Adolescent substance use is 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 et al., 2020). Youth who use alcohol by age 14 have a fivefold increased risk of alcohol use disorder compared to youth who begin using alcohol at age 21 (Substance Abuse and Mental Health Services Administration, 2009). Adolescent substance use can harm 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 help us understand the multiple levels of influence that contribute to substance use by adolescents. Cascade models posit dynamic, multilevel transactions between the youth 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 midadolescence (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 affect development, including...
using substances. Immediate socialization contexts (e.g., peers) are reflected in the microsystem and thought to affect adolescents proximally. Other socialization contexts (e.g., neighborhoods) and ideologies (e.g., politics) are reflected in the exosystem and macrosystem, respectively, to indicate their distal influence (Bronfenbrenner, 1979).

Grounded in these perspectives, in this review, we characterize processes that contribute to adolescent substance use across multiple levels of influence (see Figure 1). We provide a broad overview of key risk and protective factors within each level of influence, and touch on emerging topics in the field (see Chassin et al., 2016, for a review). We conclude with a discussion on how to integrate examinations across levels of analyses with adaptive individualized interventions.

**INDIVIDUAL FACTORS**

Adolescent substance use is a result of a sequential cascade of biological predispositions and symptomatology that emerge during early childhood (Dodge et al., 2009). Childhood temperamental traits affect the emergence of behavior problems, most commonly conceptualized as externalizing (e.g., delinquency) and internalizing (e.g., anxiety, depression) symptoms. These pathways are not deterministic, but are trajectories along which moderating factors influence youth toward adaptive or maladaptive outcomes (Hussong et al., 2017). Moreover, genetic and neural underpinnings are thought to act as catalysts setting these various pathways in motion.

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**FIGURE 1** Etiology of adolescent substance use 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 affect 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 adolescents and their social ecology, including both proximal (e.g., parents, peers) and distal (e.g., laws) socialization contexts.
Biological factors

A key vulnerability factor for adolescent substance use is a 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 fivefold (Cservenka et al., 2014). Accordingly, understanding the biological underpinnings that reflect this heritability is critical, but identifying specific genes has been challenging. Perhaps the most robust associations involve genes encoding metabolizing enzymes (see Trucco et al., 2019). Adolescents with aldehyde dehydrogenase genes (e.g., ALDH2 and ADH1B variants) experience nausea, flushing, and headaches when consuming alcohol, thus limiting their alcohol use. Although other candidate genes, such as those involved in stress response (e.g., corticotropin-releasing hormone receptor 1 [CRHRI]) and neurotransmitter functioning (dopaminergic [e.g., catechol O-methyltransferase, COMT], serotonergic [e.g., the serotonin transporter gene, 5-HTTLPR], GABAergic [e.g., γ-aminobutyric receptor α-2 subunit, GABRA2]), have been associated with substance use disorder, genome-wide association studies have generally not replicated these findings. These genes may have a more indirect association with adolescent substance use through cascading effects via intermediate phenotypes (e.g., externalizing problems) or by increasing sensitivity to socialization contexts that promote or deter use (i.e., gene × environment interactions; see Trucco et al., 2019).

FH+ adolescents also exhibit alterations in neurobiological functioning. In one study, alcohol-naive FH+ adolescents had reduced brain activity in frontoparietal regions during response inhibition on the go/no-go task compared to adolescents in a control group (i.e., youth with no family history of alcohol use disorder), despite similar behavioral performance (Schweinsburg et al., 2004). Furthermore, FH+ adolescents who drank heavily (i.e., those with 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). In research on interactions between emotion and cognition, deficits in emotional processing among FH+ adolescents may have interfered 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.

Neurogenetics, which combines research on genetic and neurobiological processes, is an emerging area of research. In one study, researchers examined the role of brain responses to negative emotional words as a potential mediator in the association between CRHRI and alcohol use (i.e., drinking volume, binge-drinking days, and alcohol-related problems) among youth (Glaser et al., 2014). CRHRI gene variation affected youth’s 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 studies can help identify the neurobiological underpinnings associated with adolescent substance use and inform targeted interventions.

Cascading effects via behavior problems

Research supports the role of behavior problems as early risk factors in the etiology of adolescent substance use. The externalizing pathway, characterized by marked deficits in behavioral inhibition, promotes 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, Shyhalla, et al., 2018). 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 socioemotional 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 socioemotional system. Furthermore, deficits in working memory combined with heightened reward seeking predict early progression into adolescent drug use (Khurana et al., 2015). Yet, some argue that the dual systems model applies only to adolescents with a disruptive behavior disorder, which exacerbates an existing externalizing trajectory (Bjork & Pardini, 2015).

