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Interactive pathways to substance abuse☆

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HIGHLIGHTS

- A model that more specifically predicts who is most vulnerable to developing addiction was offered.
- The relationship between adverse childhood events (ACE) and substance abuse was mediated by attachments and other variables.
- The path model showed new approaches to RDoC diagnoses and treatments.
- Important relationships between nomothetic, ideographic, and ecological variables were identified.
- Psychological correlates to biological reinforcement deficits were proposed.

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1. Introduction

Proponents of Research Domain Criteria (RDoC) have called for greater specifications at all levels of analysis, encouraging connections from biological to epidemiological levels and special emphases on interactions within and between these systems. However, although such interactions are valuable focuses of study, it is also important to retain a sensitivity to the individual differences between patients important to clinicians (Cuthbert, 2014; Maj, 2014). Thus, innovative and comprehensive theories of addiction should account for individual differences while maintaining strong relations between all levels of analysis. With these emphases in mind, a dynamic systems approach to interactions within and between cognitive, affective, and ecological systems intimately tied to biological systems of addiction is proposed and tested.

Although several precursors to substance abuse have been identified by previous studies, including adverse experiences in childhood, insecure attachment patterns, associations with substance using peers, and partner difficulties, few have tested relations between these sets of variables. Further, few studies have used rigorous path modeling techniques that better specify who is most susceptible to developing such disorders. The present investigation attempted to provide stringent tests of relations between these variables through a variant of structural equation modeling that analyzes several factors in interaction while maintaining sensitivity to individual differences.

1.1. Childhood trauma and substance abuse risk

Evidence for the association between early stress and later substance use has been recorded from studies conducted at multiple levels of analysis. Epidemiological evidence has found relationships between early trauma and the development of substance use disorders in adulthood (SAMHSA, 2016). Dube, Felitti, Dong, Chapman, et al. (2003) found a significant positive graded relationship between the number of traumatic events experienced in childhood and the likelihood of substance abuse in adulthood, as measured by the Adverse Childhood Events questionnaire (ACE).

Relationships between early stressful experiences and later addictive behavior have also been found at biological levels of analysis. Enoch (2011) has identified epigenetic interactions between childhood trauma and the expression of addictive phenotypes. A series of complex genetic changes are theorized to underlie the development of drug addiction, such that acute drug exposure produces epigenetic alterations that modify gene regulation and encourage abuse and development of addiction. Epigenetic modifications brought about by drug use, such as

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DNA methylation and histone acetylation, can interact with vulnerable genotypes and other environmental factors so that those who possess susceptible alleles and experience stress during sensitive periods are more prone to becoming addicted (Bodetto et al., 2013; McQuown & Wood, 2010; Wong, Mill, & Fernandes, 2011). Childhood trauma, such as parental neglect, is also associated with methylation and acetylation of genetic substrates, suggesting a molecular additive effect or interaction as a predictor of vulnerability to addiction, expressed uniquely by those experiencing childhood or adolescent stress and/or possessing addiction-prone genotypes (Unternaehrer et al., 2015; Xie, Korkmaz, Braun, & Bock, 2013).

Work in this area often implicates the hypothalamic-pituitary-adrenal (HPA) axis and the brain's mesocorticolimbic stress reactive and reward systems as targets of these genetic interactions. Blomeyer et al. (2008) found that adolescents homozygous for a particular allele of corticotropin releasing hormone receptor 1 were more likely to become heavy drinkers, and that this relationship was strengthened when they were exposed to stressful events. In rodent studies, in which early life stress was experimentally induced, chronically stressed young rats were more likely to self-administer cocaine as adults and demonstrated an altered HPA axis response to stress stimuli (Sinha, 2001). Similar effects have been observed in primate models and supported by correlational studies in humans (Enoch, 2011).

The mesocorticolimbic system, consisting of diffuse dopaminergic projections throughout the midbrain, limbic system, and cortex, is tightly linked with multiple classes of addiction as well as reward levels and functioning in social relationships. This system has evolved to evaluate the reward value of environmental stimuli, such as social interaction and food, for reward value. The mesocorticolimbic system can be "hijacked" by drugs of abuse, which most often over-stimulate dopaminergic circuits leading first to disproportional reward value placed on drug use and later to negative withdrawal states following biological compensation for exaggerated activation (e.g. receptor downregulation) (Koob & Le Moal, 2001; Volkow, Baler, & Goldstein, 2011). Baseline mesocorticolimbic functioning varies between individuals as a function of genotype, environmental factors, and interactions between them so that drug intake more significantly predisposes some to the dopaminergic alterations associated with addiction (Yacubian & Büchel, 2009). Indeed, early life stress in rats led to changes in dopaminergic signaling in the ventral tegmental area/nucleus accumbens neural pathways and altered responses to psychostimulants in adulthood (Enoch, 2011).

