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Understanding the Etiology of Adolescent Substance Use through Developmental Perspectives

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Abstract

There are notable risks associated with alcohol, cigarette, and marijuana use during adolescence. Yet, there is no single cause for adolescent substance use. The etiology of substance use is known to develop over time, across multiple levels of influence. Informed by developmental perspectives, this review aims to provide an overview of biological (e.g., genetic, neuroimaging), individual (e.g., temperament, behavior problems), and social (e.g., parents, peers) factors that increase risk for and protection against this multifaceted phenomenon. Additionally, emerging areas of research are highlighted, as well as preliminary work examining the etiology of adolescent substance use across multiple levels of influence. Understanding early factors associated with the emergence of adolescent substance use can help inform prevention programming to reduce subsequent cognitive impairments and psychopathology. Adaptive individualized interventions addressing various domains linked to adolescent substance use in real-time are likely to have significant utility given the numerous pathways to adolescent substance use.

Keywords

adolescents; etiology; alcohol; cigarettes; marijuana; temperament; socialization

Adolescent substance use remains a significant public health concern. The 30-day prevalence rates of alcohol (18.2%), marijuana (15.6%), and cigarette (3.7%) use among youth are notable despite declines from historical highs (Johnston, 2020). Youth who use alcohol by age 14 have a five-fold increased risk of alcohol use disorder compared to those initiating at age 21 (SAMHSA, 2009). Adolescent substance use can negatively impact brain development, which may contribute to risk for cognitive impairments (e.g., memory) and psychopathology (Hummel et al., 2013). Accordingly, understanding factors contributing to adolescent substance use is critical.

Adolescent substance use is a complex phenomenon best understood from a developmental viewpoint. Two perspectives have utility for understanding multiple levels of influence contributing to adolescent substance use. Cascade models posit dynamic, multilevel

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transactions between the child and the environment in emergent behaviors characterized by a sequential progression from temperamental differences in childhood (e.g., impulsivity, negative affectivity), to problem behaviors (e.g., externalizing, internalizing) in early adolescence, to riskier behaviors, such as alcohol and drug use during mid-adolescence (Dodge et al., 2009). Moreover, biologically based differences (e.g., genes, neural deficits) are believed to initiate this cascading sequence. Bronfenbrenner's Developmental Ecological Systems Theory (1979) organizes various "systems" that interact with individual differences to impact development, including engagement in substance use. Immediate socialization contexts (e.g., peers) are reflected in the *microsystem* and posited to have a proximal impact on the adolescent. Whereas other socialization contexts (e.g., neighborhoods) and influences (e.g., politics) are reflected in the *exosystem* and *macrosystem*, respectively, to indicate their distal influence (Bronfenbrenner, 1979).

Grounded in these perspectives, this review characterizes processes that contribute to adolescent substance use across multiple levels of influence (see Figure 1). A broad overview of key risk and protective factors within each level of influence is provided, as well as emerging topics. Readers are referred to Chassin and colleagues (2016) for an exhaustive review. Concluding remarks on how to effectively integrate examinations across levels of analyses with adaptive individualized interventions are offered.

Individual Factors

Prior work indicates that adolescent substance use etiology is a result of a sequential cascade of biological predispositions and symptomatology that emerge during early childhood (Dodge et al., 2009). Childhood temperamental traits have been shown to impact the emergence of behavior problems, most commonly conceptualized as externalizing (e.g., delinquency) and internalizing (e.g., anxiety, depression) symptoms. These pathways are not suggested to be deterministic. Rather, they are trajectories, along which moderating factors influence youth toward either adaptive or maladaptive outcomes (Hussong et al., 2017). Moreover, genetic and neural underpinnings are believed to act as catalysts setting these various pathways into motion.

Biological Factors

A key vulnerability factor for adolescent substance use is family history of substance use disorder (FH+), with a heritability of approximately 50% (Trucco et al., 2019). FH+ increases the likelihood of developing problematic substance use three-to-five-fold (Cservenka et al., 2014). Accordingly, understanding biological underpinnings reflecting this heritability is critical. Yet, the identification of specific genes has been challenging. Perhaps the most robust associations involve genes encoding metabolizing enzymes (see Trucco et al., 2019). Adolescents with *ALDH2* and *ADH1B* variants experience nausea, flushing, and headaches when consuming alcohol, thus limiting alcohol use. Although other candidate genes, such as those involved in stress response (e.g., *CRHR1*) and neurotransmitter functioning (dopaminergic [e.g., *COMT*], serotonergic [e.g., *5-HTTLPR*], GABAergic [e.g., *GABRA2*]), have been associated with substance use disorder, findings have generally not replicated in genome-wide association studies. These genes may have

a more indirect association on adolescent substance use through cascading effects via intermediate phenotypes (e.g., externalizing problems) or by increasing sensitivity to socialization contexts that either promote or deter use (i.e., gene × environment interactions; see Trucco et al., 2019).

