

A Longitudinal Study of Experiential Avoidance in Emotional Disorders

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The aim of this study was to examine the degree in which measurements of trait experiential avoidance (EA) are affected by current emotional disorder and whether EA is a causal factor in the course of emotional disorders (anxiety and depressive disorders) and the development of comorbidity among emotional disorders. In a sample of 2,316 adults aged 18 to 65, consisting of healthy controls, persons with a prior history of emotional disorders, and persons with a current emotional disorder, DSM-IV-based emotional disorders (CIDI: Composite Interview Diagnostic Instrument) were assessed at T2 and 2 (T4) and 4 years later (T6)

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and experiential avoidance (AAQ: Acceptance and Action Questionnaire) at T2 and T4. Results showed that EA scores were stable over a 2-year period notwithstanding state fluctuations because of current emotional disorder. Moreover, EA scores at T2 predicted changes in distress (major depressive disorder, dysthymia, generalized anxiety disorder) and in fear disorders (social anxiety disorder, panic disorder with or without agoraphobia, agoraphobia without panic) at T4. Finally, EA at T4 mediated the longitudinal association of fear disorders at T2 with distress disorders at T6 as well as of distress disorders at T2 with fear disorders at T6. These findings suggest that EA scores are more than epiphenomena of emotional disorders and that EA may be conceptualized as a relevant transdiagnostic factor affecting the course and development of comorbidity of emotional disorders.

Keywords: experiential avoidance; longitudinal study; anxiety; depression; comorbidity

EXPERIENTIAL AVOIDANCE (EA) IS described as consisting of two related parts: (a) the unwillingness to remain in contact with aversive private experience (including bodily sensations, emotions, thoughts, memories, and behavioral predispositions), and

(b) action taken to alter the aversive experiences or the events that elicit them (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). EA has been hypothesized to play an important role in the etiology, maintenance, and modification of various forms of psychopathology (Hayes et al., 2004), anxiety and depression in particular (for reviews, see Chawla & Ostafin, 2007; Hayes et al., 1996). On the basis of these results EA is considered to constitute a broad transdiagnostic risk factor as it leads to multiple disorders (Baer, 2007; Barlow, Allen, & Choate, 2004; Harvey, Watkins, Mansell, & Shafran, 2004). In support of this presupposition, available evidence indicates that EA is associated with other broad risk factors for psychopathology such as negative emotionality/neuroticism (Boelen & Reijntjes, 2008; Bond & Bunce, 2003; Hayes et al., 2004; Kashdan, Barrios, Forsyth, & Steger, 2006).

Reviewing the available research literature on EA (Chawla & Ostafin, 2007), a striking finding is that most of the studies are correlational or between-group comparisons. These research designs preclude more definitive conclusions about the causal role of EA in the onset or maintenance of emotional disorders or the causal influence of emotional disorders on EA itself. Several models for causal relations of personality traits with emotional disorders have been proposed (Klein, Kotov, & Bufferd, 2011) that may provide a useful framework for approaching the relation of EA with emotional disorders. In one group of models (common cause, continuum/spectrum, and precursor models) personality and emotional disorders are viewed as having similar causes but without mutually influencing each other. A second group of models (predisposition and pathoplasticity models) poses that personality has causal effects on the onset or maintenance of emotional disorders. The third and last group of models (concomitants and consequences models) states that emotional disorders have a causal influence on personality.

Only a few longitudinal studies on EA are available, suggesting that EA may have a causal effect on the onset and maintenance of emotional disorders. In accordance with a predisposition model, EA predicted depressive symptoms in the face of high stress in a group of community females at risk for developing depression (Shallcross, Troy, Boland, & Mauss, 2010) and PTSD symptoms 4 and 8 weeks after trauma exposure after controlling for initial levels of PTSD symptomatology (Marx & Sloan, 2005). Moreover, in accordance with a pathoplasticity model, a treatment outcome study in patients with borderline personality disorder showed that reductions in EA were predictive of subsequent improvements in depressive symptoms, suggesting that EA is not only a consequence of

depressive experiences (Berking, Neacsiu, Comtois, & Linehan, 2009). Finally, Shahar and Herr (2011) investigated how the daily relationship between state EA and negative affect varied as a function of baseline depressive symptoms using a daily diary design in a sample of students. They found that the 1-day lagged association between negative affect and state EA did not vary as a function of baseline depression, although participants with more depressive symptoms used more daily EA overall. These few studies indicate that there is a need for prospective studies to elucidate the causal role of EA in the development and course of emotional disorders and concomitant psychopathology.

The present study is based on data of the Netherlands Study of Depression and Anxiety (NESDA; Penninx et al., 2008). NESDA is an ongoing cohort study designed to investigate determinants, course, and consequences of prevalent depressive and anxiety disorders. Consequently, the term *emotional disorders* in the present study refers to anxiety and depressive disorders only. On the basis of this study it is possible to test a concomitants and pathoplasticity perspective on EA. According to the concomitants (or state-dependent) model, assessments of EA will be colored, or distorted, by the individual's emotional state. This model implies that EA scores will be enhanced after the occurrence of a disorder and will be reduced after remittance. Extrapolating from the literature on state effects of emotional disorder on the measurement of personality traits as neuroticism and extraversion (Karsten et al., 2012; Spinhoven, van der Does, Ormel, Zitman, & Penninx, 2013), it is to be expected that levels of EA will be relatively stable over time, although enhanced during disorder occurrence and reduced following disorder remittance.

According to a pathoplasticity model, EA may influence the expression of the disorder after onset. This influence may include several aspects, such as the predictive value of EA for the course of a particular disorder. However, EA is considered to be a transdiagnostic factor and therefore should also contribute to the development of comorbidity among disorders. This aspect of the pathoplasticity model would be supported by evidence that one disorder predicts a subsequent disorder and that EA mediates this longitudinal relation. This type of evidence has been gathered, for example, by McLaughlin and Nolen-Hoeksema (2011) for rumination as a transdiagnostic factor by showing that baseline depressive symptoms predicted subsequent increases in anxiety symptoms and vice versa and that rumination mediated both longitudinal associations.

In sum, the present study had the following three aims and examined: (a) the degree to which EA measurements are affected by current emotional disorder; (b) whether EA is a possible causal factor in the course of emotional disorders; (c) the extent to which EA predicts the co-occurrence of emotional disorders. We expected that EA as a true transdiagnostic factor would remain stable over time notwithstanding state fluctuations because of current emotional disorder and would prospectively predict both course and co-occurrence of emotional disorders.

