Development and Psychopathology (2023), **35**, 2028–2043
doi:10.1017/S0954579422000670

CAMBRIDGE UNIVERSITY PRESS

Regular Article

Predictors of problematic adult alcohol, cannabis, and other substance use: A longitudinal study of two samples

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Abstract

This study examined whether a key set of adolescent and early adulthood risk factors predicts problematic alcohol, cannabis, and other substance use in established adulthood. Two independent samples from the Child Development Project (CDP; $n = 585$; 48% girls; 81% White, 17% Black, 2% other race/ethnicity) and Fast Track (FT; $n = 463$; 45% girls; 52% White, 43% Black, 5% other race/ethnicity) were recruited in childhood and followed through age 34 (CDP) or 32 (FT). Predictors of substance use were assessed in adolescence based on adolescent and parent reports and in early adulthood based on adult self-reports. Adults reported their own problematic substance use in established adulthood. In both samples, more risk factors from adolescence and early adulthood predicted problematic alcohol use in established adulthood (compared to problematic cannabis use and other substance use). Externalizing behaviors and prior substance use in early adulthood were consistent predictors of problematic alcohol and cannabis misuse in established adulthood across samples; other predictors were specific to the sample and type of substance misuse. Prevention efforts might benefit from tailoring to address risk factors for specific substances, but prioritizing prevention of externalizing behaviors holds promise for preventing both alcohol and cannabis misuse in established adulthood.

Keywords: alcohol; cannabis; development; risk factors; substance use

(Received 18 December 2021; revised 24 June 2022; accepted 25 June 2022; First Published online 12 August 2022)

Problematic alcohol, cannabis, and other substance (e.g., opioids) use are problems well worth preventing, yet society has been unsure of how to prevent problematic alcohol, cannabis, and other substance use, which have high financial and human costs (WHO, 2018, 2020). In the United States alone, each year substance misuse is estimated to cost $740 billion (Morales et al., 2020). These financial costs are incurred as a result of increased healthcare burden, criminal justice system involvement, and social welfare services needed, as well as lost productivity at work, for individuals who misuse alcohol, cannabis, and other substances, compared to those who do not. For alcohol and increasingly for cannabis, society has come to accept certain levels of "non-problematic" use while recognizing problematic use as worthy of prevention. Preventing misuse of legal substances that are used in ways other than prescribed (e.g., opioids) as well as use of illegal substances is also a public health priority, particularly in the context of increasing rates of overdose deaths. According to the latest estimates released by the Centers for Disease Control and Prevention (2020), 70,237 drug overdose deaths occurred in the United States in 2017 alone – an increase of 387% from 1999 to 2017. According to the World Health Organization (WHO, 2018), 5.1% of the global burden of disease and injury is attributable to alcohol, with additional high burdens associated with opioids and other substances (WHO, 2020). Thus, prevention and intervention efforts are important given the high costs of substance misuse.

Given the importance of prevention, individual empirical studies, systematic reviews, and meta-analyses all have identified a range of predictors of problematic alcohol, cannabis, and other substance use (see Morales et al., 2020). The purpose of the present study was to understand whether a key set of adolescent and early adulthood predictors uniquely predicted problematic alcohol, cannabis, and other substance use (including misuse of prescription drugs as well as illegal drugs) in established adulthood (ages 30–45; Mehta et al., 2020). Established adulthood has been largely overlooked in developmental research, but this period represents a distinct stage characterized by some of the most demanding years due to a "career-and-care crunch" (Mehta et al., 2020, p. 436). Substance use in emerging adulthood (approximately ages 18–25) has been much better characterized than substance use in established adulthood in the developmental literature (Arnett, 2005). Although substance use is high in early adulthood and may be part of identity exploration that is salient in emerging adulthood (Arnett, 2005), problematic substance use becomes more entrenched in established adulthood. For example, substance use disorders in the United States declined between 2015 and 2018

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Cite this article: Lansford, J. E., *et al.* (2023). Predictors of problematic adult alcohol, cannabis, and other substance use: A longitudinal study of two samples. *Development and Psychopathology* 35: 2028–2043, https://doi.org/10.1017/S0954579422000670

for adolescents ages 12–17 and emerging adults ages 18–25; however, during this same time period, substance use disorders remained stable or increased for adults ages 26 and older (Bouchery, 2021). In addition, established adulthood is a developmental period that surpasses all other age groups in rates of overdose death (Centers for Disease Control and Prevention, 2020).

Predictors of problematic alcohol, cannabis, and other substance use

There are many relevant predictors that we could have selected, assessed in different developmental eras. We chose to focus on predictors from adolescence and earlier in adulthood that have received both theoretical and empirical support in a number of previous studies as especially important factors in the development of problematic alcohol, cannabis, and other substance use. We drew on several theoretical frameworks, reviews, and meta-analyses in determining which predictors to include in our models. Each of these theories, reviews, and meta-analyses is reviewed briefly to illustrate how they guided the selection of predictors. We also acknowledge that many other factors that we did not analyze have been included in conceptual frameworks and empirical studies of substance misuse, so our list of predictors is not comprehensive.

Problem-Behavior Theory has a long history as a framework for understanding the development of substance misuse and other problem behaviors (Jessor, 1987). Within this framework, personality, environmental, and behavioral characteristics are considered as potential instigators of and controls on problem behavior. The framework encompasses both more distal predictors, such as family socioeconomic status (SES), as well as more proximal predictors, such as parent support and affiliation with deviant peers (Jessor, 1987). Problem-Behavior Theory accounts for ways in which different types of problem behaviors, including both substance misuse and antisocial behavior, might stem from similar risk factors. Indeed, several meta-analyses have identified prior externalizing behavior as a robust predictor of the development of later substance use problems. For example, a meta-analysis of 12 longitudinal studies found that externalizing behavior in childhood and adolescence increases the risk of alcohol use disorders in early adulthood by 62% (Meque, Dachew, et al., 2019). Prior externalizing behavior also has emerged in other meta-analyses and reviews as one of the most important predictors of the development of substance use problems (e.g., Doran et al., 2012; Morales et al., 2020).

Externalizing and internalizing behaviors often are comorbid with each other and with substance use disorders (Helle et al., 2019, 2020), and internalizing problems, such as depression and anxiety, increase the likelihood of alcohol, cannabis, and other substance disorders (e.g., Acuff et al., 2018; Lalic et al., 2018; Lee et al., 2018; Serre et al., 2018). An additional theoretical framework delineates an internalizing pathway that has been important in understanding the development of substance use problems (e.g., Hussong et al., 2017). According to this framework, behavioral inhibition in infancy and early childhood leads to internalizing problems that contribute to interpersonal skills deficits; adolescents then begin and escalate substance use as ways to cope with internalizing problems and interpersonal skills deficits (Hussong et al., 2011). This self-medication hypothesis has been identified in a narrative review to explain how individuals experiencing psychological distress try to ease their distress through using alcohol, cannabis, or other substances (Turner et al., 2018). This theoretical framework also has been extended to incorporate an externalizing branch of the internalizing pathway, which describes how internalizing problems can contribute to the development of externalizing problems, which sometimes lead to affiliations with deviant peers who endorse substance use (e.g., Eiden et al., 2016; Hussong et al., 2018; Rothenberg et al., 2020).

The Social Development Model (Catalano et al., 1996; Hawkins et al., 1986; Kosterman et al., 2014) has integrated a wide range of predictors into a theoretical framework for understanding substance abuse. These factors include parent substance use, family relationships (including parent involvement, attachment, parenting styles, and other aspects of parent-child relationships such as communication), family structure, early antisocial behavior, school factors (such as commitment to school and academic achievement), peer factors (such as peers' drug use), personality traits, and genetic factors (Hawkins et al., 1986). The Social Development Model and developmental psychopathology framework (Cicchetti & Rogosch, 2002) were the theoretical bases for a systematic review of six domains of predictors of alcohol use in early adulthood (Meque, Salom, et al., 2019). Individuals' own history of prior substance use and their family members' history of drinking, externalizing and internalizing behaviors during adolescence, school (e.g., school bonding), and peer (e.g., affiliation with antisocial friends) were identified as main categories of predictors of alcohol use disorder in early adulthood. This review of 22 longitudinal studies of predictors of alcohol use disorder concluded that there are especially robust links between externalizing behaviors in adolescence and alcohol use disorder in young adulthood (Meque, Salom, et al., 2019).

