Original Research

Anhedonia and the Relationship Between Other Depressive Symptoms and Aggressive Behavior

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Abstract

Depression has been linked to multiple forms of aggressive behavior in college students; however, it is unclear which aspects of depression explain this connection. Anhedonia, defined as the loss of interest and/or pleasure in previously enjoyed activities, may provide unique information about relationships between depression and aggression. Using cross-sectional data from two independent samples of college students (N = 747 and N = 736 for Study 1 and Study 2, respectively), we examined whether anhedonia helped explain the relationship between broader depressive symptoms and different forms of aggressive and antisocial behavior. Anhedonia accounted for variance in both self-directed aggression and antisocial behavior independent of gender, hostility, anger, other depressive symptoms, and cognitive distortions (Study 2). In addition, there were significant indirect effects of depressive symptoms on self-directed aggression (Studies 1 and 2) and antisocial behavior (Study 2) via anhedonia. Hypotheses involving other-

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directed aggression received mixed support, with anhedonia atemporally associated with other-directed aggression independent of broader depressive symptoms in Study 1, but not in Study 2. The current findings suggest that anhedonia is an important individual difference that helps explain the relationship between depression and aggressive and antisocial acts and that anhedonia may be differentially associated with various types of aggressive and antisocial behavior.

Evidence suggests that depression is related to higher levels of aggression (Storch, Bagner, Geffken, & Baumeister, 2004), antisocial behavior (Ritakallio, Luukkaala, Marttunen, Pelkonen, & Kaltiala-Heino, 2010), and self-directed aggression such as nonsuicidal self-injury (Taliaferro & Muehlenkamp, 2015a, 2015b) in college students and adolescents. Moreover, evidence also supports an association between depression and aggression in samples with clinically significant depressive symptoms (Winkler, Pjrek, & Kasper, 2005). However, while there is much empirical support for a depression–aggression link (see Dutton & Karakanta, 2013, for a review), not all studies examining the proposed relationship have yielded significant findings (C. J. Ferguson et al., 2005).

Inconsistencies in the literature on depression and aggressive behavior may be partly due to the heterogeneity of depression. Recent research has demonstrated that (a) depression is not a consistent syndrome—the label "depression" may encompass myriad symptom profiles (Fried & Nesse, 2015) and (b) individual depressive symptoms vary in their impact on daily functioning in a range of domains (Fried & Nesse, 2014). Thus, it is possible that only some depressive symptoms (e.g., heightened negative emotion, pessimistic cognitive bias, impaired concentration or executive functioning, sleep problems, anhedonia) are associated with aggression. It is therefore necessary to examine the role of core individual symptoms of psychopathology that might link the broader construct of depression and various types of aggressive and antisocial behavior.

Several widely cited theoretical models of aggression suggest potential pathways by which individual depressive symptoms could influence aggressive behavior. The I³ ("I-cubed") theory of intimate partner violence (IPV; Finkel & Eckhardt, 2013), which posits that an impelling force, inhibiting force, and an instigating trigger are necessary for the perpetration of violence to transpire (Finkel et al., 2012), may be one such model. For example, heightened negative emotion can increase the likelihood that an individual

will experience an urge to behave aggressively toward an intimate partner in response to a given triggering event (Birkley & Eckhardt, 2015; Watkins, DiLillo, Hoffman, & Templin, 2015). Heightened negative emotion has also been found to predict IPV perpetration as well as broader criminal activity during emerging adulthood, specifically. Similarly, in the general aggression model (Anderson & Bushman, 2002), negative affect is one of the routes by which situational and cognitive input variables can influence decision making and subsequently behavior, and this idea has gained empirical support (Verona & Curtin, 2006; Verona, Patrick, & Lang, 2002). General strain theory posits that individuals may be motivated to engage in antisocial and/or criminal activities such as illicit drug use in an effort to alleviate negative emotion (Agnew, 2001).

Specific aspects of depression could also weaken an individual's ability to inhibit aggressive urges or think through possible consequences before acting—another potential pathway suggested by both the I^3 theory (Finkel & Eckhardt, 2013) and the general aggression model (Anderson & Bushman, 2002). For example, difficulty concentrating and impaired executive functioning are common features of depression (Rock, Roiser, Riedel, & Blackwell, 2014).

Anhedonia and Aggressive Behavior

In the present pair of studies, we focus on anhedonia, or the loss of pleasure or interest in people or things. Anhedonia is a core element of depression that may be uniquely associated with aggressive and antisocial behavior including sexual violence (Carvalho & Nobre, 2013), delinquency in adolescents (Cohn et al., 2015), and aggression in a laboratory task (Fanning, Berman, & Guillot, 2012). Anhedonia may also help explain the relationship between self-directed aggression[1] and depression (Fawcett et al., 1990; Nordström, Schalling, & Asberg, 1995; Sadeh, Javdani, Finy, & Verona, 2011). Research has shown that anhedonia heightens the risk for nonsuicidal self-injury and suicide attempts in both women and men (Sadeh et al., 2011). Indeed, this relationship has previously been found to be *stronger* for self-directed aggression than other-directed aggression (Sadeh et al., 2011; Zielinski, Veilleux, Winer, & Nadorff, 2017).

Anhedonia, Reward Devaluation, and Disengagement From Competing Goals

Goal pursuit engages the incentive reward system and generates interest and anticipatory pleasure (Alcaro, Huber, & Panksepp, 2007; Gollwitzer &

Moskowitz, 1996). When an individual is engaged in pursuing a highly desired goal or prospective reward, attention toward competing cues is inhibited and competing goals are viewed less positively, to the extent that pursuing them would derail progress toward the primary goal (Shah, Friedman, & Kruglanski, 2002). Thus, engagement in pursuit of highly valued long-term rewards such as academic achievement, career success, positive social relationships, or financial stability generates interest and pleasure and likely facilitates inhibition of competing cues and action impulses, such as urges to engage in aggressive, antisocial, or sensation-seeking behaviors.

However, experimental research has demonstrated that when a goal is repeatedly paired with negative affect, the prospective reward associated with achieving that goal becomes less desirable and efforts to pursue the goal cease (Aarts, Custers, & Holland, 2007). This finding is consistent with evidence that individuals devalue and disengage from pursuing goals when the perceived risk of failure, frustration, or disappointment is too high (Brandstätter, Herrmann, & Schüler, 2013; Carroll, Shepperd, & Arkin, 2009; Carver & Scheier, 1998; Klinger, 1975). As this occurs, individuals experience a reduction in positive affect and may be especially prone to aggressive and impulsive behavior (Carver & Scheier, 1998). Indeed, there is empirical support for the idea that a lack of positive affect, as opposed to exaggerated negative affect, may result in failure to inhibit aggressive and antisocial impulses (Carvalho & Nobre, 2013; Cohn et al., 2015; Zisner & Beauchaine, 2016).

