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Research paper

Social criticism moderates the relationship between anxiety and depression 10 years later

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ABSTRACT

Background: Research has consistently documented anxiety and depression as bidirectional risk factors for one another. However, little research investigates the sequential comorbidity of anxiety and depression over lengthy durations, and the influence of contextual variables on this relationship have not been fully empirically investigated.

Method: The current study examined perceived social criticism as a moderator of the relationship between a history of anxiety and a past 12-month depressive episode at least 10 years later (and vice versa) utilizing the National Comorbidity Survey Baseline (N = 8,098) and *Re*-interview data (N = 5,001). History of anxiety and depressive diagnoses were assigned at Wave 1, past year diagnosis at Wave 2, and perceived social criticism was assessed at Wave 1.

Results: Structural equation modeling indicated that when controlling for a Wave 1 latent depression factor, a positive relationship between Wave 1 latent anxiety and a Wave 2 latent depression emerged for those endorsing higher perceived social criticism from friends and relatives, respectively. Unexpectedly, when controlling for Wave 1 latent anxiety, a *negative* relationship between Wave 1 latent depression and Wave 2 latent anxiety emerged for those endorsing higher perceived social criticism from friends, but no relationship when moderated by perceived social criticism from relatives.

Limitations: Perceived social criticism was self-reported, which may introduce self-perception bias.

Conclusions: Results identified perceived social criticism as an important moderator in the sequential comorbidity of anxiety and depression over a long period of time.

Anxiety and depressive disorders are common, with lifetime prevalence estimates of 28.8% for anxiety disorders, and 16.6% for major depressive disorder (Kessler et al., 2005). Additionally, approximately 27–77% of those with a primary anxiety disorder diagnosis meet lifetime criteria for comorbid major depression, and about 75% of those diagnosed with major depression meet lifetime criteria for a comorbid anxiety disorder (Brown et al., 2001). Despite such comorbidity being associated with greater severity of psychiatric symptoms, poorer quality of life, poorer treatment outcomes and increased relapse rates (Brown et al., 1996; Keller et al., 1992; Norberg et al., 2008), the temporal dynamics of anxiety-depressive comorbidity have not been comprehensively investigated. A recent meta-analysis of longitudinal studies identified anxiety and depression as bidirectional risk factors for one another at the symptom and disorder level (Jacobson and Newman, 2017). However, only two studies have evaluated bidirectional relationships with a duration between assessments of a decade or more (Fichter et al., 2010; Kessler et al., 2008). It remains unclear how sequential anxiety-depressive diagnosis comorbidity functions over longer periods of time.

The two studies that examined anxiety-depression sequential comorbidity over a decade or more found significant bidirectional relationships between their respective measures of anxiety and depression (Fichter et al., 2010; Kessler et al., 2008). For example, Kessler et al. (2008) found that presence of a major depressive episode at baseline predicted subsequent onset of a diagnosis of GAD at a ten year follow-up, and vice versa. However, the GAD to subsequent major depressive episode relationship was slightly more likely than the reverse. Similar to Kessler et al.'s (2008) study, Fichter et al. (2010) documented bidirectional relationships between any pure anxiety syndrome and pure depression across 25 years, with the probability to shift

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from baseline pure anxiety to subsequent pure depression being greater than the reverse. These studies provide preliminary support that even over a decade or more, anxiety and depression as measured by diagnostic criteria or the equivalent are risk factors for one another. Although these studies controlled for individual-level risk factors that might influence longitudinal associations (e.g., childhood adversities, parental history of mental disorders, and respondent personality (Kessler et al., 2008); age and gender (Fichter et al., 2010)) they did not evaluate context variables (i.e., moderators) that might directly influence the strength or direction of these longitudinal relationships (Kraemer et al., 2008).

Context variables affecting the risk of developing a secondary anxiety or depression diagnosis in those with a prior episode of one of these disorders are inadequately understood. Not all individuals with a prior anxiety or depression diagnosis develop a secondary diagnosis, and context variables may clarify who is at higher risk. Understanding how risk factors function over longer periods of time presents unique opportunities for prevention. In particular, the importance of the social context in the bidirectional sequential comorbidity of anxiety and depression has been highlighted by findings that social support and perceived rejection moderated these relationships (Starr and Davila, 2012; Xu and Wei, 2013). It may be that the level of support or rejection from the interpersonal environment contributes to sequential comorbidity, such that an environment characterized by criticism contributes to greater risk.

