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# Focus on and venting of negative emotion mediates the 18-year bi-directional relations between major depressive disorder and generalized anxiety disorder diagnoses

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ARTICLE INFO	A B S T R A C T				
A R T I C L E I N F O Keywords: Generalized anxiety disorder Major depressive disorder Denial Venting Longitudinal Behavioral disengagement	<ul> <li>Background: Myriad emotion regulation and coping theories have proposed that avoidant/emotion-oriented coping is a cause and consequence of anxiety and depression. However, few studies have investigated potential mechanisms underlying the prospective anxiety-depression disorder relation. The current study examined various coping strategies (i.e., denial, focus on and venting of emotion (FOAVE), and behavioral disengagement) as potential longitudinal mediators between generalized anxiety disorder (GAD) and major depressive disorder (MDD).</li> <li>Methods: In a nationally representative sample of adults (N = 3,294), MDD and GAD were assessed at Time 1 (T1) and Time 3 (T3) (Composite International Diagnostic Interview – Short-Form), and avoidant coping strategies (denial, behavioral disengagement, and FOAVE) were measured at Time 2 (T2) (Coping Questionnaire). Assessments occurred over 18 years, each spaced approximately 9 years apart. Structural equation modeling mediation analyses examined whether T1 MDD predicted T3 GAD (and vice versa), and if T2 avoidant coping mediated these relations, above and beyond baseline comorbidity.</li> <li>Results: FOAVE mediated the T1 MDD–T3 GAD association, and vice versa. Presence of T1 MDD and GAD predicted more T2 FOAVE, and greater T2 FOAVE forecasted T3 MDD and GAD, accounting for 16–21% of the longitudinal MDD-GAD relations. However, behavioral disengagement and denial did not mediate the prospective MDD-GAD relations. Also, T1 MDD and GAD forecasted greater T2 behavioral disengagement. Conclusions: The use of FOAVE, may be a mechanism by which MDD earlier in life may lead to GAD 18 years later, and vice versa. Theoretical and potential clinical implications are discussed.</li> </ul>				

Nearly 1 in 6 adults will experience depression at some point in their lives and 29% of people meet criteria for a lifetime anxiety disorder in the United States (Kessler and Wang, 2008). Of these, generalized anxiety disorder (GAD) and major depressive disorder (MDD) are two highly prevalent mental health problems in the U.S. These disorders are characterized by chronic fatigue, irritability, difficulty focusing, and sleep problems. Moreover, GAD and MDD have been related consistently to frequent interpersonal conflicts, poorer relationship quality (Jacobson and Newman, 2016; Shin and Newman, 2019), as well as decreased life satisfaction, educational, and economic attainment (Kessler et al., 2008). Further, elevated GAD and MDD have been associated independently with less personal monthly income on average (Dismuke and Egede, 2010) and lower life satisfaction (Beutel et al., 2010). MDD and GAD can also lead to deleterious effects on physical health by impacting

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https://doi.org/10.1016/j.jad.2022.01.079 Received 6 June 2021; Accepted 18 January 2022 Available online 20 January 2022 0165-0327/© 2022 Elsevier B.V. All rights reserved. neurocognitive, physiological, cardiorespiratory, and immune systems (Bartek et al., 2021; Ottaviani et al., 2016; Zainal and Newman, 2021; Zainal and Newman, 2022). Further, a robust GAD-MDD bidirectional association exists, such that GAD predicts future MDD, and vice versa (see meta-analyses by Jacobson and Newman, 2017). Thus, understanding the mechanisms accounting for the GAD-MDD prospective relation is important.