Materials and Methods

SAMPLE

The NESDA sample of 2,981 adults (18–65 years) includes participants with a lifetime and/or current anxiety and/or depressive disorder ($n = 2,329$; 78%) and healthy controls (persons without depressive or anxiety disorders; $n = 652$; 22%). To include various developmental stages of disorders and different levels of severity, participants were recruited from general practices ($n = 1,610$; 54%), mental health organizations ($n = 807$; 27%), and the general population ($n = 564$; 19%). Community-based subjects with depressive or anxiety disorders were previously identified in two population-based studies: *Nemesis* (Bijl et al., 1998) and *Ariadne* (Landman-Peeters et al., 2005). Primary care patients were identified through a 3-stage screening procedure (involving the K10 and the CIDI short form) among patients of 65 General Practitioners consulting for any reason in a 4-month period. In secondary care, patients were recruited when newly enrolled for a depressive or anxiety disorder at one of the 17 participating mental health organization locations. General exclusion criteria were a primary diagnosis of psychotic, obsessive-compulsive, bipolar, or severe addiction disorder and not being fluent in Dutch.

As the Acceptance and Action Questionnaire was measured for the first time at T2, T1 assessments are not further analyzed in the present study. Of the 2,596 persons at T2, 2,316 (89.2%) completed the AAQ, constituting the present study sample. Mean age at T2 was 42.2 years ($SD = 13.1$), mean number of years of education was 12.4 years ($SD = 3.3$), 66.8% was female, 42% was married, and 97% had a Dutch nationality. At T2 1,488 persons (64.2%) had no current 6-month recent depressive and/or anxiety disorder. Psychiatric diagnoses among the remaining 828 disordered participants were as follows: dysthymia (DYS) = 200 (24.2%); major depressive disorder (MDD) = 486 (58.7%); generalized anxiety disorder (GAD) = 171 (20.7%); social anxiety disorder (SAD) = 302 (36.5%); panic disorder

with or without agoraphobia (PD) = 238 (28.7%); and agoraphobia without panic (AGO) = 122 (14.7%). Comorbidity among disorders was high: of the 828 participants with a disorder, 421 participants (50.8%) had a single disorder; 206 (24.9%) two disorder; 135 (16.3%) three disorders; and 66 (8.0%) four or more disorders. Subsuming individual disorders under the broader categories of distress (DYS, MDD, GAD) and fear (SAD, PD, AGO) disorders yielded the following prevalence and comorbidity rates: 578 of the affected persons (69.8%) had a distress disorder and 533 (64.4%) a fear disorder. Of the persons with a distress disorder 283 (49.0%) had a fear disorder and of the persons with a fear disorder 53.1% had a distress disorder.

PROCEDURE

A detailed description of the NESDA design and sampling procedures has been given elsewhere (Penninx et al., 2008). The baseline assessment included assessment of demographic and personal characteristics, a standardized diagnostic psychiatric interview and a medical assessment including blood sampling. The research protocol was approved by the ethical committees of the participating universities, carried out in accordance with the Declaration of Helsinki, and all respondents provided written informed consent. After 2 (T2), 4 (T4), and 6 years (T6) a face-to-face follow-up assessment was conducted with a response of 87.1% ($n = 2,596$) at T2, of 80.6% ($n = 2,402$) at T4 and 75.7% ($n = 2,256$) at T6. Experiential avoidance was measured twice at T2 and at T4.

MEASURES

Psychiatric Diagnosis

At T0, T2, T4 and T6 past or present DSM-IV depressive (MDD, DYS) or anxiety (PD, SAD, GAD, AGO) disorders were established using the Composite Interview Diagnostic Instrument (CIDI, version 2.1). The CIDI is a worldwide used fully standardized instrument, which classifies diagnoses according to DSM-IV criteria (APA, 1994). It has shown high interrater reliability, high test-retest reliability and high validity for depressive and anxiety disorders (Wittchen, 1994). The CIDI was administered by more than 40 research assistants that have been trained, including psychologists, nurses, or residents in psychiatry. Research assistants received 1 week of training by the fieldwork coordinator, and were certified to conduct assessments following approval of audiotapes of at least two complete interviews. Question wording and probing behavior of interviewers was constantly monitored by checking a random selection of about 10% of all taped interviews. In addition, a

continuous monitoring system of interviewer variances and interviewer-specific item-nonresponse was maintained through computer analyses in SPSS software.

Based on the results of several large-scale epidemiological studies of the structure of psychopathology assessing diagnostic comorbidity patterns phenotypically and/or genotypically (for reviews, see Beesdo-Baum et al., 2009; Clark & Watson, 2006) and based on a previous study of the optimal longitudinal factor structure of emotional disorders in the NESDA study (Spinhoven, Penelo, de Rooij, Penninx, & Ormel, 2014), individual diagnoses were subsumed under the broader categories of distress (DYS, MDD, GAD) and fear (SAD, PD, AGO) disorders and operationalized as an ordinal variable (range 0 – 3). In contrast to the DSM-IV/5 model, which distinguishes depressive and anxiety disorders, GAD is subsumed under the distress disorders in this categorization and not under the anxiety disorders. This categorization reflects the particularly high comorbidity of depressive disorder with GAD, possibly due to a shared single genetic diathesis (Kendler, 2006).

Experiential Avoidance

EA was measured by self-report with the Dutch version of the 9-item Acceptance and Action Questionnaire (AAQ-I; Boelen & Reijntjes, 2008; Hayes et al., 2004) at T2 and T4. Items are scored on a 7-point Likert scale ranging from 1 = *never true* to 7 = *always true*. Total scores range from 9 to 63. A previous study of the Dutch AAQ-I showed that a one-factor model, with AAQ items constituting a single dimension of EA, fitted the data well (Boelen & Reijntjes). Also, the internal consistency (.74) and temporal stability of the AAQ (.82) were satisfactory. Internal consistency of the AAQ-I in the present study was: $\alpha = 0.69$ at T2 and $\alpha = 0.74$ at T4.

