J Youth Adolescence (2017) 46:1515–1532
DOI 10.1007/s10964-017-0676-6

EMPIRICAL RESEARCH

Effect of Victimization on Impulse Control and Binge Drinking among Serious Juvenile Offenders from Adolescence to Young Adulthood

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Received: 9 February 2017 / Accepted: 11 April 2017 / Published online: 24 April 2017

Abstract A vast literature has found longitudinal effects of early life stress on substance use and self-regulatory processes. These associations may vary by period-specific development among youth involved in the juvenile justice system. The current study used an accelerated longitudinal design and auto-regressive latent trajectory with structure residuals (ALT-SR) model to examine the within-person cross-lagged associations between binge drinking, impulse control, and victimization from 15 to 25 years of age. A large sample ($N = 1100$) of justice-involved youth were followed longitudinally for 7 years ($M_{age\ baseline} = 15.8$, $M_{age\ conclusion} = 22.8$). In general, the sample was ethnically diverse (41% Black, 34% Hispanic, 21% White, 4.3% Other) and primarily male (87.2%). Participants reported on their frequency of binge drinking, impulse control, and frequency of victimization at each time point. The results indicated that, during adolescence, victimization and binge drinking attenuated impulse control, which resulted in more binge drinking and victimization during young adulthood. The current study highlights the importance of assessing developmental processes and period-specific transitions among at risk youth, especially for youth experiencing early life stress.

Keywords Stress · Child maltreatment · Alcohol use · Impulse control · Self-Regulation · Emerging adulthood

Introduction

Deficits in behavior, such as impulse control, place adolescents and young adults at greater risk of early (and continued) use of alcohol and other drugs (Carroll et al. 2006). More specifically, youth with early onset of problem behavior consistently report higher alcohol and drug use (DeWit et al. 2000), violent behavior (Champion et al. 2004), and impulse control problems (Carroll et al. 2006). This is important as adolescents and young adults have the highest rates of alcohol use (12% and 60%, respectively) and binge drinking (6 and 38%, respectively) in the United States (SAMHSA 2013). Further exacerbating the problem, experiences of early victimization (e.g., abuse, exposure to violence) has also been identified as a predictor of adolescent and young adult substance use (Kilpatrick et al. 2003). As such, a vast literature has established long term effects of early victimization on psychosocial functioning, neurological dysfunction, and binge drinking (Shonkoff and Garner 2012). Unfortunately, youth in the juvenile justice system have rates of substance use five times higher than community youth (Grisso and Underwood 2004) and over 90% of youth in the juvenile justice system (compared to 60% of community youth) report experiencing at least one violent event in their lives (Finkelhor et al. 2015; Ford et al. 2013). Further, justice involved youth with early onset of problem behaviors score lower on impulse control tasks (Carroll

Electronic supplementary material The online version of this article (doi:10.1007/s10964-017-0676-6) contains supplementary material, which is available to authorized users.

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et al. 2006) and have higher prevalence of substance use and victimization. Thus, it is important to investigate these relationships among at-risk youth using a developmental perspective.

Theoretical and Conceptual Models regarding Victimization and Impulse Control

Broadly, the seminal work by Gottfredson and Hirschi's (1990) *General Theory of Crime* aimed to explain deviant and criminal behavior in the context of deficiencies in self-control (i.e., impulse control), and has been one of the most widely cited theories in the justice-criminology literature (Pratt and Cullen 2000). Gottfredson and Hirschi (1990) argue that because criminal and analogous behaviors (e.g., binge drinking) are easy to conduct and immediately gratifying, those with diminished self-control are at greater risk for engaging in behaviors such as binge drinking. In an early meta-analysis, Pratt and Cullen (2000) provided evidence for Gottredson and Hirschi's general theory of crime and the association between diminished self-control and criminal behavior ($r = .23$) and analogous behavior (e.g., drug and alcohol use; $r = .35$). Other studies have also tested this theory to explain high-risk behaviors including drinking and driving (Keane et al. 1993) and binge drinking among college students (Gibson et al. 2004). In addition to the General Theory of Crime, larger, more overarching theoretical/conceptual models such as *developmental transitions and trajectories* (Havighurst 1948) help researchers understand variations that exists across the life span (see Schulenberg and Maggs 2002 for review). For instance, transitions refer to the actual process of change whereas trajectories are defined as patterns of systematic and successive change over time (Elder 1998). Thus, we seek to understand how certain transitions (e.g., adolescence to young adulthood) are embedded into individual trajectories (e.g., binge drinking) that help point to important periods of life, which may reflect increases or decreases in functioning (e.g., variation in binge drinking trajectories based on age related transitions) (Rutter 1996). Hirschi (2002) posited that an individual's level of self-control will increase as youth transition into adulthood. While increases in self-regulatory behavior such as impulse control have been shown in prior literature (Harden and Tucker-Drob 2011), we seek to understand how factors such as exposure to early life stress (Shonkoff et al. 2009) and early engagement in alcohol use may affect variation in impulse control processes and influence long-term developmental trajectories and period-specific transitions.

Keeping this larger framework in mind, there are two additional theories that aid our understanding of how early exposure to stress may alter impulse control processes and influence long-term dysfunction. First is the *allostatic load* model (McEwen 2012; Shonkoff et al. 2009), which posits that the amount of stress an individual experiences over time contributes to pathogenic outcomes (Juster et al. 2011). Second is the *self-control strength model*, which posits that self-control is a finite resource and, once depleted, leads to impaired self-control (Baumeister and Vohs 2003; Muraven and Baumeister 2000). It may be that excessive stress stemming from victimization may lead to diminished impulse control, which may in turn affect an individual's substance use behavior. While early life stress is a relatively broad construct that encompasses many forms, including indirect stressors (e.g., witnessing a violent act), physical stressors (e.g., car accident), and direct stressors (e.g., neglect, physical abuse, victimization) (Shonkoff and Garner 2012; Widom et al. 1999), the aim of this current study is to examine how one particular form of stress (i.e., victimization) affects subsequent impulse control and binge drinking.

The allostatic load model underscores the importance of long-term, frequent, and prolonged exposure to stressful life experiences and the body's repeated neuroendocrine response (e.g., the way our body responds to stressors). The allostatic load model represents this "wear and tear" (Juster et al. 2011; McEwen and Stellar 1993) on the body and highlights the over-activation of the stress response system (e.g., sympathetic-adrenal-medullary (SAM) and the hypothalamic pituitary adrenal (HPA) axis) (Korte et al. 2005; Sapolsky et al. 1997). Prior studies have found that among low income youth, chronic exposure to stress mediates the association between poverty and allostatic load in young adulthood (Evans and Kim 2012). This chronic exposure to stress can result in dysregulation of multiple physiological systems that predict deleterious outcomes including cognitive functioning, cardiovascular disease, mortality, impulse control problems, and substance use (Romeo and McEwen 2006). Recently, Evans and Kim (2012) explained that chronic exposure to stressors during childhood and adolescence (specifically among low-income youth) leads to disruption in self-regulatory processes (e.g., impulse control) that aid in coping with external and acute stressors. Several studies have echoed these findings such that adolescents tend to have higher concentrations of cortisol (HPA axis) when exposed to stressors (Gunnar et al. 2009).

