A developmental cascade model for early adolescent-onset substance use: the role of early childhood stress

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ABSTRACT

Backgrounds and aims Despite the link between stress and addictive behavior in adulthood, little is known about how early life stress in families predicts the early emergence of substance use in adolescence. This study tested a developmental cascade model, proposing that early stressful life events and negative parent–child interaction covary, and both disrupt the refinement of inhibitory control, which evolves into problem behavior in middle/late childhood and subsequent substance use exploration in early adolescence. **Methods** Data came from the Early Steps Multisite study, a community sample of at-risk families in the metropolitan US areas of Pittsburgh (Pennsylvania), Eugene (Oregon) and Charlottesville (Virginia) with children aged 2 years at the start of the study and 14 years at the last measurement ($n = 364$). Structural equation modeling was used to test the proposed model. **Results** Early stressful life events and negative parent–child interaction assessed at ages 2–5 were negatively related to inhibitory control at ages 7 and 8. Low levels of inhibitory control were prognostic of childhood problem behavior at ages 9 and 10. Finally, late childhood problem behavior was associated with substance use at age 14. Parental drug use was directly related to substance use at age 14. **Conclusions** Early life stress may disrupt child inhibitory control, which can cascade into behavioral and peer problem behavior in childhood and, in turn, heighten the risk for early adolescent substance use.

Keywords Adolescence, cascade model, childhood stress, development, inhibitory control, substance use.

INTRODUCTION

Substance use before age 15 years is the most robust predictor of substance dependencies and abuse by late adolescence and adulthood [1–3]. Furthermore, early substance use has several other potential negative consequences. First, early substance use is associated with abnormalities in brain functioning, including poorer neurocognitive performance, changes in white and gray matter volume and abnormal neuronal activation patterns [4]. Secondly, early substance use is associated with higher rates of depressive symptoms, depression and suicide attempts [5]. Thirdly, it is associated with driving while intoxicated [6], leading to a greater risk for motor vehicle accidents. Finally, early adolescent substance use undermines academic performance and increases school absences and the risk of dropping out of high school [7].

One of the best predictors of early onset substance use is deviant behavior. Youth with a history of antisocial behavior and deviant peer involvement are more likely to initiate alcohol, tobacco and cannabis use by age 15 [8]. To design effective prevention strategies, longitudinal research is necessary to identify malleable environmental risk factors to target and reduce the unfolding of risk over development. In developmental cascade models, risk factors assessed in early childhood are hypothesized to foster increased exposure to risk processes assessed in early, then later middle childhood, and ultimately to adolescent behavior. Previous research has tested a

developmental cascade model aimed at adolescent substance use [9]. In this longitudinal study, early parenting and contextual stress at age 5 was prognostic of peer difficulties during middle childhood, which predicted deviant peer involvement and, in turn, later adolescent substance use. Of particular interest was that family stress was presented as a non-specific risk factor underlying the unfolding of risk from childhood through adolescence. As in several developmental studies [10], Dodge *et al.* [9] posited family stress as a disruptor of parenting that leads to early child problem behavior and peer deviance, both of which are predictive of developing dependence on alcohol, tobacco and marijuana [11]. The present study builds upon this research with a focus on a potential underlying mechanism in the process in which early childhood family stress can influence the cascade from minor childhood difficulties to early adolescent drug use.

A critical factor deserving inclusion within a developmental cascade model aimed at substance use is the disruption of children's self-regulation [12,13], the core of which is inhibitory control [14]. Inhibitory control focuses on the ability to actively disengage from a dominant (i.e. rewarding) response to achieve a goal. It is thought that early childhood may be a critical period in which adverse life events (e.g. death in the family, divorce, drug use by a parent) [15] and correlated poor parenting practices [16] undermine refinement of the children's inhibitory control. Acute stress activates the hypothalamic–pituitary–adrenal (HPA)-axis and the secretion of cortisol, as well as the peripheral catecholamine system, which are all essential for survival [17]. However, when early childhood life events exceed the child's coping resources, it can undermine the child's development of affective and cognitive control functioning [18], attenuating executive control and behavior inhibition [17]. In a similar way, neglect and/or maltreatment establish environment conditions that create a state of chronic over-arousal for the child, further undermining refinement of self-regulatory functions [19]. Additionally, evidence indicates that parents' positive behavior support and low levels of coercive exchanges promote the emergence, growth and refinement of self-

regulation in general and inhibitory control in particular in early childhood [20].

