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A Model of Aggressive Behavior: Early Adversity, Impulsivity, and Response Inhibition

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Abstract

Exposure to adverse environments during childhood is robustly linked to future aggressive behavior. In this study we tested a model of emotional and neurocognitive mechanisms related to aggressive behavior in the context of childhood adversity. More specifically, we used path analysis to assess the distal contribution of childhood adversity and the more proximal contributions of emotion-related and non-emotion-related forms of impulsivity, and behavioral response inhibition to aggressive behavior. Participants were 180 undergraduates who completed well-validated self-report measures and an emotional version of the Go/No-Go task. The structural equation model was a poor fit for the data ($\chi^2(3) = 23.023$, $p < .001$; RMR = .131; CFI = .682; RMSEA = .142), though several significant paths emerged. Childhood adversity, emotion-related impulsivity, and behavioral response inhibition displayed direct effects on aggression, collectively accounting for 16.3% of variance. Findings demonstrate the specificity of emotional subtypes of impulsivity in linking childhood adversity and aggression. This study extends work on pathways to aggressive behavior by illustrating the complex relationships of early environmental, cognitive, and emotional mechanisms related to aggression.

Keywords

aggression; adversity; impulsivity; response inhibition; emotion

Aggression, defined as behavior intended to cause physical or psychological harm to another person, is a multifaceted problem driven by temperamental and environmental factors

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Disclosure of Interest Authors declare no conflicts of interest.

Ethical Standards and Informed Consent All procedures followed were in accordance with the ethical standards of the institutional review board at the University of Miami and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all participants for being included in the study.

(Vitaro, Brendgen, & Barker, 2006). Aggression is disturbingly common and exacts high societal costs (Vos et al., 2016). More than 20% of college students endorse engaging in some form of violent behavior (Schwartz, Beaver, & Barnes, 2015). As such, understanding the etiological contributions to this behavior remains of great interest. Attempts to explain aggression have focused on both distal and proximal influences.

Aggression and Adversity

At a distal level, early childhood has ubiquitously been characterized as a sensitive window of human development, with early family environment playing a catalyzing role in the development of behavioral problems (Kessler et al., 2010; Shonkoff et al., 2012). Adversity during this window (including maltreatment, physical/sexual abuse, poverty, and neglect) has been linked to various forms of aggression (Barnow & Freyberger, 2003), including bullying (Cook et al., 2010) and violent crime (Brewer-Smyth, Cornelius, & Pickelsimer, 2015). Multiple longitudinal studies have shown that early family environment and adversity significantly predict aggressive behavior (Côté, Vaillancourt, LeBlanc, Nagin, & Tremblay, 2006; Criss, Pettit, Bates, Dodge, & Lapp, 2002). Cross-sectional studies have found additional support for this link in children (e.g., Shields & Cicchetti, 1998), undergraduates (e.g., Carver, Johnson, McCullough, Forster, & Joormann, 2014), and those hospitalized for psychiatric disorders (Stinson, Quinn, & Levenson, 2016). Taken together, a robust literature links early adversity to aggression.

Impulsivity

Impulsivity has been tied to both childhood adversity and aggressive behavior (Lovallo, 2013; Carver et al., 2014). Impulsivity, however, is a broad construct, and a great deal of recent research has attempted to parse its heterogeneity (Berg, Latzman, Bliwise, & Lilienfeld, 2015; Whiteside & Lynam, 2001). In particular, factor analytic studies have distinguished between impulsivity precipitated by emotion and impulsive tendencies that do not involve emotion, such as lack of perseverance, lack of premeditation, and sensation seeking (Whiteside & Lynam, 2001; Carver et al., 2011).

The contribution of emotion-related impulsivity to psychopathology is supported by a meta-analysis of 115 studies (n = 40,432), which found that both internalizing and externalizing psychopathologies were more strongly associated with emotion-related impulsivity than with any other form of self-rated impulsivity (Berg, Latzman, Bliwise, & Lilienfeld, 2015). Multiple longitudinal studies have found that emotion-related impulsivity robustly predicts the onset and course of both internalizing and externalizing behaviors (Smith, Guller, & Zapolski, 2013; Zapolski, Cyders, & Smith, 2009).

Focusing specifically on aggression, self-report measures of emotion-related impulsivity have been linked to aggression, anger, and hostility in clinical and non-clinical samples (Hoptman, Antonius, Mauro, Parker, & Javitt, 2014; Johnson & Carver, 2016) and both reactive and proactive aggression (Hecht & Latzman, 2015). Of note, in one study, when controlling for emotion-related impulsivity, non-emotion-related impulsivity was not significantly correlated with aggression (Johnson, Carver, & Joormann, 2013).

