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Experiential avoidance predicts persistence of major depressive disorder and generalized anxiety disorder in late adolescence

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Abstract

Objective: Experiential avoidance (EA) is a transdiagnostic construct that may underlie the high comorbidity between major depressive disorder (MDD) and generalized anxiety disorder (GAD). This analysis used data from a longitudinal study (conducted September 2010 – April 2016) to examine whether adolescent EA varies by MDD/GAD symptomology trajectory and predicts said trajectories. Longitudinal associations between EA, anxiety, and depression symptoms were also examined.

Methods: Fifteen to 20 year-old adolescents (N=183) were followed for two years with a comprehensive assessment battery. Symptom trajectory modeling, using weekly symptom ratings, identified 4 MDD and 4 GAD trajectories which were collapsed to form combined MDD/GAD trajectory groups: Persistent (n = 81), High-Decreasing (n = 44), Normal-Increasing (n = 37), and Minimal (n = 21). Analyses of covariance, structural equation modeling, and linear regression analyses were performed.

Results: Persistent adolescents had higher EA versus other groups (p-values ≤ 0.001), with greater EA stability versus High-Decreasing adolescents (p = 0.008). EA predicted anxiety and depressive symptoms alike (p-values ≤ 0.005), which in turn did not predict EA (p-values ≥ 0.188). EA, at both time points, predicted combined MDD/GAD trajectories after adjusting for depressive and anxiety symptoms and other confounders (p-values < 0.001).

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Conclusion: EA appears to be an important predictor of MDD/GAD symptomology in older adolescents, potentially serving as a treatment target. Findings suggest a possible trait-like nature for EA, perhaps increasing risk for the emergence and persistence of MDD and/or GAD.

Trial Registration: ClinicalTrials.gov identifier: .

Keywords

major depressive disorder; generalized anxiety disorder; longitudinal; risk factor; experiential avoidance

Introduction

Major depressive disorder (MDD) and generalized anxiety disorder (GAD) are prevalent psychiatric disorders and leading causes of disability worldwide.[1] MDD and GAD are highly comorbid, particularly among adolescents, which results in more significant distress and functional impairment as compared to either disorder alone.[2,3] Identifying factors that underlie their comorbidity may further our understanding of MDD and GAD, leading to improved treatments.

Experiential avoidance (EA) is a multifaceted transdiagnostic construct that offers promise in this pursuit. Specifically, EA refers to an "unwillingness to remain in contact with uncomfortable private events by escaping or avoiding these experiences."[4] Avoidance of distressing experiences reduces immediate contact with the distressing experiences, delivering short-term relief. Long-term, however, it leads to greater dysfunction and increased distress.[5,6] EA is posited to relate to various forms of psychopathology through both implicit (i.e., classically-conditioned) and explicit pathways (i.e., deliberate avoidance).[7,8]

Cross-sectional adolescent studies have found positive associations between EA, depression, and anxiety at diagnostic (categorical) and symptom (dimensional) levels.[8,9] These studies demonstrated high EA among participants with current MDD and GAD. High EA has also been identified in adolescents at high-risk for MDD who have not developed a major depressive episode yet.[10] It is unknown, however, whether EA changes with disorder onset (or recurrence) and remittance in adolescents.

A few longitudinal studies in adults have examined EA in depressive and anxiety disorder occurrence and remittance, as well as symptom increase. For example, using 6-year data from the Netherlands Study of Depression and Anxiety (NESDA),[11] Spinhoven and colleagues[12] found an association between EA and current depressive and anxiety disorders (collectively termed "distress disorders"). While EA was mostly stable within individuals, it did increase and decrease with distress disorder occurrence and remittance. EA also predicted changes in diagnostic status.[12] A separate study found reduction in EA was significantly associated with reduction in depressive symptoms over the course of a 1-year treatment study of borderline personality disorder.[13] Finally, EA positively predicted daily levels of social anxiety and emotional distress over 3 weeks in a sample of college undergraduates.[14] While these findings highlight the role EA might play in the onset and

course of distress disorders, other adult studies failed to support it.[15,16] To the best of our knowledge, there has yet to be a longitudinal study of *adolescent* EA in relation to distress disorders (i.e., MDD and GAD). Moreover, no study has jointly examined longitudinal relations between EA, depression, and anxiety symptom severity.

