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Regular Article

Parent-child separation and intergenerational transmission of substance use and disorder: Testing across three generations

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Abstract

There is evidence for intergenerational transmission of substance use and disorder. However, it is unclear whether separation from a parent with substance use disorder (SUD) moderates intergenerational transmission, and no studies have tested this question across three generations. In a three-generation study of families oversampled for familial SUD, we tested whether separation between father (G1; first generation) and child (G2; second generation) moderated the effect of G1 father SUDs on G2 child SUDs. We also tested whether separation between father (G2) and child (G3; third generation) moderated the effect of G2 SUDs on G3 drinking. Finally, we tested whether G1-G2 or G2-G3 separation moderated the mediated effect of G1 SUDs on G3 drinking through G2 SUDs. G1 father-G2 child separation moderated intergenerational transmission. In families with G1-G2 separation, there were no significant effects of father SUD on G2 SUD or G3 drinking. However, in nonseparated families, greater G1 father SUDs predicted heightened G2 SUDs and G3 grandchild drinking. In nonseparated families, G1 father SUDs significantly predicted G2 SUDs, which predicted G3 drinking. However, G2-G3 separation predicted heightened G3 drinking regardless of G2 and G1 SUDs. Parental separation may introduce risk for SUDs and drinking among youth with lower familial risk.

Keywords: disorder; parent-child; separation; substance; use

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There is robust evidence for the intergenerational transmission of substance use and substance use disorders (SUDs) from parents to offspring, through gene-environment mechanisms (e.g., Chassin et al., 2013; Patrick et al., 2014; Sher et al., 1997). Additionally, a number of studies have identified moderators that may increase or decrease risk for intergenerational transmission of SUDs and substance use. For example, prior research has found evidence for familial characteristics as moderators of transmission (e.g., Castro et al., 2006; Hussong & Chassin, 1997). One such familial characteristic that may moderate intergenerational transmission of SUD is parent-child separation. Numerous studies indicate that separation is a robust risk factor for youth substance use and SUD (e.g., Arkes, 2013; Davis & Shlafer, 2017; Hamdan et al., 2013; Räikkönen et al., 2011). Many children are exposed to both parental separation and SUD, because these risk factors tend to co-occur (Becoña et al., 2012). Therefore, it is important to characterize the impact of both parental separation and SUD on offspring substance use outcomes.

In addition to serving as risk factors for offspring substance use outcomes, it is plausible that parent SUD and parent-child separation interact. Most studies testing interactions between parental separation and SUD on youth substance use have examined the moderating effects of parental divorce rather than other forms of parent-child separation. Findings have been conflicting, with three studies finding only main effects (Grant et al., 2015; Holst et al., 2020; McCutcheon et al., 2018), and others finding interactions (Jackson et al., 2016; Thompson et al., 2008, 2013; Waldron et al., 2014; Waldron et al., 2018; Windle & Windle, 2018). Based on studies of similar risk factors, this interaction is likely to take one of three forms: "vulnerable and reactive," "protective-stabilizing," and "vulnerable-stable" (Luthar et al., 2000; see Figure 1).

"Vulnerable and reactive" interaction

If parental SUD and separation interact to predict offspring SUD, one possibility is that they exert a "vulnerable and reactive" interaction effect, in which both risk factors work in tandem to compound risk (Luthar et al., 2000). In other words, parental SUD may exert a stronger effect on offspring SUD among youth who have also experienced separation, which suggests that parental separation *heightens* risk for intergenerational transmission of SUD.

There are several plausible reasons for a stronger effect of parental SUD on offspring SUD among youth who have also experienced separation. First, it is possible that the combination of these two risk factors operates through a diathesis-stress model (Cicchetti & Toth, 1998), such that genetic propensity for SUD interacts with environmental adversity (i.e., parental separation and related stressors) to promote youth substance use and disorder. Although parental separation constitutes risk for substance use among all youth, those youth with genetic propensity for substance

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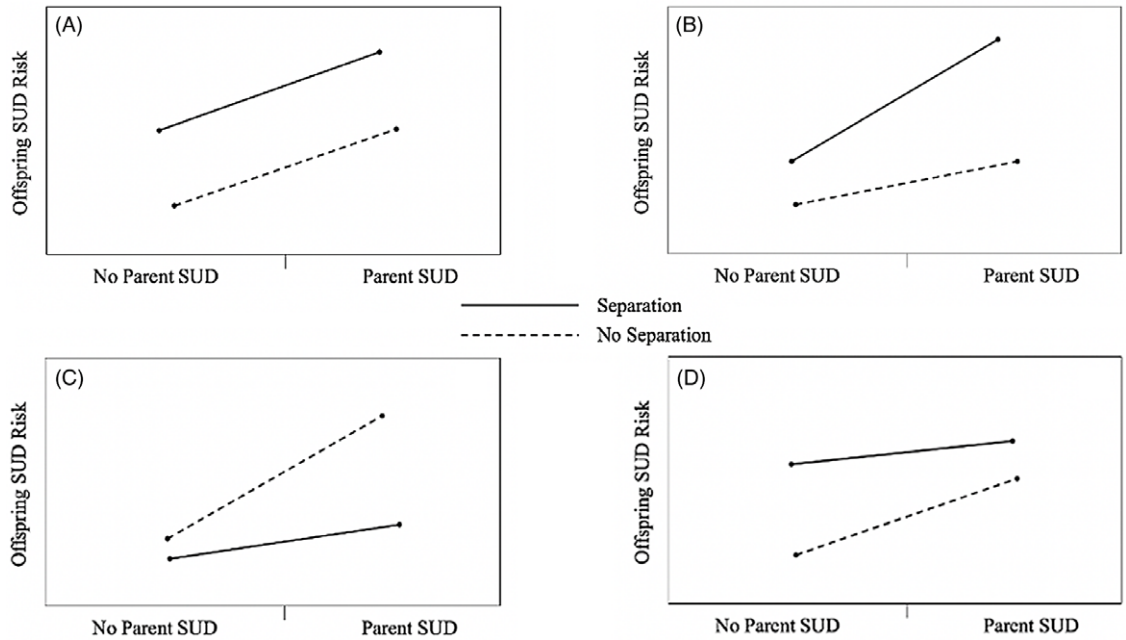

Figure 1. Potential combined effects of parental SUD and separation on offspring SUD/ substance use risk, informed by prior literature: (A) no interaction, main effects only; (B) vulnerable and reactive interaction; (C) protective-stabilizing interaction, (D) vulnerable-stable interaction.

use (indexed by parental SUD) may be at greater risk for substance involvement following parental separation. An alternative possibility is that youth exposed to parental SUDs experience a variety of adverse early environmental stressors (Solis et al., 2012), which then increases their sensitivity to the negative effects of later adverse events, such as parental separation. This "stress sensitization" framework (McLaughlin et al., 2010), would suggest that children dually exposed to parental separation and SUD fare the worst with respect to substance use outcomes. Third, it may be that children with parental SUD have fewer protective resources than youth without parental SUD to buffer the effect of parental separation on their own substance use. Indeed, the "vulnerable and reactive" interaction effect is the type of interaction most supported by prior literature. Parental SUD and separation have been found to compound risk for the initiation of substance use, adolescent substance use, and development of SUD (Jackson et al., 2016; Thompson et al., 2008, 2013; Waldron et al., 2014; Windle & Windle, 2018).

