonance imaging (MRI), pneumoencephalography (PEG), diffusion tensor imaging, and functional MRI-have become assessment tools which have enabled detailed insight into brain structural changes and function during periods of alcohol drinking, abstinence, and relapse.

Longitudinal MRI studies of alcoholics have shown that following about one month of abstinence from alcohol, cortical gray matter, overall brain tissue, and hippocampus tissue [2-4] increase in volume. With longer-term follow-up, alcoholics who maintain sobriety may show shrinkage of the third ventricular volume or a general increase in brain size significant in frontal and temporal regions [5]. Alcoholics who relapse into drinking, in contrast, show expansion of the third ventricle and shrinkage of white matter or loss of overall brain tissue relative to that seen at study entry. Cortical white matter volume may be particularly responsive to recovery with prolonged sobriety or vulnerable to further decline with continued drinking [2, 6-9]. Over a 5 year longitudinal study, prolonged sobriety was associated with improvement or stabilization of measures of brain tissue volume. By contrast, a return to drinking was related to ventricular enlargement and cortical gray matter loss, especially in the frontal lobes, and the extent of cortical volume shrinkage correlated with the amount drunk over the five years. Several factors can reduce the probability of recovery of brain structure with sobriety, including older age, simultaneous hepatic disease, and heavier alcohol consumption, history of withdrawal seizures, concurrent smoking and malnutrition [10,11].

Limitation of alcoholic studies design in humans is that it's hard to ethically administer control over drinking alcohol and other factors in humans. However, in contrast, animal studies offer control over factors contributing to change for the better or, the worse with continued or discontinued alcohol exposure. To evaluate alcoholism, animal models will be helpful in the understanding of the brain volume changes documented in the course of human alcoholism. Enlarged lateral ventricles in animal seen after administration of alcohol are markedly similar to those observed in the alcoholic man.

It has been reported that in chronic alcoholism the genetic regulation selectively affects gray but not white matter and the fate of cortical volume. Metabolic, environmental, behavioral and genetic factors that influence restoration of neuro function have yet to be identified but can be characterized with neuroimaging. With systematic longitudinal study and meticulous, classification of people with alcohol use disorders, neuroimaging in combination with neuropsychology is a quantum leap for in vivo detection and tracking of brain systems affected by alcoholism. Neuropathology is functionally relevant as it helps in determining the brain's plasticity at different ages of alcohol exposure and withdrawal, and judges neural mechanisms of insult and recovery [12,13].

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