The present investigation is a secondary data analysis from a two-year longitudinal study investigating the skeletal effects of selective serotonin reuptake inhibitors (SSRI) in adolescents.[17] The primary aim of the present study was to examine whether adolescent EA varies by combined MDD/GAD symptomology trajectory and predicts trajectories over and above depressive and anxiety symptoms.

We hypothesized that adolescents with persistent MDD and/or GAD would endorse significantly elevated EA[9,10] and exhibit more stability of EA severity longitudinally compared to adolescents of other combined MDD/GAD trajectory groups.[12] We also expected that EA would predict combined MDD/GAD trajectories, after controlling for anxiety and depression. In an exploratory analysis, we also sought to examine the directionality of longitudinal associations between EA, anxiety, and depression symptoms.

Methods

Procedures and participants

The University of Iowa Institutional Review Board approved this longitudinal, observational study[17] which was conducted from September 2010 – April 2016 (ClinicalTrials.gov identifier:). Adult participants and parents/guardians of minor participants provided written informed consent and minors provided assent. Fifteen to 20 year-olds were recruited through inpatient and outpatient clinical settings, advertisements, and word of mouth. They were enrolled either psychotropic-free or within a month of starting a SSRI, regardless of diagnostic status excluding eating disorders and substance dependence.[17] Additional exclusion criteria included significant medical history; pregnancy; concomitant treatment with other antidepressants, mood stabilizers, or antipsychotics; chronic use of medications affecting bone metabolism; or plans to soon move out-of-state.

At the baseline visit, demographic data were collected, including self-reported race/ethnicity. Participants completed the Beck Depression Inventory-II (BDI-II)[18] and the Beck Anxiety Inventory (BAI)[19] and trained research staff administered the Longitudinal Interval Follow-Up Evaluation for Adolescents (A-LIFE).[20] Participants returned for follow-up visits every four months to complete this battery, and post-visit meetings were held to reach clinical consensus on A-LIFE ratings. EA was assessed using the Acceptance and Action Questionnaire, second version (AAQ-II).[21] This was administered twice during the study, the first time being at the first follow-up visit. On average, there were 342.1 days (SD = 56.7) between the initial (V1) and second (V2) visits at which AAQ-II data were collected.

Measures

Experiential avoidance.—The AAQ-II is a self-completed questionnaire consisting of 7 items, rated on a 7-point Likert scale with higher scores indicating greater EA (range = 7 to 49).[21] The AAQ-II has demonstrated good psychometrics in late-adolescent and young-adult

samples.[22] Internal consistencies, as measured by Cronbach's alpha (α), were 0.92 and 0.93 for V1 and V2, respectively.

Depressive symptoms.—On the self-completed widely-used 21-item BDI-II[18] responses are scored on a 4-point Likert scale with higher scores indicating greater depression (range = 0 to 63). Internal consistencies were 0.93 and 0.94 for V1 and V2, respectively.

Anxiety symptoms.—On the self-completed 21-item BAI[19] responses are scored on a 4-point Likert scale with higher scores indicating greater anxiety (range = 0 to 63). Internal consistencies were 0.91 at both V1 and V2.

MDD and GAD weekly symptoms.—The clinician-administered A-LIFE was used to track weekly MDD and GAD symptoms. Clinician ratings on the A-LIFE range from 0 for no symptoms, to 2 to 4 for varying levels of symptom severity and impairment, to 5 and 6 for meeting full DSM-IV-TR criteria.

Data analytic strategy

Consistent with Spinhoven and colleagues,[12] MDD and GAD were subsumed under the broader term of distress disorders. This approach was motivated by significant overlap in symptom presentation and high rates of MDD-GAD co-occurrence.[23–26] A-LIFE-based weekly MDD and GAD ratings were analyzed using group-based trajectory modeling (GBTM), a statistical method used to identify clusters of individuals following similar patterns of progression over time.[27] Models with 3- and 4-group solutions were generated using the censored normal distribution and the polynomial function of time (linear, quadratic, or cubic) that best fit the data. In each model, individuals were assigned to the group where the GBTM-determined probability of membership was highest. The choice of best fitting model was informed by model fit indices[28] and ultimately made by maximizing the number of non-redundant MDD and GAD patterns. Four-group, rather than alternative, solutions for MDD and GAD trajectories were decided upon because they provided better fits to the data and were of greater clinical utility, allowing for more meaningful interpretation of findings. The GBTM analysis was conducted using PROC TRAJ in SAS 9.4.[29]