Perceived social criticism (PSC) is one understudied environmental variable that research has documented as a predictor of depression. Specifically, previous research has established that PSC is positively associated with depression onset (Burkhouse et al., 2012; Peterson-Post et al., 2014) and relapse (Hooley and Teasdale, 1989). Diathesis-stress models suggest that psychological symptoms are the products of an interaction between an underlying vulnerability and life stress (Monroe and Simons, 1991). Similarly, the “diathesis-anxiety” model of anxiety-depression comorbidity suggests that pre-existing vulnerabilities interact with the stress of anxiety symptoms to produce depressive symptoms (Cohen et al., 2014). Perhaps PSC either causes stress that interacts with underlying vulnerabilities present in anxiety disorders, and/or exacerbates anxiety symptoms which interact with cognitive vulnerabilities to predict an increased risk for subsequent depression.

The relationship between PSC and the generation of anxiety is less well understood. Additionally, context variables influencing the relationship from depression to subsequent anxiety remain understudied, perhaps because previous research has emphasized the path from anxiety to subsequent depression. However, a correlated liabilities model may be one explanation for the temporal relation between depression and subsequent anxiety (Mathew et al., 2011). The correlated liabilities model suggests that depressive and anxiety symptoms have similar underlying vulnerabilities, such that the same set of vulnerabilities interact with life stress to predict anxiety and depression (Klein et al., 2003; Rice et al., 2004). Perhaps negative cognitive biases for social information present in both depression and anxiety enhance sensitivity to social cues, which interact with PSC to increase stress and subsequent risk for developing an anxiety disorder (Caouette and Guyer, 2016; Feldman and Downey, 1994; Olthuis et al., 2012).

It is widely believed that PSC is an important individual difference that predicts the long-term course of depression, yet few studies have investigated the impact of PSC beyond a time course of one year. One study failed to find an association between initial PSC ratings and depression relapse five and ten-years later in a sample of hospitalized patients (Krommüller et al., 2008); however, this study was limited by a lack of power. Another study found that PSC predicted depressive symptoms at five and ten-year follow-up in a community sample (Peterson-Post et al., 2014), suggesting that PSC may be an important individual difference for predicting depression course over longer periods of time. In addition to the gap in the literature regarding long-term PSC-depression relationships, the long-term PSC-anxiety relationship has not yet been explored. The present study incrementally addresses these gaps by examining PSC as a moderator of anxiety-depression sequential comorbidity over ten years in a nationally representative community sample.

A single daily diary study examined PSC and found it was not a significant moderator of the temporal associations between anxious and depressed moods (Starr and Davila, 2012). However, a meta-analysis suggests that discrete instances of social rejection may not elicit immediate distress, but continual or chronic perceived rejection predicts overall negative affect and low self-esteem (Blackhart et al., 2009). Therefore, PSC's impact as a moderator may emerge over longer periods of time, such that the cumulative effects of frequent PSC may increase risk for anxiety-depression comorbidity. Additionally, the study's participants were those diagnosed with GAD, limiting its generalizability to other anxiety symptoms and diagnoses. The present study tested the hypotheses that (1) presence of one or more lifetime anxiety diagnoses would predict past 12-month depressive diagnoses at a time point at least 10 years later, (2) presence of one or more lifetime depression diagnoses would predict past 12-month anxiety diagnoses at a time point at least 10 years later, and (3) PSC would strengthen these relationships. Given the importance of the social environment in the sequential comorbidity of anxiety and depression, PSC may be an important and understudied moderator of the temporal relationships between anxiety and depressive disorders, such that high PSC exacerbates the risk of developing subsequent anxiety or depression.

1. Method

1.2. Data sources

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. Ethics approval was not required for this study because it involved secondary data analyses of de-identified open access data and informed consent was obtained at the time of the original data collection.

