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Aggression Protects against the Onset of Major Depressive Episodes in Individuals with Bipolar Spectrum Disorder

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Abstract

A growing body of research suggests that bipolar spectrum disorders (BSDs) are associated with high aggression. However, little research has prospectively examined how aggression may affect time to onset of hypomanic/manic versus major depressive episodes. In a longitudinal study, we tested the hypothesis that aggression would prospectively predict a shorter time to the onset of hypomanic/manic episodes and a longer time to the onset of major depressive episodes, based on the Behavioral Approach System theory of BSDs. Young adults ($N = 120$) diagnosed with cyclothymia, bipolar II disorder, or bipolar disorder not otherwise specified were followed every four months for an average of 3.55 years. Participants completed measures of depressive and manic symptoms, family history of mood disorder, impulsivity, and aggression at baseline and were followed prospectively with semistructured diagnostic interview assessments of hypomanic/manic and major depressive episodes and treatment seeking for mood problems. Cox proportional hazard regression analyses indicated that overall, physical, and verbal aggression predicted a longer time to major depressive episode onset, even after controlling for baseline depressive and manic symptoms, family history of mood disorder, treatment seeking for mood problems, and

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impulsivity. Aggression, however, did not significantly predict time to onset of hypomanic/manic episodes, controlling for the same covariates. The findings suggest that approach-related behaviors may be utilized to delay the onset of major depressive episodes among people with BSDs.

Keywords

aggression; hypomania/mania; major depression; bipolar spectrum; behavioral approach system

A well-established finding in the literature is that individuals with bipolar spectrum disorders (BSDs) exhibit high levels of aggression and anger. For example, individuals with BSDs score significantly higher on self-report trait measures of aggression (including aggressive behavior, anger, and hostility) compared to healthy controls and to individuals with non-BSD psychopathology (Ballester et al., 2014; Dervic et al., 2015; Molz et al., 2013). Additionally, individuals with BSDs report more extensive histories of lifetime aggressive behaviors in adolescence and adulthood (Corrigan & Watson, 2005; Perroud, Baud, Mouthon, Courtet, & Malafosse, 2011), and inpatients with BSDs are almost three times more likely to display aggressive behavior while in treatment compared to inpatients with other psychiatric disorders (Barlow, Grenyer, & Ilkiw-Lavalle, 2000). Although aggression may fluctuate in BSDs according to current mood state, this feature seems to persist across contexts, rather than being a more transient, reactive occurrence (Dutra, Reeves, Mauss, & Gruber, 2014).

There is also evidence to suggest that aggression may be a possible risk factor for BSD onset. A review of the research examining prodromal symptoms of BSDs identified irritability and aggressiveness as among the most frequently reported symptoms predating onset of BSD (Skjelstad, Malt, & Holte, 2010). Similarly, in children with BSDs, symptoms of irritability/dyscontrol (including temper tantrums, poor frustration tolerance, impulsivity, increased aggression, decreased attention span, hyperactivity, and irritability) were associated with transition to more classic manic and depressive symptoms (Fergus et al., 2003). High-risk children of parents with BSDs also have been shown to score higher on measures of hostility and irritability compared to children of control parents, even when accounting for the child's own psychopathology and parental non-BSD psychopathology (Farchione et al., 2007). These findings, along with evidence that aggression in individuals with BSDs is associated with poorer functioning (Ballester et al., 2014), as well as greater violence and suicidality (Fazel, Lichtenstein, Grann, Goodwin, & Långström, 2010; Oquendo et al., 2000), underscores the need to better understand the role of aggression in BSD onset and course.

Recent work suggests that, similar to mania/hypomania, aggression might be conceptualized as an "approach emotion" associated with the Behavioral Approach System (BAS) (Carver & Harmon-Jones, 2009), the system that regulates approach motivation and goal-directed behavior to attain rewards (Alloy & Abramson, 2010; Alloy, Nusslock, & Boland, 2015; Urosević, Abramson, Harmon-Jones, & Alloy, 2008). In other words, BAS activation may be linked with aggression and anger (Carver, 2004), in addition to being associated with increased energy, positive affect, and excessive goal-directed behavior (Gray, 1994).