In addition to the stress response system (e.g., HPA axis), exposure to stress also affects the prefrontal cortex, which serves as the area of the brain responsible for impulse control (Pechtel and Pizzagalli 2011). Prior studies have found prefrontal cortex dysfunction to be a phenotype important for the neural basis of addiction, and is associated with impulsivity which increases risk of alcohol neurotoxicity (Reynolds 2006). For example, studies of adolescents with alcohol use disorder show smaller white and grey matter in the prefrontal cortex compared to adolescents

without alcohol use disorder (De Bellis et al. 2005). Exposure to trauma, especially during adolescence when the brain in maturing and developing (Steinberg and Morris 2001), can lead to a cascade of negative events (McEwen 2003). This may be especially true for youth involved in the justice system as they have experienced extensively more trauma than community samples, and the biological adaptation that occurs (e.g., impairment of HPA axis and prefrontal cortex) may make them more prone to impulsive decision making, heightened emotional reactions, and disorganized coping styles (Ford and Blaustein 2013; Ford et al. 2008).

Complementing the allostatic load model is the self-control strength model (Baumeister and Vohs 2003). Briefly, self-control is the ability to override urges or desires and is otherwise used to maximize the long-term best interests of an individual (Agnew et al. 2011). The self-control strength model balances on the tenet that each time an individual self-regulates, they are drawing on a resource that, once depleted, results in reduced capacity to regulate emotions or impulses (Hagger et al. 2010). Thus, when an individual exerts repetitive efforts to self-regulate behavior, attempts thereafter should, in theory, fail, indicating a lapse in time when decision-making and impulsivity may play a larger role in problematic behaviors. For example, a recent meta-analysis found significant effects of impulse control depletion for self-control tasks, effort, negative affect, and blood glucose (Hagger et al. 2010). Others have tested this model on alcohol consumption, and have found that exerting self-control (prior to alcohol consumption) resulted in higher levels of alcohol use (Muraven et al. 2002). Considering the influence of chronic exposure to stress has on both physiology and behavior, it follows that youth exposed to heightened stress may be exerting more self-control than those not experiencing stress. Subsequently, these same individuals may have dysregulated impulse control processes that may relate to neurological dysfunction (e.g., prefrontal cortex) and increased substance use.

Empirical Studies on Exposure to Violence, Impulse Control, and Substance Use

Over the past two decades, an influx of research has focused on the behavioral and physiological effects of early trauma and victimization (Shonkoff et al. 2009; Shonkoff and Garner 2012). Specifically, prior research has found that youth with higher levels of exposure to violence (specifically victimization) are at heightened risk for a myriad of problem behaviors such as delinquency, early maturity, dampened impulse control, and substance use (Arseneault et al. 2006; Garner et al. 2014). Early stress exposure has been linked with past year binge drinking among adolescents (Hamburger et al. 2008) and victimized adolescents are at heightened risk for engaging in other high-risk behaviors (Kilpatrick et al. 2000). This is especially true for youth with early-onset of problem behaviors (Egeland et al. 2002; Moffitt and Caspi 2001). Others have found that victimization during young adulthood is associated with heightened social risk (e.g., deviant peers) and more substance use (Davis et al. 2015). Prior literature on justice involved youth has shown a link between early stress exposure with dissociative symptomology and substance use (Carrion and Steiner 2000; Ford et al. 2010), and re-victimization (Finkelhor et al. 2007).

While research has broadly unearthed evidence regarding relations between victimization and adverse behavioral outcomes, studies have also found associations between exposure to violence and problems with impulse control, in both behavioral and neurological research (Andersen et al. 2008; Monahan et al. 2015; Teicher et al. 2006). For example, behavioral evidence suggests that youth who are exposed to violence or are victimized during adolescence are less likely to develop self-regulatory skills including impulse control (King et al. 2013) and have lowered self-control during late adolescence and young adulthood (Agnew et al. 2011). Similar results have been found when assessing the concurrent or long-term effects of exposure to violence among young adults (Monahan et al. 2015). Prior evidence from neuroscience research shows that early life stress affects the prefrontal cortex, which is associated with impulse control (Thayer et al. 2009) in both animal and human studies. For example, early chronic stress has been shown to lead to poorer stress reactivity, impulse control, and HPA axis functioning in animals (Teicher et al. 2006) and young adults (Andersen et al. 2008). Similar results have been found among youth with early onset conduct problems (Speltz et al. 1999). It is theorized that those exposed to violence may become especially sensitive towards potentially threatening situations, which can be vital in the short term, but may have negative consequences on behavioral regulation and substance use in the long term (McCoy 2013). While behavioral impulse control increases during adolescence (Steinberg et al. 2008), the ability to properly self-regulate one's behavior can be altered when exposed to violence, which may have consequences for future alcohol use.

Disaggregating Two Levels of Effects

Prior studies have attempted to untangle the temporal effects of early life victimization, binge drinking, and self-regulatory processes (Begle et al. 2011; Fernie et al. 2013; Thompson et al. 2008). The most common method for testing reciprocal paths across developmental periods is the auto-regressive cross-lagged (ARCL) model. For example, Begle and colleagues (2011) found a full cross-lagged effect

between high risk behavior (drug and alcohol use) and victimization. Unfortunately, typical ARCL models yield estimates that are a combination of both between and within-person variance (Berry and Willoughby 2016). That is, when using longitudinal structural equation models (such as the ARCL) estimates are an odd amalgam of both within and between-person variance, thus making it difficult, if not impossible, to interpret. The combination of both levels of analysis into one is termed convergence, something that is incredibly rare in practice. For example, one would not assume that changes in binge drinking from an adolescent's "typical" level (i.e., individual mean) would be identical to changes in binge drinking compared to their peers (i.e., grand mean).

Current Study

The current study investigates the associations between victimization, binge drinking and impulse control from adolescence to young adulthood in a sample of early-onset justice involved youth. Prior literature has not considered these constructs simultaneously or among justice involved youth with early onset of behavior problems. We also extend the literature by using advances in modeling longitudinal data, employing the auto-regressive latent trajectory model with structured residuals (ALT-SR) (Curran et al. 2014). The ALT-SR model allows us to disaggregate and simultaneously consider *between-person* relationships (e.g., trait-like) among our variables while also modeling *within-person* (e.g., state-like) cross-lagged relationships during the transition from adolescence to young adulthood. This model sets our lagged process at a more developmentally appropriate level of analysis—*within-person*. Using this model will allow us to test, empirically, associations across three different developmental levels. That is, the ALT-SR separates the variance into three separate bins. The first bin is the portion of variance that does not change (e.g., the level or intercept), which aids in our understanding of basic differences across ages cohorts. The second is the portion of variance that changes over the entire span of the study (e.g., the slope), which aids in our understanding of basic individual differences in impulse control, victimization, and binge drinking. The third bin is the portion of variance that changes wave-to-wave (e.g., the within-person cross lags), which aids in our understanding of within-person fluctuations across adolescence and young adulthood.

Thus, the aim of the current study was to understand the effect of exposure to violence (e.g., victimization) on developmental trajectories of impulse control and binge drinking from adolescence through young adulthood. Because we were most interested in understanding the long-term effects of early exposure to violence on changes in behaviors, like binge drinking and impulse control, we sought to understand if these patterns were different for adolescents and young adults. Thus, the aim was to test three distinct hypotheses. First, in line with the allostatic load model, we sought to understand if high levels of exposure to violence in the form of direct victimization was associated with subsequent decreases in impulse control and increases in binge drinking (Hypothesis 1). Second, in line with both the allostatic load and self-control strength model, we assessed if heightened *individual* levels of victimization were associated with wave-to-wave decreases in impulse control (lower than average trajectories) and increases in binge drinking (higher than average trajectories) (Hypothesis 2). Finally, in line with the self-control strength model and the overarching theory of transitions and trajectories, we tested if early exposure to violence during adolescence was associated with incremental deficits in impulse control and if these deficits transitioned into young adulthood, resulting in a cascade of problematic behaviors (e.g., long-term effects of deficits in impulse control due to early victimization) (Hypothesis 3).

