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Developmental cascades in studies of adolescent and young adult substance use etiology: A systematic review

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ABSTRACT

Introduction: Frequently, developmental cascade models are used to examine causal linkages between early family risk and substance use etiology. When framed with longitudinal data, cascade models contribute to understanding developmental etiology by parsing stability from change in multiple domains of influence. This systematic review examines the research methods used in cascade studies of substance use etiology.

Method: A systematic literature review involved four electronic literature databases (i.e., PsycINFO, MEDLINE, EMBASE, Web of Science). Specific terms referenced substance use etiology and developmental cascade effects. Inclusion requirements included cross-domain effects and repeated measures. Studies were eliminated based on including interventions or growth modeling that failed to differentiate time-specific effects. A risk assessment indicated adequate inter-rater reliability for the 18 studies included.

Results: Conceptually, there was little evidence supporting hypothesized cascade effects that involved cross-domain risk mechanisms linking early parental socialization with later substance use. Methodologically, studies were characterized by modest sample sizes, lack of power, and relatively small effect sizes (ESavg. = 0.05 [SD = 0.046], range 0.003 - 0.19). Only half of the studies conducted formal statistical tests of indirect effects linking early socialization with later substance use.

Conclusion: This review highlights there is very little evidence for developmental cascade effects involving early parental socialization and substance use etiology. Methodological and conceptual limitations may hamper detection of developmental cascade effects and further undermine our understanding of substance use etiology. Future studies may want to follow larger samples, over extended time frames and specify intermediate mechanism that contribute to vulnerability.

1. Introduction

The concept of developmental cascades has received considerable attention in the field of developmental psychopathology (Masten & Cicchetti, 2010). Cascade models are primarily rooted in the general language of biology, embryology, and epigenetic models of development (Gottlieb, 2007); however, they also draw heavily on developmental systems theory. As a result, an important component to cascade models heralds the active role the organism plays in determining behavioral outcomes (Cox et al., 2010). The goal of cascade models is to account for the subtle intricacies that link age-salient tasks that occur at one stage of development with behaviors that occur further downstream. Rather than being construed as a passive organism with development unfolding according to a blueprint, the developing organism engages with important social actors and, through self-organization, actively constructs their world. This mutual reciprocity enables the individual to actively select behavioral responses that increase their person-environment fit. This view is consistent with Bronfenbrenner's (1977, 1979) bioecological framework specifying that development is a highly interactive process positioned within concentric circles of mutual influence. A strength of the cascade framework is that it does not impose a single nomothetic pathway that unfolds in a linear fashion across development. Rather, using the full compilation of longitudinal developmental relations, an investigator can posit that multiple paths can converge on a single outcome (i.e., equifinality) or that a single path can produce multiple outcomes (i.e., multifinality) (Cicchetti & Rogosch,

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1996).

The literature offers several renditions on what comprises a cascade model. For instance, Masten and Cicchetti (2010) referred to "cumulative consequences" in their definition of cascades and further qualified that relations could be "direct and unidirectional, direct and bidirectional, or indirect through various pathways" (p. 491). Lin et al. (2020) stated that a developmental cascade model "represents the way two or more parts of a developmental system interact cumulatively across multiple time points" (p. 126). Lynne-Landsman et al. (2010) further reinforced the need for explicit "transactions" that cover multiple domains at multiple time points and reflect the perseveration of risk. Jones et al. (2016) perhaps provided the most precise definition when they stated that developmental cascades "refer to the within- and across-domain transactional or interactive processes of development from childhood to adulthood" (p. 722). They further stated that a core feature of cascade models is "reciprocal spillover effects across domains of functioning" (p. 722).

1.1. Underlying features of cascade designs

While there is a wide variability in the conceptualization of cascade models, their design typically involves specifying a cross-lagged panel design (CLPD) using prospective, longitudinal data. Repeated measure "autoregressive" effects capture between-person rank-order stability. Within-time (concurrent) associations between domains capture "reciprocal influences" that reflect mutual interactions. With autoregression in place and appropriate controls for concurrent associations between different domains, a predictive 'cascading' (i.e., cross-lagged) path from one domain to another captures the hypothesized developmental perturbation that contributes to maladaptive outcomes. Having repeated measures also allows the testing of within-domain, developmental stationarity (i.e., stability of mean, variance, and autocorrelation structure over time). Moreover, when model configurations include direct and indirect effects and appropriate covariates, the potential for spurious relations is considerably reduced (Kenny, 1975), which strengthens causal inferences (Berry & Willoughby, 2017). Taken as a whole, the developmental cascade approach provides a means to detect critical developmental junctures where socialization agents (e.g., family, peers) may become increasingly influential or naturally abate. It is also an ideal framework to learn more about earlier family socialization processes associated with later maladaptive outcomes before they become entrenched and resistant to prevention or intervention.

The developmental cascade model has been used to examine the ontogenesis of various developmental outcomes including externalizing behaviors (Bornstein et al., 2010), depression (Goodman et al., 2019), emotional regulation (Blair et al., 2015), conduct problems (Ettekal et al., 2020), aggression (Lansford et al., 2010), antisocial behavior (Kochanska et al., 2017), crime (Fleming et al., 2010), and violence (Dishion et al., 2010). In many cases, these negative outcomes indeed have life-long consequences, but one particular outcome that may deserve particular attention is substance use. Cascade models have been applied to elucidate the mechanisms that contribute to both early onset and continued or exacerbated substance use that meets diagnostic criteria of abuse or dependence. These studies are guided by major etiological theories that provide insight into the primary socializing influences that guide child and youth development (see reviews by Petraitis et al., 1995; Scheier, 2001). More prominent theories include family interactional theory (Brook et al., 1988, 1990), problem behavior theory (Jessor & Jessor, 1977), social development model (Catalano et al., 1996), and peer cluster theory (Oetting & Beauvais, 1986, 1987). More general theories of developmental psychopathology have also been used to account for a wide range of deviant outcomes (Cicchetti & Tucker 1994; Sameroff, 2000). Overall, these theories collectively implicate intrapersonal (e.g., personality, cognitive, affective) and contextual (e.g., school, cultural) factors in the early stages of substance use.

1.2. Cascade models of substance use

Cascade models of substance use etiology vary considerably in their conceptual emphases. For instance, Eiden et al. (2016) examined the effects of mothers' and fathers' parenting practices (i.e., warmth/sensitivity) on child's psychosocial functioning in early and middle childhood and the collective influence of these domains on both early and later adolescent substance use. Barton et al. (2018) examined the relations of chronic family poverty, biological markers of stress (i.e., brain catecholamines), and supportive parenting with cigarette, alcohol, and marijuana use at ages 19 and 25. Lynne-Landsman et al. (2010) examined the effects of early developmental factors (e.g., parental discipline, child oppositional defiant behavior) on trajectories of adolescent alcohol, cigarette, and marijuana use (from 9th to 12th grades) and then monitored the effects of growth in substance use on young adult (age 21) developmental outcomes. In each of these cases, a developmental cascade framework captures a reorganization of child or adolescent behavior through both micro- and macro-transactions that occur between the focal child and powerful socializing agents (e.g., family caregivers, peers) at multiple time points across the lifespan.