FH+ adolescents also exhibit alterations in neurobiological functioning. One study demonstrated reduced brain activity in fronto-parietal regions in alcohol-naïve FH+ adolescents during response inhibition on the Go/No-Go task compared to controls, despite similar behavioral performance (Schweinsburg et al., 2004). Furthermore, FH+ adolescents engaging in heavy drinking (i.e., early onset drinking, drunkenness, drinking-related problems) demonstrated abnormal brain activity in limbic and frontal areas in response to emotional stimuli (Heitzeg et al., 2008). Work involving emotion-cognition interactions demonstrated that deficits in emotional processing among FH+ adolescents may interfere with executive functioning brain response through altered amygdalar functional connectivity (Cservenka et al., 2014), which can exacerbate risk for problematic alcohol use. Yet, greater neural activation in response to emotional stimuli (e.g., orbital frontal gyrus; Heitzeg et al., 2008) and successful inhibition (e.g., dorsolateral prefrontal cortex; Martz et al., 2018) are indicative of protective neural mechanisms against substance use disorder, even among FH+ adolescents.

An emerging area reflects neurogenetics, which combines genetic and neurobiological processes. A notable study examined the role of brain responses to negative emotional words as a potential mediator in the association between *CRHR1* and alcohol use (i.e., drinking volume, binge-drinking days, and alcohol-related problems) among youth (Glaser et al., 2014). Findings indicate that *CRHR1* gene variation impacts risk of problematic alcohol use via negative emotionality through activation in the right ventrolateral prefrontal cortex to negative emotional words (Glaser et al., 2014). Similar examinations can help identify neurobiological underpinnings associated with adolescent substance use that can ultimately inform targeted interventions.

Cascading Effects Via Behavior Problems

Prior work supports the role of behavior problems as early risk factors in adolescent substance use etiology. The externalizing pathway, characterized by marked deficits in behavioral inhibition, has been shown to promote adolescent substance use (Hussong et al., 2017). Externalizing symptomatology also promotes deleterious socialization processes (e.g., affiliation with substance using peers) that set the stage for adolescent substance use (Colder et al., 2018a). This pathway is predicated by difficult temperament in infancy that may be due in part to biological underpinnings, which often results in behavioral problems in childhood, leading to a sequential progression to adolescent substance use (Hussong et al., 2017). In addition, the dual systems model of risk provides a more developmentally normative explanation for increased risk-taking that is often observed during adolescence. The model posits that such behavior results from the temporally dissimilar development of two neurobiological systems: the socio-emotional system, which develops rapidly during early adolescence, and the cognitive-control system which develops through early adulthood (Castellanos-Ryan et al., 2013). This imbalance increases vulnerability to substance use,

especially among FH+ youth, as the cognitive-control system inhibits the impulsive behavior attributed to the rapidly developing socio-emotional system. Further, prior work indicates that deficits in working memory combined with heightened reward seeking is predictive of early progression into adolescent drug use (Khurana et al., 2015). Yet, some argue that the dual systems model only applies to adolescents with disruptive behavior disorder, simply exacerbating an existing externalizing trajectory (Bjork & Pardini, 2015).

There is also support for the role of internalizing symptoms on adolescent substance use, but this process is nuanced. Some researchers posit that temperamental characteristics reflecting internalizing symptoms (e.g., behavioral inhibition, fearfulness) may initially be protective of substance use during early adolescence, though these youth are likely to follow a more rapid escalation in use during mid- to late adolescence when substance use is more normative (Hussong et al., 2017). For example, social anxiety was protective against substance use given fewer opportunities to associate with substance using peers (Khoddam et al., 2016). Yet, prior work largely does not support the role of internalizing symptoms alone in the etiology of adolescent substance use (Colder et al., 2018b; Scalco et al., 2020).

