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Behavioral processes and risk for problem substance use in adolescents

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Abstract

This narrative review examines associations of delay discounting, response inhibition, sensation-seeking, and urgency with adolescent problem substance use. Each of these processes is linked to adult substance use disorders, is associated with conditions linked to increased risk for adolescent problem substance use, and predicts problem substance use. Notably all processes are linked to early life adversity (ELA) exposure and most appear to help explain links between ELA exposure and problem substance use. These findings are consistent with a growing body of literature indicating ELA interferes with the development of neural circuits crucial to cognitive functioning and emotion regulation. Further, developmental trajectories of these processes generally align with maturational imbalance hypotheses of adolescent risk. Ongoing and pending large longitudinal studies may be essential for better understanding how ELA and other influences shapes these processes and the role of these processes in risk for problem substance use in adolescence and beyond. Finally it is possible that risk-related processes may be useful metrics in the context of implementing and evaluating strategies to prevent problem substance use in adolescence.

1. Introduction

Adolescence is a key risk period for the emergence and progression of problem alcohol and other drug use (Grant & Dawson, 1997; Webb, Baer, McLaughlin, McKelvey, & Caid, 1991). Initial use often emerges in mid to late teens (SAMHSA, 2019), and adolescents with earlier onset substance use may be at higher risk for developing a substance use disorder (SUD) and other poor outcomes (Chen, Storr, & Anthony, 2009; Conde, Peltzer, Gimenez, & Cremonte, 2020). Substance use typically peaks in late adolescence and early adulthood, and then declines by the late 20s and early 30s (SAMHSA, 2019). Similarly, risk for developing an SUD typically peaks by early 20's and then subsequently declines (Hingson, Heeren, & Winter, 2006; Winters & Lee, 2008).

Corresponding to this period of heightened risk, adolescence is also associated with the development of specific behavioral processes linked to increased risk for problem substance

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use. This narrative review focuses on delay discounting, response inhibition, sensation-seeking, and urgency. Below I first briefly review key populations at high risk for problem substance use in adolescence. Next I describe delay discounting, response inhibition, sensation-seeking, and urgency and review their associations with substance use disorders, high risk populations, and adolescent substance use outcomes. Finally I discuss implications of early life adversity (ELA) being associated with these process, potential interactions among these processes in the context of maturational imbalance hypotheses of adolescent risk, and potential future directions for research in this area.

2. High risk adolescent populations

While adolescents in general are at increased risk for developing drug and alcohol problems, this risk is amplified in certain populations. Adolescents with family histories of alcohol or other SUDs are approximately 4 to 8 times more likely to develop an alcohol or other drug use disorder themselves (Cloninger, Bohman, & Sigvardsson, 1981; Merikangas et al., 1998; Russell, 1990). This family-history associated risk is amplified in individuals with more biological parents, grandparents, and other family members with SUDs (Dawson, Harford, & Grant, 1992; Stoltenberg, Mudd, Blow, & Hill, 1998). This elevated risk is directly linked to a pattern of phenotypic characteristics that includes increased impulsivity and externalizing and internalizing traits (Acheson, Vincent, Cohoon, & Lavallo, 2018; Haeny et al., 2020), commonly referred to as behavioral undercontrol (Sher, Walitzer, Wood, & Brent, 1991) or neurobehavioral disinhibition (Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004). Similarly, externalizing disorders such as attention deficit hyperactivity disorder (ADHD) and conduct disorder or subclinical increases in externalizing traits are linked to increased risk for problem substance use in adolescence (Groenman, Janssen, & Oosterlaan, 2017; Iacono, Malone, & McGue, 2008; Koelch, Dopfner, Freitag, Dulz, & Rosler, 2019; Zulauf, Sprich, Safren, & Wilens, 2014). Additionally, internalizing disorders such as major depression or subclinical increases in internalizing traits also increase risk for adolescent problem substance use (Groenman et al., 2017; Hussong, Jones, Stein, Baucom, & Boeding, 2011; Mason, Stevens, & Fleming, 2020), although there is somewhat mixed evidence when co-occurring externalizing traits are controlled for (Hussong, Ennett, Cox, & Haroon, 2017). Further, both having a family history of SUD and having increased externalizing and internalizing traits overlap with experiencing increased ELA (Acheson et al., 2018; Fenton et al., 2013; Kendler et al., 2012; Konstenius et al., 2017; McLaughlin, Sheridan, & Nelson, 2017), and ELA itself is linked to increased risk for problem substance use (Andersen & Teicher, 2009; S. R. Dube et al., 2003; Shanta R Dube et al., 2006; Felitti et al., 1998). How these factors contribute to risk for adolescent problem substance use is not fully understood, but growing evidence indicates the processes reviewed below may be playing significant roles.