Bivariate relations were examined with Pearson correlations. Chi-square tests of independence and Bonferroni-corrected Analyses of Covariance (ANCOVAs) were used for group comparisons. Longitudinal associations between EA, anxiety, and depressive symptoms were investigated using cross-lagged autoregressive structural equation modeling (SEM) analyses allowing for residual correlations. Acceptable model fit required the Root Mean Square Error (RMSEA) ≤ 0.08, the Comparative Fit Index (CFI) ≥ 0.90, and the Tucker Lewis Index (TLI) ≥ 0.90. Excellent model fit required RMSEA ≤ 0.06, CFI ≥ 0.95, TLI ≥ 0.95; in both instances, Chi-square (χ^2) was to be large and non-significant ($p \geq 0.05$). [3031] Finally, hierarchical linear regression models were tested with AAQ-II scores predicting combined MDD/GAD symptom trajectories. Variance Inflation Factor (VIF) and Tolerance indices indicated degree of predictor multicollinearity.[32] Covariates included age, sex, and

cumulative SSRI exposure (defined as cumulative time [in days] taking an SSRI) given potential confounding effects.[3334]

Supplemental SEM and regression analyses were performed with an abbreviated, 5-item AAQ-II score to examine whether results would replicate when the items strongly resembling anxiety symptoms ("I worry about not being able to control my worries and feelings" and "Worries get in the way of my successes") were excluded. This approach was taken to reduce potential predictor-outcome overlap.[35] SPSS v. 19[36] and MPlus v. 7.2[37] were utilized.

Results

Participant disposition

Two-hundred seventy-nine participants were recruited; however, 37 were missing all EA data, 8 were excluded for bipolar disorder and/or psychosis, and 2 were excluded for SSRI use prior to starting the study. Forty-nine participants provided EA data only at V1 and were retained for the relevant analyses. This left 183 participants with complete data, who did not significantly differ from those with partial EA data on age ($p = 0.131$), sex ($p = 0.507$), ethnicity ($p = 0.523$), EA ($p = 0.747$), anxiety symptoms ($p = 0.484$), or depressive symptoms ($p = 0.871$) though they differed on race ($p \leq 0.001$).

Identification of MDD/GAD trajectories within the sample

A 4-group trajectory model offered the best fit for both MDD and GAD regardless of whether linear, quadratic, or cubic modeling was used (see supplementary figures A and B). For MDD, group 1 exhibited no symptoms, group 2 exhibited declining symptoms from a relatively low level, group 3 exhibited remitting symptoms from a clinically-significant baseline, and group 4 exhibited persistent clinically significant symptoms. As for GAD, group 1 initially reported minimal symptoms, increasing slightly over time, group 2 had moderate baseline symptoms further increasing during follow up, group 3 had declining symptoms from a clinically-significant level, while group 4 appeared to maintain a significant symptom level over the study period. Subsequently, using these MDD and GAD trajectories, participants were assigned to one of four combined MDD/GAD trajectory categories (Table 1).

Descriptive statistics and group comparison results

Table 2 lists demographic and clinical characteristics of included participants. Several significant group differences were observed across variables. With respect to EA, Persistent adolescents reported significantly greater symptoms at both V1 and V2 than adolescents in other trajectory groups, who did not differ from one another (p-values ≥ 0.05). Adjusting for age, sex, and cumulative SSRI exposure did not substantially alter the findings.

Bivariate correlations

EA, depression, and anxiety symptoms were significantly correlated at each of the 2 visits when the EA was assessed, as well as across visits (Table 3).

Within-subject change in EA between V1 and V2

Groups significantly differed in EA change between V1 and V2 ($F(3, 175) = 4.23$, $p = 0.006$, $\eta^2 = 0.068$), after adjusting for age ($p > 0.90$), sex ($p > 0.50$), cumulative SSRI exposure ($p > 0.40$), and V1 EA ($\beta = -0.469$, $p < 0.001$, 95% confidence interval [CI] = −0.602 to −0.335). Post-hoc analyses showed that the effect was driven by a significant difference in EA change between the High-Decreasing and the Persistent groups (Mean difference in Δ EA = 4.38, 95% CI = 0.815 to 7.950, $p = 0.008$).