Response Inhibition

What mechanisms might underlie emotion-related impulsivity? Like aggression, emotion-related impulsivity has been tied to childhood adversity (Carver et al., 2011; Wardell, Strang, & Hendershot, 2016). At a more proximal level, many have suggested that impulsivity may reflect deficits in executive function (Carver, Johnson, & Joormann, 2008; Bechara & Van der Linden, 2005). Of the many facets of executive function (Miyake et al., 2000), response inhibition has been empirically linked to emotion-related impulsivity, aggression, and childhood adversity. Response inhibition is a stable, trait-like characteristic that is robustly heritable (Miyake et al., 2000; Anokhin, Golosheykin, Grant, & Heath, 2017). Meta-analyses have found that reports of impulsive responding to both positive and negative emotions relate to poorer performance on response inhibition measures (Johnson, Tharp, Peckham, Sanchez, & Carver, 2016; Cyders & Coskunpinar, 2011). This is particularly true in clinical samples ($r = .34$), suggesting that the relationship between these two constructs may be more robust as their expression becomes more severe (Johnson, Tharp, Peckham, Sanchez, & Carver, 2016). One recent study found that emotion-related impulsivity, but not non-emotion-related impulsivity, related significantly to deficits in response inhibition (Dekker & Johnson, 2018). Corroborating this finding, self-reported emotion-related impulsivity related to worse performance on an emotional Go/No-Go task given after social rejection (Chester, Lynam, Milich, & DeWall, 2017). Taken together, theory and behavioral data suggest that deficits in response inhibition may underlie impulsive responding in the face of emotions.

Like impulsivity, impaired response inhibition has been related to aggressive behavior and to early adversity. Poor response inhibition has been extensively tied to aggressive behavior in clinical and non-clinical samples (Dambacher et al., 2015; Tonnaer, Cima, & Arntz, 2016), and to self- and teacher-reported reactive aggression in children (Feilhauer, Cima, Korebrits, & Kunert, 2012; Ellis, Weiss, & Lochman, 2009). Response inhibition deficits have also been tied to childhood adversity (McDermott, Westerlund, Zeanah, Nelson, & Fox, 2012; Mittal, Griskevicius, Simpson, Sung, & Young, 2015).

A recent article by McLaughlin (2016) proposed a model for psychopathology that develops after childhood adversity, in which emotion processing and executive function are two key intermediary mechanisms. In the analyses reported here we applied this framework to aggressive behavior. We focused on response inhibition and impulsivity as proximal mechanisms relevant to the link between early adversity and aggression.

As shown in Figure 1, we tested a model in which exposure to adverse environments during childhood gives rise to poor response inhibition as a cognitive function, which in turn promotes impulsivity, and ultimately leads to (among other things) aggressive behavior. We used this model to address three aims. First, we empirically assessed a modified version[1] of McLaughlin's (2016) model of psychopathological manifestations following childhood adversity in the context of aggression. To our knowledge, this is the first study to use this

[1]McLaughlin's (2016) model specifies unique pathways between types of adversity, distinct manifestations of emotional processing and executive functioning, and different forms of psychopathology. We do not test this full model, but rather are guided by this framework in considering how adversity gives rise to certain emotional and cognitive deficits, which in turn contribute to aggression.

model as the foundation for an empirical investigation of these variables. We differ from McLaughlin (2016), however, in that we specify temporal precedence between our emotional processing and executive functioning variables based on the literature cited above, whereas McLaughlin hypothesized separate pathways for these variables. Specifically, we hypothesized that early adversity would precede response inhibition, and that response inhibition in turn would precede impulsivity in a pathway to aggression. We further hypothesized that pathways involving emotion-related impulsivity would display greater effects than those involving non-emotion-related impulsivity. Our study is novel in testing the involvement of response inhibition, emotion-related impulsivity, and other aspects of impulsivity in a pathway from childhood adversity to aggressive behavior.

Methods

Procedures were approved by the Institutional Review Board at the University of Miami before data collection began. Participants completed written informed consent procedures. Participants under the age of 18 obtained parental consent prior to providing their own assent. Previous publications from this study have reported the association of emotion-related impulsivity with early adversity and, separately, aggression (Carver et al., 2011; Johnson, Carver, & Joormann, 2013). That work did not consider the role of behavioral response inhibition, nor did it test a multivariate model. Current analyses focus on the subset that completed the response inhibition task.