Strong evidence indicates that low levels of inhibitory control in early and middle childhood lead to development of problem behavior during late childhood and adolescence [21,22]. Children with low levels of inhibitory control are less capable of delaying gratification [23] and are therefore more likely to ignore conventions and rules. In childhood, youth with antisocial behavior begin to cluster together, providing mutual reinforcement and engaging in rewarding activities and behaviors such as early drug experimentation [24]. In this way, low levels of inhibitory control may underlie frequent engagement in problem behavior and affiliation with peers who provide abundant reinforcement for breaking rules [25]. The key point of the developmental cascade framework is that early problem behavior disrupts normative development which, in turn, predicts clustering into high-risk peer environments [26]. Indeed, when examining middle childhood antecedents to early substance use onset in males, antisocial behavior, academic skill deficits and deviant peer involvement significantly predicted marijuana use by age 15.

The present study

Our study tests a more nuanced developmental substance use cascade model using a community sample of children and families who are at risk. Specifically, we focus on examining inhibitory control and deviant behavior as potential underlying mechanisms that link the association between childhood life stress, negative parent–child interaction and the onset of early adolescent substance use (see Fig. 1). Based upon previous studies we hypothesize that adverse life events and a negative parent–child interaction (i.e. the extent to which the overall interaction between parent and child can be described as dyadic coercion) in early childhood will predict lower child inhibitory control which, in turn, is associated with late childhood deviance. Higher late child deviance would predict greater early adolescent substance use. We also hypothesized that parent's drug use in the child's early childhood could be indirectly associated with early adolescent substance use through
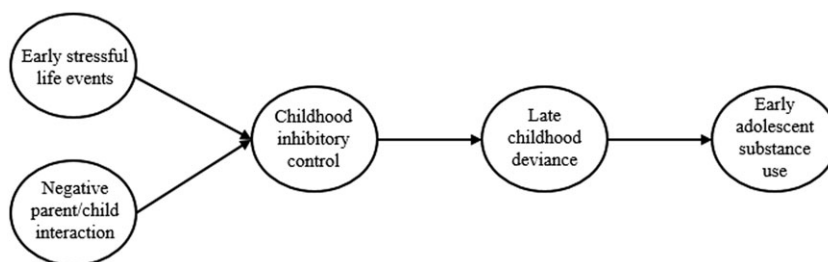

Figure 1 Developmental cascade model for the effects of early stressful life events and negative parent–child interaction on early adolescent substance use

the aforementioned pathway as an additional life stressor, or directly associated with early substance use as a genetic effect or social learning and modeling.

METHODS

Participants

The present study utilizes data from the Early Steps Multisite (ESM) study, a randomized controlled trial conducted in the United States, investigating the prevention of children's behavioral problems [27]. A detailed description of the Family Check-Up (FCU) intervention used in the ESM study is available elsewhere [28,29], but this report does not focus on the FCU. The entire sample included 731 children and their primary caregivers who were recruited between 2002 and 2003. More than 96% of the primary caregivers at the initial assessment were biological mothers; in all other cases, they were non-maternal custodial caregivers. Participants were recruited from Women, Infants and Children (WIC) Nutritional Supplement Centers in the metropolitan areas of Pittsburgh (Pennsylvania), Eugene (Oregon) and Charlottesville (Virginia). Families were eligible to participate if they had a child aged between 24 and 35 months [mean = 29.9 months, standard deviation (SD) = 3.2] and if they met risk criteria in at least two of three risk domains for future behavioral problems. Specifically, risk criteria for recruitment were defined at 1 SD or above the normative range on several screening measures within these three domains: (a) child behavior (conduct problems, high-conflict relationships with adults); (b) family problems (maternal depression, daily parenting challenges, substance use problems, teen parent status); and (c) socio-demographic risk (no more than 2 years post-high school education and low family income). Children who met criteria based on family problems and socio-demographic risk were also required to have above-normative levels of externalizing problems to ensure significant levels of problem behavior.