3. Delay discounting

3.1. Definition, assessment, and SUD association

Delay discounting refers to how delayed outcomes such as rewards are often discounted or valued less than more immediate outcomes (Rachlin, Raineri, & Cross, 1991). Delay

discounting is typically measured with procedures where participants choose between sooner smaller and larger later hypothetical monetary rewards (see Lempert, Steinglass, Pinto, Kable, & Simpson, 2019 for review). These choices are used to estimate discount curves of how the value of delayed monetary rewards decrease with increasing delay. Typically values of delayed rewards decrease more rapidly over the first several weeks to months and then decline more slowly with longer delays.

While some degree of delay discounting is normal, problem substance use is reliably associated with increased discounting of delayed rewards (Amlung, Vedelago, Acker, Balodis, & MacKillop, 2017; Bickel, Koffarnus, Moody, & Wilson, 2014; MacKillop et al., 2011). A meta-analysis of delay discounting and addictive behaviors (alcohol, tobacco, cannabis, stimulant, and opioid use as well as gambling) found strong evidence for increased delay discounting in individuals with addictive behaviors, especially for those with addictive disorders (MacKillop et al., 2011). A subsequent meta-analysis examining continuous associations of delay discounting with these same addictive behaviors found delay discounting was robustly associated increased addiction severity and greater quantity and frequency of use (Amlung et al., 2017). Based on the extensive findings linking increased delay discounting and problem substance use, increased delay discounting has been proposed as a behavioral marker and a behavioral endophenotype of addictive disorders (Bickel et al., 2014; MacKillop, 2013; Mitchell, 2019).

3.2. Alterations in SUD vulnerable populations

In normally developing youths, delay discounting decreases across adolescence and early adulthood corresponding with maturation related changes in frontal striatal circuitry (Achterberg, Peper, van Duijvenvoorde, Mandl, & Crone, 2016; Olson et al., 2009; Steinberg et al., 2009), and increased delay discounting has been observed in at risk populations. Delay discounting is increased in children, adolescents, and young adults with family histories of alcohol and other SUDs (Acheson et al., 2018; Acheson, Vincent, Sorocco, & Lovallo, 2011; Dougherty et al., 2014; Dougherty et al., 2015; VanderBroek, Acker, Palmer, de Wit, & MacKillop, 2016), supporting the notion that increased delay discounting may be an endophenotype for SUDs (MacKillop, 2013; Mitchell, 2019). Increased delay discounting has also been found in children and adolescents with ADHD (Patros et al., 2016) and conduct disorder (Fanti, Kimonis, Hadjicharalambous, & Steinberg, 2016; White et al., 2014). A recent meta-analysis found increased delay discounting was also associated with major depressive disorder as well as schizophrenia, borderline personality disorder, bipolar disorder, bulimia, and binge eating disorder (Amlung et al., 2019). Consistent with these findings, increased delay discounting has been proposed as a trans-disease process for multiple psychiatric disorders (Bickel et al., 2019).