Methods

Participants

Data were from the *Pathways to Desistance Study*, a longitudinal study of serious juvenile offenders. Participants were enrolled in the study from two locations (Maricopa County, AZ and Philadelphia County, PA) between November 2000 and March 2003. Data collection ended in March of 2010. At baseline, participants were between the ages of 14–18 and 21–25 at study completion. To be eligible for this study, youth had to be charged with a felony crime or a similar non-felony crime (e.g., misdemeanor). All eligible participants in both locations who agreed to participate signed informed consents with their parent/legal guardian. All interviews used computer-assisted personal interview (CAPI) techniques. Overall, the study achieved an average of 89.5% retention. Additional details on the study design and methods can be found in Mulvey et al. (2004) and Schubert et al. (2004).

Our sample ($N = 1100$ out of 1354) included only those between the ages of 14–17 at baseline ($M_{age} = 15.7$, SD = 1.04) with early onset (14 years or younger) problem behaviors (e.g., delinquency, substance use). Participants (Table 1) were predominantly male (87%), low socio-economic status (e.g., 80% of parents held a high school degree or less), and ethnically diverse with 41% participants identifying as African American, 34% as Hispanic, 21% as White, and 4% as Other. Participants were 10 years old, on

Table 1 Baseline characteristics

	Total sample ($N = 1100$) M (SD) or *n* (%)
Demographics	
Age, in years	15.8 (1.04)
Male *n* (%)	958 (87.2)
White *n* (%)	229 (20.8)
Black *n* (%)	450 (41.0)
Hispanic *n* (%)	374 (34.0)
Other *n (%)*	47 (4.28)
Family/school *n* (%)	
Mothers education high school or less	831 (79.4)
Father drug problem	400 (46.5)
Mother drug problem	299 (28.5)
Enrolled in school	972 (71.9)
Psychiatric disorders *n* (%)	
Clinically diagnosed depression[a]	71 (6.57)
Clinically diagnosed anxiety[b]	56 (5.45)
Post-traumatic stress disorder[a]	66 (6.11)
Substance use	
Binge drinking (past 6 months)	3.4 (2.3)
Lifetime alcohol dependence *n* (%)	113 (10.7)
Lifetime drug dependency *n* (%)	163 (15.4)
Personality and victimization	
Impulse control	2.92 (.943)
Victimization	3.80 (1.91)

Ranges: Binge drinking (1.0–9.0); Impulse control (1.0–5.0); Victimization (1.0–6.0)

[a] Disorder was derived from the Composite International Diagnostic Interview

[b] Diagnoses were derived from the brief symptom inventory

average, at time of first offense. Finally, participants reported binge drinking at least once a month, on average.

Procedures

Data were collected over a period of 7 years with bi-annual assessments during the first 3 years and annually during the last 4 years. To ensure time was spaced evenly, we averaged data for 6 and 12 month (year 1), 18 and 24 month (year 2), and 30 and 36 month (year 3) follow-ups. Thus, our analysis included seven time points spaced 1 year apart. We used data from the baseline assessment in our propensity weighting analysis and as covariates in our statistical models. Because data were set up with naturally occurring cohorts (age), we used an accelerated longitudinal cohort design. This creates planned missing data such that participants only have data for the seven time points they are in the study allowing us to model development across 10 years (15–25 years old).

Measures

Across all measures when parent/caregiver data were available we took the mean between self-report and parent/caregiver report. We should note that parent/caregiver data were only available during the adolescent phase (ages 15–18), thus data during the young adult phase are self-report only. We have included additional analyses to check results with and without the use of parent/caregiver data (see "Additional Analyses and Alternate Models" below).

Victimization

We used the 6-item victimization subscale of the Exposure to Violence Inventory (Selner-O'Hagan et al. 1998). Example items include "have you been chased when you thought the person chasing you would hurt you in the past 12 months," "have you been sexually assaulted," and "have you been attacked with a weapon?" Reliability estimates range from $\alpha = .68$ to 0.79. Construct validity of the Exposure to Violence Inventory was found using item analysis, such that the most severe forms of violence were the least commonly experienced and when they were experienced youth had higher scores on the scale (Selner-O'Hagan et al. 1998). This type of validity confirms that differences in exposure to violence is not random, but due factors such as family functioning, lifestyle, neighborhood, and peer context. Higher scores indicate more victimization.

Impulse control

We used the impulse control subscale (6 items) from the Weinberg Adjustment Inventory (Weinberger and Schwartz 1990). Participants and parents/caregivers respond on a 5-point Likert scale ranging from "False" to "True." Example items include "I say the first thing that comes to my mind without thinking enough about it," "I should try harder to control myself when I'm having fun," and "I become "wild and crazy" and do things other people might not like." Reliability estimates range from $\alpha = .78$ to 0.81. The Weinberg Adjustment Inventory has also been correlated with the Minnesota Multiphasic Personality Inventory (MMPI) with a sample of incarcerated adolescent males (Farrell and Sullivan 2000) indicating good construct validity. Others have assessed content validity by assessing how the Weinberg Adjustment Inventory subscales relate to problem behaviors (e.g., delinquency) across multiple samples (Huckaby et al. 1998). Higher scores indicate more impulse control.

Table 2 Testing cohort differences using unweighted and weighed hierarchical linear models. Results for binge drinking

	Unweighted		Weighted	
	Model 1	Model 2	Model 1	Model 2
Fixed effects				
Intercept	1.43 (.107)*	1.38 (.131)*	1.41 (.091)*	1.39 (.096)*
Linear slope	.129 (.033)*	.153 (.041)*	.127 (.030)*	.138 (.038)*
Quadratic slope	.0001 (.005)	.0001 (.005)	.003 (.005)	.002 (.005)
Cohort 2	.431 (.132)*	.418 (.166)*	.420 (.116)*	.399 (.127)*
Cohort 3	.541 (.124)*	.601 (.156)*	.419 (.1164)*	.436 (.135)*
Cohort 4	.581 (.124)*	.674 (.156)*	.532 (.118)*	.619 (.134)*
Time*cohort 2		.007 (.032)		.012 (.036)
Time*cohort 3		−.037 (.030)		−.012 (.037)
Time*cohort 4		−.052 (.003)*		−.049 (.038)
Random effects				
Intercept within (L1)	1.15 (.096)*	1.15 (.096)*	1.04 (.086)*	1.04 (.086)*
Intercept between (L2)	−.041 (.021)*	−.040 (.020)*	−.058 (.018)*	−.058 (.018)*
Linear slope	.074 (.007)*	.074 (.007)*	.079 (.007)*	.079 (.006)*
Fit indices				
-2LL	27,628.6	27,624.5	29,897.2	29,894.4
AIC	27,648.6	27,650.5	29,917.2	29,920.4
BIC	27,698.6	27,650.5	29,967.3	29,985.4

Note: The models shown above are for unweighted (e.g., raw data) and weighted (e.g., with propensity weights applied). This preliminary step is used to assess cohort differences and determine if utilization of an accelerated longitudinal design is appropriate. Cohort 1 (age 14) was the reference group

Cohort 2 15 years old, *Cohort 3* 16 years old, *Cohort 4* 17 years old, *−2LL* −2 log likelihood, *AIC* Akaike Information Criteria, *BIC* Bayesian Information Criteria

Binge drinking

Binge drinking was assessed with a single item asking participants "in the past 12 months how often have you had 5 or more drinks at a time?" Participants responded to a 10-point scale ranging from "never" to "daily."