"Protective-stabilizing" interaction

Despite these findings, it is also possible that parent-child separation is *protective* in the context of parental SUD. A "protective-stabilizing" interaction effect (Luthar et al., 2000) would indicate that parental SUD confers less risk for offspring SUD when offspring are separated from a parent, suggesting that parental separation protects against the intergenerational transmission of SUD (see Figure 1, panel C). This would presumably occur by reducing the child's environmental exposure to parental SUD and related risk factors. The highest-risk group would be those with parent SUD but no parent-child separation.

To our knowledge, only one study has demonstrated a protective-stabilizing interaction between parental SUD and separation in predicting offspring substance use. Waldron et al. (2018) found that parental SUD and separation both independently predicted earlier-onset drinking in adolescents. However, separation from mothers with alcohol use disorder (AUD) predicted a delayed onset of drinking. Examining parent risk factors other than SUD, a broader literature base has also revealed protective effects of parental separation on children's adjustment. For example, Jaffee et al. (2003) found that children of antisocial fathers displayed fewer conduct problems if they spent less time living with their fathers. Additionally, others have found that paternal incarceration may decrease physical aggression in girls (Wildeman, 2010). Most pertinent to the present study, Osborne and Berger (2009) found that children living with fathers with SUD displayed higher levels of behavior and health problems than those whose father with SUD did not reside with them.

"Vulnerable-stable" interaction

A third possibility is that parent-child separation and parental SUD display a "vulnerable-stable" interaction effect (Luthar et al., 2000). In a vulnerable-stable interaction, parent SUD would increase risk for offspring without histories of parent-child separation but offspring exposed to parent-child separation would have heightened risk for SUD regardless of parental SUD. This pattern might occur because separation is a less expected stressor for more privileged youth or because of a "ceiling effect" of the impact of stressors among children with greater initial disadvantage, in this case among children with parent SUD (Turney, 2015). This form of interaction might also reflect two separate pathways into SUD: a genetically influenced pathway for individuals with familial risk for SUD and an environmentally influenced pathway (e.g., through separation) for those with less familial risk.

Although no research to date on offspring substance use outcomes has found this pattern of effect, several studies have found that lower-risk (e.g., higher socioeconomic status, White) youth experience greater harm from parental separation in other behavioral and socioemotional domains (Bosick & Fomby, 2018; Fomby et al., 2010; Mollborn et al., 2012; Womack et al., 2019; Räikkönen et al., 2011; Ryan et al., 2015).

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Interaction effects across three generations

Given that studies on the intergenerational transmission of SUD have recently begun to demonstrate transmission across three generations (e.g., Bailey et al., 2006; Hill et al., 2018), it is plausible that parent-child separation may also moderate transmission of SUD across three generations. Parent-child separation may moderate both (a) the G1 SUD to G2 SUD link, and (b) the G2 SUD to G3 alcohol use link in the three-generation mediation model.

Moderation of G1-G2 transmission

If parent-child separation moderates the effect of first-generation ("G1") SUD on second-generation ("G2") SUD, this effect may also carry over to the transmission of SUD or substance use to the third generation ("G3"). Previous research suggests a longitudinal mediated effect of G1 SUD to G2 SUD to G3 alcohol use. That is, separation of the G1 parent and G2 child may moderate the mediated effect of G1 grandparent SUD on G3 grandchild substance use through G2 parent SUD (see Figure 2, Model 2). Such a finding would provide important implications for SUD prevention by demonstrating that parent-child separation in the context of parental SUD not only provides risk or protection for child SUD, but also has downstream intergenerational effects on substance use on the child's G3 offspring.

In line with the three potential patterns of interaction previously outlined for separation as a moderator of intergenerational transmission of SUD from G1 parents to G2 children, these patterns may carry over to influence substance use among G3 grandchildren. For example, if separation of the G1 parent and G2 child amplifies risk for transmission of SUD from G1 to G2, it may then increase risk for substance use among the G2's offspring. Alternatively, if G1-G2 separation *protects* against transmission of SUD from G1 to G2, it may also reduce the later risk of substance use among G3 grandchildren. Finally, if G1-G2 separation only increases risk for SUD among G2s without parental SUD, it may act as a distal risk factor that "sets off" the intergenerational transmission of SUD from G2 to G3 in the absence of prior familial risk.

Moderation of G2-G3 transmission

If SUD is transmitted from the first generation to the second generation, but parent-child separation moderates the effect of second-generation ("G2") SUD on third-generation ("G3") SUD, subsequent transmission of SUD from G2 to G3 would be altered. Stated differently, separation of the G2 parent and G3 child may moderate the mediated effect of G1 grandparent SUD on G3 grandchild substance use or disorder through G2 parent SUD (see Figure 2, Model 3). Parent-child separation in the G2-G3 family may moderate previous generational patterns of substance use or disorder in the family in several ways. If SUD is transmitted from G1 to G2, and separation of the G2 parent and G3 child further amplifies risk for transmission of substance use and disorder from G2 to G3, G3 children may be at even greater risk. Conversely, if G2-G3 separation *protects* against transmission of substance use and disorder from G2 to G3 following previous transmission from G1 to G2, it may interrupt the intergenerational transmission to G3 children. Lastly, if G2-G3 separation only increases risk for substance use and disorder among G3 children without parental SUD, it may act as a risk factor that increases the probability of substance use or disorder despite low levels of prior familial risk.

Current study

In a three-generation, longitudinally followed sample of families with and without SUD, the present study had the following aims (see Figure 2).

Separation as moderator in G1-G2 family (Model 1a)

To test whether G1 father SUDs during G2 children's lifetimes (coded as 0 "none," 1 "AUD *or* drug use disorder (DUD)," 2 "AUD *and* DUD") and G1 father-G2 child separation increased the risk of G2 child lifetime SUDs. Because there were low rates of mother-child separation in the current study (9% in G1-G2 family and 12% in G2-G3 family), we focused on father-child separation. To test whether G1 father-G2 child separation moderated the effect of G1 SUDs on G2 SUDs. We predicted that G1 father SUDs and G1 father-G2 child separation would predict greater risk of G2 SUDs, but had no predictions about the form of interaction between separation and SUDs.