Specifically, anger provocation and aggressiveness are hypothesized to occur when goal striving is thwarted (e.g., obstacles or insults are presented) (Fowles, 1988; Harmon-Jones & Sigelman, 2001). In support of this, work by Harmon-Jones et al. (2002) showed that left hemisphere activation was associated with both anger and manic/hypomanic symptoms in individuals with BSD. Additionally, Molz et al. (2013) found that aggression was associated with life events that activate the BAS, and the association between BSD status and such BAS-activating events was mediated by aggression and impulsivity.

Existing research also suggests that trait aggression may be a predisposition towards experiencing BAS activation, which may put individuals at risk for experiencing hypomania/mania, but protect them against experiencing depression. For example, cognitive-behavioral deactivation strategies, such as dampening high success expectancies and participating in calming activities, have been found to reduce the likelihood of manic relapse (Lam, Wong, & Sham, 2001), whereas behavioral activation strategies protect individuals against full-blown depression (Ekers et al., 2014; Mazzucchelli, Kane, & Rees, 2009). Therefore, given the role of these activation and deactivation strategies in the development and prevention of mood episodes, trait aggression, as an approach-related tendency (Carver & Harmon-Jones, 2009), may be a risk factor for BAS activation. Such evidence implicating aggression in approach motivation suggests that aggression may be highly associated with hypomania/mania. Conversely, given that depression is theorized to be associated with low motivation or excessive BAS deactivation states (Alloy et al., 2015), it may be less compatible with aggression.

Indeed, the BAS theory of BSDs posits that vulnerability to BSDs is the result of an overly sensitive reward system that is hyperreactive to goal- and reward-relevant cues (Alloy et al., 2015). This hypersensitivity leads to excessive reward motivation and approach-related affect in response to BAS-activating events (e.g., goal-striving events), which, in turn, may lead to hypomania/mania. Importantly, this model also proposes that BSD individuals experience an excessive downregulation or decrease in behavioral approach in response to BAS-deactivating events (e.g., irreconcilable goal-thwarting events), which, in turn, may lead to depression. Thus, the hypothesized vulnerability to BSDs in the BAS model is a propensity toward both excessive reward system activation and deactivation states, which account for mania/hypomania and depression, respectively. Given that trait aggression is linked to approach motivation and BAS activation (Carver & Harmon-Jones, 2009), it may trigger hypomanic/manic symptoms and episodes by increasing the likelihood of excessive BAS activation, but at the same time, protect against depressive symptoms and episodes by decreasing the likelihood of excessive BAS deactivation among individuals with BSDs. However, no study to date has specifically examined the hypothesized association between aggression and manic/hypomanic versus depressive episodes of BSD.

The current longitudinal study examined the impact of trait aggression on time to onset of manic/hypomanic and major depressive episodes in a sample of young adults with BSDs. Based on the BAS theory of BSDs, we expected, first, that higher scores on trait-level measures of aggression would predict a shorter time to onset of manic/hypomanic episodes. Second, we hypothesized that higher trait aggression would predict a longer time to onset of major depressive episodes.

Method

Participants

Participants in this study were drawn from the Longitudinal Investigation of Bipolar Spectrum Disorders (LIBS) Project, which examined factors that influence the course of BSDs. Based on an expanded Schedule for Affective Disorders and Schizophrenia—Lifetime diagnostic interview (exp-SADS-L) (Endicott & Spitzer, 1978), which was expanded to generate both *DSM-IV-TR* (American Psychiatric Association, 2000) and Research Diagnostic Criteria (RDC) (Endicott & Spitzer, 1978) diagnoses, participants were eligible if they were diagnosed with a current or lifetime diagnosis of bipolar II disorder, cyclothymia, or bipolar not otherwise specified (BDNOS), per the *DSM-IV-TR* or RDC. The BDNOS group was comprised of individuals who had experienced: (a) hypomanic episode(s) but no diagnosable depressive episodes, (b) a cyclothymic mood pattern with periods of affective disturbance that did not meet frequency/duration criteria for hypomanic and depressive episodes, or (c) hypomanic and depressive episodes not meeting frequency criteria for a diagnosis of cyclothymia. Participants were excluded if they had experienced a manic episode as defined by the *DSM-IV-TR* or RDC criteria, as this would suggest a bipolar I diagnosis (and an aim of the overall LIBS Project was to predict conversion to bipolar I). However, individuals who eventually converted to bipolar I disorder over the course of the study were not excluded because a goal of the current study was to examine the prospective effects of trait aggression on time to onset of episodes of both hypomania and mania. All potential participants completed written informed consent procedures. All measures and procedures were approved by the institutional review boards at Temple University and the University of Wisconsin-Madison.