There is stronger support in the role of internalizing symptomatology on adolescent substance use in the context of externalizing symptomatology. Consistent with the unfolding of substance use via cascading effects, the dual failure hypothesis (Capaldi, 1992) posits that externalizing symptoms in early adolescence may lead to later co-occurring symptomatology resulting primarily from peer rejection and alienation from parents, which together increase risk in deviant peer group affiliation that promotes substance use. The stable co-occurring hypothesis (Craig et al., 2018b) proposes that adolescents who are characterized by co-occurring symptomatology across development are at increased risk for substance use given disturbances in executive function and impairments in social adaptation (e.g., peer rejection). One study modeling pure externalizing symptomology, pure internalizing symptomatology, and co-occurring symptomatology from late childhood to late adolescence demonstrated that even though externalizing symptoms in the absence of internalizing symptoms represented the most salient etiological pathway to alcohol use consumption (i.e., quantity × frequency), co-occurring internalizing and externalizing symptomatology also conferred risk consistent with the dual failure and stable co-occurring hypotheses (Scalco et al., 2020). Accordingly, externalizing and internalizing pathways to substance use are likely not distinct. Rather, cascading transactions between adolescent externalizing and internalizing symptomatology that arise from biological predispositions and early temperamental factors likely characterize etiological pathways to adolescent substance use.

An emerging area of research supporting another relevant factor in cascading effects to adolescent substance use via problem behaviors are sleep disturbances. Sleep problems reflect deficits in sleep duration, sleep continuity, and variable weekday and weekend sleep-wake timing (i.e., circadian misalignment). Sleep problems specific to wakefulness in bed was found to precede adolescent anxiety and depression (Blake & Allen, 2020). In contrast, circadian misalignment has been linked to disruption in reward processes and externalizing pathways to substance use. Namely, misalignment impairs executive functioning and inhibitory control, which may influence reward-related decision-making and the decision to use substances (Hasler & Clark, 2013). Future work examining longitudinal

bidirectional associations and neurobiological underpinnings linking specific sleep problems to adolescent substance use could identify intervention targets.

Social Factors

Another important domain in adolescent substance use is the social context. Consistent with Bronfenbrenner's theory, parents and peers are two of the most salient social contexts that impact adolescent substance use. Similarly, cascade models propose that children with challenging temperaments make it difficult for their caretakers to parent effectively. Problematic parenting, in turn, may lead children to behave disruptively upon school entry leading to increased peer rejection. Stress caused by these dynamics may paradoxically lead parents to withdraw from adaptive parenting practices. This withdrawal may increase opportunities for the adolescent to associate with deviant peers, which likely potentiates substance use onset. Accordingly, a focus is placed on risk and protective processes within these domains.

Parent Socialization Context

Although various aspects of the parent context contribute to the etiology of substance use, parenting practices and styles have received significant attention. Parental warmth represents behavior directed toward the adolescent reflecting a message that they are loved, such as praise and involvement, while parental control represents actions intended to shape adolescent behavior, such as supervision and discipline (Calafat et al., 2014; Luk et al., 2017). One study found that adolescents perceiving lower parental involvement were at greater risk for using alcohol than those perceiving highly involved parents (Gottfredson & Hussong, 2011). Parent-adolescent relationships characterized by low warmth may contribute to an adolescent's inability to effectively regulate behavior, increasing risk for substance use initiation (Hummel et al., 2013).

Within the parental control domain, monitoring/knowledge is perhaps the most widely supported protective parental practice against adolescent substance use. Monitoring reflects a parent's knowledge of their adolescent's whereabouts, activities, and relationships. One study demonstrated that lower parental monitoring predicted the initiation of alcohol use, binge drinking, and marijuana use (Rusby et al., 2018). Parental monitoring/knowledge may be protective against adolescent substance use by minimizing exposure to substance-using peers.

Other work has derived typologies of parenting styles based on combinations of warmth and control: authoritarian (low in warmth and high in control), permissive (high in warmth and low in control), authoritative (high in both), and neglectful (low in both). Prior research suggests that authoritative parents represent the most optimal style given the strong association with resilience, self-esteem, and reduced involvement in behavior problems (Calafat et al., 2014). Yet, cross-cultural studies question this assertion. For example, authoritarian parenting was associated with independence, assertiveness, and engagement across Black and Hispanic/Latinx adolescents (see Calafat et al., 2014) and social-emotional adjustment and low problem behaviors in collectivist Asian cultures (see Luk et al., 2017). All parenting styles were protective against adolescent substance use except for

the neglectful style in a South American sample (Valente et al., 2017). In sum, prior work indicates that aspects of parental warmth and control may minimize adolescent substance use, but the optimal combination of these factors likely differs across race, ethnicity, and country of origin.

