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Regular Article

Leveraging bifactor modeling to test prospective direct and indirect effects of adolescent alcohol use and externalizing symptoms on the development of task-general executive functioning

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Abstract

Adolescence is a period of substantial maturation in brain regions underlying Executive Functioning (EF). Adolescence is also associated with initiation and escalation of Alcohol Use (AU), and adolescent AU has been proposed to produce physiological and neurobiological events that derail healthy EF development. However, support has been mixed, which may be due to (1) failure to consider co-occurring externalizing symptoms (including other drug use) and poor social adaptation, and (2) heterogeneity and psychometric limitations in EF measures. We aimed to clarify the AU-EF association by: (1) distinguishing general externalizing symptoms from specific symptoms (AU, aggression, drug use) using bifactor modeling, (2) testing prospective associations between general externalizing symptoms and specific symptoms, and task-general EF, as indexed by a well-validated computational modeling framework (diffusion decision model), and (3) examining indirect pathways from externalizing symptoms to deficits in task-general EF through poor social adaptation. A high-risk longitudinal sample ($N = 919$) from the Michigan Longitudinal Study was assessed at four time-points spanning early adolescence (10–13 years) to young adulthood (22–25). Results suggested a critical role of social adaptation within peer and school contexts in promoting healthy EF. There was no evidence that specific, neurotoxic effects of alcohol or drug use derailed task-general EF development.

Keywords: Adolescence; alcohol use; bifactor modeling; externalizing; executive functioning

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Introduction

Executive functioning refers to a hypothesized family of top-down mental processes that support concentration, inhibition, attention, and behavioral regulation (Diamond, 2013). Healthy development of executive functioning is essential for mental and physical health, academic achievement, and success in life (Moffitt et al., 2011; Royall et al., 2002). While executive functioning may be biologically based, it is also influenced by environmental experiences (Müller & Kerns, 2015). It has been proposed that early adolescent alcohol use produces a cascade of physiological and neurobiological events that adversely affect trajectories of brain development (Crews et al., 2007), including the development of executive functioning (Squeglia et al., 2014). Despite some support for this idea (Wetherill et al., 2013; Thoma et al., 2011), other studies report no association between early alcohol use and subsequent executive functioning (Squeglia et al., 2009; Tapert et al., 2002). Indeed, in a review of this literature, Spear (2018) concluded that many of the studies that report negative neurocognitive consequences of adolescent alcohol use may overstate this conclusion; while there may be negative associations between alcohol use and performance on one or two cognitive tasks, alcohol-using youth compared to non-drinking controls did not exhibit altered performances on the majority of cognitive tasks examined. A better understanding of the ways in which early alcohol use may impact executive functioning development is of critical public health importance, and the current study aims to address this gap.

The extant literature that has investigated the association between adolescent alcohol use and subsequent executive functioning has two notable limitations that may explain the mixed empirical evidence. First, most studies have not considered alcohol use in the broader context of problem behavior (Squeglia et al., 2009; Wetherill et al., 2013). This leaves open the question of whether poor executive functioning may be due to the neurotoxic effects of alcohol or drug use, or a complex cascade involving externalizing symptoms and poor social adaptation. There is empirical evidence that the broader domain of externalizing symptoms increases risk for poor social adaptation which, in turn, hampers healthy executive functioning development (Eisenberg et al., 2015). Accordingly, it is important to consider the developmental context of adolescent alcohol use, and the potential pathways from externalizing problems, including consideration of indirect effects through social adjustment across several domains

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when examining associations between alcohol use and executive functioning. Failure to do so may yield a confusing and mixed pattern of results.

Second, the executive functioning literature faces significant challenges related to definition and measurement (Nigg, 2017). Indeed, the dominant framework posits that executive functioning (also commonly referred to as "self-regulation," or "self-control") is a broad construct comprised of a set of complex, modular functions (Miyake et al., 2000). However, this framework has led to considerable heterogeneity in measurement and terminology (Nigg, 2017) and has not been well-supported by subsequent psychometric studies (Karr et al., 2018). This has likely contributed to confusion regarding the link between adolescent alcohol use and cognitive development. An alternative framework that has emerged from the computational modeling literature posits that individuals' efficiency of accumulating goal-relevant evidence is a cognitive individual difference that underlies many of the modular executive functions (Löffler et al., 2024; Weigard et al., 2021; Weigard & Sripada, 2021). This alternative framework shows great promise in unifying executive functions and has not been considered with respect to how alcohol may impact executive functioning.

To address these aforementioned gaps, the current study proposes to: (1) Utilize bifactor modeling to parse general externalizing symptoms and the unique, neurotoxic effects of alcohol use and other drug use, (2) Examine indirect effects to better understand how adolescent alcohol use, other drug use, the broader constellation of externalizing behavior, and social adaptation may be associated with the development of executive functioning, and (3) Apply diffusion decision modeling, a cognitive modeling approach, to behavioral data in order to provide a precise index of task-general executive functioning (efficiency of evidence accumulation; drift rate).

The externalizing context and social development

Externalizing symptoms have received little attention in studies examining the alcohol use-executive functioning link (Squeglia et al., 2009; Wetherill et al., 2013). The omission of externalizing symptoms is notable because past work considering the alcohol use-executive functioning association posits that the specific neurotoxic effects of alcohol use derail healthy executive functioning development (López-Caneda et al., 2013); however, an alternative possibility is that, separate from alcohol use but highly comorbid, externalizing symptoms (e.g., aggressive and rule-breaking behaviors) increase risk for poor social adaption, which in turn, hampers executive functioning development. Externalizing symptoms and alcohol use (and perhaps other drug use) may each lead to poor executive functioning, but through different mechanisms (equifinality).

Several threads of research support the idea of executive functioning operating through social adaptation. The development of executive functioning is facilitated by strong interpersonal relationships and experiences that help scaffold development of these cognitive abilities. Indeed, there is empirical evidence that social factors, such as parental scaffolding and social modeling, facilitate executive functioning development (Carlson, 2003; Geeraerts et al., 2021). This is important because externalizing symptoms may disrupt critical social interactions that promote executive functioning. Indeed, externalizing symptoms negatively impact relationships with peers and parents, and school engagement (Baumeister et al., 2005; Buist et al., 2004; Moilanen et al., 2010). Poor social adaptation and low engagement in school, in turn, may hamper healthy development of executive functioning (Eisenberg et al., 2015; Farley & Kim-Spoon, 2014; Heylen et al., 2017, 2019). Hence, failure to consider externalizing symptoms when testing associations between alcohol use and executive functioning leaves open the possibility that the relationship between alcohol use and poor executive functioning is due to externalizing symptoms rather than the neurotoxic effects of alcohol use. Consideration of the broader externalizing context is consistent with recent calls for developmental psychopathology research to account for the co-occurrence of related symptoms and the hierarchical structure of psychopathology (Colder et al., 2013; Forbes et al., 2016; Kotov et al., 2017). Yet, no studies, to our knowledge, have tested mechanisms of the association between externalizing symptoms and poor executive functioning in adolescence, and this remains a significant limitation in the literature. The current study addresses this gap.

Measurement of executive functioning

The majority of studies examining links between alcohol use and executive functioning have used assessments designed to measure distinct executive functions (e.g., attentional shifting, working memory, inhibitory control, etc.; Nigg, 2017), which often suffer from psychometric issues (Eisenberg et al., 2019; Enkavi et al., 2019; Hedge et al., 2018). For example, the Stop Signal Task (SST) measures the ability to suppress or stop prepotent responses inhibitory control, a key executive function (Diamond, 2013; López-Caneda et al., 2013). Most studies using the SST and similar tasks rely on reaction time-based indices to measure inhibitory control. However, there is growing evidence that measures of Stop Signal Reaction Time (SSRT), response time (RT), and accuracy-based indices display poor to moderate test-retest reliability over short periods of time (e.g., three weeks; Hedge et al., 2018; Thunberg et al., 2023). Additionally, as noted above, fractionated approaches to conceptualizing and measuring executive functioning have demonstrated poor construct validity as individual difference dimensions (Karr et al., 2018; Löffler et al., 2024; Eisenberg et al., 2019). Poor construct validity is likely due in part to the fact that indices derived from a given executive function task are known to be influenced by multiple construct-irrelevant sources of variability, including measurement artifacts associated with participants' level of response caution (e.g., speed/accuracy tradeoffs) and motor response speed (Hedge et al., 2019; Lerche & Voss, 2019; Stafford et al., 2020).

An emergent alternative to this "fractionation" perspective uses formal mathematical models to describe the neurocognitive mechanisms that underlie task performance. Evidence accumulation models are a well-validated framework from mathematical psychology and computational neuroscience that explain individuals' choices and RTs on a wide array of behavioral tasks (including executive function tasks). The models posit a process in which people gather evidence for possible responses until a critical evidence threshold is reached for a given choice (Smith & Ratcliff, 2004; Heathcote & Matzke, 2022). The diffusion decision model (DDM; Ratcliff, 1978; Ratcliff et al., 2016), one of the most commonly used evidence accumulation models, decomposes task performance into parameters representing several mechanisms of influence on behavioral performance: (1) efficiency of evidence accumulation toward the correct choice ("drift rate"; v), (2) degree of response caution (boundary separation; a), (3) response biases, which tend to favor decisions with higher probabilities (start point; z), and (4) processes unrelated to the decision process, such as time

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for stimulus encoding and motor response execution (nondecision time; *Ter*; Voss et al. 2013). The DDM has previously been used to measure these mechanisms on the SST by accounting for construct-irrelevant influences on SST performance (Epstein et al., 2023; Fosco et al., 2019b; Karalunas & Huang-Pollock, 2013).

Application of the DDM to large task batteries has repeatedly found that the model's drift rate parameter forms a cohesive general factor that drives individual differences in performance across many complex cognitive tasks and shows strong psychometric properties, including good test-retest reliability (Eisenberg et al., 2019; Lerche et al., 2020; Schubert et al., 2016; Schmiedek et al., 2007). In contrast to assumptions of the "fractionation" perspective, recent work demonstrates that the vast majority of the variance in individuals' performance on a variety of tasks thought to measure specific executive functions can instead be explained by this task-general factor (Löffler et al., 2024; Weigard et al., 2021; Weigard & Sripada, 2021). As drift rate can be conceptualized as individuals' efficiency of selectively gathering goal-relevant evidence to make adaptive choices across contexts, it also has strong conceptual links to traditional definitions of executive function (Weigard & Sripada, 2021). Taken together there is strong evidence to support the drift rate parameter as an index of task-general executive functioning. Accordingly, the current study applied the DDM to the SST to characterize general executive functioning (Weigard et al., 2020; Zucker et al., 1996, 2000).