Measures

Buss-Perry Aggression Questionnaire (AQ)—The AQ (Buss & Perry, 1992) is a 29-item self-report questionnaire that assesses trait levels of aggression. Participants completed this measure at the baseline visit by answering each question using a five point Likert scale that ranged from “extremely uncharacteristic of me” to “extremely characteristic of me.” The AQ contains four subscales: Physical Aggression (e.g., “Once in a while I can’t control the urge to strike another person”), Verbal Aggression (e.g., “I can’t help getting into arguments when people disagree with me”), Anger (e.g., “I sometimes feel like a powder keg ready to explode”), and Hostility (e.g., “I wonder why sometimes I feel so bitter about things”), and an overall score that is the sum of the subscales (Overall Aggression). The AQ has demonstrated good retest reliability as well as concurrent validity with other measures of trait aggression (Buss & Perry, 1992). The original article reported that in a sample of college students, the mean score of Overall Aggression was 77.8 ($SD = 16.5$) for males and 68.2 ($SD = 17.0$) for females; Physical Aggression was 24.3 ($SD = 7.7$) for males and 17.9 ($SD = 6.6$) for females; Verbal Aggression was 15.2 ($SD = 3.9$) for males and 13.5 ($SD = 3.9$) for females; Anger was 17.0 ($SD = 5.6$) for males and 16.7 ($SD = 5.8$) for females; and Hostility was 21.3 ($SD = 5.5$) for males and 20.2 ($SD = 6.3$) for females (Buss & Perry, 1992). Possible scores range from 29 to 145.

Impulsive Nonconformity Scale (INS)—The INS (Chapman et al., 1984) is a self-report scale that evaluates impulsive behavior. It contains 51 true-false items (e.g., “When I want something, delays are unbearable”). Individuals scoring high on the INS in a prior study were more likely to endorse antisocial, psychotic, depressive, and hypomanic/manic symptoms than control participants (Chapman et al., 1984), and were likely to exhibit high BAS sensitivity (Alloy et al., 2006, 2009), providing support for the use of the INS as a measure of impulsivity. The INS has good retest reliability ($r = .84$) (Chapman et al., 1984) and good internal consistency ($\alpha = .79-.84$) (Alloy et al., 2006, 2009; Chapman et al., 1984). The original article reported that the mean scores on the INS were 19.0 for male control participants and 14.5 for female control participants (Chapman et al., 1984). Possible scores range from 0 to 51.

Beck Depression Inventory (BDI)—The BDI (Beck, Rush, Shaw, & Emery, 1979) is a 21-item self-report questionnaire that assesses the presence and severity of current symptoms of depression. The BDI has been used successfully to assess symptoms of depression in individuals with, and at risk for, BSDs (Ng & Johnson, 2013; Stange et al., 2012). It has demonstrated excellent psychometric properties (Beck, Steer, & Garbin, 1988). Beck (1988) reported that the mean scores on the BDI were 10.9 ($SD = 8.1$) for minimal depression, 18.7 ($SD = 10.2$) for mild depression, 25.4 ($SD = 9.6$) for moderate depression, and 30.0 ($SD = 10.94$) for severe depression. Possible scores range from 0 to 63.