STATISTICAL ANALYSES

In order to analyze whether changes in EA scores are associated with changes in psychopathology status, four psychopathology course groups were composed: (a) persons with no disorder at T2 and T4 (unaffected group); (b) persons with no disorder at T2 and a disorder at T4 (occurrence group); (c) persons with a disorder at T2 and no disorder at T4 (recovery group); and (d) persons with a disorder at T2 and at T4 (affected group). For each psychopathology course group paired *t*-tests were performed to compare uncorrected T2 and T4 EA scores. In addition, we calculated change scores by subtracting T2 scores from T4 EA scores and computed Pearson correlations of T2 EA scores with uncorrected EA change scores in each psycho-

pathology course group to get an indication for regression to the mean. To prevent bias into the estimate of change by regression to the mean, we next compared the EA change scores across psychopathology status groups using an analysis of covariance (ANCOVA) with T2 EA scores as covariates. We calculated within groups Cohen's *d* and between groups Cohen's *d* (unaffected versus occurrence group and recovery versus affected group) to get an estimate of the magnitude of the changes and differences, respectively. We considered effects sizes of .20 to represent a small, of .50 a moderate, and of .80 or larger a large effect (Cohen, 1988). In order to examine possible differential effects of fear and distress disorder on state fluctuations in EA, the above analyses were repeated for participants without distress disorder at T2 and T4 and for participants without fear disorder at T2 and T4.

We conducted several analyses to examine whether EA is associated with emotional disorders. First, using analysis of variance (ANOVA) we compared T2 EA scores of the different psychopathology course groups (i.e., the unaffected, occurrence, recovery, and affected group) in the total sample and separately for participants without distress disorder at T2 and T4 and for participants without fear disorder at T2 and T4. Next, in order to determine the direction of the relationship of EA with changes in distress and fear disorders, using structural equation modeling (SEM) we examined a fully cross-lagged autoregressive model with the autoregressive effects and EA, distress and fear disorders at T2 predicting each other at T4 while controlling for interrelationships among distress disorders, fear disorders, and experiential avoidance at T4.

Finally, using SEM we tested two longitudinal mediation models examining the role of EA as a putative mediator of the longitudinal association of distress with fear disorders (and vice versa). More specifically, we determined (a) the association of T2 distress with T6 fear disorders (and T2 fear with T6 distress disorders); (b) the association of T2 distress/fear with T4 EA, controlling for T2 EA; (c) the association of T4 EA with T6 distress/fear, controlling for T2 distress/fear; and (d) the attenuation of the association of T2 distress with T6 fear after accounting for EA at T4 (and vice versa). In this way we could analyze whether T2 distress disorders predicted EA at T4 and whether EA at T4 predicted subsequent fear disorders at T6 (and vice versa whether the relation of T2 fear disorders with subsequent distress disorders at T6 was mediated by EA at T4). The significance of the indirect effect of fear disorders on distress disorders through EA (and vice versa) was determined using a bootstrap

approximation with 1,000 iterations to obtain biased-controlled confidence intervals.

In our SEM analyses we used a weighted least squares estimator with a diagonal weight matrix and robust standard errors and a mean- and variance-adjusted χ^2 -statistic (WLSMV). The WLSMV estimator is appropriate for categorical and nonmultivariate normal data and provides consistent estimates when data are missing at random with respect to covariates (Asparouhov & Muthén, 2010). In these models pathways to ordered categorical outcome variables (e.g., presence of emotional disorders) are denoted by standardized probit regression coefficients and pathways to continuous outcome variables (e.g., level of EA) by standardized linear regression coefficients. In case of nonsaturated models, model fit was evaluated using the Tucker-Lewis Index (TLI), the Comparative Fit Index (CFI), and the Root Mean Square Error of Approximation (RMSEA). For the TLI and CFI, values between 0.90 and 0.95 are considered acceptable, and > 0.95 as good. For the RMSEA, acceptable models have values of < 0.10 , and good models of < 0.05 . As for models with more than 400 cases the χ^2 is almost always statistically significant, in the present study we did not consider a significant χ^2 value to indicate an unsatisfactory model fit.

Analyses were run using SPSS v. 21 (Corp., IBM, 2012) and MPlus v. 7.1 (Muthén & Muthén, 1998-2012). A significance level of $p < .05$ was used for all analyses.

Results

CHANGES IN EXPERIENTIAL AVOIDANCE FOLLOWING CHANGES IN PSYCHOPATHOLOGY STATUS OVER 2-YEAR PERIOD

Changes in AAQ scores were analyzed in the 2,085 participants with complete T2 and T4 AAQ measurements. AAQ measurements proved to be highly correlated, $r = .70$, $p < .001$. Table 1 shows EA scores at T2 and T4 for the different psychopathology course groups (i.e., the unaffected, occurrence, recovery, and affected group) and tests of within-group differences. All groups—except the affected group—showed significant changes in uncorrected EA scores over time ($p < .001$). The significant decline in EA in the unaffected group had a negligible effect size ($d < .20$), while the decline in EA in the recovery group and the increase in EA in the occurrence group had a small effect size ($.20 < d < .50$). In each group the correlation of T2 EA scores with uncorrected EA change scores was significant at $p < .001$ and varied from .36 – .42. Table 1 also shows the results of the ANCOVA of EA difference scores between psychopathology course groups corrected for T2 EA. This analyses showed a significant effect for group, $F(3, 2080) = 58.93$, $p < .001$. Post hoc Bonferroni comparisons showed a significant difference in change in EA between the unaffected and occurrence group ($p < .001$) and the recovery and affected group ($p < .001$). The between-group effect sizes for these differences are

Table 1
Experiential Avoidance (EA) at 2- and 4-Year Follow-up by Psychopathology Status and Test of Change in EA Scores ($n = 2,085$)

Group	n	T2	T4	Difference 2- and 4- year follow-up ^a			Difference in change across groups ^b				
		M (SD)	M (SD)	M (95% CI)	t	p	d	M (95% CI)	F	p	d
<u>Distress/Fear</u>											
Unaffected	1160	29.1 (6.7)	28.3 (7.2)	-8 (-1.1 - -.4)	-4.46	<.001	.11	-2.0 (-2.3 - -1.6)	58.91	<.001	.65 ^c
Occurrence	191	33.3 (6.6)	34.8 (7.2)	1.5 (.7 - 2.3)	3.57	<.001	.22	1.8 (1.0 - 2.6)			
Recovery	296	35.7 (6.2)	33.5 (6.9)	-2.2 (-2.9 - -1.6)	-6.42	<.001	.34	-1.1 (-1.7 - -.4)			.57 ^d
Affected	438	38.7 (6.7)	38.8 (6.9)	.1 (-.5 - .7)	.41	ns	.01	2.3 (1.7 - 2.9)			
<u>Distress only</u>											
Unaffected	1160	29.1 (6.7)	28.3 (7.2)	-8 (-1.1 - -.4)	-4.46	<.001	.11	-1.2 (-1.5 - -.9)	23.93	<.001	.74 ^c
Occurrence	97	32.5 (5.9)	34.8 (7.1)	2.3 (1.1 - 3.5)	3.76	<.001	.35	3.0 (1.9 - 4.2)			
Recovery	138	35.7 (6.4)	33.3 (7.0)	-2.4 (-3.5 - -1.4)	-4.48	<.001	.36	-.6 (-1.6 - .4)			.53 ^d
Affected	78	36.8 (6.3)	37.1 (6.5)	.3 (-1.0 - 1.6)	.39	ns	.05	2.5 (1.7 - 3.7)			
<u>Fear only</u>											
Unaffected	1160	29.1 (6.7)	28.3 (7.2)	-8 (-1.1 - -.4)	-4.46	<.001	.11	-1.2 (-1.5 - -.8)	18.17	<.001	.47 ^c
Occurrence	70	33.9 (7.2)	34.4 (7.7)	.5 (-.8 - 1.8)	.77	ns	.07	1.7 (.4 - 3.1)			
Recovery	90	35.6 (5.7)	33.4 (6.2)	-2.2 (-3.3 - -1.1)	3.98	<.001	.37	-.4 (-1.6 - .8)			.55 ^d
Affected	82	37.0 (8.1)	37.9 (7.8)	.9 (.6 - 2.4)	1.22	ns	.11	3.1 (1.9 - 4.4)			