Association of EA, depression, and anxiety over time

SEM analyses were performed to examine the association between V1 and V2 EA, depression, and anxiety symptoms. Interrelations were controlled for with residual correlations (Figure 1), while adjusting for age, sex, and cumulative SSRI exposure. The specified model fit the data excellently (χ^2 (5) = 8.16, $p > 0.10$; RMSEA = 0.059; CFI = 0.993; TLI = 0.951). EA at V1 predicted V2 anxiety and depressive symptoms, with V1 EA being the sole significant predictor of V2 EA. Therefore, EA held significant associations with both anxiety and depression across time points. Notably, the model yielded the same pattern of results when using abbreviated AAQ-II scores.

EA predicts MDD/GAD symptom trajectories

Hierarchical linear regression models examined V1 and V2 EA as predictors of combined MDD/GAD trajectories (1 = Minimal, 2 = Normal-Increasing, 3 = High-Decreasing, and 4 = Persistent). Findings were similar whether the outcome was treated as an ordinal or dimensional variable, and therefore data are presented with dimensional outcomes for ease of interpretation. Age, sex, and cumulative SSRI exposure were entered as covariates in step 1 and the model accounted for 25% of outcome variance (Table 4). V1 EA, anxiety, and depressive symptoms were entered at step 2, predicting an additional 15% of outcome variance with V1 EA as the only significant symptom-level predictor. V2 analyses yielded similar results with EA the only significant symptom-level predictor of combined MDD/GAD trajectories. Variance Inflation Factor (VIF, $Mdn \leq 1.154$) and Tolerance ($Mdn \leq 0.867$) indices revealed no threat of multicollinearity in either model.[32]

After imputing for missing data, increasing the sample to $N = 239$, V2 analyses yielded the same pattern of results (data available upon request). Moreover, V1 and V2 results were little changed when performing analyses with abbreviated AAQ-II scores.

Discussion

The present investigation is the first longitudinal study to examine the role of EA in late-adolescence distress *disorders*. Moreover, this study is the first to examine directionality of longitudinal relations between EA, depression, and anxiety *symptoms* in any age group. Levels of EA were consistently elevated among adolescents with Persistent combined MDD/GAD symptom trajectories relative to other groups. Moreover, among symptom trajectory groups, EA was significantly less likely to change in the Persistent versus the High-Decreasing group, suggesting that consistently elevated EA levels may portend chronic MDD and/or GAD symptoms. Notably, EA was the only significant symptom-level

predictor of combined MDD/GAD trajectories, even after accounting for potential confounders. In fact, SEM analyses showed EA to predict future anxiety and depressive symptoms but not vice versa.

Significantly greater EA among adolescents with persistent MDD/GAD symptoms echoes prior findings showing EA to be elevated among adults and adolescents with current MDD and GAD.[8–10,12] These adolescents may more consistently utilize emotion and thought suppression strategies, which can perpetuate psychopathology.[6] Patterns of inner experience and behavior (i.e., thought suppression, emotional suppression, and avoidance coping)[8] among these adolescents may be enduring, similarly present during times of persistent MDD-GAD symptoms and remission. Experimental evidence and laboratory studies on emotional and thought avoidance strategies support the idea that experiential avoidance may be a core feature associated with mood and anxiety disorders, including MDD and GAD.[38–40] In fact, efforts to suppress an unwanted thought can lead to a temporary relief, followed by a period of increased thought frequency and emotional distress.[41] Thus, although these strategies may be reinforcing in the short term, they can result in a vicious cycle of cognitive and emotional avoidance followed by more intense emotional distress associated with reemergence of the unwanted thoughts.[38,39] Interrupting the cycle of avoidance is a key component of several psychotherapeutic interventions.

Prospectively, EA significantly predicted subsequent anxiety and depressive symptoms but the opposite was not the case. This finding is consistent with prior research demonstrating that EA in adults predicts depressive symptom reduction.[42] The fact that earlier depression and anxiety failed to predict subsequent EA suggests unidirectional relations between EA and depressive and anxiety symptoms.

EA, rather than anxiety or depressive symptoms, predicted MDD/GAD symptomology consistently across longitudinal SEM and hierarchical regression analyses. This aligns with the finding of higher EA in adolescent girls at risk for MDD compared to healthy controls[10] and provides support for EA being conceptualized as a transdiagnostic process in late-adolescent distress disorders. An important consideration deals with the degree to which EA is a multifaceted construct, operationally overlapping with anxiety- and depression-related symptoms (i.e., worry, neuroticism, and rumination).[8,43] To mitigate this concern, we performed supplemental analyses with abbreviated AAQ-II scores, omitting GAD-related items to avoid predictor-criterion contamination; the results remained largely unchanged. Remaining AAQ-II items on this abbreviated version includes items relating to dysfunctional distress (i.e., “It seems like most people are handling their lives better than I am”).[44] Therefore, Dysfunctional distress, regardless of excessive worrying, appears to have significant cross-sectional and longitudinal associations with depressive and anxiety symptomology.

