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The Effects of Aggression on Symptom Severity and Treatment Response in a Trial of Cognitive Behavioral Therapy for Panic Disorder

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Abstract

Background—Previous research suggests that patients with panic disorder exhibit higher levels of aggression than patients with other anxiety disorders. This aggression is associated with more severe symptomatology and interpersonal problems. However, few studies have examined whether higher levels of aggression are associated with a worse treatment response in this population.

Methods—The present study sought to examine the association of aggression with panic disorder symptom severity in a sample of 379 patients who participated in a trial examining long-term strategies for the treatment of panic disorder.

Results—We found that aggression was significantly associated with higher baseline levels of panic disorder symptoms, anxiety, depression, and functional impairment. Further, we found that patients higher in aggression did not achieve the same level of improvement in general anxiety symptoms during treatment compared to patients lower in aggression, even when controlling for baseline anxiety symptom severity.

Conclusion—These results suggest that more research is needed concerning patients with anxiety disorders with higher aggression, as they may be a group in need of additional treatment considerations.

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Keywords

Panic Disorder; Anxiety; Anger; Aggression

Introduction

Aggression is often considered a maladaptive behavioral manifestation of anger. While anger is a normal reaction to perceived mistreatment and can motivate individuals to stand up for themselves or defend loved ones in a healthy, assertive way, dysregulated anger and aggression can disrupt personal health and functioning¹ and can lead to behaviors that harm others^{2,3}. In general, individuals with anxiety or depressive disorders appear to be susceptible to experiencing elevated anger and aggression^{4,5,6}. Importantly, individuals who have elevated anger in the context of an anxiety disorder are seemingly unique from those who are high in trait anger (e.g., inmates, spouse abusers, etc.) because their anger develops in the context of the disorder and tends to remit when the anxiety disorder is treated⁸.

Elevated anger and aggression in anxiety disorders can result in higher levels of distress and impairment in a variety of ways^{8,9,10,11}. Patients with co-morbid anger and anxiety exhibit higher levels of suicidality than patients without co-morbid anger^{12,13}. Additionally, anger strains interpersonal relationships, which may already be weakened by the presence of an anxiety disorder, and potentially attenuates needed social support^{8,14}. Further, patients with emotional disorders and co-morbid anger often have a poorer prognosis than patients without co-morbid anger. Even though dysregulated anger may remit when the disorder is treated, evidence suggests that anger plays a role in the maintenance of anxiety disorders, making it more difficult to treat anxiety when anger is present and is associated with less favorable treatment outcomes^{15,16}.

Some research suggests that anger may be differentially elevated across the anxiety disorders and that patients with panic disorder (PD) may have significantly higher levels of anger and aggression compared individuals experiencing other anxiety or related disorders (i.e. obsessive-compulsive disorder, social anxiety disorder, and generalized anxiety disorder) and healthy controls^{4,9,17,18}. Further, one study¹⁷ found that patients with PD make attempts to control their anger more than healthy individuals and the authors suggested that this control or “bottling up” of anger may represent emotional processing difficulties (i.e., a maladaptive way of processing stressful life events). It is also possible that patients concerned with emotional control might find strong emotions, including anger, threatening and have difficulty tolerating their emotional experiences¹⁹. In another study, lifetime problems with anger expression remained associated with PD even when controlling for demographic variables, other anxiety disorders, bipolar disorder, substance use disorder, borderline personality disorder, and depression⁴. This result suggests a unique relationship between lifetime PD and difficulties with anger expression.

Although aggression would appear to be an important element of anger to understand given its social (and potentially legal) costs, the correlates and effects of aggression in the context of PD have not been well explored. Existing, albeit limited, research suggests that, similar to aggression in other anxiety disorders, aggression in PD is associated with greater symptom

severity, impulsivity, and suicide risk²⁰. In treatment, higher scores on patient aggression are associated with lower ratings of therapist adherence to treatment protocols, suggesting that patients with higher aggression and hostility may be engaging in treatment- or therapist-rejecting behaviors that could reduce the effectiveness of the treatment being delivered²¹.