Note. M = mean; SD = standard deviation; 95% CI = 95% confidence interval; ^a Based on paired samples t-test between 2- and 4-year follow-up EA scores; ^b ANCOVA for difference scores for EA across groups, corrected for baseline EA; ^c Effect size difference between unaffected and occurrence group; ^d Effect size difference between recovery and affected group.

moderate, confirming state-dependent influences on level of EA.

Next, we differentiated between distress (DYS, MDD, GAD) and fear (PD, SAD, AGO) disorders in studying change in EA following changes in psychopathology (see Table 1). Also, in the subgroup of unaffected participants with no fear disorder at T2 and T4, change of EA was associated with the occurrence of and recovery from distress disorders ($p < .001$), while in the subgroup of participants with no distress disorder at T2 and T4, change of EA was associated with the occurrence and recovery from fear disorders ($p < .001$). In both subgroups the effect size of the difference in adjusted change scores between the unaffected versus occurrence group and the affected versus the recovery group was moderate, suggesting that state dependent influences on level of EA did not critically depend on type of disorder.

CROSS-SECTIONAL DIFFERENCES IN EXPERIENTIAL AVOIDANCE BETWEEN PSYCHOPATHOLOGY COURSE GROUPS

ANOVA revealed a significant main effect for group in analyzing T2 EA measurements, $F(3, 2081) = 254.09, p < .001$ (see Table 1 for descriptives). Post hoc comparisons showed significantly higher EA scores in participants with a disorder at T2 (recovery and affected group) than without a disorder at T2 (unaffected and occurrence group). Interestingly, the occurrence group also showed significantly higher levels of EA than the unaffected group, while the

recovery group showed significantly lower levels of EA than the affected group. These results indicate that among the participants with no disorder at T2, those with higher levels of EA were more prone to develop a disorder, while among the participants with a disorder at T2, those with lower levels of EA were more prone to remit. Repeating these analyses separately for participants without distress disorder at T2 and T4, $F(3,1398) = 63.92, p < .001$, and for participants without fear disorder at T2 and T4, $F(3, 1469) = 72.57, p < .001$, yielded similar results except that there were no significant differences in EA between the recovery and affected group.

DIRECTION OF THE RELATIONSHIP OF EXPERIENTIAL AVOIDANCE WITH DISTRESS AND FEAR DISORDERS

In order to assess the direction of the relation of level of EA with changes in the number of distress and fear disorders (and vice versa), we used a fully cross-lagged panel approach allowing residual correlations among number of distress disorders and fear disorders and level of EA at T4 (see Fig. 1) ($n = 2,316$). The model showed an acceptable fit to the data, $\chi^2(2) = 10.45, p < .01$; RMSEA = .04; CFI = 1.00; TLI = .98. As can be derived from this model, distress and fear disorders at T2 significantly predicted EA at T4, $\beta = .06, p < .001$, respectively, $\beta = .08, p < .001$. EA at T2 significantly predicted both distress disorders, $\beta = .33, p < .001$, and fear disorders, $\beta = .34, p < .001$, at T4 in an

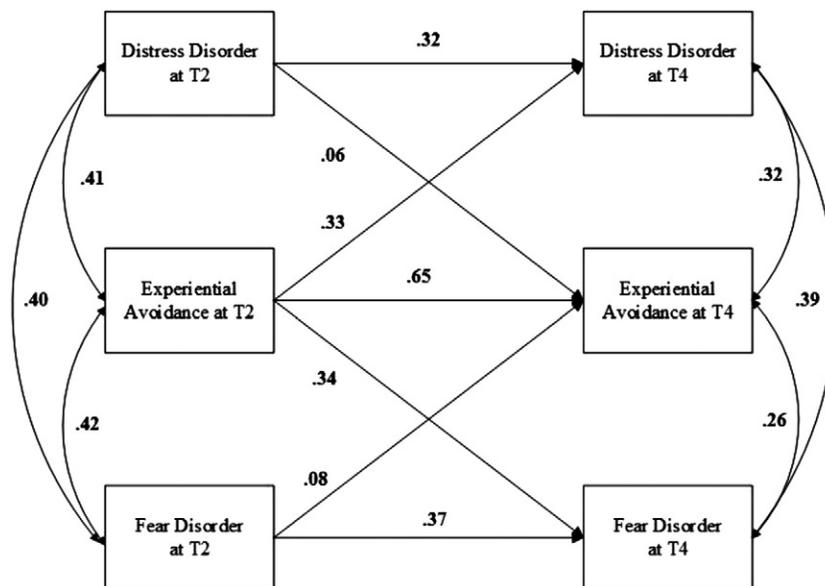


FIGURE 1 Standardized parameter estimates for fully cross-lagged autoregressive model with the autoregressive effects and experiential avoidance, distress and fear disorders at T2 predicting each other at T4 ($n = 2,316$).

equal magnitude. Standardized probit regression coefficients of EA as a predictor of distress and fear disorders at T4 were comparable to those of distress and fear disorders as predictors.

EXPERIENTIAL AVOIDANCE AS A MEDIATOR OF LONGITUDINAL ASSOCIATIONS OF DISTRESS DISORDERS WITH FEAR DISORDERS

Next, we analyzed the associations between number of distress disorders at T2 and number of fear disorders at T6 while controlling for number of fear disorders at T2 (and vice versa) ($n = 2,316$). T2 fear disorders significantly predicted T6 distress disorders, controlling for T2 distress disorders, $\beta = .19$, $p < .001$. T2 distress disorders were significantly associated with T6 fear disorders, controlling for T2 fear disorders, $\beta = .11$, $p < .001$.

