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ACETALDEHYDE ENDOCANNABINOIDS AND DOPAMINE: IS THIS –JUST- “A MENAGE A TROIS”?
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As ethanol, its first metabolite, acetaldehyde (ACD), enhances dopamine neurotransmission and exerts rewarding and motivational effects in animal models tailored for studying addictive-like behaviours. The endocannabinoid system fine-tuning dopamine cell activity, affects distinct drug-related behaviours and specific drug-induced effects. In light of this, it becomes urgent to investigate the implications of a direct manipulation of the DAergic synapse, and the contribution of the endocannabinoid system on oral ACD self-administration in rats. ACD drinking-behaviour was evaluated in an operant paradigm consisting of acquisition and maintenance; extinction; deprivation and relapse; conflict. D2-receptor agonists, quinpirole (0.03 mg/Kg, i.p.) and ropinirole (0.03 mg/Kg, i.p.), and CB1-receptor antagonist, AM281 (1 mg/Kg, i.p.), were administered during the different phases of the paradigm. Our results show that oral ACD readily induced the acquisition and maintenance of an operant drinking-behaviour, even during reinstatement and conflict. Quinpirole decreased lever presses for ACD during extinction (p < 0.05) and relapse (p < 0.01; p < 0.001) Ropinirole, administered during abstinence, reduced ACD intake during reinstatement (p < 0.001). AM281 significantly decreased lever presses for ACD during extinction (p < 0.001), relapse (p < 0.001) and conflict (p < 0.001). These data suggest that whereas the direct modulation of the dopaminergic synapse influences drug-seeking and relapse behaviour, the endocannabinoid system may also play a role in shock-paired compulsive ACD intake, probably also through the involvement of the NPY-ergic system. These findings highlight the mandatory need for further investigation on the “addiction-scavenger” potential played by the endocannabinoid system taking into account its modulatory role in classic and peptidergic neurotransmission.