Reciprocal relationships

There is considerable empirical evidence that relationships between executive functioning, externalizing symptoms, and alcohol and drug use operate bidirectionally. Indeed, past work has reported that healthy levels of executive functioning are protective against a wide range of adverse psychosocial outcomes (Duckworth, 2011; Royall et al., 2002), including externalizing symptoms (Alessandri et al., 2022; Hentges et al., 2020; Wiker et al., 2023), and alcohol and drug use (Nigg et al., 2006; Piehler et al., 2012). There is also evidence of bidirectional relationships between social adaptation and problem behavior, with healthy social adjustment predicting lower levels of externalizing symptoms and substance use across adolescence (Burke et al., 2008; Jacobson & Crockett, 2000; Prinstein & La Greca, 2004). Although not central to the current study, we examine possible reciprocal relationships between task-general executive functioning, externalizing symptoms, alcohol and drug use, and social adaptation across parents, peers, and school.

The current study

The current study uses data from the Michigan Longitudinal Study (MLS). Of note, the sample is enriched for children of parents with an Alcohol Use Disorder (AUD), who are at risk to exhibit high levels of early adolescent alcohol use and externalizing psychopathology and low levels of executive functioning (Meldrum et al., 2018; Puttler et al., 2017). Greater representation of the high-end of problem behavior will strengthen our ability to detect the proposed associations (Paige et al., 2021). The hypotheses, design, and analyses of the current study were preregistered: https://osf.io/75zwg/?view_only = 46d2a2b0aa8d485d8acb860f4bdc1044. Some deviations from the preregistration should be noted. Specifically, we initially proposed to utilize several measures of executive functioning, including two behavioral tasks (the go/no-go in addition to the SST) as well as neuroimaging measures, in order to explore how hypothesized associations may differ across measures of executive functioning. After accessing the full dataset from the MLS, the sample sizes of the SST data (N range = 360–553) were substantially larger than the go/no-go (N range = 106–532) and neuroimaging (N range = 93–214) data across the four waves. Additionally, our own psychometric work with the neuroimaging data raised concerns about its reliability and validity. We elected to publish this psychometric work (Paige et al., 2024) separately and focus here on the central aim of the preregistered study, testing the ways in which adolescent alcohol use, externalizing symptoms, and social adaptation impact the development of executive functioning.

Aim 1

Test longitudinal bifactor models of externalizing symptoms from adolescence to early adulthood, which provides a way to separate alcohol use specific variance from general externalizing symptoms at four assessments spanning early adolescence to early adulthood and to examine whether the structure of co-occurring and unique symptoms vary across age. We hypothesize that a latent factor characterized by general externalizing symptoms and domain specific factors for alcohol use, drug use, aggressive behaviors, and rule-breaking behaviors will emerge at each assessment (see Figure 1).

Aim 2

Test prospective associations between general externalizing symptoms and domain specific facets and task-general executive functioning. We hypothesize that higher-order factors of externalizing symptoms will be related to poor task-general executive functioning. We also hypothesize that the specific factors of alcohol use, drug use, aggression, and rule-breaking will be related to poor task-general executive functioning.

Aim 3

Test potential indirect associations that may underlie the relationship between externalizing symptoms and deficits in task-general executive functioning. The relationship between alcohol use (and perhaps drug use) and task-general executive functioning is thought to represent a direct association consistent with the neurotoxic effects of adolescent alcohol use on the developing brain (Squeglia et al., 2014). In contrast, associations between externalizing symptoms and poor task-general executive functioning are posited to operate through negative socio-environmental experiences (Fay-Stammbach et al., 2014). This aim tests potential indirect pathways involving parents, peers, and school (see Figure 2). We hypothesize that high levels of externalizing symptoms will be associated with disrupted parental monitoring, low peer connectedness, and poor school engagement, which in turn, will be related to poor task-general executive functioning.

Method

Participants

Risk level of the offspring in the MLS was varied through recruitment of a population-based sample that differed in level of Alcohol Use Disorder (AUD) among the fathers (Zucker et al., 2000). The highest risk group (41%) were drunk drivers with at least 0.15% blood alcohol levels who also met other ascertainment criteria. A moderate risk group (29%) were community members with an AUD, uncovered during the canvass for controls. All moderate and high-risk individuals had an AUD diagnosis, were coupled with a partner

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Figure 1. Conceptual model of bifactor model for externalizing symptoms (Aim 1). *Note*. To reduce the number of model parameters, the rule-breaking and aggressive behavior subscales of the Achenbach measures were each parceled into five bundles at each wave.

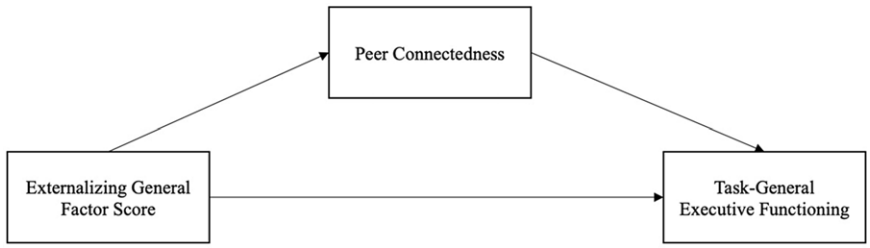

Figure 2. Conceptual model depicting indirect pathways from general externalizing symptoms factor scores to task-general executive functioning operating through peer connectedness (Aim 3). *Note*. Peer connectedness is depicted here, and separate models were estimated for each proposed social adaptation variable (e.g., parental monitoring, peer connectedness, and school engagement).

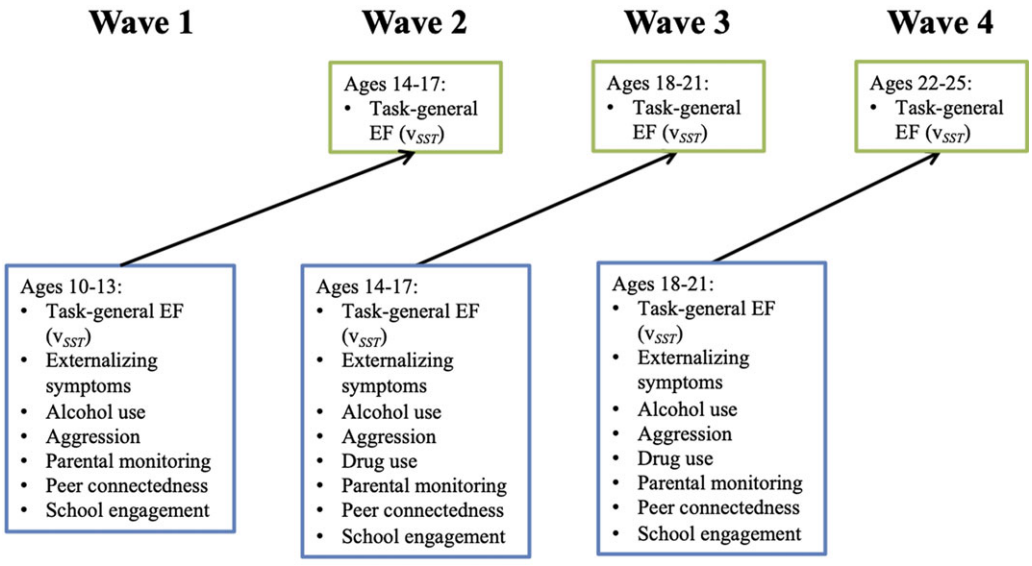

Figure 3. Conceptual model depicting multilevel model for testing main effects between general externalizing symptoms, specific alcohol use, specific aggression, social adaptation, and task-general executive functioning. *Note*. For simplicity, the random intercept and covariates are not depicted here. Sex, racial/ethnic status, parental education, computer version and remaining drift diffusion model parameters, nondecision time, response conservativeness, and response bias were included as covariates.

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Table 1. Sample sizes

Wave	Mean Age (range)	Bifactor (N)	Parental Monitoring (N)	Peer Connectedness (N)	School Engagement (N)	Stop Signal Task (N)
1	12.14 (10–13)	875	793	988	983	360
2	15.98 (14–17)	919	903	1170	989	542
3	19.88 (18–21)	782	76	1031	130	552
4	23.93 (22–25)	652	0	774	0	470

Note. We selected four waves to represent developmental periods of adolescence, each that spans multiple years. Therefore, some participants contributed more than one observation within each developmental period. In these cases, we selected the observation that was closest to the average age within that wave.

and had a son who was 3 to 5 years old. The low risk group (30%) was an ecologically comparable set of families with no parental AUD at the time of recruitment, who were recruited from neighborhoods where the high-risk participants lived. Families were excluded from the MLS if the target child displayed signs of fetal alcohol syndrome or the mother reported drinking during pregnancy. Full biological siblings were also included in the MLS if they were within eight years of age of the male target child. Due to initial MLS recruitment, the majority of the sample was male (61.7%) and White (80.7%). Among adolescents of color, the largest groups were African American (7.4%), Hispanic (5.1%), and Biracial (2.7%). In subsequent years, families were added to the MLS to increase demographic diversity. The current study uses a subset of the MLS sample selected to represent four critical developmental windows: early adolescence (ages 10–13), middle adolescence (ages 14–17), late adolescence (ages 18–21), and early adulthood (ages 22–25). Sample sizes for all data across the four waves are presented in Table 1. For simplicity, these four developmental windows will be described as Waves (W) 1, 2, 3, and 4 henceforth.

The larger psychosocial sample used in the current study was comprised of majority males (67.2%) and was majority non-Hispanic White (80.5%). Additionally, on average, parents of target adolescents had completed some college (48.8%), with an additional 24.3% of parents completing college and 13.5% of parents possessing postgraduate education.

All procedures were approved by an Institutional Review Board. If participants were under the age of 18, consent from caregivers and assent from adolescents were obtained prior to participation. Adolescents provided informed consent after age 18.

Procedure

The MLS involves repeated measures of alcohol use, drug use, behavioral and psychological functioning, environment, and psychopathology that were all assessed across the four developmental windows described above. Annual assessments occurred between ages 10 and 25 for a subset of variables, including all alcohol use, drug use, and psychosocial variables used in the current study. Participants also completed behavioral tasks, including the SST, in a lab setting.

Measures

Demographics

Participant sex (0 = male, 1 = female), racial/ethnic status (0 = people of color, 1 = Caucasian/non-Hispanic White), and parental education (1 = Less than High School, 2 = High School, 3 = Some College, 4 = Bachelors, 5 = Post. Grad.) were included as covariates. Socioeconomic resources is a multidimensional construct that includes parental education, household income, and neighborhood disadvantage (Ensminger & Fothergill, 2003). A recent well-powered, multisite study using longitudinal Adolescent Brain Cognitive Development Study (ABCD) data found that parental education (controlling for household income-to-needs and neighborhood disadvantage) exhibited strong, consistent effects on brain development (Sripada et al., 2022). Therefore, the current study used highest reported parental education as a measure of socioeconomic resources.