If loss of interest and devaluation of a previously valued goal or prospective reward is part of a normative process of goal disengagement, then anhedonia may reflect overly broad, extended disengagement and devaluation of rewards. This conceptualization is consistent with the central role of anhedonia in depression; in fact, recent meta-analytic evidence indicates that some depressed persons actively devalue and avoid prospective rewards, as predicted by Reward Devaluation Theory (Winer & Salem, 2016). Moreover, depressed individuals who are unable to reengage with pursuit of substitute goals and prospective rewards are at increased risk for suicidal ideation and behavior (Carver & Scheier, 1998; O'Connor, Fraser, Whyte, MacHale, & Masterton, 2009; O'Connor, O'Carroll, Ryan, & Smyth, 2012). Thus, anhedonia may act as a pathway linking depression and negative affect to self-directed aggression.

Recent Changes in Anhedonia

Studies examining anhedonia and aggression have produced some inconsistent results. For instance, anhedonia has been found to predict aggression on a behavioral task; however, in the same investigation, anhedonia and

self-reported aggression did not yield a similar relationship (Fanning et al., 2012). Thus, further research is needed to clarify the nature of the relationship between anhedonia and aggression.

One possible explanation for the discrepancy in findings regarding anhedonia and aggression is the lack of specificity of trait-based anhedonia measures (Winer, Veilleux, & Ginger, 2014). Recent changes in anhedonia (i.e. a *loss* rather than a stable lack of interest or pleasure) may prove more predictive of aggressive and antisocial responses (even a lifetime history of such responses) than trait-based anhedonia. This is because (a) depressive episodes often reoccur, (b) experiencing a depressive episode is associated with elevated risk of subsequent episodes (Burcusa & Iacono, 2007), and (c) loss of interest or pleasure may reoccur whenever valued prospective rewards are repeatedly paired with negative affect (Winer & Salem, 2016). Thus, a recent decrease in positive affect could indicate a person who is likely to experience more volatile states (Treadway & Zald, 2011) and depression (Winer, Veilleux, & Ginger, 2014). A metric of change from baseline may, therefore, be uniquely capable of uncovering relationships between shifting anhedonic tendencies and aggressive tendencies in individuals who are prone to depression. However, until recently, available measures of anhedonia indexed either trait anhedonia or state anhedonia. Thus, whether changes in anhedonia may signal increased risk for aggressive and antisocial behavior remains an empirical question.

In the present study, we used measures of aggression and antisocial behaviors previously used to investigate candidate relationships with anhedonia (Fanning et al., 2012). We also employed a novel measure of anhedonia, the Specific Loss of Interest and Pleasure Scale (SLIPS; Winer, Veilleux, & Ginger, 2014), which allows for the assessment of recent changes in anhedonia separately from trait anhedonia and therefore offers an incremental advance for examining acute loss of interest and pleasure associated with depression. Previously, changes in anhedonia from baseline have only been measured using selections of relevant items pulled from broader measures, but this approach has uncovered relationships between anhedonia and depression (Joiner, Brown, & Metalsky, 2003) and anhedonia and suicidality (Winer, Nadorff, et al., 2014). As the first measure specifically designed to measure recent changes in anhedonia, the SLIPS is more likely than previous measures of anhedonia to reveal a relationship between changes in anhedonia and aggressive, violent, and antisocial tendencies, if such a relationship exists.

The purpose of the present study was to examine multifaceted aspects of aggressive and antisocial behavior in relation to recent changes in anhedonia in college students. We hypothesized that recent changes in anhedonia would account for the relationship between other current depressive symptoms (i.e., besides anhedonia) and history of aggression in all forms (self-directed,

other-directed, antisocial). Our hypothesis was based on the idea that repeated pairing of valued goals with depressive symptoms such as negative emotions and beliefs eventually results in anhedonia as an individual devalues prospective rewards and disengages from goal pursuit, which in turn weakens inhibitory control over aggressive and antisocial impulses.

Finding a link between self-reported changes in anhedonia and aggressive and antisocial behavior in college students would be important for a number of reasons. First, social anhedonia may reflect interpersonal dysfunction and proneness to psychopathology (S. M. Thompson et al., 2015), as well as increase the chance that an individual may become aggressive (Fanning et al., 2012). Second, discovering that anhedonia underlies the relationship between depression and aggressive or antisocial acts would help specify one way that depression and aggression are linked. Third, this understanding could increase ability to predict the risk of an individual directing violence toward self or others if he or she is experiencing recent changes in anhedonia (Sadeh et al., 2011). Predictive information of this kind could be especially important in a college population, given that trajectories of aggressive behavior have been found to change significantly during college (M. P. Thompson, Swartout, & Koss, 2013).

Study 1

Method

Participants. Participants were obtained from a survey of 747 undergraduate students in introductory-level psychology classes at a large, public university in the Southeastern United States collected as part of a larger study (Nadorff, Anestis, Nazem, Harris, & Winer, 2014). The sample was 57% female, with an age range of 18-33 years (mean age = 18.9 years, *SD* = 1.4 years). Approximately 74% of participants identified themselves as Caucasian, 21% African American, 2% other, and less than 1% Hispanic, Asian or Pacific Islander, and Native American. Six hundred and seventy participants completed each of the measures with no missing data and were thus included in the following analyses. Participants were recruited using the SONA system, an online survey management system, and received course credit for participating in the study.