Separation as moderator in G2-G3 family (Model 1b)

To replicate this finding in the G2-G3 generation, by testing whether G2 father SUDs during G2 children's lifetimes and G2 father-G3 child separation increased levels of G3 child drinking. G3 drinking was used as the outcome variable because there were no available data on G3 SUD or drug use. We predicted that G2 father SUDs and G2 father-G3 child separation would be associated with greater G3 drinking, but had no predictions about the form of interaction between separation and SUDs.

Separation in G1-G2 family as moderator of indirect effect (Model 2)

We tested whether father-child separation in the G1-G2 family moderated the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. We predicted that G1-G2 separation would moderate this indirect effect but had no predictions about the form of moderation.

Separation G2-G3 family as moderator as moderator of indirect effect (Model 3)

We tested whether parent-child separation in the G2-G3 family moderated the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. We predicted that G2-G3 separation would moderate this indirect effect but had no predictions about the form of moderation. Importantly, this model focused on separation between G3 child and G2 parent (both mothers and fathers) because the "a path" in this moderated mediation model required G2s to be the biological children of G1 fathers but did not specify G2 sex (see Model 3 diagram in Figure 2).

Method

Sample

Participants were from a larger three-generation study of familial AUD (Chassin et al., 1992). The original cohort of participants included 454 families with an adolescent (G2) and his/her parents (G1). Each were interviewed over six waves of data, spanning from 1988 to 2010. The sample included 246 G2s with at least one biological, custodial parent with AUD and 208 G2s from demographically-matched comparison families (matched on family

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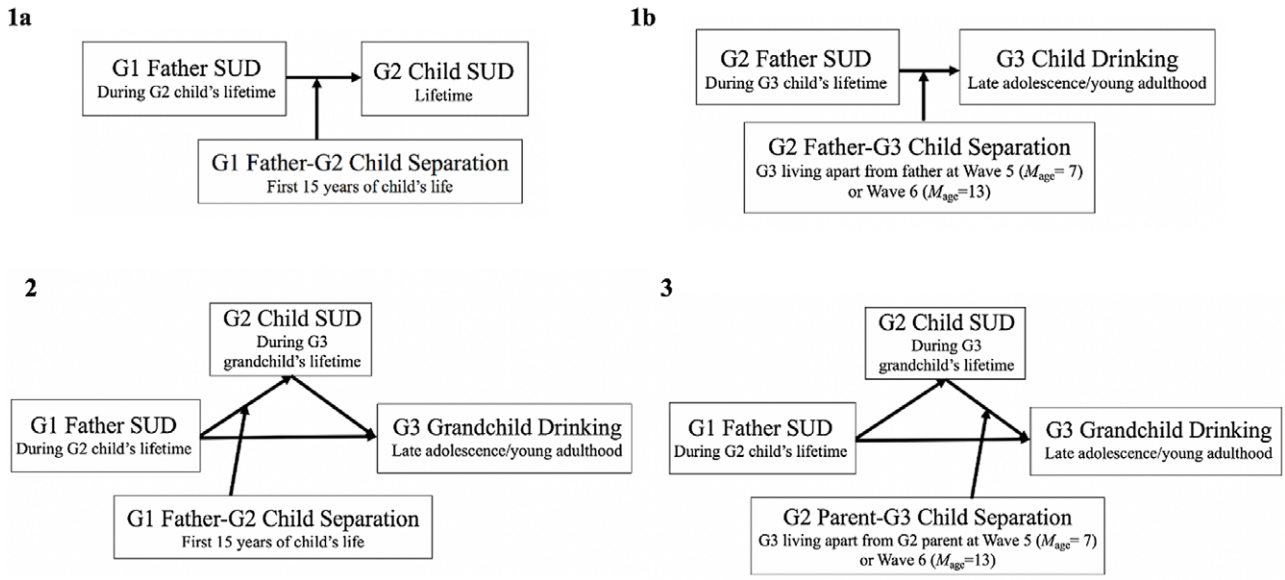

Figure 2. Study models testing parent-child separation as a moderator of the intergenerational transmission of substance use and disorder: (1a) Simple moderation model in G1-G2 family; (1b) Simple moderation model (replication) in G2-G3 family; (2) Moderated mediation model with parent-child separation in G1-G2 family; (3) Moderated mediation model with parent-child separation in G2-G3 family.

composition, ethnicity, socioeconomic status, and adolescent age) with no parental AUD. G2 siblings within the same age range ("age-eligible siblings" also between 11 and 15 years old at Wave 1) entered the study at Wave 4. At Wave 5, siblings outside the same age range ("age-ineligible siblings") were recruited. At Waves 5 and 6, assessments were conducted with generation 3 ("G3s"), the children of the G2s, and with G2s' partners (the G3's other parent). G3s also provided drinking data in adolescence and young adulthood (four surveys, from 2011 to 2021). Figure A in the supplemental material presents a timeline outlining waves of enrollment for each of these groups.

Participants were initially recruited through court records, community telephone screenings, and health maintenance organization (HMO) questionnaires. G1 parents were required to be of Latinx or non-Latinx Caucasian ethnicity, have a date of birth between 1926 and 1960, live in Arizona, and have at least one child between 10.5 and 15.5 years old. Additionally, at least one biological, custodial parent was required to meet criteria for lifetime alcohol abuse or dependence according to the DSM-III (American Psychiatric Association, 1980) or the Family History-Research Diagnostic Criteria (FH-RDC; Endicott et al., 1975). Comparison families (families in which neither biological nor custodial parents met DSM-III or FH-RDC criteria for lifetime alcohol abuse or dependence) were recruited through phone screenings from the same neighborhood as families with AUD. Recruitment procedures are described in greater detail elsewhere (Chassin et al., 1992).

The current study includes two subsamples of the total sample of G2 adults and their partners/spouses. Analyses for Model 1a (testing effects of G1 father SUD and separation on G2 SUDs) use Subsample 1, which includes all G2 children of G1s ($N = 829$). This subsample includes both original "target" G2s who were recruited at baseline and their siblings, who were recruited at Waves 4 and 5. However, G2s' partners and spouses were excluded due to lack of information about onset and recency of their parents' SUDs, precluding tests of the effect of parental SUD during their childhoods. We compared those who were included in Subsample 1 with those who were excluded (i.e., the G2s' partners/spouses). Those who were excluded were older and more likely to be male and to have been separated from their mothers and fathers.

Subsample 2 was used for Model 1b (testing effects of G2 father SUDs and separation on G3 child drinking) and Models 2 and 3 (models testing parent-child separation as a moderator of the effect of G1 SUD on G3 drinking through G2 SUDs). This subsample includes 347 cases consisting of one G2 adult who was the biological child of G1 parents and who also was the parent of a G3 child, and another adult who was the G3 child's other parent. G2 adults without children were excluded. The other parents' SUDs and separation from the child were used as control variables. We compared those who were included in Subsample 2 with those who were excluded (i.e., G2 adults without children and their spouses/partners). Excluded participants were younger and less likely to be non-Latinx White. Descriptive statistics for the two study subsamples and the variables used in all models are presented in Table 1.