Of the 1666 parents approached at WIC sites across the three study sites and who had children in the appropriate age range, 879 families met the eligibility requirements; of these, 731 agreed to participate. No differences in family problems, socio-demographic risk or problem behavior appeared between those who agreed to participate and those who did not. Of the 731 families (49% female children), 272 (37%) were in Pittsburgh, 271 (37%) in the Eugene site and 188 (26%) in Charlottesville. Of the 731 families, 662 (91%), 627 (86%), 621 (85%), 568 (78%), 565 (77%), 588 (80%), 573 (78%) and 592 (81%) participated in the follow-ups at the ages of 3, 4, 5, 7, 8, 9, 10 and 14 years, respectively. A total 367 of the 731 families were given the FCU intervention. These families were excluded from analyses in the present study, as the intervention could affect the mediators and outcomes considered, leading to a sample of 364 families used for the present study.

Procedure

Assessments took place during home visits when children were aged 2–10 (except age 6) and age 14. Families received compensation for their effort and time.

Measures

Early stressful life events and parental substance use

When children were aged 2, 3, 4 and 5, parents answered this question: 'Which of the following factors has had an impact on your family's wellbeing, or ability to make healthy changes?' [30]. Among the 18 factors listed, items were included, such as a recent remarriage or a death in the family (see Supporting information, Appendix S1 for the complete list). During the four successive years a sum score of stressful events was calculated over all items, except for parental substance use, because this could be genetic or a consequence of modeling or socialization. Hence, this item was included as a separate factor in the model. Higher sum scores on the early stressful life events measure indicate the experience of a higher number of early stressful life events.

Negative parent–child interaction

To assess the quality of the parent–child relationship we looked the parent–child interaction. At ages 2–5, videotaped interaction tasks involving the child and the parent were coded using the Relationship Affect Coding System (RACS [31]). The RACS is a micro-social coding system that reflects the three dimensions of behavior (i.e. verbal, physical and affect) for each of the family participants simultaneously. The cues used for code selection are based on facial expression, vocal tone and non-verbal cues, such as body posture and/or orientation. The RACS coding was recorded using Noldus Observer XT, version 11.0 (Noldus Information Technology, Leesburg, VA, USA, 2012), which allows for continuous coding of an interaction. At any given moment during an interaction, the parent and child can have one code (or event or state) recorded from each of these three data streams. Because there are three simultaneous data streams for each participant in the interaction tasks, six behavior clusters are created that summarize the three data streams for each person in the interaction. The six behavior summary clusters are positive, neutral, directives, negative, no talk and ignore (for more details see [32]). Behavior clusters observed at each time-point link the child's and parent's behavior at the same time, thereby arriving at dyadic states [33]. Using this approach, it is possible to calculate durations and frequencies for the dyadic states. For the purposes of this study, summary scores were created for observed dyadic coercive engagement (the duration of time that the caregiver and child was engaged in negative

behavior and for positive engagement). These scores were used to construct latent factors of negative parent–child interaction at the ages of 2, 3, 4 and 5 years. Finally, these five latent constructs were used to construct an overall latent construct Negative Parent–Child Interaction ages 2–5. Reliability coefficients were in the 'good' to 'excellent' range [34,35].

Childhood inhibitory control

At the child's age 7, inhibitory control was measured by using the 13-item inhibitory control subscale from the Child Behavior Questionnaire (CBQ) [36]. Parents rated each item on a seven-point Likert scale ranging from 'extremely untrue' to 'extremely true'. Sample items include: 'My child can lower his/her voice when asked to do so' and 'My child has a hard time following instructions'. The CBQ has shown adequate reliability and validity [36]. At age 8, items from the CBQ were adapted to apply to older children, resulting in the eight-item inhibitory control subscale of the parent-report Temperament in Middle Childhood Questionnaire (TMCQ) [37]. Sample items include: 'My child has a hard time stopping him/herself when told to do so' and 'My child likes to plan carefully before doing something'. Respondents rated each item on a five-point Likert scale, ranging from 'almost always untrue' to 'almost always true'. Alpha reliabilities were between 0.64 and 0.80. Item scores were calculated such that higher scores reflect more inhibitory control.