Alcohol and drug use

The Drinking and Drug History Forms for Children/Youth and Adults were administered to provide developmentally appropriate assessments of alcohol, cannabis, cigarette, and illicit drug (e.g., cocaine, hallucinogens, and nonprescription opioids) use annually (Zucker et al., 1990, 1993). These measures assessed quantity, frequency, and variability of alcohol and drug use as well as past year alcohol and drug use problems. At ages 18–23, 64% of MLS participants met criteria for AUD, validating the high-risk nature of the sample (Puttler et al., 2017).

Externalizing symptoms

The Youth Self-Report (YSR) and Adult Self-Report (ASR) from the Achenbach System of Empirically Based Assessments were used to provide developmentally appropriate assessments of externalizing symptoms annually (Achenbach et al., 2003; Achenbach & Rescorla, 2001). Externalizing problems on the YSR and ASR included two subscales, rule-breaking and aggressive behaviors. Items that measured alcohol and drug use were removed from the externalizing dimension in order to avoid confounding with variables measured by the Drinking and Drug History Forms for Children/Youth and Adults. Internal consistencies of the externalizing scale on the YSR and ASR were good across W1-4 (α range = 0.87–0.88), and were fair to good across W1-4 for the rule-breaking (α range = 0.70–0.77) and aggressive behaviors (α range = 0.83–0.84) subscales.

Parenting context

The Parent Monitoring-Youth Form is a 10-item measure that was administered to children annually beginning at age 11 to measure the supervision and monitoring provided by parents or caretakers outside of the school environment (Chilcoat, & Anthony, 1996). It was adapted for age appropriateness by the MLS project from work by Patterson and Capaldi (1988). In order to be developmentally appropriate, this measure was administered until targets were 18-years-old. Therefore, the current study only had parental monitoring data across W1-3. Parental monitoring was assessed through seven items that asked the target child to respond how often their parents are involved, are present with them, or how often their parents are involved in monitoring their behavior or whereabouts. Sample items included, "How often, before you go out, do you tell your [mom/dad/mom and dad/caregiver] when you will be back?"

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and "If you are at home when your parents are not, how often do you know how to get in touch with them?". Responses were rated on a 5 point Likert scale (1 = All of the time, 5 = Never), and were reverse-coded in the current study so that higher scores indicated higher parental monitoring. Adolescents could also respond 9 = if the item did not apply to them. These responses were coded as missing data. The seven items were averaged within wave to compute scale scores of parental monitoring. Internal consistencies were fair to good across W1-3 (α range = 0.68–0.88).

Peer context

The 10-item Peer Relationships subscale of the Self-Image Questionnaire for Young Adolescents was used to measure peer connectedness (Petersen et al., 1984). This measure was administered annually beginning at age 11 and continued through early adulthood, spanning W1-4 in the current study. Adolescents were asked to answer questions, "about the way you interact with your friends." Sample items included, "I find it extremely hard to make friends," and "I think that other people just do not like me." Responses to the scale items range from 1 = "very strongly agree" to 6 = "very strongly disagree." Responses were reverse-scored and scale scores were computed so that higher scores indicate higher levels of peer connectedness. Instrument validity has been demonstrated (Petersen et al., 1984), and internal consistencies were good across W1-4 (α range = 0.81–0.89).

School context

The Teacher Report Form (TRF; Achenbach & Rescorla, 2001) was administered annually and was used to measure school engagement. The TRF was completed by the one of the participant's primary teachers. This measure was administered until targets were 18-years-old; therefore, the current study measured school engagement across W1-W3. Throughout the MLS, the return rate for teacher participation was just under 90%. School engagement was assessed using four items that asked teachers to compare the target adolescent's functioning to typical pupils of the same age. Sample items included, "how hard is he/she working?" and, "how happy is he/she?". Responses to the scale items range from 1 = "much less" to 6 = "much more." Internal consistencies were good across W1-3 (α range = 0.88–0.90).

Executive functioning

Stop-signal task. The Stop-Signal Task (SST; Logan, 1994), a computerized choice reaction time task designed to measure inhibition, was administered annually in the MLS. Participants were presented with an "X" or "O" on a computer screen and instructed to respond by pressing one of two keys as quickly as possible upon seeing the stimulus. They were also instructed to withhold response (stop themselves) upon hearing a tone (stop signal) following the stimulus on the screen. The stop signal occurred on 25% of the trials. A stochastic process occurred in which participants' response on prior trials was used by the program to set stop signal delay (warning time) on subsequent trials, in order to maintain a successful inhibition rate of 50%. Four blocks (256 trials), following two practice blocks, were administered in the MLS. The first practice block of 32 trials was used to practice the "go" part of the task and to set participants expectancy to respond as rapidly as possible to the "X" or "O" choice task. The second practice block was used to teach them to recognize the auditory stop signal.

Initially, the DOS computer program was used to administer the SST. As the study continued, an EPrime version of the SST was used. About 21% of the current sample complete the SST using the earlier DOS computer program. We considered only including cases that completed the SST using EPrime; however, we were hesitant to reduce the sample size as well as exclude participants who were more likely to be female and people of color. Therefore, we elected to utilize all SST data and covary computer program, in addition to sex, racial/ethnic status, and parental education in all analyses using SST data.

Data analytic strategy

Diffusion decision model

Stop-signal task. The diffusion model was fit to SST data using functions from the Dynamic Models of Choice suite of R functions (Heathcote et al., 2019). The central four drift diffusion model parameters were estimated for each individual: drift rate (v_{SST}), response conservativeness (a_{SST}), nondecision time (Ter_{SST}), and response bias (z_{SST}). Between-trial variability in *Ter* was also estimated but ther other between-trial variability parameters were fixed to 0 given the difficulty of recovering these parameters at low trial numbers and evidence that they have a minimal impact on estimates of the main parameters (Lerche et al., 2017). Individual-level Bayesian estimation was implemented using the differential evolution Markov chan Monte Carlo (DE-MCMC: Turner et al., 2013) approach with broad, uninformative priors. Upon examination of the response bias (z_{SST}) variables, data at W1 and W3 were kurtotic. Two cases at W1 and one case at W3 fell more than 3 standard deviations below the mean, suggesting noncompliance with the task. These cases were removed, and the data were normally distributed. Drift rate (v_{SST}) represents efficiency of gathering goal-relevant evidence to make an adaptive choice; larger drift rate values indicate faster information accumulation or better task-general executive functioning. The a_{SST}, Ter_{SST}, and z_{SST} parameters were included as covariates across all models predicting v_{SST}.

Hypothesis testing

Analyses were performed in SAS ® 9.4 and Mplus Software (Muthén & Muthén, 1998-2018; SAS Institute Inc., 2013). Sex, racial/ethnic status, and parental education were included as statistical control variables in regression models (Karreman et al, 2009; Squeglia et al., 2009; Windle, 1990).

Aim 1. To test aim 1, confirmatory factor analysis with Robust Maximum Likelihood estimation was used to estimate bifactor models of externalizing symptoms at four assessments spanning early adolescence to early adulthood. Bifactor modeling allows for all items to load onto a general factor and domain specific factors, thereby separating cross-sectional alcohol use specific variance from general externalizing symptoms variance (Figure 1). The general factor was indicated by items on the YSR and ASR, and items on the Drinking and Drug History Forms. The alcohol use specific factor was indicated by five items which assess alcohol use in the past year: past year frequency of alcohol use, number of drinking days in the past six months, quantity of alcohol use on drinking days in the past six months, number of days past year binge-drinking, and a count variable of past year alcohol use problems. The drug use specific factor was also indicated by five items: past year frequency of cigarette use, past month quantity of cigarette use, past year frequency of cannabis use, a count variable of past year drug use problems, and a count variable of past year other illicit drug use (e.g., hallucinogens, cocaine, nonprescription

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opioids, etc.). To reduce the number of model parameters, the rule-breaking and aggressive behavior subscales of the Achenbach measures were each parceled into five bundles at each wave (Little et al., 2013). Fit of the bifactor models was assessed using conventional (e.g., comparative fit index [CFI], root mean square error approximation [RMSEA], standardized root mean square residual [SRMR]) as well as alternative fit statistics developed for bifactor models (e.g., omega, omega hierarchical subscale (ω_{HS}), and construct replicability (H); Dueber, 2017; Rodriguez et al., 2016a, 2016b). ω and ω_s are the latent variable analogs to coefficient alpha for the general externalizing factor and specific factors, respectively. ω_H differs from ω in that it only represents the variance from a single latent variable whereas ω is a function of all common variance. ω_{HS} is an index of the reliability of the specific factors after partitioning out the variance attributable to the general factor. The only computational difference between ω_S and ω_{HS} is that in the numerator, the term associated with variance on the general factor is removed, leaving only the variance associated with the specific factor. ω, ω_S, ω_H, and ω_{HS} values greater than .70 indicate acceptable reliability and values greater than .80 indicate good reliability (Cortina, 1993; Rodriguez et al., 2016b; Santos, 1999; Tavakol & Dennick, 2011). To evaluate fit of the bifactor model, values lower than .80 for Percentage of Uncontaminated Variance and greater than .70 for Explained Common Variance indicate that data are unidimensional enough to introduce bias in fitting a multidimensional model (Quinn, 2014; Reise et al., 2013). Values of construct replicability, H, > .70 indicate that measured items adequately represent the latent factors (Hancock & Mueller, 2001; Rodriguez et al., 2016a). Factor score determinacies > .70 indicate adequate correlation of factor scores with latent factors (Rodriguez et al., 2016b). Nested tests were used to evaluate the invariance of the factor structure across time. In order to reduce model complexity, which is a common concern for longitudinal bifactor models (McElroy et al., 2018), factor scores were computed to be used in our cross-lagged MLM regression models.

Aim 2 (Figure 3). To test aim 2, MLMs were estimated in PROC MIXED procedure in SAS 9.4 with maximum likelihood estimation (ML). MLM allows for varying times of assessments between participants and easily accommodates missing data (Tabachnick, & Fidell, 2013). Repeated measures (level 1) were nested within participants (level 2), and data were arranged to test cross-lagged associations (e.g., externalizing symptoms, alcohol use, and aggression at W1 predicts drift rate at W2, controlling for W1 drift rate, and this structure was repeated through W4). The models included a random intercept, and sex, racial/ethnic status, and parental education as level 1 covariates. All independent variables were standardized at the sample level to aid in the interpretation of the results (Hamer & Simpson, 2009). The MLMs that included drift rate as outcomes also included covariates from the DDM (nondecision time; *Ter*, response conservativeness; *a*, and response bias; *z*)[1].

In order to examine bidirectional associations, the models were restructured to test task-general executive functioning (drift rate) as a prospective predictor of each symptom factor. Separate models were run for each symptom factor (general externalizing, alcohol use, drug use, and aggression).