Despite its strengths, this study is not without limitations. Assessing baseline EA would have been preferable; however, the AAQ-II was a post-hoc addition to the study battery. Social phobia may have been relevant but was excluded due to oversensitivity of diagnostic measures to subthreshold symptoms. Additionally, a significant portion of participants were taking SSRIs, which treat MDD and GAD effectively.[45,46] Although accounted for in the

analyses, it is not fully clear how SSRI use may have moderated the results. Moreover, whether the findings apply to early adolescents is to be determined. Identifying which EA processes, if any, are more dominant than others given stage of childhood development may also be worth investigating. Finally, generalizability of the findings could have been hampered by the limited ethnic/racial diversity of the participants.

The findings of the current study suggest that an intervention targeting EA in adolescents may lead to valuable outcomes. Acceptance and Commitment Therapy (ACT) is a behavioral intervention that aims to help patients overcome EA with acceptance, mindfulness, and behavioral change strategies.[39] ACT has been identified as an empirically supported treatment for MDD and anxiety disorders, as well as other chronic health problems.[47] Moreover, ACT treatment trials in adults have shown that decreases in experiential avoidance are associated with depressive and anxiety symptom reduction.[4849] Future studies should examine whether changes in EA among adolescents mediate treatment outcomes in depressive and anxiety symptoms.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Clinical points:

- Experiential Avoidance (EA) is a multifaceted transdiagnostic construct implicated in major depressive disorder (MDD) and generalized anxiety disorder (GAD). However, investigation of EA's role in MDD and GAD symptom trajectories, as well as EA's longitudinal associations with depressive and anxiety symptoms, are lacking in adolescence.
- EA was found to associate with persistence of MDD and/or GAD symptomology. Moreover, longitudinal analyses showed EA to predict subsequent depressive and anxiety symptoms, as well as severity of combined MDD/GAD symptoms.
- Findings suggest EA is an important predictor of MDD/GAD symptomology in older adolescents, perhaps serving as a treatment target for extant evidence-based psychotherapeutic interventions.

