

Previous research has indicated that drug abuse has significant impacts on astrocytes. Morphological deficits as well as reductions in synaptic colocalization are observed in nucleus accumbens (NAc) core astrocytes following long access (LgA; 6 h/d) cocaine self-administration and subsequent 45-day abstinence. Despite these pronounced effects, the underlying mechanisms of these effects remain unknown. Based on previous literature, there is an emerging hypothesis that chronic dopaminergic stimulation mediates cellular adaptations resulting in the atrophic phenotype. To begin exploring this, we employed a yoked paradigm involving LgA intravenous cocaine self-administration and yoked cocaine to determine if volition was a requirement to induce the abstinence-dependent morphological and synaptic colocalization deficits associated with LgA cocaine self-administration.

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