Increased delay discounting is also linked to ELA exposure (Gondre-Lewis et al., 2016; Kang et al., 2017; Lovallo et al., 2013; Oshri, Liu, Duprey, & MacKillop, 2018; Simmen-Janevska, Forstmeier, Krammer, & Maercker, 2015). Studies in elderly ELA exposed populations suggest ELA induced delay discounting increases may persist across the lifespan (Krammer, Kleim, Simmen-Janevska, & Maercker, 2016; Simmen-Janevska et al., 2015). Findings in young adults suggest ELA may be additively contributing to the

increased delay discounting associated with having family histories of alcohol and other drug use disorders (Acheson, Vincent, Cohoon, & Lovallo, 2019).

3.3 Association with problem substance use in adolescence

In addition to being found in at risk populations, increased delay discounting in adolescents can predict alcohol and other drug use outcomes (Audrain-McGovern et al., 2009; Fernie et al., 2013; Khurana, Romer, Betancourt, & Hurt, 2017; Kim-Spoon, McCullough, Bickel, Farley, & Longo, 2015; Martinez-Loredo, Fernandez-Hermida, De La Torre-Luque, & Fernandez-Artamendi, 2018). Increased delay discounting in mid adolescence predicted increased odds of tobacco smoking in late adolescence and early adulthood (Audrain-McGovern et al., 2009). Increased delay discounting across early to mid-adolescence predicted alcohol involvement at 6 month follow up (Fernie, Cole, Goudie, & Field, 2010). Decreased delay discounting in early adolescence mediated protective effects of religiousness against alcohol, tobacco, and cannabis use in mid-adolescence (Kim-Spoon et al., 2015). Young adolescents with increased delay discounting, poor working memory, and increased tendencies to act without thinking were more likely to engage in early substance use and develop alcohol, cannabis, or tobacco dependence by early adulthood (Khurana et al., 2017). Similarly, increased delay discounting and lack of premeditation in early to mid-adolescence also increased risk for tobacco, cannabis, and heavy alcohol use (Martinez-Loredo et al., 2018). Among adolescents undergoing different behavioral treatments for cannabis use disorder, increased delay discounting was a more robust predictor of poorer abstinence outcomes than type of treatment they received (Stanger et al., 2012). Similarly, increased delay discounting predicted poorer treatment outcomes in adolescents undergoing a contingency management smoking cessation program (Harvanko, Strickland, Slone, Shelton, & Reynolds, 2019).

4. Response Inhibition

4.1. Definition, assessment, and SUD association

Response inhibition refers to the ability to withhold a prepotent response (Logan, Van Zandt, Verbruggen, & Wagenmakers, 2014). Primary measures for assessing response inhibition include stop signal tasks, which require rapid responding to “go” signals that must be withheld when go signals are followed by a “stop” signal (Logan, 1994). Go/no go tasks where participants must rapidly respond to “go” stimuli but not “no go” stimuli are also commonly used (Newman, Widom, & Nathan, 1985). Additionally, Stroop tasks (Stroop, 1935) are also considered by some to be a measure of response inhibition (Moeller, Bederson, Alia-Klein, & Goldstein, 2016). Stroop tasks compare response latencies on trials with congruent stimuli (e.g., naming the font color of the word “red” written in red ink) versus incongruent stimuli (e.g., naming the font color of the word “blue” written in green ink), with longer response latencies to respond correctly to incongruent stimuli indicating poorer inhibition.

Poor response inhibition is linked to problem substance use (Kozak et al., 2019). For instance, a meta-analysis of stop signal and go-no go tasks performance and addictive behaviors found poor response inhibition was associated with heavy use/dependence of

MDMA, methamphetamine, tobacco, and alcohol but not opioids or cannabis (J. L. Smith, Mattick, Jamadar, & Iredale, 2014). In contrast, a mega-analysis of stop signal and go-no task performances in recreational (mostly non-abusing) substance users found lifetime cannabis use was associated with poorer response inhibition on stop signal tasks (Liu et al., 2019). It is not clear that poor performance on these tasks equates to inability to inhibit substance use or other problem behaviors rather than more basic cognitive dysfunction.

