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Preliminary examination of the role of striatal dopamine function in risk-taking behavior in binge drinkers
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Purpose: Binge drinking accounts for almost ¾ of US economic costs related to excessive alcohol consumption and is associated with profound public health consequences [1,2]. Nevertheless, surprisingly little is known about underlying biobehavioral mechanisms. In this study, we evaluated the role of striatal dopamine (DA) function in this behavior.

Methods: Forty-four healthy M/F drinkers, ages 18–29 years, completed the Iowa gambling task (IGT) and underwent two 90-min PET studies with [11C]Craclopride (RAC). Twenty-one (M = 16) were classified as non-bingers and 23 (M = 13) were classified as bingers based on a history of at least one binge episode in the past year. The first scan was preceded by injection of 10 ml 0.9% NaCl; the second by 0.3 mg/kg amphetamine (AMPH). D2 receptor nondisplaceable binding potential (BPND) was obtained by multilinear reference tissue method (MRTM) [3]. ΔBPND was estimated as % change from the placebo to the AMPH scan. Six volumes of interest (VOIs) were defined: left (LvS) and right (RvS) ventral striatum, anterior (aPU) and posterior (pSU) putamen, and anterior (aCN) and posterior (pCN) caudate nucleus.

Results: The two groups did not differ by gender, age of first drink or first intoxication, sensation-seeking traits, or IGT performance. However, bingers consumed a greater quantity of alcohol than non-bingers, 2.7 (3.2) vs. 0.46 (0.57) drinks/week (p = 0.003). ΔBPND was lower in bingers than in non-bingers in all VOIs, but differences were not significant. The two groups also did not differ in baseline BPND or subjective drug effects. Nevertheless, findings of regression analyses adjusted for gender and drinks/week revealed significant interactions (p < 0.003) between IGT scores and binge drinking status in predicting regional ΔBPND. Findings of subsequent stratified analyses showed that greater risk-taking was associated with greater DA responses in the aPU (p = 0.001) and aCN (p = 0.005) in bingers, whereas no relationship was found between IGT scores and ΔBPND in non-bingers.

Conclusion: Although preliminary, the findings are consistent with evidence of DA involvement in risk-taking in rodents [4,5] and of altered corticollimbic brain activation during decision-making tasks in binge drinkers [6,7]. The findings suggest that risks for binge drinking may be related more to behavioral effects of altered DA activity in associative regions mediating action-outcome contingencies than to drug sensitivity per se.

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References

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