To our knowledge, no study has examined the relationship between aggression and treatment outcomes in the context of PD specifically, despite the evidence indicating that aggression in PD is associated with a variety of negative outcomes. The purpose of this study was to 1) explore the association of aggression with PD severity, overall anxiety severity, overall depression severity, and functional impairment in a study of long-term treatment strategies for patients initially treated with cognitive behavioral therapy (CBT) for PD and 2) examine whether aggression is associated with less improvement during acute treatment and later phases. Our hypotheses were as follows: 1) higher aggression would be significantly associated with greater symptom severity and interference at baseline, 2) higher aggression would predict a lower likelihood of response to CBT in the acute treatment phase, 3) higher aggression would predict a higher likelihood of treatment attrition, and 4) higher aggression would predict less symptom improvement during acute treatment and later phases.

Method

Participants

A total of 379 adults completed a baseline interview and met criteria for a primary diagnosis of PD (see Aaronson et al., 2008, for detailed inclusion/exclusion criteria)²². The majority of the patients were female (65.4%) and identified as Caucasian (86.8%). Additionally, 5.3% of the patients identified as African American, 5.8% identified as Asian or Pacific Islander, 1.1% identified as American Indian or Alaskan Native, and 1.1% identified as “other”. The patient’s ages ranged from 18-70 ($M = 37.5$, $SD = 11.89$). This study took place at four sites and was approved by the institutional review board at each site. A human subjects review board (IRB) approved all study procedures and all participants signed an informed consent form before completing any study procedures.

Measures

Interpersonal aggression—The interpersonal aggression subscale of the abbreviated version of the Inventory of Interpersonal Problems-Personality Disorders screening instrument²³ (IIP-PD), hereafter referred to as aggression, was completed at baseline to assess pre-treatment levels of aggression (active hostility/aggression towards others). Individuals indicated the extent to which they agreed with each of five statements (i.e., “I argue with other people too much,” “I lose my temper too easily,” “I fight with other people too much,” “I get irritated and annoyed too easily,” and “I am too aggressive toward other people”) on five-point scales, which were then averaged to achieve the subscale score. The interpersonal aggression subscale has strong internal consistency with a Cronbach’s alpha of .86²⁴.

Panic Disorder Severity—The Panic Disorder Severity Scale²⁵ (PDSS) was used in this study to specifically assess the severity of PD symptoms. This scale is a seven item clinician-administered interview that assesses seven dimensions associated with panic attacks: 1) frequency of panic attacks; 2) distress during panic attacks; 3) anticipatory anxiety (worry about future panic attacks); 4) agoraphobic fear and avoidance; 5) interoceptive fear and avoidance (i.e., apprehension and avoidance of bodily sensations); 6) impairment of or interference in work functioning; and 7) impairment of or interference in social functioning. Based on the patient's response to each question, the clinician rates the response on a scale of zero (none) to four (extreme). A summary score is obtained by summing all items and higher scores indicated greater severity. This measure has good internal consistency ($\alpha = .65$) and has demonstrated sensitivity to change throughout treatment²⁵.

Overall Anxiety—The Hamilton Anxiety Rating Scale²⁶ assessed overall anxiety severity in the current study. This assessment is a 14-item clinician-administered interview. Each item assesses a symptom of anxiety (ex. insomnia, anxious mood) and scores range from zero (none) to four (very severe). An overall score is obtained by summing all items and higher scores indicate greater symptom severity. This measure was administered with the Structured Interview Guide for the Hamilton Anxiety Rating Scale (SIGH-A) and has strong internal reliability ($\alpha = .85$). It has been used extensively in clinical trials as a measure of anxiety severity²⁷.