The *scar theory* proposes that elevated anxiety and depression can reinforce maladaptive habits (e.g., avoidant coping, suboptimal lifestyle choices) across prolonged durations by impacting vulnerability factors over time (Lewinsohn et al., 1981). The idea is that heightened depression and anxiety symptoms could entrench sedentary lifestyles (e. g., physical inactivity, addictive behaviors that trade off short-term momentary pleasure or relief for long-term increased negative affect)

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(Bonnet et al., 2005), unhelpful beliefs, and construal biases, over long durations (Just et al., 2001). Consequently, scar theory argues that heightened depression and anxiety might chronically result in increased risk of diverse physical illness (Kessler et al., 2003; Stewart et al., 2009), and adversely affect close relationships (Newman and Zainal, 2020; Santini et al., 2020), thereby elevating risk for more future mental health problems. Based on the scar theory, it is thus plausible that avoidant coping strategies, such as excessive focus on and venting of emotion, denial, and behavioral disengagement that were not as chronically present prior to the disorders may emerge and persist even as symptoms abate over time.

In support of the scar model, five studies have shown that depression and anxiety disorders predicted future heightened use of avoidant and related dysfunctional coping strategies. For example, across 3 years, individuals with multiple sclerosis with increased depressive mood displayed significant decrements in active coping styles (Arnett and Randolph, 2006). Moreover, adolescents with elevated levels of depression and anxiety subsequently showed more reliance on disengagement, rumination, and social avoidance (Zimmer-Gembeck, 2015). Consistent with those findings, patients diagnosed with amyotrophic lateral sclerosis who had more symptoms of anxiety and depression utilized less active and positive mood-inducing coping strategies over time (Jakobsson Larsson, Nordin, and Nygren, 2016). Recently, a study showed that a 9-year increase in counts of MDD, GAD, and panic disorder diagnoses predicted larger future 9-year declines in goal persistence and positive appraisal (Zainal and Newman, 2019). Collectively, these studies support the idea that over long periods, depression and anxiety disorders render people vulnerable to using avoidant coping mechanisms in the future.

In the past 50 years, myriad coping theories have proposed that avoidant behavioral and maladaptive cognitive patterns predispose individuals to future MDD and GAD. The transactional model of stress and coping posits that avoidant or emotion-oriented coping strategies in response to controllable stressors (i.e., persistent attempts to evade problems or to deal with them indirectly) negatively impact the wellbeing of an individual across time (Lazarus and Folkman, 1984). Relatedly, the *learned helplessness theory* postulates that risk factors for MDD can include the tendency to avoid responsibilities, assume the worst, lack of initiative, prolonged inactivity, and reduced participation in meaningful pursuits (Maier and Seligman, 2016; Seligman and Ollendick, 1998). The response styles theory posits that rumination exacerbates depression by enhancing negative thinking and impairing problem solving and is correlated with higher levels of general anxiety; rumination is positively related to avoidant coping (Nolen-Hoeksema et al., 2008). Focus on and venting of emotion is similar to rumination, as both processes involve excessively attending to past events in non-constructive ways. Taken together, theories suggest that avoidant coping strategies (e.g., denial, focus on and venting of emotion, and behavioral disengagement) may present as risk factors of MDD and GAD.

Thus far, seven longitudinal studies have examined whether avoidant coping strategies preceded and predicted anxiety and depression disorders. One study tested if there was a positive relation between avoidant coping and anxiety or depression severity across 12 months among women undergoing fertility treatments (Verhaak et al., 2005); however, this study observed a null relation between avoidant coping and mental health symptoms. Conversely, supporting the stress-coping transactional model, chronically ill patients who used more avoidant coping were at higher risk for developing depression (Rabinowitz and Arnett, 2009). Similarly, avoidant coping was detrimental to the physical and psychological health of undergraduate students (Elliot et al., 2011); however, as this study was conducted over a 15-week period, it might not generalize across adulthood development. Likewise, an 18-month study indicated that increased trait-level passivity was related to a rise in depressive symptoms among breast cancer patients (Avis et al., 2013). Moreover, across four weeks, women with high anxiety sensitivity tended to utilize more avoidant coping mechanisms,

including disengagement, denial and suppression of competing activities (Sigmon et al., 2004). Further, lower acceptance toward life events and use of emotion- (vs. problem-) focused coping toward controllable stressors predicted more anxiety symptoms in Western undergraduates over time (Szabo et al., 2016). In addition, a recent 2-year longitudinal study noted that denial and avoidance was associated with higher future levels of anxiety and depression among dementia caregivers (del-Pi-no-Casado et al., 2019). On balance, the findings from these studies suggest that avoidant coping patterns would precede and forecast future depression and anxiety disorders.