Late childhood deviance

We used the SRD (Self Report of Deviance) with 27 questions to assess child deviancy at ages 9 and 10 [26]. On a three-point scale (0 = never, 1 = once/twice and 3 = more often), this questionnaire asks children about their behaviors during the past year (e.g. 'In the past year, have you on purpose broken or damaged or destroyed something belonging to your parent or other people in your family?' and 'In the past year, have you taken something from a store without paying for it?'). Alpha reliabilities were 0.67 and 0.69, respectively.

In addition to their own problem behavior at ages 9 and 10, children answered questions about the friends with whom they spent the most time and estimated how many of those friends engaged in problem behaviors (e.g. 'How many of your peers have used marijuana?' and 'How many of your peers have dropped out of school?'). Children responded to these questions on a five-point scale (0 = none; 1 = one, 2 = two, 3 = three, 4 = four or more). Internal consistency for the 11-item scale was 0.76 and 0.73, respectively, for ages 9 and 10 [38]. Higher scores indicate more deviant behavior.

Early adolescent substance use

Substance use onset at age 14 was established if children responded 'yes' to one of these questions: 'Did you use tobacco, alcohol, marijuana, or any other drug?' [8].

Covariates

Family income and children's gender were included as covariates in the model loading on all main study variables. Early adolescent substance use may be a consequence of social modeling, probably from their parents. Therefore, parent recent substance use at age 10, which was established if parents responded positively on questions about alcohol use, cannabis use, daily tobacco use or use of hard drugs, was included as control variable of early adolescent substance use. Finally, inhibitory control at ages 7 and 8 and child deviance at ages 9 and 10 were controlled for by inhibitory control [36] and oppositional defiant problems at age 2 [26].

Data analysis

First, we computed descriptive statistics. Secondly, using Mplus version 7.4 [39], we employed structural equation modeling to test the hypothesized mediation model. To determine model fit, we used the comparative fit index (CFI, critical value $\geq$ 0.90; [40], the Tucker Lewis Index (TLI, critical value $\geq$ 0.90; [41]) and the root mean squared estimate of approximation (RMSEA, critical value $\leq$ 0.08 [36]). As we had a mix of categorical and continuous variables, we used the weighted least squares means and variances estimator (WLSMV [42,43]). Finally, using a Bayesian mediation analysis in Mplus [44], we tested whether inhibitory control and problem behavior during middle/late childhood would mediate the effects of adverse life events, parent drug use and negative parent–child interaction on early substance use. Attrition analyses, by means of logistic regression, showed no systematic relationship between covariates, the main outcome measure and attrition. Hence, missing data were handled by full information maximum likelihood (FIML) based on the missing at random assumption [45].

RESULTS

Descriptive statistics

More than two-thirds of those families enrolled had an annual income of less than $20 000; the average number of family members per household was 4.5 (SD = 1.63). Forty per cent of the sample had a high school diploma (HSD) or general educational development (GED) equivalency and an additional 35.8% had 1–2 years of post-high school training. Across sites, the children belonged to the following racial groups: 26.9% African American, 50.8%

European American, 13.2% biracial and 9.1% other races (e.g. American Indian, Native Hawaiian). In terms of ethnicity, 13.4% of the sample reported being Hispanic American. At age 14, 22% of youth reported substance use, which was driven by alcohol, tobacco and cannabis. Prevalence for the different substances for this sample were sometimes more than twofold compared to that of the normative age norm in the United States (e.g. marijuana [46]). Skewness (cut-off < 2) and kurtosis (cut-off < 7) of most variables fell within the acceptable range [47], except child deviant behavior and peer deviant behavior at ages 9 and 10. Multivariate outlier analyses identified no influential cases.