Secondary data analysis was conducted utilizing the National Comorbidity Survey: Baseline 1990–1992 (NCS-1; Kessler et al., 1994) and the National Comorbidity Survey: Reinterview 2001–2002 (NCS-2). This epidemiological study employed a naturalistic, longitudinal design. Diagnostic interviews were administered with 8098 participants during Wave 1, and 5001 participants were reinterviewed 10 years later during Wave 2 (46.8% male; M age = 43.03 (SD = 10.48); 78.3% White, 10.3% African American, 8.3% Hispanic, 3.1% Other). NCS-2 interviews were administered to a probability subsample and weighted to adjust for differential probabilities of selection and non-response bias, including non-response bias based on NCS-1 diagnoses (Kessler et al., 1994).

During Wave 1, the Composite International Diagnostic Interview (CIDI) was administered by non-clinician trained interviewers (Robins et al., 1988). The CIDI has evidenced excellent interrater reliability (Wittchen et al., 1991), and high concordance with the Diagnostic and Statistical Manual of Mental Disorders, 3rd Edition, Revised (DSM-III-R; American Psychiatric Association, 1987) criteria for depressive disorders (K = 0.84), and anxiety disorders (K = 0.76; Janca et al., 1992). Wave 1 anxiety diagnoses included lifetime GAD, panic disorder, social phobia, and simple phobia, and Wave 1 depression diagnoses included lifetime dysthymia, and major depressive disorder (MDD).

Additionally, PSC was measured during Wave 1 with three items assessing frequency of friend-PSC (F-PSC), relative-PSC (R-PSC), and spousal-PSC (S-PSC) on a 4-point Likert scale from *always* (1) to *not at all* (4), respectively (e.g., “How often do [they] criticize you?”). These items were reverse coded, such that higher scores indicated higher

frequency of PSC. These items were part of a larger social support and conflict scale (Schuster et al., 1990; Walen and Lachman, 2000) that was used in the Midlife in the United States Survey (MIDUS). Data from the MIDUS study indicated good internal reliability of the total sale (Walen and Lachman, 2000). We conducted bivariate correlation analyses and an exploratory factor analysis (EFA) to investigate whether the PSC items could be examined as a general PSC factor or as single item indicators to best represent our construct of interest. Bivariate correlations indicated that S-PSC was only correlated to a small extent with F-PSC ($r = -0.20$), and R-PSC ($r = -0.15$). Additionally, an EFA demonstrated that S-PSC did not considerably contribute to a PSC factor with a factor loading of -0.31. Based on these analyses S-PSC did not represent our construct of interest, so we omitted it given the potential weaknesses of single-item measures. This lack of convergence may be expected as these items have not been analyzed as a subscale in previous research, and are conceptualized as single indicators in three separate social domains. With the exclusion of the S-PSC item there were no longer sufficient items to comprise a factor. Therefore, we decided to analyze these items separately as single item measures. Of note, the precedent in the literature is to measure PSC with a single item similar to the items used here (e.g., "How critical is your [spouse, friend, family] of you?" Hooley and Teasdale, 1989), which evidences high retest reliability (Chambless et al., 1999), and good convergent and discriminant validity (Chambless and Blake, 2009; Riso et al., 1996; White et al., 1998).

Between Wave 1 and Wave 2, the DSM-IV was published (American Psychiatric Association, 1994). Therefore, during Wave 2, the CIDI was utilized to assign DSM-IV diagnoses. The CIDI evidenced adequate to excellent concordance with DSM-IV anxiety and mood diagnoses (Reed et al., 1998). Wave 2 anxiety diagnoses included past 12-month GAD, panic disorder, social phobia, and simple phobia, and Wave 2 depression diagnoses included past 12-month dysthymia, and MDD. We chose to utilize past 12-month diagnoses for Wave 2 (instead of lifetime diagnosis) to preserve the temporal precedence of diagnoses at Wave 1.

