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The Impact of Experiential Avoidance on the Reduction of Depression in Treatment for Borderline Personality Disorder

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Abstract

Background—Reducing symptoms of depression is an important target in the treatment of borderline personality disorder (BPD). Although current treatments for BPD are effective in reducing depression, the average post-treatment level of depression remains high.

Aim—To test whether experiential avoidance (EA) impedes the reduction of depression during treatment for BPD.

Method—EA and depression were assessed in 81 clients at baseline and 4-month intervals during one year of therapy. Simple correlations, hierarchical linear modeling, and latent difference score models were used to investigate the association between self-reports of EA and both self-reports and observer-based ratings of depression.

Results—EA was positively associated with greater severity of depression at all points of assessment, and changes in EA were positively associated with changes in depression. Moreover, EA significantly predicted less subsequent reduction in depression whereas no such effect was found for depression on subsequent EA.

Conclusion—The findings are consistent with the hypothesis that EA impedes the reduction of depression in the treatment of BPD and should thus be considered an important treatment target.

Keywords

borderline personality disorder; experiential avoidance; depression; treatment; outcome

Strong evidence suggests that depression is a common experience among individuals meeting criteria for borderline personality disorder (BPD). For example, in carefully controlled studies,

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comorbidity rates between BPD and current mood disorder range between 31–61% for major depressive disorder (MDD) and between 12–24% for dysthymia (Comtois, Cowley, Dunner, & Roy-Byrne, 1999; Skodol et al., 1999; Zimmerman & Mattia, 1999). Moreover, up to 83% of individuals suffering from BPD report a history of MDD, up to 39% report a history of dysthymia (Zanarini et al., 1998), and 37% meet criteria for depressive personality disorder (Grilo, Sanislow, & McGlashan, 2002). Additionally, individuals meeting criteria for both BPD and MDD exhibit greater severity of depression in self-reports than individuals with MDD but without BPD (e.g., Abela, Payne, & Moussaly, 2006; Stanley & Wilson, 2006). Finally, individuals with BPD often suffer from depressive symptoms even when they do not meet full criteria for any affective disorder. For example, chronic dysphoric mood states, negative self-evaluations, and feelings of hopelessness and helplessness are commonly found in individuals suffering from BPD (Gunderson & Phillips, 1991; Hooley, 2007; Trull, 2001; Zittel-Conklin & Westen, 2005). These findings imply that reducing the suffering associated with depressive symptoms is an important way of reducing the suffering of BPD individuals.

Fortunately, the results of major outcome-studies of the past decade indicate that today's treatments for BPD are effective in reducing depression (Bateman & Fonagy, 1999; Bohus et al., 2004; Bohus et al., 2000; Brown, Newman, Charlesworth, Crits-Christoph, & Beck, 2004; Koons et al., 2001; Kröger et al., 2006; Linehan et al., 2006; Turner, 2000). Pre-post effect sizes (Cohen's d) in these studies range from 0.54 to 2.1 (*Mdn* = 1.1) for the Beck Depression Inventory (BDI), and from 0.17 to 2.55 (*Mdn* = 0.93) for the Hamilton Rating Scale for Depression (HRSD). However, these studies also demonstrate a significant post-treatment level of depression, with mean values ranging between *M* = 13.4 to 25.1 for the BDI (*Mdn* = 20.9) and *M* = 7.5 to 19.1 for the HRSD (*Mdn* = 14.0). In fact, most of these studies report post-treatment mean depression scores corresponding to moderate or even severe levels of depression. Thus, even after carefully conducted state-of-the-art BPD treatments, a great amount of depression-related suffering remains in these patients. Considering that residual symptoms of depression are known to be important predictors of relapse after treatment for unipolar depression (Judd et al., 1998) and that residual depressive symptoms are likely to trigger more typical symptoms of BPD (which have been conceptualized as dysfunctional attempts to avoid aversive inner experiences; Linehan, 1993a), it can be concluded that there is a considerable need to identify factors impeding the reduction of depression during treatment for BPD.