Using many of the same constructs included in the Social Development Model, a dynamic cascade model of the development of substance use onset in adolescence found that child factors, ecological factors, conduct problems, parenting, and peer relationships all contributed to the prediction of onset of substance use in adolescence (Dodge et al., 2009). For example, family stress had both direct and indirect effects on substance use onset, by increasing depression, anxiety, and problematic social relationships that are themselves predictors of increased substance use (Dodge et al., 2009). Family stress also has been found to predict early initiation of substance use in adolescence in other developmental cascade models (Otten et al., 2019), and stressful life events often are associated with an increase in substance use and disorders in adulthood (Keyes et al., 2011).

Parts of these overarching theoretical frameworks for understanding the development of substance use are also elaborated in theories regarding specific components of the broad models. For example, the theory of deviant peer contagion explains mechanisms, such as positive reinforcement, through which affiliation with peers who engage in problem behaviors contributes to the spread of these behaviors within peer groups and that ineffective parental monitoring exacerbates the risks of affiliation with deviant peers (Dishion et al., 1995; Piehler & Dishion, 2014). Empirical studies support this theoretical framework, as alcohol, cannabis, and other substance use are often initiated during adolescence in the presence of peers (Henneberger et al., 2019). Adolescents' perceptions of their best friend's alcohol use are strongly related to their own alcohol use (Schuler et al., 2019), and adolescents whose peers use cannabis are more likely to use cannabis themselves (Marmet et al., 2021). Peer approval also is associated with more adolescent misuse of opioids (Tucker et al., 2020). Longitudinal research also suggests that young adults' perceptions of peer substance use predict adults' own subsequent substance use (Lansford et al., 2021).

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Focusing on the parenting component of larger theoretical models, a meta-analysis of 131 studies identified parental monitoring, parent-child relationship quality, parental support, and parental involvement as longitudinal predictors of both alcohol initiation and levels of later alcohol use and misuse (Yap et al., 2017). Likewise, a meta-analysis of family-based prevention programs for adolescent substance use, found that parental involvement and parent-adolescent relationship quality are important aspects of family relationships that are related to adolescents' substance use (Van Ryzin et al., 2016). Parental monitoring encompasses a range of behaviors such as setting rules and limits (e.g., curfews) and soliciting information about adolescents' activities, friends, and whereabouts (Lionetti et al., 2019). More parental monitoring, better parent-adolescent relationship quality, and more parental involvement are generally related to less adolescent alcohol, cannabis, and other substance use (e.g., Scholes-Balog et al., 2020). Poor relationships with parents prior to the age of 18 also have been found to predict cannabis use disorder in early adulthood (Marmet et al., 2021), although a meta-analysis of longitudinal studies found that parenting predictors appear to wane in importance in adulthood (Meque, Salom, et al., 2019).

Additional predictors that have not traditionally been incorporated in theoretical frameworks regarding the development of substance use onset or problems may be particularly important in understanding specific types of substance use. In particular, pain management has been central to the opioid crisis (Papp et al., 2020). Individuals often first begin using opioids legally with a prescription from a physician to manage pain. For those who become addicted, opioid use continues beyond the need to manage physical pain, with opioids increasingly used more frequently or in higher doses than prescribed and eventually without a prescription (Lalic et al., 2018). Beyond pain, poor physical health more broadly has been related to alcohol, cannabis, and other substance misuse (Onyeka et al., 2019), although these associations are likely bidirectional, with substance use worsening physical health and poor physical health leading to an increase in substance use.

Theoretical models of the development of substance use and meta-analyses sometimes include demographic factors such as gender, race/ethnicity, and SES either as substantive predictors or control variables. Gender differences often are found in alcohol, cannabis, and other substance use, with men more likely to use and misuse these substances than women (e.g., Blanco et al., 2018; Boden et al., 2020; Haardörfer et al., 2021). Patterns of differences in substance use and misuse by race/ethnicity are more complex and less consistent (e.g., Haardörfer et al., 2021; Schiavon et al., 2018; Substance Abuse and Mental Health Services Administration, 2019). SES also has a complex association with alcohol, cannabis, and other substance use, with SES differences in use often varying as a function of the specific substance involved, geographic location, and developmental timing. For example, some studies have found that in the United States, adolescents from lower SES families use more alcohol and cannabis than adolescents from higher SES families (Andrabi et al., 2017), but other studies have found that youth from higher SES families use alcohol and cannabis more frequently than youth from lower SES families (Martin, 2019).

Beyond main effects, gender and race/ethnicity might also moderate associations between other risk factors and problematic alcohol, cannabis, and other substance use. For example, one study found that anxiety in adolescence is a predictor of alcohol and cannabis misuse in adulthood for women but not men (Buckner & Turner, 2009). A study using data from the nationally representative Monitoring the Future sample found that race/ethnicity moderated the strength of the relation between parental involvement and substance use for 8th grade girls (Pilgrim et al., 2006). However, previous research has been inconclusive regarding the possible moderating roles of gender and race/ethnicity with some studies finding evidence of moderation for particular race/ethnic groups, for particular risk factors, and for particular substance use outcomes (e.g., Kulis et al., 2012; Vaughan et al., 2018; Wiesner et al., 2005). For example, analyses of a nationally representative sample in the United States found that adolescents' perceptions of their close friends' attitudes about substance use predicted adolescents' own substance use in the last month across gender and race/ethnic groups, but the strength of relations across some demographic groups varied for some specific substances (Mason et al., 2014). Taken together, these studies suggest that further attention to gender and race/ethnicity in understanding longitudinal predictors of substance use is warranted.

The present study

Although a large body of research has documented a range of predictors of problematic alcohol, cannabis, and other substance use, our study is novel both in its long-term developmental approach, with adolescent and early adulthood predictors of problematic use in established adulthood, and in its simultaneous inclusion of a wide range of diverse predictors to be able to test whether each is significant above and beyond the others. A review of prospective longitudinal studies of childhood and adolescence predictors of adult substance use disorders concluded that because alcohol and cannabis use have higher base rates than other substance use, prospective longitudinal studies have focused primarily on predictors of alcohol and cannabis use, resulting in less understanding of prospective predictors of other substance use (Morales et al., 2020), a gap we address in the present study.

Following from the gaps in the literature reviewed above, we tested whether substance use and internalizing and externalizing problems in adolescence and early adulthood are risk factors for problematic alcohol, cannabis, and other substance use in established adulthood and whether parental monitoring, relationship quality, and involvement in adolescence that are important predictors of adolescent substance use would continue to be significant predictors of problematic alcohol, cannabis, and other substance use in established adulthood. We also examined family stress in adolescence as a predictor of problematic alcohol, cannabis, and other substance use in established adulthood and whether peer substance use in adolescence continues to predict alcohol, cannabis, and other substance use in established adulthood. In addition, we included poor physical health in adolescence and poor physical health and pain in early adulthood as possible predictors of alcohol, cannabis, and other substance use in established adulthood because of the important role pain management has played in the opioid crisis. We tested main effects of gender and race/ethnicity on problematic alcohol, cannabis, and other substance use during established adulthood, as well as whether gender and race/ethnicity moderated links between adolescent and early adulthood risk factors and problematic alcohol, cannabis, and other substance use in established adulthood. We controlled for SES in models predicting problematic alcohol, cannabis, and other substance use during established adulthood.

We address two central questions. First, do similar adolescent and early adulthood risk factors predict problematic alcohol,

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

cannabis, and other substance use in established adulthood, or are some risk factors substance-specific? We hypothesize that problematic "other" substance use, because of its inclusion of opioids, will be more strongly predicted by poor physical health, pain, and internalizing problems, whereas problematic alcohol and cannabis use will be more strongly predicted by peers' substance use, lack of parental monitoring and involvement in adolescence, and externalizing behaviors. However, we also expected the range of predictors to be implicated in more problematic use of all three categories of substances (alcohol, cannabis, and other) because previous research demonstrates that some risk factors consistently predict use of a range of substances, whereas other risk factors are substance-specific (e.g., Blanco et al., 2018). Second, are links between risk factors and problematic alcohol, cannabis, and other substance use moderated by gender or race/ethnicity? We did not have specific hypotheses regarding gender or race/ethnicity, as previous research has been inconsistent in whether links between risk factors and problematic substance use are moderated by gender or race/ethnicity.