Participants

Participants were 180 undergraduates (127 females) at the University of Miami who participated in the research study for partial course credit. The mean age was 18.68 (SD = 2.39, range = 17–39). Self-reported ethnicity was 57.2% non-Hispanic white, 23.3% Hispanic, 6.7% Asian, 5.6% African American, 3.3% Caribbean, and 3.9% identified as other.

Measures

Bryant Aggression Questionnaire (BAQ).—Trait anger and aggression were measured using the 12-item self-rated Bryant Aggression Questionnaire (BAQ) (Bryant & Smith, 2001), a shortened form of the Buss-Perry Aggression Questionnaire (BPAQ) (Buss & Perry, 1992). The BAQ contains four separable subscales, each comprised of three items: Verbal Aggression (e.g., “I often find myself disagreeing with people”), Physical Aggression (e.g., “I have threatened people I know”), Anger (e.g., “I have trouble controlling my temper”), and Hostility (e.g., “I wonder why I am so bitter about things”). Responses were made on a scale ranging from 1 to 5. The BAQ has remarkably high correlations with both the BPAQ (r = .96) and the Brief Aggression Questionnaire (r = .91), and replicates the four-factor structure of the BPAQ (Webster et al., 2013). Further, the BAQ has demonstrated strong relationships to other measures of verbal aggression, physical aggression, anger, and hostility (Tremblay & Ewart, 2005).

Analyses did not incorporate hostility, as that subscale captures trait cynicism and would be less pertinent to understanding aggressive behavior that results from an inability to inhibit

responses. Internal consistency for each remaining subscale was moderate in this sample (Anger = .69; Physical Aggression = .68; Verbal Aggression = .78). An aggression composite score was created by averaging subscale scores, as the remaining subscales were all highly correlated with one another (all *r's* > .43, $p < .001$).

Risky Families.—Childhood adversity was measured using the Risky Families questionnaire (Taylor, Lerner, Sage, Lehman, & Seeman, 2004). This 13-item self-rated measure was designed to assess the relationship between family stress and health outcomes in adulthood. The items cover 13 aspects of the childhood family environment, such as the extent to which the respondent felt loved, was verbally and physically assaulted or threatened, or witnessed household violence or assault. Respondents rate items on a scale ranging from 1 ("not at all") to 5 ("very often"). The Risky Families scale has been validated against clinical interviews of adversity in the early family environment (Taylor, Lerner, Sage, Lehman, & Seeman, 2004) and has been shown to predict key psychopathological outcomes (Lehman, Taylor, Kiefe, & Seeman, 2005; Taylor et al., 2006). Internal consistency is high in the current sample ($\alpha = .86$).

Three-Factor Impulsivity index.—The Three-Factor Impulsivity index is a composite of well-validated self-report questionnaires relevant to impulsivity, which includes both emotion- and non-emotion-related forms of impulsivity, plus some newly developed scales (see Carver et al., 2011, for scale development). There are three factor-analytically derived components. Factor 1, labeled Pervasive Influence of Feelings, taps the tendency for emotions to reflexively affect one's outlook, including one's worldview and overall sense of self-worth (e.g., "When I have emotional experiences, they strongly influence how I look at life"). This factor is composed primarily of items from the following scales: Negative Generalization (Carver, 1998), Sadness Paralysis, Emotions Color One's Worldview, and Inability to Overcome Lethargy (Carver et al., 2011). Negative Urgency (Whiteside & Lynam, 2001) and Laziness, from the Behavioral Indicators of Conscientiousness (Jackson et al., 2010) scales also cross-load on this factor.

Factor 2, labeled Lack of Follow-Through, is composed primarily of scales that do not reference emotion, but rather distractibility (item, Carver et al., 2011), perseverance (item, Whiteside & Lynam, 2001), and self-control (e.g., "It's hard to get myself moving, even when I know what I want to do" (Tangney, Baumeister, & Boone, 2004)).

Factor 3, labeled Feelings Trigger Action, measures the tendency for emotions to reflexively drive behavior (e.g., "When I am really excited, I tend not to think of the consequences of my actions"). It is composed primarily of items from Reflexive Reaction to Feelings (Carver et al., 2011), an abbreviated version of the Positive Urgency Measure (Cyders et al., 2007), and an abbreviated version of Negative Urgency (Whiteside & Lynam, 2001).