Evidence-based maintaining factors for depression include: a depressogenic attributional style (Abramson, Metalsky, & Alloy, 1989), hopelessness (Beck, Weissman, Lester, & Trexler, 1974), low self-esteem (Brown & Harris, 1978), dysfunctional attitudes (Beck, 1967) and rumination (Nolen-Hoeksema, 1991). All of these factors are significantly associated with BPD (Abela, Payne, & Moussaly, 2006). Another concept that is currently discussed as a putative risk-factor for the development and maintenance of depression is the general tendency to react towards aversive experiences with avoidance-oriented response patterns (e.g., Ottenbreit & Dobson, 2004; Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005). These response tendencies are thought to lead to: loss of reinforcement (Ferster, 1973; Jacobson, Martell, & Dimidjian, 2001), rumination (Cribb, Moulds, & Carter, 2006), impaired emotional processing (Borkovec, Ray, & Stoeber, 1998), increased negative cognitions (Abramowitz, Tolin, & Street, 2001; Wegner & Zanakos, 1994; Wenzlaff & Bates, 1998) and emotions (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Eifert & Heffner, 2003; Levitt, Brown, Orsillo, & Barlow, 2004; Feldner, Zvolensky, Stickle, Bonn-Miller, & Leen-Feldner, 2006; Feldner, Zvolensky, Eifert, & Spira; 2003), and consequently to depression.

Hayes and colleagues (1996) have proposed the term of *experiential avoidance* (EA) to summarize a broad range of potentially problematic behaviors that individuals apply in order to avoid aversive experiences. According to Hayes and colleagues (2004, p. 554), EA is "a

phenomenon that occurs when a person is unwilling to remain in contact with particular private experiences (e.g., bodily sensations, emotions, thoughts, memories, images, behavioral predispositions) and takes steps to alter the form or frequency of these experiences or the contexts that occasion them, even when these forms of avoidance cause behavioral harm." In order to measure EA, Hayes and colleagues (2004) developed the *Acceptance and Actions Questionnaire* (AAQ), a self-report measure that assesses constructs considered as important indicators of EA. The total score of the AAQ has been demonstrated to be strongly associated with self-report measures of depression (for a review see Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Moreover, two longitudinal studies in non-clinical samples provide further preliminary support for the assumed causal effect of EA on depression (Bond & Bunce, 2003; Kashdan, Barrios, Forsyth, & Steger, 2006). However, due to the striking lack of more rigorous tests of causal effects in clinical populations, it is unclear whether EA is a cause or merely a consequence of depression.

Several findings suggest that EA might be particularly important for the maintenance of depression in BPD: First, BPD individuals report more frequently avoidance-oriented response patterns in coping inventories (Bijttebier & Vertommen, 1999; Kruedelbach, McCormick, Schulz, & Gruenreich, 1993; Vollrath, Alneas, & Torgensen, 1998) and in the AAQ (Rüsch et al., 2006) than do normal controls or patients suffering from social phobia, respectively. Second, both symptoms of BPD and symptoms of other mental disorders often co-occuring with BPD (such as posttraumatic stress disorder; Zanarini et al., 1998) are associated with EA (Chapman, Specht, & Cellucci, 2005; Marx & Sloan, 2005; Chapman, Gratz, & Brown, 2006). Third, the tendency to suppress negative thoughts was shown to moderate the effect of negative affect on borderline characteristics (Rosenthal, Cheavens, Lejuez, & Lynch, 2005). However, at this point no study has explicitly investigated the association between EA and depression in individuals treated for BPD.

Given that a) the reduction of depression is an important target in the treatment for BPD, b) there is a need to improve the effects of BPD-treatments on depression, and c) EA has repeatedly been demonstrated to be associated both with depression and BPD, the aim of this study is to clarify whether EA impedes the reduction of depression during treatment for BPD by testing the following hypotheses:

1. EA is significantly associated with higher levels of depression before, during and after treatment for BPD.
2. The reduction of EA during treatment for BPD is significantly associated with a greater reduction of depression.
3. The level of EA predicts subsequent reduction of depression during treatment for BPD, whereas the level of depression does not predict subsequent changes in EA.