Halberstadt Mania Inventory (HMI)—The HMI (Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999) is a 28-item self-report questionnaire modeled after the BDI that assesses current cognitive, motivational, affective and somatic symptoms associated with mania/hypomania. The HMI has exhibited high internal consistency ($\alpha = .82$), adequate convergent validity ($r = .32$) with the mania scale of the Minnesota Multiphasic Personality Inventory (MMPI) (Hathaway & McKinley, 1943), and discriminant validity ($r = -.26$ with the depression scale of the MMPI and $r = -.12$ with the BDI) (Halberstadt & Abramson, 2007). The construct validity of the HMI has been supported by Alloy et al. (1999). Alloy et al. (1999) reported that the mean score on the HMI for healthy individuals was 17.9 ($SD = 5.6$). Possible scores range from 0 to 84.

Revised General Behavior Inventory (GBI)—The GBI (Depue et al., 1981; Depue, Krauss, Spoont, & Arbisi, 1989) is a 73-item self-report questionnaire used to identify and distinguish between potential bipolar spectrum participants and controls. The GBI has been validated among many populations including undergraduates, psychiatric outpatients, and relatives of bipolar I probands (Depue et al., 1989; Klein, Depue, & Slater, 1985). Its psychometric properties are strong, with internal consistencies of $\alpha = .90-.96$, test-retest reliability of $r = .71-.74$, good sensitivity, and excellent specificity for bipolar spectrum disorders (Depue et al., 1981, 1989). Items were designed to assess various experiences related to depressive, hypomanic/manic, or biphasic symptoms, and how these experiences range in terms of intensity, duration, and frequency. Ratings are made on a 4-point rating scale, ranging from 1 (*not at all*) to 4 (*very often or almost constantly*), and items received a score of one point when rated as 3 (*often*) or 4 (*very often or almost constantly*) on the scale (Depue et al., 1989). Points are summed to obtain two subscores: depression (GBI-D score)

and hypomania/mania and biphasic (GBI-HB score). The following cutoff scores were used to identify potential bipolar spectrum and control participants: GBI-D score of 11 and GBI-HB score of 13 for potential bipolar spectrum participants (Depue et al., 1989). A pilot study validated this high- and low-GBI group assignment procedure against diagnoses obtained via the exp-SADS-L interviews (Alloy et al., 2008). The mean scores on the GBI reported by the original article were 10.73 ($SD = 9.69$) for males and 12.59 ($SD = 11.73$) for females (Depue et al., 1981). Possible scores range from 73 to 292.

Exp-SADS-L—The exp-SADS-L (Alloy et al., 2008; Alloy, Urošević, et al., 2012) is a semistructured interview assessing symptoms related to mood, anxiety, eating, psychotic, and substance use disorders over the lifetime. All interviews were conducted by trained interviewers who were blind to GBI scores. To obtain consensus diagnoses and monitor inter-rater reliability, interviews were audiotaped, and expert psychiatric consultants were used as the third diagnostic tier for diagnostic consensus. Inter-rater reliability was high ($K > .95$ for major depressive disorder diagnoses; $K > .96$ for bipolar spectrum diagnoses) (Alloy et al., 2008). Nusslock et al. (2007) provide further details about exp-SADS-L diagnoses and the extensive interviewer training.

Expanded Schedule for Affective Disorders – Change Version (exp-SADS-C)—

The exp-SADS-C (Endicott & Spitzer, 1978) is a semistructured interview that obtains information about the occurrence, duration, and symptom severity of psychiatric disorders. The exp-SADS-C was used to diagnose *DSM-IV-TR* episodes of major depression and hypomania/mania occurring across the prospective follow-up period (Alloy, Bender, et al., 2012; Alloy et al., 2008; Francis-Raniere, Alloy, & Abramson, 2006; Stange et al., 2015; Weiss et al., 2015). Exp-SADS-C interviewers were blind to participants' GBI scores, exp-SADS-L diagnoses, and baseline aggression, impulsivity, and mood symptom scores. Validity and inter-rater reliability for the exp-SADS-C were strong ($K > .80$) (Alloy et al., 2008; Francis-Raniere et al., 2006). Nusslock et al. (2007) provide further details about exp-SADS-C diagnoses and interviewer training.