4.2. Alterations in SUD vulnerable populations

Performance on response inhibition improves across adolescence corresponding with maturation related changes in frontal striatal circuitry (Fosco, Hawk, Colder, Meisel, & Lengua, 2019; Liston et al., 2006; Madsen et al., 2010; Seghete, Herting, & Nagel, 2013), and there is evidence for persistent response inhibition deficits in SUDs vulnerable populations. Response inhibition has been reported to be impaired in children, adolescents, and adults with family histories of SUDs (Acheson, Richard, Mathias, & Dougherty, 2011; Dougherty et al., 2014; Dougherty et al., 2015; Lovallo et al., 2013; Lovallo, Yechiam, Sorocco, Vincent, & Collins, 2006). A systematic and quantitative review of meta-analyses on effects of ADHD on cognitive performance found ADHD was associated with medium effect size response inhibition deficits (Pievsky & McGrath, 2018). ELA has also been linked to poorer response inhibition on go/no-go and Stoop tasks in young adults (Kim, Kim, Jin, Im, & Lee, 2018; Lu et al., 2017; Marshall et al., 2016).

4.3 Association with problem substance use in adolescence

Poor performance on response inhibition tasks predicts development and progression of problem alcohol and other drug use (Moeller et al., 2016). For instance among a sample of high risk youths and community controls, poor response inhibition on a stop signal task in early and mid-adolescence predicted alcohol-related problems and illicit drug use independent of parental histories of alcohol use disorders (Nigg et al., 2006). A follow up analysis on high risk youths from the same cohort found that poor sleep in childhood predicted poor adolescent response inhibition, and poor adolescent response inhibition partially mediated links between poor childhood sleep and early adult illicit drug use (Wong, Brower, Nigg, & Zucker, 2010). Poorer response inhibition on stop signal tasks across early to mid-adolescence predicted alcohol involvement at 6 month follow up, similar to findings on delay discounting reported in the same cohort (Fernie et al., 2010). Among adolescents with externalizing problems, poorer inhibition on Stroop Task performance together with increased alcohol approach tendencies predicted alcohol use at 6 month follow up (Peeters et al., 2013). Among adolescent smokers in treatment, poor performance on response inhibition tasks has predicted relapse (Krishnan-Sarin et al., 2007).

5. Sensation-seeking

5.1. Definition, assessment, and SUD association

Sensation-seeking refers to the tendency to seek out novel, stimulating, and often risky experiences (Zuckerman & Link, 1968). It is typically assessed with self-report measures such as the Sensation Seeking Scale (Zuckerman, 2007b) or the UPPS Impulsive Behavior scale (Whiteside & Lynam, 2001). Increased sensation-seeking is found in substance

abusing populations (Zuckerman, 2007a). For instance, a recent meta-analysis found sensation-seeking was one of the most commonly studied and robustly altered personality traits in binge drinkers (Adan, Forero, & Navarro, 2017). Similarly, another meta-analysis found increased sensation-seeking was associated with smoking and nicotine dependence (Kale, Stautz, & Cooper, 2018). Finally, another recent meta-analysis found SUD psychotherapy treatment was associated with small but significant decreases in sensation-seeking (Hershberger, Um, & Cyders, 2017).

5.2. Alterations in SUD vulnerable populations

Sensation-seeking increases during mid-adolescence and plateaus or decreases by early adulthood (Harden & Tucker-Drob, 2011; Steinberg et al., 2008), and increased sensation-seeking may contribute to SUD risk in vulnerable populations. FH+ adolescents and young adults with high family densities of alcohol use disorders or early onset substance use have been reported to have elevated sensation-seeking (Acheson et al., 2016; Finn, Earleywine, & Pihl, 1992; Handley et al., 2011). Consistent with this, a combined impulsivity and sensation-seeking measure was reported to mediate links between parental alcoholism and the development of problem and alcohol and other drug use across adolescence and early adulthood in sample of FH+ and FH– youths (King & Chassin, 2004). Similarly, increased sensation-seeking was associated with ELA and partially mediated ELA effects on alcohol and cannabis use in a large sample of adults (Oshri, Kogan, et al., 2018).

