Abstract

Executive Dysfunction and Reward Dysregulation: Interactions in Drug Addiction

by

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Cocaine addiction is a serious public health hazard, and contributes to disastrous outcomes for individuals who suffer from it. Addiction is accompanied by an inability to control one's own behavior, and a preoccupation with cocaine at the expense of other rewarding pursuits. Previous research has suggested that difficulties with executive function and reward processing may underlie these problems, but the extent to which each contributes to addiction severity, or how these two factors may interact, remains to be elucidated. By using event related potential (ERP) measures in combination with information about self-reported anhedonia over three experiments, we set out to more clearly define the phenotype of cocaine addiction and to investigate the extent to which executive dysfunction and reward dysregulation are associated with addiction severity. A model was designed to examine these factors. In addition, in a fourth study we investigated the integrity of executive functioning in both neutral and emotional contexts in abstinent cocaine users. We found that cocaine users show much more anhedonia than controls, and this anhedonia is associated with addiction severity. In addition, anhedonia is associated with poorer ability to monitor behavior when working toward reward, with increased reward motivation in controls and cocaine users, and also with reduced consummatory reward response in cocaine users. Intriguingly, however, anhedonia is not associated with executive function deficits that are found in cocaine users, and these same executive function deficits are not associated with addiction severity. Finally, we show these executive function deficits to be normalized in abstinent cocaine abusers, and show that abstinent cocaine abusers do not modulate inhibitory response in response to emotional stimuli. Combined, these findings suggest that addiction is a phenotype defined by the presence of both reward dysregulation and executive dysfunction, and that reward dysregulation especially is associated with increased severity of the syndrome. These findings are then discussed in terms of a possible mechanistic model.