Consequently, we next examined EA both as a mediator of the longitudinal fear-distress as well as of the distress-fear association. T2 fear was associated with T4 EA, controlling for EA at T2, $\beta = .10$, $p < .001$. T4 EA was associated with T6 distress, controlling for T2 distress, $\beta = .37$, $p < .001$. In the final mediation model, T2 fear remained a significant predictor of T6 distress, controlling for T2 distress and T2 EA, when T4 EA was added to the model, $\beta = .07$, $p < .05$ (see Fig. 2). The covariance between T2 distress and T2

fear disorders and between T2 EA and both T2 distress and fear was accounted for in the final model. Fit indices indicated that the model had an acceptable fit to the data: $\chi^2(2) = 23.773$, $p < .001$; RMSEA = .07, TLI = .99; CFI = .94. Bootstrapping estimates showed that the indirect effect of fear through EA on distress ($\beta = .04$; 95% BCI = .03 – .05) was significant.

Finally, we analyzed EA as a putative mediator of the longitudinal distress–fear association in a similar way. T2 fear was associated with T4 EA, controlling for EA at T2, $\beta = .08$, $p < .001$. T4 EA was associated with T6 fear, controlling for T2 fear, $\beta = .29$, $p < .001$. In the final mediation model, T2 fear was no longer a significant predictor of T6 distress, controlling for T2 distress and T2 EA, when T4 EA was added to the model, $\beta = .01$, $p = .63$ (see Fig. 3). Fit indices indicated that the model had an acceptable fit to the data: $\chi^2(2) = 26.622$, $p < .001$; RMSEA = .07, TLI = .99; CFI = .93. Bootstrapping estimates showed that the indirect effect of fear through EA on distress ($\beta = .03$; 95% BCI = .02 – .04) was significant.

Discussion

The general aim of the present study was to examine longitudinal relations of experiential avoidance (EA) with distress (MDD, DYS, GAD) and fear disorders

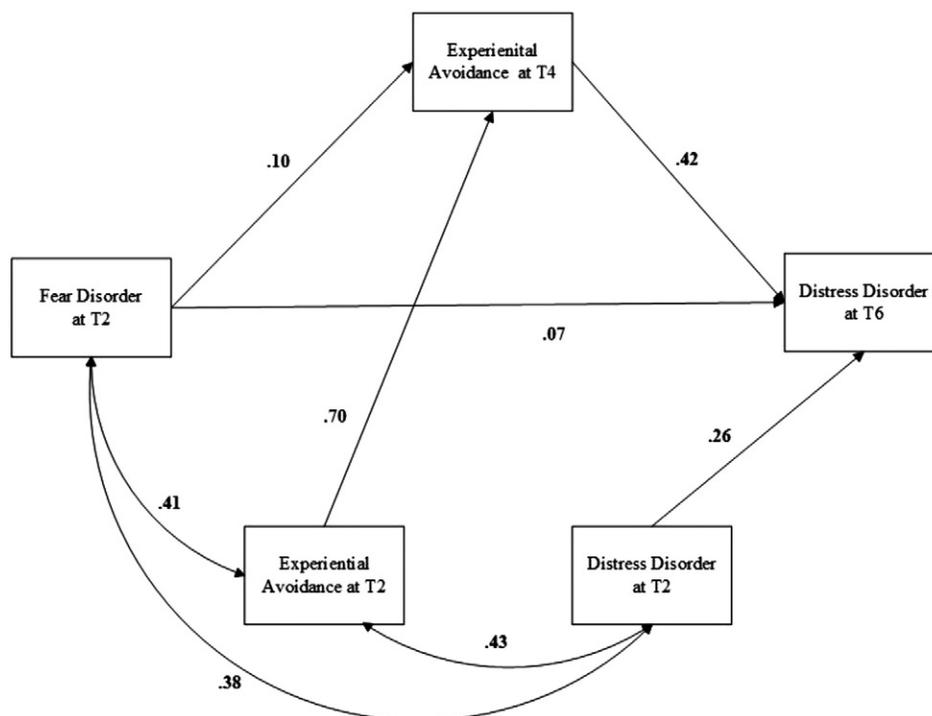


FIGURE 2 Standardized parameter estimates for the longitudinal mediation model of experiential avoidance at T4 mediating the prospective relations between fear disorders at T2 and distress disorders at T6 ($n = 2,316$).

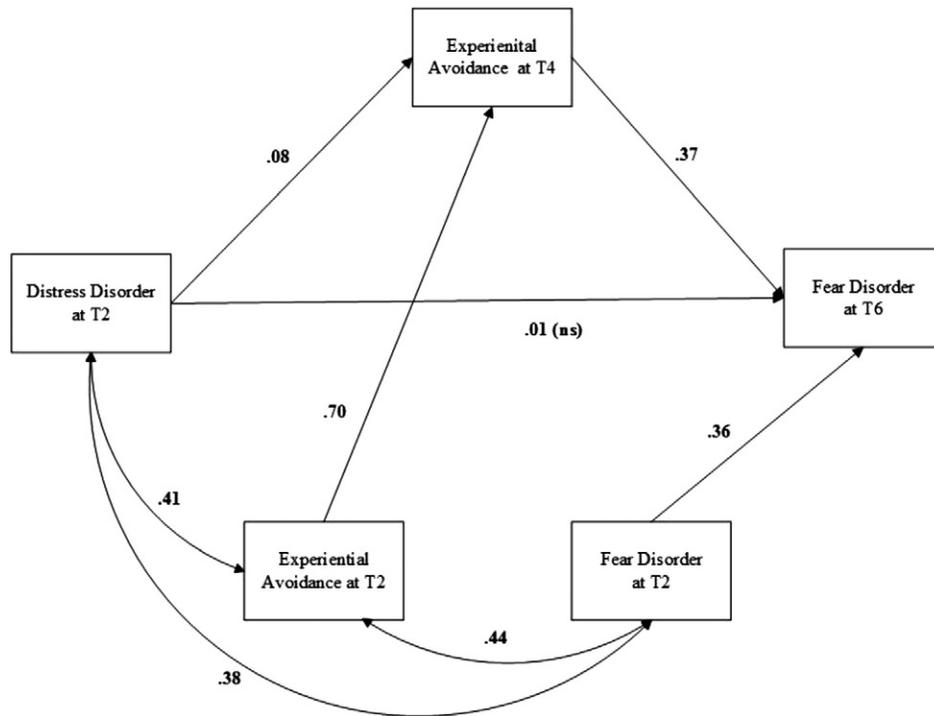


FIGURE 3 Standardized parameter estimates for the longitudinal mediation model of experiential avoidance at T4 mediating the prospective relations between distress disorders at T2 and fear disorders at T6 ($n = 2,316$).