Scales were standardized, and factor scores were constructed by weighting all of the contributing scales according to estimates published in Carver et al. (2011). Factors were scored such that higher scores reflect higher impulsivity. Each scale showed high internal consistency (Pervasive Influence of Feelings: $\alpha = .80$; Lack of Follow-Through: $\alpha = .87$; Feelings Trigger Action: $\alpha = .84$). The factors have demonstrated robust associations with

early adversity (Carver et al., 2011), internalizing and externalizing disorders (Johnson et al., 2013; Johnson, Tharp, Peckham, Carver, & Haase, 2017), suicidal ideation and action (Auerbach, Stewart, & Johnson, 2017), and aggression, in bivariate analyses (Johnson et al., 2013).

Facial Go/No-Go.—The Go/No-Go is a widely used computerized task to measure response inhibition, a key facet of executive functioning (Georgiou & Essau, 2011). A version using emotional stimuli was designed to test whether response patterns differed by emotional valence. During the task, faces depicting either happy or sad expressions were displayed on a computer screen for 500 milliseconds. Trials were introduced by a fixation cross in the center of the screen that was randomized to last either 1250, 1500, or 1750 milliseconds. Participants were instructed to strike a key in response to one emotional facial expression ("go" trials) and to withhold a response to the other expression ("no-go" trials). The emotional valence of go trials alternated with each block.

Participants completed two practice blocks (four trials each), then completed four blocks of 24 trials each (50% male faces and 50% female faces in each block, randomly ordered). For each block, go stimuli appeared on 75% of the trials and no-go stimuli appeared on 25% (randomly ordered). Signal detection analysis was used to measure false alarms (striking the key in response to a no-go trial). Sensitivity scores (d') are calculated as the Z-score for correctly identified go stimuli ("hits") minus the Z-score for false alarms. Response inhibition scores were calculated as the proportion of false alarms to no-go stimuli shown, such that higher scores reflect a greater false alarm rate. Go/No-Go tasks using faces as emotional stimuli are widely used (Tottenham, Hare, & Casey, 2011; Grunewald et al., 2015), and have sufficient convergent validity ($r = .51–.74$) with non-emotional versions of the same task and with other measures of behavioral inhibition, demonstrating that the use of emotional stimuli preserves the basic inhibition construct of the original Go/No-Go task (Schulz et al., 2007).

Procedure.—Participants completed the questionnaires described above in group testing sessions. The Go/No-Go task was completed in individual sessions, as were other tasks not described here. Tasks were administered in random order.

Data Analytic Plan.—All variables in Figure 1 displayed approximately normal distributions and were Z-scored for uniformity. Preliminary analyses indicated that false alarm scores for negative versus positive valence trial types did not differ in their relationships with other key constructs and were robustly correlated ($r = .39$, $p < .001$); accordingly, we created a composite false alarm rate score by averaging positive and negative valence trial scores. To address missing data for 32 cases on the Three-Factor Impulsivity index (which was not administered to one group of participants), multiple imputation was conducted using SPSS, in which patterns in the available data are analyzed and values are probabilistically derived using regression parameters. To robustly protect against bias of imputed values, imputation for missing values was conducted 20 times, resulting in 20 different datasets. Analyses were run for each dataset and pooled estimates were created according to Rubin's (1987) rules, in which point estimates for model parameters are estimated in each dataset and then averaged.

As preliminary descriptive analyses, we first generated univariate distributions and bivariate correlations. To test our model, we conducted a path analysis to assess whether adversity, response inhibition, and self-reported impulsivity have unique or overlapping effects on aggression, and to determine whether our hypothesized model was a good fit for the data. The Amos package in SPSS was used to construct the path model and determine fit indices. Goodness-of-fit was assessed using four criteria: a chi-square test (χ^2), the Root Mean Square Residual (RMSR), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA) (see Kenny, 2015 for details of fit determination). Fit indices were run for each imputed dataset and then averaged. Path coefficients were created by pooling unstandardized regression weights across imputed datasets. We report standardized coefficients. Significance of the path coefficients was determined by pooling the corresponding critical ratio and treating it as a Z-score (Khine, 2013).

Results

Assessment of Model Appropriateness

We assessed statistical assumptions of regression with the gvlma package in R (alpha = .05) to test for skew, kurtosis, homoscedasticity, and linearity. No significant deviations from regression assumptions were noted.

Correlations

As preliminary descriptive information before considering the multivariate model, the bivariate correlation matrix, along with descriptive statistics, is presented as Table 1[2]. Primary variables were not significantly related to age, gender or ethnicity (*r*'s < ±.137, *p*'s > .07). Nonetheless, we controlled for age, gender, and ethnicity in the multivariate model.