Control variables

Because our developmental trajectory analysis began with year 1 (baseline variables were used in a propensity weighing procedure) we controlled for Gender, Race/ethnicity, and baseline victimization, impulse control, and binge drinking. Baseline values for impulse control, victimization, and binge drinking were all measured as described above. Further, given this sample is comprised of juvenile justice youth and being incarcerated or placed in a secure confinement can influence victimization and drinking behavior, we controlled for the proportion of time spent in a controlled environment. Finally, all variables used in the propensity weighting procedure were also used as control variables in our models.

Analytic Approach

One of the major assumptions that accompanies accelerated longitudinal designs is each cohort can be considered part of the "same" cohort (e.g., single developmental trajectory). Given our cohorts were naturally occurring (e.g., age) we expected cohort differences to emerge as cohort 1 (14 year olds) are likely different in terms of binge drinking compared to, say, cohort 4 (17 year olds). Thus, we took an initial step and tested for cohort differences (Miyazaki and Raudenbush 2000) by testing several nested hierarchical linear models (Table 2). We started by testing nested models with: (1) linear and quadratic time and the cohort variable (model 1), and (2) linear time, quadratic time, cohort variable, and the interaction with cohort and time (model 2). In our unweighted model, we found a significant cohort by time interaction indicating cohort differences exist (see Table 2 unweighted models). To adjust for these cohort differences we used propensity weighting (Rosenbaum and Rubin 1983) procedures to adjust for differences across our cohorts. Typically, researchers use basic regression adjustment to account for differences across

individuals. More recently, procedures that use propensity weighting have been proposed to adjust for potential differences across groups. Because we have four cohorts (ages 14–17 at baseline), we used the R package TWANG McCaffrey et al. 2013; R. Core 2016) and the multinomial propensity scores (MNPS) function to control for imbalance across *multiple* groups. TWANG uses machine learning techniques, specifically the Generalized Boosted Model, which involves an iterative process using multiple regression tree logic to capture any nonlinear relationships between treatment assignment and any baseline variables (Friedman 2001; McCaffrey et al. 2004). Further, the Generalized Boosted Model method is superior to traditional logistic regression methods in terms of bias reduction (e.g., standard errors, mean-square error, variable selection) (Harder et al. 2010; Lee et al. 2009) and can be used to find the best balance between groups through its iterative procedure (McCaffrey et al. 2013). We used the R package TWANG to weight individuals across 24 baseline variables that included aspects such as early onset of problem behavior, parental variables (e.g., parental drug and alcohol use, family incarceration), mental health (i.e., major depressive disorder), and peer delinquency. We then we re-tested our hierarchal liner models using the weighted data. Results indicated no significant cohort differences (see Table 2 weighted models), indicating we can treat our data as one continuous cohort (age 15–25). We used the final propensity weight in all subsequent analyses.

Next, we fit a taxonomy of auto-regressive latent trajectory with structured residuals (ALT-SR) models (Curran et al. 2014) to examine the simultaneous between and within-person effects of victimization, binge drinking and impulse control across both adolescence and young adulthood. Using the ALT-SR specification, the between-person effects are captured by correlating (or regressing) our latent intercepts and growth parameters (represented by $\varphi_{standarized}$ below). Thus, the remaining within-person variance is "pushed" into the residual cross-lagged portion of the model (see Online Resources for full model). One advantage of the ALT-SR over traditional auto-regressive cross lag models is that we can capture variance that doesn't change (intercept), variance that changes over the course of the study (slope), and latent growth *within* person cross-lagged associations. This allows us to gain a more nuanced understanding of age specific developmental processes. To determine if the latent linear and quadratic growth parameters should vary randomly, we tested each separately using likelihood ratio tests. Finally, while our weighting procedure aided in producing equal cohorts to guard against any residual potential bias, we controlled for all 24 variables in our weighting analysis and participant gender, race, and time spent in the community in each of our ALT-SR models to ensure doubly robust estimation.

To understand specific developmental processes, we ran three separate models. First, Model 1established basic auto-regressive associations between our variables of interest. Second, Model 2 established an *overall* association between binge drinking, impulse control, and victimization where cross-lag effects were constrained to be the same across both developmental periods. Finally, in Model 3 we assessed the *developmental variation* in the magnitude of time-specific associations among binge drinking, impulse control, and victimization by constraining our within-person cross-lag effects to vary across adolescence (ages 15–17) and young adulthood (ages 18–25), separately. Fit statistics (Comparative Fit Index (CFI), Root Mean Square Error of Approximation (RMSEA), and Standardized Root Mean Square Residual (SRMR)) were used to assess improvement in model specification.

Though missing data were minimal (~10% across 7 years), we utilized full-information maximum likelihood (FIML) estimator in Mplus version 7.4 (Muthén and Muthén 1998–2012) for all analyses. FIML treats all observed indicators as latent factors and allows each person to contribute whatever data is available instead of removing individuals with missing data. To adjust for non-normality, all standard errors were bootstrapped (iterations = 10,000).

Results

All model results with parameter estimates and standard errors are in Table 3. Below we report standardized estimates (β) and unstandardized estimates(b). Between-person correlations (i.e., intercept and random slopes) are represented by $\varphi_{standarized}$ below.

Propensity Weighting

Overall, our propensity weighting resulted in reduced maximum standard effect sizes across all four cohorts (see Table 4). One of our matching variables, age first sold drugs retained an effect size above the suggested threshold of 0.25 (Stuart and Rubin 2008) at 0.34, but it was greatly reduced from the unweighted difference of 1.21. However, to ensure that estimates in our models were not due to unbalanced covariates, we included age first sold drugs along with the remaining 23 variables used in the propensity weighting as covariates to ensure doubly robust control. See Online Resources for additional propensity weighting output.

Between-Person Associations

Overall mean trajectories showed increases in binge drinking (B = .367, SE = .043, $p < .01$) and impulse control

Table 3 Associations between binge drinking, impulse control, and victimization from the ALT-SR model. Parameter (SE)