5.3 Association with problem substance use in adolescence

Increased sensation-seeking is linked to substance use in adolescents and young adults (Quinn & Harden, 2013; Stautz & Cooper, 2013; VanderVeen, Hershberger, & Cyders, 2016). A meta-analysis of adolescent alcohol use and impulsivity-related personality traits found increased sensation-seeking had the largest associations with binge and heavy drinking, and increased sensation-seeking was also associated with alcohol use initiation in younger adolescents (Stautz & Cooper, 2013). Similarly, an analysis of a large sample of adolescents followed into early adulthood as part of the National Longitudinal Survey of Youth found slower decreases in sensation-seeking in late adolescence and early adulthood were associated with larger increases in alcohol use (Quinn & Harden, 2013). Additionally, a meta-analysis of impulsivity-related traits and adolescent cannabis use found that increased sensation-seeking was related to both use and negative consequences of use (VanderVeen et al., 2016).

6. Positive and Negative Urgency

6.1. Definition, assessment, and SUD association

Positive and negative urgency refer to behavioral propensities to act rashly when in negative or positive emotional states, respectively (Cyders & Smith, 2008). Positive and negative urgency are assessed with self-report measures such as the UPPS Impulsive Behavior scale (Berg, Latzman, Bliwise, & Lilienfeld, 2015; Whiteside & Lynam, 2001). Positive and negative urgency appear to be distinct from tending to act without thinking and tending to experience positive or negative moods (Peterson & Smith, 2019).

Urgency shows robust links to problem substance use (Cyders & Smith, 2008). A meta-analysis of impulsivity traits and alcohol use found that drinking problems were strongly related to both negative and positive urgency, and alcohol dependence was strongly related to negative urgency (Coskunpinar, Dir, & Cyders, 2013). The meta-analysis by Kale and colleagues linking sensation-seeking with smoking and nicotine dependence found stronger associations with both those variables and positive urgency (Kale et al., 2018). Finally, the meta-analysis by Hershberger and colleagues reporting small decreases in sensation-seeking with SUDs psychotherapy reported similar decreases in negative urgency (Hershberger et al., 2017).

6.2. Alterations in SUD vulnerable populations

Among normally developing adolescents, urgency appears to peak in mid adolescence and decline in early adulthood (Kasen, Cohen, & Chen, 2011), and increased urgency is found in SUDs vulnerable populations (Berg et al., 2015; Shin, McDonald, & Conley, 2018). A meta-analysis on impulsivity related traits and psychopathology found negative urgency was linked to depression, suicidality, aggression, anxiety, borderline personality disorder, and disordered eating (Berg et al., 2015). Increased urgency is also linked to ELA exposure (Shin et al., 2018), and increased negative urgency has been reported to mediate links between ELA severity and alcohol and cannabis problems in late adolescence (Wardell, Strang, & Hendershot, 2016). Similarly, a study in undergraduate students found positive urgency partially mediated links between ELA and problem drug use (Ramakrishnan, McPhee, Sosnowski, Rajasingam, & Erb, 2019).

6.3 Association with problem substance use in adolescence

Elevated urgency is linked to adolescent substance use (G. T. Smith & Cyders, 2016). The Stautz and Cooper (2013) meta-analysis linking sensation-seeking to adolescent drinking also found similar relationships for positive urgency and found problematic alcohol use in older adolescents was linked to both positive and negative urgency. Similarly the VanderVeen et al. (2016) meta-analysis linking sensation-seeking with adolescent cannabis use also reported both positive and negative urgency were associated with cannabis use and that positive urgency was associated with negative consequences of use. Overall urgency in pre-adolescence was found to predict drinking problems in mid-adolescence in a large prospectively monitored sample of adolescents (Cole, Peterson, & Smith, 2018). Negative urgency has also been reported to interact with alcohol and smoking reinforcement expectancies respectively to predict drinking and smoking by mid-adolescence (D'Agostino, Peterson, & Smith, 2019).