Aim 3. MLM using the PROC MIXED procedure in SAS 9.4 with ML and Monte Carlo simulation with 20,000 samples was used to test aim 3 (Preacher & Selig, 2012). Cross-lagged models were arranged such that externalizing symptoms at W1 predicted peer connectedness at W2, controlling for W1 peer connectedness, and this structure was repeated through W4. Given that no data were collected on parental monitoring and school engagement at W4, as was developmentally appropriate, cross-lagged models to test indirect effects through parental monitoring and school engagement were arranged such that externalizing symptoms at W1 predicted peer connectedness at W2, controlling for W1 peer connectedness, and this structure was only repeated through W3. In order to examine univariate effects, separate models were estimated for each proposed social adaptation variable (e.g., parental monitoring, peer connectedness, and school engagement; MacKinnon, 2000). A model that included all the proposed indirect assocaitions was also estimated to examine unique effects. Then, a cross-lagged model was estimated such that parental monitoring, peer connectedness, and school engagement at W1 predicted drift rate at W2, controlling for W1 drift rate, and this structure was repeated through W4. All models included a random intercept and sex, racial/ethnic status, and parental education as level 1 covariates. All predictor variables were standardized (Tabachnick, & Fidell, 2013). Estimates (e.g., A and B paths) were input into a Monte Carlo simulation to test indirect effects (Preacher & Selig, 2012).

[1]MLMs without the DDM covariates (*Ter*, *a*, and *z*) were also estimated. Patterns of results across models that did and did not include DDM parameters as covariates were consistent with respect to hypothesized pathways.

Results

Descriptive statistics

At W1, 96% of adolescents endorsed no alcohol use, which is not surprising given the age of the sample (10–13-years-old). Drinking increased across adolescence and early adulthood, with 30.3%, 71.6% and 86.3% of participants reporting past year alcohol use across Waves 2, 3, and 4, respectively. Specifically, at W2 (ages 14 to 17) about 13.7% of adolescents reported less than monthly drinking, 4.5% drank monthly, 11.3% drank about once a week, and less than 1% drank more than once a week. At W3 and W4, adolescents and early adults reported drinking 4.07 and 6.62 days per month on average, respectively. Alcohol use problems also increased across development. Whereas only 1.2% of adolescents at W1 endorsed any problems, this number increased to 21.7%, 48%, and 56.5% across waves 2, 3, and 4, respectively. Regarding illicit drug use, as expected, use was low at W1; 98.8% and 96.7% of adolescents reported no cannabis and no cigarette use, respectively. Cannabis use increased across adolescence, with 22.0% (W2) and 43.7% (W3) of adolescents reporting past year cannabis use, and then decreased slightly in early adulthood (38.5% endorsed past year use at W4). Endorsement of past year cigarette use increased across waves 2 (19.3%), 3 (41.9%), and 4 (46.4%). Experiencing problems related to illicit drug use was uncommon; 98.4%, 91.9%, 92.7%, and 96.8% endorsed 0 drug problems across waves 1, 2, 3, and 4, respectively.

Bifactor models

At W1 (ages 10–13), endorsement of illicit drug use was very low; 4.26% and 1.26% of the sample endorsed any cigarette and cannabis use in the past year, respectively. Therefore, these data were highly skewed and kurtotic, and we did not include drug use items in bifactor modeling at W1. Additionally, only 0.17% of the sample endorsed past year binge-drinking at W1, and this item was omitted from bifactor modeling at W1. Bifactor models of externalizing symptoms were estimated at each wave. Initial estimation of bifactor models produced a negative residual

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Table 2. Bifactor results for single wave and multiple wave models

Model	χ^2 (df), p value	CFI	TLI	RMSEA	SRMR
Early and Middle Adolescence					
W1 Bifactor	108.30(67), $p = .001$	0.98	0.97	0.03	0.03
W2 Bifactor	529.98(150), $p < .001$	0.95	0.93	0.05	0.09
W1 and W2 Combined Bifactor	941.58(480), $p < .001$	0.95	0.94	0.03	0.07
Late Adolescence and Early Adulthood					
W3 Bifactor	409.88(150), $p < .001$	0.96	0.95	0.05	0.09
W4 Bifactor	298.98(148), $p < .001$	0.97	0.97	0.04	0.06
W3 and W4 Combined Bifactor	1276.87(674), $p < .001$	0.96	0.95	0.03	0.08
Early Adolescence to Early Adulthood					
W1-W4 Combined Bifactor	4059.59(2500), $p < .001$	0.94	0.94	0.02	0.07

Note. CFI = comparative fit index. TLI = Tucker-Lewis index. RMSEA = root mean square error. Approximation. SRMR = standardized root mean square residual. *W* = wave.

variance for the rule-breaking specific factors. A negative residual variance in this context suggests that after accounting for the general externalizing symptoms factor there was little variability left in the rule-breaking specific factors (Bollen, 1989). Removing the rule-breaking specific factors and allowing the rule-breaking items to load only on the general externalizing factors led the models to estimate without negative variance estimates. Rule-breaking specific factors likewise produced negative residual variances in our previous work utilizing a community sample of adolescents to estimate bifactor models of externalizing symptoms (Paige et al., 2021). Results for individual bifactor models estimated at each wave (W1-W4) are presented in Table 2.

Then, the independent measurement models at each wave were combined into an overall measurement model with factors correlated across time. Details regarding longitudinal measurement invariance of bifactor models can be found in Supplemental Material 1. Fit information of final models is presented in Table 2. The final longitudinal externalizing symptoms bifactor measurement model including W1-W4 provided acceptable fit to the data ($\chi^2 = 4059.59(2500)$, $p < .001$, CFI = .94, TLI = .94, RMSEA = .02, SRMR = .07). Standardized factor loadings and alternative fit statistics across all bifactor models are presented in Table 3.

The alcohol use item loadings ranged from 0.21 to 0.51 and from 0.55 to 0.93 on the general externalizing factors and alcohol use specific factors, respectively, across W1-4. The drug use item loadings had similar weak loadings on the general externalizing factor across W2-4 (range = 0.24–0.53), and they also had weak loadings on the drug use specific factors (range = 0.26–0.55), with the exception of the annual cannabis use frequency item (range = 0.61–0.74). Regarding aggression, the item loadings generally fell within the acceptable range on the general externalizing factors (range = 0.43–0.67). Consistent with our previous work estimating bifactor models of externalizing symptoms (Paige et al., 2021), the aggression item loadings on the specific factors were weak across early and middle adolescence, and were stronger across late adolescence and early adulthood (see Table 3). Finally, with the exception of one weak loading in early adolescence, rule-breaking behavior item loadings on the general externalizing symptoms factor were acceptable, ranging from 0.52 to 0.73.

In sum, the conventional and alternative fit statistics suggest that the longitudinal bifactor model provided an adequate fit to the data. The general externalizing factors and alcohol use specific factors were well specified (strong factor loadings), while the drug use and aggression specific factors were not (weak factor loadings), suggesting that there was less common variance in these specific factors after accounting for variance in general externalizing symptoms. Additional fit indices for the bifactor models are provided in Supplemental Material 1.

Main effects of externalizing symptoms, specific alcohol and drug use, and aggression predicting task-general executive functioning

Main effects MLMs were run to test prospective main effects of externalizing symptoms, specific alcohol and drug use, and aggression on task-general executive functioning across adolescence and emerging adulthood, controlling for prior task-general executive functioning and demographic variables (e.g., sex, racial/ethnic status, and parental education; Path C; see Figure 2). The results of main effects models for externalizing symptoms, specific alcohol use[2] and aggression predicting task-general executive functioning are reported in Table 4 (see Model 1).

Results from the intercept only model revealed that the ICC was 0.5185, indicating that 51.85% of the variance for drift rate during SST was due to clustering within individuals. This represents fair test-retest reliability by conventional standards, but relatively robust given the average measurement interval of 2–3 years (whereas test-retest reliability is typically estimated across much shorter intervals)[3]. Stability of task-general executive functioning

[2]Because results from bifactor modeling did not support a specific factor of drug use at W1, we estimated a separate set of MLMs with data arranged to test cross-lagged associations (externalizing symptoms, alcohol use, drug use, and aggression at W2 predicts drift rate at W3, controlling for W2 drift rate, and this structure was repeated through W4, such that I was able to test two cross-lags). The models included a random intercept, sex, racial/ethnic status, and parental education as level 1 covariates. Results were consistent with the main effects MLMs testing associations across W1-4. Specific drug use did not significantly predict drift rate (v_{SST}; $\beta = -0.01$, $p = .65$).

[3]We conducted additional analyses to examine correlations between traditional metrics of behavioral performance on the SST and drift rate. Within-time correlations between drift rate and SSRT were small to moderate and negative, such that adolescents with faster drift rate (better task-general executive functioning) had lower (faster) reaction times (r range = −0.21 – −0.37). Additionally, within-time correlations between drift rate and go accuracy were positive and moderate in size across waves 2 ($r = 0.58$), 3 ($r = 0.44$), and 4 ($r = 0.43$). The within-time correlation was strong at W1 ($r = 0.74$). These correlations indicate that that adolescents with faster drift rate (better task-general executive functioning) had better accuracy on go trials. These associations support the psychometric

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Table 3. Standardized factor loadings for bifactor models across waves 1, 2, 3, and 4

	Factor														
Item	*G W1*	*AU W1*	*AB W1*	*G W2*	*DU W2*	*AU W2*	*AB W2*	*G W3*	*DU W3*	*AU W3*	*AB W3*	*G W4*	*DU W4*	*AU W4*	*AB W4*
Other drug use				0.29	0.26			0.31	0.37			0.24	0.44		
Cigarette quantity				0.42	0.48			0.39	0.33			0.37	0.34		
Cigarette frequency				0.49	0.55			0.38	0.39			0.35	0.33		
Cannabis frequency				0.53	0.71			0.44	0.64			0.45	0.61		
Drug use problems				0.41	0.44			0.30	0.38			0.25	0.41		
Binge-drinking				0.48		0.72		0.38		0.87		0.32		0.90	
AU problems	0.29	0.61		0.49		0.66		0.43		0.57		0.35		0.55	
Drinks per day	0.23	0.90		0.51		0.80		0.38		0.74		0.32		0.71	
Freq drinking days	0.21	0.93		0.52		0.74		0.40		0.80		0.25		0.81	
Annual AU Freq	0.21	0.86		0.50		0.75		0.35		0.77		0.22		0.66	
AB Parcel 1	0.57		0.27	0.52			0.41	0.54			0.58	0.43			0.64
AB Parcel 2	0.59		0.39	0.58			0.33	0.51			0.62	0.51			0.67
AB Parcel 3	0.62		0.60	0.63			0.35	0.50			0.52	0.51			0.51
AB Parcel 4	0.67		0.26	0.56			0.52	0.49			0.60	0.53			0.53
AB Parcel 5	0.61		0.42	0.59			0.39	0.56			0.39	0.61			0.27
RB Parcel 1	0.38			0.52				0.63				0.61			
RB Parcel 2	0.66			0.66				0.70				0.72			
RB Parcel 3	0.63			0.68				0.62				0.64			
RB Parcel 4	0.59			0.52				0.71				0.73			
RB Parcel 5	0.68			0.66				0.68				0.67			
ECV	0.52			0.54				0.51				0.48			
H	0.86	0.94	0.53	0.90	0.67	0.86	0.50	0.90	0.57	0.89	0.69	0.88	0.56	0.89	0.70
ω/ω_S	0.91	0.92	0.85	0.93	0.79	0.95	0.83	0.93	0.70	0.96	0.87	0.91	0.68	0.89	0.86
$\omega_{H/}\omega_{HS}$	0.69	0.85	0.24	0.77	0.45	0.65	0.27	0.75	0.40	0.68	0.45	0.70	0.42	0.77	0.44
FD	0.91	0.97	0.75	0.92	0.85	0.94	0.72	0.96	0.79	0.95	0.86	0.92	0.77	0.96	0.85

Note. *G* = General externalizing factor, *W* = Wave, AU = Alcohol Use, Freq=Frequency, AB = Aggressive Behavior, RB = Rule-breaking. ECV = Explained common variance. FR = Factor Determinacy.