Depression—The Hamilton Rating Scale for Depression²⁸ assessed depression severity. This measure was administered with the Structured Interview Guide for the Hamilton Rating Scale for Depression (SIGH-D). This measure is a clinician-administered interview containing 17 items. Each item assesses a symptom of depression including depressed mood, suicide, and loss of interest. The clinician rates each patient's response on a scale ranging from zero (absent) to four (severe). Some items, for which quantification is difficult or impossible (e.g., insomnia), are scored on a scale ranging from zero (absent) to two (clearly present). An overall score is obtained by summing all 17 items and higher scores indicate greater symptom severity. This measure has been used extensively in studies of depression and it has strong reliability at the total score level²⁹ ($\alpha = .81$).

Functional Impairment—The Work and Social Adjustment Scale³⁰ (WSAS) was used to assess impairment in work and social activities due to psychopathology. This measure is a five item self-report questionnaire with items assessing how the way a patient feels impacts his or her functioning. Responses are given on a Likert scale ranging from zero (not impaired at all) to eight (severely impaired). A total score is obtained by summing all items and higher scores indicate greater levels of severity. The measure has been used in several studies examining anxiety disorders and evidences strong internal consistency with Cronbach's alpha estimates ranging from .79 to .94 across different studies³⁰.

Procedure

The current study was conducted using data from a large clinical trial assessing long-term strategies (LTS) for the treatment of PD and consisted of three phases. See Figure 1 for an

illustration of the participant flow through the study. The first phase of the trial consisted of 12 weeks of acute treatment with cognitive behavioral therapy (CBT). A total of 256 patients completed this phase. After the first phase, patients were classified as responders or non-responders and re-randomized for the second phase of treatment. Patients were considered treatment responders if they: 1) received a score of “2” (much improved) or “1” (very much improved) on the Clinical Global Impressions scale³¹, 2) they had at least a 40% reduction in PDSS score, and 3) had no panic attacks in the past month. Non-responders did not meet at least one of these conditions. During the second phase, responders were randomized to one of two conditions: no treatment (assessment only) or maintenance CBT for nine months. In the third phase of the study, all responders received assessments only (no treatment) for an additional twelve months. In total, responders were followed for 21 months after the acute CBT phase.

Non-responders, on the other hand, were randomized to one of two conditions for three months: pharmacotherapy with paroxetine, or continued CBT. In the third phase, non-responders who achieved responder status from either the CBT or paroxetine condition continued to receive the same treatment for an additional nine months. Patients classified as non-responders after the three-month trial of CBT or paroxetine were referred outside of the study, but were still followed for assessments. In total, non-responders were followed for a total of 12 months. An assessment (major assessment or MA) occurred at baseline and after the completion of each phase. Thus, MA1 was the baseline assessment; MA2 was the post-acute assessment for the first 12 weeks of CBT; MA3 followed the second phase; and MA4 followed the third phase. Each post assessment served as the baseline for the next phase. The assessments of interest in this study were only given at major assessment points. Results from the acute phase of the study are described in Aaronson et al. (2008)²². Results from the second and third phases for the responder study are described in White et al. (2013)³². Results for the non-responders study are described in Payne et al (under review)³³.

Data Analytic Strategy

Hierarchical linear modeling using maximum likelihood estimation (HLM) was utilized to assess a subset of the study hypotheses given the longitudinal, multi-assessment nature of the data³⁴. Separate models were examined for each dependent variable (SIGH-A, PDSS, SIGH-D, WSAS) using the Proc Mixed procedure in SAS software, version 9.4. Each model examined the effect of baseline aggression and time interval (i.e., the number of months since post-acute treatment) on the dependent variable in question. Due to significant correlations between aggression and baseline symptom measures, we included the respective baseline measure for each dependent variable as a covariate. To facilitate interpretation, baseline aggression and baseline symptom measures were grand-mean centered. Random effects were estimated for intercept only (not slope) during the post-treatment interval. Preliminary analyses indicated the necessity of fixing the random effect of slope for each model to correct for boundary constraints; thus, we do not examine systematic residual variation in slope over the post-acute-treatment period.