1.3. Rationale for the current study

Despite a plethora of individual studies that have implemented a cascade framework to examine substance use etiology, to our knowledge, no systematic review has been conducted that summarizes this area of research. Such a review can help to provide much needed evidence for the developmental significance of certain risk mechanisms, their timing, and relative magnitude of influence. Understanding the developmental etiology of substance use is important for several reasons. First, there is now considerable evidence from longitudinal studies that early and prolonged substance use interferes with adult role socialization (Newcomb, 1987; Staff et al., 2010), including educational attainment beyond high school (Horwood et al., 2010; Register et al., 2001) and economic well-being in young adulthood (e.g., occupational prestige, earning potential; Belfield & Levin, 2007; Thompson et al., 2019). Evidence also shows deleterious effects of drug use on neuropsychological development (Lisdahl & Tapert 2012) with effects extending to mental (Newcomb et al., 1993; Patton et al., 2002) and physical health (Brook et al., 2008; Chen et al., 1996). Furthermore, substance use in the early stages of adolescence can interrupt academic pursuits leading to early school dropout (Townsend et al., 2007), which in turn is associated with increased risks of mental health problems and crime (Maynard et al., 2015). Given a wide range of sequelae associated with substance use, understanding precisely how earlier developmental vulnerabilities influence each other and eventually lead to substance use in adolescence and young adulthood should guide the development of evidence-based prevention, intervention, and treatment programs.

2. Method

2.1. Eligibility criteria

We formulated the inclusion criteria so that we would only include studies that involved the following features: (a) non-clinical samples from the general population as well as high-risk communities, (b) repeated measures with more than two waves (required to empirically confirm "cascade" effects), and (c) a cross-domain configuration (required to confirm reciprocal transactions). We did not mandate a cross-lagged panel design (CLPD) with repeated measures at every wave because in longitudinal studies transpiring over extended periods of time, measures have to change to accommodate unique developmental tasks that surface with increasing age (e.g., Bornstein et al., 2018; Magnusson & Cairns, 1996). Moreover, we did not consider the age at which the study incepted. In this review, several studies followed individuals from birth onwards, but most studies monitored development

through adolescence and emerging and young adulthood. In some cases, a lengthy time frame bridging unique developmental periods was required to model the intricate processes through which early poor parenting increases the risk of child behavioral and socioemotional maladjustment and how this risk perturbation influences subsequent adolescent substance use (e.g., Eiden et al., 2016).

2.2. Exclusion criteria

We eliminated studies that specified causal mediation chains in the absence of any repeated measures (e.g., Kerr et al., 2012; Martel et al., 2009; Mun et al., 2018; Otten et al., 2018). We also did not include intervention studies targeting mediating processes in an effort to disrupt developmental cascades (e.g., Wolchik et al., 2016). We made this decision to preserve the etiological focus and also to provide a more sweeping view of cascade effects rather than "disruption" of cascade effects. We did not include cascade models that relied on latent growth models because this type of model configuration does not separate time-specific effects dissected on a wave-by-wave basis, essentially neglecting a hallmark feature of the cascade framework. However, the exceptions to this exclusion (thus included in the review) are cascade studies that modeled the following: growth to capture intra-individual trajectories of change and statistically related the growth parameters (slope and intercept) to distal outcomes (e.g., Brody et al., 2010), cascade effects for psychosocial risk in conjunction with substance use growth (e.g., Lynne-Landsman et al., 2010), or growth with structured residuals to parse time-specific effects (e.g., Davis et al., 2018).

2.3. Information sources and search strategy

We conducted searches of the peer-reviewed literature using four major electronic databases including PsycINFO (1887-March 2020), MEDLINE (1946-March 2020), EMBASE (1965-March 2020), and Web of Science (1900- March 2020). The search terms included "developmental cascade" in conjunction with the following keywords (using "OR" in between the keywords and using quotation marks for the entire word/phase): "drug use," "drug abuse," "substance use," "substance abuse," "smoking," "tobacco," "cigarette," "alcohol," "marijuana," and "cannabis." We limited the search to published peer review articles and the English language, excluding theses/dissertations, pre-published materials, and government reports. Once we eliminated duplicates across the four databases and obtained full text articles, we determined whether they qualified or not based on the inclusion and exclusion criteria. Finally, we manually searched the reference list from the qualifying articles to retrieve additional candidate articles.

2.4. Risk bias assessment

We assessed risk of bias using a slightly modified systematic review checklist from the Critical Appraisals Skills Programme (CASP; https://www.casp-uk.net/casp-tools-checklists) appropriate for cohort longitudinal studies. Both authors of this review independently coded each of the qualifying articles. A total of 12 evaluation criteria were included based on the original CASP checklist. The few modifications included strengthening the reliability/validity evaluation, adding qualifications of whether different features of the developmental cascade were tested thoroughly (i.e., bidirectional or reciprocal influences, transactions across time, cascade effects) and whether the study findings were relevant and corresponded with the original research hypotheses. A cut-off of 8 out of 12 possible points was used for the evaluation. The two raters substantially agreed ($K = 0.68$, $S_E = 0.11$, p less than 0.01). This was verified by both a logistic regression that failed to differentiate between the raters, χ^2 (9) = 12.81, $p = 0.171$, and a Generalizability Coefficient (reliability estimate) indicating substantial agreement, IE $\rho^2 = 0.77$, $F =$ 4.37, p less than 0.01.