Research also supports the role of internalizing symptoms in adolescent substance use, but the process is nuanced. Some researchers argue that temperament characteristics reflecting internalizing symptoms (e.g., behavioral inhibition, fearfulness) may initially protect against substance use during early adolescence, though for these youth, use is likely to escalate more rapidly during mid- to late adolescence when
substance use is more normative (Hussong et al., 2017). For example, in one study, social anxiety protected against substance use given fewer opportunities to associate with substance-using peers (Khoddam et al., 2016). Yet, research largely has not supported the role of internalizing symptoms alone in the etiology of adolescent substance use (Colder, Frndak, et al., 2018; Scalco et al., 2020).

Research supports more strongly the role of internalizing symptomatology in 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; together, these increase the risk of affiliation with a deviant peer group that promotes substance use. The stable co-occurring hypothesis (Colder, Shyhalla, et al., 2018) proposes that adolescents 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). In one study, researchers modeled pure externalizing symptomatology, pure internalizing symptomatology, and co-occurring symptomatology from late childhood to late adolescence; even though externalizing symptoms in the absence of internalizing symptoms represented the most salient etiological pathway to alcohol use (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 probably characterize etiological pathways to adolescent substance use.

Sleep disturbances are another emerging area of research that supports a relevant factor in cascading effects to adolescent substance use via problem behaviors. Sleep problems reflect deficits in sleep duration, sleep continuity, and variable weekday and weekend sleep–wake timing (i.e., circadian misalignment). In one study, sleep problems specific to wakefulness in bed preceded 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. Misalignment impairs executive functioning and inhibitory control, which may influence reward-related decision making and the decision to use substances (Hasler & Clark, 2013). Research that examines the longitudinal bidirectional associations and neurobiological underpinnings linking specific sleep problems to adolescent substance use could identify targets for intervention.

**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 affect 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. Paradoxically, stress caused by these dynamics may lead parents to withdraw from adaptive parenting practices. This withdrawal may increase opportunities for adolescents to associate with deviant peers, which probably increases the likelihood of substance use. Accordingly, in this review, we focus 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 in research. Parental warmth represents behavior directed toward adolescents, reflecting a message that they are loved, such as praise and involvement, whereas parental control represents actions intended to shape adolescent behavior, such as supervision and discipline (Calafat et al., 2014; Luk et al., 2017). In one study, adolescents who perceived lower parental involvement were at greater risk for using alcohol than those who perceived that their parents were very involved (Gottfredson & Hussong, 2011). Parent–adolescent relationships characterized by low warmth may contribute to adolescents’ inability to regulate behavior effectively, increasing youth’s risk of initiating substance use (Hummel et al., 2013).

Within the domain of parental control, monitoring and knowledge may be the most widely supported protective practices against adolescent substance use. Monitoring means parents know about their adolescents’ whereabouts, activities, and relationships. In one study, low parental monitoring predicted the initiation of alcohol use, binge drinking, and marijuana use in adolescent offspring (Rusby et al., 2018). Parental monitoring and knowledge may protect against adolescent substance use by minimizing exposure to substance-using peers.

Other work has identified 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). In general, authoritative parents represent the most optimal style for overall adolescent adaptive development given their 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 in the United States (see Calafat et al., 2014), and with social-emotional adjustment and low problem behaviors in collectivist Asian cultures (see Luk et al., 2017). In a study of South American families, all parenting styles protected against adolescent substance use except for the neglectful style of parenting (Valente et al., 2017). In summary, research suggests 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 affect adolescent substance use more directly via their own use of and attitudes toward substances. Parents communicate a message of acceptance to adolescents when they use substances (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. In one study, parental permission for children to taste alcohol prior to adolescence predicted increased alcohol consumption and alcohol-related problems in late adolescence via alcohol-specific cognitive appraisals (Colder, Shyhalla, et al., 2018). Similarly, in another study, parental provision of sips of alcohol was associated with levels of early adolescent alcohol consumption (i.e., sippers vs. drinkers, sippers vs. abstainers, and drinkers vs. abstainers; Wadolowski et al., 2015); for example, drinkers were more likely than abstainers to report receiving sips of alcohol from their parents. Taken together, this work indicates that sipping alcohol with parents’ permission is not benign and may not necessarily promote responsible drinking.

A more nascent area of research supports the role of maltreatment during childhood in adolescent substance use. Although perpetrators of maltreatment may include nonrelatives, biological parents are the most common offenders (Benedini & Fagan, 2018), and the maltreatment domain is very nuanced. In one study, even though youth who reported neglect, physical abuse, and sexual abuse were more likely to use alcohol, cigarettes, and marijuana in adolescence than youth without a history of maltreatment, the association between physical abuse and alcohol use was weakest while the association between sexual abuse and marijuana use was strongest (Hussey et al., 2006). In another study, the association between physical abuse and substance use was mediated by internalizing problems for girls, whereas the association between sexual abuse and substance use was mediated by both internalizing problems and anger for girls, but only by anger among boys (Benedini & Fagan, 2018).