Table 4. Multilevel modeling results for general externalizing symptoms, specific alcohol use, specific aggression, and social adaptation predicting task-general executive functioning (v_{SST})

	Model 1			Model 2			Model 3			Model 4			Model 5		
Variable	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value
Intercept	2.84	0.29	**9.64**	2.76	0.43	**6.44**	2.79	0.29	**9.49**	2.96	0.37	**7.95**	3.01	0.45	**6.64**
Sex	− 0.03	0.02	− 1.35	− 0.01	0.03	− 0.40	− 0.03	0.02	− 1.40	− 0.06	0.03	− 1.78	− 0.01	0.04	− 0.36
Racial/ethnic status	0.03	0.03	1.01	0.02	0.04	0.69	0.03	0.03	1.20	0.06	0.03	2.01*	0.05	0.04	1.35
Parental Education	0.09	0.03	**3.43**	0.07	0.04	1.78	0.09	0.03	**3.58**	0.05	0.03	1.52	0.05	0.04	1.11
Computer version	− 0.02	0.09	− 0.13	0.004	0.17	0.03	− 0.01	0.09	− 0.15	0.007	0.16	0.04	0.02	0.16	0.09
Task−general EF (v_{SST}) stability	0.43	0.02	**17.92**	0.45	0.04	**12.51**	0.42	0.02	**17.70**	0.38	0.03	**11.68**	0.38	0.04	**9.71**
Nondecision time (Ter_{SST})	2.23	0.33	**6.79**	1.92	0.47	**4.11**	2.25	0.33	**6.84**	1.93	0.43	**4.53**	1.93	0.49	**3.91**
Response conservativeness (a_{SST})	− 0.42	0.05	− **8.32**	− 0.35	0.07	− **5.06**	− 0.43	0.05	− **8.62**	− 0.37	0.06	− **5.94**	− 0.37	0.07	− **5.06**
Response bias (z_{SST})	0.88	0.57	1.53	1.14	0.83	1.36	1.02	0.57	1.78	0.72	0.73	0.97	0.66	0.89	0.75
General externalizing symptoms	− 0.04	0.02	− 1.72	0.003	0.04	0.09	− 0.03	0.02	− 1.44	− 0.01	0.04	− 0.34	0.02	0.05	0.48
Specific alcohol use	0.005	0.02	0.22	− 0.06	0.04	− 1.59	− 0.005	0.02	− 0.27	− 0.02	0.03	− 0.76	− 0.06	0.04	− 1.60
Specific aggression	0.03	0.02	1.24	− .002	0.04	− 0.06	0.04	0.02	1.56	0.02	0.04	0.44	0.03	0.05	0.64
Parental monitoring				0.04	0.30	1.18							0.004	0.04	0.11
Peer connectedness							0.07	0.02	2.84**				0.10	0.04	2.70**
School engagement										0.14	0.03	**4.16**	0.14	0.04	**3.46**

Note: Model 1 indicates the main effects model for general externalizing symptoms, specific alcohol use, and specific aggression predicting task-general executive functioning (v_{SST}). Models 2-5 display the step-wise process of adding the social adaptation variables in order to examine univariate effects. Standardized coefficients are presented, with the exception of the drift diffusion covariates, which were included concurrently with the dependent variables. Racial/ethnic status was coded such that 0=People of color and 1= Caucasian/non−Hispanic White. EF = Executive functioning. Bolded values = $p < .001$, ** = $p < .01$, * = $p < .05$.

(v_{SST}) across adolescence and emerging adulthood was supported ($\beta = 0.43$, $p < .001$; see Table 4). Regarding covariates, parental education ($\beta = 0.09$, $p < .001$) was related to task-general executive functioning (v_{SST}), such that adolescents whose parents reported high levels of education had higher levels of task-general executive functioning (v_{SST}). Nondecision time (Ter_{SST}) was positively associated with task-general executive functioning (v_{SST}), while response conservativeness (a_{SST}) was negatively related to task-general executive functioning (v_{SST}). General externalizing symptoms, specific alcohol use, and specific aggression did not significantly predict task-general executive functioning (v_{SST}) across time[4].

Main effects of social adaptation predicting task-general executive functioning

Next, we added social adaptation (i.e., parental monitoring, peer connectedness, and school engagement) to the main effects MLM to test prospective main effects on task-general executive functioning across adolescence and emerging adulthood, controlling for prior task-general executive functioning and demographic variables (e.g., sex, racial/ethnic status, and parental education; Path B; see Figure 2). These variables (i.e., parental monitoring, peer connectedness, and school engagement) were first tested in separate models and then tested simultaneously in a combined model. This allowed examination of unadjusted and adjusted associations. The results of main effects models for parental monitoring, peer connectedness, and school engagement predicting task-general executive functioning are reported in Table 4 (see Models 2-5).

When estimated in separate models, peer connectedness ($\beta = 0.07$, $p < .01$), and school engagement ($\beta = 0.14$, $p < .001$) prospectively predicted task-general executive functioning (v_{SST}), such that high levels of peer connectedness and school engagement were related to better task-general executive functioning (v_{SST}) across adolescence and emerging adulthood, controlling for previous levels of task-general executive functioning (v_{SST}) and covariates. The prospective association between parental monitoring and task-general executive functioning (v_{SST}) was nonsignificant.[5]

Next, an MLM with all social adaptation variables predicting task-general executive functioning (v_{SST}) was estimated to examine univariate effects (see Table 4; Model 5). In this model, peer connectedness ($\beta = 0.10$, $p < .01$) and school engagement ($\beta = 0.14$, $p < .001$) still significantly predicted task-general executive functioning (v_{SST}), suggesting that these social adaptation variables had unique effects on task-general executive functioning.

Main effects of general externalizing symptoms, specific alcohol use, and aggression predicting social adaptation

Main effects MLMs were run to test prospective main effects of general externalizing symptoms, specific alcohol use, and aggression predicting social adaptation (i.e., parental monitoring, peer connectedness, and school engagement) across adolescence and emerging adulthood, controlling for prior social adaptation, and covariates (e.g., sex, racial/ethnic status, and parental education; Path A; see Figure 2). The results of main effects models for general externalizing symptoms, specific alcohol use, and aggression predicting social adaptation are reported in Table 5.

Parental monitoring

Stability of parental monitoring across adolescence was supported ($\beta = 0.24$, $p < .001$). Sex, racial/ethnic status, and parental education were not related to parental monitoring. Regarding hypothesized pathways, general externalizing symptoms prospectively predicted low levels of parental monitoring across adolescence ($\beta = -0.14$, $p < .001$). Specific alcohol use and specific aggression did not predict changes in parental monitoring.

Peer connectedness

Stability of peer connectedness across adolescence and early adulthood was supported ($\beta = 0.35$, $p < .001$). Regarding covariates, sex, racial/ethnic status, and parental education were not related to peer connectedness. With respect to hypothesized pathways, high levels of general externalizing symptoms ($\beta = -0.05$, $p < .01$) and specific aggression ($\beta = -0.05$, $p < .01$) prospectively predicted low levels of peer connectedness across adolescence and early adulthood. Additionally, domain specific alcohol use predicted changes in peer connectedness across time in the unexpected direction, such that high levels of specific alcohol use predicted high peer connectedness ($\beta = 0.04$, $p < .05$).

School engagement

Stability of school engagement across adolescence was supported ($\beta = 0.58$, $p < .001$). Regarding covariates, sex ($\beta = 0.16$, $p < .01$) and parental education ($\beta = 0.26$, $p < .001$) were significantly related to school engagement, such that girls and adolescents whose parents reported high levels of education had high levels of school engagement. With respect to hypothesized pathways, high levels of general externalizing symptoms prospectively predicted low levels of school engagement across adolescence ($\beta = -0.21$, $p < .01$). Specific alcohol use and specific aggression were not prospectively related to school engagement.

Indirect effects

A conceptual figure depicting significant indirect effects is provided in Figure 4.

Parental monitoring

Given that parental monitoring did not prospectively predict task-general executive functioning, there were no plausible indirect effects involving parental monitoring.

Peer connectedness

Given the pattern of main effects reported above, a plausible indirect pathway was from general externalizing symptoms to

properties of drift rate, as they are in the expected direction; however, they are generally moderate in size. These moderate correlations provide further evidence of criterion validity by demonstrating that drift rate is related to traditional measures of SST behavioral performance, but it is also a distinct construct.

[4]With regard to developmental timing, we created a dummy variable that was coded such that 0 = early adolescence (ages = 10–13) and 1 = middle (ages = 14–17) and late (ages = 18–21) adolescence. This dummy coded age variable was used to create interaction terms with independent variables that were hypothesized to predict changes in task-general executive functioning development across time. Interaction terms were added to full main effects models reported above. These interaction terms allowed me to examine a whether early adolescence represents a sensitive period of neurobiological development during which alcohol use may be especially harmful to cognitive development. Interaction terms for early adolescence and specific alcohol use did not significantly predict task-general executive functioning (v_{SST}; $\beta = -1.21$, $p = .33$).

[5]Given my hypothesis that either very high or very low levels of parental monitoring could precipitate poor executive functioning, I also examined curvilinear associations. The quadratic term of parental monitoring predicting task-general executive functioning (v_{SST}) was nonsignificant ($\beta = -0.02$, $p = .28$).