Parents also have a more direct effect on adolescent substance use via their own use of and attitudes towards substances. Parents communicate a message of acceptance to adolescents when they engage in substance use behavior (Rusby et al., 2018). Similarly, parents convey a message to children that drinking is unlikely to lead to negative consequences when permitting their children to consume alcohol. One study found that parental permission to taste alcohol prior to adolescence predicted increased alcohol consumption and alcohol-related problems in late adolescence via alcohol-specific cognitive appraisals (Colder et al., 2018a). Similarly, other work found that parental provision of alcohol sips was able to distinguish between levels of early adolescent alcohol consumption (i.e., sippers vs. drinkers, sippers vs. abstainers, and drinkers vs. abstainers; Wadolowski et al., 2015). Taken together, this work indicates that sipping alcohol with parental permission is not benign and may not necessarily promote responsible drinking.

A more nascent area of research supports the role of childhood maltreatment on adolescent substance use. Although perpetrators may consist of non-relatives, biological parents are the most common offenders (Benedini & Fagan, 2018). Prior work indicates important nuances is this domain. One study found that even though youth reporting neglect, physical, and sexual abuse had greater odds of adolescent alcohol, cigarette, and marijuana use compared to youth without a history of maltreatment, the association between physical abuse and alcohol use was weakest, and the association between sexual abuse and marijuana use was strongest (Hussey et al., 2006). One study found that the association between physical abuse on substance use was mediated by internalizing problems for girls, while the association between sexual abuse and substance use was mediated by both internalizing problems and anger for girls, but only anger among boys (Benedini & Fagan, 2018).

Peer Socialization Context

Peers also have a direct and indirect effect on shaping adolescent substance use. Similarities between peers and adolescents result from two processes. *Selection* refers to an adolescent's decision to join certain peer groups based on similarity in substance use attitudes and/or behaviors, while *socialization* reflects an adolescent's change in substance use attitudes and/or behaviors to assimilate to peers. Prior work indicates that both processes bring about high similarity among adolescents and peers regarding substance use. One study found that adolescent alcohol use predicted peer drinking, supporting selection, and that peer alcohol use predicted adolescent drinking, supporting socialization (Patrick et al., 2016).

Importantly, the relative influence of selection vs. socialization has been demonstrated to vary across substances and development. Modeling and socialization influences had a strong effect size among "soft drug" (e.g., alcohol, marijuana, tobacco) use but only a moderate to average effect size among "hard drug" use (e.g., cocaine, heroin; Kruis et al., 2020). Other work supported both peer socialization and selection for alcohol use, but only peer selection for marijuana (Becker & Curry, 2014) and cigarette (Kiuru et al., 2010) use.

One possible explanation involves the more social nature of drinking among peers (e.g., drinking games) compared to the more solitary act of smoking cigarettes (Kiuru et al., 2010). Moreover, although both processes are believed to operate simultaneously to impact adolescent substance use, prior research indicates that peer socialization is the primary mechanism of influence during early adolescence (Patrick et al., 2016). This is due in part to the increased salience of the peer context during early adolescence and the increase in resistance to peer influence in mid- to late adolescence (Patrick et al., 2016).

Social norms theory (Azjen & Fishbein, 1980) posits two processes through which peers impact substance use: *descriptive norms* (i.e., an adolescent's belief regarding substance use prevalence) and *injunctive norms* (i.e., an adolescent's belief regarding substance use approval). One study found that when asking popular adolescents directly about their cigarette and marijuana use, they reported almost no use (Helms et al., 2014). However, classmates perceived that these same popular teens were smoking up to three cigarettes per day and using marijuana up to nine times per month. Moreover, higher perceptions of popular peers' substance use in Grade 9 were associated with a steep growth in substance use (Helms et al., 2014).