Methods

Participants

Levels of EA and depression were assessed in 81 female outpatients during one year of treatment for BPD. Assessment took place at baseline and at 4-month intervals during the treatment. All subjects were participants in a randomized controlled trial in which dialectical behavioral therapy (DBT; Linehan 1993a, 1993b) was compared to treatment by community experts (TBE; Linehan et al., 2006). Of the 101 participants in the intent-to treat sample of this study, the current study included the 81 individuals who completed the Acceptance and Action Questionnaire at least once during the first year of treatment[2]. Participants were diagnosed with BPD based on both the International Personality Disorders Examination (IPDE; Loranger, 1995) and the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-

II; First, Spitzer, Gibbons, Williams, & Benjamin, 1996). Participants were enrolled if they had a recent and chronic history of non-suicidal self-injury or suicide attempts (i.e. 2 or more episodes in the previous 5 years, with one episode occurring in the previous 8 weeks). All participants gave informed consent prior to participation in the studies, and all procedures were approved by the University of Washington review board. Participants were between 18 and 44 years of age (M = 28.90, SD = 7.47); 75.6% earned below $15,000 per year; 40.7% had some college education; 14.8% were college graduates. The majority of the sample (86.4%) was Caucasian; 61.7% had never been married, and 14.8% were currently married. Most participants (95%) qualified for other Axis I disorders: Approximately 50% had two or three other Axis I diagnoses, with the most common being anxiety disorders (80.3%), major depressive disorder (77.8%) and substance abuse disorder (25.9%); about one third (32.8%) were diagnosed with an Axis II disorder other than BPD. Participants had between 0 and 151 lifetime suicide attempts (Mdn = 3, Q_1 = 1, Q_3 = 7) and between 2 and 301 lifetime episodes of self-injury (Mdn = 26, Q_1 = 11; Q_3 = 68). All participants in the study received psychotherapeutic treatment for BPD. About half of the participants (N = 43) were treated with DBT. The other 38 participants were treated by non-behavioral therapists who had been nominated as experts in the treatment of BPD by community mental health leaders. Both types of treatment, as well as the randomization and assessment procedures, have been described in detail by Linehan and colleagues (2006).

Measures

EA was assessed as the total score of the Acceptance and Action Questionnaire (AAQ; Hayes et al., 2004). The AAQ is a self-report measure that assesses different aspects of EA utilizing a 7-point Likert-type scale. Higher scores on the AAQ indicate more avoidance, while lower scores indicate more acceptance. We used the sixteen-item version of the AAQ (Hayes et al., 2004), as recommended by Hayes at the time the study was conducted. This version contains seven items that are part of the nine-item version that has lately been recommended for use, and it correlates at a .89 level with the new nine-item version (Hayes et al., 2004). The sixteen-item version has been applied in several studies and has shown acceptable internal consistency and convergent validity with measures of depression, dissociation and avoidance (e.g., Roemer, Salters, Raffa, & Orsillo, 2005). In the present study, cronbach's α for the AAQ was .83 (baseline).

Depression was assessed with the modified Hamilton Rating Scale for Depression (HRSD; Miller, Bishop, Norman, & Maddever, 1985) and the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The original HRSD (Hamilton, 1960) is an interviewer-rated scale which has been widely used in clinical trials of cognitive behavioral and psychopharmacological treatments for depression. We used the modified 25-item version in this study, because of its superior validity and reliability (Miller et al., 1985; Bagby, Ryder, Schuller, & Marshall, 2004). The BDI is a 21-item self-report measure of depression. Depressive affect is measured based on numerical ratings of somatic, behavioral, emotional, and cognitive signs of depression. The BDI is a commonly used measure with good reliability and validity (e.g., Beck & Steer, 1984). In the present study, cronbach's α was .80 for the HRSD and .83 for the BDI (baseline).

Analytic Strategies

In order to test Hypothesis 1 and determine the strength of the cross-sectional association between the AAQ and both indicators of depression, we computed Pearson's correlations

[2]Since the AAQ was not yet available when the Linehan et al. (2006) study began in 1994, a significant number of missing values (36, 27, 23, and 13 of the N=101 intent-to-treat sample at t1, t2, t3, and t4, respectively) was due to the AAQ not being part of the assessment battery.

separately for all four points of measurement. In order to test Hypothesis 2, we used the multidimensional extension of the generalized linear mixed effects model proposed by MacCallum, Kim, Malarkey, and Kiecolt-Glaser (1997) to model individual (intercepts and) slopes and determine how strongly slopes of the AAQ are associated with slopes of the HRSD and the BDI, respectively. Models were run separately for HRSD and BDI. Model fit was evaluated using diagnostics discussed by Jiang (2001). Diagnostics include assessment of residuals, outliners, and influential observations. In order to test Hypothesis 3, we used latent difference score models (LDS), a structural equation method that tests time lagged effects between two variables while simultaneously controlling for the autoregression of these variables. This method was developed by McArdle and colleagues (McArdle, 2001, 2009; McArdle & Hamagami, 2001) and is increasingly applied to investigate causal relationships in social and clinical psychology (e.g., Ferrer & McArdle, 2004; Hawley, Ho, Zuroff, & Blatt, 2006; Lövdén, Ghisletta, & Lindenberger, 2005). Figure 1 shows the structure of the two models (one for each indicator of depression) used in the LDS analyses.