Factor loadings of the latent variables

Regarding negative parent–child interaction at ages 2–5, latent constructs were developed of positive engagement and coercive engagement. Factor loadings (λs) for the eight measures ranged from 0.64 to 0.85. On a second level, these latent constructs were used as indicators for the overall parent–child interaction measure (range λs = 62–0.82). The inhibitory control latent variable consisted of two indicators (i.e. assessments at ages 7 and 8) with $\lambda = 0.80$ and $\lambda = 0.76$, respectively. Regarding late childhood deviance at ages 9 and 10, two latent constructs were developed of own deviancy ($\lambda = 0.81$, $\lambda = 0.73$) and peer deviancy ($\lambda = 0.61$, $\lambda = 0.52$). On a second level, these two latent constructs were used as indicators for the overall late childhood deviance measure ($\lambda = 0.71$, $\lambda = 0.87$). All factor loadings were higher than 0.50 and all were statistically significant. Supporting information, Appendix S2 shows the CFI for pooled measurement models (i.e. all factor loadings).

Correlations between the study variables

Table 1 shows the zero-order correlation coefficients between the main study variables. What stands out are the positive and consistent relations between early stressful life events and positive engagement at ages 3 and 4, parent drug use at ages 2–5 and negative relations between early stressful life events and inhibitory control at ages 10 and 11. In addition, there were significant negative relations between inhibitory control and own and peer deviance. Own deviancy at age 9 was positively related with early substance use.

Structural equation model

Figure 2 presents findings of the structural equation model. Model fit indices suggested a good fit to the data, $\chi^2_{(162)} = 260.827$, $P = 0.000$; CFI = 0.921; TLI = 0.888; RMSEA = 0.041.

Early stressful life events and negative parent–child interaction were negatively associated with inhibitory control in childhood ($P < 0.01$ and $P < 0.01$, respectively). Childhood inhibitory control was negatively associated with later problem behavior ($P < 0.01$) and finally, late childhood deviance was prognostic of early substance use at age 14 ($P < 0.01$). Drug use by parents was not related to any of the mediating variables, but positively associated directly with early substance use ($P < 0.01$). Regarding the covariates, lower income was associated with lower levels of parental substance use ($B = 0.12$, $P < 0.05$) and being a girl was associated with higher levels of inhibitory control ($B = 0.17$, $P < 0.01$). Together, the included variables in the model explain 12.8% of the variance. Analyses in the data set that only included completers on substance use were similar.

Mediated effects were obtained from Mplus with Bayesian mediation analysis with a Bayes estimator. There were significant mediated effects from early stressful life events on early adolescent substance use via childhood inhibitory control and late childhood deviance (estimate = 0.015; 95% CI = 0.001, 0.043) and from negative parent–child interaction on early adolescent substance use via inhibitory control and late childhood deviance (estimate = 0.01; 95% CI = 0.001, 0.039).

DISCUSSION

This study examined a developmental model for early adolescent substance use. We hypothesized that stressful life events, parental drug use and negative parent–child interaction in early childhood would increase the risk of early adolescent substance use via disruptions in inhibitory control in early childhood and elevation of late childhood deviance.

In line with the findings of previous studies [16], we found that early stressful life events and negative parent–child interaction significantly predicted inhibitory control at school age, while controlling for early childhood inhibitory control. Drug use by parents during early childhood was not related to the mediators. However, it was directly related to early substance use.

The findings of the present study extend existing knowledge on the impact of early exposure to family stress on child development in at least two main ways. First, they support the claim that inhibitory control can be malleable and susceptible for environmental influences [48–50]. In particular, environmental factors that cause stress and exceed available coping resources may undermine development of areas in the brain responsible for inhibitory control, such as the prefrontal cortex. Only recently, research has started to provide clues as to why environmental factors can disrupt prefrontal cortical functioning. For instance, studies have shown that the effects of stress on the prefrontal cortex are mediated by disinhibiting intracellular signaling pathways [51]. Future research

Table 1 Correlations between the main study constructs.