	Model 1[a]	Model 2[b]	Model 3[c]	
			Adolescence	Young adulthood
Within-Person cross-lags				
$Binge_t$ *on* IC_t	–	−.147 (.052)*	−.089 (.066)	−.180 (.056)*
IC_t *on* $Binge_t$	–	−031 (.008)*	−.028 (.014)*	−.025 (.006)*
$Binge_t$ *on* $Vict_t$	–	.001 (.049)	−.008 (.058)	.085 (.086)
$Vict_t$ *on* $Binge_t$	–	.020 (.007)*	.022 (.010)*	.033 (.009)*
IC_t *on* $Vict_t$	–	−.031 (.021)	−.066 (.033)*	−.009 (.033)
$Vict_t$ *on* IC_t	–	−.008 (.018)	−.012 (.022)	.001 (.030)
Auto-regressive				
$Binge_t$ *on* $Binge_t$	.343 (.030)*	.359 (.031)*	.314 (.043)*	.251 (.044)*
IC_t *on* IC_t	.249 (.025)*	.156 (.028)*	.220 (.031)*	.283 (.030)*
$Vict_t$ *on* $Vict_t$	.040(.020)*	.031 (.020)	−.004 (.043)	.020 (.031)
(Co) Variances (between-person)				
$Binge_{int}$ *with* IC_{int}	−.099 (.015)*	−.233 (.026)*	−.351 (.021)*	
$Binge_{int}$ *with* $Vict_{int}$	.048 (.014)*	.330 (.015)*	.224 (.13)	
IC_{int} *with* $Vict_{int}$	.034 (.004)*	−.161 (.009)	−.350 (.007)*	
$Binge_{int}$	1.98 (.380)*	2.04 (.393)*	1.71 (.344)*	
IC_{int}	1.62 (.211)*	1.28 (.281)*	1.59 (.222)*	
$Vict_{int}$	.455 (.121)**	.428 (.118)*	.368 (.148)*	
Residual (Co) variances				
$Binge_{cit1-cit7}$	.325 (.078)*	.275 (.076)*	.035 (.051)	
$IC_{cit1-cit7}$	.266 (.016)*	.197 (.038)*	.255 (.017)*	
$Vict_{cit1-cit7}$	.036 (.009)*	.035 (.011)*	.026 (.077)*	
Fit statistics				
-2LL	45,798.48	45,648.68	45,071.73*	
AIC	45,956.48	45,836.68	45,289.73	
BIC	46,341.46	46,294.76	45,820.90	
χ^2	2495.58	2345.78	1768.83	
df	79	94	109	
RMSEA[d]	.050	.049	.040	
SRMR[e]	.096	.090	.067	
CFI[f]	.787	.803	.900	

Note: Estimates for all control variables on all latent intercept, and linear growth parameters are not shown for readability

In the table above, subscripts identify time of measurement. For example, a single *t* indicates paths were constrained to be equal over time. Subscript *int* indicates latent intercept (mean level) to obtain between-person parameter estimates. Subscripts with an epsilon (*cit*) indicate residual variance measured from Time 1 to Time 7

[a] Model 1 includes estimates for autoregressive paths only

[b] Model 2 includes binge drinking, impulse control, and victimization. Model building results indicated both linear and quadratic slope of impulse control should vary randomly

[c] Model 3 includes all estimated cross-lag paths. Model building results indicated linear binge drinking and quadratic victimization should vary randomly [d]RMSEA (Root Mean Square Error) indices below 0.05 are considered to be representative of good model fit

[e] SRMR (Standardized Root Mean Square Residual) indices below 0.08 are considered to be representative of good model fit

[f] CFI (Comparative Fit Index) scores above 0.90 are indicative of good model fit

df degrees of freedom, *Binge* Binge drinking, *IC* Impulse Control, *Vict* Victimization

*$p < .05$

Table 4 Propensity score weighting results from TWANG

Variable	Unweighted				Weighted			
	Max ASMD	Min P	Max KS	Min KS Pval	Max ASMD	Min P	Max KS	Min KS Pval
Social support	0.173	0.094	0.088	0.409	0.128	0.23	0.08	0.576
Binge drinking	0.333	0.001	0.205	<0.001	0.039	0.711	0.03	1.000
Alcohol	0.474	<0.001	0.223	<0.001	0.094	0.376	0.049	0.973
Exposure to violence	0.631	<0.001	0.232	<0.001	0.123	0.245	0.073	0.678
Impulse control	0.100	0.291	0.071	0.639	0.115	0.315	0.087	0.469
Parent marital status	0.086	0.389	0.038	0.995	0.084	0.456	0.063	0.886
Proportion family jail	0.393	<0.001	0.252	<0.001	0.091	0.482	0.092	0.615
Mom drug use	0.278	0.008	0.113	0.129	0.138	0.25	0.046	0.994
Father drug use	0.184	0.089	0.108	0.244	0.104	0.344	0.058	0.955
Mom alcohol use	0.067	0.503	0.022	1.000	0.067	0.562	0.022	1.000
Family mental health	0.140	0.195	0.045	0.985	0.196	0.136	0.062	0.934
Major depressive disorder	0.092	0.361	0.022	1.000	0.022	0.841	0.005	1.000
Alcohol dependence	0.447	<0.001	0.098	0.234	0.167	0.161	0.037	1.000
Drug abuse	0.324	0.002	0.129	0.051	0.095	0.46	0.038	1.000
Early onset alcohol/ drug	0.107	0.302	0.019	1.000	0.137	0.266	0.024	1.000
Early onset steal	0.057	0.564	0.023	1.000	0.094	0.382	0.037	1.000
Early onset fight	0.179	0.064	0.086	0.374	0.178	0.119	0.085	0.571
Psychosocial maturity	0.109	0.274	0.095	0.318	0.054	0.61	0.063	0.835
Age first sold drugs	**1.208**	<0.001	0.467	<0.001	**0.337**	0.059	0.162	0.339
Age first offense	0.265	0.010	0.088	0.368	0.228	0.054	0.090	0.505
Age first suspended	0.305	0.003	0.146	0.024	0.142	0.250	0.088	0.588
Socio economic status	0.088	0.403	0.067	0.747	0.052	0.624	0.044	0.992
Stoop test	0.117	0.264	0.018	1.000	0.213	0.187	0.033	1.000
IQ	0.058	0.550	0.08	0.462	0.063	0.570	0.063	0.951

Note: All variables listed above were used as covariates in our ALT-SR models to ensure doubly robust control. Values for Max ASMD are compared across unweighted and weighted variables. Here, we can assess how well the weighting analysis equated individuals across all cohorts. Lower ASMD values indicate a reduction in the standardized mean difference across cohorts. Bold indicates the only variable that was above the recommended standardized mean difference of 0.25

Max ASMD maximum absolute standardized mean difference, here reflecting the maximum difference from all group pairings, *Min Pval* *p*-value associated with the maximum ASMD value, *Max KS* maximum Kolmogrov-Smirnov test statistic of all group pairings, *Min Pval* value associated with the maximum KS value

(B = .492, SE = .0128, $p < .01$), and decreases in victimization (B = −.021, SE = .018, $p = .38$).

The intercept and slope factors represented by the latent growth model indicated moderate to strong associations for between-person binge drinking, victimization, and impulse control in our overall model (see Table 3). In line with our first hypothesis (Hypothesis 1), between-persons, individuals who had high impulse control were less likely to engage in binge drinking ($\varphi_{standarized} = -.23$) and less likely to experience victimization ($\varphi_{standarized} = -.16$). Further, individuals who experienced more victimization were more likely to engage in binge drinking ($\varphi_{standarized} = .33$). Similar results were found in our developmental model.