So far, two studies have examined avoidant coping mechanisms as mediators of the prospective depression-anxiety relationship. In a fourwave study among children and adolescents, more anxiety symptoms significantly predicted greater depression severity over a decade later, and this anxiety-depression symptom relation was mediated by midtime-point avoidance patterns (Jacobson and Newman, 2014); however, the construct of avoidance was assessed with a one-item measure, which could yield unstable estimates. The present study built on the prior study by utilizing four-item measures of avoidant coping and recruited a sample of community adults to test if their pattern of findings could generalize across adulthood development. Another study observed that the bidirectional association between depression and anxiety symptoms across one year was either partially or fully mediated by trait rumination in adults (McLaughlin and Nolen-Hoeksema, 2011); despite that, their two-time-point mediation analyses could have yielded biased parameter estimates (Cole and Maxwell, 2003). The current study thus extended prior ones by conducting more rigorous mediation analyses across three waves of measurement (Maxwell and Cole, 2007), and investigating multiple maladaptive coping mechanisms as mediators across a longer timeframe of 18 years. In addition, the present study built on the literature by providing analyses at the disorder (vs. symptom) level. This is important as both disorder- and symptom-level information are necessary to guide the development of empirically-supported transdiagnostic treatments and optimize established disorder-specific protocol approaches (Everaert and Joormann, 2019; Spijker et al., 2020).

Building on the foregoing theories and existing data, the present study aimed to examine if avoidant coping strategies (i.e., focus on and venting of emotion, behavioral disengagement, denial) would mediate the 18-year relations between MDD and GAD in community adults. Based on theory and literature, we hypothesized that the 18-year bidirectional and positive association between MDD and GAD would be mediated by denial (Hypotheses 1 and 2), focus on and venting of emotion (Hypotheses 3 and 4), and behavioral disengagement (Hypotheses 5 and 6). Specifically, we predicted that elevated baseline MDD and GAD would forecast increased denial, focus on and venting of emotion, and behavioral disengagement 9 years later, above and beyond baseline disorder presence. Moreover, we expected that higher denial, focus on and venting of emotion, and behavioral disengagement would thereby independently predict presence of MDD and GAD following 9 years.

# 1. Method

#### 1.1. Participants

This present study used the publicly available Midlife Development in the United States (MIDUS) database, and measured data at three different waves, 1995–1996 (Time 1; T1), 2004–2005 (T2), and 2012–2013 (T3) (Brim et al., 2019; Ryff et al., 2017, 2019). Due to the publicly available nature of this dataset, the present study was exempt from Institutional Review Board (IRB) approval. Nonetheless, the original MIDUS investigators received IRB approval from the participating institutions. The 3294 adults in the sample used for this study participated in all three waves and were able to speak to our research question. At T1, the average age of participants was 45.62 years (range = 20–74, SD = 11.41), 54.61% of participants were female, and 42% were college educated. 89.01% of individuals identified as White, 3.25% African American, and 7.73% Native American, Asian, Pacific Islander or other.

#### 1.2. Measures

MDD and GAD were assessed via an in-person semi-structured clinical interview at T1 and T3. Coping strategies were measured using selfreport at T2.

## 1.2.1. Major depressive disorder

The Composite International Diagnostic Interview – Short Form (CIDI-SF; Kessler et al., 1998) aligned with the Diagnostic and Statistical Manual–Third Edition-Revised (DSM-III-R; American Psychiatric Association, 1980) criteria and was used to diagnose MDD at T1 and T3. Participants reported on any episodes experienced in the past 12 months. To determine overall severity, we summed depression symptoms from a binary response scale (0 = No and 1 = Yes) across 7-items (e. g., depressed mood, changes in appetite, fatigue, ideation of suicide, sleep disturbances; range = 0–7). For MDD, the specificity (93.9%) and sensitivity (89.6%) of the CIDI-SF were good. The CIDI-SF MDD severity scale also had strong internal consistency (T1 and T3 Cronbach's  $\alpha = 0.94$  herein) and good retest reliability (Wittchen, 1994).