A burgeoning field with respect to socialization contexts is the impact of media exposure and communication on adolescent substance use. Although research finds associations between more traditional substance use-related media exposure (e.g., television, radio), technological advances providing 24-hour access to videos, text messaging, and social networks have created new opportunities for peers, marketing companies, and celebrities to influence an adolescent's attitudes towards substance use (Boyle et al., 2016). Nearly ubiquitous smartphone usage among teens has fueled increased online activities whereby approximately 45% of teens report being online on a near-constant basis (Pew Research Center, 2018). With increased time online comes greater exposure to images depicting substance use on websites and social networking platforms. Greater exposure to alcohol-related advertising and social media content predicted alcohol use onset (Boyle et al., 2016). Moreover, exposure to substance-related content via social media likely impacts use via perceived norms and the formation of favorable attitudes towards substance use (Davis et al., 2019). This is partly due to the glamorized portrayal of substance use on social media, offering an overabundance of misleading media content. Yet, the content and context of media exposure is critical with respect to adolescent substance use. That is, exposure to educational programs and screen-to-screen social interactions via apps (e.g., FaceTime) and active parental mediation of media influence (i.e., discussions exploring and clarifying media content; Collier et al., 2016) offer health benefits to adolescents, including lower levels of substance use.

Conclusions

Adolescent substance use is a notable public health concern. The etiology of adolescent substance use is complex and multifaceted. To gain an accurate understanding of how risk for substance use unfolds, a firm grounding in developmental perspectives is critical. Namely, adolescent substance use often reflects a sequential progression of risk that emerges in childhood characterized by deficient or elevated behavioral inhibition which, in turn,

contributes to the development of externalizing, internalizing, or co-occurring behaviors, leading to progressively more problematic behaviors, including substance use (Figure 1). These developmental processes all occur within the context of various social ecologies that impact adolescent decision-making and behaviors. Accordingly, future work should continue to incorporate multiple levels of influence to mirror the inherent complexity in the etiology of adolescent substance use. A notable example includes the work of Buil and colleagues (2017) that examined individual temperament, peer difficulties, and overt antisocial and substance use outcomes in adolescence. This work highlights the synergistic effects between social contexts and individual development. In addition, Marceau and colleauges (2020) examined developmental pathways comprised of polygenic risk, prenatal stress, warm parenting, and internalizing and externalizing problems in the prediction of adolescent substance use. Similar work examining multiple levels of influence is critical to informing multidimensional intervention programming.

Adolescent substance use behavior is nested within multiple spheres of socialization contexts, as well as biologically based individual risk factors. A particularly promising area of scientific work leverages technology that is ingrained in adolescents' lifestyles by focusing on digital interventions. These include web- and computer-based (eHealth) strategies, as well as mobile health (mHealth) options (Nahum-Shani et al., 2018). For example, Climate Schools-Combined is a digital online intervention that integrates modules focused on the prevention of substance use, depression, and anxiety (Teeson et al., 2020), which is consistent with cascade models supporting the role of internalizing problems to adolescent substance use. The Climate Schools-Combined intervention produced increased knowledge of marijuana use and less growth in odds of drinking compared to those receiving a standalone preventive intervention for substance use (Teeson et al., 2020). Yet, given empirical support for the prominent role of externalizing and co-occurring symptomatology as pathways to adolescent substance use, youth demonstrating a biological predisposition to risk taking behavior could benefit from additional modules that incorporate brain training to improve emotion regulation and enhance effortful control (Sher, 2016). Similarly, future work incorporating real-time normative feedback interventions to modify perceptions regarding the frequency and approval of peer substance use to mobile applications holds great promise (Davis et al., 2019).

Additionally, just-in-time-adaptive interventions (JITAIs) that are often administered through wearable technology and mobile phones through user-specific input, geolocation, and passive sensing allow for the detection and real-time deployment of interventions (Nahum-Shani et al., 2018). Wearable sensors and mobile phones can inform the unique social ecologies (e.g., peer group), contexts (e.g., sporting events), and individual factors (e.g., mood, physiological changes) that contribute to increased risk for substance use that align with developmental cascade and ecological models. Together this information can be used to trigger timely interventions that are ecologically valid to create personalized strategies to manage an individual's risk contexts and stressors to help prevent the use of substances. Accordingly, continued efforts in developing technology-delivered personalized treatment packages that target multiple domains of influence impacting adolescent substance use in real-time will be invaluable.