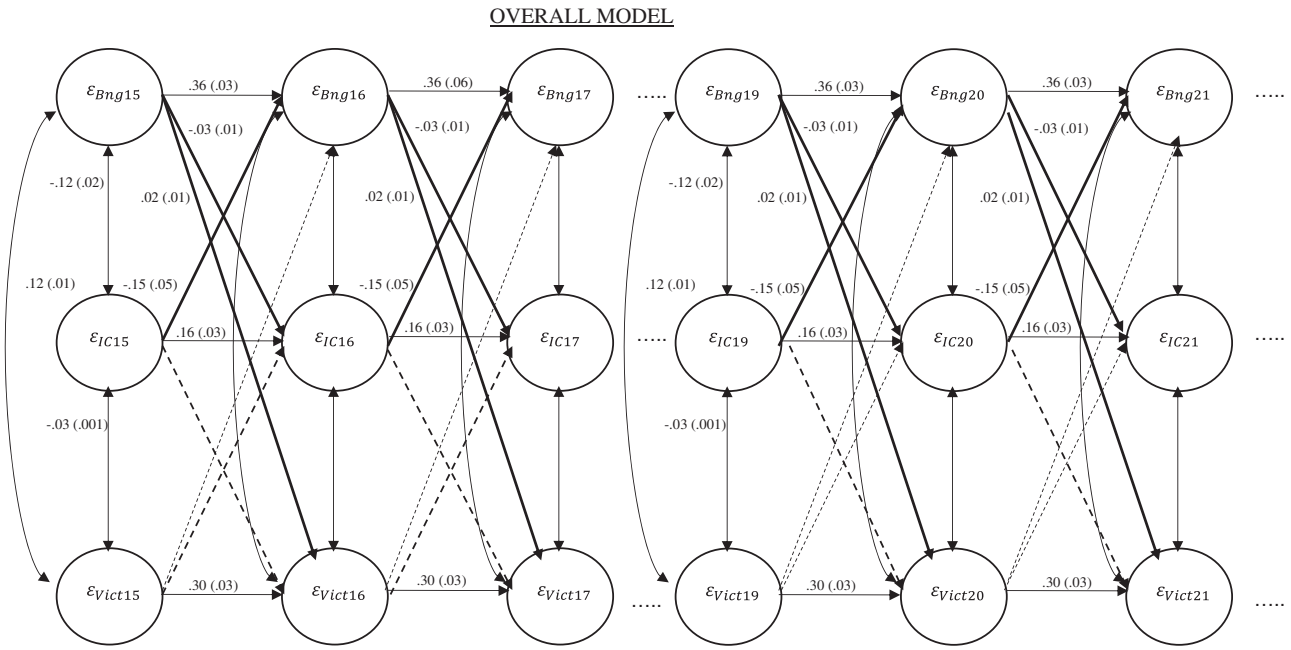

Fig. 1 ALT-SR model displaying within-person cross lagged effects for our overall model (no developmental split). Significant parameter estimates (Standard Error) are only shown. Full parameter estimates can be found in Table 3. *Note*: *Bold line* indicate significant path, *dashed line* represents non-significant path. *Bng* Binge drinking, *IC* Impulse Control, *Vict* Victimization. Each time point represents a year lag (e.g. Bng15 to IC16 represents binge drinking at age 15 to impulse control at age 16). In this model, we constrained cross-lag effects to be equal across both developmental periods

Within-Person Associations

Overall model

When constraining trajectories to be equal across time (see Fig. 1; Table 3, Model 2) we found when individuals reported more impulse control than their typical levels, they were less likely to report higher binge drinking the next year ($\beta = -.05$; $b = -.1595\%CI$ [−.27,−.02]). Conversely, reporting more binge drinking than normal was associated with subsequent decreases in impulse control a year later ($\beta = -.09$; $b = -.03,95\%CI$ [−.05,−.01]). Increased binge drinking was associated with experiencing more victimization ($\beta = .06$; $b = .02,95\%CI$ [.001,.04]) than normal the following year in age. Interestingly contrary to our second hypothesis (Hypothesis 2), we did not find within-person associations between victimization and binge drinking ($\beta = .01$; $b = .001,95\%CI$ [−.13,.13]) or victimization and impulse control ($\beta = -.03$; $b = -.03,95\%CI$ [−.08,.02]).

Developmental model

We found full support for our third hypothesis (Hypothesis 3). During adolescence, we did not find evidence of victimization or impulse control predicting binge drinking (see Fig. 2, Table 3 Model 3). However, we did find that individuals who reported more binge drinking ($\beta = -.06$; $b = -.028,95\%CI$[−.06,−.01]) and victimization ($\beta = -.05$; $b = -.067,95\%CI$ [−.13,−.01]) than their typical level had lower than typical impulse control the following year. Further, when individuals reported more binge drinking ($\beta = .07$; $b = .022,95\%CI$ [.01,.04]) than their typical level they also reported more victimization the following year.

Interestingly, during young adulthood, victimization did not predict more binge drinking($\beta = .03$; $b = .09,95\%CI$ [−.06,.23]). However, we found when individuals reported less impulse control than their typical level they reported more binge drinking($\beta = -.07$; $b = -.18,95\%CI$ [−.33, −.03]). Further, reporting higher binge drinking than average was associated with less impulse control ($\beta = -.07$; $b = -.03,95\%CI$ [−.04,−.01]) and experiencing more victimization ($\beta = .10$; $b = .03,95\%CI$ [.01,.05]) than typical.

Post-hoc Mediation

We tested potential post-hoc indirect effects based on results from Model 3. Specifically, we tested the indirect effect between binge drinking and impulse control via victimization in adolescence and the indirect effect between impulse control and victimization via binge drinking in young adulthood. We did not find a significant indirect effect (standardized indirect effect = .006, SE = .03, $p = .21$) during adolescence. However, we did find, though moderately, that binge drinking significantly mediated the relationship between impulse control and victimization

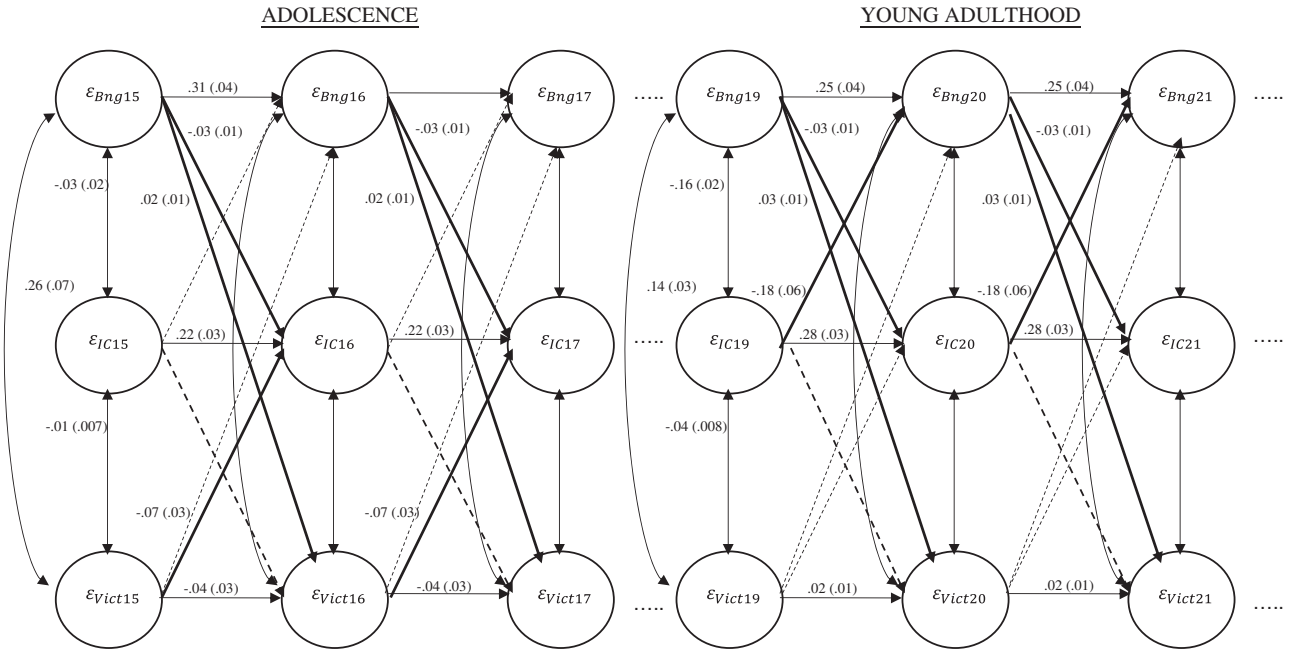

Fig. 2 ALT-SR model displaying within-person cross lagged effects for our developmental model. Significant parameter estimates (Standard Error) are only shown. Full parameter estimates can be found in Table 3. *Bng* Binge drinking, *IC* Impulse Control, *Vict* Victimization. Each time point represents a year lag (e.g. Bng15 to IC16 represents binge drinking at age 15 to impulse control at age 16). In this model we constrained cross-lag effects to vary across adolescence and young adulthood, separately

(standardized indirect effect $= -.01$, SE $= .001$, $p = .03$) during young adulthood.