3. Results

3.1. Search results

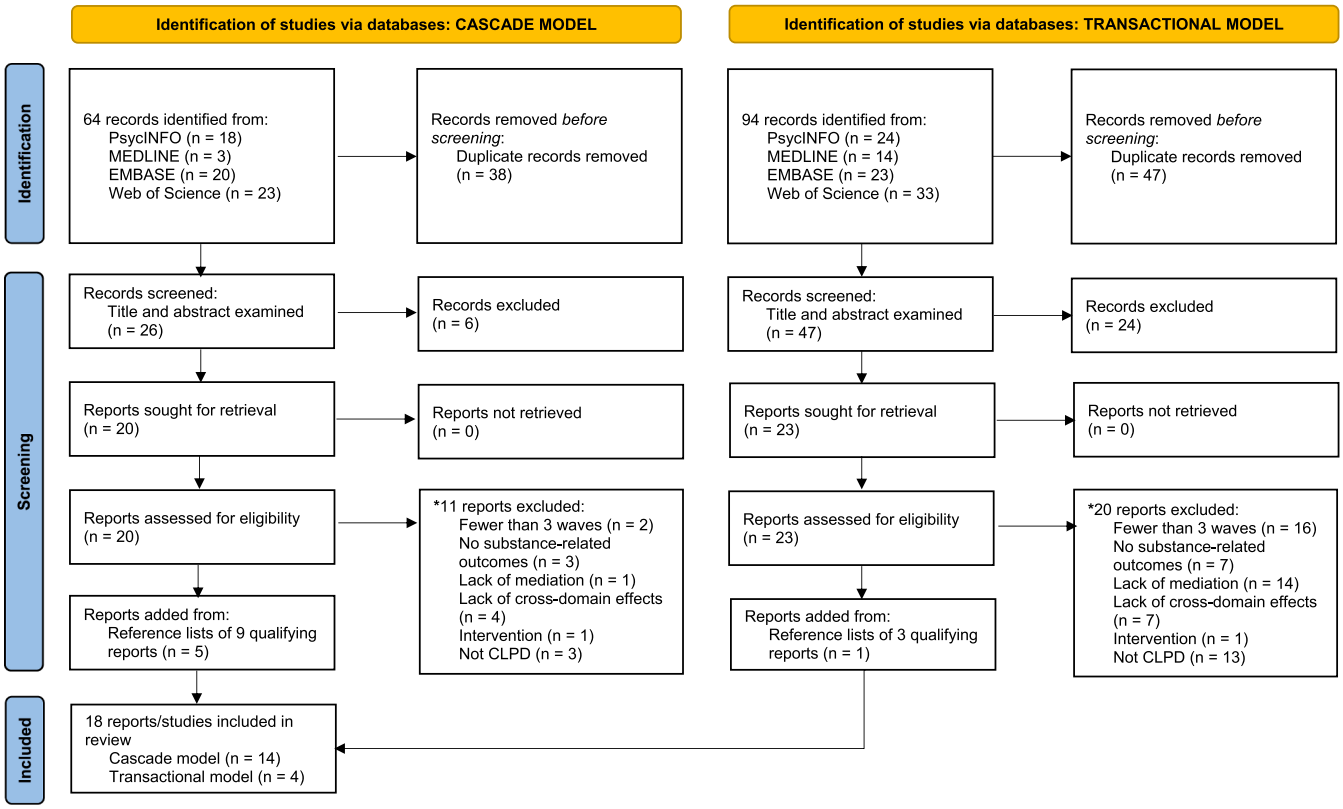
A search of the four electronic databases produced a total of 65 articles based on the search criteria described above: PsycINFO produced 18 articles, MEDLINE produced 3, EMBASE produced 20, and Web of Science produced 23. Elimination of 38 duplicates resulted in 26 unique articles. Based on titles and abstracts, 6 of them were excluded. Each author then independently scanned through each of the 20 articles to determine its suitability as a developmental cascade paper and determined whether the article emphasized substance use as the endpoint, which eliminated 11 articles and resulted in the final set of 9 unique articles. Manual searches of the references of these articles then added 5 more articles.

While screening the articles for qualification, we noted that "transactional model" was a term used in many articles that did not make it through the fine sieve for "developmental cascade" articles. In many cases, investigators failed to mention "developmental cascade" but used a cross-lagged panel design (CLPD) with three or more waves of repeated measures, thus satisfying the inclusion criteria. In order to capture studies that meet the inclusion criteria despite no direct mention of "developmental cascade," we conducted fresh searches using the additional key word "transactional" in conjunction with each of the same key words/phrases as above. These additional searches produced 94 potentially relevant articles, including 24 articles from PsycINFO, 14 for MEDLINE, 23 from EMBASE, and 33 from Web of Science. With the elimination of 47 duplicates as well as 24 articles solely based on titles and abstracts, the remaining 23 articles were carefully evaluated for qualification. Twenty of them were deemed unqualifying, resulting in 3 unique additions. Manual searches of their references added one additional article, producing a total of 4 articles from this second set of online searches that qualified under the inclusion criteria as true "cascade" models. The two online searches combined produced the 18 articles that met the inclusion criteria and thus were included in this review. Following the PRIMSA declaration guidelines (Moher et al., 2009), Fig. 1 shows the flow chart detailing the two sets of searches (i.e., for cascade and transactional models) and qualification processes for the articles.

3.2. Overview of the reviewed studies

Table 1 provides an overview of the 18 studies that qualified for inclusion in the systematic review. The table outlines key sample characteristics for each study and provides pertinent information about the study design, sample descriptions, assessment protocol, statistical techniques, inclusion of power analyses, whether mediation was formally tested, and a brief summary of the main findings (including effect sizes). While this information is not meant to be exhaustive, it should give readers a sense of the full breadth and execution of substance use cascade studies. The section examining study findings addresses only specific tests of the cascade process as they attend to substance use and, for the sake of brevity, excludes mentions of baseline or within-domain associations, covariates, or alternative non-hypothesized pathways.

Several interesting findings can be noted from the table. First, of the 18 studies, only Lee et al. (2015) presented power analyses, which was based on a Monte Carlo simulation for a path analysis. Sample sizes ranged from a low of $N = 120$ to $N = 1{,}875$, and 11 out of the 18 studies (61.11%) had less than 500 participants, indicating the dire need for power analyses to allay concerns about sample sufficiency for reliably detecting effects. In addition, only 9 out of 18 studies (50.00%) included formal statistical tests of these hypothesized indirect effects. Cascade models are built on the premise of extended causal mediation chains including reciprocal and cross-domain transactional effects. Therefore, the merits of a developmental cascade model can only be realized upon testing the intervening mechanisms that represent the risk perturbation



*The numbers of different reasons for exclusions add up to more than the number of reports excluded as a report can be excluded for multiple reasons.

Fig. 1. PRISMA Flow Chart.

producing deficits in adaptive functioning.

The data collection methodology varied considerably across the studies, although almost all studies (except for Haller et al., 2010) used self-report questionnaires in some form or another (Supplementary Table 1). The next most prevalent form was interviews, including in-person interviews, web-based interviews, and diagnostic interviews (13 of 18). A quarter of the studies (4 out of 18; 25.00%) also used computer-assisted personal interviews (CAPI). It was quite common to use multi-informant studies (12 of 18), acquiring data from sources in addition to the focal child (e.g., parents, teachers, peers). Other sources of data included archival data such as standardized academic testing or information culled from county health, clinic, and hospital records (5 of 18). Several studies used observations or coded videotaped interactions, for example, between a caregiver and a child (4 of 18). Two studies collected saliva for genetic testing, fMRI to detect neural changes, or neonatal cord blood to assay lead concentrations.

Many of the studies reviewed specified multiple intervening mechanisms as developmental risk factors (Supplementary Table 2). Specifically, more than half of the studies (13 of 18) emphasized the factors in the family context (e.g., family conflict, child maltreatment, parental alcoholism, parenting) with four of them specifically focusing on parental alcoholism (Eiden et al., 2016; Elam et al., 2016; Haller et al., 2010; Lee et al., 2015) and one on child maltreatment (Rogosch et al., 2010). Half of the studies (9 of 18) included intrapersonal factors (e.g., impulsivity, behavioral disinhibition, self-esteem) and interpersonal skills (e.g., social competence). Similarly, half (or about half) of the studies included maladaptive behaviors among peers as well as focal children/adolescents: peer deviance (e.g., peer delinquency, peer drug use) (9 out of 18), internalizing problems (e.g., depression, anxiety, negative emotions) (9 of 18), and externalizing problems (e.g., physical fighting, bullying perpetration) (8 of 18). Fewer studies included academic factors (e.g., academic achievement, student engagement) (5 of 18), different sources of stress (e.g., negative life events, polygenic risk) (2 of 18), and other risk factors that did not involve parents or family (e.g., unsafe sex, risky driving, environmental toxin exposure) (4 of 18).