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Table 5. Multilevel modeling results for general externalizing symptoms, specific alcohol use, and specific aggression predicting social adaptation

	Parental monitoring			Peer connectedness			School engagement		
Variable	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value
Intercept	4.06	0.03	**130.45**	4.46	0.02	**267.53**	4.67	0.05	**97.13**
Sex	0.001	0.03	0.03	− 0.006	0.02	− 0.38	0.16	0.05	3.22**
Racial/ethnic status	− 0.05	0.03	− 1.48	0.02	0.02	1.04	0.02	0.06	0.29
Parental education	0.07	0.03	1.87	− 0.01	0.02	− 0.67	0.26	0.05	**5.22**
General externalizing symptoms	− 0.14	0.04	− 3.47**	− 0.05	0.02	− 3.22**	− 0.21	0.06	− 3.33**
Specific alcohol use	− 0.05	0.08	− 0.61	0.04	0.02	2.31*	0.14	0.08	1.81
Specific aggression	− 0.03	0.03	− 1.09	− 0.05	0.02	− 2.80**	0.005	0.05	0.11
Parental monitoring stability	0.24	0.03	**7.69**						
Peer connectedness stability				0.35	0.02	**20.77**			
School engagement stability							0.59	0.05	**11.54**

Note: Sex was coded such that 0=male, 1=female. Racial/ethnic status was coded such that 0=People of color and 1= Caucasian/non-Hispanic White. Standardized coefficients are presented. Bolded values = $p < .001$, ** = $p < .01$, * = $p < .05$.

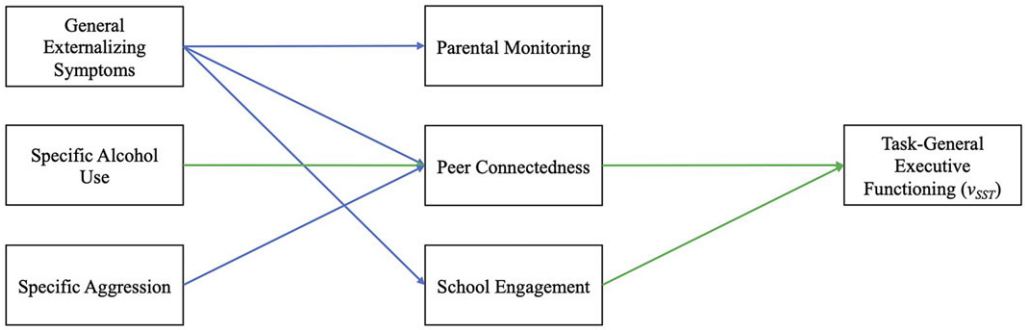

Figure 4. Conceptual model depicting significant indirect effects between general externalizing symptoms, specific alcohol use, specific aggression, social adaptation, and task-general executive functioning. *Note*. Significant pathways are presented here. Blue paths indicate negative direction of effects, while green paths indicate positive direction of effects. Multilevel models predicting each social adaptation variables (e.g., parental monitoring, peer connectedness, and school engagement) were run separately, but significant results are depicted together here in a conceptual figure. Social adaptation only significantly predicted changes in task-general executive functioning as measured by drift rate on the stop signal task. v_{SST} = drift rate on the stop signal task.

task-general executive functioning (v_{SST}) through peer connectedness. There were significant, prospective indirect effects from externalizing symptoms to task-general executive functioning (v_{SST}) through peer connectedness (95% CI[−0.076, −0.007]). High levels of general externalizing symptoms predicted poor peer connectedness, which in turn, predicted low levels of task-general executive functioning (v_{SST}).

Further, the pattern of main effects suggested an indirect pathway from specific aggression to task-general executive functioning (v_{SST}) through peer connectedness. There were significant, prospective indirect effects from specific aggression to task-general executive functioning (v_{SST}) through peer connectedness (95% CI[−0.143, −0.010]). In other words, high levels of specific aggression predicted poor peer connectedness, which was subsequently related to low levels of task-general executive functioning (v_{SST}).

Additionally, results from multilevel models suggested an indirect pathway from specific alcohol use to task-general executive functioning (v_{SST}) through peer connectedness. There were significant, prospective indirect effects from specific alcohol use to task-general executive functioning (v_{SST}) through peer connectedness (95% CI[0.001, 0.031]). The direction of this indirect effect was somewhat surprising; high levels of specific alcohol use predicted better peer connectedness, which subsequently predicted high levels of task-general executive functioning (v_{SST}).

School engagement

Given the pattern of findings above, another plausible indirect pathway was from general externalizing symptoms to task-general executive functioning (v_{SST}) through school engagement. There were significant, prospective indirect effects from externalizing symptoms to task-general executive functioning (v_{SST}) through school engagement (95% CI[−0.277, −0.040]). Results suggest that high levels of externalizing symptoms predicted poor school engagement, which was subsequently related to low levels of task-general executive functioning (v_{SST}).

Reciprocal relationships

Main effects MLMs were run to test prospective main effects of task-general executive functioning and social adaption on general externalizing symptoms, specific alcohol use, and specific aggression across adolescence and emerging adulthood,

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Table 6. Multilevel modeling results for task-general executive functioning and social adaptation predicting general externalizing symptoms, specific alcohol use, specific drug use, and specific aggression

	General externalizing symptoms			Specific alcohol use			Specific aggression			Specific drug use		
Variable	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value	β	S.E.	t value
Intercept	− 0.009	0.008	− 1.13	− 0.05	0.02	− 2.39*	0.005	0.005	1.07	− 0.02	0.04	− 0.50
Sex	0.008	0.007	1.19	− 0.02	0.02	− 1.12	0.01	0.004	2.84**	− 0.003	0.003	− 0.97
Racial/ethnic status	0.01	0.007	1.97*	0.07	0.02	**3.51**	− 0.004	0.004	− 1.14	0.01	0.003	3.20**
Parental Education	− 0.01	0.008	− 1.32	− 0.002	0.02	− 0.13	− 0.007	0.005	− 1.60	− 0.004	0.003	− 1.29
Computer version	0.02	0.02	0.93	0.02	0.05	0.51	0.006	0.01	0.55	0.02	0.04	0.42
Task−general executive functioning (v_{SST})	− 0.01	0.008	− 1.63	− 0.003	0.02	− 0.17	0.003	0.005	0.58	− 0.006	0.003	− 1.82
Nondecision time (Ter_{SST})	0.009	0.007	1.23	0.03	0.02	1.49	− .0001	0.005	− 0.03	− 0.002	0.003	− 0.72
Response conservativeness (a_{SST})	0.001	0.007	0.13	0.03	0.02	1.78	− 0.002	0.005	− 0.49	0.003	0.003	0.96
Response bias (z_{SST})	− 0.01	0.007	− 1.70	0.02	0.02	0.89	0.001	0.004	0.27	0.003	0.003	0.10
Parental monitoring	− 0.006	0.007	− 0.85	− 0.004	0.02	− 0.21	− 0.005	0.004	− 1.18	0.001	0.003	0.42
Peer connectedness	− 0.002	0.007	− 0.26	0.05	0.02	2.95**	− 0.003	0.004	− 0.60	0.001	0.003	0.38
School engagement	− 0.009	0.008	− 1.10	0.02	0.02	0.80	− 0.009	0.005	− 1.97*	− 0.005	0.003	− 1.63
General externalizing symptoms stability	0.14	0.008	**16.60**									
Specific alcohol use stability				0.13	0.02	**6.76**						
Specific aggression stability							0.08	0.004	**17.53**			
Specific drug use stability										0.05	0.003	**17.99**

Note: Sex was coded such that 0=male, 1=female. Racial/ethnic status was coded such that 0=People of color and 1= Caucasian/non-Hispanic White. Standardized coefficients are presented. Because results from bifactor modeling did not support a specific factor of drug use at wave 1, we estimated a separate multilevel model with data arranged to test cross-lagged associations predicting specific drug use (task-general executive functioning and social adaptation at wave 2 predicts specific drug use at wave 3, controlling for wave 2 specific drug use, and this structure was repeated through wave 4, allowing for the test of two cross-lags). Bolded values = $p < .001$, ** = $p < .01$, * = $p < .05$.

controlling for prior levels of the dependent variable and demographic variables (e.g., sex, racial/ethnic status, and parental education). The results of models testing reciprocal relationships are reported in Table 6.

General externalizing symptoms

General externalizing symptoms were stable across adolescence and emerging adulthood ($\beta = 0.14$, $p < .001$). Racial/ethnic status ($\beta = 0.01$, $p < .05$) was related to general externalizing symptoms, such that non-Hispanic White adolescents had higher levels of externalizing symptoms, but this was a very small effect. Task-general executive functioning (v_{SST}) did not predict externalizing symptoms across time. Social adaption (parent monitoring, peer connectedness, and school engagement) was unrelated to externalizing symptoms.

Specific alcohol use

Specific alcohol use was stable across time ($\beta = 0.13$, $p < .001$). Racial/ethnic status ($\beta = 0.07$, $p < .001$) was related to specific alcohol use, such that non-Hispanic White adolescents endorsed higher levels of specific alcohol use. Task-general executive functioning (v_{SST}) did not prospectively predict specific alcohol use. With respect to social adaptation, peer connectedness prospectively predicted specific alcohol use across adolescence and emerging adulthood ($\beta = 0.05$, $p < .01$). Here again, this effect was in an unexpected direction, such that high peer connectedness was related to high levels of specific alcohol use.

Specific aggression

Specific aggression was stable across adolescence and emerging adulthood ($\beta = 0.08$, $p < .001$). Regarding covariates, sex ($\beta = 0.01$, $p < .01$) was related to specific aggression, such that females exhibited higher levels of specific aggression. Task-general executive functioning (v_{SST}) did not prospectively predict changes in specific aggression across time. High levels of school engagement predicted low levels of specific aggression ($\beta = -0.009$, $p < .05$).

Specific drug use

Because results from bifactor modeling did not support a specific factor of drug use at W1, we estimated a separate MLM with data arranged to test cross-lagged associations predicting specific drug use (v_{SST} and social adaptation at W2 predicts specific drug use at W3, controlling for W2 specific drug use), and this structure was repeated through W4, allowing for testing of two cross-lags. Specific drug use was stable across adolescence and emerging adulthood ($\beta = 0.05$, $p < .001$). Regarding covariates, racial/ethnic status ($\beta = 0.01$, $p < .001$) was related to specific drug use, such that non-Hispanic White adolescents endorsed higher levels of specific drug use. Task-general executive functioning (v_{SST}) did not predict specific drug use. Social adaptation across parents, peers, and school did not prospectively predict specific drug use.

Discussion

The existing literature has yielded inconsistent findings regarding whether the effects of alcohol use during adolescence, a sensitive

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period of brain development, are neurotoxic and derail healthy cognitive development (Lees et al., 2019). Notably, previous studies have failed to consider that adolescent alcohol use occurs within a broader developmental context of externalizing symptoms (including other drug use) and poor social development. Additionally, fractionated approaches and heterogeneity in the measurement of executive functioning (Nigg, 2017) may contribute to mixed findings. This study aimed to provide clarity to the alcohol use-executive functioning association by (1) distinguishing general externalizing symptoms from domain specific symptoms (alcohol use, aggression, rule-breaking, drug use) using bifactor modeling, (2) examining prospective associations between general externalizing symptoms and domain specific symptoms, and task-general executive functioning across adolescence and early adulthood, (3) using computational modeling of performance on the SST to provide a more psychometrically sound index of task-general executive functioning, and (4) testing indirect pathways from externalizing symptoms to deficits in task-general executive functioning through poor social adaptation across parents, peers, and school. A large high-risk longitudinal sample enriched for children of parents with an AUD (the Michigan Longitudinal Study; Zucker et al., 1996) was used to test study aims.