1.3. Data analysis

Structural equation modeling (SEM) using the Mplus 8 software package with case weights applied (Kessler et al., 1994; Muthén and Muthén, 2017) was used to evaluate change in latent anxiety and depression variables. A latent approach was chosen because recent research developing an empirically-based classification of mental disorders emphasizes the importance of studying latent diagnoses given limitations of the current classification system, including the use of categorical diagnoses, limited reliability of diagnoses, hetereogeneity within diagnoses, and comorbidity across diagnoses (e.g., Kotov et al., 2017). Missing data at Wave 1 and Wave 2 were handled using full-information maximum likelihood, which is preferred over listwise deletion. Listwise deletion is well-documented to produce potentially biased parameter estimates under missing at random or missing not at random conditions. Full-information maximum likelihood produces better parameter estimates in the presence of missing data (Graham, 2009). Model fit was assessed based on standard fit indices, including the observed χ^2, Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and the Tucker-Lewis Index (TLI). Good fit is suggested to occur at RMSEA ≤ 0.05, CFI ≥ 0.95, and TLI ≥ 0.95, and acceptable fit is indicated when RMSEA ≤ 0.08, CFI ≥ 0.90, and TLI ≥ 0.90 (Brown, 2015; Marsh et al., 2004; Sharma et al., 2005).

First, we conducted a measurement model with Wave 1 lifetime GAD, panic disorder, social phobia, and simple phobia loading onto a latent Wave 1 anxiety factor, Wave 1 lifetime dysthymia and MDD loading onto a latent Wave 1 depression factor, Wave 2 past 12-month GAD, panic disorder, social phobia, and simple phobia loading onto a latent Wave 2 anxiety factor, and Wave 2 past 12-month dysthymia and MDD loading onto a latent Wave 2 depression factor. These latent constructs were formed in accordance with the latent structure found by Jacobson (2016) using the same dataset. All latent variables were allowed to correlate. Second, we conducted a baseline structural model with auto-correlation and cross-lag paths between Wave 1 and Wave 2 latent variables. Wave 1 anxiety and depression latent variables were allowed to correlate, as were the residuals of the Wave 2 anxiety and depression latent variables. This model tested whether Wave 1 anxiety predicted Wave 2 depression while controlling for Wave 1 depression, and whether Wave 1 depression predicted Wave 2 anxiety while controlling for Wave 1 anxiety.

For the final models, two latent interaction variables were created to test moderation. The indicators for the F-PSC x Anxiety latent interaction variable were computed by multiplying the Wave 1 anxiety

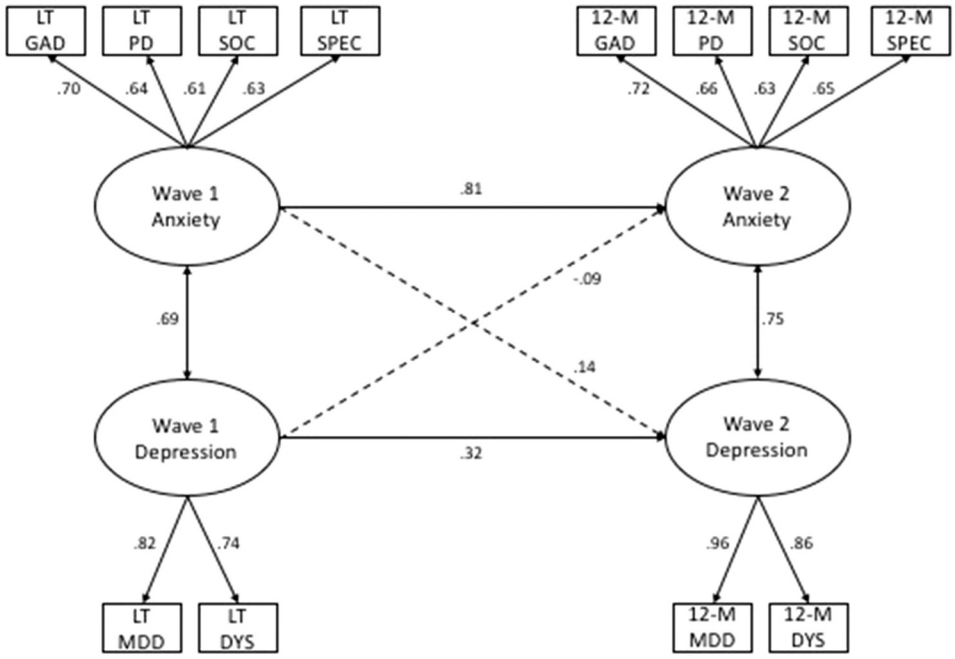

Fig. 1. This model depicts the cross-lag results where anxiety and depression at Wave 1 predict anxiety and depression at Wave 2. Solid lines represent significant relationships, whereas dotted lines represent nonsignificant relationships. LT = lifetime prevalence, 12-M = 12-month prevalence. GAD = generalized anxiety disorder, PD = panic disorder, SOC = social phobia, SPEC = specific phobia. MDD = major depressive disorder, DYS = dysthymia.