3.3. Summary statistics of the design features

The average age at the beginning of the studies was 10.67 (SD = 5.20) and increased to 11.85 (SD = 4.16) with the exclusion of the two studies that included birth cohorts beginning at 12 months of age (Eiden et al., 2016; Sitnick et al., 2014). The average age at termination was 20.47 (SD = 6.35), indicating coverage of a substantial portion of the lifespan including early adolescence through emerging adulthood. The longest time frame studied was 23 years (Jones et al., 2016), which began with interviewing children at ages 10–12 and followed up on them until they were 30–33 years old. On average, studies examined 5.44 waves (SD = 2.36) and spanned 9.80 years (SD = 6.61). Across all of the studies except for Desrochers-Couture et al. (2019), which focused solely on Inuit children in northern Québec, the average racial composition for whites was 47.23% (SD = 31.44). The exclusion of two studies that included only African American participants (Brody et al., 2010; Hsieh et al., 2015) increased this to 53.13% (SD = 26.93). Female participants constituted slightly under half (43.75%, SD = 16.41) and increased to 45.74% (SD = 4.04) when two studies that only examined male participants (Dishion & Owen, 2002; Sitnick et al., 2014) were excluded. The average sample size at follow-up was 549.61 (SD = 445.98). This number shrunk to 410.87 (SD = 247.72) with the exclusion of the two largest studies (>1000 youth) that assessed youth in schools (Davis et al., 2018; Espelage et al., 2014). Cascade studies gathered information from an average of 1.94 (SD = 0.94) individuals

Table 1
Detail of reviewed studies.

Study Authors	# of Waves	Age[1]	Sample Size	Sample Info[2]	Assessment Protocol[3]	Analysis[4] Power Mediation[5]	Findings[6,7]
Brody & Ge, 2001	3	W1: 11–12 yrs W2: 1-yr FU W3: 2-yr FU	120 dual-parent families out of 175 initial families	48.3% Female 100% Euro Am	Y-/P-WB + SRQ (CAPI), HV, T-SRQ, OBS of parent–child interactions	- SEM - No - No	Harsh parenting (W1) → child self-regulation (W2) → alcohol use (W3), controlling for prior W1 measures of child's alcohol involvement. $ES_{avg.} = 0.085$
Brody et al., 2010	3	W1: 11th-12th grade W2: 5-m FU W3: 1-yr FU	W1,2: 347 youths W3: 302	58.5% Female 100% Af Am	Y-SRQ + I, HV	- SEM/FIML + LGM - No - No	ΔLife stress (W1-W2) → Δnegative emotions (W1-W2) → Δdeviant companions (W1-W2) → ΔSU (W2-3), controlling for prior SU (W1). Wave 1–2 change score (Δ) for deviant companions and W1-W2 Δ score for negative emotions associated with 3-wave growth in SU, controlling for W1 life stress. $ES_{avg.} = 0.19$
Danzo et al., 2017	4	W1: 6th grade W2: 7th W3: 8th W4: 9th	593 families	49% Female Ethnically diverse – 36% Euro Am – 18% Latino – 15% Af Am – 7 % Asian	Y-SRQ	- SEM/FIML - No - Yes	Females: depression (W1) → peer deviance (W2) → peer deviance (W3) → alcohol use (W4) (β = 0.08). Males: no sign of cross-domain influences and indirect effects. $ES_{avg.} = 0.01$
Davis et al., 2018	3	W1: 11–13 yrs. W2: 1-yr FU W3: 2-yr FU	1,875 students	50.8% Female Ethnically diverse – 44.3% Af Am – 29.1% Euro Am – 16.5% Multirace – 7.2% Latino	Y-SRQ	- ARLT-SR, FIML - No - Yes	Depression and grades (W1) → drinking (W2). Depression & lower grades (W2) → drinking (W3). Depression & lower grades (W3) → drinking (W4). Drinking (W2) → grades (W3) & drinking (W4). Drinking (W3) → grades (W4). Bullying victimization (W1) → grades (W2) → depression (W3) → drinking (W4) (β = 0.01). $ES_{avg.} = 0.01$
Desrochers-Couture et al. (2019)	3	W1a: prenatal W1b: infancy W2a + b: 10–12 yrs. W3 + b: 17–19 yrs. (a & b = 2 separate cohorts)	W1a: 491 mothers W1b: 221 mothers W2ab: 294 youths W3ab: 212 youths	55.7% Female (W1) 100% Inuit from Northern Québec	Umbilical cord blood, venous blood, Y-IPI, P-/T-SRQ, Y-DI	- LR/SEM, FIML - No - Yes	Child blood lead (W1) → EBP (W2) → binge drinking (β = 0.09) and cannabis use (W3) (β = 0.05). $ES_{avg.} = 0.07$
Dishion & Owen, 2002	6	W1: 13–14 yrs. W2: 15–16 W3: 17–18 W4: 20–21 W5: 21–22 W6: 22–23	201 students	100% Male 90% Euro Am	Y-IPI, Y-SRQ OBS of youth-peer interactions, school/court records	- SEM/FIML - No - No	Deviant friendships (W1) → tobacco use (W2) → deviant friendships (W3) → smoking (W4-6). Deviant friendships (W1) → alcohol use (W2) → deviant friendships (W3).Deviant friendships (W1) → marijuana use (W2) → deviant friendships (W3) → marijuana use (W4-6). Controlling for antisocial behavior at age 12 did not change model findings. $ES_{avg.} = 0.05$
Eiden et al., 2016	9	W1: 12 mos. W2: 18 W3: 24 W4: 36 W5: 48 W6: 5–6 yrs. W7: 9–10/11–12 W8: 13–14 W9: 15–19	227 families - Control: 102 - Alcoholic: 125 (W9: 202)	51.1% Female 90.5% Euro Am	Y-/P-SRQP-DI (alcoholism) OBS of child & parent–child interactions	- SEM/FIML - No - Yes	For alcoholic families: maternal warmth/sensitivity (W3) → self-regulation (W4) → EBP (W7) → delinquency, drug use (W8) → alcohol use (W9) (β = 0.04). $ES_{avg.} = 0.04$
Elam et al., 2016	5	W1: 6.98 yrs. (average) W2: 12.30 W3: 13.67	380 families (W5: 169)	46.7% Female 66.1% Euro Am	Biological samples for children and parentsP-DI (alcoholism), Y-/P-CAPI, Y-/P-SRQ,	- SEM/FIML, control for ICCs to adjust for family clustering - No - Yes	Child polygenic risk (W1) → impulsivity (W1,2) → SU (W3) (β = 0.015).With fewer waves, family conflict (W2) → child reported impulsivity (W4) → SU (W5) (β = 0.046). $ES_{avg.} = 0.03$

(*continued on next page*)

Table 1 (*continued*)