Procedure

A two-phase participant selection process was used. In Phase I, 20,500 students from Temple University and the University of Wisconsin-Madison completed the revised General Behavior Inventory (GBI) (Depue et al., 1989). Based on the GBI cutoffs (see *Measures*), 1,730 potentially eligible individuals completed the exp-SADS-L for Phase II screening. At a baseline visit following Phases I and II, participants eligible for the longitudinal study completed self-report measures of aggression, impulsivity, and symptoms of depression and hypomania. Diagnostic interviews assessing *DSM-IV-TR* episodes of major depression and hypomania/mania and treatment-seeking for mood problems were completed every four months after the baseline visit for an average of 3.55 years of follow-up ($M = 1297.52$ days; $SD = 359.20$ days). The maximum number of prospective assessments participants completed was 12. The minimum number was one. The average number of prospective assessments completed was 5.85 ($SD = 3.45$). The average number of days between assessments was 153.45 ($SD = 129.99$).

Statistical Analysis

Cox proportional hazard regression (survival) analyses were used to evaluate the hypothesis that aggression predicted a longer time to onset of episodes of major depression and a shorter time to onset of episodes of hypomania/mania. We utilized survival analysis because it allows for different follow-up lengths and varying intervals between assessments, minimizes biases due to attrition, utilizes all available data at each time point, and accommodates right-censored cases, who leave the study before a target event (i.e., mood episode onset) occurs or who do not experience a target event over follow-up (Willett & Singer, 1993). Aggression scores were entered as the independent variables, and time to onset of a hypomanic/manic or major depressive episode were entered as the dependent variables. Baseline depressive and manic symptoms were entered as covariates to control for any effects of baseline mood symptoms on time to onset of mood episodes. Treatment seeking for mood problems during follow-up (yes/no) was included as a covariate to control for the possibility that treatment might alter time to onset of mood episodes. Family history of mood disorder in first-degree relatives was entered as a covariate to control for familial effects. We also included impulsivity as a covariate to ascertain that any prediction of the onset of a mood episode by aggression was above and beyond the effects of impulsivity on the recurrence of mood episodes (Ng et al., 2016). We also examined Variance inflation factor (VIF) statistics to assess possible multicollinearity problems. None of the VIF statistics for any variable was above 1.5, suggesting multicollinearity was not a problem.

Results

The final sample for this study consisted of 120 participants (47 males, 73 females) aged 17–28 years ($M = 19.71$ years, $SD = 1.76$ years). The ethnic composition of the sample was 75.00% Caucasian, 12.50% African American, 5.00% Hispanic, 2.50% Asian, 0.80% Native American, and 4.20% Other/Prefer not to answer. At the initial diagnostic interview (Phase II), there were 31 participants with cyclothymia or BDNOS and 89 participants with bipolar II disorder. Sixty-two (51.70%) of the 120 BSD participants had a positive family history of mood disorder among first-degree relatives; 82 (68.30%) sought treatment for mood symptoms at some time during the 3.55 year follow-up; 91 (75.80%) participants experienced at least one manic or hypomanic episode; and 72 (60%) experienced at least one major depressive episode over the follow-up period. Table 1 presents the descriptive statistics and correlations between the variables examined in the current study.

To test the hypothesis that aggression predicted time to onset of depressive or hypomanic/manic episodes, we conducted Cox proportional hazard regression (survival) analyses with days to onset of depressive or hypomanic/manic episode since baseline as the outcome. Baseline depressive and hypomanic symptoms, treatment-seeking status, family history of mood disorder, and impulsive non-conformity (INS) score were entered in Step 1 to account for effects of any of these characteristics on time to onset of episodes of major depression or hypomania/mania, while aggression (Overall Aggression, Anger, Hostility, Verbal Aggression, and Physical Aggression) score on the AQ was entered in Step 2 in separate models. Controlling for these covariates, higher baseline Overall Aggression predicted longer time to onset of major depressive episodes (Wald = 4.957, $p = .026$, OR = .983, 95%