Method

Participants were drawn from two multisite longitudinal studies. The first longitudinal study is the Child Development Project (CDP; Dodge et al., 1990; $n = 585$; 48% girls; 81% White, 17% Black, 2% other race/ethnicity). The CDP recruited a sample from three geographic areas: Knoxville, TN; Nashville, TN; and Bloomington, IN. Parents were approached at random during kindergarten registration in 1987 and 1988 and asked to participate in a longitudinal study of child development; approximately 75% of those approached agreed to participate. Kindergarteners were selected to represent a range of socioeconomic backgrounds in each geographic area. The present analyses included data from assessments when the participants were ages 16, 27, and 34. At age 34, data were available from 78% of the still-living original participants. Participants who provided data at age 34 relative to those without age 34 data scored higher on year 1 SES and were more likely to be female.

The second longitudinal study is Fast Track (FT; Conduct Problems Prevention Research Group, 2020), an intervention designed to decrease conduct problems for children at risk of aggression in kindergarten. Fifty-five at-risk elementary schools were selected based on neighborhood crime and poverty rates in Durham, NC; Nashville, TN; rural Pennsylvania; and Seattle, WA. Within each site, clusters of schools were randomly assigned to intervention and control conditions. The first of three cohorts was recruited in 1991 when all kindergarteners in study schools were screened by teachers for conduct problems using the Teacher Observation of Classroom Adaptation-Revised Authority Acceptance Scale (Werthamer-Larsson et al., 1991). Parents of children scoring in the top 40% of teacher-reported conduct problems within site were solicited to complete a 22-item instrument based on the Child Behavior Checklist (Achenbach, 1991) and similar scales to capture behavior problems at home. Standardized teacher and parent scores were combined to create a severity-of-risk screen score. Children within site were rank ordered based on this screen score, and study children were recruited starting with the highest risk until designated sample sizes were reached within site and condition. Ninety-one percent of recruited families consented. In addition to the intervention and control sample, FT includes a normative/community sample selected to represent the entire distribution of risk among kindergarteners in the control schools. Within each site, a stratified sample of 100 children (except Seattle; $n = 87$) from control schools was recruited based on ethnicity, gender, and decile of the Teacher Observation of Classroom Adaptation-Revised score to create the normative sample. By chance, 79 of the children in the control group were also recruited as members of the normative sample. The present study includes the normative sample (ranging from low to high aggression) and the control participants from cohort 1 (who were rated as high in aggression by teachers and parents but not randomized into the intervention group) ($n = 463$; 45% girls; 52% White, 43% Black, 5% other race/ethnicity). Thus, the FT sample is higher risk than the CDP sample because participants who were eligible for the intervention (and therefore the control group, which comprises the participants in the present study as the intervention participants were not included) were prescreened and found to engage in high levels of externalizing behavior based on teacher and mother reports in kindergarten; however, the FT sample also included a normative community sample that represented the entire distribution of risk among kindergarteners in the control schools so early externalizing in the FT sample as a whole is oversampled at the high-risk end of the distribution but ranges from low to high. The present analyses included data from assessments when the participants were ages 16, 25, and 32. Age 32 data were available from 83% of the still-living original participants. Participants who provided data at age 32 did not differ from those without age 32 data on year 1 SES or race/ethnicity but had lower initial risk screen scores and were more likely to be female.

Procedure and measures

Institutional review boards at universities participating in the data collection approved all study procedures and measures each year. Predictors of adult substance use were assessed at age 16 (in both CDP and FT) based on adolescent and parent reports during in-home interviews or mailed questionnaires and at age 27 (CDP) or 25 (FT) based on adult self-reports, primarily in telephone interviews but also through in-person and mail interviews. Adults reported their own substance use at age 34 (CDP) or 32 (FT), primarily in online interviews but also through telephone, in-person, and mail interviews. See Tables 1 and 2 for descriptive statistics for CDP and FT, respectively.

Adolescence predictors

At age 16 in both CDP and FT, we assessed adolescents' internalizing problems, externalizing behavior, poor physical health, parental monitoring, adolescent-parent relationship quality, parental involvement, family stress, perceptions of peer substance use, and adolescents' substance use. Somewhat different measures of these constructs were used in the two samples, so they are described separately.

In the CDP, internalizing problems and externalizing behavior were assessed via mothers' reports on the Child Behavior Checklist (Achenbach, 1991). Mothers reported whether each of 31 items for internalizing (e.g., whether the child feels worthless or inferior) and 33 items for externalizing (e.g., whether the child gets in many fights) was true for their child (0 = not true, 1 = somewhat or sometimes true, 2 = very or often true). Items were averaged to create an internalizing problems scale ($\alpha = .89$) and an externalizing behavior scale ($\alpha = .93$). Physical health was assessed as mothers' reports of whether the adolescent experienced a chronic illness or major medical problem in the past year (0 = no, 1 = yes).

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Table 1. Child Development Project study variables: descriptive statistics and correlations

	1	2	3	4	5	6	7	8	9	10	11	12
Variables												
1. Gender (1 = male)	1											
2. Race/ethnicity (1 = Black)	−.03	1										
3. SES	.05	−.41***	1									
4. Age 16 internalizing	−.15**	−.02	−.06	1								
5. Age 16 externalizing	.01	.15**	−.25***	.62***	1							
6. Age 16 poor health	−.06	.07	.04	.27***	.24***	1						
7. Age 16 monitoring	−.06	−.06	.10*	−.27***	−.45***	−.16**	1					
8. Age 16 relationship quality	.02	.04	.01	−.40***	−.56***	−.10*	.38***	1				
9. Age 16 involvement	−.14**	−.09*	.15**	−.16**	−.24***	−.03	.37***	.35***	1			
10. Age 16 family stress	−.10*	.10*	−.14**	.26***	.27***	.33***	−.22***	−.09	−.05	1		
11. Age 16 peer substance use	−.04	−.10*	.06	.14**	.24***	.16**	−.30***	−.12**	−.14**	.16**	1	
12. Age 16 own substance use	−.02	−.14**	.04	.07	.24***	.10*	−.37***	−.15***	−.15**	.12*	.69***	1
13. Age 27 internalizing	−.07	.01	−.07	.30***	.20***	.22***	−.13*	−.12*	−.06	.21***	.13**	.14**
14. Age 27 externalizing	.15**	.04	−.15**	.19***	.34***	.19***	−.17**	−.16**	−.09	.18**	.19***	.23***
15. Age 27 poor health	−.04	−.00	−.00	.08	−.01	.08	−.04	.04	−.02	.03	.03	.13*
16. Age 27 pain	−.01	.03	−.05	.10	.07	.12*	−.03	−.04	−.00	.09	.06	.06
17. Age 27 own substance use	.16***	.03	.01	.04	.19***	.12*	−.24***	−.10*	−.08	.05	.27***	.40***
18. Age 34 alcohol misuse	.21***	−.08	.17***	−.09	.03	−.02	−.06	−.09	−.10	−.09	.16**	.24***
19. Age 34 cannabis misuse	.17***	.09	−.04	.05	.18**	.05	−.18**	−.00	−.09	.05	.12*	.20***
20. Age 34 other misuse	.13**	.00	−.06	03	.09	.02	−.26***	−.01	−.17**	.15**	.19***	.15**
Descriptives												
M	.52	.17	39.53	6.72	8.28	.17	.01	.00	.00	.26	2.20	.34
SD	.50	.38	14.01	6.28	7.72	.38	.79	.75	.72	.14	1.04	.38
Range	0–1	0–1	8–66	0–32	0–43	0–1	–3 to 1	–3 to 1	–3 to 1	0 to 1	1–5	0–1
Missing (%)	0	1.9	2.6	23.8	23.4	23.2	20.7	20.5	20.5	23.2	20.9	22.1

	13	14	15	16	17	18	19	20
Variables								
1. Gender (1 = male)								
2. Race/ethnicity (1 = Black)								
3. SES								
4. Age 16 internalizing								
5. Age 16 externalizing								
6. Age 16 poor health								
7. Age 16 monitoring								
8. Age 16 relationship quality								
9. Age 16 involvement								
10. Age 16 family stress								
11. Age 16 peer substance use								
12. Age 16 own substance use								
13. Age 27 internalizing	1							
14. Age 27 externalizing	.66***	1						
15. Age 27 poor health	.20***	.14**	1					
16. Age 27 pain	.31***	.23***	.18***	1				
17. Age 27 own substance use	.15**	.30***	.20***	.10*	1			

(*Continued*)

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Table 1. (*Continued*)

	13	14	15	16	17	18	19	20
18. Age 34 alcohol misuse	.01	.17**	.02	−.09	.31***	1		
19. Age 34 cannabis misuse	.18***	.36***	.07	.03	.44***	.29***	1	
20. Age 34 other misuse	.21***	.24***	.18***	.10*	.16**	.06	.17***	1
Descriptives								
M	15.19	13.06	.12	.14	.52	3.84	1.42	.46
SD	10.89	8.17	.33	.35	.31	4.64	3.50	2.08
Range	0–54	0–49	0–1	0–1	0–1	0–30	0–21	0–16
Missing (%)	21.2	21.2	22.1	22.1	21.4	23.8	24.6	25.0

Note. $^{*}p < .05$; $^{**}p < .01$; $^{***}p < .001$.