Measures

SLIPS. The SLIPS is a 23-question self-report measure that assesses changes in the ability to become interested in or take pleasure from primarily social experiences (Winer, Veilleux, & Ginger, 2014). The SLIPS has demonstrated high internal consistency as well as incremental validity beyond and divergent validity from other trait and state anhedonia measures (Winer,

Veilleux, & Ginger, 2014). The SLIPS items are scored from 0 to 3 to indicate change in interest or pleasure in the last 2 weeks, relative to the past. The first statement (coded as 0) indicates no loss of interest or pleasure (e.g., "I have not lost interest in my favorite activities"). The second statement (coded as 1) indicates some loss of interest or pleasure (e.g., "I have less interest in my favorite activities"). The third statement (coded as 2) indicates a loss of most interest or pleasure (e.g., "I have lost most interest in my favorite activities"). The fourth statement (coded as 3) indicates that there has never been any interest in or pleasure gained (e.g., "It's always been hard to get interested in activities"). Scores of 3 are recoded as 0, thus allowing responses that represent trait-level anhedonia to be scored the same as responses that represent no loss of interest; the SLIPS thus has a range from 0 to 46. This recoding procedure ensures that only responses indicating a change from baseline comprise the SLIPS. In the current sample, the mean was 5.37 (SD = 7.81) with acceptable reliability (α = .94), consistent with previous studies (Winer, Nadorff, et al., 2014; Winer, Veilleux, & Ginger, 2014).

Life History of Aggression. The Life History of Aggression (LHA) is an 11-item self-report measure that assesses an individual's history of verbally or physically aggressive behavior, self-injurious and suicidal behavior, and antisocial behavior (Coccaro, Berman, & Kavoussi, 1997). Items are scored from 0 to 5 to indicate number of occurrences of each type of behavior since age 13 years (0 = none, 1 = one occurrence, 2 = two or three occurrences, 3 = four to nine occurrences, 4 = 10 or more occurrences, and 5 = too many occurrences to be counted). In the present study, we utilized the aggression subscale as our measure of other-directed aggression. The LHA aggression subscale consists of five items, measuring anger outbursts, physical fighting, verbal aggression, assaults, and aggression toward objects. Scores on this subscale have evidenced positive correlations with aggressive behavior observed in laboratory settings, as well as with biological variables associated with aggressive behavior (Berman, McCloskey, Fanning, Schumacher, & Coccaro, 2009; Coccaro, Berman, Kavoussi, & Hauger, 1996). In addition, we used the antisocial behavior subscale (assessing rule-breaking and illegal behavior such as lying, stealing, and violating the rights of others) as our measure of antisocial behavior. Finally, we used the self-aggression subscale to assess nonsuicidal self-injury and suicide attempts. In the present sample, reliability for the other-directed aggression (α = .77), antisocial behavior (α = .80), and self-directed aggression (α = .70) subscales were acceptable. Subscale means were as follows: other-directed aggression (M = 7.36, SD = 4.82), antisocial behavior (M = 2.17, SD = 3.41), and self-directed aggression (M = .97, SD = 1.86). The LHA full scale mean was 10.46 (SD = 8.31).

Buss–Perry Aggression Questionnaire. The Buss–Perry Aggression Questionnaire (BPAQ) is a 29-question self-report measure of aggressive disposition with scaled responses on a 5-point Likert-type scale ranging from "Extremely unlike me" to "Extremely like me" (Buss & Perry, 1992). The BPAQ has previously evidenced adequate reliability, and in the present study we used the anger and hostility subscales, for which reliability was acceptable (anger: $\alpha = .82$; hostility: $\alpha = .86$). Trait anger and hostility were included as covariates in our regression models, replicating previous analytic methods (Fanning et al., 2012).

Center of Epidemiologic Studies Depression Scale. The Center of Epidemiologic Studies Depression Scale (CES-D) is a 20-item self-report measure of depressive symptoms (Radloff, 1977). The CES-D is scored on a 4-point scale (0-3) with total scores ranging from 0 to 60, where higher scores represent greater depression severity. The scale has acceptable internal consistency for both the general ($\alpha = .85$) and clinical ($\alpha = .90$) populations (Radloff, 1977) and is a valid screening measure for detecting depressive symptoms (Weissman, Sholomskas, Pottenger, Prusoff, & Locke, 1977).

To ensure no overlap with the SLIPS, items 8 ("I felt hopeful about the future") and 12 ("I was happy") were removed from the scale. Remaining CES-D items index current depressed state (e.g., "I felt depressed") over the past week. In the current sample, the mean was 12.92 ($SD = 9.41$) with acceptable reliability ($\alpha = .90$), consistent with previous studies. With the anhedonia items removed, the CES-D and the SLIPS were moderately correlated ($r = .63$).

Procedure. Data were collected online using the SONA online survey system. Participants logged onto the SONA system and selected the study from a list of all of the available studies. The study was institutional review board (IRB) approved, and a summary consent sheet served as the consent document for the study. Participants were provided with referral information for available mental health servicesand awarded course credit for participating at the study's conclusion.

Examination of distributions and data analysis. The data for the present study were obtained from a broader study. The dependent measures were examined for normality. The distribution of the aggression (skew = .64, kurtosis = .25), antisocial behavior (skew = 1.89, kurtosis = 2.98), and self-aggression (skew = 2.32, kurtosis = 5.42) subscales were within normal limits (Kline, 2005). Bivariate correlations between study variables are presented in Table 1.

Table 1. Correlations Between Variables in Study 1.

Variable	1	2	3	4	5	6	7	8
1. Other-directed aggression	—	.49**	.36**	.53**	.45**	.29**	.31**	−.17**
2. Antisocial consequences		—	.61**	.43**	.39**	.36**	.52**	−.19**
3. Self-directed aggression			—	.39**	.40**	.46**	.54**	−.05
4. Anger				—	.62**	.36**	.35**	.01
5. Hostility					—	.53**	.46**	−.03
6. Depressive symptoms						—	.63**	.10*
7. Recent changes in anhedonia							—	−.01
8. Gender								—

Note. Correlations with gender performed using a point-biserial correlation with male coded as 1 and female coded as 2. *N*s range from 666 to 670.
*$p < .05$. **$p < .01$.

We examined our hypotheses via the mediation model (model 4) of the SPSS (v. 22) PROCESS macro (Hayes, 2013). PROCESS tests for mediation by using bootstrapping to estimate direct and indirect effects. In the bootstrapping method, the original *n* is treated as a miniature population, and random resamples with replacement of the original *n* are drawn. For each hypothesis, 1,000 bootstrap samples were generated and 95% bias-corrected bootstrap confidence intervals were used to determine the significance of indirect effects. An indirect effect of zero means that the mediator variable had no impact on the relationship between the predictor and outcome variables; so, when a confidence interval does not include zero, the indirect effect is considered to be significant (Hayes, 2013). Sobel tests of significance of indirect effects are also reported for comparative reference. In addition to direct and indirect effects, the effect of depressive symptoms on each outcome variable when recent changes in anhedonia are *not* included in the model is also reported for comparison.