Additional Analyses and Alternate Models

To better understand our results, we conducted several additional analyses. First, we considered limiting the number of control variables used in the model as our model utilized both propensity weighting and the weighting variables as controls for each model (doubly robust). We ran a model that used the following variables as controls to ensure our estimates were not due, simply, to covariates: Race/ethnicity, gender, proportion of time spent in the community, and age first sold drugs (this variable remained above the recommended standardized mean difference of 0.25 after propensity weighting). Results of our model resulted in better model fit (expected given fewer parameters to estimate; CFI $= .998$, RMSEA $= .028$, SRMR $= .030$) with no changes to parameter estimates in both the overall and developmental model. Second, we replicated our overall and developmental model using just self-reported data from participants. That is, we eliminated the use of parent/caregiver data to ensure our results did not vary due to utilizing multi-informant data. Results of our models directly replicated our findings in both significance and direction of effects. However, it appears that beta estimates were slightly higher when excluding parent/caregiver data. For example, in our developmental model the unstandardized beta for victimization on impulse control during adolescence was $b = -.066$ whereas in the additional model utilizing only self-reported data the unstandardized beta weight was $b = -.070$.

Discussion

Prior literature has shown long-term effects of early life victimization on behavioral outcomes (Shonkoff and Garner 2012). Unfortunately, youth in the juvenile justice system experience a higher prevalence of early life victimization, placing them at heighted risk of experiencing long term problems (Ford et al. 2010). In general, our results follow prior research on exposure to stressful life events and alterations in impulse control and substance use trajectories over time. That is, we found that mean levels of victimization (e.g., between-person) were associated with decreased impulse control and increased binge drinking. We also found evidence for variation in trajectories of impulse control when including early life victimization and early binge drinking into the model. When investigating wave-to-wave deviations (e.g., within-person), our results indicate that the cumulative effects of victimization during adolescence may have detrimental effects on self-regulatory processes such as impulse control during the adolescent phase. Subsequently, this resulted in more binge drinking and victimization during young adulthood. These results

indicate a potential developmental effect of early victimization and its long-term impact on impulse control, substance use, and continued experiences of victimization later in life. Further, victimization during young adulthood did not predict dampened impulse control, thus indicating that exposure to violence during adolescence may have a much longer impact on impulse control processes (e.g., carry over into young adulthood) than previously thought (Baumeister and Vohs 2003). Finally, one of the more interesting findings was a lack of association at the within-person level between victimization and binge drinking. It appears that, while this association exists as a general individual difference trajectory (e.g., between-person effect), this association may be more nuanced and include various "mechanisms" depending on the developmental period (e.g., impulse control during adolescence and binge drinking during young adulthood).

Adolescence is a period of development often characterized by high levels of risk taking, novelty and sensation seeking (Steinberg 2007). Relatedly, adolescence is also a time during which individuals are most likely to initiate alcohol use (Brown and Tapert 2004). This is problematic partly because adolescence is a time of increased brain development in areas such as the prefrontal cortex (Crews et al. 2007) and alcohol use has shown to have negative effects on this development. In particular, the prefrontal cortex is responsible for executive functioning such as abstract thinking, flexibility in response to cues, as well as using prior knowledge to assess current behavior and select an appropriate response (Crews and Boettiger 2009). We found that higher victimization *and* binge drinking during adolescence resulted in decreased impulse control. This translated into deficits in young adulthood, thus leading to more binge drinking episodes and eventually more victimization in young adulthood. It may be that the current sample of early onset juvenile delinquents have higher rates of binge drinking and early violence exposure, which could adversely impact areas of the brain associated with impulse control (e.g., prefrontal cortex). Further, it is possible that chronic exposure to trauma or victimization during adolescence impacts areas of the brain associated with stress regulation and impulse control, and these effects are realized during young adulthood when the brain completes its maturation process. This hypothesis becomes more interesting as some researchers have found chronic exposure to stressful experiences are associated with a "blunted" hypothalamic pituitary adrenal axis, which has been associated with numerous physiological and psychological problems later in life, including substance use (Clarke et al. 2008). Future research should examine the physiological and developmental processes that underlie the associations found in the present study.

It is curious that we did not find a direct (within-person) association between victimization and binge drinking during adolescence or young adulthood. However, this association did exist at the between-person level. While these results are perplexing, it may be that trajectories, in general, for exposure to violence and impulse control are related; however, when assessing wave-to-wave fluctuations, this relationship becomes more complicated. That is, we are not suggesting that there is a lack of relationship between experiences of victimization and subsequent alcohol consumption. On the contrary, we are suggesting that the association between victimization and binge drinking among youth may be a between-person effect (e.g., in general youth higher in victimization are more likely to engage in more binge drinking than their low victimization peers). However, the lack of within-person findings may be due, in part, because the sample in the present study consisted primarily of male juvenile offenders. Previous research has posited that gender differences exist in the relation between victimization and alcohol use (Thompson et al. 2008). Specifically, it may be that alcohol use is a risk factor for future victimization among males, as males are more likely to partake in situations promoting drinking (Huizinga et al. 2000). Conversely, alcohol use may be a consequence of victimization among females, as they may use alcohol as a maladaptive and avoidant coping mechanism (Widom et al. 2006).

Finally, post-hoc mediation analyses point to an important developmental difference between adolescence and young adulthood. That is, during adolescence, chronic exposure to violence may be a stronger predictor of impulse control problems, whereas during young adulthood binge drinking may be a way to cope with the continued efforts to self-regulate due to early traumatic experiences.

Theoretical Contributions

Overall, these results support to several theoretical orientations. Specifically, our results lend to further support Gottfredson and Hirschi's (1990) *General Theory of Crime*, which posits that deviant and criminal behaviors result from diminished self-control. Specifically, it may be that the current sample of early-onset juvenile offenders have initial deficits in self-control, thus putting them at greater risk for subsequent deviant behaviors (i.e., binge drinking) and further victimization. Thus, this theory would suggest that youth involved in the justice system are already at a disadvantage in terms of self-control. Our results expand this theory such that youth who experience early life victimization *and* engage in binge drinking during adolescence are likely to experience even more deficits in impulse control. We also found support for the larger, more overarching theory of *developmental transitions and trajectories*

(Havighurst 1948). That is, we found developmental variation in the transition from adolescence to young adulthood in terms of individual levels of impulse control and victimization.