LDS models are based on the repeated assessments of indicators across time, with the models accounting for the covariance structure as well as for the mean structure. The empirical indicators (denoted as $AAQ_t1–4$ and $ID_t1–4$, respectively, in Figure 1) are explained by latent true score variables ($EAt_{1–4}$ and $Dt_{1–4}$) and uniqueness terms. The true score variables (except for Time 1) are explained by the true scores on the preceding assessment and by latent difference score variables ($\Delta EA_{1–3}$ and $\Delta D_{1–3}$). The model accounts for several different types of change. First, as in standard growth curve analyses, the model includes a latent slope variable (accounting for constant change in the variables across time). The slope variable has a constant effect on all difference scores; for identification purposes, the parameter must be fixed to 1 (McArdle, 2001). Given that the LDS model also includes latent intercepts and covariances between all slopes and intercepts, all parts of growth curve models are represented in the LDS model. In addition to growth curve models, LDS models also account for a second type of change, i.e., proportional change, represented by effects from the true score of one assessment on the difference score of the next assessment (coefficients β_{EA} and β_D in Figure 1). Finally, the bivariate LDS model includes *coupling*, or *cross-lagged* effects between the true scores of one variable and the latent difference scores of the other variable (coefficients γ_{EA} and γ_D in Figure 1). The cross-lagged paths indicate whether intraindividual change in one variable is explained by the true score of the other variable measured on the preceding assessment, controlling for constant and proportional change. These paths constitute the relevant statistics for Hypothesis 3. For all specifications of the model, we followed the suggestions of McArdle (2001) and McArdle and Hamagami (2001). Of note is that in LDS models, standardized path coefficients are not applicable, and therefore we report the unstandardized coefficients and their significance levels.

Structural equation modeling was conducted using Amos 5 (Arbuckle, 2003; Arbuckle & Wothke, 1999). The Full-Information Maximum Likelihood (FIML) procedure included in Amos was applied for handling missing values. Methods of missing value imputation are recommended because the results are less biased and are more reliable when compared to conventional methods such as listwise or pairwise deletion (Allison, 2003; Schafer & Graham, 2002). For the mixed effects analyses we used SAS 9.1.3, and for all other computations SPSS 14.00.

Evaluation of LDS-model fit was based on current recommendations (Hu & Bentler, 1998, 1999; MacCallum & Austin, 2000). The three fit indices we used were: the Tucker-Lewis-Index (TLI), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). Hu and Bentler (1998, 1999) suggested that good fit is indicated by values greater than or equal to .95 for TLI and CFI, and less than or equal to .06 for RMSEA. In addition to these indices, we reported χ^2-statistics and the confidence interval for RMSEA.

Results

Preliminary analyses indicated that all necessary statistical assumptions were met. As can be seen in Table 1, sample sizes vary between instruments and time points, with a range of 62 to 81 (*Mdn* = 70.5) for the relevant combinations of assessment instruments.

Mean values of the AAQ, the HRSD, and the BDI decreased over time. This indicates that patients became less avoidant and less depressed during treatment.

Cross-Sectional Associations and Associations between Slopes

The AAQ was significantly associated with both the HRSD and the BDI on a 1%-alpha level (one-way test) at all four points of assessment; with r_{t1} = .44, r_{t2} = .62, r_{t3} = .61, r_{t4} = .66 for the HRSD, and r_{t1} = .34, r_{t2} = .63, r_{t3} = .63, and r_{t4} = .67 for the BDI. Thus, the results confirm Hypothesis 1. Correlations were slightly higher within the TBE condition than in the DBT condition (HRSD: Mdn_{DBT} = .52, Mdn_{TBE} = .68, BDI: Mdn_{DBT} = .57, Mdn_{TBE} = .69).

As a first-step analysis of the relationship of changes in the AAQ and changes in indicators of depression, we modeled the linear individual change over time for each combination of the AAQ and one indicator of depression (MacCallum et al., 1997). According to the diagnostics discussed by Jiang (2001), there appeared to be no significant issues with respect to fit.