Results

To test the hypothesis that recent changes in anhedonia would account for the relationship between other depressive symptoms (i.e., those besides anhedonia) and history of aggression, we used depressive symptoms (CES-D sans anhedonia) as the predictor, recent changes in anhedonia (SLIPS) as the mediator, and hostility (BPAQ subscale), anger (BPAQ subscale), and gender were included in the model as covariates (Figure 1a). First, we ran the analysis with other-directed aggression (LHA subscale) serving as the outcome variable.
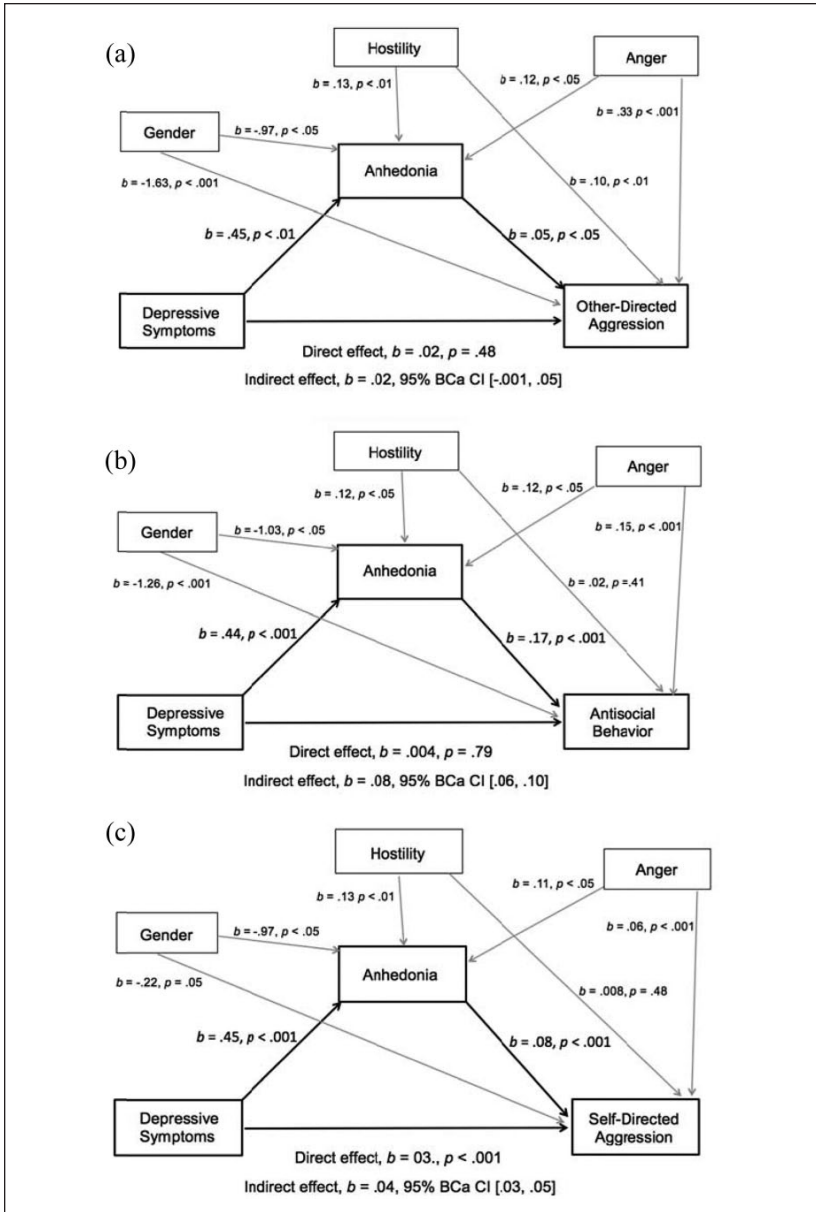
Figure 1. Mediational models for Study 1 for (a) Hypothesis 1, (b) Hypothesis 2, and (c) Hypothesis 3.
Note. Paths in gray denote covariates.

Without recent changes in anhedonia in the model, other depressive symptoms accounted for a significant amount of variance in other-directed aggression, $N = 670$,[2] $b = .04$, 95% confidence interval (CI) = [.001, .08], $p = .04$. With recent changes in anhedonia included in the model, the effect of depressive symptoms on other-directed aggression was not significant, $b = .02$, 95% CI = [−.03, .06], $p = .48$, whereas the effect of recent changes in anhedonia on other-directed aggression was significant, $b = .05$, 95% CI = [.001, .10], $p < .05$. We examined the indirect effect of depressive symptoms on other-directed aggression via recent changes in anhedonia and found that this was not significant based on bias-corrected 95% confidence intervals, $b = .02$, 95% CI = [−.001, .05], although the Sobel test did indicate a significant indirect effect, $p < .05$. Overall, the model accounted for 34.08% ($F = 68.65$, $p < .01$) of the variance in other-directed aggression.

Next, we tested the same model with antisocial behavior (LHA subscale) serving as the outcome variable (Figure 1b). Again, without recent changes in anhedonia in the model, other depressive symptoms accounted for a significant amount of variance in antisocial behavior, $b = .08$, 95% CI = [.05, .11], $p < .01$. With recent changes in anhedonia included in the model, the effect of depressive symptoms on antisocial behavior was no longer significant, $b < .01$, 95% CI = [−.03, .03], $p = .79$, whereas the effect of recent changes in anhedonia on antisocial behavior was significant, $b = .17$, 95% CI = [.14, .21], $p < .01$. We also found a significant indirect effect of depressive symptoms on antisocial behavior via recent changes in anhedonia, based on confidence intervals, $b = .08$, 95% CI = [.05, .10], as well as the Sobel test, $p < .01$. This indicates that recent changes in anhedonia accounted for the relationship between other depressive symptoms and antisocial behavior. Overall, the model accounted for 37.82% ($F = 80.77$, $p < .01$) of the variance in antisocial behavior.