Our findings further support the theory of *allostatic load* (McEwen 2003) and the *self-control strength model* (Baumeister and Vohs 2003). For example, the theory of allostatic load suggests that continued exposure to stressors early in life result in long-term deficits in physiological (e.g., cortisol) and behavioral (e.g., impulse control) outcomes. Our results provide continued support for this model and add more pointed look at the effects of exposure to stress on developmental processes into young adulthood among a sample of at risk youth. We also note that nearly 80% of the youth in this study would be considered low income (79% of mothers had a high school education or less), thus providing further support for the allostatic load model among low-income adolescents (Gunnar et al. 2009). Finally, parallel to the self-control strength model, our results indicate long-term deficits in impulse control due, in part, to victimization experiences *and* early binge drinking. This theory would suggest that continued efforts to control impulses or utilize emotional regulatory processes become depleted over time and, thus, diminish the capacity for decision making and controlling urges (Muraven and Baumeister 2000). The results provide continued support for this theoretical model and suggest that the effects of early life experiences may, in fact, have a lasting effect on impulse control into young adulthood.

Limitations

The results, however, should be interpreted with caution. First, our sample is only generalizable to early onset juvenile delinquents. Given the importance of understanding the long-term effect of early life stress on behavioral outcomes such as impulse control and binge drinking, it is vitally important that future research replicate these findings in a non-justice involved sample. Second, we were unable to assess early childhood adversity, which may have given more insight into the chronicity of victimization. That is, the study variables available for modeling did not include a retrospective account of early childhood abuse or neglect. Future research may want to consider latent constructs of victimization (e.g., early adversity, direct victimization, indirect victimization) to assess long-term effects on impulse control and binge drinking. Third, while our modeling procedure offers more nuanced insight, we did not have parental reports at each time point (adolescent phase only), which increases risk for biased responding. Future research may consider multi-informant reporting on longitudinal developmental studies. Finally, our substance use variable (binge drinking) was measured with a single item asking participants how often they engaged in binge drinking in the previous year. While single item measures have previously been used to assess binge drinking frequency (Guilamo-Ramos et al. 2005), this can be problematic in terms of the amount of information (variance) that it can provide. Although, individuals can assess, generally, whether they binge drank relatively frequently or infrequently in the past year, future research may consider multiple indicators of binge drinking or alcohol use in the form of a latent variable.

Clinical Implications

The results from the current study also have important clinical implications regarding treatment and prevention practices for youth who have experienced early life stress and those involved in the criminal justice system. Practitioners may wish to pay attention to adolescent clients who display heightened binge drinking episodes and report experiencing current victimization given that our results indicate potential impulse control problems later in life. Further, practitioners may also consider focusing on improving impulse control problems during adolescence, as this improvement may mitigate long-term behavioral (drinking) and experienced (victimization) problems later in life. Prior literature has found several psychotherapeutic approaches to be effective in attenuating impulsivity including cognitive behavioral therapy (Hofmann et al. 2012) and motivational interviewing (Hettema et al. 2005). It is thought that therapy sessions can activate frontal-cortical executive functioning, which may aid in controlling impulsive behavior (Crews and Boettiger 2009). Motivational interviewing is thought to work through evoking change talk (e.g., client pro-change language) and reducing sustain talk (e.g., client pro-use language) (Magill et al. 2014). One study used magnetoencephalography to measure neural responses when participants listened to their own change talk and found activation in the right-hemisphere network indicating that therapists that can evoke change talk are able to activate neural change, which may be associated with behavioral change. Further, it would be advantageous for future intervention and prevention efforts to include trauma-informed or trauma-focused practices into both substance use treatment programs and personality driven (e.g., impulse control) interventions. Our results suggest that youth who have experienced heightened early life stress would benefit from interventions that aid in mitigating the effects of traumatic memories, post-traumatic stress disorder, and other variations of trauma. One intervention option is TARGET (Trauma Affect Regulation: Guide for Education and Therapy (Ford and Russo 2006). TARGET focuses on interrupting automatic pilot (e.g., reacting to negative experiences). This could be considered

interrupting impulsive decision making among youth who have been exposed to early life stress. Prior research has found evidence for successfully reducing post-traumatic stress disorder symptoms and substance use among youth assigned to TARGET as compared to individuals assigned to usual care (Ford and Russo 2006; Frisman et al. 2008). Others may wish to examine interventions for young adults that aid in increasing impulse control, especially for individuals with a history of victimization or early life stress and adolescent binge drinking. For example, mindfulness based interventions have been found to have positive effects on both substance use and impulsivity (Murphy and MacKillop 2012; Witkiewitz et al. 2013a, b). While these studies have been primarily with adults, future research may wish to investigate the effect of mindfulness-based interventions among adolescent or young adult populations. Finally, a number of studies have investigated psychopharmacological treatments for impulsivity (see Moeller et al. 2001 for review). One treatment in particular, the use of naltrexone, has been shown to aid in alcohol dependent patients as well as increase frontal-cortical activation (Boettiger et al. 2009), similar to results found for motivational interviewing. It may be useful for future practitioners to use pharmacotherapy treatments in combination with psychotherapy treatments, especially for individuals who display impulsivity deficits and heightened victimization.

Conclusion

Given the high prevalence of alcohol use and victimization among early onset serious juvenile offenders, it is important for both research and public health efforts to take into consideration the developmental context in which the relations between impulse control, alcohol use, and victimization take place. To our knowledge, this is the first study to investigate the developmental effects of early life stress on long-term impulse control and binge drinking among a large sample of at risk youth. Overall, we found developmental differences during adolescence and young adulthood on binge drinking, impulse control, and victimization. However, one of the more important findings was the cascade of problems that followed heightened victimization and binge drinking during adolescence. Specifically, victimization and binge drinking during adolescence resulted in attenuated impulse control. This deficit in impulse control during adolescence was associated with increased individual level binge drinking and continued victimization in young adulthood. Our results provide a more nuanced look at the cycle of victimization and how it can influence long-term behavioral (impulse control and binge drinking) and experiences of direct victimization (e.g., re-victimization during young adulthood). Our results also have implications for prevention and intervention research. For example, previous research has identified personality-targeted interventions as a potential way to help ameliorate alcohol use among adolescents (Conrod et al. 2011), and future prospective studies should examine whether similar interventions may help serious juvenile offenders as well. Future studies should prioritize understanding the long term behavioral and neurological effects of early life stress among at risk populations as they represent a large proportion of victimized youth. In short, this study provides a deeper understanding of adolescent and young adult development and self-regulatory mechanisms that play a role in the relation between early life stress and binge drinking. Interventions aimed at self-regulatory processes may mitigate long-term problems later in life, especially among victimized youth.

Author Contributions J.D. conceived the study, performed statistical analyses, interpreted the data, and drafted the manuscript; T.D. aided in the interpretation of the data and helped draft the manuscript; B.B. helped draft the manuscript; G.M. performed statistical analyses and helped draft the manuscript; J.C.R. performed statistical analyses; B.R. participated in the design of the study and helped draft the manuscript. All authors read and approved the final manuscript.

Compliance with Ethical Standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflict of Interest The authors declare that they have no competing interests.

Ethical Approval This study was deemed exempt from ethics by the lead authors IRB as this was an analysis of secondary data.

Informed Consent Informed consent was received from each participant and their parent/caregiver prior to data collection. Please see Mulvey et al. (2004) and Schubert et al. (2004) for more details or visit the study website http://www.pathwaysstudy.pitt.edu/.

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