CI = .968–.998), but it did not significantly predict time to hypomanic/manic episodes (Wald = 1.148, $p = .248$, OR = .992, 95% CI = .978–1.007). Scores on two of the four subscales of the AQ, Physical Aggression and Verbal Aggression, also predicted longer time to onset of depressive episodes (Physical Aggression, Wald = 11.432, $p = .001$, OR = .933, 95% CI = .896–.971; Verbal Aggression, Wald = 8.251, $p = .004$, OR = .917, 95% CI = .865–.973), but they did not significantly predict time to hypomanic/manic episodes (Physical Aggression, Wald = 1.954, $p = .162$, OR = .978, 95% CI = .948–1.009; Verbal Aggression, Wald = .002, $p = .962$, OR = .999, 95% CI = .948–1.052). The other two subscales of the AQ, Anger and Hostility, did not significantly predict time to onset of episodes of hypomania/mania (Anger, Wald = .095, $p = .758$, OR = .993, 95% CI = .949–1.039; Hostility, Wald = .585, $p = .445$, OR = .984, 95% CI = .946–1.025) or major depression (Anger, Wald = .075, $p = .784$, OR = 1.007, 95% CI = .960–1.055; Hostility, Wald = .016, $p = .898$, OR = 1.003, 95% CI = .962–1.045). The survival curves for high versus low Overall Aggression, Physical Aggression, and Verbal Aggression (plotted using a median split) predicting time to onset of major depressive episodes are presented in Figures 1–3 respectively.

Discussion

Given the high rates of mood episode recurrence in BSDs, it is of high priority to identify risk and protective factors for the recurrence of mood episodes. The current study provided a theoretically guided and methodologically rigorous examination of the hypothesis that trait aggression would put people with BSDs at risk for episodes of hypomania/mania, but at the same time, protect them against episodes of major depression, based on the BAS sensitivity theory of BSDs (Alloy et al., 2015). The hypothesis was partially supported. The results of the survival analyses indicate that whereas overall, physical, and verbal aggression predicted a longer time to onset of major depressive episodes, none of the aggression scales predicted time to onset of hypomanic/manic episodes over an average of 3.55 years of follow-up, after taking into account baseline depressive and manic symptoms, impulsivity, family history of mood disorder, and treatment-seeking status over follow-up.

The findings of the current study are congruent with the theory that BAS-activating emotions and behaviors may be protective in delaying the occurrence of depression (Alloy et al., 2015). Aggressive tendencies may decrease withdrawal and low energy and motivation, and thus, prolong the time to onset of major depressive episodes. However, it may be that aggression is only BAS-activating enough to prevent people from experiencing excessive BAS deactivation, which is hypothesized to cause major depression, but not enough to lead to excessive BAS activation, which is hypothesized to cause hypomania/mania. In a similar vein, anger and hostility, which may trigger less BAS activation than physical and verbal aggression, may not activate the BAS enough to protect individuals with BSDs from excessive BAS-deactivation states, which explains the current results that anger and hostility did not predict longer time to major depression onset.

The current study has several methodological strengths. These include the use of a longitudinal sample not recruited from a clinic population, semistructured diagnostic interviews, standardized diagnostic criteria, diagnosticians blind to the aggression levels of participants, a conservative statistical analysis approach, relatively long overall follow-up

period, and relatively short follow-up assessment intervals, which provided high sensitivity for the detection of mood episodes over follow-up.