# 1.2.2. Generalized anxiety disorder

The DSM-III-R-consistent CIDI-SF was also used to diagnose GAD at T1 and T3. Participants reported the degree to which they experienced symptoms as due to their worries for about half to most days over the past year on a 4-point Likert scale (0 = Never to 3 = Most days) (10-item scale; e.g., restlessness, feeling keyed up, irritability, trouble sleeping, trouble focusing; range = 0–10). For GAD, the CIDI-SF specificity (99.8%) and sensitivity (96.6%) were excellent. Further, the CIDI-SF GAD severity scale had good internal consistency ( $\alpha s = 0.80$  and 0.83 at T1 and T3, respectively herein) and strong retest reliability (Abel and Borkovec, 1995; Wittchen, 1994).

#### 1.2.3. Coping strategies

Table 1 presents all of the items used to measure coping strategies at T2, assessed with the COPE questionnaire (Carver et al., 1989). Participants rated items on a four-point Likert scale (1 = not at all; 2 = only a little; 3 = a medium amount; 4 = a lot) assessing trait-level denial, focus on and venting of emotion, and behavioral disengagement. Denial was measured with four items (e.g., "I say to myself 'This isn't real'"). The denial scale had good internal consistency ( $\alpha = 0.76$  in this study), strong two-week retest reliability, and good convergent and discriminant validity (Carver et al., 1989). Additionally, four items tapped into focus on and venting of emotion (e.g., "I feel a lot of emotional distress and find myself expressing those feelings a lot"). The 4-item focus on and

#### Table 1

Descriptive data and correlation matrix of study variables of interest.

venting of emotion scale has shown good internal consistency ( $\alpha = 0.82$  herein), acceptable two-week retest reliability, and strong construct validity (Carver et al., 1989). Likewise, a four-item scale captured behavioral disengagement (e.g., "I admit to myself that I can't deal with it and quit trying"). This scale had strong internal consistency ( $\alpha = 0.72$  in this study), good two-week retest reliability, and excellent construct validity (Carver et al., 1989).

### 1.3. Data analytic strategy

Longitudinal structural equation modeling analyses were conducted with the Lavaan package using RStudio software (Version 1.3.959). In terms of evaluating the fit of our various models, we utilized the confirmatory fit index (CFI) (Bentler, 1990; McDonald and Marsh, 1990), root mean square error of approximation (RMSEA) (Browne and Cudeck, 1993; Steiger, 1990), and standardized root mean square residual (SRMR) (Hu and Bentler, 1999). As the manifest indicators of the latent variables of coping strategies, MDD, and GAD were ordinal in nature. We performed all analyses using maximum likelihood with robust estimators to maximize power and reduce biases in the parameter estimates (Zhong and Yuan, 2011). Using a series of CFAs, the measurement models were found to have satisfactory to good fit ( $\gamma^2 = 2.952$ ) - 69.609, p < .770, CFI = 0.968-1.000, RMSEA = 0.000-0.127, SRMR = 0.008-0.034). Additionally, we observed statistically significant standardized factor loadings (all ps < 0.001) for the indicators of latent T1 MDD ( $\lambda s = 0.712-0.953$ ), T1 GAD ( $\lambda s = 0.558-0.756$ ), T2 focus on and venting of emotion ( $\lambda s = 0.686-0.812$ ), T2 denial ( $\lambda s = 0.551-0.775$ ), T2 behavioral disengagement ( $\lambda s = 0.572-0.685$ ), T3 MDD ( $\lambda s =$ 0.712–0.962), and T3 GAD ( $\lambda s = 0.606-0.755$ ).