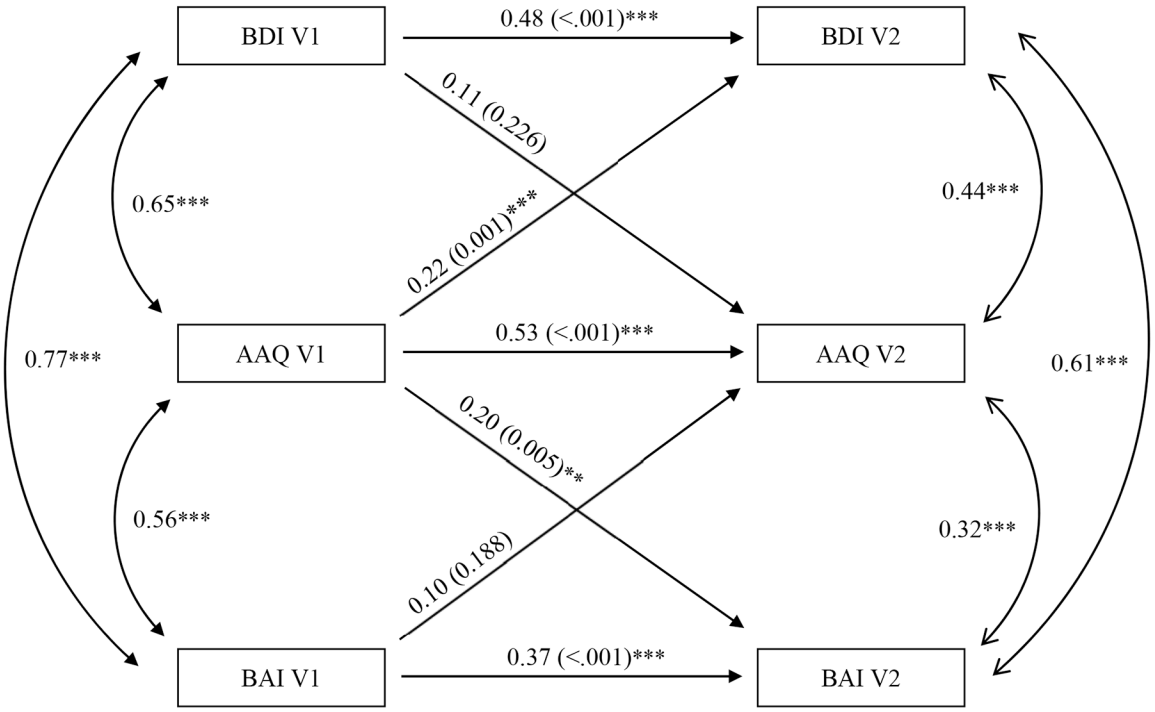

Figure 1. Associations between V1 and V2 EA, anxiety, and depressive symptoms

Note data are standardized parameter estimates (*p*-values). BDI = Beck Depression Inventory-II; AAQ = Action and Acceptance Questionnaire-II; BAI = Beck Anxiety Inventory; V1 and V2 = first and second study visits at which all 3 assessments were administered.

*p < 0.05, **p < 0.01, ***p ≤ 0.001.

Table 1

Participants' combined MDD/GAD trajectory group assignments

Group	*n*	Baseline Symptom Severity	Follow-up Symptom Severity
Persistent	81	Elevated MDD and/or GAD	Elevated MDD and/or GAD
High-Decreasing	44	Elevated MDD and/or GAD	decreasing MDD and GAD
Normal-Increasing	37	Low MDD and GAD	increasing MDD and/or GAD
Minimal	21	Low to absent MDD and GAD	Low to absent MDD and GAD

Clinical significance of symptomology determined via weekly scores on the A-LIFE Psychiatric Status Ratings.

J Clin Psychiatry. Author manuscript; available in PMC 2019 November 14.

Table 2

Participant characteristics and group comparison results on data from visits at which EA data were collected

	Complete data sample *N* = 183	Persistent (P) *n* = 81	High-Decreasing (HD) *n* = 44	Normal-Increasing (NI) *n* = 37	Minimal (M) *n* = 21	*Test Statistic (p-value)*	Group difference
Age (in years)	18.95 (1.61)	19.22 (1.39)	18.41 (1.87)	19.22 (1.40)	18.52 (1.86)	3.40 (0.019)	P > HD
Sex (%female)	61.7	77.8	50.0	54.1	38.1	17.28 (<0.001)	P > HD, NI, M; M < HD, NI
Race (%Caucasian)	77.6	70.4	81.8	78.4	95.2	6.73 (0.077)	-
Hispanic (%no)	81.4	74.1	88.6	78.4	100.0	3.51 (0.136)	-
EA Visit 1 *							
SSRI (%yes)	38.3	53.2	48.1	9.1	0.0	39.13 (<0.001)	P > NI, M
AAQ	17.02 (8.33)	21.57 (8.37)	15.10 (6.31)	11.45 (4.75)	10.71 (4.56)	27.86 (<0.001)	P > HD > NI, M
BAI	4.68 (6.29)	7.42 (7.44)	3.25 (4.20)	1.98 (3.11)	0.29 (0.62)	16.80 (<0.001)	P > HD, NI, M
BDI	5.83 (7.55)	9.93 (8.56)	4.21 (6.24)	2.00 (2.47)	0.42 (0.78)	17.97 (<0.001)	P > HD, NI, M
EA Visit 2							
SSRI (%yes)	21.7	28.6	26.2	5.9	0.0	15.45 (0.001)	P > NI, M
AAQ	16.56 (8.68)	21.51 (9.54)	13.80 (5.56)	12.57 (5.22)	10.24 (4.41)	22.40 (<0.001)	P > HD, NI, M
BAI	4.25 (6.38)	7.36 (7.82)	2.00 (3.11)	1.89 (3.89)	1.14 (2.57)	14.09 (<0.001)	P > HD, NI, M
BDI	5.13 (7.62)	9.30 (9.39)	2.61 (3.92)	1.59 (2.68)	0.57 (0.87)	19.31 (<0.001)	P > HD, NI, M

Data are mean (standard deviation), unless noted otherwise. AAQ = Acceptance and Action Questionnaire-II; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory-II; SSRI = selective serotonin reuptake inhibitors. *Note:* 26 participants, collectively, elected not to self-report ethnicity.

