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We previously developed rat experimental models based on the conditioned place preference (CPP) paradigm in which only four 15-min episodes of dyadic social interaction with a sex- and weight-matched male Sprague Dawley (SD) rat (1) reversed CPP from cocaine to social interaction despite continuing cocaine training, and (2) prevented the reacquisition/re-expression of cocaine CPP. In a concurrent conditioning schedule, pairing one compartment with social interaction and the other compartment with 15 mg/kg cocaine injections, rats spent the same amount of time in both compartments and the most rewarding sensory component of the composite stimulus social interaction was touch (taction). In the present study, we validated our experimental paradigm in C57BL/6 mice to investigate if our experimental paradigm may be useful for the considerable number of genetically modified mouse models. Only 71% of the tested mice developed place preference for social interaction, whereas 85% of the rats did. Accordingly, 29% of the mice developed conditioned place aversion (CPA) to social interaction, whereas this was true for only 15% of the rats. In support of the lesser likelihood of mice to develop a preference for social interaction, the average amount of time spent in direct contact was 17% for mice vs. 79% for rats. In animals that were concurrently conditioned for social interaction vs. cocaine, the relative reward strength for cocaine was 300-fold higher in mice than in rats. Considering that human addicts regularly prefer drugs of abuse to drug-free social interaction, the present findings suggest that our experimental paradigm of concurrent CPP for cocaine vs. social interaction is of even greater translational power if performed in C57BL/6 mice, the genetic background for most transgenic rodent models, than in rats.

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Cocaine abuse has significant and varied implications for the prognosis and long-term outcomes of cardiovascular disease (CVD) and diabetes. Single markers of these diseases may lack predictive power, but a state of inflammation and insulin resistance may potentiate the effect of cocaine abuse on cardiovascular and diabetes-related outcomes. The objective of this study is to characterize and quantify the systemic inflammation and insulin resistance that could potentially be increased by cocaine abuse. We examined two chronic cocaine administration regimens (100 mg/kg every 6 hours for 3 days and 5 mg/kg every 4 hours for 4 weeks), a cocaine-abstinent state and a matched placebo-treated control group, for blood markers of systemic inflammation and insulin resistance. Chronic cocaine was associated with increased levels of plasma C-reactive protein, intercellular adhesion molecule-1 (ICAM-1), and soluble vascular cell adhesion molecule-1, as well as lower levels of adiponectin and insulin-like growth factor-1 (IGF-1). These associations remained unchanged, suggesting that chronic cocaine administration potently induces systemic inflammation and insulin resistance. These effects of chronic cocaine on inflammation and insulin resistance were largely lost after acute cocaine cessation. Systemic inflammation and insulin resistance are both components of an inflammatory state that is a risk factor for CVD and diabetes. Chronic cocaine administration may be a mechanism by which cocaine may contribute to the accelerated rates of CVD and diabetes. One of the cocaine's brain metabolic targets is monoamine oxidase-A (MAO-A) in the brain, which is thought to be the most important site of cocaine clearance. However, it remains unknown how changes in MAO-A activity might modulate acute and chronic cocaine's effects. In this study, we used a genetic approach to inhibit or activate MAO-A in mice. For acute effects, mice were treated with 40 mg/kg cocaine and whole-brain gene expression was evaluated by microarray. 5ec8ef588b

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