|	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. Early stressful life events age 2–5	0.10	0.13*	0.18**	0.08	-0.08	-0.00	-0.03	0.09	0.26**	-0.17**	-0.16**	0.03	0.03	0.03	0.10	-0.05	-0.02	-0.07
2. Positive engagement age 2	–	0.47**	0.37**	0.39**	-0.62**	-0.32**	-0.25**	-0.27**	0.06	0.05	0.07	-0.14*	-0.14*	-0.18	-0.12	-0.04	0.10	0.00
3. Positive engagement age 3		–	0.53**	0.45**	-0.32**	-0.52**	-0.32**	-0.25**	-0.00	0.02	0.07	-0.13	-0.09	-0.12	-0.08	-0.03	-0.03	0.01
4. Positive engagement age 4			–	0.30**	-0.27**	-0.19**	-0.45**	-0.12**	0.07	0.07	0.08	-0.02	-0.03	-0.04	-0.11	0.02	-0.05	0.10
5. Positive engagement age 5				–	-0.18**	-0.20**	-0.15**	-0.56**	0.00	0.13*	0.07	-0.16*	-0.09	-0.13	-0.07	0.06	-0.01	0.05
6. Coercive engagement age 2					–	33*	41**	26*	-0.08	-0.11	-0.09	0.08	0.14*	0.14*	0.12*	0.04	-0.10	-0.03
7. Coercive engagement age 3						–	0.30**	0.33**	-0.01	-0.03	-0.09	0.07	0.13*	0.15*	0.01	0.05	-0.06	0.04
8. Coercive engagement age 4							–	0.19**	-0.08	-0.14*	-0.02	0.09	0.09	0.12	-0.03	0.14*	-0.09	-0.13*
9. Coercive engagement age 5								–	0.06	-0.15**	-0.09	0.10	0.03	0.12	0.02	0.01	-0.06	-0.08
10. Parent drug use age 2–5									–	0.01	-0.15*	0.02	0.02	0.02	-0.04	0.13***	0.02	-0.03
11. Childhood inhibitory control age 6										–	0.57**	-0.14*	-0.19**	-0.14*	-0.15*	0.07	0.17**	0.06
12. Childhood inhibitory control age 7											–	-0.17**	-0.18**	-0.06	-0.15*	-0.04	0.21**	0.02
13. Child deviant behavior age 8												–	0.52**	0.34**	0.07	0.07	-0.18**	-0.01
14. Child deviant behavior age 9													–	0.23**	0.28**	0.18**	-0.03	-0.09
15. Peer deviant behavior age 8														–	0.33**	0.06	-0.06	-0.06
16. Peer deviant behavior age 8															–	0.07	0.05	-0.08
17. Early adolescent substance use age 14																–	0.03	0.08
18. Gender																	–	0.06
19. Income																		–

*P < 0.05; **P < 0.01; ***P = 0.054.
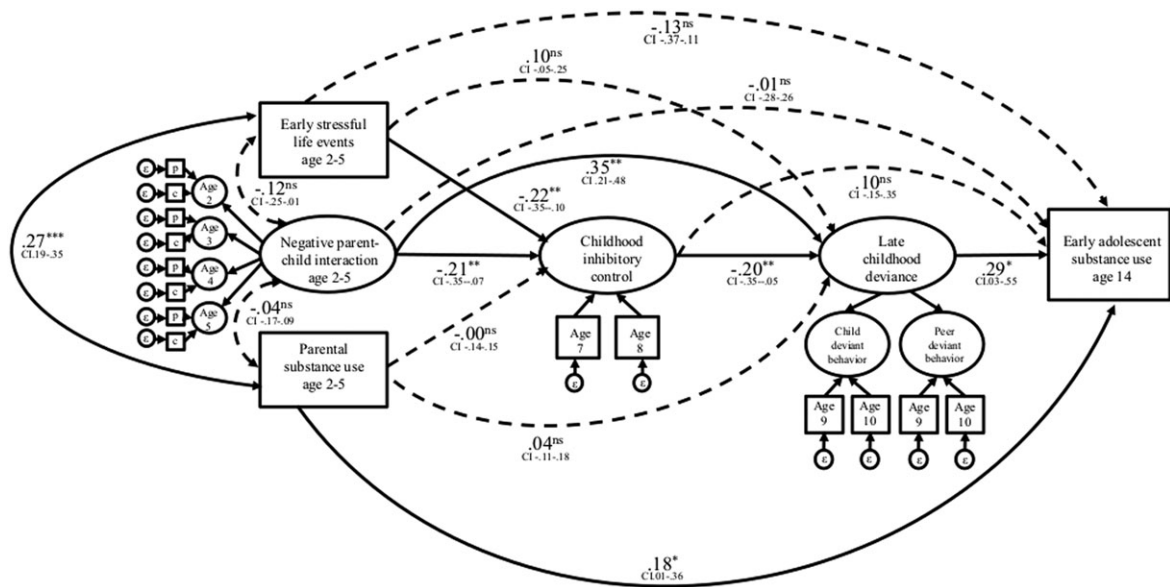
Figure 2 Findings from the ecological model for early adolescent substance use. Numbers are standardized estimates. *$P < 0.05$; **$P < 0.01$; ***$P < 0.001$