7. Overview of Findings

Delay discounting, response inhibition, sensation-seeking, and urgency are linked to SUDs and problem substance use in adults, are altered in at risk adolescent populations, and are linked to risk for adolescent problem substance use. One common theme that emerges is that ELA exposure is consistently linked to more risk prone alterations in these processes, and emerging evidence has suggested many of these processes may mediate links between ELA exposure and problem substance use. How these behavioral processes interact to influence

risk is not fully understood, but their developmental trajectories generally align with maturational imbalance hypotheses of adolescent risk. Specifically poor ability to value delayed rewards and inhibit responding coupled with increased tendencies to seek out novel and exciting stimuli and react impulsively when emotional could be placing adolescents at high risk for problem substance use. Ongoing and planned large scale studies of childhood development hold considerable promise for clarifying how these processes influence adolescent substance use, and these processes may be useful to examine in the context of SUD prevention strategies.

8. Contributions of Early Life Adversity

The associations of ELA and altered delay discounting, response inhibition, sensation-seeking, and urgency are consistent with associations between ELA exposure and alterations in neural circuits critical for cognition and emotion regulation (Andersen, 2019; Bick & Nelson, 2016). There is a growing consensus that ELA may be altering brain development in ways that promote risk for addictions and other psychopathology (Andersen & Teicher, 2009; Heim, Entinger, & Buss, 2019). Thus increased delay discounting, sensation-seeking, and urgency along with poorer response inhibition may be expressions or facets of an overarching phenotype resulting from ELA exposure. Further, given that ELA induced brain alteration patterns appear to vary based on the type and timing of ELA exposure (Andersen, 2019), it is plausible that these processes also be differentially impacted depending on what ELA exposure occurred. For example neglect in early childhood may affect these processes differently than trauma exposure in middle childhood. Further, it is also important to consider that factors such as environmental enrichment and quality of parental and other caregiver support may likely play a robust role in moderating the impact of ELA on these processes (Kentner, Cryan, & Brummelte, 2019).

It is important to consider ELA associated alterations in these processes may not necessarily reflect direct neurobehavioral effects but rather may reflect more indirect mechanisms. For example individuals with increased ELA may have increased experience with delayed rewards being unreliable and thus may learn to discount delayed rewards more to minimize risk (Acheson et al., 2019). Additionally, ELA exposure has widespread consequences such as lasting disruptions in sleep quality (Agorastos, Pervanidou, Chrousos, & Baker, 2019). Poor sleep quality could be impairing decision-making and inhibitory control (Diamond, 2013), increasing sensation-seeking (Hasler & Clark, 2013), and increasing urgency (Guerrero et al., 2019). More research is needed to determine specifically how ELA exposure impacts cognition and emotion regulation.

Further, it is also important to consider that it is difficult discern ELA effects from effects of family histories of SUDs or other psychopathology in much of the existing literature. Studies on ELA or family history often do not take the other into account despite the extensive overlap(i.e., a participant with a high number of ELA exposures is more likely to have a family history of SUD, and a participant with a family history of SUD is more likely to have experienced more ELA). Despite this, findings on ELA are often assumed to reflect environmental effects while finding on family history are often assumed to reflect hereditary

influences. It seems likely that a combination of environmental and hereditary influences is the actual driver of risk-related outcomes.

9. Interactions among processes

How these processes may interact to directly influence risky behavior in adolescents is a complex and challenging question to answer. The measures used to assess these processes had been carefully designed to isolate and quantify these processes under controlled settings where outside influences and distractions are minimized. Real world behavior involves fluid interactions among multiple processes in the context of complex environments. Notably, risk related-conditions such as having a family history of SUD may be linked to poorer ability to transition between cognitive states (Amico et al., 2020). While it is difficult to evaluate how these processes influence real time behavior, how these processes interact as trait level characteristics in relation to adolescent substance use outcomes can be more easily studied.