Finally, we tested the same model a third time, with self-directed aggression (LHA subscale) serving as the outcome variable (Figure 1c). Without recent changes in anhedonia in the model, other depressive symptoms accounted for a significant amount of variance in self-directed aggression, $b = .07$, 95% CI = [.05, .08], $p < .01$. With recent changes in anhedonia included in the model, the effect of depressive symptoms on self-directed aggression remained significant, $b = .03$, 95% CI = [.01, .05], $p < .01$, and the effect of recent changes in anhedonia on self-directed aggression was also significant, $b = .08$, 95% CI = [.07, .10], $p < .01$. Next, we examined the indirect effect of depressive symptoms on self-directed aggression via recent changes in anhedonia and found that this was significant based on confidence intervals, $b = .04$, 95% CI = [.03, .05], as well as the Sobel test, $p < .01$. This indicates that recent changes in anhedonia partially accounted for the relationship between other

depressive symptoms and self-directed aggression, although depressive symptoms also accounted for unique variance. Overall, the model accounted for 35.84% ($F = 120.58$, $p < .01$) of the variance in self-directed aggression.

Study 2

Our findings from Study 1 point to changes in anhedonia as a meaningful marker of elevated risk for aggression and antisocial behavior in college students, independent of other depressive symptoms. These findings are consistent with other recent work showing that depressive symptoms are not interchangeable, but rather are differentially related to other symptoms of psychopathology and to environmental risk factors (Bringmann, Lemmens, Huibers, Borsboom, & Tuerlinckx, 2015; Fried et al., 2015; Fried & Nesse, 2015; Fried, Nesse, Guille, & Sen, 2015). The purpose of Study 2 was to replicate Study 1 and to extend our investigation to examine cognitive distortions that could also explain the relationship between anhedonia, other symptoms of depression, and aggression in a college population. Indeed, previous research suggests that cognitive distortions, or irrational negative beliefs about oneself, the future, or the world (Beck, 1976) might bridge depressive symptoms and self-directed aggression (Miller & Esposito-Smythers, 2013), and it is possible that distorted thinking may account for the relationship between anhedonia and aggression. Thus, a second goal of Study 2 was to examine the relative value of anhedonia, as compared to the specific symptom of cognitive distortions, in identifying risk for aggression and explaining the link between aggressive behavior and broader depressive symptoms. In Study 2, we hypothesized that recent changes in anhedonia would account for the relationship between other current depressive symptoms and history of aggression (other-directed, self-directed, antisocial), independent of gender, hostility, anger, and cognitive distortions.

Method

Participants. Participants were obtained from a survey of 736 undergraduate students in introductory-level psychology classes at a large, public university in the Southeastern United States. Participants responded to a battery of measures including those used in the present study. The sample was 67% female, with an age range of 18-37 years (mean age = 19.5 years, SD = 1.9 years). Approximately 73% of participants identified themselves as Caucasian, 19% African American, 2% Hispanic, 2% Asian or Pacific Islander, <1% Native American, and 3% other. A total of 615 participants (199 males and 416 females) completed each of the measures used in Study 2 with no missing data and were thus included in the following analyses.

Measures. All measures used in Study 2 were the same as those used in Study 1 except for the addition of a cognitive distortion measure and a minor change to the depressive symptoms measure, which are described in the following sections. Measures in Study 2 evidenced adequate reliability: other-directed aggression, $\alpha = .81$; antisocial behavior, $\alpha = .75$; self-directed aggression, $\alpha = .65$; anger, $\alpha = .72$; hostility, $\alpha = .87$; and recent changes in anhedonia, $\alpha = .93$. Subscale means for the LHA were as follows: other-directed aggression ($M = 6.30$, $SD = 4.78$), antisocial behavior ($M = 1.24$, $SD = 2.52$), and self-directed aggression ($M = .74$, $SD = 1.61$). The LHA full-scale mean was 8.22 ($SD = 7.33$).

Cognitive distortions. A licensed clinical psychologist and advanced doctoral candidate in clinical psychology independently selected items from a 20-item form of the Taylor Manifest Anxiety Scale (TMAS; Bendig, 1956) that assess cognitive distortions, which were defined as irrational negative beliefs about oneself, the future, or the world. A high level of agreement was obtained ($\kappa = .88$), and the following six items were adopted as the measure of cognitive distortions for Study 2: Item 10, "I have sometimes felt that difficulties were piling up so high that I could not overcome them," Item 15, "At times I think I am no good at all," Item 16, "I am certainly lacking in self-confidence," Item 17, "I certainly feel useless at times," Item 19, "I sometimes feel that I am about to go to pieces," and Item 20, "I shrink from facing a crisis or difficulty." Items are scored on a true/false scale with one point assigned for each "true" response. In the current sample, the 6-item measure of cognitive distortions evidenced adequate reliability ($\alpha = .76$). Moreover, deletion of any single item resulted in lower reliability; thus, all six of the selected items were retained.

Depressive symptoms. As in Study 1, the CES-D was used to measure depressive symptoms, but for this study, Item 9 ("I felt that my life was a failure") was excluded to avoid any potential overlap with the measure of cognitive distortions. Items 8 and 12 were also excluded, consistent with Study 1, to avoid overlap with the SLIPS. The remaining items of the CES-D evidenced adequate reliability, $\alpha = .89$, and were moderately correlated with our measure of cognitive distortions, $r = .64$, and with recent changes in anhedonia, $r = .63$.

Procedure. Participant recruitment and data collection procedures for Study 2 were the same as those used in Study 1.

Examination of distributions and data analysis. The dependent measures were examined for normality. The distribution of the aggression (skew = .69, kurtosis = .08) and self-aggression (skew = 2.56, kurtosis = 6.39) subscales were

Table 2. Correlations Between Variables in Study 2.

Variable	1	2	3	4	5	6	7	8	9
1. Other-directed aggression	—	.53**	.39**	.53**	.43**	.32**	.21**	.21**	−.23**
2. Antisocial consequences		—	.43**	.41**	.35**	.27**	.26**	.16**	−.24**
3. Self-directed aggression			—	.28**	.33**	.38**	.34**	.30**	−.01
4. Anger				—	.55**	.38**	.27**	.28**	−.07
5. Hostility					—	.49**	.42**	.48**	−.02
6. Depressive symptoms						—	.63**	.64**	.03
7. Recent changes in anhedonia							—	.44**	−.02
8. Cognitive distortions								—	.08
9. Gender									—

Note. Correlations with gender performed using a point-biserial correlation with male coded as 1 and female coded as 2. $N = 615$.
**$p < .01$.

within normal limits [(Kline, 2005)]. The antisocial behavior subscale exhibited substantial kurtosis (skew = 3.33, kurtosis = 14.49); therefore, a log transformation was carried out. The transformed antisocial behavior subscale fell within normal limits (skew = 1.48, kurtosis = 1.14). Bivariate correlations between study variables are presented in Table 2.