Peer socialization context

Peers also shape adolescent substance use directly and indirectly. 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 attitudes or behaviors related to substance use, whereas socialization reflects an adolescent’s change in attitudes or behaviors related to substance use to assimilate to peers. Both processes result in high similarity among adolescents and peers regarding substance use. In one study, adolescent alcohol use predicted peer drinking, supporting the selection process, and peer alcohol use predicted adolescent drinking, supporting the socialization process (Patrick et al., 2016).

The relative influence of selection versus socialization varies across substances and development. Modeling and socialization influences had a strong effect on adolescents’ use of soft drugs (e.g., alcohol, marijuana, tobacco), but only a moderate to average effect on their use of hard drugs (e.g., cocaine, heroin; Kruis et al., 2020). Other work has supported peer socialization and selection processes for alcohol use, but only peer selection for marijuana use (Becker & Curry, 2014) and cigarette use (Kiuru et al., 2010). 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 operate simultaneously to affect adolescent substance use, 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 affect substance use: descriptive norms (i.e., an adolescent’s belief about the prevalence of substance use) and injunctive norms (i.e., an adolescent’s belief about approval of substance use). In one study, popular adolescents who were asked directly about their use of cigarettes and marijuana reported almost no use (Helms et al., 2014). However, classmates perceived that the same popular teenagers were smoking up to three cigarettes a day and using marijuana up to nine times a month. Moreover, higher perceptions of popular peers’ substance use in ninth grade were associated with a steep increase in substance use (Helms et al., 2014).

Studying the impact of media exposure and communication on adolescent substance use is a burgeoning field related to socialization contexts. Researchers have identified associations between more traditional media exposure related to substance use (e.g., seeing actors smoke cigarettes on television) and adolescent substance use. But technological advances providing 24-h
access to videos, text messaging, and social networks have created new opportunities for peers, marketing companies, and celebrities to influence adolescents’ attitudes toward substance use (Boyle et al., 2016). Nearly ubiquitous smartphone use among teenagers has fueled increased online activities to the point that approximately 45% of youth report being online nearly constantly (Pew Research Center, 2018). With increased time online comes greater exposure to images depicting substance use on websites and social networking platforms. In one study, greater exposure to alcohol-related advertising and social media content predicted higher levels of alcohol consumption (Boyle et al., 2016). Moreover, exposure to substance-related content via social media likely affected substance use via perceived norms and the formation of favorable attitudes toward substance use (Davis et al., 2019), partly due to the glamorized portrayal of substance use on social media, which offers an overabundance of misleading media content. Yet, the content and context of media exposure are critical with respect to adolescent substance use: Exposure to educational programs, 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

Substance use by adolescents is a major 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. Adolescent substance use often reflects a sequential progression of risk that emerges in childhood and is characterized by deficient or elevated behavioral inhibition; this, 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 affect adolescent decision making and behaviors. Accordingly, researchers should continue to incorporate multiple levels of influence in their work to mirror the inherent complexity in the etiology of adolescent substance use. Consider, for example, a study that examined individual temperament, peer difficulties, and overt antisocial and substance use outcomes in adolescence (Buil et al., 2017). This work highlights the synergistic effects between social contexts and individual development.

Another study examined the role of developmental pathways, including polygenic risk, prenatal stress, warm parenting, and internalizing and externalizing problems, in predicting adolescent substance use (Marceau et al., 2020). Work that examines multiple levels of influence is critical to informing multidimensional interventions.

Adolescent behavior associated with substance use is nested within multiple spheres of socialization contexts, as well as biologically based individual risk factors. A 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 for youth that integrates modules focused on preventing substance use, depression, and anxiety (Teeson et al., 2020), which is consistent with cascade models supporting the role of internalizing problems in adolescent substance use. Students receiving the Climate Schools-Combined curriculum reported increased knowledge of marijuana use and rated their likelihood of drinking as lower than students receiving a stand-alone 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 with 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, work that incorporates real-time normative feedback interventions to modify perceptions regarding the frequency and approval of peer substance use to mobile applications is promising (Davis et al., 2019).

Additionally, just-in-time-adaptive interventions that are often administered through wearable technology and mobile phones via user-specific input, geolocation, and passive sensing allow for the detection and initiation of interventions (Nahum-Shani et al., 2018). Wearable sensors and mobile phones can inform the unique social ecologies (e.g., peer groups), contexts (e.g., sporting events), and individual factors (e.g., mood, physiological changes) that contribute to increased risk for substance use and 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 to develop personalized treatment packages delivered via technology that target multiple domains of influence affecting adolescent substance use will be invaluable.

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