(PD, SAD, AGO). Results showed that EA scores were stable over a 2-year period notwithstanding state fluctuations because of current emotional disorder. Moreover, EA scores at T2 predicted occurrence and recovery, as well as persistence of distress and fear disorders during 2-year follow-up. In addition, EA scores at T2 predicted number of distress and fear disorders at T4 after controlling for number of distress and fear disorders at T2. Finally, EA at T4 mediated the longitudinal association of fear disorders at T2 with distress disorders at T6 and vice versa. These findings suggest that EA scores are more than epiphenomena of emotional disorders (concomitants model) and that EA may be conceptualized as a relevant transdiagnostic factor affecting the course of emotional disorders (pathoplasticity model).

Our first study aim was to examine the degree to which EA measurements are affected by current emotional disorder. The occurrence of a disorder was associated with increased EA and the remittance of a disorder with decreased EA scores. When differentiating between the occurrence and remittance of distress and fear disorders, changes in EA scores were associated with the occurrence and remittance of distress disorders as they were with the occurrence and remittance of fear disorders. The effect size of the state effects, however, was moderate, while the

association of EA scores over time was large. These results concur with those of studies of state effects in the measurement of other personality traits (Karsten et al., 2012; Spinhoven et al., 2013) and suggest that although in accordance with the concomitants model (Klein et al., 2011) the assessments of EA were colored by the individual's emotional state, these changes occur within the context of a relative stability of EA scores.

Our second study aim was to assess whether in accordance with a pathoplasticity perspective (Klein et al., 2011) EA is a possible causal factor in the course of emotional disorder. We found: (a) that participants with a fear or distress disorder showed higher levels of EA than participants without disorder; (b) that among the participants with no distress or fear disorder at T2, higher EA was a risk factor to develop such a disorder; and (c) that among the participants with a disorder at T2, higher EA was a risk factor for persistence of the disorder. In addition, we observed that EA at T2 predicted the number of distress as well as of fear disorders at T4, also after controlling for the number of distress and of fear disorders at T2. Although these results need replication, they suggest in accordance with previous studies (Berking et al., 2009; Marx & Sloan, 2005; Shallcross et al., 2010) that level of EA is more than an epiphenomenon of emotional disorder and

may have a causal effect on the further course of emotional disorders. However, it has to be kept in mind that, also on the basis of longitudinal correlational data, no firm conclusions about causality can be drawn. Although we were able to collect data about covariation (i.e., EA is related to presence of disorder) and temporal precedence (i.e., EA is predictive of course of disorder), critically nonspuriousness cannot be ruled out (Garber & Hollon, 1991). Other possible causes (third variables) such as more basic personality variables (e.g., neuroticism or behavioral inhibition) or more specific affective-cognitive processes (e.g., repetitive negative thinking or suppression) could underlie cross-sectional and prospective associations of EA with emotional disorders.

Our third and last study aim was to determine the extent to which EA predicts the co-occurrence of emotional disorders. EA significantly mediated the longitudinal association of fear disorders with distress disorders. Importantly, the three waves of our study allowed us to study EA as a variable mediating the longitudinal association of fear with distress disorders (and vice versa). We found that fear disorders at T2 predicted EA scores at T4, which subsequently predicted distress disorders at T6 (and vice versa). These mediation results are also in accordance with a pathoplasticity perspective on EA and suggest that EA not only affects the course of particular disorders, but also increases the risk of a future distress disorder when suffering from a fear disorder and vice versa. This cross-disorder effect provides compelling evidence for EA as a broad transdiagnostic risk factor for the occurrence and co-occurrence of multiple disorders.

Harvey et al. (2004) have identified 12 transdiagnostic maintenance processes across the domains of attention, reasoning, memory, thought, and behavior (e.g., selective attention, interpretation bias, over-general memory, worry, rumination, thought suppression and avoidance), and still more processes have been proposed since then. As most existing studies investigated one or only a limited number of putative transdiagnostic factors in relation to a limited number of disorders, it remains unknown whether factors will still show significant associations with particular forms of psychopathology within models that include other putative transdiagnostic factors, while taking comorbidity between disorders into account. Moreover, it has to be examined whether transdiagnostic factors potentially load on one or more latent factors, possibly providing a more parsimonious explanation of their contribution to psychopathology. For example, Aldao and Nolen-Hoeksema (2010) found that rumination, suppression, and reappraisal were indicative of a latent factor

of cognitive emotion regulation, which was associated with anxiety, depression, and eating disorders. Relatedly, it has been found that worry, thought suppression, and experiential avoidance were separate predictors of anxiety and depression, but that these cognitive processes loaded on a single factor of perceived inability to control negative thinking that explained more variance in symptoms than the processes separately (Bird, Mansell, Dickens, & Tai, 2013). These results underscore the need to study commonalities between transdiagnostic factors and identify possible higher-order factors. As the boundaries between EA and related constructs, such as thought suppression, thought control, avoidance coping, and emotional suppression, are unclear (Chawla & Ostafin, 2007) future studies including multiple measures are needed to examine whether EA is a broad overarching independent construct or shares a lot of variance with related constructs.

Common transdiagnostic processes such as EA across emotional disorders constitute the rationale for transdiagnostic or unified therapies that apply the same underlying treatment principles across mental disorders without tailoring the protocol to specific diagnoses. Reducing experiential avoidance or increasing emotional awareness is a core component in acceptance-based treatments such as Acceptance and Commitment Therapy (Hayes, 2004) as well as in therapies applying traditional CBT-strategies in a generic way (such as the Unified Protocol for Transdiagnostic Treatment of the Emotional Disorders; Barlow, 2011). The few available outcome studies only cautiously support evidence for the efficacy of these treatment forms in anxiety and depression (Hunot et al., 2013; Churchill et al., 2013; Reinholt & Krogh, 2014), showing that these treatments are more effective than no treatment and not less effective than disorder-specific treatments. Strikingly and in accordance with the transdiagnostic nature of these treatments are the higher rates of remission from comorbid depressive and anxiety disorders than following disorder-specific treatments. More high-quality and large-scaled studies on the efficacy of transdiagnostic treatments focusing on core transdiagnostic processes in patients with comorbid disorders are urgently needed.