Table 2. Fast Track study variables: descriptive statistics and correlations

	1	2	3	4	5	6	7	8	9	10	11	12
Variables												
1. Gender (1 = male)	1											
2. Race/ethnicity (1 = Black)	.05	1										
3. SES	.05	−.23***	1									
4. Age 16 internalizing	−.10	.14*	−.15**	1								
5. Age 16 externalizing	.15**	.06	−.02	.11*	1							
6. Age 16 poor health	−.07	.05	.02	.08	−.02	1						
7. Age 16 monitoring	−.16**	−.23***	.08	−.12*	−.11*	.02	1					
8. Age 16 relationship quality	−.18**	−.09	−.12*	−.20***	−.21***	.03	.39***	1				
9. Age 16 involvement	−.04	−.12*	.14*	−.08	−.10	.10	.51***	.41***	1			
10. Age 16 family stress	.06	.06	−.13*	.10	.12*	.16**	−.11*	−.16**	.01	1		
11. Age 16 peer substance use	.09	.05	−.11	.21***	.37***	.04	−.07	−.14**	−.04	.09	1	
12. Age 16 own substance use	.08	−.05	.02	.23***	.32***	.06	−.12*	−.26***	−.02	.13*	.54***	1
13. Age 25 internalizing	.01	.11*	−.20***	.31***	−.05	.02	−.07	−.15**	−.07	.06	.12*	.08
14. Age 25 externalizing	.26***	.11*	−.16**	.22***	.10	.00	−.16**	−.23***	−.13*	.05	.20***	.14*
15. Age 25 poor health	.03	−.05	−.03	.08	−.03	.10	.06	−.13*	−.03	.11	.02	−.01
16. Age 25 pain	−.01	−.04	−.17**	.11*	−.06	.10	−.04	−.09	.02	.06	.02	.01
17. Age 25 own substance use	.21***	−.05	−.01	.02	.11	.04	.05	−.17**	.05	.05	.17**	.28***
18. Age 32 alcohol misuse	.19***	−.09	.18**	.06	.10	−.00	.05	−.08	−.04	−.00	.02	.14*
19. Age 32 cannabis misuse	.26***	.07	.02	.04	.07	.04	−.07	.18**	−.09	.02	.08	.14*
20. Age 32 other misuse	.21***	−.02	−.02	−.02	.06	.02	.02	−.06	.05	.09	.06	.12*
Descriptives												
M	.55	.43	26.31	1.66	.03	.06	4.60	3.89	4.06	.15	1.76	.26
SD	.50	.50	13.28	.44	.08	.25	.46	.76	.59	.14	.89	.33
Range	0–1	0–1	5–66	1–3	0–1	0–1	3–5	1–5	2–5	0–1	1–4	0–1
Missing (%)	0	0	0	25.7	25.5	23.5	24.6	23.5	23.5	23.8	25.7	25.5

	13	14	15	16	17	18	19	20
Variables								
1. Gender (1 = male)								
2. Race/ethnicity (1 = Black)								
3. SES								
4. Age 16 internalizing								
5. Age 16 externalizing								

(*Continued*)

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Table 2. (*Continued*)

	13	14	15	16	17	18	19	20
6. Age 16 poor health								
7. Age 16 monitoring								
8. Age 16 relationship quality								
9. Age 16 involvement								
10. Age 16 family stress								
11. Age 16 peer substance use								
12. Age 16 own substance use								
13. Age 25 internalizing	1							
14. Age 25 externalizing	.70***	1						
15. Age 25 poor health	.21***	.14**	1					
16. Age 25 pain	.35***	.15**	.18***	1				
17. Age 25 own substance use	.26***	.36***	−.01	.12*	1			
18. Age 32 alcohol misuse	.18**	.23***	.17**	−.05	.27***	1		
19. Age 32 cannabis misuse	.15**	.34***	.07	−.01	.39***	.23***	1	
20. Age 32 other misuse	.15**	.24***	−.03	.01	.17**	.24***	.27***	1
Descriptives								
M	17.40	15.34	.12	.12	.56	3.21	2.07	.60
SD	12.02	10.44	.32	.33	.33	4.52	4.58	2.57
Range	0–59	0–58	0–1	0–1	0–1	0–35	0–26	0–18
Missing (%)	15.6	15.6	15.6	15.6	16.2	19.2	21.0	20.1

Note. *$p < .05$; **$p < .01$; ***$p < .001$.

Parental monitoring was assessed as the mean of three items (e.g., whether an adult is present when the child goes to friends' homes) reported by mothers (coded on a 5-point scale from 1 = never to 5 = always or almost always, with items reverse scored as needed). The three items were averaged to create a scale (α = .71). Mother-adolescent relationship quality was assessed as the mean of five items reported by mothers (e.g., how well the mother and adolescent get along). Each item was rated on a 5-point scale with items reverse scored as needed; items were averaged to create a scale (α = .81). Parental involvement was the mean of four items reported by the mother capturing how frequently the mother and adolescent talked about daily events, happy things, and problems (each coded on a 5-point scale from 1 = never to 5 = very frequently) as well as how frequently the mother helped with or attended activities (coded on a 4-point scale from 1 = never to 4 = more than once per month). The four items were standardized and averaged to create a scale (α = .68). Family stress was the proportion of 19 possible stressors (e.g., death in the family, parents' divorce) that the mother reported the family experienced in the last year. Adolescents reported how often their friends smoke cigarettes, drink alcohol, and smoke cannabis (each rated on a 5-point scale from 1 = never to 5 = very often). The three items were averaged to create a scale (α = .84). Adolescents were asked whether they used cigarettes and cannabis in the last year and whether they used alcohol or drugs for non-medical reasons in the last 6 months (0 = no, 1 = yes). A composite variable was calculated as the proportion of these substances the adolescent used, ranging from 0 if the adolescent responded "no" to using each substance to 1 if the adolescent responded "yes" to using each substance.

In FT, we assessed adolescents' internalizing problems as the mean of 30 self-reported items capturing symptoms of depression experienced (1 = almost never to 4 = most of the time; Reynolds, 1987); items were averaged to create a scale (α = .92). Adolescents' externalizing behaviors were assessed as the mean of 25 self-reported dichotomous items capturing acts of delinquency committed in the past year (Elliott et al., 1985; α = .85). Physical health was assessed as parents' reports of whether the adolescent experienced a chronic illness or major medical problem in the past year (0 = no, 1 = yes). Parental monitoring was assessed by parents' reports of their knowledge of their child's activities, companions, and whereabouts rated on a 5-point scale (1 = almost never to 5 = almost always) using a measure adapted from Loeber and Stouthamer-Loeber (1986). Items were averaged to create a scale (α = .67). Adolescent-parent relationship quality was the mean of parent reports of satisfaction with their role as a parent, perceptions of the child's difficultness, perceptions of their relationship with the child, and satisfaction with the child's behavior with higher scores reflecting better relationships (Dodge et al., 1990; α = .79). Parental involvement was the mean of five items capturing the frequency of parent-child discussion regarding the child's activities and problems with higher scores indicating more frequent communication (α = .68). Family stress was the proportion of 18 possible stressors (e.g., death in the family, parents' divorce) that the parent reported the family experienced in the last year. Adolescents reported how often each of their three best friends smokes cigarettes/uses tobacco, drinks alcohol, and uses illegal drugs including cannabis (1 = not at all to 4 = very much). The nine items were averaged to create a scale (α = .91). Adolescents were asked whether they used alcohol, cigarettes,

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

and cannabis in the last year. A composite variable was calculated as the proportion of these three substances the adolescent used.

