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Adolescent Drug Abuse, Vasopressin and Aggression: Divergent Drugs Yet Convergent Paths to the Development of the Aggressive Phenotype

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In the clinical literature, exposure to drugs of varied classes, including anabolic/androgenic steroids (AAS) and psychostimulants such as cocaine, is linked to increases in irritability, aggression, and violence in adolescent males. Yet, few preclinical studies have examined the impact of developmental exposure to these substances on biobehavioral processes regulating aggression. In recent studies we have shown that exposure to AAS or cocaine during pubertal development facilitates the generation of animals possessing a highly aggressive phenotype. From a behavioral standpoint, these pharmacologic models are particularly useful for the study of escalated aggression as drug treated animals display intense and appropriately targeted agonistic responses in the absence of established social interactions and cues, implicating the direct activation of neural mechanisms controlling aggressive responding, i.e., the anterior hypothalamic-arginine vasopressin (AH-AVP) neural system. Indeed, adolescent AAS- and cocaine- treated hamsters displayed highly escalated offensive aggression that could be reversed by blocking the activity of AVP receptors within the AH, implicating a role for AH-AVP in adolescent-drug induced aggression. Perhaps developmental exposure to these vastly different drugs of abuse facilitate aggression by enhancing the expression and/or activity of AH-AVP? While aggressive, adolescent AAS-treated animals showed significant increases in AH-AVP neural development and expression, their aggressive, cocaine-treated counterparts showed no differences in AH-AVP development and/or peptide levels. However, aggressive animals from both drug treatment groups showed increased AVP release within the AH. Together, these data suggest that adolescent AAS- and cocaine- exposure increases aggression by enhancing AVP activity within the AH, providing direct evidence for a causal role of AH-AVP in adolescent drug-induced aggression. A model for how alterations in AH-AVP neural functioning may facilitate the development of the aggressive phenotype in adolescent-drug exposed animals will be presented.

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