However, while robust correlations between vulnerable genotypes, early stress, and substance use have been found, many genetically vulnerable individuals who experience trauma never demonstrate dysfunction in adulthood (Enoch, 2011). We theorized that parental attachment patterns and peer influences might act as important mediators of the relationship between genetic vulnerability/adverse childhood events and substance use disorders in adulthood.

1.2. Attachment theory, adolescence, and substance abuse

Relationships between early trauma and later psychopathology have been theorized to be mediated by attachments (Cicchetti & Doyle, 2016). According to Lindberg, Fugett, and Carter's (2015); Lindberg, Fugett, Adkins, and Cook's (2015) theories, lowered levels of security are also predicted to exacerbate many clinical issues (such as anxiety, anger, shame, mistrust, etc.). Koob's (2013) "anti-reward" theory of addiction highlights the importance of the acquisition of a negative allostatic state as a predictor of drug dependence. We theorize that, in humans, this negative allostatic state may manifest as clinical issues and insecure attachments alongside its production of withdrawal states. Thus, in addition to priming the individual to be more responsive to addictive substances for positive affect (Baker , Piper, McCarthy, Majeskie, & Fiore, 2004; Blomeyer et al., 2008; Enoch, 2011; Koob & Le Moal, 2001; Volkow et al., 2011), it is theorized that insecure attachment behaviors and related clinical issues are the cognitive, affective, and behavioral counterparts of Koob's (2013, 2015) anti-reward model.

Evidence for the importance of childhood attachments in the development of substance use disorders has been repeatedly supported through correlational evidence (Caspers, Cadoret, Langbehn, Yucuis, & Troutman, 2005; Hawkins, Catalano, & Miller, 1992; Molnar, Sadava, De Courville, Colin, & Perrier, 2010; Schindler et al., 2005; Thorberg & Lyvers, 2006, 2010). Experimental animal models pointing to causal connections have converged on these findings as well. For example, it has been found that maternally deprived monkeys were more likely to develop patterns of drinking resembling those of human alcohol abusers (Higley, Hasert, Suomi, & Linnoila, 1991). Indeed, animal models of early life adversity as predictors of substance abuse often utilize social strain to simulate stress (Moffett et al., 2007).

In line with more recent attachment theory (Lindberg, Fugett, & Thomas, 2012), and animal models of substance use (Spear & Varlinskaya, 2010; Volkow et al., 2011; Yacubian & Büchel, 2009), it has been demonstrated that a sensitive period of growth occurs during adolescence and early adulthood. Adolescent maturation is accompanied by neural modifications involving the refinement of synaptic structure and functioning (Spear, 2000). The mechanisms responsible for this restructuring are theorized to be similar or identical to those alerted by stress and drug use, suggesting that the adolescent brain responds uniquely to these stimuli and is especially vulnerable to developing drug dependence (Andersen & Teicher, 2009; Selemon, 2013). Two sets of crucial social relations during this period must therefore be considered: partner relationships and peer relations. Although several classic studies have pointed to the importance of stable marriages as protective factors in the development of substance dependence, (Vaillant, 1995), more recent evidence for late adolescence as a sensitive period for attachment has come from physiological work, which suggests that the neural circuits involved in social behaviors and cognitions overlap with those involved in the addictions (Spear & Varlinskaya, 2010; Volkow et al., 2011; Yacubian & Büchel, 2009). As discussed above, this work theorizes that alcohol and other drugs serve as "super-normal" stimuli for the brain's reward and security centers normally responsive to evolutionarily valuable events, including healthy social interactions and relationships (Lindberg et al., 2015; Koob & Le Moal, 2001). Another influential set of factors to consider in this interacting model are variable peer influences in adolescence, a topic to which we now turn.

1.3. Peer influences

As discussed above, adolescence appears to be a sensitive period in the development of substance use disorders, during which significant neural growth in reward/security centers occurs, sensitizing the young brain to the effects of alcohol and drugs (Spear, 2000, 2007, 2011). Thus, in addition to partner attachments, it is also important to consider the broad social peer contexts in which substance abuse develops. Peer influences have repeatedly been shown to act as significant factors in the development of deviant behavior and substance use, (Hops, Andrews, Duncan, Duncan, & Tildesley, 2000; Kandel, 1978). It can be further theorized that growing up in a stressful environment can steer a child toward frequent interactions with substance using peers. When combined with recurrent substance use, these peer relations may also provoke problems with romantic partners, furthering the use of substances as substitute security mechanisms. In line with these ideas, a dynamic cascade model (Dodge et al., 2009) of longitudinal data identified parenting problems and peer contexts as important variables in the development of substance abuse. However, because this cascade model did not include measures of partner relations or strong measures of adverse childhood events or parental attachments, these potentially significant factors went untested.

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