Early adulthood predictors

At age 27 (CDP) or age 25 (FT), participants reported on their own internalizing problems, externalizing behavior, physical health, pain, and substance use. Internalizing problems were assessed using 39 items from the Adult Self-Report (Achenbach & Rescorla, 2003). Participants reported whether each item (e.g., "I feel worthless or inferior") was true for them (0 = not true, 1 = somewhat or sometimes true, 2 = very or often true), and items were averaged to create a scale (α = .93 in both CDP and FT). Externalizing problems were assessed using 35 items from the Adult Self-Report (Achenbach & Rescorla, 2003). Participants reported whether each item (e.g., "I get in many fights") was true for them (0 = not true, 1 = sometimes true, 2 = often true), and items were averaged to create a scale (α = .88 in CDP and .91 in FT). Poor physical health was coded 1 if the participant reported poor general health or living with a chronic disease/condition. Pain was assessed through participants' reports (yes or no) of whether they had experienced moderate to severe pain in the past 4 weeks (Ware & Sherbourne, 1992). Young adults were asked whether they used alcohol, cigarettes, and cannabis in the last year. A composite variable was calculated as the proportion of these three substances the young adult used.

Substance use in established adulthood

At age 34 (CDP) or age 32 (FT), participants reported on their problematic substance use with three measures based on the impact of substance use on daily functioning beyond frequency of use: Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993), Cannabis Use Disorders Identification Test-Revised (CUDIT-R; Adamson et al., 2010), and Drug Abuse Screening Test (DAST-20; Skinner & Goldberg, 1986). The AUDIT includes six items (e.g., "How often during the last year have you found that you were not able to stop drinking once you had started?" "How often during the last year have you been unable to remember what happened the night before because of your drinking?") rated on 5-point scales ranging from 0 = never to 4 = daily or almost daily. The AUDIT also includes four items that use different rating scales (e.g., "How many drinks containing alcohol do have on a typical day when you are drinking?" rated on a 5-point scale ranging from 0 = 1 or 2 to 4 = 10 or more). The items were standardized and summed to create a measure reflecting problem alcohol use. The Cannabis Use Disorders Identification Test-Revised first asks whether the participant had used any cannabis during the last 6 months. Participants who responded yes to this initial question then completed eight additional items. Five of these items (e.g., "How often during the past 6 months did you fail to do what was normally expected of you because of using cannabis?" "How often during the past 6 months have you devoted a great deal of your time to getting, using, or recovering from cannabis?") were rated on a 5-point scale ranging from 0 = never to 4 = daily or almost daily. The remaining three items use different rating scales (e.g., "How many hours were you 'stoned' on a typical day when you were using cannabis?" rated on a 5-point scale ranging from 0 = less than 1 to 4 = 7 or more). The items were standardized and summed to create a measure reflecting problem cannabis use. The DAST-20 includes 20 items (e.g., "Have you had 'blackouts' or 'flashbacks' as a result of drug use?" "Have you ever experienced withdrawal symptoms (felt sick) when you stopped taking drugs?") rated as 0 = no or 1 = yes. The DAST-20 captures misuse of prescription drugs as well as illegal drug use. The items were summed to create a measure reflecting problem drug use.

Data analyses

Path analyses were conducted using Mplus 8 (Muthén & Muthén, 2017). Models were estimated using full-information maximum likelihood with robust standard errors (Rubin & Little, 2002) and evaluated according to the sample adjusted Bayesian Information Criterion (aBIC), chi-square (χ^2) value, Comparative Fit Index (CFI), and Root Mean Square Error of Approximation (RMSEA) with confidence intervals (CI). Models with the lowest aBIC are preferred. A non-significant χ^2 value ($p > .05$), CFI values greater than .90, and RMSEA less than .06 indicate adequate fit. However, with larger sample sizes as per the present study, a non-significant χ^2 value is not necessary (Hu & Bentler, 1999).

Models were conducted separately for CDP and FT. All predictors at the same time point were specified to covary, and each model included all three substance outcomes so we were able to test for unique correlates. That is, the models included all three outcomes simultaneously, so we accounted for alcohol and cannabis problems when assessing other drug problems, accounted for alcohol and other drug problems when assessing cannabis problems, and accounted for cannabis and other drug problems when assessing alcohol problems. Thus, variance predicted in each outcome is above and beyond the shared variance accounted for by the other outcomes. For CDP, we tested the full sample and a gender multi-group model. For FT, we tested the full sample, a gender multi-group model, and a race/ethnicity multi-group model. The race/ethnicity composition of CDP (81% White) precluded the possibility of examining race/ethnicity as a multi-group model in this sample. For multi-group models, we compared two nested models. The first nested model allowed the predictors regressed onto outcome parameters to be freely estimated, and the second nested model held these parameters equal across the two groups. Models were then compared using a χ^2 test with significance indicating that the model differs across groups. For models that differed across groups, we also examined whether each parameter was significantly different across groups by freeing each parameter in turn and comparing whether these models differed from the fully constrained model using a χ^2 test. We report the full sample model when multi-group models were not significant, otherwise we report the significant multi-group models. Gender, race/ethnicity, and SES were included as covariates in the full sample models, and the remaining two variables were included as covariates in the multi-group models (e.g., race/ethnicity and SES were included as covariates in the gender multi-group models).

Results

Descriptives

Descriptive statistics and correlations of main study variables are presented in Table 1 for CDP and Table 2 for FT.

Missing data

Main study variable rates of missingness are presented in Table 1 for CDP and Table 2 for FT. Participants with and without missing data at each time point were compared on baseline demographic data (i.e., gender, race/ethnicity, and SES).

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Child Development Project

Participants with and without missing data at age 16 did not differ on SES or race/ethnicity. However, those participants with missing data at age 16 were more likely to be male, χ^2 (1, $N = 585$) = 4.38, $p = .036$. At age 27, participants without data scored lower on SES, $t(568) = -5.12$, $p < .001$, and were more likely to be male, χ^2 (1, $N = 585$) = 12.65, $p < .001$, and Black, χ^2 (1, $N = 585$) = 13.28, $p < .001$. Similarly, at age 34, participants without data scored lower on SES, $t(568) = -4.31$, $p < .001$, and were more likely to be male, χ^2 (1, $N = 585$) = 19.60, $p < .001$, and Black, χ^2 (1, $N = 585$) = 5.58, $p = .018$.

Fast Track

Participants with and without missing data at age 16 did not differ on SES; however, those participants with missing data were more likely to be male, χ^2 (1, $N = 463$) = 5.25, $p = .022$, and White, χ^2 (1, $N = 463$) = 4.30, $p = .038$. At age 25, participants with and without data did not differ on SES and race/ethnicity; however, those participants with missing data were more likely to be male, χ^2 (1, $N = 463$) = 7.32, $p = .007$. Similarly, at age 32, participants with and without data did not differ on SES and race/ethnicity; however, those participants with missing data were more likely to be male, χ^2 (1, $N = 463$) = 22.86, $p < .001$.

Structural models

All correlation coefficients between predictors and outcomes from the structural models are shown in the Supplementary Tables for CDP (see Supplementary Table S1) and for the FT gender multi-group model (see Supplementary Tables S2 and S3).

Child Development Project

Both the full sample model ($\chi^2(45) = 163.54$, $p < .001$, aBIC = 22,892.85, CFI = .93, RMSEA = .07, 90% CI[.06, .08]) and the freely estimated gender multi-group model ($\chi^2(90) = 221.39$, $p < .001$, aBIC = 22,917.26, CFI = .92, RMSEA = .08, 90% CI[.06, .09]) provided adequate fit to the data. The nested gender multi-group models revealed no significant differences across gender, $\Delta\chi^2(42) = 48.61$, $p = .224$, and thus, we report findings from the full sample model (see Table 3). Being male, higher SES, lower parent-adolescent relationship quality at age 16, higher externalizing problems and own substance use, and lower pain at age 27 predicted problematic alcohol use at age 34 ($R^2 = .23$). Only higher externalizing problems and own substance use at age 27 predicted problematic cannabis use at age 34 ($R^2 = .28$). Finally, lower parental monitoring and higher peer substance use at age 16, and poorer physical health at age 27 predicted problematic other substance use at age 34 ($R^2 = .18$).