As can be seen in Table 2, there is a significant covariance between the individual slopes of the AAQ and HRSD and between the slopes of the AAQ and BDI. This covariance corresponds to a strong correlation between both the AAQ and HRSD and the AAQ and BDI (with *r* = .85 for both measures). In sum, the results confirm Hypothesis 2: A reduction in the AAQ total score is strongly associated with a reduction in both indicators of depression. This effect was somewhat stronger in the TBE than in the DBT condition (HRSD-AAQ_{DBT}: *r* = .70, HRSD-AAQ_{TBE}: *r* = .95, BDI-AAQ_{DBT}: *r* = .71, BDI-AAQ_{TBE}: *r* = .96).

Additional mediation analyses for clustered data (Krull & MacKinnon, 2001; Bauer, Preacher, & Gil, 2006) and two active treatment conditions (Doss & Atkins, 2006) revealed a significant indirect effect (IE = $a_{time\text{-}AAQ}$ x $b_{AAQ\text{-}depression}$) of the time spent in treatment on indicators of depression via changes in the AAQ (HRSD: IE = −1.03, 95%*CI* = −1.54 - −0.53; BDI: IE = −1.26, 95%*CI* = −1.84 - −0.68). When these analyses were broken down by treatment, indirect effects were significant only for the DBT condition (DBT-HRSD: IE = −1.6, 95%*CI* = −2.37 - −0.83; DBT-BDI: IE = −1.53, 95%*CI* = −2.32 - −0.73; TBE-HRSD: IE = −0.45, 95%*CI* = −1.12 - 0.23; TBE-BDI: IE = −0.79, 95%*CI* = −1.69 - 0.10).

Latent Difference Score Analyses

Fit was good for HRSD-AAQ model, with χ^2 = 29.05, *df* = 23, *p* = .18; TLI = .97, CFI = .98, RMSEA = .051 and 90%-*CI* of RMSEA = .000 - .102. Fit for the BDI-AAQ model was still satisfactory, with χ^2 = 38.8, *df* = 23, $p < 0.05$; TLI = .92, CFI = .95, RMSEA = .083 and 90%-*CI* of RMSEA = .032 - .127. The crucial structural parameters of the LDS models are reported in Table 3.

The proportional effects (coefficients β_{EA} and β_{D})were significant for both indicators of depression but not for the AAQ. Thus, over and above the constant change explained by the growth curve part of the LDS model, there is evidence for proportional change in both indicators of depression but no evidence for proportional change in the AAQ. This indicates that the severity of depression is positively associated with the subsequent reduction of depression whereas the level of EA is not associated with subsequent changes in EA.

The cross-lagged effects of EA and depression (coefficients γ_{EA} and γ_D in Figure 1) are the relevant statistics for testing Hypothesis 3. The results demonstrate that the AAQ is significantly associated with subsequent changes in both the HRSD and the BDI. Contrastingly, neither the HRSD nor the BDI scores are significantly associated with subsequent changes in the AAQ. Thus, the results confirm Hypothesis 3.

Discussion

The goal of this study was to clarify whether EA impedes the reduction of depression during treatment for BPD. In a four-wave panel data set from a sample of 81 females treated for BPD over a one-year period, EA was significantly correlated with greater depression scores at all four points of assessment. These results replicate previous findings by demonstrating that EA is cross-sectionally associated with depression. Moreover, the present study showed that the *reduction* of EA during treatment for BPD was significantly associated with the *reduction* of depression. This finding is an important contribution to the literature, as it provides more direct evidence that reducing EA may be important goal in BPD treatment. However, these results do not yet clarify whether EA is an antecedent or a consequence of depression. Therefore, we consider the results of the LDS analyses as a particularly noteworthy extension of previous findings. The finding that levels of EA were associated with less *subsequent* reduction of depression, whereas no such effect was found for depression on subsequent EA, provides unprecedented support for the hypotheses that a) EA impedes the reduction of depression and that b) EA is not merely a consequence of depression.