Structural Model

To assess whether the model shown in Figure 1 fit the data, we ran a path analysis for each imputed data set using the Amos package in SPSS. Path coefficients across each dataset were then pooled to create the final model, which is displayed as Figure 2. Fit indices indicated that the hypothesized model fit the data poorly ($\chi^2(3) = 23.023$, $p <. 001$; RMR = .131; CFI = .682; RMSEA = .142). Several significant paths, however, emerged.

Consistent with previous work, there was a highly significant effect of childhood adversity on aggression. Consistent with our hypotheses, response inhibition, Pervasive Influence of Feelings, and Feelings Trigger Action all had significant direct and unique associations with aggression. Lack of Follow-Through did not have a significant relation to aggression, corroborating our hypothesis that emotion-related impulsivity would be more integrally related to aggression. Contrary to hypotheses, the response inhibition measure (Go/No-Go) was not significantly related to childhood adversity or to any impulsivity factor.

[2]To ensure that the associations between the Three-Factor impulsivity index and the BAQ were not artificially inflated due to construct overlap, we purged 3 items from the BAQ Anger scale, and one from the Three-Factor impulsivity scale ("In the heat of an argument, I will often say things that I later regret") then re-estimated bivariate correlations. Correlations between the BAQ and factors of emotion-relevant impulsivity were slightly attenuated, as expected (*r*BAQ with Pervasive Influence of Feelings = .326, *r*BAQ with Feelings Trigger Action =.279). Both emotion-related impulsivity scales remained significantly correlated with the BAQ ($p < .001$), and there were no significant differences in the magnitudes of the correlations after excluding these items (all *p*'s > .17)."

In exploratory analyses, we tested whether the poor fit of our model might be due to the treatment of response inhibition as a predecessor of impulsivity and due to the inclusion of non-emotion-related impulsivity, which theoretically should be less relevant for aggressive behavior. To test this, we fit a revised model in which all indirect paths from response inhibition through impulsivity were removed, and replaced by direct links between childhood adversity and emotion-related impulsivity. The exploratory model, shown in Figure 3, contained direct effects of response inhibition and childhood adversity on aggression, and indirect effects of childhood adversity on aggression through Pervasive Influence of Feelings and Feelings Trigger Action. Goodness-of-fit statistics indicated that this revised model is an excellent fit for the data ($\chi^2(3) = 4.420$, $p = .220$; RMR = .033; CFI = .971; RMSEA = .044).

Discussion

The current study tested an integrative model of three factors related to aggression in order to examine the role of distal and proximal mechanisms of aggressive behavior. Previous work has linked childhood adversity, response inhibition, and impulsivity to aggression, as well as to each other. To our knowledge, however, no one has tested a unified model of the relationships among them. Using multivariate path analysis, we tested the hypothesis that childhood adversity would have both a direct and an indirect effect on aggression, and that the indirect effect would involve impaired response inhibition and higher levels of self-reported impulsivity. We placed a particular emphasis on emotion-related forms of impulsivity, given that recent work has highlighted the importance of emotion in impulsivity underlying aggression and other psychopathological outcomes (Berg, Latzman, Bliwise, & Lilienfeld, 2015). We further hypothesized that response inhibition would be a driver of emotion-related impulsivity, as it has been shown to be heritable and stable (Miyake et al., 2000; Anokhin, Golosheykin, Grant, & Heath, 2017) and has been related to emotion-related impulsivity previously (Johnson, Tharp, Peckham, Sanchez, & Carver, 2016; Cyders & Coskunpinar, 2011; Dekker & Johnson, 2018).

Childhood adversity, response inhibition, and both forms of emotion-related impulsivity displayed significant direct and unique effects on aggression. Collectively, these variables accounted for 16.3% of the variance in aggression. Consistent with hypotheses, we found that emotion-related impulsivity, but not non-emotion-related impulsivity, was particularly relevant to both early adversity and to aggression. We also found that both deficits in response inhibition and impulsive responding to emotion concurrently and uniquely impact aggression.

Nonetheless, our multivariate path analysis did not support the hypothesized model. One reason for the poor fit is that response inhibition, though significantly related to aggression, was not related to the measures of impulsivity, and thus did not act through them. The hypothesized pathway strays from the model outlined by McLaughlin (2016), which gives no temporal precedence to executive functioning or emotional processing deficits. Our exploratory model, which did not treat response inhibition as a mediator, confirmed that considering executive functioning and emotional processing deficits as separable contributors to aggression yielded considerably better model fit. Our results thus suggest that

difficulty inhibiting prepotent responses per se does not affect aggression by manifesting as impulsive personality traits, but rather is a correlate of aggressive behavior independent of impulsive personality traits.