Results

Following the analytic design used in Study 1, we examined our hypothesis that recent changes in anhedonia would account for the link between other depressive symptoms and history of aggression, independent of gender, hostility, anger, and cognitive distortions, via the mediation model (model 4) of the SPSS (v. 22) PROCESS macro (Hayes, 2013). Depressive symptoms served as the predictor, recent changes in anhedonia as the mediator, and hostility, anger, cognitive distortions, and gender were included in the model as covariates. We first ran the analysis with other-directed aggression as the outcome variable (Figure 2a). Contrary to our hypothesis, recent changes in anhedonia were not independently associated with other-directed aggression, $b = -.04$, 95% CI = [−.10, .02], $p = .19$, and the indirect effect of depressive symptoms on other-directed aggression via recent changes in anhedonia was not significant. Cognitive distortions also were not significant in this model, $b = -.11$, 95% CI = [−.31, .10], $p = .30$. However, other depressive symptoms were significantly positively associated with other-directed aggression, $b = .08$, 95% CI = [.03, .13], $p < .01$. Overall, the model accounted for 35.56% ($F = 55.92$, $p < .01$) of the variance in other-directed aggression.
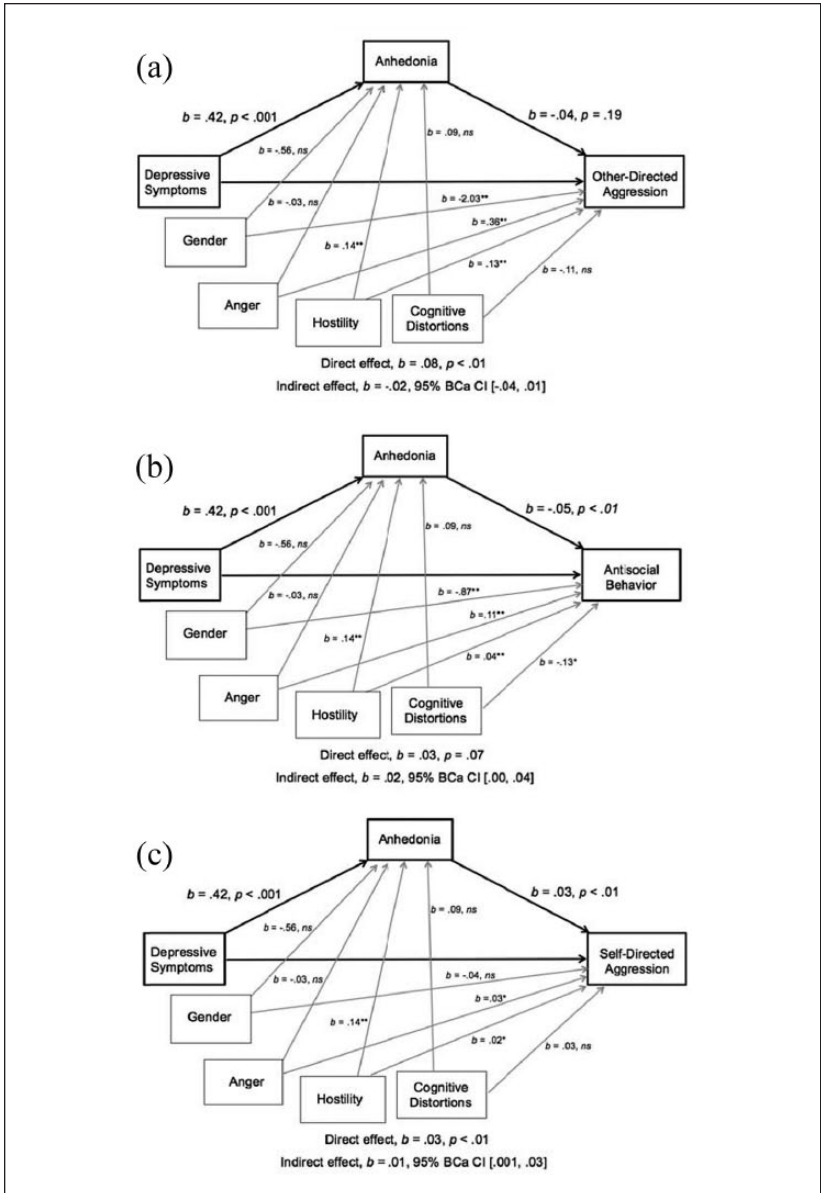
Figure 2. Mediational models for Study 2 for (a) Hypothesis 1, (b) Hypothesis 2, and (c) Hypothesis 3.
Note. Paths in gray denote covariates. *ns* = nonsignificant.
*p < .05. **p < .01.

Next, we repeated the analysis with antisocial behavior as the outcome variable (Figure 2b). Without recent changes in anhedonia in the model, depressive symptoms accounted for significant variance in antisocial behavior, $b = .004$, 95% CI = [.001, .007], $p < .01$, whereas cognitive distortions did not, $b = –.009$, 95% CI = [–.02, .01], $p = .22$. With recent changes in anhedonia included in the model, depressive symptoms were no longer associated with antisocial behavior, $b = .002$, 95% CI = [–.001, .006], $p = .16$, and the effect of recent changes in anhedonia on antisocial behavior was significant, $b = .005$, 95% CI = [.001, .008], $p = .02$. We examined the indirect effect of depressive symptoms on antisocial behavior via recent changes in anhedonia and found that this was not significant based on bias-corrected 95% confidence intervals, $b = .002$, 95% CI = [–.0002, .004], although the Sobel test did indicate a significant indirect effect, $p = .02$. Overall, the model accounted for 25.22% ($F = 34.17$, $p < .01$) of the variance in antisocial behavior.

We ran the same analysis a third time with self-directed aggression as the outcome variable (Figure 2c). With anhedonia included in the model, both anhedonia, $b = .03$, 95% CI = [.01, .05], $p < .01$, and other depressive symptoms, $b = .03$, 95% CI = [.01, .05], $p < .01$, accounted for significant variance in self-directed aggression. Cognitive distortions were not significant in this model, $b = .03$, 95% CI = [–.04, .12], $p = .40$. The indirect effect of depressive symptoms on self-directed aggression via recent changes in anhedonia was significant based on confidence intervals, $b = .01$, 95% CI = [.0000, .03], as well as the Sobel test, $p < .01$. Thus, recent changes in anhedonia partially accounted for the relationship between other depressive symptoms and self-directed aggression, although depressive symptoms also accounted for unique variance. Overall, the model accounted for 19.25% ($F = 24.15$, $p < .01$) of the variance in self-directed aggression.