Study Authors	# of Waves	Age[1]	Sample Size	Sample Info[2]	Assessment Protocol[3]	Analysis[4] Power Mediation[5]	Findings[6,7]
		W4: 16.80 W5: 19.31			Y-/P-SRQ + TI, Y-WB + SRQ		
Englund & Siebenbruner, 2012	4	W1: 7 yrs. W2: 9 W3: 12 W4: 16	191 children	45% Female83% (mother)/66% (child) Euro Am	Y-/P-/T-SRQ, clinic/hospital records	- SEM/FIML, TPSC - No - No	AC (W1) → AC (W2) → EBP (W3) → EBP (W4) → alcohol use/nonuse (W4) (β = 0.002).IBP (W1) → IBP (W2) → AC (W3) → AC (W4) → alcohol use/nonuse (β = 0.01) and level of use (β = 0.013).AC (W1) → AC (W2) → EBP (W3) → EBP (W4) → Cannabis Use /Nonuse (β = 0.017) and level of use (β = 0.01). $ES_{avg.}$ = 0.01
Espelage et al., 2014	3	W1: 5th-7th grades W2: 1-semester FU W3: 2-semester FU	1,232 students	49.8% Female Ethnically diverse – 51% Af Am – 34% Euro Am – 9% Other	Y-SRQ	- SEM/MI - No - Yes	Family violence (W1) → fighting perpetration (W2) → SU (W3) for boys only (β = 0.03).Family violence (W1) → bullying perpetration (W2) → SU (W3) for boys only (β = 0.09). $ES_{avg.}$ = 0.06
Haller et al., 2010	4	W2: 14.2 yrs. (average) W3: 15.2 W5: 25.8 W6: 32.1	W2: 449 families W3: 445 W5: 411 W6: 404 - Control: 196 - Alcoholic: 209	48.1% Female 71.9% Euro Am	P-DI (alcoholism), Y-/P-CAPI or TI	- SEM/FIML - No - No	Parental alcoholism → binge drinking (W2) → binge drinking (W3) → binge drinking (W5) → alcohol dependence (W6). Parental alcoholism → academic achievement (W2) → SU (W3) → peer SU (W5) → drug disorder (W6). Only tested independent legs of mediation chain. Parental alcoholism → SU and binge drinking (W5) (β_{avg} = 0.007). $ES_{avg.}$ = 0.004
Hsieh et al., 2015	3	W1: 16 yrs. (10th grade) W2: 18 (12th) W3: 21	681 students	51% Female 100% Af Am	Y-IPI, Y-SRQ	- SEM/FIML - No - No	Delinquent peers (W1) → risky driving (W2). Alcohol & marijuana use (W1) → delinquent peers (W2). Alcohol & marijuana use (W2) → delinquent peers (W3). Delinquent peers (W2) → risky driving (W3). No paths involving negative family environment to other domains significant. $ES_{avg.}$ = 0.05
Jones et al., 2016	13	W1: 10–12 yrs. W2: 13–14 W3: 15–18 W4: 21 W5: 24–27 W6: 30–33	808 families (W6: 765)	49% Female Ethnically diverse – 47% Euro Am – 26% Af Am – 22% Asian Am – 5% Native Am	Y-/P-/T-SRQ + I,Y-DI (youth mental health)	- SEM/FIML - No - No	Positive family environment (W1) → SU (W2) → peer SU (W3) → SA/Dep Sx (W4) → partner SU (W5) → SA/Dep Dx (W6) (β = 0.001).Positive family environment (W1) → BD (W2) → peer SU (W3) → SA/Dep Sx (W4) → partner SU (W5) → SA/Dep Dx (W6) (β = 0.001).Family SU (W1) → peer SU (W3) → SA/Dep Sx (W4) → partner SU (W5) → SA/Dep Dx (W6) (β = 0.001).Family SU (W1) → peer SU (W3) → partner SU (W5) → SA/Dep Dx (W6) (β = 0.009). $ES_{avg.}$ = 0.003
Lee et al., 2015	4	W1: 21.3 yrs. (average) W2: 24.5 W3: 29.0 W4: 34.3	W1: 465 students - Control: 236 - Alcoholic: 229 W2: 451 W3: 404 W4: 377	47% Female 86% Euro Am 5% Af Am 1% Asian 8% Nat Amer	Y-SRQ,Y-DI (parental alcoholism) , FH-RDC for AUD	- ARCL with DTSA + TVC/FIML - Yes - Yes	Family AUD (W1) → disinhibition (W1) → role transition (W2) → problem drinking (W3) → problem drinking (W4) (β = 0.016).Family AUD (W1) → conscientiousness (W1) → role transition (W2) → problem drinking (W3) → problem drinking (W4) (β = 0.019). Models also trimmed of AUD (W1) significant:Disinhibition (β = 0.205); Conscientiousness (β = -0.009). $ES_{avg.}$ = 0.06
Lynne-Landsman et al., 2010	9	W1: 1st grade W2: 3rd W3: 7th W4: 8th W5-W8: 9th-12th W9: 21 yrs.	678 families	46.8% Female 86.8% Af Am 13.2% Euro Am	P-/T-SRQ + I, Y-/P-/T-SRQ, Y-CAPI, school/court records	- GMM/FIML, LGR - No - Yes	Many early childhood measures (defiant or aggressive behaviors) were not indirectly related to early adolescent measures (SU latent class membership) or distal young adult outcomes. Effect sizes for significant indirect effects linking early childhood to 3rd grade outcomes or 7th and 8th grade behaviors were relatively small ($\beta_{avg.}$ = 0.08). $ES_{avg.}$ = 0.07
Rogosch et al., 2010	4	W1: 7–9 yrs. W2: 10–12	415 children - Control: 156-Maltreated: (W4: 259)	39.5% Female Ethnically diverse – 55.2% Af Am	Y-SRQ + I, Y-/T-SRQ, P-IPI, state records,	- SEM - No - No	Maltreatment status (<age 8) → IBP (W1) → EBP (W2) → CAD (W3) → CAD (W4) (β = 0.0015).Maltreatment status (<age 8) → IBP (W1) → EBP (W2) → EBP

(*continued on next page*)

Table 1 (*continued*)

Study Authors	# of Waves	Age[1]	Sample Size	Sample Info[2]	Assessment Protocol[3]	Analysis[4] Power Mediation[5]	Findings[6,7]
		W3: 13–15 W4: 15–18		− 31.6% Euro Am − 13.3% other	peer nominations,Y-DI (youth alcoholism)		(W3) → CAD (W4) (β = 0.002). Maltreatment status (<age 8) → IBP (W1) → IBP (W2) → IBP (W3) → CAD (W4) (β = 0.006). $ES_{avg.}$ = 0.003
Sitnick et al., 2014	7	W1: 1.5 yrs. W2: 2 W3: 3.5 W4: 11 W5: 12 W6: 15 W7: 17	W1: 310 families W4-5: 276 W6: 270 W7: 251	100% Male Ethnically diverse − 53% Euro Am − 36% Af Am − 6% other	OBS of mother–child interactions, P-SRQ + I, Y-SRQ, peer-SRQ, HV/Lab tasks	- SEM - No - Yes	Nurturance (W2) → parental knowledge (monitoring + disclosure) (W6) → SU (W7) (β = 0.11).Childhood EBP (W2,3) → parental knowledge (W6) → SU (W7) (β = -0.14).Mother Dep Sx (W1-3) → adolescent EBP (W4,5) → parental knowledge (W6) → SU (W7) (β = 0.04).Childhood EBP (W2,3) → adolescent EBP (W4,5) → parental knowledge (W6) → SU (W7) (β = 0.04). $ES_{avg.}$ = 0.08
Wang & Fredricks, 2014	4	W1: 7th grade W2: 9th W3: 11th W4: 1 year post high school	W1: 1272 families W2: 1157 W3: 1084 W4: 997	51% Female Ethnically diverse − 58% Af Am − 36% Euro Am − 6% other	Y-/P-SRQ, school records	- SEM/FIML, LGM, controlled for ICCs - No - No	SU (W1) → behavioral engagement (W2); SU (W2) → behavioral engagement (W3). Behavioral engagement (W1) → SU (W2). Behavioral engagement (W2) → SU (W3). Emotional engagement → SU (W2 & W3). SU (W1) → engagement (W2). SU (W2) → emotional engagement (W3). $ES_{avg.}$ = 0.01

[1] **Age**: FU = Follow-Up; W = Wave; Yr = Year.