Developmental trajectories of delay discounting, response inhibition, sensation-seeking, and urgency generally align with maturational imbalance hypotheses of adolescent risk (Casey, Jones, & Somerville, 2011; Ernst, Pine, & Hardin, 2006; Shulman, Harden, Chein, & Steinberg, 2014; Steinberg et al., 2008). These hypotheses postulate adolescents are at heightened risk for substance misuse and other problem behaviors because motivational and emotional drives peak in early adolescence, while impulse control develops more slowly. Essentially, the “gas pedal” in the adolescent brain matures early while the “brake pedal” is still developing. This resultant “gap” or lack of effective behavioral regulation is thought to not be resolved until early adulthood when mature levels of impulse control are achieved and motivational and emotional drive trajectories have either leveled off or declined. Consistent with this dual system developmental pattern, delay discounting and response inhibition steadily change across adolescence and into young adulthood, with older individuals valuing delayed rewards more and having improved capability to inhibit pre-potent responses (Achterberg et al., 2016; Fosco et al., 2019; Liston et al., 2006; Madsen et al., 2010; Olson et al., 2009; Seghete et al., 2013; Steinberg et al., 2009). In contrast, sensation-seeking, and urgency appear to peak by mid to late adolescence and then level off or subside as individuals mature into adulthood (Harden & Tucker-Drob, 2011; Kasen et al., 2011; Steinberg et al., 2008).

Concerns have been noted about the need for more rigorous and in-depth evaluations of maturational imbalance hypotheses (Meisel, Fosco, Hawk, & Colder, 2019). Notably, many studies investigating these hypotheses have used relatively brief or limited assessments of inhibitory control and motivation related processes. More complex models addressing interactions among more specific behavioral processes like the ones reviewed here may enhance understandings of how maturational imbalances in neurodevelopment contribute to risk for problem substance use.

10. Future Directions

A better understanding of how behavioral processes contribute to risk for problem substance use in adolescents may be essential for improved prevention and early intervention

strategies. Much of the existing findings were generated from studies using relatively small to modest sample sizes, and many of these studies were cross-sectional or had limited longitudinal follow-up. Large-scale prospective studies assessing multiple processes and other relevant variables in the same individuals, such as the ongoing Adolescent Brain Cognition and Development Study (Volkow et al., 2018), hold considerable promise for better understanding how these behavioral processes influence addiction risk. Further, increasing evidence suggest that ELA may be altering neurodevelopment to increase risk for SUDs and other psychopathology. However most studies on ELA in humans have to rely on retrospective accounts of events that occurred years prior, and large sample sizes are needed to feasibly attempt to differentiate effects of different patterns of ELA exposure as well as disentangle ELA influences from frequently co-occurring factors such as family histories of SUDs and other psychopathologies. Studies such as the pending HEALthy Brain Childhood Development Study, which intends to enroll a large cohort of pregnant women and monitor impacts of family and environmental influences on their children's development for at least 10 years (NIH, 2020), may be essential for better understanding how ELA impacts the early development of behavioral processes associated with risk for problem substance use in adolescence.

Given links between problem substance use and delay discounting, response inhibition, sensation-seeking, and urgency, these processes may be useful to examine in the context of SUD prevention strategies. Children who appear more risk prone on these processes may benefit from targeted intervention strategies to protect against problem substance use. Further, these processes could potentially be used to help measure the effectiveness of prevention strategies. For instance there is evidence that early interventions for childhood maltreatment may reduce response inhibition deficits and decision-making impairments (Jankowski et al., 2017; Weller, Leve, Kim, Bhimji, & Fisher, 2015). Potentially, risk-related behavioral processes could be used as metrics to provide relatively rapid feedback about the effectiveness of different prevention strategies aimed at addressing underlying dysfunctions that promote risk for problem substance use.

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Highlights

- Delay discounting, response inhibition, sensation-seeking, and urgency are all linked to risk for problem substance use in adolescence.
- Early life adversity may be playing a significant role in these processes.
- Differences in developmental trajectories of these processes may contribute to risk.