Results

Preliminary Analyses

First, descriptive statistics were examined. No outliers were discovered. Means and standard deviations for all study variables at every assessment time point can be found in Table 1.

Relationship between Aggression and Symptom Severity at Baseline

Next, we examined the relationship between aggression and symptom severity with a series of zero-order correlations between aggression scores and measures of PD symptom severity, anxiety, depression, and functional impairment (see Table 2). Overall, the results indicated that aggression was significantly related to all outcome variables, with moderate effect sizes, such that higher aggression was associated with higher symptom severity at baseline.

Relationship Between Treatment Response and Initial Aggression

Treatment response and attrition—As we have already established relationships between baseline aggression and symptom severity, and with the knowledge that severity may account for some variance in predicting responder status or risk for attrition, we controlled for baseline PD severity (using PDSS score) in the following logistic regression analyses. To aid in interpretation, we standardized the aggression variable ($M = 0$, $SD = 1$). When predicting responder status, controlling for baseline PD severity, a test of the full model against an intercept-only model was not statistically significant, ($\chi^2(1) = 2.45$, $p = .29$), suggesting that the predictor variables (baseline PD severity and aggression) as a set do not reliably distinguish between responders and non-responders. The Wald criterion demonstrated that aggression did not make a significant contribution to predicting responder status ($p = .45$). An odds ratio (OR) of 1.15, 95% CI [.80, 1.65] suggests that a one-unit increase in aggression (i.e., one SD above the mean) is associated with 15% greater likelihood of not achieving responder status after the acute CBT treatment.

When evaluating the relationship between attrition and aggression, controlling for baseline PD severity, a test of the full model against an intercept-only model was statistically significant at every time point, indicating that the predictors (baseline PD severity and aggression) as a set reliably distinguished between patients who did or did not drop out of treatment at the end of each phase ($\chi^2(1) = 6.42$, $p = .04$; $\chi^2(1) = 10.88$, $p = .004$; $\chi^2(1) = 20.34$, $p < .0001$, respectively).

The Wald criterion demonstrated that aggression was related to attrition at the end of the acute phase at the level of a nonsignificant trend ($p = .098$). Odds ratios suggested that when aggression increased by one unit, the likelihood of attrition was 21% (OR = 1.21, 95% CI [.97, 1.51]). Similar analyses indicated that baseline aggression did significantly predict differences in attrition at the end of the second phase ($p = .001$) and third phase ($p < .001$). Odds ratios suggested that when aggression increased by one unit, the likelihood of remaining in treatment decreased by 43% (OR = 1.43, 95% CI [1.15, 1.79]) at the end of the second phase and 66% by the end of the third phase (OR = 1.66, 95% CI [1.31, 2.10]).

Initial Unconditional Models—We first examined the unconditional means models for each variable to evaluate their respective magnitude of within-person and between-person variability. Intraclass correlation coefficients (ρ), which describe the proportion of variance in each outcome variable that is between-persons, indicated there was substantial between-person, and thus also within-person, variability in outcome variables ($\rho = .48$ for the SIGH-D, $\rho = .54$ for the PDSS, and $\rho = .56$ for the WSAS and SIGH-A). An evaluation of the unconditional growth models for each variable indicated that each variable's intercept (which is centered at the post-treatment assessment) was significantly different than zero and that each variable's slope during the post-treatment period was significantly different from zero (see Table 3).