As already mentioned in the introduction, such an effect may results from various processes. For example, a high level of EA may prevent clients from: a) engaging in a supportive relationship with the therapist (Horvath & Bedi, 2002; Lynch et al., 2006), b) “processing” important information and experiences (Greenberg & Pascual-Leone, 2006), c) habituating to phobic stimuli (Mowrer, 1960), d) engaging in behavioral experiments necessary to invalidate dysfunctional attitudes (Beck, 1995), e) engaging into activities that will (eventually) lead to an increase in reinforcement (Ferster, 1973), and f) refraining from rigid attempts to suppress negative cognitions/emotions, which may have paradoxical effects of enhancing the accessibility of these thoughts (e.g., Abramowitz, Tolin, & Street, 2001) and/or the intensity of these emotions (e.g., Berking, Orth et al., 2008; Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Levitt, Brown, Orsillo, & Barlow, 2004). Identifying which of these specific effects of EA is particularly important for impeding the treatment of depression in BPD is certainly an important question for further research. However, it can also be argued that while the specific consequences of EA may differ between patients, there is a high possibility that EA will have at least some of these adverse effects, and that EA should therefore be targeted in all clients with strong tendencies to avoid aversive inner experiences.

Thus, the results of this study have important implications for clinical practice. They identify EA as a promising target for interventions aimed at the reduction of depression in BPD individuals. It can be assumed that today’s treatments often already address proximate antecedents of EA. For example, DBT teaches the skills of *distress-tolerance*, *mindfulness*, *opposite action*, and *acceptance* (Linehan, 1993a, 1993b). All these interventions target EA: The application of *distress-tolerance skills* is supposed to help clients tolerate aversive inner states without engaging in dysfunctional avoidance behaviors; exercising *mindfulness-skills* helps replace avoidance behavior with a focused, non-judgmental perception of the sensations associated with aversive experiences; practicing *opposite action* helps patients actively engage in behavior that contrasts the (avoidance-oriented) action tendencies of the emotion they are feeling; and *acceptance skills* are supposed to “turn the mind” from fighting against reality to “willingly” accepting it (Linehan, 1993a; p. 148). Unfortunately, at this point there is almost no data available on the effectiveness of specific “anti-EA” interventions employed in DBT or

other treatments addressing EA (e.g., Berking, Wupperman et al., 2008; Gratz & Gunderson, 2006; Hayes et al., 2006). Thus, future research needs to evaluate existing interventions with regard to their effectiveness in reducing EA in BPD. Depending on the results, new and more-effective strategies that focus on EA may need to be developed and integrated into BPD-treatments. In order to facilitate these efforts, future research should also focus on identifying proximal antecedents of EA.

Although this study provides striking evidence for the importance of EA, it also indicates that there are limits to the relevance of this concept. The finding that EA (partially) mediates the effects of treatment on depression in the DBT but not in the TBE condition, suggests that there may be other ways to reduce depression in BPD. Unfortunately, the present study was not able to address other potential antecedents of depression in BPD. Therefore, it is important that future studies simultaneously assess both EA and other potentially relevant factors (e.g., Abela, Payne, & Moussaly, 2006) and investigate the differential and incremental validity of EA beyond these other factors.

Further limitations of this study include a) the use of an older version of the AAQ; b) the assessment of EA via a self-report instrument that subsumes possibly independent concepts (such as “excessively negative evaluations of private experiences or negative self-references”, Hayes et al., 2004, p. 556) which may greatly overlap with depression under the concept of EA; c) the application of an SEM-based approach with a sample size smaller than recommended by some authors (e.g., Anderson & Gerbing, 1988) and with a relatively high rate of missing values; d) the high comorbidity of anxiety (and other) disorders, which are closely associated with EA in the present sample; and e) the lack of treatment-specific LDS analyses.

However, the version of the AAQ used in this study is highly correlated with the current version of the AAQ (Hayes et al., 2004), and significant effects were found even when a possible overlap between the measures for EA and depression was controlled (via controlling for autoregression effects in the LDS models). Moreover, some authors consider sample sizes of $N > 75$ as sufficient for SEM depending on the number of latent and manifest variables in the model (e.g., Bollen & Long, 1993; Geweke & Singleton, 1980). In addition, the reliability of the LDS-based analyses is enhanced by the use of structural constraints derived from plausible prepositions, multiple points of assessment, and application of full information maximum likelihood (FIML) procedure to deal with missing data. The sufficient/good model fit and the convergent findings for different measures of depression are in line with this assumption. Finally, a high comorbidity of anxiety (and other) disorders is typical for BPD individuals; excluding patients also meeting criteria for anxiety (or other) disorders would greatly endanger the external validity of the study.