That behavioral response inhibition displayed no significant relationship with impulsivity runs counter to both previous theory (Carver, Johnson, & Joormann, 2008) and research (Dekker & Johnson, 2018; Johnson, Tharp, Peckham, Sanchez, & Carver, 2016). Meta-analysis, though, indicates that the relationship between emotion-related impulsivity and response inhibition is more robustly present in clinical samples, and thus may only hold at more severe presentations of these constructs (Johnson, Tharp, Peckham, Sanchez, & Carver, 2016).

The lack of effect of child adversity on response inhibition also runs counter to previous theory and data. McLaughlin's model of childhood adversity (2016) contains more nuanced patterns of associations, for example, between different types of early adversity and specific emotional or cognitive endophenotypes. Our use of a single broad index of adversity and only one index of response inhibition may have been overly general and led to a loss of information.

Several other limitations are noteworthy. First, our reliance on self-report measures of adversity, impulsivity, and aggression may have inflated observed relationships among these variables due to shared method variance. Further, given that response inhibition was measured using an in-laboratory paradigm, whereas all other variables were measured with self-report questionnaires, there is a potential methodological issue regarding the temporal sequencing of the measures. Future research would benefit from assessing aggression using in-lab behavioral measures to get a temporally proximate assessment of the impact of response inhibition on aggression. Second, given that we placed a particular emphasis on the role of emotion in understanding failures to control impulse, we aimed to test response inhibition using emotion-relevant stimuli. Recent research, though, indicates that emotional and non-emotional versions of response inhibition tasks mostly overlap in their effects (Wright et al., 2014), and that emotional versions of the task still preserve measurement of response inhibition (Schulz et al., 2007). Further, we observed no differential effect of the positive versus negative go/no-go stimuli on any key variable in our model. Because our task did not include neutral stimuli, we were unable to contrast emotional versus non-emotional stimuli. Future research would benefit from more precisely measuring the role of emotion in response inhibition, and how emotion, response inhibition, and impulsivity interact to give rise to aggressive behavior. Lastly, given the use of a multifactorial model, our study is limited by its relatively small sample size. Future research would benefit from using larger and more diverse samples to return more precise parameter estimates.

Conclusions

Aggression is a complex behavioral phenomenon that has both distal and proximal influences. McLaughlin (2016) provided a clear model of how to consider the developmental progression of these multiple influences. This is, to our knowledge, the first empirical investigation to incorporate a form of this model as a foundation to conjointly consider

adversity, executive function, multiple forms of impulsivity, and aggression. We consider our findings an important step towards helping to empirically refine this model by investigating the contribution of two key proximal mechanisms to aggressive behavior following early adversity. Our findings support the well-established conclusion that aggressive behavior arises from both early experiences of the individual as well as more immediate cognitive and emotional responses. We build upon this work by demonstrating that these proximal mechanisms exact unique influences on aggressive behavior. Our findings also reiterate that impulsivity is a heterogeneous and nuanced construct, and that certain forms of emotion-related impulsivity may be particularly relevant to understanding links between childhood adversity and aggression.