A product-of-coefficients approach was utilized to conduct mediation analyses of the indirect effects for T1 MDD and T1 GAD predicting T2 avoidant coping (*a* path), and T2 avoidant coping predicting T3 GAD and T3 MDD severity (*b* path), beyond the indirect effect (*c* path). The unstandardized regression coefficients ( $\beta$ ) and standard errors were presented, and bootstrapping with 10,000 resampling draws was utilized (Cheung and Lau, 2008). The ratio of the indirect effect (a\*b) to the total effect was the mediation effect size, c = a\*b + c', which was expressed in the percentage variance that the T2 avoidant coping mediator accounted for in the bi-directional relation between MDD and GAD across T1 and T3 (Preacher and Kelley, 2011; Wen and Fan, 2015). For a robust analysis, we controlled for baseline symptoms in all of our models (Maxwell and Cole, 2007). In total, 0.1% of the data was missing, and was handled using full information maximum likelihood, the recommended approach (Graham, 2009).

	1	2	3	4	5	6	7
1 T2 FOAVE	-	-	-	-	-	-	-
2 T2 Denial	.95***	-	-	-	-	-	-
3 T2 BD	.92***	.94***	-	-	-	-	-
4 T1 MDD	.07***	.03	.03	-	-	-	-
5 T1 GAD	.05***	.04*	.03	.29***	-	-	-
6 T3 MDD	.05***	.02	.02	.28***	.18***	-	-
7 T3 GAD	.04*	.03	.02	.16***	.35***	.34***	-
Mean	5.30	1.99	2.82	0.69	0.14	0.60	0.13
SD	2.80	2.18	2.26	1.82	0.86	1.71	0.92
Minimum	0.00	0.00	0.00	0.00	0.00	0.00	0.00
Maximum	12.00	12.00	12.00	7.00	10.00	7.00	10.00
Skewness	0.42	1.25	0.58	2.44	7.47	2.69	7.89
Kurtosis	-0.28	1.44	0.09	4.35	60.84	5.74	65.66

Note. \*\*\* p < .001, \*\* p < .01, \* p < .05.BD = behavioral disengagement; FOAVE = focus on and venting of emotion; GAD = generalized anxiety disorder; MDD = major depressive disorder; SD = standard deviation.

# 2. Results

# 2.1. Latent mediation analyses

#### 2.1.1. T2 denial as a mediator

First, we tested the hypothesis that the relationship between T1 MDD and T3 GAD (and vice versa) would be mediated by T2 denial. Based on the practical fit indices, this mediation model showed good fit ( $\chi^2$  (*df* = 12) = 85.97, *p* < .001, CFI = 0.968, RMSEA = 0.056, SRMR = 0.032). Supporting Hypothesis 1, higher T1 MDD significantly predicted greater T2 denial ( $\beta$  = 0.102, *SE* = 0.034, *z* = 3.026, *p* = .002), and more T2 denial was significantly related to greater T3 GAD ( $\beta$  = 0.020, *SE* = 0.010, *z* = 2.065, *p* = .039). However, the indirect effect (T1 MDD  $\rightarrow$  T2 denial  $\rightarrow$  T3 GAD) was not statistically significant ( $\beta$  = 0.002, *SE* = 0.001, *z* = 1.729, *p* = .084). Thus, Hypothesis 1 was not supported.

Next, we examined if T2 denial would mediate the T1 GAD–T3 MDD relation. This T1 GAD  $\rightarrow$  T2 denial  $\rightarrow$  T3 MDD mediation model showed acceptable fit ( $\chi^2$  (df = 12) = 108.02, p < .001, CFI = 0.971, RMSEA = 0.052, SRMR = 0.025). T1 GAD was significantly associated with more T2 denial ( $\beta = 0.300$ , SE = 0.093, z = 3.209, p = .001), but T2 denial did not significantly predict T3 MDD ( $\beta = 0.019$ , SE = 0.015, z = 1.282, p = .200). Simultaneously, the indirect effect (T1 GAD  $\rightarrow$  T2 denial  $\rightarrow$  T3 MDD) was not significant ( $\beta = 0.006$ , SE = 0.005, z = 1.167, p = .243). Therefore, Hypothesis 2 was not supported.

