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Drug Dependence and Synaptic Plasticity

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Is there a relation between drug dependence and synaptic plasticity? Drugs of abuse produce a variety of CNS effects resulting in cognitive deficits which include reduced memory performance, poor performance on attention tasks and learning deficits. It has been suggested that the use of these drugs may alter normal brain functions and possibly have neurotoxic effects. Supporting this hypothesis is the association between drug abuse and decreased white matter volume. There is sufficient evidence that drug abuse causes neurotoxicity although the mechanisms have not been fully elucidated. There are a number of experimental evidences indicating that neurotrophins levels may be altered by psychoactive drugs. The most common drugs studied in relation to neurotrophins were cannabis, nicotine, amphetamines, alcohol, cocaine and opioids. The synaptic plasticity is defined as experience dependent changes in the efficacy or strength of synaptic transmission. Deficits in the production and utilization of neurotrophines can lead to a variety of CNS dysfunctions as impaired brain development and psychiatric disorders e.g., schizophrenia. Till now, no researches establish if the addictive drugs could affect the structural plasticity or not. So it is important to study the relationship between plasticity and addiction-associated behaviors and study the correlation between the excitatory synaptic transmission and drug-dependent behaviors.

Drugs of abuse can alter synaptic plasticity in the mesolimbic dopamine system, a region that is implicated in a variety of addictive behaviors. In particular, long-lasting changes at excitatory synapses in the nucleus accumbens and the VTA result from *in vivo* administration of drugs of abuse. The lateral hypothalamus (LH) sends a substantial projection to the VTA and is a critical element in motivation and reward circuits activated by drugs of abuse, including cocaine.

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