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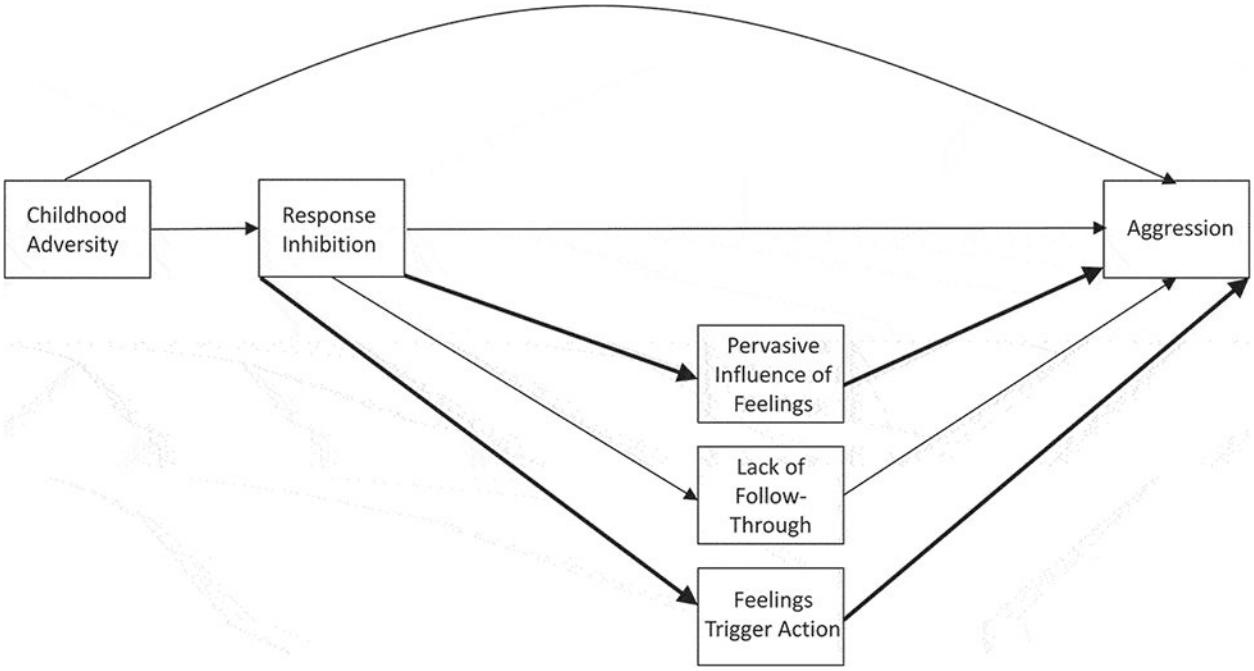

Figure 1. Hypothesized Model of Risk for Aggression, after
McLaughlin (2016) Note. Bold lines indicate emotion-relevant pathways.

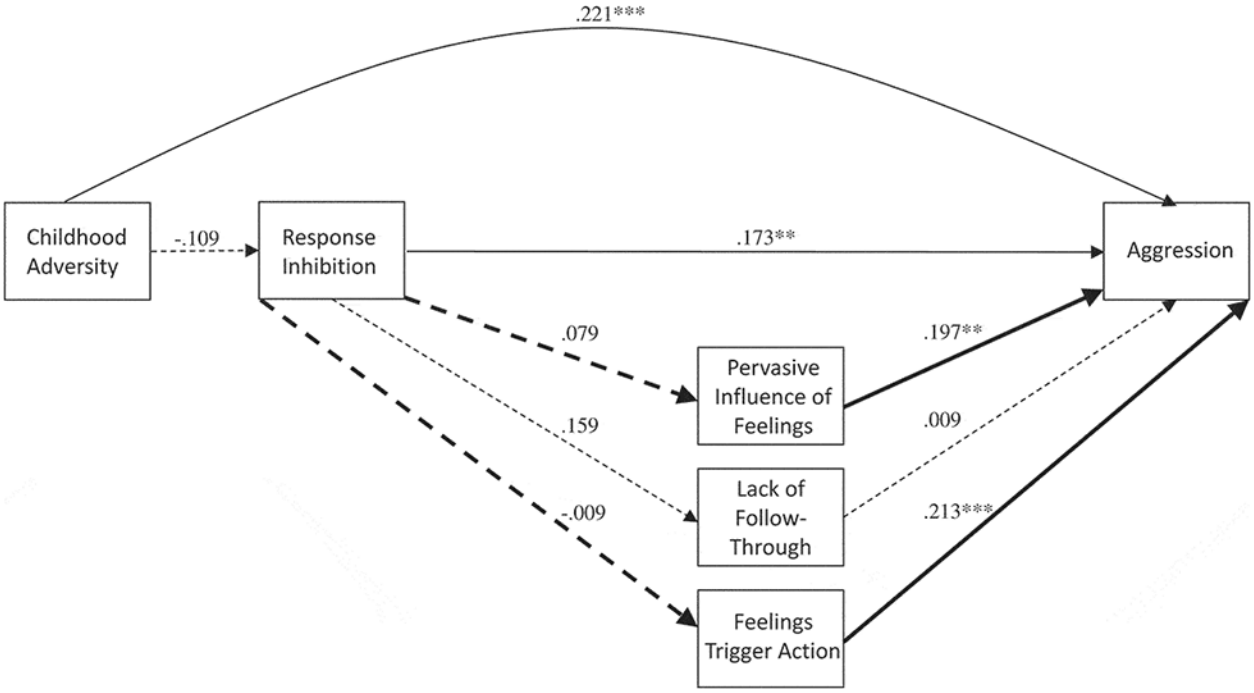

Figure 2. Aggression Model with Path Coefficients

Note. Dashed lines represent non-significant pathways. Bold lines represent emotion-relevant pathways. * $p < .05$ ** $p < .01$ *** $p < .001$