Aim 1: hierarchical structure of externalizing symptoms

In line with calls to examine developmental changes in the structure of psychopathology (Forbes et al., 2016), the current study estimated longitudinal bifactor models of externalizing symptoms across early adolescence (age 10) to early adulthood (age 25). Our first hypothesis that a latent factor characterized by general externalizing symptoms and domain specific factors for alcohol use, drug use, aggressive behaviors, and rule-breaking behaviors would emerge at each assessment was largely supported. Results from bifactor modeling supported a hierarchical structure of externalizing psychopathology at four assessments spanning early adolescence to early adulthood; a general externalizing factor and specific factors emerged at each assessment. This is consistent with a wide body of research on the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017), and supports the notion that substance use is part of a larger constellation of problem behavior (Krueger et al., 2021).

Specific factors of alcohol use and aggression emerged at each wave. However, specific factors of drug use did not emerge until Wave 2 (ages 14–17), due to very low endorsement of drug use at Wave 1 when the sample was 10–13-years-old. This finding is novel as no other studies have considered such a bifactor model starting in early adolescence, and results support Forbes et al. (2016) contention that exposure to environmental risks (e.g., drugs) guides the manifestation of increasingly specific factors of externalizing symptoms across development. It should also be noted that while the general externalizing and specific alcohol use factors represented the data well and were reliable, the specific factors of aggression and drug use were less well-represented and less reliable. This pattern replicates our prior work which found poorly specified specific factors of aggression and substance use compared to general factors of externalizing symptoms in an adolescent sample (Paige et al., 2021). Additionally, due to the poorly represented and less reliable specific factors, results concerning specific factors of aggression and drug use should be interpreted with caution.

Specific factors of rule-breaking behavior did not emerge. This is somewhat inconsistent with theory that behaviors like delinquency are distinguishable from general externalizing symptoms and other specific externalizing factors in adolescence (Forbes et al., 2016; Krueger & Tackett, 2014). However, in a separate sample, we similarly found no evidence specific factors of rule-breaking behaviors in a longutidinal bifactor model (Paige et al., 2021). Additionally, factor analytic work by Krueger et al. (2007) suggests that externalizing symptoms are characterized by a hierarchical structure that includes a general externalizing factor and two lower-order factors of aggression and substance use. Our results were consistent with this model, suggesting that the majority of rule-breaking variance is accounted for by factors of general externalizing symptoms, and there is likely little variance "leftover" to specify domain specific factors of rule-breaking behaviors (Krueger et al., 2007). In conclusion, findings from the current study support the notion that externalizing symptoms are best understood through a hierarchical structure, including a general factor characterized by rule-breaking, aggression, and substance use behaviors as well as domain specific factors of aggression, alcohol use, and drug use, which emerges as adolescents age.

Aim 2: direct effects of externalizing symptoms, specific alcohol and drug use, and aggression on the development of task-general executive functioning

Direct effects of general externalizing symptoms on task-general executive functioning

General externalizing symptoms were hypothesized to be related to poor task-general executive functioning as a direct effect and this was not supported. This is somewhat inconsistent with prior studies that have reported prospective associations between problem behavior and questionnaire-assessed executive functioning (Atherton et al., 2019; Paige et al., 2021; Wiker et al., 2023). However, measurement of executive functioning in the current study differed from this prior work. While Fosco et al. (2019a) reported links between SSRT and subsequent delinquency across adolescence, no previous studies, to our knowledge, have examined the consequential effects of problem behavior on behavioral performance on the SST.

It should be noted that empirical evidence linking externalizing symptoms to executive functioning during adolescence and early adulthood is lacking. This is especially surprising, given the developmentally normative increase in deviant behavior (Moffitt, 1993) as well as critical changes in neurodevelopment (Blakemore & Choudhury, 2006) that occur during this developmental period. It may be that the small number of published studies in this area is due to nonsignificant associations between externalizing symptoms and executive functioning across adolescence (e.g., the "file drawer problem"; Rosenthal, 1979). While nonsignificant direct effects from the current study support this notion, importantly, findings suggest that the broader problem behavior context still confers risk for poor executive functioning (v_{SST}) development, and indirect pathways through social adaption are discussed in detail under Aim 3.

Neurotoxic effects of alcohol use on task-general executive functioning

Our third hypothesis that the alcohol use specific factors would be related to poor task-general executive functioning was not supported. Additionally, with respect to developmental timing, there was no evidence that alcohol use during the sensitive period of early adolescence (ages 10–13) had deleterious effects on the

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development of executive functioning. As noted earlier, the literature testing neurotoxicity effects of alcohol and executive function is limited and mixed. The current study accounted for the broader context of problem behavior and poor social development in which adolescent alcohol use often occurs, and also used a reliable measure of task-general executive functioning (drift rate), which may enhance construct validity in measurement of individual differences that underlie performance on executive functioning tasks (Löffler et al., 2024; Weigard et al., 2021; Weigard & Sripada, 2021).

Indeed, as variance in specific alcohol use was separated from variance accounted for by general externalizing symptoms, specific drug use, and specific aggression, after accounting for these behaviors, remaining specific alcohol use variance may be an indicator of normative developmental trajectories. Alcohol use is normative in American culture (Johnston et al., 2018), and some amount of use may be an indicator of healthy psychosocial adjustment in certain contexts (Peele & Brodsky, 2000). Notably, non-Hispanic White adolescents had higher scores on the specific alcohol use factors. It is possible that these specific factors represented less harmful alcohol use that may be unique to non-Hispanic White Americans. Indeed, there is substantial empirical evidence that compared to non-Hispanic White Americans, Black Americans experience higher levels of alcohol problems despite later initiation of drinking and lower levels of use across almost all age groups, likely due to a complex interaction of factors related to discrimination, racism, and within-group social disapproval (Zapolski et al., 2014). Further, there were positive associations between specific alcohol use and peer connectedness, discussed in more detail below. Notably, recent studies that have accounted for the broader context of problem behavior have reported that adolescent and emerging adult alcohol use is not correlated with executive functioning (Meehan et al., 2013; Paige et al., 2021; Piehler et al., 2012). It should be stated that evidence does *not* suggest that adolescent drinking directly promotes healthy adjustment, but rather, emphasizes the need to consider that adolescent alcohol use occurs in a broader context of problem behavior. In summary, it may be that the relationship between early alcohol use and poor executive functioning operates within a larger constellation of externalizing symptoms and socio-environmental experiences.

Neurotoxic effects of drug use on task-general executive functioning

As expected, there was lower variability in drug use in early adolescence compared to alcohol use (Swendsen et al., 2012), which presented some challenges in examining the unique effects of drug use on the development of task-general executive functioning. Indeed, the data did not support a specific factor of drug use in early adolescence. This limited examination of prospective effects of specific drug use on executive functioning to two cross-lags (middle to late adolescence, and late adolescence to early adulthood). Our hypothesis that the drug use specific factors would be related to poor task-general executive functioning was not supported. However, it should be noted that drug use specific factors were not well-defined by the indicators and had poor reliability, and so some caution is warranted in the conclusion that that early drug use does not have deleterious effects on long-term executive functioning. Notably, past work has largely failed to account for the broader context of problem behavior in which drug use occurs (Squeglia et al., 2009; Wetherill et al., 2013), and it is possible that previously observed direct links between drug use and poor executive functioning are actually due to co-occurring externalizing symptoms and poor social development.

Unique effects of aggression and rule-breaking behaviors on task-general executive functioning

There was no evidence that the specific factors of aggression and rule-breaking behaviors were related to low levels of task-general executive functioning. Bifactor modeling did not support estimation of specific factors of rule-breaking behavior, and this precluded examination of unique effects of rule-breaking behaviors on executive functioning. With respect to aggression, these specific factors were not well-defined by the indicators and had poor reliability. Further, results suggested that this domain specific factor likely did not represent physical aggression; as girls endorsed higher levels of specific aggression, and physical aggression has been reliably established to be more prevalent among males across development (Card et al., 2008). Some items on the YSR/ASR aggression subscale measure emotional lability (e.g. "My mood or feelings change suddenly"), while others measure forms of aggression that may not be as clearly violent (e.g., "I argue a lot"). After accounting for general externalizing variance, unique variance for the aggression specific factors may have been representative of emotional lability or hostility. Indeed, adolescent females are more likely to endorse emotional lability (Stringaris & Goodman, 2009). Additionally, results are consistent with our past work found no prospective associations between factors of specific aggression, which likely represented emotional lability or hostility, and effortful control across five assessments spanning ages 11–20 (Paige et al., 2021). No prior studies, to our knowledge, have examined the prospective effects of emotional lability on cognitive development as measured by behavioral tasks. This may be a useful direction for future research.

Aim 3: indirect pathways from general externalizing symptoms to task-general executive functioning through social adaptation

Indirect effects through parental monitoring

Our hypothesis that there would be indirect effects between externalizing symptoms and deficits in task-general executive functioning through parental monitoring was not supported. Indeed, parental monitoring was not related to task-general executive functioning (ν_{SST}). This is inconsistent with recent longitudinal work by Hong et al. (2024) which demonstrated that lower parental acceptance and higher rejection was associated with weaker improvements in adolescent executive functioning. Notably, no previous studies, to our knowledge, have examined the consequences of parental monitoring, specifically, on adolescent executive functioning. The current study suggests that that examining the impact of parenting practices related to parent-adolescent relationship quality, specifically, may be a more fruitful future direction.

Notably, general externalizing symptoms predicted changes in parental monitoring, such that high levels of externalizing symptoms predicted low levels of parental monitoring across adolescence. This is an important finding because research in this area has yielded mixed results, with some early childhood studies demonstrating that externalizing symptoms elicit parental over-control (Eisenberg et al., 2015). Conversely, cross-sectional associations in an adolescent sample suggest that externalizing symptoms elicit low parental monitoring, above and beyond prior levels of parental monitoring (Fite et al., 2006). This divergent pattern of associations between externalizing symptoms and

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parental monitoring emphasizes the importance of developmental timing when considering these trajectories. Indeed, Patterson's (2002) coercion model suggests that when faced with problem behavior, parents often initially respond by becoming increasingly involved in attempts to control their child's behavior. However, over time, parents who are repeatedly faced with problem behavior and lack the skills to effectively intervene likely feel increasingly ineffective and become alienated from parenting (Patterson, 2002). By the time that children who exhibit high levels of problem behavior have reached adolescence, parents have likely reached this point in the coercion cycle, leading them to decrease their monitoring in response to elevated externalizing symptoms across time. Findings from the current study advance our understanding by demonstrating prospective effects between offspring externalizing symptoms and decreases in parental monitoring across early to late adolescence, supporting the notion that offspring problem behavior leads to parental disengagement across long periods of time.