# 2.1.2. T2 focus on and venting of emotion as a mediator

Next, we tested the hypothesis that the association between T1 MDD and T3 GAD (and vice versa) would be mediated by T2 focus on and venting of emotion. This mediation model had good fit ( $\chi^2$  (df = 12) = 137.18, p < .001, CFI = 0.974, RMSEA = 0.061, SRMR = 0.028). Supporting Hypothesis 3, higher T1 MDD significantly predicted greater T2

focus on and venting of emotion ( $\beta = 0.406$ , SE = 0.046, z = 8.857, p < .001), and more T2 focus on and venting of emotion was significantly related to greater T3 GAD ( $\beta = 0.017$ , SE = 0.005, z = 3.257, p = .001). Additionally, the indirect effect (T1 MDD  $\rightarrow$  T2 focus on and venting of emotion  $\rightarrow$  T3 GAD) was significant ( $\beta = 0.007$ , SE = 0.002, z = 2.996, p = .003). T2 focus on and venting of emotion accounted for 20.59% of the T1 MDD–T3 GAD association. Thus, Hypothesis 3 was supported.

The T1 GAD  $\rightarrow$  T2 focus on and venting of emotion  $\rightarrow$  T3 MDD mediation model displayed acceptable fit ( $\chi^2$  (df = 12) = 190.71, p < .001, CFI = 0.961, RMSEA = 0.072, SRMR = 0.051). Lending support to Hypothesis 4, T1 GAD was significantly associated with higher T2 focus on and venting of emotion ( $\beta = 0.484$ , SE = 0.097, z = 4.993, p < .001), and greater T2 focus on and venting of emotion significantly predicted higher T3 MDD severity ( $\beta = 0.048$ , SE = 0.010, z = 4.793, p < .001). Also, the indirect effect (T1 GAD  $\rightarrow$  T2 focus on and venting of emotion  $\rightarrow$  T3 MDD) was significant ( $\beta = 0.023$ , SE = 0.007, z = 3.390, p = .001). T2 focus on and venting of emotion explained 16.08% of the T1 GAD–T3 MDD relation. Thus hypothesis 4 was supported. Figs. 1 and 2 depict the regression path coefficients, standard errors, factor loadings, item error variances, and T1 MDD-GAD correlation, of the parameter estimates for Hypotheses 3 and 4.

# 2.1.3. T2 behavioral disengagement as a mediator

Following this, we examined the hypothesis that the T1 MDD–T3 GAD association (and vice versa) would be mediated by T2 behavioral disengagement. Based on the practical fit indices, this mediation model showed good fit ( $\chi^2$  (df = 12) = 22.39, p = .033, CFI = 0.997, RMSEA = 0.015, SRMR = 0.018). T1 MDD significantly predicted more T2 behavioral disengagement ( $\beta = 0.127$ , SE = 0.034, z = 3.753, p < .001), but T2 behavioral disengagement did not significantly relate to T3 GAD ( $\beta = 0.014$ , SE = 0.009, z = 1.574, p = .116). Additionally, the indirect



**Fig. 1.** Focus on Emotion and Venting as a Mediator of MDD Predicting GAD 18 Years Later. Note. \*\*\* p < .001; \*\* p < .01; \* p < .05. MDD = major depressive disorder; GAD = generalized anxiety disorder.



**Fig. 2.** Focus on Emotion and Venting as a Mediator of GAD Predicting MDD 18 Years Later. Note. \*\*\* p < .001; \*\* p < .01; \* p < .05. MDD = major depressive disorder; GAD = generalized anxiety disorder.

effect (T1 MDD  $\rightarrow$  T2 behavioral disengagement  $\rightarrow$  T3 GAD) was not significant ( $\beta = 0.002$ , SE = 0.001, z = 1.409, p = .159). Therefore, Hypothesis 5 was not supported.