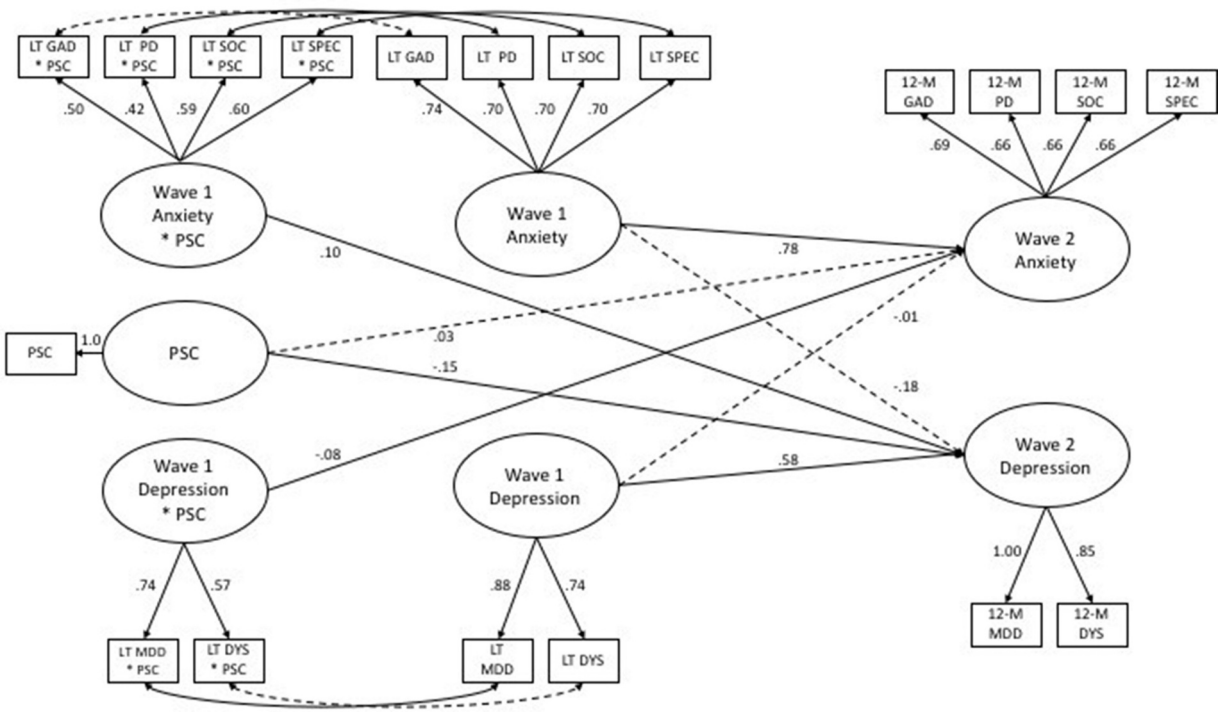

Fig. 2. This model depicts the moderation results with a latent interaction between Wave 1 anxiety and F-PSC predicting depression at Wave 2, and a latent interaction between Wave 1 depression and F-PSC predicting anxiety at Wave 2. Concurrent associations were modeled but are not depicted here for readability. Solid lines represent significant relationships, whereas dotted lines represent nonsignificant relationships. LT = lifetime prevalence, 12-M = 12-month prevalence. GAD = generalized anxiety disorder, PD = panic disorder, SOC = social phobia, SPEC = specific phobia. MDD = major depressive disorder, DYS = dysthymia. F-PSC = friend perceived social criticism.

indicators by F-PSC (mean-centered), and the indicators for the F-PSC x Depression latent interaction variable were computed by multiplying the Wave 1 depression indicators by F-PSC (mean-centered) and the Wave 1 depression indicators. The F-PSC variable and the two F-PSC interaction variables (with anxiety and depression) were then added as predictors of the Wave 2 anxiety and depression latent variables (Marsh et al., 2004). This model tested whether F-PSC moderated the relationship between Wave 1 anxiety and Wave 2 depression while controlling for Wave 1 depression, and whether F-PSC moderated the relationship between Wave 1 depression and Wave 2 anxiety while controlling for Wave 1 anxiety. Manifest variable interactions were allowed to correlate with the main effect residuals. An identical procedure was followed utilizing R-PSC in place of F-PSC.