Measures

G1 SUDs

G1 SUD diagnoses were obtained at Wave 1 from the computerized Diagnostic Interview Schedule (C-DIS, Version 3; Robins et al., 1981) supplemented with spousal report data from the FH-RDC (Version 3; Endicott et al., 1975) if needed. Information about age of onset and recency of symptoms was used to determine whether the G1 parent had SUDs (drug or alcohol disorders) during the G2 child's lifetime. Separate ordered categorical variables for mothers and fathers were created to capture: (0) no AUD or DUD, (1) either AUD or DUD, and (2) both AUD and DUD. In Subsample 1, 54% of G1 fathers had no SUDs, 32% had either AUD or DUD, and 14% had both (Table 1). In Subsample 2, rates for fathers were 57%, 30%, and 13%, respectively. In Subsample 2, 88% of G2 mothers had no SUDs. 10% had either AUD or DUD, and 2% had both. In Subsample 2, rates for mothers were 86%, 11%, and 3%, respectively.

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Table 1. Descriptive statistics

	Subsample 1 (Model 1a; $N = 829$)	Subsample 2 (Models 1b, 2, and 3; $N = 347$)
	G2s biological children of G1s	G2s must be parents of at least one G3
Subsample inclusion criteria	%/*M*(*SD*); range	%/*M*(*SD*); range
G1 variables		
G1 father SUDs during G2 child's life	54% No SUD	57% No SUD
	32% AUD or DUD	30% AUD or DUD
	14% AUD & DUD	13% AUD & DUD
G1 mother SUDs during G2 child's life	88% No SUD	86% No SUD
	10% AUD or DUD	11% AUD or DUD
	2% AUD & DUD	3% AUD & DUD
G1 (M or F) parent MDD Dx.	5% Yes	7% Yes
G1 (M or F) parent ASPD Dx.	7% Yes	7% Yes
G2 variables		
G2 age	32.86 (3.65); 21–50	34.74 (4.12); 21–50
G2 ethnicity	30% Latinx	36% Latinx
G2 sex	50% Female	62% Female
G2 separation from father	18% Separated	22% Separated
G2 separation from mother	8% Separated	9% Separated
G2 lifetime SUDs	38% No SUD	–
	35% AUD or DUD	
	27% AUD & DUD	
G2 SUDs during G3 child's life	–	55% No SUD
		27% AUD or DUD
		18% AUD & DUD
G2 father SUDs during G3 child's life		44% No SUD
		33% AUD or DUD
		23% AUD & DUD
G2 (M or F) MDD Dx.	–	30% Yes
G2 ASPD symptoms (greater # between M or F)	–	1.69 (2.36); 0–6
G3 variables		
G3 sex	–	47% Female
G3 age (Wave 7–10; age-band)	–	20.45 (2.47); 16–27
G3 ethnicity	–	42% Latinx
G3 separation from G2	–	20% Yes
G3 separation from mother	–	12% Yes
G3 separation from father	–	41% Yes
G3 typical drinking quantity (Wave 7–10; age-band)	–	3.29 (2.36); 0–9
G3 drinking frequency (Wave 7–10; age-band)	–	3.32 (2.00); 0–8
G3 most drinks in a day (Wave 7–10; age-band)	–	7.05 (8.11); 0–48

Note. MDD = Major Depressive Disorder. ASPD = Antisocial Personality Disorder. Typical drinking quantity: (0) "none" to (9) "9 or more." Typical drinking frequency: (0) "never" to (8) "every day."

G1 psychopathology

G1 diagnoses of psychopathology were tested as covariates. Diagnoses of affective disorder (major depression and dysthymia) and antisocial personality disorder were assessed with the DIS-III (Robins et al., 1981) at Wave 1. Information about onset and recency of symptoms was used to determine whether parents had a diagnosis of psychopathology during the child's lifetime. Two separate dichotomous variables were created to capture whether at least one parent had a diagnosis of (a) affective disorder, or (b) antisocial personality disorder at any time during the G2's first 18 years of life (i.e., had symptoms after the child's birth and before the child turned 18). Five percent of G2s in Subsample 1 and

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7% of G2s in Subsample 2 had a parent with an affective disorder (Table 1). Seven percent of G2s in both Subsamples 1 and 2 had a parent with antisocial personality disorder.

G2 psychopathology

Indicators of G2 psychopathology were also tested as covariates. G2 diagnoses of affective disorder (major depression and dysthymia) were assessed with the C-DIS (Robins et al., 1981) at Wave 6. Information about onset and recency of symptoms was used to create a dichotomous variable capturing whether at least one parent had a diagnosis of an affective disorder during the G3's childhood (i.e., had symptoms after the child's birth and before the child turned 18). Thirty percent of G3 children had at least one parent with an affective disorder (Table 1). However, clinical diagnoses of parent antisocial personality disorder were not possible because we did not assess C-DIS conduct problems before age 18. Therefore, a count of adult antisocial personality disorder symptoms during the child's lifetime was used as a proxy. To aggregate across the child's two parents, the count of symptoms that was higher among mother versus father was used ($M = 1.69$; range: 0–6).

G1-G2 parent-child separation

Adult G2 participants answered several questions about history of parent-child separation on the computerized Diagnostic Interview Schedule (DIS, Version 4; Robins et al., 1995) as part of a developmental history for the DSM-IV diagnostic interview at Wave 6. Participants responded to the question, "Before age 15, did you live away from your biological mother for at least 6 months?" and repeated this question for their biological father. Eighteen percent and 22% of G2s experienced father-child separation in Subsamples 1 and 2, respectively (Table 1). Eight percent of G2s in Subsample 1 and 9% in Subsample 2 experienced mother-child separation.

G2-G3 parent-child separation

Because G3 children did not complete diagnostic interviews, a parallel measure of parent-child separation for the G2-G3 generation was not available. Instead, G3 reports of family structure at Waves 5 ($M_{age} = 7.41$; range: 4.59–13.94) and 6 ($M_{age} = 12.98$; range: 7.73–17.64) were used to create a proxy variable for parent-child separation. If their response at either wave indicated that they were not living with their mother or father, they were coded as having experienced maternal or paternal separation. Forty-one percent of G3s experienced father-child separation (the separation variable used for Model 1b), whereas 12% experienced mother-child separation (Table 1). Twenty percent of G3s were separated from their G2 parent (i.e., the biological child of G1 grandparents. This variable was used for Models 2 and 3 because the mediation model required G2 parents to be the biological children of G1s).