Effect of Baseline Aggression on Treatment Response—HLM analyses, as described previously, assessed whether patients' baseline aggression levels resulted in poorer treatment response above and beyond initial symptom severity. Table 4 includes the parameter estimates. Results indicated each variable exhibited significant improvement over the post-acute-treatment period. Controlling for baseline severity and condition, individuals with higher baseline aggression exhibited significantly higher scores on the SIGH-A over the post-acute-treatment period; the relationship between baseline aggression and SIGH-D was at the level of a non-significant trend. Controlling for baseline severity and condition, individuals with higher baseline aggression did not exhibit significantly higher scores on the WSAS or PDSS over the post-acute-treatment. While scores on all the measures continued to improve during the post-acute-treatment period, the magnitude of the relationship between baseline aggression and the SIGH-A was such that individuals scoring one standard deviation above the mean on baseline aggression scored approximately 2.1 points higher on the SIGH-A than individuals scoring one standard deviation below the mean on baseline aggression across the post-acute-treatment period.

Discussion

As expected, higher aggression was significantly associated with higher panic symptoms, overall anxiety, depression, and functional impairment at baseline, which is consistent with previous research that has found relationships between these constructs²⁰. Additionally as expected, patient aggression was also associated with a significantly higher likelihood of attrition. A review of previous literature indicates the identification of factors related to attrition in individuals with PD has been somewhat difficult. In three large studies of cognitive behavioral therapy for PD, this sample included³⁷, researchers examined many potential factors related to attrition (in one study, over 50 variables). Across these previous investigations, greater negative attitudes toward treatment³⁵, lower motivation for treatment³⁶, lower household income³⁵, younger age³⁷, and lower education³⁶ were the only variables that emerged as predictors of attrition. Thus, the identification of aggression as a significant predictor of attrition is of interest in a small literature characterized by limited findings. While aggression-attrition relationships have rarely been examined, increased anger and hostility have been found to predict attrition in individuals with borderline personality disorder³⁸, bulimia nervosa³⁹, and posttraumatic stress disorder⁴⁰. To the best of our knowledge, this relationship has not been previously examined in PD. It is possible that

patients with higher aggression have greater difficulty forming a positive therapeutic relationship that connects them to treatment, are more likely to discontinue treatment if they become frustrated with some aspect of the therapy process, or have difficulties effectively communicating with their therapist, which can disrupt the therapeutic alliance. Any of these possibilities may account for their higher attrition rate.

However, while the specific reasons for the aggression-attrition relationship are not fully clear, our results also suggest that, for those patients who stay in treatment, aggression did not have a significant impact on likelihood of being a treatment responder during the acute CBT period (first 12 weeks) and did not affect the likelihood of experiencing improvement in PD severity and functional impairment at the end of the acute CBT period and over the remainder of the study. As these results were not as initially hypothesized, it is possible that the greater likelihood of attrition for patients with higher aggression may have lessened the ability to observe relationships between aggression and PD severity and functional impairment. However, our results are also consistent with the possibility that PD-focused CBT treatments are sufficient for reducing targeted symptoms regardless of aggression level if patients remain in treatment. Taken together, our findings suggest that therapists who identify PD patients with higher levels of aggression at the beginning of treatment should take steps to address this aggression directly, such as discussing treatment fidelity with the patients early on, expressing openness to discussing patients' concerns or frustrations during the course of treatment, and checking in periodically with patients regarding their reactions and attitudes about the treatment. These practices might increase the likelihood that patients with higher levels of aggression will complete treatment and see comparable improvement in symptoms targeted by this PD-focused CBT treatment.

Importantly, we also observed that patients with higher aggression did not experience as much improvement in terms of overall anxiety and (to a lesser extent) depressive symptoms during acute CBT treatment, and that this pattern did not change during the post-acute-treatment period. This set of results indicates that PD-focused CBT may not be as successful in reducing comorbid symptoms in those with higher than average aggression. There are a couple plausible reasons for the poorer prognosis of PD patients observed in our study. First, previous research indicates that therapists are often less adherent to treatment protocols when working with aggressive patients, possibly due to patient hostility or therapist-rejecting behaviors in session. Second, these protocols are not designed to address aggression in the context of an anxiety disorder²¹. Therefore, when a patient with PD and higher than average aggression is identified at their intake assessment, therapists could decide to target aggressive behaviors early in treatment, which may increase patients' chances of more broadly benefitting from (and completing) a PD-focused treatment. Alternatively, or additionally, therapists could focus on providing highly adherent therapy in the face of increased in-session hostility.

