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L-Type Calcium Channel Blockers are used to Prevent Drug Addiction in the Past, Present, and Future

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Abstract

L-type Ca2+ channel (LTCC) blockers have been investigated as potential therapeutics to treat the signs and symptoms of substance addiction for the past three decades. The existence of LTCCs in the brain dopaminergic circuits, which are thought to be crucial in the emergence and expression of addictive behaviours, and the widespread use of LTCC blockers in the treatment of hypertension, which may allow for the off-label use of these drugs with good brain penetration as therapeutics for mental disorders, have both been cited as supporting evidence for this theory. Addiction can be seen as a maladaptive kind of learning in which strong memories of stimuli and behaviours connected to drugs drive compulsive drug use. We will primarily use this paradigm to concentrate on the dopaminergic system, which is known to play a crucial role in drug-associated learning.

Keywords: L- tyoe calcium; Drug addiction; Addiction

Introduction

The dopaminergic system of the brain facilitates the development of strong memories of drug-associated stimuli (sensory cues) and behavioural activities that ultimately result in compulsive drug use. The mesolimbic and nigrostriatal pathways are formed by dopamine (DA) neurons in the ventral tegmental area (VTA) and substantia nigra pars compacta (SNc), which primarily transmit dopaminergic projections to the nucleus accumbens (NAc) and the dorsal striatum, respectively. These two dopaminergic projections regulate learning and reward-driven behaviour, including as drug addiction. While the SNc-to-dorsal striatum projections of the nigrostriatal pathway will be gradually recruited during later phases where stimulus-response habits are formed with gradual loss of outcome contingency, the mesolimbic system (VTA-to-NAc pathway) is thought to play a major role in the initial learning of the stimulus-action-outcome associations [1,2].

Rodent studies on the effects of LTCC blockers on the dopaminergic system and addiction

Studies showing that the dihydropyridine LTCC blockers nimodipine and isradipine suppress cocaine-induced DA release in the ventral striatum and locomotor stimulation in rats, but other types of LTCC blockers (verapamil and diltiazem) are ineffective led to the first animal studies reporting the potential involvement of LTCCs in the dopaminergic system in the action of addictive drugs. According to a number of studies, systemic administration of LTCC blockers prevents learning of drug-induced conditioned place preference (CPP), a type of Pavlovian learning where unique contextual cues of the conditioning box are coupled with drug exposure, as well as suppresses learning of operant responding (self-administration) for cocaine and morph [3,4].

How does the function of LTCCs in the dopaminergic system relate to these observations? Dihydropyridine LTCC blockers have been shown in several investigations to have inhibitory effects on the tonic pacemaker firing of DA neurons recorded in mouse brain slice preparations. In addition, these investigations described how these LTCC blockers inhibited burst firing caused by K+ channel blocking or LTCC activation, in which repeated bursts are seen during enhanced membrane potential oscillations [5, 6]. These data support the theory that LTCC blockers limit drug-induced DA release by inhibiting DA neuron activity, which prevents DA-dependent synaptic plasticity in the NAc and dorsal striatum from developing [7, 8].

Conclusion

Researchers have investigated the effects of LTCC blockers used as antihypertensives on measurements of physiological effects (such as psychostimulant-induced hypertension) and psychological effects (such as subjective sensation) in people in parallel with the rodent studies mentioned above. Early research in this field assessed the impact of several LTCC blockers on the effects of cocaine. Rowbotham et al. (1987) gave diltiazem to the patients [9, 10].

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Conflict of Interest

None.

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