Fast Track

The full sample model ($\chi^2(45) = 82.94$, $p < .001$, aBIC = 13,862.89, CFI = .96, RMSEA = .04, 90% CI[.03, .06]), the freely estimated gender multi-group model ($\chi^2(90) = 156.07$, $p < .001$, aBIC = 13,298.19, CFI = .94, RMSEA = .06, 90% CI[.04, .07]), and the freely estimated race/ethnicity multi-group model ($\chi^2(90) = 145.54$, $p < .001$, aBIC = 13,956.77, CFI = .95, RMSEA = .05, 90% CI[.04, .07]) provided adequate fit to the data. Whereas the nested gender multi-group models revealed significant differences across gender, $\Delta\chi^2(42) = 78.47$, $p = .001$, the nested race/ethnicity multi-group models revealed no significant differences across

Table 3. Estimates of predictors and outcomes for the Child Development Project – full sample

	B(*SE*)	β	*p*
Age 34 alcohol misuse			
Gender (1 = male)	1.31(.41)	.29	.001
Race/ethnicity (1 = Black)	.26(.61)	.06	.675
SES	.05(.02)	.17	.001
Age 16 internalizing	−.04(.04)	−.05	.382
Age 16 externalizing	.00(.05)	.00	.964
Age 16 poor health	−.28(.60)	−.02	.638
Age 16 monitoring	.64(.37)	.11	.088
Age 16 relationship quality	−.71(.35)	−.12	.044
Age 16 involvement	−.17(.35)	−.03	.630
Age 16 family stress	−2.70(1.61)	−.09	.093
Age 16 peer substance use	.25(.28)	.06	.374
Age 16 own substance use	1.14(.86)	.10	.188
Age 27 internalizing	−.03(.03)	−.08	.275
Age 27 externalizing	.12(.05)	.21	.016
Age 27 poor health	.03(.68)	.00	.963
Age 27 pain	−1.52(.67)	−.12	.023
Age 27 own substance use	3.48(.82)	.24	<.001
Age 34 cannabis misuse			
Gender (1 = male)	.42(.31)	.12	.178
Race/ethnicity (1 = Black)	.71(.49)	.21	.141
SES	.01(.01)	.05	.295
Age 16 internalizing	.01(.03)	.01	.870
Age 16 externalizing	.04(.04)	.10	.250
Age 16 poor health	−.46(.44)	−.05	.295
Age 16 monitoring	−.13(.24)	−.03	.599
Age 16 relationship quality	.55(.30)	.12	.068
Age 16 involvement	−.14(.32)	−.03	.652
Age 16 family stress	.00(1.42)	.00	.999
Age 16 peer substance use	−.15(.21)	−.05	.464
Age 16 own substance use	.35(.66)	.04	.593
Age 27 internalizing	−.01(.02)	−.04	.565
Age 27 externalizing	.13(.04)	.29	<.001
Age 27 poor health	−.24(.56)	−.02	.671
Age 27 pain	−.44(.51)	−.04	.393
Age 27 own substance use	3.85(.57)	.34	<.001
Age 34 other substance misuse			
Gender (1 = male)	.39(.20)	.19	.054
Race/ethnicity (1 = Black)	−.24(.30)	−.11	.416
SES	−.00(.01)	−.02	.681
Age 16 internalizing	−.03(.03)	−.08	.272
Age 16 externalizing	−.00(.03)	−.00	.976
Age 16 poor health	−.46(.41)	−.08	.259
Age 16 monitoring	−.56(.25)	−.21	.024
Age 16 relationship quality	.23(.23)	.08	.308

(*Continued*)

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Table 3. (*Continued*)

	B(*SE*)	β	*p*
Age 16 involvement	−.31(.18)	−.11	.088
Age 16 family stress	1.66(1.32)	.11	.207
Age 16 peer substance use	.32(.16)	.16	.043
Age 16 own substance use	−.57(.35)	−.10	.103
Age 27 internalizing	.02(.01)	.11	.113
Age 27 externalizing	.02(.02)	.09	.277
Age 27 poor health	.97(.48)	.15	.042
Age 27 pain	.13(.34)	.02	.706
Age 27 own substance use	.17(.35)	.02	.635

race/ethnicity, $\Delta\chi^2(42) = 52.82$, $p = .122$, and thus, we report findings from the freely estimated gender multi-group model (see Table 4). Among the male sample, greater internalizing problems and own substance use, poorer physical health, and lower pain at age 25 predicted problematic alcohol use at age 32 ($R^2 = .29$), and greater externalizing problems and own substance use at age 25 predicted problematic cannabis use at age 32 ($R^2 = .30$). Among the female sample, higher SES, internalizing problems, externalizing problems, and monitoring, better physical health, and lower peer substance use at age 16, and higher externalizing problems and own substance use at age 25 predicted problematic alcohol use at age 32 ($R^2 = .31$). In addition, higher own substance use at age 25 predicted problematic cannabis use at age 32 ($R^2 = .14$). There were no significant effects for problematic other substance use in this gender multi-group model (male: $R^2 = .15$; female: $R^2 = .09$).

Table 4 (see last two columns) also reports χ^2 tests comparing whether each parameter was significantly different across groups by freeing each parameter in turn. For alcohol misuse, internalizing problems at age 25 differed across groups. For cannabis misuse, internalizing and externalizing problems, and own substance use at age 25 differed across groups; however, internalizing problems was not significant for males or females. Finally, for other substance misuse, monitoring at age 16, and internalizing and externalizing problems at age 25 differed across groups; however, none of these parameters were significant for males or females.

Discussion

This study aimed to understand whether a set of theoretically and empirically supported adolescent and early adulthood risk factors uniquely predicted problematic alcohol, cannabis, and other substance use in established adulthood. Our first research question was whether adolescent and early adulthood risk factors predicted problematic alcohol, cannabis, and other substance use in established adulthood similarly, or whether some risk factors are substance-specific. We did not find support for our hypothesis that poor physical health, pain, and internalizing problems would be more strongly related to problematic other substance use than to problematic alcohol or cannabis use, but we did find support for the hypothesis that some of the predictors would be substance-specific. These findings are broadly consistent with prior studies that have found that some risk factors consistently predict use of a range of substances, whereas other risk factors are substance-specific (e.g., Blanco et al., 2018). We extend this previous research by demonstrating that a wider range of risk factors in adolescence and early adulthood predicted problematic alcohol and cannabis use than other substance use in established adulthood.

Our second research question was whether links between risk factors and problematic alcohol, cannabis, and other substance use are moderated by gender or race/ethnicity. Previous research on whether gender and race/ethnicity moderate the association between risk factors and subsequent problematic substance use has been inconsistent (e.g., Bachrach & Chung, 2020; Kulis et al., 2012; Vaughan et al., 2018; Wiesner et al., 2005). We did not find gender differences in predictors of problematic alcohol, cannabis, or other substance use in the CDP sample. In the FT sample, we found evidence that males and females differed with respect to links between several adolescent and early adulthood risk factors and subsequent problematic substance use in established adulthood, but as with previous research, this pattern of findings did not provide evidence for a clear pattern of consistently greater risk for subsequent problematic substance use based on prior risk factors for females versus males. Instead, the specificity principal in developmental science appears to apply to these findings, suggesting that specific conditions of specific people at specific times moderate specific relations by specific processes (Bornstein, 2017). Race/ethnicity did not moderate associations between the risk factors and subsequent problematic alcohol, cannabis, or other substance use. Whether gender and race/ethnicity serve as moderators may depend on other factors, such as developmental stage and the particular risk factors and outcomes involved.

Overall, the model predicted the most variance in cannabis use in the CDP sample and the FT male sample and the most variance in alcohol use in the FT female sample, although in the FT male sample, the model explained 29% of the variance for alcohol use and 30% variance for cannabis, so these percentages are not meaningfully different. However, more individual predictors in the multivariate path models were significantly related to alcohol misuse (6 of 17 predictors in the CDP, 4 of 16 in FT males, and 8 of 16 in FT females) than to cannabis misuse (2 of 17 predictors in the CDP, 2 of 16 in FT males, and 1 of 16 in FT females) or other substance misuse (3 of 17 predictors in the CDP and none in FT males or females). We may have had more power to detect predictors of alcohol misuse given the greater prevalence of alcohol misuse than cannabis or other substance misuse both in our sample and in epidemiological studies (e.g., McCabe et al., 2021), perhaps in part accounting for this pattern of findings.