Consistent with our results (and previous research) establishing an association between panic and aggression and the implications for treatment of PD with CBT, it is worth noting that another treatment for PD, panic-focused psychodynamic psychotherapy, does more explicitly recognize the panic-anger association^{41,42}. This therapy views conflicts about anger as a core dynamic that contributes to the onset and maintenance of panic disorder.

Panic-focused psychodynamic psychotherapy has demonstrated initial efficacy in the treatment of PD, but the extent to which this treatment reduces anger and aggression is less known⁴³. As it is likely that aggression would be managed differently in CBT than psychodynamic therapy (i.e., a focus on anger-related thoughts and behaviors in CBT versus a focus on the conflict caused by anger in psychodynamic therapy), research on CBT treatment that targets aggression directly in patients with PD is necessary to identify effects that might be specific to either treatment.

Finally, the results of this study should be considered in the context of its limitations. First, the assessment of aggression used is typically a screening measure and does not provide in-depth information regarding a patient's aggression levels. Further, as the IIP-PD is a self-report measure, there is the possibility that responses could be affected by a tendency to misperceive behaviors as aggressive when they are not. Results from previous research are mixed as to whether individuals with PD are more likely to have impairments in the ability to effectively perceive and express emotions^{44,45}. With this in mind, it is possible that the relationships we observed between initial aggression and initial symptom severity could be in part due to more severe patients interpreting their behavior as more aggressive. However, we controlled for initial symptom severity in our longitudinal analyses, which minimizes the potential for this alternative explanation to have driven those results. We encourage future research on aggression in clinical populations to consider patients' perceptions of their aggression and include measures of objective aggressive behaviors and perceived aggression in order to identify the relative contributions of each.

Second, we were unable to examine differences in response based on aggression level within treatment conditions due to a lack of power. Third, this study did not have a control group. Therefore the trajectory of patients with PD and higher aggression levels who do not receive CBT is unknown, limiting inferences regarding the utility of CBT for PD with heightened aggression compared to no treatment. These limitations can be addressed in future research by including more comprehensive measures of aggression, including a clinician-rated measure of aggression, as well as examining the effects of elevated patient aggression across a variety of treatments and including control groups in future treatment studies.

Given the lack of research regarding the effects of aggression on treatment outcomes, this study is among the first to draw attention to the role aggression plays within the context of such patients' presenting treatment characteristics and how they progress in treatment. These results indicate that greater attention should be paid to assessing the extent to which patients presenting with PD also struggle with elevated aggression. Second, these results indicate that our current treatments may not be maximally effective for patients high in aggression. Thus, an assessment of aggression at the start of treatment provides therapists with the opportunity to alter their treatment if necessary, perhaps by adding a treatment target related to decreasing aggressive behaviors. Therapists may be in a better position to maximize the benefits for their patients by incorporating some targeted approaches for aggression. Finally, it is our hope that future research examines the extent to which aggression is a transdiagnostic issue. It is possible that patients with a variety of emotional disorders (e.g., depression, other anxiety disorders) are more susceptible to higher aggression and may not receive the full benefits of their treatment as a result. This possibility should be further

explored, as this patient population may represent a group in need of additional treatment considerations.

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Figure 1. Participant flow through the LTS study. MA indicates major assessment time points.