2. Results

The measurement model exhibited acceptable fit (χ^2/df = 7.68; CFI = 0.94, TLI = 0.92; RMSEA = 0.04). Correlations between Wave 1 and Wave 2 depression and anxiety latent variables ranged from 0.42 (Wave 1 anxiety and Wave 2 depression) to 0.84 (Wave 2 depression and Wave 2 anxiety). The second model with the addition of cross-lag paths similarly exhibited acceptable fit (χ^2/df = 7.68; CFI = 0.94, TLI = 0.92; RMSEA = 0.04) and is depicted in Fig. 1. Wave 1 anxiety significantly predicted Wave 2 anxiety (b = 0.81, 95% CI [.69, 0.93], SE = 0.07, t = 11.07, p < .001), and Wave 1 depression significantly predicted Wave 2 depression (b = 0.33, 95% CI [.21, 0.44], SE = 0.07, t = 4.51, p < .001), as expected. However, contrary to our hypothesis, the path from Wave 1 anxiety to Wave 2 depression was not significant (b = 0.14, 95% CI [.01, 0.26], SE = 0.07, t = 1.84, p = .067), and the path from Wave 1 depression to Wave 2 anxiety was not significant (b = −0.09, 95% CI [−0.22, 0.03], SE = 0.08, t = 1.24, p = .215).

The F-PSC final moderation model exhibited acceptable fit (χ^2/df = 13.16; CFI = 0.96, TLI = 0.94; RMSEA = 0.05) and is depicted in Fig. 2. F-PSC significantly moderated the relationship between Wave 1 anxiety and Wave 2 depression when controlling for Wave 1 depression (b = 0.09, 95% CI [.04, 0.14], SE = 0.03, t = 3.10, p = .002), and between Wave 1 depression and Wave 2 anxiety when controlling for Wave 1 anxiety (b = −0.08, 95% CI [−0.12, −0.03], SE = 0.03, t = 2.81, p = .005). The moderation effects were such that for those with higher F-PSC at Wave 1, anxiety was positively associated with depression at Wave 2; however, for those with lower F-PSC, anxiety was not associated with Wave 2 depression. On the other hand, for participants exhibiting higher F-PSC at Wave 1, depression was negatively associated with Wave 2 anxiety, but depression was not associated with Wave 2 anxiety for those endorsing lower F-PSC.

The R-PSC final moderation model exhibited good fit (χ^2/df = 8.86; CFI = 0.97, TLI = 0.96; RMSEA = 0.04) and is depicted in Fig. 3. R-PSC significantly moderated the relationship between Wave 1 anxiety and Wave 2 depression when controlling for Wave 1 depression (b = 0.12, 95% CI [.04, 0.20], SE = 0.03, t = 3.91, p < .001), but not between Wave 1 depression and Wave 2 anxiety when controlling for Wave 1 anxiety (b = 0.005, 95% CI [−0.07, 0.08], SE = 0.03, t = 0.16, p = .869). The significant moderation effect was such that for those with higher R-PSC at Wave 1, anxiety was positively associated with depression at Wave 2; however, for those with lower R-PSC, anxiety was not associated with Wave 2 depression.[1]

[1] Results of the moderation models remained the same when controlling for age. The F-PSC moderation model with the addition of age at Wave 2 exhibited acceptable fit (χ2 (df = 67) = 2065.50; CFI = .96, TLI = .94; RMSEA = .05). F-PSC moderated the path from Wave 1 anxiety to Wave 2 depression when controlling for Wave 1 depression (b = .09, 95% CI [.05, .13], SE = .03, t = 3.46, p = .001), and from Wave 1 depression to Wave 2 anxiety when controlling for Wave 1 anxiety (b = −.07, 95% CI [−.11, −.02], SE = .03, t = 2.31, p = .021). The R-PSC moderation model with the addition of age at Wave 2 also exhibited acceptable fit (χ2 (df = 67) = 1504.96; CFI = .96, TLI = .95; RMSEA = .04). R-PSC moderated the path from Wave 1 anxiety to