Finally, we tested the hypothesis that behavioral disengagement would mediate the T1 GAD-T3 MDD relation. The T1 GAD  $\rightarrow$  T2 behavioral disengagement  $\rightarrow$  T3 MDD mediation model showed good fit ( $\chi^2$  (df = 12) = 28.03, p = .005, CFI = 0.994, RMSEA = 0.021, SRMR = 0.020). T1 GAD was significantly associated with higher T2 behavioral disengagement ( $\beta = 0.260$ , SE = 0.081, z = 3.223, p = .001), but T2 behavioral disengagement did not significantly predict T3 MDD ( $\beta = 0.027$ , SE = 0.014, z = 1.895, p = .058). Also, the indirect effect (T1 GAD  $\rightarrow$  T2 behavioral disengagement  $\rightarrow$  T3 MDD) was not significant ( $\beta = 0.007$ , SE = 0.005, z = 1.558, p = .119). Thus, Hypothesis 6 was not supported.

#### 3. Discussion

Results suggested that focus on and venting of emotion may be a mechanism by which GAD may lead to MDD 18 years later (and vice versa), accounting for 16.08% of the T1 GAD-T3 MDD association, and 20.59% of the T1 MDD-T3 GAD relation. Also, although T1 MDD and T1 GAD predicted higher T2 denial, and greater T2 denial thereby forecasted T3 GAD, denial did not mediate the 18-year bi-directional relations between MDD and GAD. Further, although T1 GAD and T1 MDD preceded and predicted greater T2 behavioral disengagement, T2 behavioral disengagement did not present as a risk factor for T3 MDD and GAD. Overall, our findings partially supported our hypotheses and the *transactional model of stress and coping* and *learned helplessness* theories (Lazarus and Folkman, 1984; Swanson et al., 2012), furthering the idea that GAD and MDD are linked to maladaptive coping behavior complex pattern of findings.

Why did greater T1 MDD and GAD render people more vulnerable to higher focus on and venting of emotion, denial, and behavioral disengagement at T2? In general, these findings lend credence to cognitive and behavioral scar theories which propose that experiences of psychopathology can reinforce poor habits, such as chronic rumination, avoidance, passivity, and adoption of limiting mindsets across long durations (Hong, 2007; Zainal and Newman, 2019). Stated differently, prior MDD and GAD might "scar" individuals and render them susceptible to future long term relapses via continual information processing biases, suboptimal lifestyles, dissatisfying social relationships, and associated factors (see empirical review by Burcusa and Iacono, 2007). Relatedly, it is plausible that people with MDD and GAD were inclined to rigidly avoid dealing with their problems instead of confronting them head-on by using a wide repertoire of coping approaches. This construal is aligned with data that whereas healthy controls tended to flexibly and optimally utilize myriad situation-specific coping mechanisms (approach, avoidance, escape), GAD- and MDD-disordered persons tended to rigidly choose avoidant coping approaches (Haskell et al., 2020). Denial, focus on and venting of emotion, and behavioral disengagement are also likely to be intrinsically linked to unhelpful self-referential repetitive thinking (i.e., focusing on negative feelings triggered by dwelling on past stressors or worrying excessively) common in MDD and GAD, as demonstrated in diverse samples (Jakobsson Larsson et al., 2016; Liu et al., 2020; Michl et al., 2013; Sumner et al., 2010). Consistent with this interpretation, depression and anxiety disorders were found to be associated with non-constructive repetitive negative thinking over 3 years (Spinhoven et al., 2018). Also, the finding that GAD predicted more focus on and venting of emotion might be related to pseudo-problem-solving tendencies in habitual worriers (Llera and Newman, 2020; Provencher et al., 2004). Upcoming

longitudinal studies can test these ideas.