Notwithstanding these strengths, it is also important to note the limitations of the study. First, we relied on a self-report measure to assess trait aggression. Future studies should examine whether objective measures of aggression also predict a longer time to onset of major depressive episodes. Second, our sample consisted of university students. Therefore, our results may not be generalizable to other populations. However, it is important to note that the participants met *DSM-IV-TR* and/or RDC diagnostic criteria for a BSD and exhibited features that characterize individuals with BSDs (e.g., 68% of the participants sought treatment for mood symptoms at some time during the 3.55 year follow-up). In addition, the participants were relatively early in the course of their BSD, which might decrease the confounding effects of years of treatment. However, it is also important to highlight that the current findings may not generalize to healthy controls, given that the study consisted of only participants with BSDs. Third, family history of mood disorder was obtained indirectly by interviewing the participants rather than their relatives. Fourth, we did not measure state aggression, which should be a more proximal predictor of states of BAS activation and deactivation. Future studies might examine the exact role trait and state aggression play in the course of BSDs and whether state aggression mediates the relationship between trait aggression and time to onset of major depressive episodes. Future research also should examine how to more directly measure BAS-activation and -deactivation states because these states are hypothesized to lead to hypomania/mania and depression in the BAS theory of BSDs. Fifth, the current study did not examine how cultural differences in aggression may affect the relationship between trait aggression and time to mood episodes (Archer, 2006; Martin, Manuel, & Fujihara, 2001). This is an important area for future research. Sixth, a few aspects of the BAS theory of BSDs were not examined in the current study. For example, we did not test whether trait aggression interacts with BAS-related events to predict mood symptoms and episodes. In addition, we did not measure BAS activation and deactivation states and examine how they may predict hypomania/mania and depression. Finally, we did not examine the mechanisms that may underlie the association between aggression and longer time to onset of depressive episodes in BSDs, such as sleep disturbance (Ng et al., 2015; Ng, Chung, Lee, Yeung, & Ho, in press). Future studies should address these questions.

The findings of the current study raise the interesting possibility that approach-related or BAS-activating behaviors may be utilized to delay the onset of major depressive episodes among people with BSDs. Specifically, it may be useful for people with BSDs to engage in behaviors that will activate the BAS enough to protect them against major depression, but not so much as to trigger hypomania/mania. The current study does not suggest that aggression should be prescribed as a form of prevention. Rather, there are other activation strategies that may be more adaptive and practical than aggression (Manos, Kanter, & Busch, 2010). For example, there is a large body of literature demonstrating that behavioral activation strategies protect individuals against full-blown depression (Ekers et al., 2014; Mazzucchelli et al., 2009). Given that people with BSDs spend more days depressed than manic (Kupka et al., 2007), studies examining what behaviors are able to trigger a balanced

level of BAS activation may be warranted and may lead to more fine-tuned interventions that help reduce the impact of major depression in BSDs.

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Figure 1.
Time to major depressive episode as a function of high versus low overall aggression (plotted using a median split).

Figure 2.
Time to major depressive episode as a function of high versus low physical aggression (plotted using a median split).

Figure 3. Time to major depressive episode as a function of high versus low verbal aggression (plotted using a median split).

Table 1

Correlations and Descriptive Statistics of Study Variables

	OA	PA	VA	Anger	Hostility	INS	BDI	HMI	Tx	FamHist	DaysHM	DaysMD
OA	—	.766**	.664**	.796**	.720**	.451**	.161	.036	.044	.025	-.022	.032
PA		—	.400**	.392**	.331**	.376**	.173	.009	.101	-.020	.024	.145
VA			—	.501**	.237**	.378**	.019	.064	-.150	-.103	-.107	.131
Anger				—	.526**	.356**	.116	.033	.054	.061	-.045	-.067
Hostility					—	.235**	.129	.017	.060	.113	.025	-.118
INS						—	.086	.185*	.018	-.060	-.185*	-.118
BDI							—	-.148	.282**	.083	.080	-.250**
HMI								—	-.073	-.094	.073	.290**
Tx									—	.238**	.114	-.169
FamHist										—	.096	.004
DaysHM											—	.328**
DaysMD												—
<i>M</i>	70.25	18.73	15.22	17.12	19.18	17.58	10.29	14.45	—	—	463.06	674.37
<i>SD</i>	17.08	7.25	4.30	5.44	6.03	8.39	10.68	9.68	—	—	536.20	592.47
<i>α</i>	.91	.80	.82	.79	.82	.86	.94	.83	—	—	—	—

Note. OA = Buss-Perry Aggression Questionnaire (AQ) Overall Aggression; PA = AQ Physical Aggression; VA = AQ Verbal Aggression; Anger = AQ Anger; Hostility = AQ Hostility; INS = Impulsive Nonconformity Scale; BDI = Beck Depression Inventory; HMI = Halberstadt Mania Inventory; Tx = Treatment-seeking for mood problems; FamHist = Family history of mood disorders; DaysHM = Days without a hypomanic/manic episode; DaysMD = Days without a major depressive episode.

* $p < .05$.

** $p < .01$.