G2 SUDs

G2 lifetime AUD and DUD (each dichotomous; yes/no) were assessed with the C-DIS (Robins et al., 1981) or FH-RDC (Endicott et al., 1975) at Wave 6. An ordered categorical variable was created to capture: (0) no AUD or DUD, (1) either AUD or DUD, and (2) both AUD and DUD. Forty-four percent of G2 fathers had no SUDs, 33% had either AUD or DUD, and 23% had both (Table 1). Among G3 youth's G2 parents (i.e., the biological children of G1 grandparents), 55% had no SUDs, 27% had AUD or DUD, and 18% had both.

G3 drinking

In order to maximize the use of data and deal with missingness at different waves, an age-band approach was used to capture G3 drinking. Because the majority of the data came from Wave 9, when only alcohol use (not other drug use) data were collected, only alcohol use was examined in G3s. During G3 adolescence and young adulthood (ages 16–27; $M_{age} = 20.45$), alcohol use data were chosen from waves 7–10 based on (a) when data were available, and (b) when the youth were closest to their early twenties (when levels of drinking typically peak; Lee & Sher, 2018). Alcohol use was measured with a latent variable comprised of three indicators: frequency of past-year drinking, (0) never to (8) every day ($M = 3.32$); average number of drinks consumed, (0) none to (9) 9 or more ($M = 3.29$); and greatest number of drinks consumed in one day ($M = 7.05$). Loadings for the drinking latent variable were strong and significant in all models. Standardized coefficients were not available due to the use of categorical mediators in the full structural equation model that included the measurement model for drinking. Unstandardized coefficients ranged from 1.51 to 4.61; all $p < .001$.

Analytic plan

Intercorrelations among study variables were examined in SPSS Version 27.0 to assess for potential multicollinearity between predictors. Pearson correlations are presented in Table 2. Despite some associations between predictors of interest, there was no evidence of multicollinearity; VIFs were in an acceptable range (<10) for all predictors.

Analyses for all four models were conducted in *MPlus* Version 8 (Muthén & Muthén, 2018). Model 1a tested main and interactive effects of G1 father SUDs and G1 father-G2 child separation on G2 child SUDs, using ordinal logistic regression to estimate effects of predictors on an ordered categorical dependent variable. In ordinal logistic regression, predictors with a statistically significant odds ratio above 1 are associated with greater odds of being above a particular level of SUD (one SUD vs. none, or two SUDs vs. one) as opposed to being at or below that level, whereas predictors with a statistically significant odds ratio below 1 are associated with lower odds of being above a particular level of SUD (Harrell, 2015). Initial covariates included G1 parent psychopathology during G2 child's lifetime, G1 mother SUDs, G1 mother-G2 child separation, G2 sex, G2 age, and G2 ethnicity. Nonsignificant covariates were trimmed. Model 1b tested main and interactive effects of G2 father SUDs and G2 father-G3 child separation on G3 drinking, using linear regression. Covariates included G2 parent psychopathology during G3 child's lifetime, G2 mother SUDs, G2 mother-G3 child separation, G3 age, G3 sex, and G3 ethnicity. Nonsignificant covariates were trimmed. Models 2 and 3 used bootstrapping-based mediation and syntax developed by Stride et al. (2015) to test whether father-child separation in the G1-G2 family (Model 2) or parent-child separation in the G2-G3 family (Model 3) moderated the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. In models 2 and 3, initial covariates included G1 parent psychopathology during G2 child's lifetime, G1 mother SUDs, G1 mother-G2 child separation, G2 parent psychopathology during G3 child's lifetime, G2 spouse/partner SUDs, G2 partner-G3 child separation, G3 age, G3 sex, and G3 ethnicity. FIML was implemented to account for missing data (7% for Model 1a, 15% for Model 1b, 16% for Model 2, and 14% for Model 3). Standard errors were corrected to account for clustering among

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Table 2. Bivariate Pearson's correlations

	1	2	3	4	5	6	7	8	9	10	11
1	–	−0.01	.17**	.22**							
2	−0.08	–	−0.01	.19**							
3	.26*	0.06	–	.20**							
4	.29**	.14**	.24**	–							
5	.26**	−0.02	.12**	.62**	–						
6	0.04	−0.08	−0.06	−0.03	0.09	–					
7	−0.01	.29**	−0.04	.29**	.33**	−0.06	–				
8	.19**	−0.1	0.07	.35**	.32**	−0.01	.51**	–			
9	0.1	−0.08	−0.07	.14**	.18**	.33**	0.04	0.06	–		
10	.16*	−0.1	−0.02	.16**	.21**	.16**	0.12	.16**	.70**	–	
11	.17*	0.08	0.11	.20**	.20**	.27**	0.01	0.09	.48**	.54**	–

Note. *$p < .05$, **$p < .01$. Correlations above the diagonal are for Subsample 1 (Model 1a), below the diagonal are for Subsample 2 (Models 1b, 2, and 3). Blank correlations above diagonal are due to lack of G3 variables in Model 1a. To preserve space, only variables used in final study models included (excluded covariates that were trimmed from final models due to nonsignificance). (1) G1 Father SUD During G2 Child's Life. (2) G2 Sex. (3) G2 Separation from Father. (4) G2 SUDs (Lifetime for Model 1a, During G3 Child's Life for Models 2 and 3). (5) G2 Father SUDs During G3 Child's Life. (6) G3 Age. (7) G3 Separation from G2. (8) G2 Separation from G2 Father. (9) G3 Typical Drinking Quantity. (10) G3 Drinking Frequency. (11) G3 Most Drinks in a Day.

Table 3. Model 1a: Father-child separation as moderator of the effect of G1 father SUDs on G2 child lifetime SUDs

				95% CI	
	B	*SE*	OR	Lower	Upper
G1 father SUDs**	0.739	.142	2.093	1.584	2.765
G1 father-G2 child separation**	1.721	.371	5.593	2.704	11.565
G1 father SUDs × separation**	−1.114	.354	0.328	0.164	0.657
G2 sex**	0.707	.147	2.027	1.520	2.704

Note. **$p < .01$, ***$p < .001$. Nonsignificant covariates (G1 parent psychopathology, G1 mother SUDs, G1 mother-G2 child separation, G2 age, and G2 ethnicity) were trimmed. OR = Logistic regression odds ratio. CI = confidence intervals. Confidence intervals for odds ratios are not significant if 1 is included. Significant effects **bolded** for emphasis.

G2 siblings, due to nonindependence in variables such as G1 SUD and psychopathology, ethnicity, and G1 parent-G2 child separation. Model fit indices were not available because of the use of ordered categorical mediators and outcomes.

Results

Model 1a

Model 1a tested main and interactive effects of G1 father SUDs and G1 father-G2 child separation on G2 SUDs (see Table 3 for results). G1 Father SUDs and G1 father-G2 child separation were both associated with increased G2 odds of having a higher level of SUD. There was a significant interaction between G1 father SUDs and separation. Probing this interaction showed that among families without G1 father-G2 child separation, G1 father SUDs predicted greater G2 odds of having a higher level of SUD (OR = 2.093, 95% CIs: 1.491, 2.695), but in families with father-child separation, G2 odds of having a higher level of SUD were similarly high regardless of father SUDs (OR = 0.687, 95% CIs: 0.219, 1.156). This "vulnerable-stable" interaction is displayed in Figure 3. Males also had greater odds of having a higher level of SUD.