[2] **Sample Info**: Af = African; Am = American; Euro = European.

[3] **Assessment Protocol**: AUD = Alcohol Use Diagnosis; CAPI = Computer-assisted personal interview; DI = Diagnostic Interview; FH-RDC – Family History – Research Diagnostic Criteria; HV = Home Visit; IPI = In-Person Interviews; OBS = Observations; P = Parent; SRQ = Self-Reported Questionnaire; SRQ + I = Self-Reported Questionnaire in an Interview Format; T = Teacher; TI = Telephone Interview; WB = Web-Based; Y = Youth.

[4] **Analysis**: ARCL = Autoregressive Cross-Lagged Model; ARLT-SR = Autoregressive Latent Trajectory with Structured Residuals; DTSA + TVC = Discrete Time Survival Analysis with Time-Varying Covariates; FIML = Full Information Maximum Likelihood; GMM = Growth Mixture Modeling; ICC = Intraclass Correlation; LGM = Latent Growth Model; LGR = Logistic Regression; LR = Linear Regression; MI = Multiple Imputation; SEM = Structural Equation Modeling; TPSC = Two-Part Semicontinuous.

[5] **Mediation**: Mediation analyses included with decomposition of effects based on standard practice. Not all effects listed are significant.

[6] **Findings**: Δ = Change; AC = Academic Competence; CAD = Cannabis Abuse and Dependence; Dx = Diagnosis; Dep = Depression; EBP = Externalizing Behavior Problems; ESavg = Effect Size, averaged across multiple pathways; IBP = Internalizing Behavior Problems; SA = Substance Abuse; SU = Substance Use; Sx = Symptoms.

[7] Discussion of findings are limited to whether evidence of cascade effect was found (reciprocal and transactional effects controlling for stability) and comported with explicit hypotheses. Numbers in parentheses (β) is indirect effect.

(e.g., focal child, parent, teacher, peers) and modeled an average of 3.22 target measures (*SD* = 1.26). However, several studies modeled latent variables with multiple indicators (e.g., Elam et al., 2016; Espelage et al., 2014; Sitnick et al., 2014), created composite substance use measures (e.g., Brody et al., 2010; Haller et al., 2010; Hsieh et al., 2015), or used diagnostic nosology for abuse and/or dependence (e.g., Jones et al., 2016; Rogosch et al., 2010).

About two-thirds of the studies (14 out of 18) used full information maximum likelihood (FIML) estimation for statistical treatment of incomplete data and thus reported no attrition. One study (Espelage et al., 2014) used multiple imputation but failed to indicate how many imputations or what auxiliary variables were included in the estimation. The average indirect effect size across all studies (averaging indirect effects within studies with multiple outcomes) was β = 0.046 (*SD* = 0.046), reinforcing a relatively small effect for the length of time covered and the multiple hypothesized pathways.

In order to ascertain whether study design features influenced effect sizes, we conducted several non-parametric tests predicting the average effect sizes (effect sizes in studies with multiple outcomes were averaged). Predictors included several study design characteristics including the arithmetic subtraction of beginning age from the follow-up age, the number of waves, % white, % female, the number of reporting sources, the follow-up sample size, and the number of target measures. These are exploratory analyses; however, they may point to structural differences in the studies that can account for variability in the effect sizes. For continuous and ordinal predictors, we used a Spearman rank-order correlation (ρ) with bootstrapping initially (Sheskin, 2003). For nominal predictors (with two categories), we used a Mann-Whitney *U* test (*U*) initially. If any results were statistically significant, we analyzed predictors and effect sizes using a nonparametric regression (scatterplot smoothing). For all tests, we used bootstrapping with 5000 samples and bias-corrected accelerated intervals (BCa) (with ρ) or exact tests (with *U*) to determine statistical significance. We modeled each predictor separately one by one to avoid suppression.

The results indicated that neither the difference between beginning and ending ages of individuals in the various studies, $\rho = -0.22$, BCa 95% Confidence Interval (−0.66, 0.34), nor age as a dichotomous measure (greater or less than 10 years), $U = 33.00$, $p = 0.53$, was significantly related to variation in effect sizes. The total number of waves of data used in the studies was not significant, $\rho = -0.27$, BCa 95% CI (−0.72, 0.26), and likewise the percent white individuals was not significant, $\rho = 0.02$, BCa 95% CI (−0.49, 0.55). Percent female composition was not significant, $\rho = 0.13$, BCa 95% CI (−0.30, 0.56) with the same result modeling a dichotomous measure (percent female greater or less than 50%), $U = 0.30.50$, $p = 0.63$. The number of sources providing information was not significant, $\rho = 0.27$, BCa 95% CI (−0.28, 0.75), and this did not change when the number of sources was modeled as greater or less than one, $U = 34.00$, $p = 0.62$, greater or less than two, $U = 21.00$, $p = 0.12$, and greater or less than three, $U = 21.00$, $p = 0.12$. The same held for sample size in the studies, $\rho = -0.40$, BCa 95% CI (−0.71, 0.02), and the number of targeted measures, $\rho = 0.24$, BCa 95% CI (−0.40, 0.70).

4. Discussion

To our knowledge, this is the first systematic review to organize empirical studies that examined substance use etiology using a developmental cascade framework. Although this review focused exclusively on substance use, many of the findings are relevant to the developmental cascade literature more generally. In this respect, the studies reviewed point to specific advances in understanding human development because they involved multiple sources of influence, included prospective, longitudinal designs covering substantial portions of the lifespan, and often used multiple informants (e.g., parent, teacher, child, peers). In addition, all of the studies blended diverse explanatory systems to account for the risk mechanisms contributing to substance use. Some studies went beyond a focus on the early stages of substance use by examining factors contributing to diagnoses of abuse/dependence in young adulthood. This is a strength of the cascade approach and is particularly important for understanding substance use etiology as it captures multiple sources of influence that cumulatively build, exert their effects over an extended period of time, and manifest in maladaptive behaviors later in life that take their toll on human functioning and well-being.

4.1. Conceptual demands of the developmental cascade model

Despite growing application in developmental studies, cascade models face several challenges. First, the research design must be methodologically rigorous so as to capture as many appropriate and critical "developmental junctures" as possible. It is incumbent on the investigator to space assessments appropriately in order to detect cross-domain influences that may be specific to a particular developmental period. For instance, the transition from middle to high school is considered stressful because of changes in the school context and pedagogical structure as well as the increasing influence of peer networks (Barber & Olsen, 2004; Eccles et al., 1991). Obtaining data before, during, and even after these transitions can be vital to explain the effects of role transitions and peer influences as intervening mechanisms that contribute to substance use in early adolescence and beyond.