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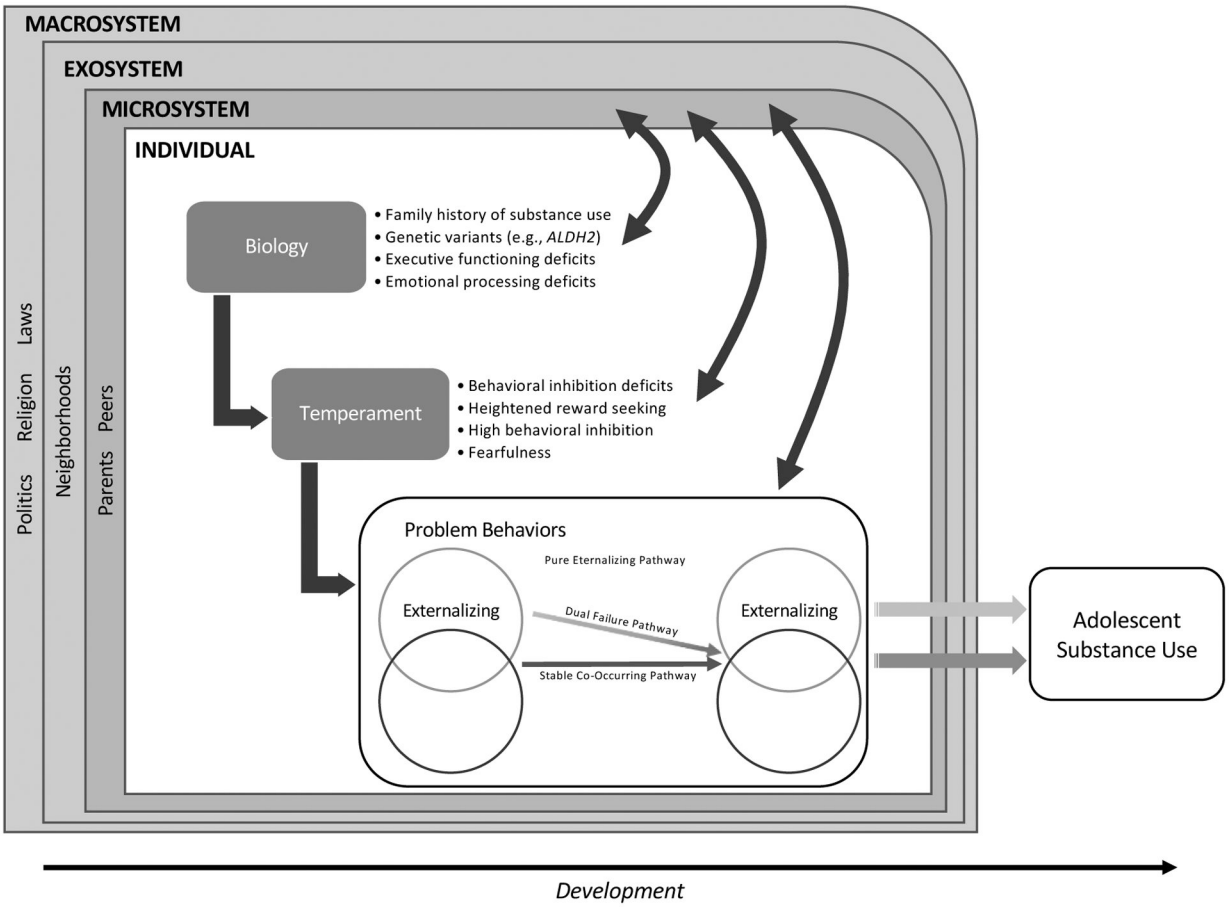

Figure 1. Adolescent Substance Use Etiology via Cascade and Ecological Models Across Development

Note: At the individual level, cascade models highlight the sequential processes that characterize the etiology of adolescent substance use, which originate from biological factors, leading to the emergence of temperamental risk factors, that in turn impact the onset of problem behaviors. Processes via problem behaviors can be conceptualized by transactional associations between externalizing and internalizing symptomatology: a stable or pure externalizing pathway, a pathway from externalizing symptomatology to co-occurring (externalizing and internalizing) symptomatology (i.e., dual failure pathway), and a stable co-occurring pathway. These individual processes are best understood as transactions between the adolescent and their social ecology, comprised of both proximal (e.g., parents, peers) and distal (e.g., laws) socialization contexts.