One of the assumptions of ordinal logistic regression is the proportional odds assumption (Harrell, 2015), which states that (a) the effects of predictors on the lowest versus all higher categories of the dependent variable (i.e., zero G2 SUDs vs. one or two G2 SUDs) are the same as the coefficients for (b) the effects of predictors on the relationship between the next lowest category and all higher categories (i.e., zero or one G2 SUDs vs. two G2 SUDs). We evaluated this assumption by converting the ordered categorical variable into two binary variables and performing binary logistic regression analyses with the two outcomes. Results suggested that coefficients for the two binary dependent variables were similar (see Supplemental Table A), indicating that the proportional odds assumption was met.

Model 1b

Model 1b tested main and interactive effects of G2 father SUDs and G2 father-G3 child separation on G3 child drinking (see Table 4). A higher level of fathers' SUDs predicted greater child drinking. However, there was no effect of father-child separation on child drinking and no interaction between father SUDs and separation. Lastly, older G3s had greater drinking.

Model 2

Model 2 tested father-child separation in the G1-G2 family as a moderator of the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. Main and interactive effects of study variables on G2 SUDs and G3 drinking are presented in Table 5, and moderated mediation effects are presented in Table 6. G1 father SUDs, G1 father-G2 child separation, and older G2 age predicted greater G2 odds of having a higher level of SUD. There was also an interaction between G1 father SUDs and G1 father-G2 child separation. This interaction was probed, revealing a pattern similar to that seen in Model 1a. G1 father SUDs increased G2 odds of having a higher level of SUD in the absence of father-child separation (OR = 2.427, CIs: 1.616, 3.582), but among G2s who experienced father-child separation, G2 odds of having a higher level of SUD were similarly high regardless of G1 father SUDs (OR = 0.735, CIs: 0.251, 2.157). A higher level of G2 SUDs was associated with greater drinking in G3 grandchildren. However, G1 grandfather SUDs did not have a significant

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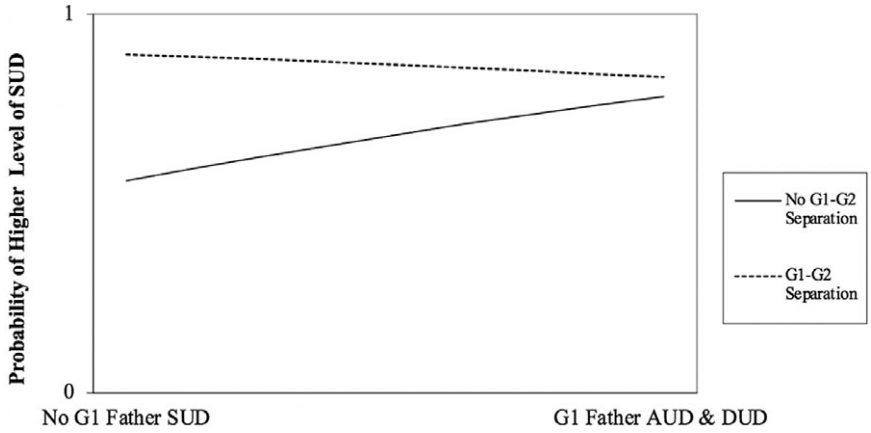

Figure 3. G1 father-G2 child separation moderates the effect of G1 father SUDs on G2 SUDs (from model 1a).

direct effect on G3 grandchild drinking. Older G3s also drank more.

Indirect and total effects of G1 grandfather SUDs on G3 grandchild drinking in nonseparated and separated families appear in Table 6. Following procedures created by Stride et al. (2015), an index of moderated mediation was calculated, which multiplied the unstandardized beta coefficient for the "b path" (M on Y) by the unstandardized beta coefficient for the effect of the interaction term on the mediator. The index of moderated mediation was significant, suggesting that G1 father-G2 child separation moderated the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. In families without G1 father-G2 child separation, there were significant total and indirect effects of G1 grandfather SUDs on G3 grandchild drinking. However, in families with histories of G1 father-G2 child separation, these effects were nonsignificant.

Model 3

Model 3 tested G2 parent-G3 child separation as a moderator of the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. Main and interactive effects of study variables on G2 SUDs and G3 drinking are presented in Table 7, and moderated mediation effects are presented in Table 8. G1 father SUDs, male G2 sex, and younger G2 age predicted increased G2 odds of having a higher level of SUD. Greater G2 SUDs, G2 parent-G3 child separation, and older G3 age were all associated with greater drinking in G3 grandchildren. G1 grandfather SUDs did not predict G3 grandchild drinking. There was also an interaction between G2 parent SUDs and G2-G3 separation. When this interaction was probed, G2 SUDs predicted greater G3 drinking in families that did not experience G2-G3 separation ($B = .333$, $p = .006$), but G2 SUDs predicted similarly high levels of G3 drinking in families with G2-G3 separation ($B = -.152$, $p = .408$; see Figure 4).

Indirect and total effects of G1 grandfather SUDs on G3 grandchild drinking in nonseparated and separated families appear in Table 8. The index of moderated mediation multiplied the unstandardized beta coefficient for the "a path" (X on M) by the unstandardized coefficient for the effect of the interaction term on the outcome. This index was significant, suggesting that G2 parent-G3 child separation moderated the indirect effect of G1 grandfather SUDs on G3 grandchild drinking through G2 parent SUDs. In families without G2 parent-G3 child separation, there were significant total and indirect effects of G1 SUDs on G3 drinking. However, in families with histories of G2-G3 separation, these effects were nonsignificant.

Finally, to contextualize these findings, percentages of G2 SUDs in Models 1a and 2, as well as means of each outcome variable (G2 SUDs or G3 drinking) in all four models, were calculated by group (parental SUD × parent-child separation) in SPSS Version 27.0 to explore patterns in the data (see Supplemental Tables B and C). These patterns indicated that in Models 1a, 2, and 3, parental SUDs predicted greater offspring substance use and disorder in nonseparated families, but in separated families, offspring substance use and disorder was similarly high regardless of parental SUDs. In Model 1b, means of G3 child drinking only appeared to increase with G2 father SUDs.