Indirect effects through peer connectedness

Our hypothesis that there would be indirect effects between externalizing symptoms and deficits in task-general executive functioning through peer connectedness, such that high levels of externalizing symptoms would be related to low levels of peer connectedness, which in turn, would be related to low levels of task-general executive functioning was supported. It has been well-documented that externalizing symptoms elicit poor relationships with peers (Baumeister et al., 2005), and our findings are consistent with this literature. The significant, prospective association between poor peer connectedness and decreases in task-general executive functioning (ν_{SST}) across adolescence and early adulthood is novel and important. Indeed, findings from the current study support the notion that adolescents who are rejected by prosocial peers may have fewer opportunities for the practice of social competence skills that involve executive functioning (Calkins, 1994; Fox & Calkins, 2003), and receive little feedback and guidance for their behavior in the peer context, which likely derails healthy cognitive development (ν_{SST}; Farley & Kim-Spoon, 2014; Leflot et al., 2011).

Additionally, while not specifically hypothesized, there was evidence to support indirect effects between specific aggression and poor task-general executive functioning (ν_{SST}) through peer connectedness. The prospective effect between specific aggression, which likely represents emotional lability, and poor peer connectedness is consistent with the reliably documented effects of aggression-related emotional lability on social maladjustment (Kamper & Ostrov, 2013; Murray-Close, 2013; Ostrov, 2008). The current study extends past work in this area, which has largely been concentrated in childhood, by demonstrating that emotional lability indirectly predicts task-general executive functioning through poor peer connectedness across four assessments that span ages 10 to 25. However, results bear replication due to poor reliability and low H values on the aggression specific factors. Future work in this area may aim to utilize scaled scores of emotional lability or hositility when investigating indirect effects from specific aggression to poor executive functioning through social maladaptation.

It should also be noted that specific alcohol use impacted the development of task-general executive functioning (ν_{SST}) indirectly through peer connectedness; however, this indirect effect was in the opposite direction of what was expected. Indeed, high levels of specific alcohol use predicted better relationships with peers, which in turn, was subsequently related to high levels of task-general executive functioning (ν_{SST}). Importantly, there is *no* evidence that adolescent alcohol use has positive, direct, neurobiological effects on cognitive development. Rather, our interpretation of the alcohol use specific factor is that when variance related to drinking is separated from the broader context of problem behavior, this specific variance is likely a marker of positive social adjustment in some contexts (Peele & Brodsky, 2000). In other words, findings from the current study do *not* suggest that adolescent drinking is protective. Rather, results suggest that when using bifactor modeling to parse externalizing variance, variance related to drinking that is separate from variance related to rule-breaking, aggression, and other drug use is indicative of something positive, such as sociability.

Indirect effects through school engagement

Our hypothesis that there would be an indirect pathway between externalizing symptoms and deficits in task-general executive functioning through school engagement, such that high levels of externalizing symptoms would be related to poor school engagement, which in turn, would be related to low levels of task-general executive functioning was supported. Results corroborate substantial evidence that externalizing symptoms elicit poor school engagement throughout childhood (Stipek & Miles, 2008) and adolescence (Masten et al., 2005). Moreover, our findings forward the idea that school engagement is critical for supporting cognitive development (Berry, 2012), and provide novel empirical evidence that school engagement plays a central role in strengthening task-general executive functioning (ν_{SST}) across ages 10–25. School provides a rich context for adolescents to observe modeled executive functioning skills, as well as practice behaviors related to healthy cognitive skills and receive direct and indirect feedback on these behaviors.

Reciprocal relationships

While not a primary aim of the current study, examination of reciprocal relationships was illuminating. Notably, drift rate did not predict externalizing symptoms across time, diverging from a large literature on the protective effects of healthy executive functioning in guarding against problem behavior (Alessandri et al., 2022; Fosco et al., 2019a; Hentges et al., 2020; Wiker et al., 2023). This study was the first, to our knowledge, to apply drift diffusion modeling to the SST across ages 10–25. Notably, the lack of significant effects in task-general executive functioning (ν_{SST}) predicting general externalizing symptoms increases confidence in the notion that the direction of this within-person association flows from externalizing symptoms to poor task-general executive functioning (ν_{SST}). This is consistent with a recent longitudinal study of adolescents that reported prospective associations between externalizing symptoms and poor executive functioning, but found no support for bidirectional associations (Brieant et al., 2022). In contrast, analyses in the ABCD consistently find prospective relations between executive functioning and externalizing psychopathology, potentially due to the statistical power of this larger sample (Romer & Pizzagalli, 2021; Wiker et al., 2023). It is also relevant that, under ideal measurement conditions (e.g., multiple tasks), drift rate appears to be a relatively stable trait (Schubert et al., 2016; Weigard et al., 2021). If the rank ordering of individuals' drift rate stays relatively stable across development, between-subjects associations of drift rate with psychopathology may reflect directional relations between these constructs at the level of individuals' time-invariant traits, while within-subjects

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relations may be more subtle and difficult to detect. More work utilizing drift diffusion modeling to index task-general executive functioning when examining prospective associations between reaction time-based measures of executive functioning and problem behavior across adolescence is warranted.

Relatedly, drift rate did not significantly predict changes in specific factors of alcohol use, drug use, and aggression. This is somewhat inconsistent with empirical evidence that healthy cognitive development reduces levels of substance use and aggression across adolescence and early adulthood (Nigg et al., 2006; Piehler et al., 2012). However, our interpretation of the alcohol use specific factor suggests that once variance unique to drinking is separated from problem behavior, it is likely an indicator of sociability. Indeed, previous studies that account for the broader context of problem behavior report nonsignificant associations between effortful control and low levels of adolescent alcohol use (Paige et al., 2021; Piehler et al., 2012). Moreover, nonsignificant associations are consistent with our past work estimating bifactor models of externalizing symptoms and examining bidirectional associations with effortful control across adolescence, which suggests that healthy cognitive development does not prospectively predict specific factors of substance use nor aggression (Paige et al., 2021).

Additionally, parental monitoring, peer connectedness, and school engagement did not predict changes in general externalizing symptoms across early adolescence to early adulthood. This diverges from past research that has reported prospective associations between healthy social adjustment and decreases in adolescent problem behavior (Jacobson & Crockett, 2000; Prinstein & La Greca, 2004). Here again, the lack of significant effects in the model predicting general externalizing symptoms increases confidence in the notion that the direction of this association flows from externalizing symptoms to poor social adjustment. This supports findings by Burke et al. (2008) who investigated bidirectional relationships between parenting and child behaviors, and concluded that evidence for child problem behavior influencing parenting behaviors was stronger than evidence for parenting behaviors predicting child behaviors.

Finally, prospective associations between healthy social adaptation and specific alcohol use and aggression was illuminating. Indeed, adolescents who reported high levels of school engagement prospectively decreased in their levels of specific aggression across time, corroborating past work in this area (Loukas et al., 2010). Additionally, strong connections to peers predicted high levels of specific alcohol use, lending further support to our interpretation of this factor as healthy and normal trajectories of adolescent drinking. Indeed, a 2023 systematic review and meta-analysis of peer connectedness and substance use in adolescence reported that alcohol use was more likely than other substances to be associated with peer connectedness (Cole et al., 2023). Contrary to expectation, social adaptation did not predict changes in specific drug use. Analyses testing prospective effects on specific drug use were limited to only two cross-lags, and the stability of specific drug use across time was high.

Limitations

Findings from the current study should be interpreted within the context of certain limitations. First, a large proportion of the sample was male and non-Hispanic White; thus, results may not generalize to samples that are more diverse with respect to sex and race. Additionally, the MLS is a high-risk sample containing a high proportion of children of parents with an AUD. This allowed us to test study hypotheses in a sample of adolescents at risk for high levels of alcohol use, drug use, externalizing problems, and task-general executive functioning deficits; on the other hand, results may not generalize to community samples or typically developing adolescents.

Second, adolescent provided self-report on externalizing symptoms and alcohol and drug use, and also peer connectedness. Reliance on a single reporter may have inflated assocaitions with peer connectedness and potentially deflated relations with other variables from different measurement domains. Still, adolescents' own perceptions of their ability to make and maintain friendships are likely important in understanding effects of social adjustment on executive functioning development. Future work in this area may elect to use multiple informants when measuring adolescents' problem behavior, substance use, and social adjustment and examining their impact on executive functioning development.

Relatedly, third, our measure of parental monitoring was limited in several ways. Namely, parental monitoring may not be the ideal measure to utilize when considering the ways in which parents support adolescent cognitive development. Indeed, recent work suggests that measures which more closely assess aspects of parent-adolescent relationship quality (i.e., acceptance and rejection) may shape trajectories of adolescent executive functioning (Hong et al., 2024). Additionally, our measure of parental monitoring demonstrated fair reliability estimates at several waves, raising concerns about its psychometric properties. On the other hand, general externalizing symptoms predicted changes in parental monitoring in the expected direction, supporting predictive validity of the measure. Still, future work in this area should aim to utilize measures which more precisely and reliably assess parent-adolescent relationship quality when exploring the role that parents play in supporting the development of adolescent executive functioning.

Conclusion and future directions

Findings from the current study demonstrate that the problem behavior context indirectly predicts poor efficiency of goal-relevant evidence accumulation, a critical ability that underlies individual differences across executive functioning tasks, through social maladaptation. This study was the first, to our knowledge, to provide strong empirical evidence for the role of social adaptation in supporting task-general executive functioning development throughout adolescence and early adulthood. These findings call for research on adolescence to continue to examine the role of social relationships in supporting healthy executive functioning, and support the importance of interventions that target increasing social skills and are based in schools when treating problem behavior (Leff et al., 2010). Moreover, our results emphasize the need for research in this area to adopt a developmental psychopathology perspective. Findings did not support the notion that there are unique, neurotoxic effects of adolescent alcohol and drug use. It is critical for the field to account for the hierarchical structure of psychopathology, namely the broader externalizing context, when considering the consequences of adolescent alcohol use.

Finally, findings from the current study demonstrate that the application of the DDM model to behavioral tasks of executive functioning may be a fruitful approach in enhancing reliability and construct validity in the measurement of efficiency of evidence accumulation, a construct which is thought to underlie executive

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functioning (Löffler et al., 2024; Weigard et al., 2021; Weigard & Sripada, 2021). Indeed, drift rate (v_{SST}) demonstrated strong psychometric properties in the form of temporal stability that is fair by conventional standards (ICC = 0.52), but relatively robust given the average measurement interval of 2–3 years, and predictive validity in its positive, significant associations with peer relationship quality and school engagement. On the other hand, the application of the DDM model is a relatively new venture within clinical psychology, and further work examining whether results differ when utilizing traditional reaction time-based approaches, as well as a broader battery of assessments, to measure executive functioning tasks is warranted.

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