Discussion

In the present pair of studies, we examined relationships between recent changes in anhedonia, other depressive symptoms (e.g., depressed mood, sleep problems, loss of appetite, difficulty concentrating), cognitive distortions (Study 2), and history of aggressive and antisocial behavior. We hypothesized that anhedonia would account for the relationship between other depressive symptoms and history of aggression; this hypothesis was based on the rationale that repeated pairing of valued goals with depressive symptoms such as elevated negative emotion is associated with anhedonia, which in turn is associated with weaker inhibitory control over aggressive and antisocial impulses. Ultimately, our hypothesis was partially supported.

Across both studies, anhedonia was significantly atemporally associated with antisocial behavior independent of other depressive symptoms, anger, hostility, gender, and in Study 2 cognitive distortions, whereas other depressive symptoms were not significantly related to antisocial behavior when anhedonia was included in the model. The indirect effect of other depressive symptoms on antisocial behavior via anhedonia was significant in Study 1 and neared significance in Study 2. Similarly, recent changes in anhedonia explained unique variance in history of self-directed aggression even when accounting for other depressive symptoms, anger, hostility, gender (Study 1 and Study 2), and cognitive distortions (Study 2). Furthermore, in both studies, the indirect effect of depressive symptoms on self-directed aggression via recent changes in anhedonia was significant.

Evidence from the present studies suggests that anhedonia might be a key symptom driving atemporal associations between depression and self-directed aggression (i.e., nonsuicidal self-injury and suicide attempts) and between depression and antisocial behavior (i.e., behavior leading to arrests or disciplinary action at work or school, illegal activities). These findings are especially compelling given the high correlation between anhedonia and other depressive symptoms ($r = .63$ in both studies). The SLIPS (Winer, Veilleux, & Ginger, 2014) strictly measures anhedonia and does not include items pertaining to other depressive symptoms, and we were careful to remove items assessing anhedonia from the measure of depressive symptoms prior to conducting analyses, so we can be confident that the independent variable and mediator in our analyses did capture separable constructs. Although it is not surprising that anhedonia and other depressive symptoms were highly related, the correlation necessarily made it more difficult to detect a unique contribution of anhedonia in accounting for variance in self-directed aggression and antisocial behavior.

The present findings regarding self-directed aggression and antisocial behavior are consistent with previous evidence indicating that anhedonia confers elevated risk of self-directed aggression, even within depressed populations (Fawcett et al., 1990; Nordström et al., 1995) and those with a history of arrests or legal convictions (Sadeh et al., 2011). Thus, it appears that positive affect such as interest, pleasure, and anticipatory excitement acts as a protective factor against the use of self-destructive coping strategies when individuals are faced with high levels of negative emotion.

One potential explanation for the protective nature of positive affect is that it maintains engagement with goals and prospective rewards. Theoretical models of self-regulation and goal pursuit broadly conceptualize positive affect as a central component of approach motivation and pursuit of prospective rewards (Carver & Scheier, 1998; M. J. Ferguson & Bargh, 2004; Kuhl,

2000). This idea is also supported by neuroscientific evidence (Alcaro et al., 2007). Engagement with adaptive goals and prospective rewards, in turn, may compete with and facilitate inhibition of antisocial and self-directed aggressive urges (Shah et al., 2002). Indeed, positive affect and brain pleasure center functioning is paramount in decisional processing and inhibition (Fellows & Farah, 2005; Kringelbach & Berridge, 2009). Conversely, loss of interest and pleasure may reflect a devaluation of prospective rewards and/or disengagement from reward pursuit (Bryant, Winer, Salem, & Nadorff, 2017; Carver & Scheier, 1998; Winer & Salem, 2016). Losing the ability to experience positive affect may negatively impact decision-making processes (Sherdell, Waugh, & Gotlib, 2012) and constitute an important individual difference that relates to aggressive acts.

When examining other-directed aggression as the outcome variable, we found a significant atemporal association with recent changes in anhedonia independent of gender, hostility, anger, and other depressive symptoms in Study 1. However, in Study 2, neither recent changes in anhedonia nor cognitive distortions accounted for significant variance in other-directed aggression. The indirect effect of depressive symptoms on other-directed aggression via recent changes in anhedonia was not significant in either study.

Thus, recent changes in anhedonia might account for some part of the relationship between other depressive symptoms and history of other-directed aggression, but for this form of aggression, anhedonia's role as a cross-sectional mediator might not be as large or robust as we initially predicted.

In formulating our hypothesis for the present pair of studies, we had conceptualized aggressive and antisocial behaviors as actions that satisfy immediate urges but ultimately are maladaptive and may reduce access to larger rewards in the future. Thus, we reasoned that interest and engagement in pursuing prospective rewards would facilitate inhibition of aggressive and antisocial urges of all types, and conversely that anhedonia would weaken individuals' ability to override urges to engage in maladaptive behaviors of all types (i.e., self-directed, other-directed, antisocial). However, based on the present pair of studies, recent changes in anhedonia appear to be more closely linked with self-directed aggression and antisocial behavior than with other-directed aggression.

There is precedent for this outcome in the literature on depressive symptoms and aggression (Sadeh et al., 2011). One possibility is that the link between anhedonia and other-directed aggression may be weaker due to the tendency for anhedonic individuals to withdraw from social interactions, thus limiting the opportunities for other-directed aggression (Sadeh et al., 2011). Another possible explanation is that depressed individuals may experience stronger or more frequent urges to engage in self-directed aggression and

antisocial behaviors than other-directed aggression, because the former may be viewed as means of regulating emotions—for example, engaging in non-suicidal self-injury in an effort to reduce negative affect (Klonsky, 2009), or using illegal drugs in an effort to generate positive affect and/or reduce negative affect (Kober, 2014). In turn, factors that weaken inhibitory control, such as loss of interest in or disengagement from pursuit of more adaptive prospective rewards, can logically be expected to have a greater impact on maladaptive behaviors that are associated with stronger or more frequent urges.