Discussion

The present study sought to understand whether parent-child separation moderated the intergenerational transmission of substance use and disorder across three generations. Specifically, we were interested in whether (a) father-child separation moderated the effect of father SUDs on child SUDs in the G1-G2 family, (b) father-child separation moderated the effect of father SUDs on child drinking in the G2-G3 generation family, (c) father-child separation in the G1-G2 family moderated the indirect effect of grandfather SUDs on grandchild drinking through parent SUDs, and (d) parent-child separation in the G2-G3 family moderated the indirect effect of grandfather SUDs on grandchild drinking through parent SUDs. Our study was well-positioned to test these questions given its longitudinal, three-generation design and oversampling of families with SUDs. Although previous research has demonstrated parent-child separation as a moderator of the intergenerational transmission of SUD, our findings contribute to the literature by testing these effects across three generations (both through replication across generations and through moderated mediation analyses that test parent-child separation in different generations).

Interactive effects of father-child separation and father SUDs

In G1-G2 families with father-child separation, G2 offspring had a similarly high level of lifetime SUDs regardless of paternal SUDs. However, in G1-G2 families without father-child separation, paternal SUDs increased risk for offspring SUDs. This moderated effect reflects a "vulnerable-stable" pattern (Luthar et al.,

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Table 4. Model 1b: Father-child separation as moderator of the effect of G2 father SUDs on G3 child drinking

	B	*SE*	*p*
G2 father SUDs*	0.343	.149	.021
G2 father-G3 child separation	0.354	.260	.173
G2 father SUD × separation	−0.174	.224	.439
G3 age***	0.129	.036	<.001

Note. *$p < .05$, ***$p < .001$. Nonsignificant covariates (G2 parent psychopathology, G2 mother SUDs, G2 mother-G3 child separation, G3 sex, and G3 ethnicity) were trimmed. Significant effects **bolded** for emphasis.

Table 5. Model 2: Direct effects in mediation model with moderation by G1-G2 separation

					95% BCa bootstrap CI	
	B	SE	*p*	OR	Lower	Upper
G2 child SUDs						
G1 father SUDs***	0.887	.203	<.001	2.427	1.616	3.582
G1 father-G2 child sep*	1.893	.736	.010	6.638	1.763	28.888
G1 father SUDs × sep*	−1.194	.587	.042	0.303	0.096	0.967
G2 age*	−0.079	.035	.023	0.924	0.866	0.990
G3 child drinking						
G2 parent SUDs*	0.246	.102	.016	–	–	–
G1 grandfather SUDs	0.189	.117	.107	–	–	–
G3 age***	0.138	.036	<.001	–	–	–

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Nonsignificant covariates (G1 parent psychopathology, G1 mother SUDs, G1 mother-G2 child separation, G2 sex, G2 parent psychopathology, G2 partner SUDs, G2 partner-G3 child separation, G3 sex, and G3 ethnicity) were trimmed. Significant effects **bolded** for emphasis. Logistic regression odds ratio results only for effects with categorical outcome (G2 SUDs).

Table 6. Model 2: Total and specific indirect effect of G1 SUDs on G3 drinking through G2 SUDs: Moderation by G1-G2 separation

		95% BCa bootstrap CI	
	B	Lower	Upper
Indirect effect: Nonseparated	.218	.042	.451
Indirect effect: Separated	−.076	−.410	.183
Total effect: Nonseparated	.407	.140	.705
Total effect: Separated	.113	−.269	.489
Index of moderated mediation	−.293	−.761	−.018

Significant effects **bolded** for emphasis.

Table 7. Model 3: Direct effects in mediation model with moderation by G2-G3 separation

					95% BCa bootstrap CI	
	B	*SE*	*p*	OR	Lower	Upper
G2 child SUDs						
G1 father SUDs***	0.847	.213	<.001	2.333	1.552	3.523
G2 sex**	0.768	.248	.002	2.155	1.331	3.434
G2 age*	−0.084	.037	.023	0.920	0.855	0.988
G3 child drinking						
G2 parent SUDs*	0.333	.122	.006	–	–	–
G2 parent-G3 child sep*	0.637	.273	.020	–	–	–
G2 parent SUD × sep*	−0.485	.220	.027	–	–	–
G1 grandfather SUDs	0.199	.115	.085	–	–	–
G3 age***	0.137	.036	<.001	–	–	–

Note. *$p < .05$, **$p < .01$, ***$p < .001$. Nonsignificant covariates (G1 parent psychopathology, G1 mother SUDs, G1 mother-G2 child separation, G2 parent psychopathology, G2 mother SUDs, G2 mother-G3 child separation, G3 sex, and G3 ethnicity) were trimmed. Significant effects **bolded** for emphasis. Logistic regression odds ratio results only for effects with categorical outcome (G2 SUDs).

Table 8. Model 3: Total and specific indirect effect of G1 SUDs on G3 drinking through G2 SUDs: Moderation by G2-G3 separation

		95% BCa bootstrap CI	
	B	Lower	Upper
Indirect effect: Nonseparated	.282	.081	.591
Indirect effect: Separated	−.129	−.550	.163
Total effect: Nonseparated	.481	.168	.804
Total effect: Separated	.070	−.264	.441
Index of moderated mediation	−.411	−.963	−.079

Note. Significant effects **bolded** for emphasis.

2000). This is consistent with prior findings indicating that youth with lower levels of a given risk factor (e.g., youth from higher socioeconomic backgrounds, White youth) prior to a parent-child separation experience greater harm from the separation (Bosick & Fomby, 2018; Fomby et al., 2010; Mollborn et al., 2012; Womack et al., 2019; Räikkönen et al., 2011; Ryan et al., 2015). From a developmental psychopathology framework, father-child separation in lower-risk families may represent a "turning point," in which a disruptive event transpires that is markedly different from what the child has generally experienced previously over their life course (Rutter, 1996). Such a turning point may facilitate discontinuity in development by eliciting risk for SUD that was previously low. On the other hand, among families that already have risk for SUD, the likelihood of SUD may already be great enough that father-child separation exerts little impact on this risk. An alternative interpretation is that these results highlight two distinct pathways into SUD: a genetically influenced pathway for individuals with familial risk for SUD and an environmentally influenced pathway, through separation, for those with less familial risk.