Strengths of this study include a prospective and longitudinal design in a large sample of participants with depressive and/or anxiety disorder from different recruitment settings and the use of a structured diagnostic interview to assess presence of depressive and anxiety disorders. Although we consider analyzing psychiatric diagnoses as dependent variables as a study strength, it should be kept in mind that elevated levels of anxiety and depression may also be

present in persons meeting criteria for subthreshold diagnosis showing degrees of distress and impairment comparable to those of persons meeting formal diagnostic criteria (Merikangas et al., 2003). As most of the studies on the relation of EA with level of psychopathology have been conducted in student samples or clinical groups without formal diagnosis (Chawla & Ostafin, 2007), our results expand upon previous studies reporting a relationship of EA with level of psychopathology using continuous measures of symptom severity by showing that such a relation of EA also exists with emotional disorders using dichotomous diagnostic categories.

The results of this study also need to be considered in light of several limitations: (a) The AAQ-I as used in the present study has been criticized for having overly complex items or items showing some overlap with other concepts (Chawla & Ostafin, 2007). A new 7-item AAQ-II has been developed to improve its psychometric consistency (Bond et al., 2011). However, the correlation of the AAQ-I with the AAQ-II is very high ($r = .97$). (b) As the AAQ was only assessed at two waves, the consequences (or scar) model could not be evaluated. An adequate test of this model requires assessing persons before and after a first episode to determine whether EA scores remain higher after disorder occurrence. (c) The size of the standardized estimates of the indirect paths from fear disorder to distress disorder through EA (and vice versa) as established by bootstrapping are hard to interpret as they involve the multiplication of a standardized linear regression coefficient (path to EA) with a standardized probit regression coefficient (path to distress or fear disorder). (d) As the NESDA study only investigates anxiety and depressive disorders, the relation of EA as a transdiagnostic factor with other emotional disorders (e.g., substance use disorder or borderline personality disorder) warrants further study. (e) Finally, selective attrition during follow-up may have resulted in biased parameter estimates using the WLSVM estimator in analyzing all available information for all estimators.

Conclusions

The findings from the present study suggest that experiential avoidance constitutes a relatively stable trait, which as a broad transdiagnostic risk factor may determine course and comorbidity of emotional disorders. In transdiagnostic treatment interventions for emotional disorders, it seems warranted to include interventions specifically targeting this transdiagnostic factor.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

References

- Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy*, 48, 974–983. <http://dx.doi.org/10.1016/j.brat.2010.06.002>
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Asparouhov, T., & Muthen, B. (2010). *Weighted least squares estimation with missing data*. Retrieved from <http://www.statmodel.com/download/GstrucMissingRevision.pdf>
- Baer, R. A. (2007). Mindfulness, assessment, and transdiagnostic processes. *Psychological Inquiry*, 18(4), 238–242. <http://dx.doi.org/10.1080/10478400701598306>
- Barlow, D. H. (2011). *Unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide*. New York: Oxford University Press.
- Barlow, D. H., Allen, L. B., & Choate, M. L. (2004). Toward a unified theory for emotional disorders. *Behavior Therapy*, 35, 205–230. [http://dx.doi.org/10.1016/S0005-7894\(04\)80036-4](http://dx.doi.org/10.1016/S0005-7894(04)80036-4)
- Beesdo-Baum, K., Hofler, M., Gloster, A. T., Klotsche, J., Lieb, R., Beauducel, A., . . . Wittchen, H. U. (2009). The structure of common mental disorders: A replication study in a community sample of adolescents and young adults. *International Journal of Methods in Psychiatric Research*, 18, 204–220. <http://dx.doi.org/10.1002/Mpr.293>
- Berking, M., Neacsiu, A., Comtois, K. A., & Linehan, M. M. (2009). The impact of experiential avoidance on the reduction of depression in treatment for borderline personality disorder. *Behaviour Research and Therapy*, 47, 663–670. <http://dx.doi.org/10.1016/j.brat.2009.04.011>
- Bird, T., Mansell, W., Dickens, C., & Tai, S. (2013). Is there a core process across depression and anxiety? *Cognitive Therapy and Research*, 37, 307–323. <http://dx.doi.org/10.1007/s10608-012-9475-2>
- Bijl, R. V., van Zessen, G., Ravelli, A., de Rijk, C., & Langendoen, Y. (1998). The Netherlands Mental Health Survey and Incidence Study (NEMESIS): Objectives and design. *Social Psychiatry and Psychiatric Epidemiology*, 33, 581–586. <http://dx.doi.org/10.1007/s001270050097>
- Boelen, P. A., & Reijntjes, A. (2008). Measuring experiential avoidance: Reliability and validity of the Dutch 9-item Acceptance and Action Questionnaire (AAQ). *Journal of Psychopathology and Behavioral Assessment*, 30, 241–251. <http://dx.doi.org/10.1007/s10862-008-9082-4>
- Bond, F. W., & Bunce, D. (2003). The role of acceptance and job control in mental health, job satisfaction, and work performance. *Journal of Applied Psychology*, 88, 1057–1067. <http://dx.doi.org/10.1037/0021-9010.88.6.1057>
- Bond, F. W., Hayes, S. C., Baer, R. A., Carpenter, K. M., Guenole, N., Orcutt, H. K., . . . Zettle, R. D. (2011). Preliminary psychometric properties of the Acceptance and Action Questionnaire-II: A revised measure of psychological inflexibility and experiential avoidance. *Behavior Therapy*, 42, 676–688. <http://dx.doi.org/10.1016/j.beth.2011.03.007>
- Chawla, N., & Ostafin, B. (2007). Experiential avoidance as a functional dimensional approach to psychopathology: An empirical review. *Journal of Clinical Psychology*, 63, 871–890. <http://dx.doi.org/10.1002/jclp.20400>
- Churchill, R., Moore, T. H. M., Furukawa, T. A., Caldwell, D. M., Davies, P., Jones, H., Shinohara, K., Imai, H., Lewis, G., & Hunot, V. (2013). "Third wave" cognitive and behavioural therapies versus treatment as usual for depression. *Cochrane Database of Systematic Reviews* 2013, 10. Art. No.: CD008705. <http://dx.doi.org/10.1002/14651858.CD008705.pub2>