Further, these results indicate that increased focus on and venting of emotion, but not denial or behavioral disengagement, present as a risk factor for future MDD. In addition, the data suggests that denial and focus on and venting of emotion, but not behavioral disengagement, may serve as predictors of subsequent GAD. These findings do not come as a surprise. Extensive cross-sectional research has documented strong relations between more frequent passive coping behaviors and elevated depression and anxiety across diverse cultures (e.g., North American, European, Asian samples; Marguerite et al., 2017; Suzuki et al., 2018). Further, focus on and venting of emotion can be viewed as part of cognitive and behavioral deficits repertoire across social and nonsocial domains (Ottenbreit et al., 2014). It may also relate to the tendency to avoid positive emotions as a negatively reinforcing way to experience temporary relief at the cost of prolonged, chronic, and increased negative affect; a feature consistently observed in clinical anxiety and depression (Buhk et al., 2020; Newman and Llera, 2011). Similarly, by venting emotions and avoiding addressing problems head-on, individuals tend to prolong stressful events, rather than actively working to find and implement effective, concrete, and actionable solutions to alleviate their mental health symptoms and prevent a downward spiral into anxiety and depression disorders. The excessive use of ruminative brooding akin to focus on and venting of emotion has been shown to be related to increased depression and anxiety in various clinical and community-dwelling adult samples (Lee et al., 2001; Michl et al., 2013; Pollard and Kennedy, 2007). Similar findings have been found in adolescents and children, with maladaptive coping (e.g., avoidance, denial), resulting in later higher levels of psychopathology symptoms (Compas et al., 2017). Future studies can test these speculations by further investigating the impact that focus on and venting of emotion and related problem-solving strategies can have on individuals' mental health at different life stages.

Several limitations deserve consideration. The sample used in the current study was comprised of predominately White, middle-to-upper income individuals. Future studies may test whether these patterns of findings extend to more diverse cultural and socio-economic backgrounds. Additionally, MDD and GAD were measured in this study using DSM-III-R instead of the latest DSM-5, which future replication efforts should utilize. Unmeasured third variables (e.g., gender differences in problem-solving and coping approaches; Sigmon et al., 2004) should also be considered in upcoming studies. Nonetheless, strengths of this study include its prospective design that allowed for the establishment of temporal precedence and evaluation of participants' disorder development over a longer time course than previous studies that used cross-sectional designs or shorter durations. Additionally, the present study used psychometrically reliable and valid measures for the purposes of assessing coping behaviors as well as gold standard clinical interviews to determine MDD and GAD diagnoses. Future studies could expand the age range of the sample to investigate the timeline of this relationship from childhood or adolescence to older adulthood. Such endeavors could offer more information on the long-term developmental ramifications of avoidant coping tendencies during youth.

Our findings present with several clinical implications. Interventions used to treat depression and anxiety disorders could target the coping mechanisms we studied, in particular, venting of emotion. By targeting venting, clinicians may be able to relieve both GAD and MDD symptoms more effectively, whereas focusing on remediating behavioral disengagement or denial may only impact symptoms of one or the other. For example, applied relaxation, problem-solving training, and cognitive therapy have been shown to be clinically effective in reducing symptoms of GAD and MDD (Beaudreau et al., 2019; Kirkham et al., 2015; Newman et al., 2020; Provencher et al., 2004). To this end, clinicians could apply the theories examined herein for persons with MDD and GAD by helping them to utilize active coping (e.g., exercising agency) versus passive coping toward life situations and vulnerabilities largely within their control.

#### Statement of ethics

This study was conducted in compliance with the American Psychological Association (APA) ethical standards in the treatment of human participants and approved by the institutional review board (IRB). Further, this research was conducted was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Informed consent was obtained from participants as per IRB requirements at Harvard University, Georgetown University, University of California at Los Angeles, and University of Wisconsin. Since this study used a publicly available dataset, it was exempt from IRB approval.

# CRediT authorship contribution statement

**Natalie S. Marr:** Data curation, Formal analysis, Methodology, Investigation, Funding acquisition, Writing – original draft, Writing – review & editing. **Nur Hani Zainal:** Conceptualization, Resources, Formal analysis, Methodology, Software, Formal analysis, Supervision, Data curation, Investigation, Funding acquisition, Writing – original draft, Writing – review & editing. **Michelle G. Newman:** Conceptualization, Resources, Formal analysis, Formal analysis, Supervision, Data curation, Methodology, Investigation, Funding acquisition, Writing – original draft, Writing – review & editing.

#### **Declaration of Competing Interest**

My research team, Ms. Zainal, Dr. Newman, and I, do not have any conflicts of interest in regards to the authorship, research, and/or publication of this article, and have enclosed the manuscript and tables in Microsoft word format. All authors are affiliated with PSU.

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