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A POSSIBLE ENDOCANNABINOID MECHANISM IN RELAPSE TO DRUG - SEEKING

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Detoxification from drug abuse is strongly threatened by the occurrence of renewed episodes of drug intake. In human addicts, relapse to drug seeking may take place even after a considerably long period from the last drug consumption. Over the last decade, the endocannabinoid system has received remarkable attention due to its unique features, including its rewarding properties closely resembling those of the most commonly abused substances and its multiple therapeutic implications. Although limited at present, evidence is now emerging on a possible participation of the endogenous cannabinoid system in the regulation of relapsing phenomena. Animal models have been extremely useful in demonstrating that, under certain conditions, CB₁ receptor stimulation may elicit relapse not only to cannabinoid seeking but also to cocaine, heroin, nicotine, alcohol, methamphetamine. Both stimulation and blockade of the central cannabinoid CB₁ receptor have proved to play an important role in drug- as well as in cue-induced reinstatement of drug-seeking behavior. Indeed, pretreatment with the CB₁ receptor antagonist rimonabant significantly prevents reinstatement of seeking behavior for several drugs. However, corroborating data on the involvement of the cannabinoid system in stress-induced reinstatement are still rather scarce. Here, we will discuss data obtained from our and other laboratories using diverse experimental approaches, to provide a comprehensive picture of the recent evidence of a relationship between the cannabinoid system and the neurobiological mechanisms leading to relapse.

References:

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