When this interaction effect was tested in the G2-G3 family, however, it was not replicated. There are several possible explanations for this discrepancy. Perhaps father-child separation has a greater impact on substance disorder, rather than alcohol use, or is more predictive of drug use or polysubstance use than alcohol use. Because we did not have data on G3 substance disorder or drug use, we could not test these questions. Another possibility is that this interaction effect may not be visible until youth age into adulthood, when there is less age heterogeneity in substance use than in adolescence/young adulthood. G3 youth in the current analyses ranged in age from 16 to 27, and there was a strong effect of

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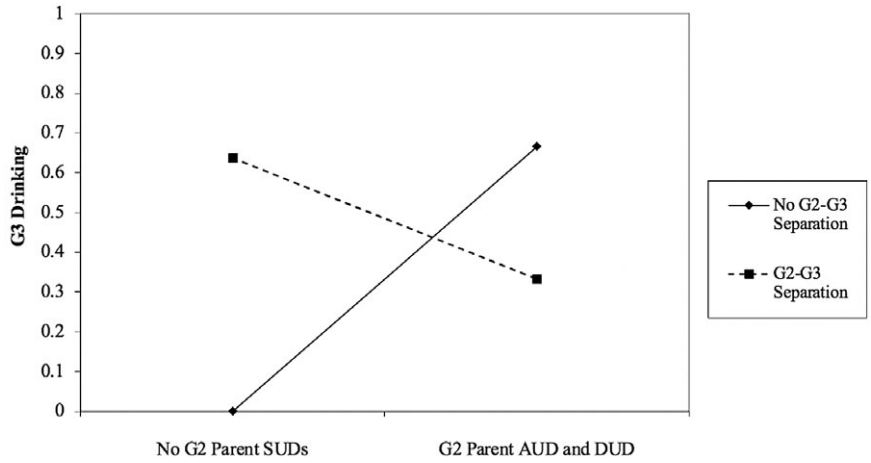

Figure 4. G2 parent-G3 child separation moderates the effect of G2 SUDs on G3 drinking (from model 3, moderated mediation model with parent-child separation in G2-G3 family).

age on drinking. Finally, father-child separation may have been more impactful for G2 children rather than for their offspring, as G2 children grew up during a time when two-parent households were more commonplace (Parker & Horowitz, 2015).

Parent-child separation as a moderator across three generations

Our first moderated mediation analyses expanded upon previous studies by testing whether father-child separation not only moderated effects of father SUDs on child SUDs, but also had downstream effects on grandchildren's drinking. We found evidence for moderated mediation, suggesting that father-child separation only increased substance use and disorder risk in G2 children and G3 grandchildren in the *absence* of prior familial risk for SUD (i.e., in the absence of grandfather SUDs). In other words, the indirect effect of grandfather SUDs on grandchild drinking through parent SUDs was only significant among nonseparated families. In families with father-child separation in the G1-G2 family, risk for G2 child SUDs (and subsequently, G3 grandchild drinking) was higher than in the group of families with neither father SUDs nor separation, regardless of G1 grandfather SUDs. This finding underscores the strong impact of father-child separation on substance use and disorder for several generations. Not only did father-child separation increase risk for SUDs in children with otherwise lower levels of familial risk for SUD; it also appeared to act as a distal predictor that "set off" a chain of intergenerational transmission of substance use problems to those children's later offspring. Individuals who develop SUDs due to father-child separation may pass along risk for substance use and disorder to their own offspring, potentially through mechanisms such as substance-specific parenting, adversity exposure, or other environmental risk.

Parent-child separation in the G2-G3 family was also found to moderate intergenerational transmission across three generations. These results suggest that even when SUDs are transmitted from parent to child, separation between child and grandchild may moderate effects on grandchild drinking. Here, too, the interaction between parental SUD and separation reflected a "vulnerable-stable" pattern, in which G2 parent-G3 child separation only increased G3 drinking among G3s with low levels of parental SUD. In other words, G2 SUDs predicted greater G3 drinking in families without G2 parent-G3 child separation, whereas in families with separation, G3 drinking levels were similarly high regardless of G2 SUDs. The emergence of this pattern of interaction across two generations in our moderated mediation analyses supports the robustness of our findings. Moreover, the test of moderated mediation indicated that in families without G2-G3 separation, there was transmission of G1 grandfather SUDs to G2 parent SUDs to G3 child drinking. However, this indirect effect was nonsignificant in families with G2-G3 separation. G3s who were separated from a G2 parent demonstrated heightened levels of drinking, regardless of parent and grandparent SUDs. Importantly, this "vulnerable-stable" pattern of interaction was found across two generations in the present study, which increases confidence in these findings. However, it stands in contrast to prior literature, most of which has found that individuals who experience *both* parental separation and SUD have the greatest substance use risk (Jackson et al., 2016; Thompson et al., 2008, 2013; Waldron et al., 2014; Windle & Windle, 2018). Perhaps this is because, unlike the samples of the aforementioned studies, the current sample is enriched for families with SUD. This may have allowed us to better model the high level of risk that parental SUD confers for offspring substance use outcomes, regardless of history of parent-child separation.

It is notable that this interaction effect was seen in the current model (Model 3), but not in Model 1b, which tested whether G2 father-G3 child separation moderated the effect of G2 father SUDs on G3 drinking. Importantly, Model 3 included both female and male G2 parents, because the moderated mediation model required these parents to be the biological children (male or female) of G1 parents. It is possible that the detrimental effect of parent-child separation in this generation was carried by mother-child separation. Although the low rate of mother-child separation in the present sample precluded us from testing this with sufficient power, we tested in a sensitivity analysis whether mother SUDs and mother-child separation interacted to predict child drinking, controlling for child age (other covariates were nonsignificant and thus trimmed). Maternal SUDs predicted greater child drinking, but mother-child separation and the interaction were nonsignificant (see Supplemental Table D). Overall, it appears that an aggregation of maternal and paternal separation interact with parental SUDs to predict offspring drinking in the G2-G3 generation, but reasons for this are unclear. Future studies with higher rates of mother-child separation should aim to disentangle these effects.

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Limitations and future directions

The current findings should be interpreted in light of several limitations. First, because some G3 youth were still adolescents, it is possible that we did not capture escalations in drinking that occurred as they aged into young adulthood. Similarly, the original sample of data was selected from one state consisting of a unique sample of parents with and without SUD that may not reflect other samples. Second, there was a relatively low rate of maternal SUD and mother-child separation, which prevented us from testing effects of these variables in primary analyses due to lack of power. We were also underpowered to test the effects of timing of separation, despite research indicating that parental separation earlier versus later in childhood may exert differential effects on youth outcomes (Cavanagh & Fomby, 2012). Future research should address these limitations. Finally, although we tested a number of covariates, there may have been unmeasured confounders, such as reason for separation, socioeconomic status, and child co-residence with other caregivers. An important direction for future research is to incorporate advanced methods for causal inference with observational data, such as propensity score matching or instrumental variables, to more accurately estimate the effects of parent-child separation on substance use and disorder.

Implications

Our findings have several key implications for research and prevention. First, given that parent-child separation may promote offspring SUD at lower levels of parental SUD, prevention efforts should be targeted towards youth experiencing paternal separation during this key developmental "turning point," especially since these consequences seem to exert later downstream effects on their own offspring. Similarly, parent-child separation also appears to set off risk for drinking among youth with low familial (parent and grandparent) risk for SUD, further highlighting the potential role of parent-child separation as a turning point. Finally, because the effects of parent-child separation appear to depend on parental SUD and other risk factors, it is possible that studies only examining effects of separation on youth outcomes will produce misleading effects. Thus, future research should aim to further disentangle the separate and joint roles of separation and other parental risk factors in promoting or protecting against youth maladjustment.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579422000876

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Conflicts of interest. None.

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