Second, even when studies designate developmental relations that are appropriately timed, there is a chance that hypothesized paths may not be significant. For instance, some studies specified relations between parenting and child or youth development as both reciprocal (cross-domain, within-time) and transactional (cross-domain, over time), but failed to obtain significant results (e.g., Danzo et al., 2017; Elam et al., 2016; Hsieh et al., 2015). Other studies noted a cross-domain effect linking adolescent substance use with deviant peer associations perhaps reinforcing a peer selection mechanism. However, in attempting to explain why youth select deviant peers, the same studies were not able to find significant relations linking family environment (i.e., family conflict, parental substance use) with any downstream effects (e.g., Brody & Ge, 2001; Sitnick et al., 2014). Conceivably, parental fighting and conflict encourages youth to spend more time outside of the home to avoid escalating conflict. Disruption of the home environment and lack of parental monitoring may precipitate a peer selection mechanism that can account for substance use etiology.

4.2. Difficulty modeling risk and vulnerability

In addition to parental monitoring as one contributing factor, other family processes may be involved to account for downstream effects. One, in particular, is the role of parental alcohol or substance abuse, which can sustain a more expansive set of adverse child rearing conditions. Notably, homes with parental substance abuse are often rife with family conflict, harsh and inconsistent parenting, and low emotional support (Eiden et al., 2016; Elam et al., 2016). Children of alcoholics are frequently exposed to parental psychopathology as well as deviant role models (e.g., Chassin et al., 1999; King & Chassin, 2004). However, it is interesting to note that even though Haller et al. (2010) found adverse effects of parental alcoholism on later youth substance use, the path was still meted through affiliation with substance-using peers, reaffirming the importance of deviant peer associations as part of the developmental process linking early harsh parenting and later substance use.

These findings come on the heels of considerable evidence linking peer deviance and peer affiliation with substance use etiology (Pandina et al., 2010). Peers act as sounding boards and also represent social learning mechanisms through which youth adopt certain behaviors. Their prominence is consistent with personality theorists who suggest that peers provide interactional continuity that enhances person-environment fit (Roberts & Caspi, 2003). In other words, troubled youth who experience rejection in environments promoting conventional behavior (e.g., home, school) select social groups that support their interactional styles, which involves rejection of conventional norms. This view is also consistent with problem behavior theory (Jessor & Jessor, 1977), which suggests that certain personality characteristics including social criticism and alienation act as "instigations" that, in the absence of controls and regulations promoting conventionality, lay the foundation for deviant peer associations. Associations with deviant peers provide the desired support, acceptance, and sense of belongingness that enhance the self-esteem of troubled and disenfranchised youth (Vega et al., 1996). Thus, it makes sense that half of the studies reviewed included some measure of deviant peer affiliations and demonstrated that these associations mediated the effects of family socialization on later substance use. Other intermediate factors that were examined as bridges between early parenting and later adolescent substance use included intrapersonal and interpersonal factors such as impulsivity, self-regulation, self-esteem, and social competence (Brody & Ge, 2001; Elam et al., 2016; Lee et al., 2015; Rogosch et al., 2010), as well as internalizing and externalizing behaviors (Englund & Siebenbruner, 2012; Lynne-Landsman et al., 2010; Sitnick et al., 2014). In all of these instances, the sequential dynamic process involving a causal mediation chain, which is the backbone of developmental cascades, offers a more ecologically valid explanation of substance use etiology.

4.3. Methodological limitations and concerns raised by the review

The findings from this systematic review also raise several additional methodological concerns. By their very nature involving extensive longitudinal follow-up, cascade studies inevitably face issues related to sample size, attrition, and power. This was evidenced by relatively small samples with more than a half of the studies having fewer than 400 participants at follow-up. A small sample with a relatively large number of model parameters strains the robustness of the statistical methods used to model cascade effects. Moreover, it is well established that participating families or children who experience adverse cascade effects or drop out of studies may be financially poorer, less educated, socially marginalized, psychologically vulnerable (e.g., depressed), lacking in social or cognitive skills, and in poorer physical health, all of which increase their reluctance or difficulty to continue to participate (Gustavson et al., 2012; Weinberger et al., 1990). In the absence of these individuals, the final models underestimate the severity of risk, which can reduce the external validity of findings (Ahern & Le Brocque, 2005; Cox et al., 2010).

Numerous studies used the full-information maximum likelihood (FIML) procedures as a missing data treatment, which is most optimal when data is missing at random or completely at random (Schafer & Graham, 2002). An important component to using statistical treatment of missing data requires auxiliary measures that boost the available information in the covariance matrix for estimating missing data values (e.g., Collins et al., 2001). None of the studies reviewed acknowledged sample bias due to non-random patterns of subject loss (Little, 1995) or elaborated which measures were used to augment the imputation, increase power, and provide less biased and more accurate estimates of model parameters.

Moreover, power was not addressed in all but one of the studies (Lee et al., 2015). Both Kenny (1975) and Rogosa (1980) commented that CLPD is, by its very nature, a low power test and may have fallen out of favor for several reasons (Berry & Willoughby, 2017; Hertzog & Nesselroade, 1987). One reason is that CLPD is quite stringent with respect to estimating predictor effects over and above stability. When the within-domain stability over time is high, there is little residual variance to predict, and lagged cross-domain (transactional) effects tend to be small. The consensus among the studies reviewed was that there was moderate (>0.50) stability for most behaviors. Moreover, the presence of moderate to large autoregressive effects is further exacerbated by moderate within-time association between domains (i.e., reciprocal influences) at an earlier time point, which diminishes the magnitude of longitudinal "lagged" association. The addition of covariates and other control measures that account for meaningful variance in the outcome enhances precision but also further reduces the residual variance net of predictors. This is what led Adachi and Willoughby (2015) to suggest that relatively small effect sizes ($\beta = \sim 0.10$) may actually be meaningful when stability is high and that there is a moderate concurrent association between domains at an earlier point in time. Many of the studies in this review encountered this problem, suggesting that developmental cascades would appear trivially small but be quite meaningful when all else is considered.

With one exception (i.e., Espelage et al., 2014), investigators who specified latent constructs in their model did not test for longitudinal factorial invariance or differential item functioning (for dichotomous or polytomous measures) to establish measurement equivalence over time (e.g., Dimitrov, 2010). This is a critical oversight as constructs must have the same meaning over time in order to infer change in the underlying psychological processes. At the very least, imposing both metric and scalar invariance would rule out confounding by changes in the meaning of the factor (specifying equivalent factor loadings across time) or the mean structure of items over time (specifying equivalent indicator intercepts over time).

Suppression was also evident in several of the studies. Suppression occurs in multiple regression or path analysis when multicollinearity and criterion irrelevant variance are present in a model (Beckstead, 2012; Tzelgov, & Jenik, 1991). Suppression can be detected when the sign of an effect switches (from + to − or from – to +), or there is considerable diminishment of effect size between predictor and criterion with the introduction of a third variable. Simply stated, in suppression, a third variable "steps on" the predictive component and squashes relevant variance. In the studies reviewed, there were several instances of suppression (i.e., null effects or changes in the sign of effects) that remained unexplored. For instance, Danzo et al. (2017) reported a counterintuitive finding that more family conflict was associated with *less* alcohol use from 8th to 9th grade (after controlling for earlier alcohol use). Englund and Siebenbruner (2012) found that both the likelihood and separately level of alcohol use increased with *higher* academic competence. These effects run counter to the literature, where family conflict raises the risk of alcohol use and academic competence is protective lowering the risk of alcohol use. Haller et al. (2010) noted the possibility of suppression; however, they did not test this further with statistical models to correct for multicollinearity, which involves teasing apart criterion-relevant from criterion-irrelevant variance with specific modeling techniques in a confirmatory framework (Beckstead, 2012).