The present findings represent a step toward understanding how specific depressive symptoms may link to aggressive and antisocial behavior. In addition, this pair of studies suggests possible ways in which Reward Devaluation Theory might be integrated with theoretical models of self-regulation. Although our results were generally consistent with our theoretical rationale, further research is needed to more directly test the roles of reward devaluation and engagement with adaptive goals in determining whether and when individuals engage in aggressive and antisocial behavior. In addition, future longitudinal research can examine the temporal order of anhedonia, broader depressive symptoms, and aggressive and antisocial behavior and test for causal effects of anhedonia. Future research can also help to determine whether and why recent changes in anhedonia are more closely associated with some forms of antisocial and aggressive behavior than with others, as was found in the present studies. Ultimately, this line of investigation can inform the development of intervention strategies to reduce such behaviors in depressed or at-risk college students.

Limitations

There are several limitations to the present studies. First, because these were cross-sectional designs, findings cannot be used to infer causality, despite mediational analyses (MacKinnon, Fairchild, & Fritz, 2007). Without longitudinal data, analyses such as those employed in the present studies can only demonstrate *statistical* mediation, which does not provide any information about the order in which variables emerged. The results of the present studies must therefore be interpreted as evidence of atemporal associations and not as evidence of causal processes that might be hypothesized to unfold over time (Winer et al., 2016). We chose to structure our statistical models with depressive symptoms as the independent variable (X), recent changes in anhedonia as the mediator (M), and subscales from the LHA as dependent variables (Y) because there was established precedent in the literature for doing so (Fanning et al., 2012) and because this structure made sense given the theoretical underpinnings of our hypotheses. However, our specific

hypotheses for the present pair of studies were neither temporal nor causal in nature, and one could just as easily test atemporal relationships by setting subscales from the LHA as the independent variables and depressive symptoms as the dependent variable.

This lack of a clear pathway is not a theoretical limitation unique to this study, but rather one that must be considered whenever modeling trait-level constructs (Borsboom, Mellenbergh, & van Heerden, 2003; Markus & Borsboom, 2013). In traditional psychometrics, traits are commonly considered to be reflected by various items (i.e., aggression represented via the items on the LHA) and thus do not have clear temporal structure. In other words, traits are considered to be relatively established by early adulthood (McCrae & Costa, 1996) and by definition difficult to model related to change processes. Thus, we again note that our exploration of atemporal associations in the current manuscript is one that assesses direct and indirect influences of depression and anhedonia on aggression cross-sectionally, without making any causal or temporal claims.

Further research with longitudinal data collection is needed to test what, if any, causal relationships might exist among depressive symptoms, anhedonia, and various types of aggressive or antisocial behavior. Despite this caveat, the cross-sectional model is a vital starting point for examining new hypotheses (Carlson & Morrison, 2009). In addition, the use of a measure of recent changes in anhedonia allowed for an assessment of change from baseline, thus providing a precise investigation of the relationship between self-reported anhedonia, aggression, and antisocial behavior.

A second limitation is that the samples used in the current studies were predominantly Caucasian, and therefore, these findings may not generalize to college students from diverse racial and ethnic groups. In addition, because we focused specifically on examining relationships between anhedonia, depressive symptoms, and aggression in college students, it is unclear whether the present findings generalize to different age ranges or to populations with fewer years of education. Although emerging adults provide a uniquely strong population for examining emerging aggressive and violent behaviors (M. P. Thompson et al., 2013), future work with nationally representative samples can assess whether anhedonia accounts for the relationship between depression and aggression and depression and antisocial acts across different demographic groups and to what extent trait and recent changes in anhedonia diverge in their ability to predict aggressive and antisocial behavior. In addition, future research may also examine the relation between anhedonia, depressive symptoms, and aggression in samples evidencing clinically significant depressive symptoms (e.g., CES-D total scores above 16), given emerging evidence suggesting a link between depressive symptoms and aggression in individuals diagnosed with depressive disorders (Fazel et al., 2015).

A third limitation is the use of a nonvalidated measure to assess cognitive distortions in Study 2. For example, the true/false format of the scale could have resulted in social desirability or demand characteristics (e.g., "I certainly feel useless at times"), which we could not assess in the present study. However, the high level of agreement between clinicians determining the measure ($\kappa = .88$) provides strong evidence that the resulting internally consistent ($\alpha = .76$) subscale indeed measured negative self-beliefs as intended. Moreover, accounting for this variable allowed for stronger conclusions regarding the impact of our individual difference variables. Nonetheless, despite this novel constellation of findings, further evaluation of the independence of distorted thinking and anhedonia in relation to depression and aggression is warranted.

Finally, although the LHA is a validated measure that has been used in previous research examining depression and aggressive behavior (Berman et al., 2009; Coccaro et al., 1996), it does have limitations. For instance, the assessment of our three dependent variables using subscales derived from the same scale runs the risk of artifactually converging the constructs of other-directed aggression, antisocial behaviors, and self-directed aggression. In addition, the other-directed aggression subscale includes only one item to measure each aspect of the construct, such as verbal aggression and aggression toward inanimate objects. However, our hypotheses did not include any comparative predictions about differential effects of anhedonia in accounting for different types of aggressive behavior, so the potential overlap was not a primary concern for the purposes of the present studies. Moreover, although the subscales were inter-correlated (*r*s ranging from .36 to .61 across both studies), those correlations were far from perfect, indicating that the constructs did not overlap entirely. Finally, in both studies, divergent findings emerged from our three sets of analyses, suggesting that despite this possible limitation, we were still assessing three separable constructs, although future research may benefit from using different measures to assess these constructs, particularly those that fall under the realm of other-directed aggression (e.g., verbal and physical aggression separately).

Conclusion

The current findings suggest that anhedonia is an important individual difference that helps explain the relationship between depression and aggressive and antisocial acts, and that anhedonia may be differentially associated with various types of aggressive and antisocial behavior. Future research can investigate the manner in which these processes unfold over time and help identify other variables that predict individuals at greatest risk for aggressive and self-destructive behavior in the context of a change in anhedonia.

Declaration of Conflicting Interests

Funding

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Notes

1. In keeping with previous work on the topic (Coccaro, Berman, & Kavoussi, 1997; Sadeh, Javdani, Finy, & Verona, 2011), we use the term self-directed aggression to refer to nonsuicidal self-injury and suicide attempts.
2. A total of 670 participants were included in every instance of the model. When other-directed aggression and antisocial behavior were used as outcome variables, 286 men and 385 women comprised the total *N*. When self-directed aggression was used as the outcome variable, there were 285 men and 385 women. The discrepancy in gender breakdown is due to slight differences in participants who completed all relevant measures and thus were eligible for inclusion in analyses.

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