* Figures for EA visit 1 include participants with partial EA data, *N* = 229: Persistent (*n* = 109), High-Decreasing (*n* = 52), Normal-Increasing (*n* = 44), and Minimal (*n* = 24).

Table 3

Pearson correlations among primary study variables

	1	2	3	4	5	6	7	8	9	10
1. Age	-									
2. Sex	.027	-								
3. AAQ at V1†	.163*	−.231***	-							
4. BAI at V1†	.063	−.183**	.564***	-						
5. BDI at V1†	.081	−.154*	.645***	.759***	-					
6. SSRI at V1†	−.101	−.076	.262***	.398***	.309***	-				
7. AAQ at V2	.085	−.170*	.649***	.516***	.527***	.243***	-			
8. BAI at V2	.011	−.208**	.399***	.529***	.421***	.312***	.522***	-		
9. BDI at V2	−.033	−.202**	.528***	.584***	.631***	.273***	.655***	.710***	-	
10. SSRI at V2	−.095	−.057	.224**	.369***	.288***	.931***	.207**	.318***	.274***	-

Age (in years) from baseline visit; Sex, female = 0; AAQ = Action and Acceptance Questionnaire-2; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory-II; SSRI = cumulative SSRI exposure (in days); V1 and V2 = first and second visits at which EA data were collected.

*$p < 0.05$,

**$p < 0.01$,

***$p \leq 0.001$

†Participants with partial EA data (EA collected at V1 only) included, $N = 229$.

Table 4

Hierarchical regression results showing that EA at V1 and V2 predicts combined MDD/GAD trajectories

	B	SE	β	*t*	*p*	F	R^2	Adj. R^2	Δ R^2
EA visit 1 [†]									
Step 1	-	-	-	-	-	24.234	0.248	0.238	-
Constant	1.247	0.709	-	1.760	0.080				
Age	0.091	0.037	0.145	2.464	0.015 *				
Sex	−0.558	0.125	−0.261	−4.454	<0.001 ***				
SSRI	0.004	0.001	0.389	6.609	<0.001 ***				
Step 2	-	-	-	-	-	23.748	0.396	0.380	0.148
Constant	1.283	0.641	-	2.003	0.046 *				
Age	0.049	0.034	0.078	1.443	0.151				
Sex	−0.381	0.116	−0.179	−3.285	0.001 ***				
SSRI	0.003	0.001	0.259	4.448	0.001 ***				
AAQ	0.041	0.009	0.325	4.541	< 0.001 ***				
BAI	−0.001	0.014	−0.003	−0.036	0.972				
BDI	0.018	0.012	0.132	1.485	0.139				
EA visit 2									
Step 1	-	-	-	-	-	18.056	0.232	0.219	-
Constant	1.191	0.827	-	1.439	0.152				
Age	0.093	0.043	0.141	2.147	0.033 *				
Sex	−0.557	0.142	−0.258	−3.930	<0.001 ***				
SSRI	0.002	0.000	0.383	5.819	<0.001 ***				
Step 2	-	-	-	-	-	18.008	0.380	0.359	0.148
Constant	0.896	0.752	-	1.191	0.235				
Age	0.072	0.040	0.110	1.826	0.070				
Sex	−0.397	0.132	−0.184	−3.012	0.003 **				
SSRI	0.002	0.000	0.284	4.557	<0.001 ***				
AAQ	0.036	0.010	0.284	4.504	< 0.001 ***				

	B	SE	β	*t*	*p*	F	R^2	Adj. R^2	Δ R^2
BAI	−0.001	0.014	−0.008	−0.088	0.930				
BDI	0.021	0.013	0.154	1.595	0.112				

B = unstandardized beta; SE = standard error; β = standardized beta; *t* = *t* statistic; *p* = significance level; *F* = *F* statistic; R^2 = variance; Adj. R^2 = Adjusted R^2 (adjusted for the number of model predictors); Δ R^2 = change in explained variance. SSRI = Cumulative SSRI exposure (in days); AAQ = Action and Acceptance Questionnaire-II; BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory-II.

$^{*}p < 0.05$

$^{**}p < 0.01$

$^{***}p < 0.001$.

†Participants with partial EA data (EA collected at V1 only) included, $N = 229$.