Not surprisingly given the links between externalizing behavior and substance use in theoretical frameworks such as Problem-Behavior Theory (Jessor, 1987) and the Social Development Model (e.g., Kosterman et al., 2014), as well as previous meta-analytic work (Meque, Dachew, et al., 2019) and reviews (Meque, Salom, et al., 2019), we found that individuals' own prior substance use in early adulthood as well as prior externalizing behaviors were the most consistent predictors of substance use in established adulthood across samples. Individuals' own prior substance use in adolescence and externalizing behaviors in adolescence (with the exception of the FT female subsample) did not significantly predict substance use in established adulthood above and beyond substance use and externalizing behavior in early adulthood and the other predictors in the models. Internalizing pathways to substance misuse also have been incorporated in previous theoretical frameworks and supported empirically (e.g., Hussong et al., 2011). We also found evidence for internalizing

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

Table 4. Estimates of predictors and outcomes for Fast Track – gender multi-group model

	Male sample			Female sample			χ^2 test	
	B(*SE*)	β	*p*	*B*(*SE*)	β	*p*	$\Delta\chi^2$	*p*
Age 32 alcohol misuse								
Race/ethnicity (1 = Black)	−1.69(.93)	−.30	.070	−.53(.37)	−.19	.154		
SES	.06(.04)	.15	.069	.04(.02)	.17	.020		
Age 16 internalizing	−.32(1.39)	−.02	.819	1.34(.44)	.22	.002	.01	.914
Age 16 externalizing	.76(6.45)	.01	.907	11.14(3.60)	.20	.002	.95	.329
Age 16 poor health	4.48(3.93)	.18	.255	−1.48(.61)	−.13	.015	1.30	.254
Age 16 monitoring	.09(1.38)	.01	.951	1.45(.46)	.23	.002	1.15	.283
Age 16 relationship quality	.45(.84)	.06	.588	.62(.38)	.16	.102	.79	.373
Age 16 involvement	−.66(.88)	−.06	.455	−.49(.41)	−.11	.229	.95	.330
Age 16 family Stress	−.96(3.29)	−.02	.770	−1.03(1.46)	−.05	.479	1.78	.182
Age 16 peer substance use	.44(.60)	.07	.465	−.62(.31)	−.16	.047	1.25	.263
Age 16 own substance use	1.41(1.90)	.09	.457	1.43(.83)	.15	.084	.39	.531
Age 25 internalizing	.20(.07)	.42	.004	−.06(.03)	−.22	.054	12.29	<.001
Age 25 externalizing	−.11(.06)	−.21	.056	.09(.03)	.25	.003	.10	.748
Age 25 poor health	4.64(1.82)	.25	.011	1.19(1.02)	.12	.245	3.44	.063
Age 25 pain	−3.20(1.47)	−.18	.030	.30(.78)	.03	.702	.01	.943
Age 25 own substance use	3.49(1.20)	.20	.004	1.40(.69)	.16	.041	2.09	.148
Age 32 cannabis misuse								
Race/ethnicity (1 = Black)	.69(.75)	.12	.361	.30(.38)	.12	.432		
SES	.04(.04)	.11	.258	−.01(.01)	−.06	.227		
Age 16 internalizing	1.04(1.49)	.08	.484	−.61(.36)	−.11	.087	2.64	.104
Age 16 externalizing	−3.78(6.95)	−.06	.586	.26(3.02)	.01	.932	.06	.809
Age 16 poor health	.01(5.38)	.00	.999	.38(.87)	.04	.665	.09	.770
Age 16 monitoring	1.21(1.30)	.10	.353	.45(.38)	.08	.241	.10	.747
Age 16 relationship quality	−.09(.66)	−.01	.887	−.77(.44)	−.22	.082	.00	.965
Age 16 involvement	−.69(.82)	−.07	.396	−.31(.36)	−.08	.388	.98	.322
Age 16 family stress	−4.31(3.88)	−.11	.266	−.69(1.25)	−.03	.584	1.00	.317
Age 16 peer substance use	−.42(.79)	−.07	.594	−.18(.37)	−.05	.629	.32	.573
Age 16 own substance use	1.60(2.64)	.10	.544	.38(.59)	.04	.518	.23	.631
Age 25 internalizing	−.08(.05)	−.18	.105	−.01(.02)	−.02	.841	3.94	.047
Age 25 externalizing	.20(.06)	.41	.001	.00(.03)	.01	.939	17.92	<.001
Age 25 poor health	1.26(1.80)	.07	.484	.76(.69)	.09	.271	.35	.555
Age 25 pain	1.26(1.50)	.07	.399	−1.10(.57)	−.14	.054	2.50	.114
Age 25 own substance use	5.48(1.25)	.32	<.001	2.05(.84)	.26	.015	9.75	.002
Age 32 other substance misuse								
Race/ethnicity (1 = Black)	−.54(.57)	−.16	.339	.03(.07)	.09	.655		
SES	−.03(.03)	−.11	.266	.01(.00)	.19	.076		
Age 16 internalizing	−.53(1.10)	−.07	.629	.02(.06)	.03	.743	.02	.888
Age 16 externalizing	1.18(3.59)	.03	.742	−.50(.38)	−.08	.191	.79	.373
Age 16 poor health	.53(1.39)	.04	.702	−.08(.06)	−.05	.177	.12	.729
Age 16 monitoring	.82(.55)	.12	.139	−.10(.08)	−.12	.212	14.33	<.001
Age 16 relationship Quality	−.14(.35)	−.03	.688	.02(.04)	.04	.623	.00	.955
Age 16 involvement	.74(.80)	.12	.358	.09(.07)	.16	.168	1.92	.166
Age 16 family stress	−.59(2.41)	−.02	.807	.15(.20)	.05	.458	.18	.675

(*Continued*)

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Table 4. (*Continued*)

	Male sample			Female sample			χ^2 test	
	B(*SE*)	β	*p*	*B*(*SE*)	β	*p*	$\Delta\chi^2$	*p*
Age 16 peer substance use	−.27(.43)	−.08	.524	−.03(.03)	−.06	.309	.39	.531
Age 16 own substance use	1.33(1.32)	.14	.312	.11(.08)	.09	.172	.49	.482
Age 25 internalizing	.04(.03)	.14	.210	.00(.00)	.10	.410	7.11	.008
Age 25 externalizing	.05(.03)	.15	.084	−.00(.00)	−.06	.478	6.62	.010
Age 25 poor health	−.82(.70)	−.07	.239	−.08(.05)	−.07	.117	2.14	.143
Age 25 pain	−.72(.81)	−.07	.377	−.09(.05)	−.08	.108	.73	.391
Age 25 own substance use	.22(.68)	.02	.744	.03(.08)	.03	.739	1.61	.205

at age 16 (for FT females) and at age 25 (for FT males), as predictors of alcohol misuse in established adulthood, above and beyond the other predictors in the models.

Other notable predictors in our models included parent-adolescent relationship quality and peer substance use. Consistent with developmental cascade models (Dodge et al., 2009) and prior meta-analytic work (Yap et al., 2017) documenting the importance of parenting in the development of substance use problems, we found that higher age 16 parent-adolescent relationship quality predicted less alcohol misuse; and more parental monitoring at age 16 predicted less other substance misuse in established adulthood in the CDP sample. Several aspects of parent-adolescent relationships are robustly associated with early onset of substance use and the developmental course of substance use during adolescence (Van Ryzin et al., 2016). It is notable that aspects of parent-adolescent relationships continued to predict alcohol misuse and other substance misuse almost two decades later in the CDP sample, perhaps because lower quality parent-adolescent relationships contributed to a developmental cascade including earlier substance use in adolescence. Consistent with theories of deviant peer contagion (Dishion et al., 1995; Piehler & Dishion, 2014), we found that peers' substance use when participants were age 16 predicted substance misuse in established adulthood in the CDP sample, accounting for peers' substance use during early adulthood and all other variables in the models. It is possible that in the FT sample that was at higher risk for externalizing problems than the CDP sample, parent-adolescent relationships would have had more problematic behaviors to overcome to have long-term protective effects on problematic alcohol and substance use during established adulthood and that peer substance use in adolescence may have conferred less future risk if adolescents themselves were already on a risky trajectory.

Although most of the associations were in the expected directions, a few were counterintuitive. It is possible that multicollinearity could have resulted in suppressor effects or other statistical anomalies, although with the exception of the correlation between internalizing and externalizing problems in early adulthood, the bivariate correlations among the predictors in both samples are well below the absolute values that would typically raise concerns about multicollinearity. Although the multivariate path analyses showed unexpected relations between more pain at age 27 and less alcohol misuse at age 34 in the CDP sample, more pain at age 25 and less alcohol misuse at age 32 in the FT male sample, and poorer health, more parental monitoring, and less peer substance use at age 16 and less alcohol misuse at age 32 in the FT female sample, we urge caution in interpreting these findings. In the FT female sample, the bivariate correlation between poor health at age 16 and alcohol misuse at age 32 is not significant, more parental monitoring at age 16 is correlated with less substance use concurrently at age 16 as expected, and less peer substance use at age 16 is correlated with less of individuals' own substance use at ages 16 and 25 as expected. Because the finding of more pain in early adulthood related to less alcohol misuse in established adulthood replicated in the CDP and FT male samples, this unexpected finding deserves future attention. However, we also note that poorer health in early adulthood was related to more alcohol misuse in the FT male sample and more other substance misuse in the CDP sample.