Nevertheless, future studies need to replicate these findings with other indicators of generalized avoidance tendencies (e.g., Berking & Znoj, 2008; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006; Gross & John, 2003; Ottenbreit & Dobson, 2004), with larger sample sizes, and with fewer missing values. In combination with a systematic variation in treatment, the latter would allow for extending the treatment-specific analyses to the LDS approach. Additionally, these studies should address possible moderating effects of comorbid (anxiety) disorders, clarify whether EA is important for treatment outcomes other than depression, and investigate whether the results of this study can be generalized to other BPD-populations, such as men and/or non-suicidal women.

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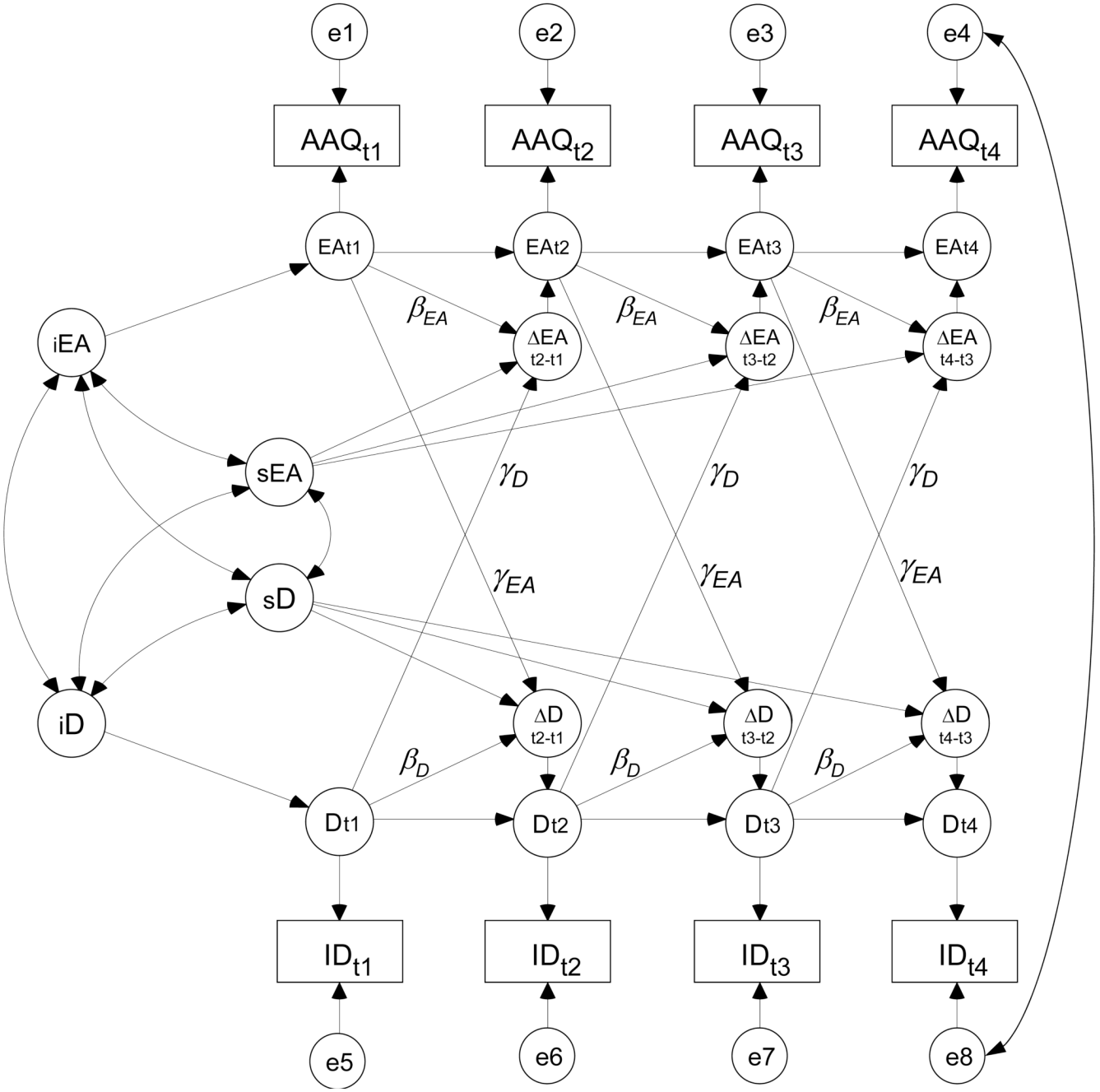

Figure 1.