Almost all of the CLPD models adjusted for time-invariant confounds at baseline without considering the potential time-varying nature of these relations. Race cannot change, but mother's education can, so can income, the number of siblings, and other demographic factors that are not immutable. Unlike its growth modeling counterpart, CLPD does not efficiently model time-varying covariates well, which are specified as static effects. Moreover, many of the cascade models reviewed did not observe the requirement for *stationarity*, choosing instead to model new developmental factors at different developmental periods. While this is an important element of modeling change over time given new developmental milestones that surface at different ages, it can complicate matters because new structural components representing causal mechanisms are introduced into the model.

4.4. Criticisms of CLPD

Many of the studies reviewed specified causal mediation chains to represent cascades as sequential "ripple" effects that entail one domain influencing another over time. This model structure is meant to capture perturbations of risk that eventually promotes negative developmental outcomes. These theoretical linkages are the bread and butter of cascade models, which exemplify the interaction of risk-engendering forces at multiple levels and across time. Notwithstanding the importance of these relations, there is growing dissatisfaction with CLPD to capture cascade effects in a methodologically rigorous manner. Rogosa (1980) and others (Hamaker et al., 2015) have criticized CLPD because it cannot appropriately model different "rates of change" from one point to another between different constructs. In other words, if one domain is changing quite rapidly (e.g., maternal harshness) and another domain changes at a much slower rate (e.g., child behavior), the relative velocity of these constructs is muted.

Related to this point, CLPD initially represented a means to establish causal predominance; in other words, it was designed to determine the relative magnitude of cross-domain (i.e., transactional) influences over time while controlling for stability. This is what Rogosa (1980) termed the "causal winner," although he went on to note the CLPD's deficiencies. Take, for example, a simple two-construct model positing maternal harsh parenting (X) and youth behavior problems (Y), framed in a multi-wave longitudinal panel design. This model would provide a means to determine whether X causes Y or Y causes X or whether these effects are reciprocal and of equal magnitude. While harsh parenting can conceivably contribute to youth behavior problems, a child can foster a negative or coercive feedback loop by acting out and exacerbate harsh parenting. The absence of any causal predominance for X or Y can also be attributed to spuriousness whereby a third variable causes both X and Y.

Only three of the 18 studies—Danzo et al. (2017), Davis et al. (2018), and Lee et al. (2015)—conducted precise tests of causal dominance in a manner consistent with developmental cascade hypotheses. They specifically tested whether autoregressive and cross-lagged paths differed in magnitude by time (i.e., testing stationarity) using nested model comparisons. The lack of more rigorous tests of dominance in the remaining studies is somewhat surprising. Indeed, all that is required is computing a χ^2 difference test relative to the degrees of freedom (df) contrasting a model constraining paths to equivalence nested with a model freely estimating these parameters.

Also of interest is that many models specified complex mediational chains to capture socialization effects on child or youth behavior. Positing transactional cross-domain effects with multiple waves necessitates statistically testing the significance of indirect effects using the product of coefficients method with 95% bias-corrected bootstrap confidence intervals for the product (MacKinnon, 2008; MacKinnon et al., 2004). As this review points out, half of the studies reviewed omitted this statistical test; therefore, all we can determine from the path coefficients between any two constructs without mediation being explicitly tested is that one domain influenced another in a temporally related manner. Without formally testing the sequence of mediating effects, we are left unable to determine whether the intervening mechanisms hypothesized to be responsible for cascading effects on the outcome are indeed significant (Cole & Maxwell, 2003). Returning to an earlier point regarding small samples, there are now methods available that involve resampling strategies with multiple mediators, which can be used to effectively determine the relative dominance of cross-lagged mediational paths (Preacher & Hayes, 2008).

4.5. Conclusion and recommendations for future studies

Although the use of developmental cascade models to account for (mal)adaptive development has been steadily growing, its application is not without drawbacks. The combination of small samples, high attrition, complex multilevel models, and the lack of statistical remedies to address artifacts of multicollinearity presents hurdles that can limit the heuristic utility of these models. New and quite powerful analytic techniques are becoming more widely available that can address many of the methodological pitfalls discussed in this article (e.g., Little et al., 2007). Included in these techniques is modeling growth with structured residuals. This provides a novel approach to handle data with repeated measures and gives investigators another tool to frame developmental processes (Curran et al., 2014). One feature of growth models with structured residuals is the ability to map different components of change, allocating them to within- and between-person components. In the traditional world of CLPD, stability effects represent between-person variation, but lost to this modeling process is within-person change. People change over time not only relative to other people, but relative to their own initial levels and their rate of growth. Modeling a person's relative position to the overall group mean is only one facet of development. A person's own rate of growth over time (and their initial beginning point) also influences the behaviors they may engage (e.g., drinking alcohol) because it reflects their personal psychological milieu. Not everyone starts at the same level of family dysfunction, nor do they share the same rate of change in externalizing behaviors. Some youth may even overcome the effects of a negative family environment through various protective mechanisms that offset vulnerability. The ability to blend between- and within-person information (i.e., fixed and random effects) to understand change is a crucial piece of evidence needed for cascade models.

In addition to modeling nuances, there is also tremendous emphasis in cascade models to select the right set of factors that convey risk across time. The studies reviewed here point to a myriad of factors that may be involved in the etiology of substance use including family socialization (e.g., family conflict, harsh vs. nurturing/warm parenting, monitoring), children's capacity for self-regulation or lack thereof (e.g., behavioral disinhibition, impulsivity), internalizing problems (e.g., depression), externalizing problems (e.g., bullying perpetration, aggression), deviant peer associations, and academic achievement, all of which exert some influence on later substance use. Even genetic risk should be considered as part of the cascade as it can pave the way for creating contexts that evoke maladaptive behaviors (Elam et al., 2016). Unfortunately, lacking from all of these models is a cohesive framework that links the different processes into a unified whole to account for substance use etiology. By necessity, models of this scope and magnitude require large samples, tracked longitudinally from a very early age, and with minimal attrition to avoid biased results. This type of research is costly in terms of both time and money, albeit necessary in order to increase the field's knowledge of developmental etiology. Regardless of the precise risk mechanisms involved (e.g., equifinality, multifinality), a more comprehensive model is required to account for the derailment of a youth's life into a world of substance use and even abuse. Despite these concerns, we are cautiously optimistic that with continued methodological refinements and more precise specification of theory-driven models, results of longitudinal panel studies will converge on the simple premise that a great deal of adversity encountered in the home, neighborhood, or school can place some individuals at risk and detract from normal healthy development.

CRediT authorship contribution statement

Lawrence M. Scheier: Conceptualization, Methodology, Software, Formal analysis, Writing – original draft, Supervision. **Aya Shigeto:** Data curation, Visualization, Investigation, Software, Validation, Writing – review & editing.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.abrep.2022.100420.

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