- Clark, L. A., & Watson, D. (2006). Distress and fear disorders. An alternative empirically based taxonomy of the 'mood' and 'anxiety' disorders. *British Journal of Psychiatry*, *189*, 481–483. <http://dx.doi.org/10.1192/bjp.bp.106.03825>
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). New York: Lawrence Erlbaum Associates.
- Garber, J., & Hollon, S. D. (1991). What can specificity designs say about causality in psychopathology research? *Psychological Bulletin*, *110*, 129–136. <http://dx.doi.org/10.1037/0033-2909.110.1.129>
- Harvey, A. G., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders*. Oxford: Oxford University Press.
- Hayes, S. C. (2004). Acceptance and Commitment Therapy: Relational frame, theory, and the third wave of behavioral and cognitive therapies. *Behavior Therapy*, *35*, 639–665. [http://dx.doi.org/10.1016/S0005-7894\(04\)80013-3](http://dx.doi.org/10.1016/S0005-7894(04)80013-3)
- Hayes, S. C., Strosahl, K., Wilson, K. G., Bissett, R. T., Pistorello, J., Toarmino, D., . . . McCurry, S. M. (2004). Measuring experiential avoidance: A preliminary test of a working model. *Psychological Record*, *54*, 553–578.
- Hayes, S. C., Wilson, K. G., Gifford, E. V., Follette, V. M., & Strosahl, K. (1996). Experimental avoidance and behavioral disorders: a functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology*, *64*, 1152–1168. doi: 0.1037//0022-006X.64.6.1152.
- Hunot, V., Moore, T. H. M., Caldwell, D. M., Furukawa, T. A., Davies, P., Jones, H., Honyashiki, M., Chen, P., Lewis, G., & Churchill, R. (2013). "Third wave" cognitive and behavioural therapies versus other psychological therapies for depression. *Cochrane Database of Systematic Reviews* 2013, 10. Art. No.: CD008704. <http://dx.doi.org/10.1002/14651858.CD008704.pub2>
- Karsten, J., Penninx, B. W. J. H., Riese, H., Ormel, J., Nolen, W. A., & Hartman, C. A. (2012). The state effect of depressive and anxiety disorders on big five personality traits. *Journal of Psychiatric Research*, *46*, 644–650. <http://dx.doi.org/10.1016/j.jpsychires.2012.01.024>
- Kashdan, T. B., Barrios, V., Forsyth, J. P., & Steger, M. F. (2006). Experiential avoidance as a generalized psychological vulnerability: Comparisons with coping and emotion regulation strategies. *Behaviour Research and Therapy*, *44*, 1301–1320. <http://dx.doi.org/10.1016/j.brat.2005.10.003>
- Kendler, K. S. (2006). Major depression and generalized anxiety disorder. Same genes, (partly) different environments – revisited. *British Journal of Psychiatry*, *168*(Suppl. 30), 68–75.
- Klein, D. N., Kotov, R., & Bufferd, S. J. (2011). Personality and depression: Explanatory models and review of the evidence. *Annual Review of Clinical Psychology*, *7*, 269–295. <http://dx.doi.org/10.1146/annurev-clinpsy-032210-104540>
- Landman-Peters, K. M., Hartman, C. A., van der Pompe, G., den Boer, J. A., Minderaa, R. B., & Ormel, J. (2005). Gender differences in the relation between social support, problems in parent–offspring communication, and depression and anxiety. *Social Science & Medicine*, *60*, 2549–2559. <http://dx.doi.org/10.1016/j.socscimed.2004.10.024>
- Marx, B. P., & Sloan, D. M. (2005). Peritraumatic dissociation and experiential avoidance as predictors of posttraumatic stress symptomatology. *Behaviour Research and Therapy*, *43*, 569–583. <http://dx.doi.org/10.1016/j.brat.2004.04.004>
- McLaughlin, K. A., & Nolen-Hoeksema, S. (2011). Rumination as a transdiagnostic factor in depression and anxiety. *Behaviour Research and Therapy*, *49*, 186–193. <http://dx.doi.org/10.1016/j.brat.2010.12.006>
- Merikangas, K. R., Zhang, H., Avenevoli, S., Acharyya, S., Neuenchwander, M., & Angst, J. (2003). Longitudinal trajectories of depression and anxiety in a prospective community study. The Zurich Cohort Study. *Archives of General Psychiatry*, *60*, 993–1000. <http://dx.doi.org/10.1001/archpsyc.60.9.993>
- Muthén, L. K., & Muthén, B. O. (1998–2012). *Mplus user's guide* (7th ed.). Los Angeles, CA: Muthen & Muthen.
- Penninx, B. W., Beekman, A. T., Smit, J. H., Zitman, F. G., Nolen, W. A., Spinhoven, P., . . . Van Dyck, R. (2008). The Netherlands Study of Depression and Anxiety (NESDA): Rationale, objectives and methods. *International Journal of Methods in Psychiatric Research*, *17*, 121–140. <http://dx.doi.org/10.1002/mpr.256>
- Reinholt, N., & Krogh, J. (2014). Efficacy of transdiagnostic cognitive behaviour therapy for anxiety disorders: A systematic review and meta-analysis of published outcome studies. *Cognitive Behaviour Therapy*. <http://dx.doi.org/10.1080/16506073.2014.897367>
- Shahar, B., & Herr, N. R. (2011). Depressive symptoms predict inflexibly high levels of experiential avoidance in response to daily negative affect: A daily diary study. *Behaviour Research and Therapy*, *49*, 676–681. <http://dx.doi.org/10.1016/j.brat.2011.07.006>
- Shallcross, A. J., Troy, A. S., Boland, M., & Mauss, I. B. (2010). Let it be: Accepting negative emotional experiences predicts decreased negative affect and depressive symptoms. *Behaviour Research and Therapy*, *48*, 921–929. <http://dx.doi.org/10.1016/j.brat.2010.05.025>
- Spinhoven, P., Penelo, E., de Rooij, M., Penninx, B. W., & Ormel, J. (2014). Reciprocal effects of stable and temporary components of neuroticism and affective disorders: results of a longitudinal cohort study. *Psychological Medicine*, *44*, 337–348. <http://dx.doi.org/10.1017/S0033291713000822>
- Spinhoven, P., van der Does, W., Ormel, J., Zitman, F. G., & Penninx, B. W. J. H. (2013). Confounding of big five personality assessments in emotional disorders by comorbidity and current disorder. *European Journal of Personality*, *27*, 389–397. <http://dx.doi.org/10.1002/Per.1885>
- Wittchen, H. U. (1994). Reliability and validity studies of the WHO Composite International Diagnostic Interview (CIDI): A critical review. *Journal of Psychiatric Research*, *28*, 57–84. [http://dx.doi.org/10.1016/0022-3956\(94\)90036-1](http://dx.doi.org/10.1016/0022-3956(94)90036-1)

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