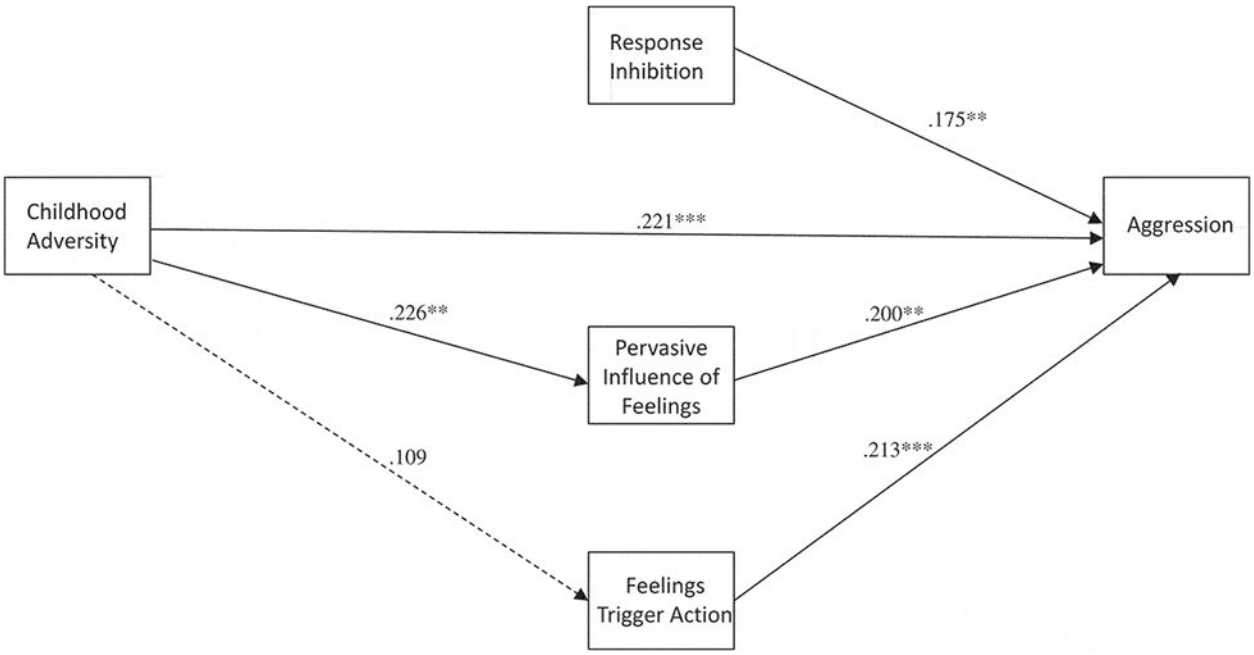

Figure 3. Revised Exploratory Model of Aggression.

Note. Dashed lines represent non-significant pathways. * $p < .05$ ** $p < .01$ *** $p < .001$

Table 1.

Descriptive Statistics and Bivariate Correlations among BAQ, Risky Families, Go/No-Go, and Three-Factor Impulsivity (N = 180)

	Descriptive Statistics			Bivariate Correlations					
Variable	***M***	***SD***	***Range***	***1***	***2***	***3***	***4***	***5***	***6***
1. Aggression Composite (BAQ)	2.446	0.826	1.00 – 5.00	-					
2. Risky Families	1.817	0.557	1.00 – 3.85	0.328***	-				
3. False Alarm Rate (Go/No-Go Composite)	0.183	0.115	0.02 – 0.73	0.161*	−0.117	-			
4. Pervasive Influence of Feelings	0.038	0.965	−2.07 – 2.59	0.386***	0.213**	0.068	-		
5. Lack of Follow-Through	0.099	0.963	−2.19 – 2.38	0.199**	0.151	0.138	.323***	-	
6. Feelings Trigger Actions	0.075	1.067	−2.62 – 3.01	0.373***	0.101	−0.007	.294***	.175*	-

Note. Descriptive statistics for Pervasive Influence of Feelings, Follow-Through, and Feelings Trigger Actions reflect z-scored values, as these had been previously transformed in the data set. Pooled imputed values were used for the 32 missing cases on these measures. Correlation coefficients for relationships with these scores reflect pooled estimates created in SPSS. BAQ = Bryant Aggression Questionnaire.

* p < .05

** p < .01

*** p < .001.

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