Several influential calls for replication have been made in the last decade, asserting the need for psychological and developmental science to test the generalizability and robustness of findings (Bonett, 2012; Duncan et al., 2014). Underlying these calls for replication is the acknowledgement that developmental processes may not generalize across diverse populations but instead may be dependent on the nature of particular research samples (Bornstein et al., 2013). The present study included two independent samples, which differed in their initial level of risk for externalizing behaviors in childhood, which have been found to be risk factors for the development of substance use in adolescence (e.g., Colder et al., 2013). Including both a high-risk and low-risk sample contributes to our ability to test the replicability and generalizability of findings across samples. We found many similarities in the findings in these two samples. However, there were differences as well. Gender also moderated relations between risk factors and problematic alcohol, cannabis, or other substance use in the higher-risk FT but not lower-risk CDP sample (although male gender itself was a risk factor for more problematic alcohol use in the CDP sample). Externalizing behavior was a particularly consistent risk factor for subsequent problematic substance use across substances and samples, despite the FT sample's initial higher risk of externalizing behavior and therefore potential restriction of range that would have made it less likely to find significant associations between prior externalizing and substance use outcomes.

Strengths and Limitations

Our study had several notable strengths, particularly the inclusion of two independent samples that were recruited in early childhood and followed prospectively to age 32 (FT) or 34 (CDP), with

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predictors of problematic substance use in established adulthood assessed in adolescence and earlier in adulthood. We also examined predictors of problematic alcohol, cannabis, and other substance use in the same model to be able to test whether predictors generalize across substances or are substance-specific while including substances other than alcohol or cannabis. In this way, our study responds to a call for such work identified in a previous systematic review of prospective longitudinal studies of childhood and adolescence predictors of adult substance use disorders (Morales et al., 2020).

However, our study also had limitations. First, as we acknowledged at the outset, many risk and protective factors that we did not investigate also are related to problematic alcohol, cannabis, and other substance use. For example, parents' substance use, through either modeling or genetic factors, affects individuals' own substance use (Spechler et al., 2019), stress hormones are related to more alcohol and cannabis use (Barton et al., 2018), and brain areas related to inhibitory control, reward processing, and executive functioning are implicated in alcohol initiation and misuse (O'Halloran et al., 2017). Second, we were able to test race/ethnicity as a moderator of associations only for the FT sample, which was 43% Black and 52% White. Future research is needed to test whether the findings hold in more racially and ethnically diverse samples. Third, with the exception of testing gender and race/ethnicity as moderators, our models tested whether a range of risk factors predicted problematic alcohol, cannabis, and other substance use above and beyond the other risk factors. Future research will be needed to test long-term mediation models of developmental processes and whether risk is moderated by factors beyond the sociodemographics we examined in the present study. Fourth, although predictors assessed in adolescence were reported by both adolescents and parents, predictors assessed in early adulthood and substance use in established adulthood were based on self-reports and are thus subject to self-report biases (Althubaiti, 2016). Fifth, the FT sample included participants from a high-risk control group that had a limited, high range of externalizing behaviors at the beginning of the study, which likely elevated their risk for subsequent substance use problems, although the FT sample also included a normative community sample without this restricted range of externalizing at the beginning of the study. In addition, although attrition was low for a study spanning almost three decades, participants who provided data at age 34 (CDP) or age 32 (FT) were at somewhat lower risk of substance use problems by virtue of their higher kindergarten SES (CDP), lower-risk screen scores in kindergarten (FT), and female gender (both samples). Thus, base rates of problematic alcohol, cannabis, and other substance use in established adulthood may have been lower than if the full original samples had provided data. In addition, predictors associated with higher risk and male gender may have had attenuated associations with substance misuse in established adulthood, but this would have increased the risk for Type II rather than Type I errors. Finally, although we tested specificity of predictors of problematic alcohol, cannabis, and other substance use, the "other" substance use category was not further differentiated into specific other substances. Future research could examine finer-grained distinctions, such as misuse of prescribed opioids compared to specific other substances. In addition, we may have found fewer significant predictors of other substance use problems compared to problematic alcohol and cannabis use due to low power associated with less prevalence of problems with other drug use compared with problems with alcohol or cannabis use.

Implications for practice, policy, and future research

Our findings have at least three implications for policy, practice, and future research. First, interventions designed to reduce different types of substance misuse may need to target different risk factors. In particular, although both alcohol misuse and cannabis misuse in established adulthood were predicted by externalizing problems and individuals' own prior substance use earlier in adulthood, other substance misuse in established adulthood was not predicted by externalizing problems or prior substance use in either the CDP or FT sample. The prevalence of problems with illicit drug use in this community sample during established adulthood was relatively small compared to problems with alcohol and cannabis use, as would be expected from prior epidemiological research (McCabe et al., 2021). An important direction for future research will be to use larger, nationally representative samples to fully understand predictors of illicit substance use with samples large enough to accommodate low base rates of problematic illicit substance use as well as to understand whether and how predictors differ in the full range of population-representative subgroups.

A second implication of our findings is that early interventions that seek to prevent alcohol and cannabis misuse might be best served by reducing externalizing behavior problems. When time and resources are scarce and difficult decisions about what to prioritize in interventions must be made, questions often arise about the extent to which programs should target particular risk factors (Miller & Hendrie, 2008). Our findings do not imply that risk factors that were not significantly associated with substance misuse in established adulthood above and beyond all the other risk factors in the models were unimportant, as developmental cascade models have demonstrated that early risk factors can cascade into other more temporally proximal risk factors that ultimately predict substance use (e.g., Dodge et al., 2009; Eiden et al., 2016; Otten et al., 2019). However, the findings do suggest that above and beyond other risk factors in adolescence and early adulthood, including prior substance use, externalizing behaviors in early adulthood continue to predict alcohol and cannabis misuse in established adulthood, highlighting the utility of targeting externalizing problems in prevention and intervention programs for substance misuse.

A third implication of our findings is that prevention and intervention efforts will be well served by better understanding specific predictors of specific substance use in specific populations, consistent with the specificity principle in developmental science (Bornstein, 2017). Although we found some consistencies across the CDP and FT samples and across predictors of different types of substance misuse, we also found differences. Future research to delve into explanations for inconsistencies across demographic groups, developmental periods, and other factors will be important for tailoring interventions to address the specific needs of specific individuals at specific points in their development.

Conclusions

The present study built on previous prospective longitudinal studies of predictors of adult substance use by taking a long-term developmental approach examining a wide range of adolescent and early adulthood predictors of problematic alcohol, cannabis, and other substance misuse in established adulthood in two independent samples. In both samples, compared to problematic cannabis use and other substance use, problematic alcohol use in established adulthood was predicted by more of the predictors from adolescence and early adulthood, which included gender, race/ethnicity,

https://doi.org/10.1017/S0954579422000670 Published online by Cambridge University Press

SES, internalizing problems, externalizing behaviors, parental monitoring, parent-adolescent relationship quality, parental involvement, family stress, peer substance use, own substance use, poor physical health, and pain. Individuals' own prior substance use and externalizing behaviors in early adulthood were the only consistent predictors of subsequent alcohol and cannabis misuse across samples; other predictors were specific to the sample and type of substance misuse. Taken together, the findings suggest that prevention efforts might benefit from being tailored to address risk factors for specific substances but also that prioritizing prevention of externalizing behavior problems holds promise for predicting both alcohol and cannabis misuse in established adulthood.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579422000670

Funding statement. The Fast Track project has been supported by National Institute of Mental Health (NIMH) Grants R18 MH48043, R18 MH50951, R18 MH50952, R18 MH50953, K05MH00797, and K05MH01027; National Institute on Drug Abuse (NIDA) Grants DA016903, K05DA15226, RC1DA028248, and P30DA023026; National Institute of Child Health and Human Development Grant R01 HD093651; and Department of Education Grant S184U30002. The Center for Substance Abuse Prevention also provided support through a memorandum of agreement with the NIMH. Additional support for this study was provided by a B.C. Children's Hospital Research Institute Investigator Grant Award and a Canada Foundation for Innovation award to Robert J. McMahon. The Child Development Project has been funded by grants MH56961, MH57024, and MH57095 from the National Institute of Mental Health, HD30572 from the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development, and DA016903 from the National Institute on Drug Abuse.

Conflicts of interest. None.

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