should focus on these and other potential underlying mechanisms. The direct link between parental drug use and early substance use may point to a genetic effect [52] or aspects of early learning drug and alcohol use [53,54]. Currently, early childhood prevention programs do not directly intervene and treat parental drug and alcohol problems, but rather advise against heavy use in the presence of children [29]. Perhaps early family-based prevention would benefit from an emphasis on the disruptive effect of heavy alcohol and/or drug use to family formation and long-term youth health and wellbeing. Unexpectedly, there was no significant correlation between early life stress events and the latent construct of negative parent–child interaction. It is possible that the association between early life stress events and parent–child interaction is curvilinear; perhaps the relationship between the two is significant only after passing a certain threshold of number of early life stress events.

In addition to the focus on the family, these data support an emphasis on prevention of problem behavior in general and improving self-regulation capacity specifically. Several evidence-based interventions suggest the benefits of such a focus; for example, the emphasis on social influence skills in the Life Skills Training universal strategy [55] as well as the Coping Power program for high-risk youth [56]. However, some caution is in order when conducting group interventions that focus on developing self-regulation in early adolescence when the participants are high risk [57,58].

Strengths and limitations

This study had two key methodological strengths: we longitudinally observed participants from early childhood to middle adolescence and all variables were measured by multiple indicators and were longitudinal. Nevertheless, the results should be interpreted in the context of some limitations. First, in addition to examining negative parent–child interaction as a potential stressor, we examined early stressful life events by summing all the events under the assumption that each event has equal magnitude of impact on one's life. However, this may not be true, because the experience of each life event is subjective. Secondly, our sample may have some limits in generalizing the present findings, as families were recruited in nutritional supplement centers and some of the participants were sampled based on existing conduct problems. Thus, it is important to replicate our findings in other samples that are more generalizable. Thirdly, although we emphasize the importance of an ecological approach, in this study we primarily focused on assessing the parent and child dynamics. Future studies should employ more diverse ecological assessments of inhibitory control, childhood deviance and substance use based upon teacher- and peer-reports, direct observations and drug screening tests. Finally, future studies should also focus on other potential mechanisms in this model (e.g. executive control, intelligence). This would also contribute to an increase of the predictive value of the model.

Conclusion

In conclusion, these study results highlight that early childhood life events and negative parent–child interaction can indirectly influence early substance use onset in adolescence via changes in inhibitory control and early adolescent deviance, while drug use by parents during

childhood is directly related to early substance use onset in adolescence. These observed longitudinal processes suggest important prevention implications for high-risk populations who are much more vulnerable to being exposed to chronic family stress. Although recent efforts to prevent substance use focus on parenting practices, data from this study suggest that an ecologically focused approach to family support may be useful for prevention, with attention given to reducing stress, poverty and the associated family disruption while also supporting improved parenting skills.

Declaration of interests

None.

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Appendix S1 Early stressful events that were included in the sum score.

Appendix S2 The pooled factor analysis of the measurement model.