Table 1

Descriptive Statistics for All Study Variables at All Time Points

	Mean (SD)			
	Baseline	MA2	MA3	MA4
IIP-Aggression	1.06 (.88)	.64 (.66)	.65 (.72)	.53 (.60)
PDSS	14.20 (4.87)	6.73 (4.54)	4.95 (4.52)	3.87 (4.19)
SIGH-A	18.77 (9.84)	11.55 (8.11)	9.31 (7.20)	7.55 (6.53)
SIGH-D	11.85 (7.37)	7.73 (6.17)	6.53 (5.61)	5.40 (5.22)
WSAS	14.94 (8.88)	5.67 (6.37)	4.64 (5.97)	3.39 (4.91)

Note. MA2 assessment occurred post treatment, MA3 occurred at the end of phase 2 and MA4 occurred at the end of the study

Table 2

Correlations between Aggression and Symptom Severity Measures at Baseline

Measure	IIP-Aggression
PDSS	.30**
SIGH-A	.32*
SIGH-D	.32**
WSAS	.35**

Note.

*
p<.05,**
p<.01

Table 3

Unconditional Growth Models

Measure	Estimate	SE	<i>t</i>	<i>df</i>	<i>p</i>
SIGH-A					
Intercept (MA2)	11.35	.45	25.00	255	<.0001
Slope over Time (MA2-MA4)	-.16	.03	-5.65	351	<.0001
PDSS					
Intercept (MA2)	6.51	.26	24.85	255	<.0001
Slope over Time (MA2-MA4)	-.13	.02	-7.89	367	<.0001
SIGH-D					
Intercept (MA2)	7.64	.34	22.18	255	<.0001
Slope over Time (MA2-MA4)	-.10	.02	-4.27	351	<.0001
WSAS					
Intercept (MA2)	5.60	.36	15.51	251	<.0001
Slope over Time (MA2-MA4)	-.08	.02	-3.55	320	.0004

Note. MA2 assessment occurred post-acute treatment. MA2-MA4 is the period following post-acute-treatment. Intercept refers to the estimated score of each measure at MA2. Slope estimates refer to changes in the score of each measure per month during MA2-MA4.

Table 4

Models Examining the Effect of Baseline Aggression on Each Outcome Variable Across the Post-Acute-Treatment Period Controlling for Baseline Severity

Measure	Estimate	SE	t	df	p
SIGH-A					
Intercept	11.52	.40	28.76	253	<.0001
Slope over Time (MA2-MA4)	-.16	.03	-5.93	351	<.0001
SIGH-A (MA1)	.31	.04	7.79	351	<.0001
Aggression (MA1)	1.18	.44	2.67	351	.0079
PDSS					
Intercept	6.61	.25	26.14	253	<.0001
Slope over Time (MA2-MA4)	-.13	.02	-8.17	367	<.0001
PDSS (MA1)	.22	.05	3.96	367	<.0001
Aggression (MA1)	.43	.28	1.53	367	.127
SIGH-D					
Intercept	7.76	.31	25.02	253	<.0001
Slope over Time (MA2-MA4)	-.10	.02	-4.45	351	<.0001
SIGH-D (MA1)	.30	.04	7.35	351	<.0001
Aggression (MA1)	.66	.33	1.96	351	.051
WSAS					
Intercept	5.71	.32	17.94	249	<.0001
Slope over Time (MA2-MA4)	-.09	.02	-3.96	320	<.0001
WSAS (MA1)	.29	.03	8.19	320	<.0001
Aggression (MA1)	.28	.36	0.79	320	.43

Note. MA1 assessment occurred pre-acute treatment. MA2 assessment occurred post-acute treatment. MA2-MA4 is the period following post-acute-treatment. Intercept refers to the score of each measure at MA2, controlling for MA1. A significant intercept value indicates that the score at MA2 significantly differed from zero, controlling for MA1. Slope over Time estimates refer to changes in the score of each measure per month during MA2-MA4, controlling for MA1. Significant Slope over Time estimates indicate that a variable showed significant improvement over MA2-MA4. Each measure at MA1 represents the use of baseline measures to control for initial severity. Significant values for a measure indicates that baseline estimates significantly differed from zero. Aggression (MA1) estimates indicate whether individuals with higher baseline aggression exhibited higher scores on the respective measures during MA2-MA4. Significant p-values for Aggression (MA1) indicate that individuals higher in aggression showed less improvement on the respective measure over MA2-MA4.