Model illustrating the bivariate latent difference score (LDS) analysis. For purposes of clarity, cross-sectional correlations of uniqueness terms are only shown for Time 4, but are also included in the models for Time 1 to Time 3. For further details on model specifications, see McArdle (2001) and McArdle & Hamagami (2001). AE = Experiential Avoidance; D = Depression; d = difference; i = intercept; s = slope; e = error; ID = Indicator of Depression; β_{EA}= Experiential Avoidance proportional effects, β_D= Depression proportional effect, γ_{EA} = Experiential Avoidance ➔$_{\Delta}$Depression cross-lagged effects, and γ_D= Depression ➔$_{\Delta}$Experiential Avoidance cross-lagged effects; $_{\Delta}$ = difference between two subsequent points of assessment.

Table 1

Means, Standard Deviations and Missing Values

Measure	Time 1			Time 2			Time 3			Time 4		
	M	*SD*	*N*	*M*	*SD*	*N*	*M*	*SD*	*N*	*M*	*SD*	*N*
AAQ	4.97	0.70	65	4.80	0.73	66	4.69	0.67	62	4.40	0.82	72
HRSD	33.20	9.24	76	28.46	10.52	72	25.18	11.24	67	22.44	11.44	66
BDI	34.07	9.24	81	25.87	11.68	71	21.27	11.92	70	19.58	12.56	71

Note. AAQ = Acceptance & Action Questionnaire; HRSD = Hamilton Rating Scale for Depression; BDI = Beck Depression Inventory.

Table 2

Estimated Intercepts and Slopes, and Associations between Slopes

Model	Label	*Estimate*	*SE*	*DF*	*t/z*
AAQ-HRSD	HRSD-Intercept	35.67	1.06	387	33.50***
	AAQ-Intercept	5.14	0.08	387	65.25***
	HRSD-Slope	−3.21	0.53	75	−6.06***
	AAQ-Slope	−0.17	0.04	80	−4.70***
	Covariance of the Random[a]	0.51 (0.85)	0.11	N/A	4.68***
AAQ-BDI	BDI-Intercept	37.02	1.14	394	32.48***
	AAQ-Intercept	5.15	0.08	394	65.43***
	BDI-Slope	−4.63	0.56	80	−8.31***
	AAQ-Slope	−0.17	0.04	80	−4.71***
	Covariance of the Random[a]	0.54 (0.85)	0.11	N/A	4.66***

Note.

*** $p < .001$.

z = test statistic for Covariance of the Random, t = test statistic for all other effects.

N/A = Non-applicable for z-statistics (Standard normal).

[a] correlation in parentheses

Table 3

Bivariate LDS Analyses: Estimates of Coefficients

Parameter	Model 1. HRSD	Model 2. BDI
Regression Coefficients		
EA→ $_{\Delta}$EA proportional effects (β*EA*)	0.115 (0.381)	−0.092 (0.320)
Depression→ $_{\Delta}$Depression proportional effects (β*D*)	−0.958 (0.25)***	−0.727 (0.164)***
EA→ $_{\Delta}$Depression crossed-lagged effects (γ*EA*)	12.858 (5.357)*	9.568 (4.745)*
Depression→ $_{\Delta}$EA crossed-lagged effects (γ*D*)	−0.002 (0.018)	0.011 (0.011)
Means		
EA Intercept	4.911 (0.082)***	4.953 (0.085)***
Depression Intercept	32.655 (0.966)***	33.798 (0.976)***
EA Slope	−0.660 (1.448)	−0.05 (1.325)
Depression Slope	−37.265 (20.399)(*)	−31.148 (19.627)(*)
Variances		
EA Intercept	0.336 (0.081)**	0.38 (0.089)***
Depression Intercept	44.934 (13.815)***	48.246 (13.86)***
EA Slope	0.049 (0.031)	0.044 (0.015)**
Depression Slope	51.695 (25.974)*	36.16 (15.726)*
EA Error	0.177 (0.023)***	0.167 (0.021)***
Depression Error	44.363 (5.647)***	47.991 (5.668)***

Note. Numbers in parentheses represent standard errors of estimates.

Coefficients β*EA* β*D* γ*EA*, and γ*D* as denoted in Figure 1; EA = Experiential Avoidance; Δ = difference between two subsequent points of assessment.

(*) $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.