Title

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Objective: Increased alcohol consumption amplifies the risk for substance use disorders, neural injury, and neurocognitive dysfunction. Although at-risk alcohol use, defined as drinks per day rates of >3 for women and >4 for men, is prevalent in methamphetamine (MA) users, it is unclear whether this elevation modulates MA-associated neurocognitive impairment. We hypothesized that persons who reported MA-dependence (MA+), per DSM-IV criteria, and lifetime rates of at-risk alcohol use (ALC+) would exhibit more prominent neurocognitive deficits than individuals who met criteria for only one, or none, of these conditions.

Methods: Neuropsychological and substance use assessments were administered to four groups: MA+/ALC+ (n=53), MA+/ALC- (n=34), MA-/ALC+ (n=37), MA-/ALC- (n=80). Groups were compared univariately on a global deficit score (GDS) based on demographically-corrected neurocognitive test performances, and on rates of global neurocognitive impairment (NCI) based on GDS≥0.50. Linear and logistic regressions predicted GDS and NCI from substance group status, controlling for estimated premorbid verbal IQ (VIQ).

Results: Between-group differences were observed for mean GDS ($F_{3,200}$ =5.24, p<.01) and NCI rates (χ^2 =11.37, p<.01). As expected, the MA-/ALC- group displayed the lowest (best) GDS [mean (SD)=0.22 (0.25)] and NCI rate (12.5%). The MA-/ALC+ and MA+/ALC+ groups displayed intermediate GDS and NCI rates [0.33 (0.31), 24.3% and 0.34 (0.36), 18.9%, respectively] while the MA+/ALC- exhibited the worst neurocognitive performance [0.43 (0.33), 41.2%]. Adjusting for premorbid VIQ,

substance group status predicted GDS ($F_{3,199}$ =3.34, *p*<.05) and likelihood of NCI (χ^2 [3]=9.54, *p*<0.05). For MA+/ALC-, follow-up analyses indicated higher GDS than MA-/ALC- (*t*=3.07, *p*<.01) and likelihood of NCI than MA-/ALC- (OR=4.08, *p*<.01) and MA+/ALC+ (OR=3.38, *p*<.05).

Conclusions: Whereas heavier drinking increased the likelihood of neurocognitive deficits in the absence of MA-dependence, at-risk alcohol use was associated with reduced likelihood of neurocognitive problems in MA-dependent individuals. Given the known neurotoxic and neurobehavioral consequences of high alcohol use, these results must be interpreted with caution. Our findings, however, are consistent with prior studies suggesting that alcohol-induced neurovascular and metabolic processes may attenuate stimulant-associated neural injury. Further examination of neurophysiologic mediators (e.g., brain temperature during combined use), as well as potential neurobehavioral confounds (e.g., cannabis use), is warranted to elucidate whether alcohol may confer a degree of neuroprotection in MA-dependence.

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