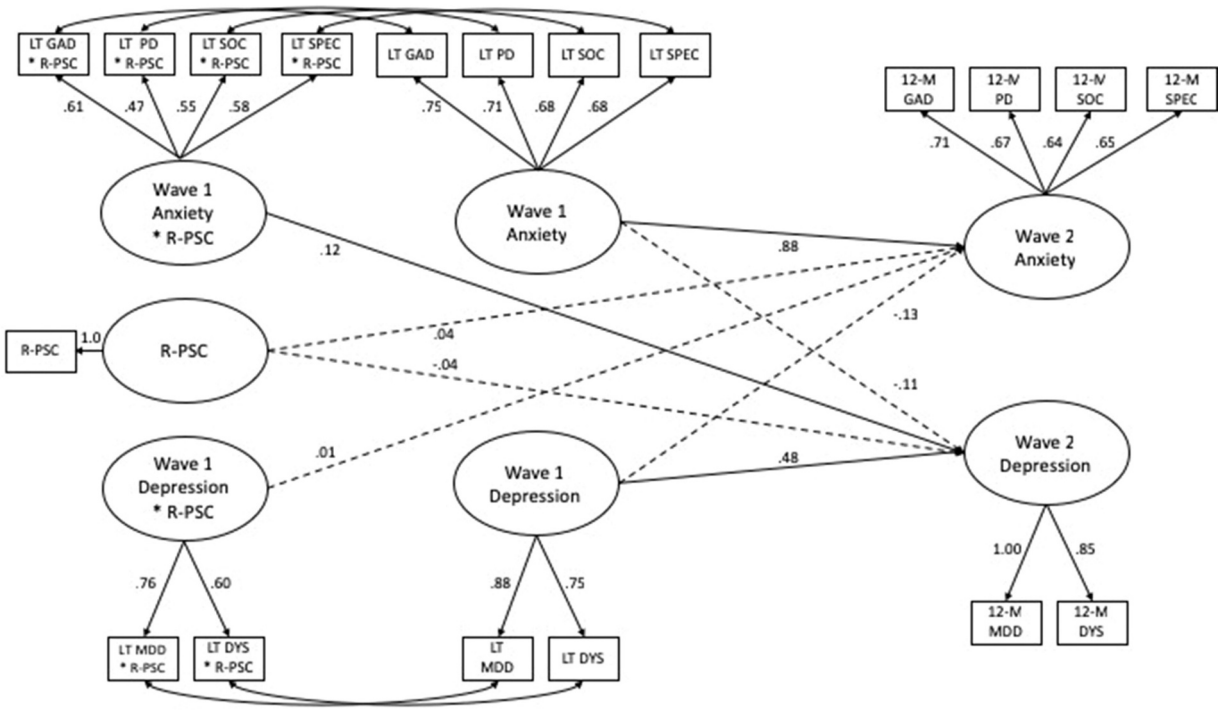

Fig. 3. This model depicts the moderation results with a latent interaction between Wave 1 anxiety and R-PSC predicting depression at Wave 2, and a latent interaction between Wave 1 depression and R-PSC predicting anxiety at Wave 2. Concurrent associations were modeled but are not depicted here for readability. Solid lines represent significant relationships, whereas dotted lines represent nonsignificant relationships. LT = lifetime prevalence, 12-M = 12-month prevalence. GAD = generalized anxiety disorder, PD = panic disorder, SOC = social phobia, SPEC = specific phobia. MDD = major depressive disorder, DYS = dysthymia. R-PSC = relative perceived social criticism.

In sum, hypotheses 1 and 2 were not supported as Wave 1 anxiety did not predict Wave 2 depression while controlling for Wave 1 depression, and vice versa, in the cross-lag model. Hypothesis 3 was partially supported, such that F-PSC and R-PSC increased the likelihood of someone with anxiety at Wave 1 developing a depressive diagnosis at Wave 2, but F-PSC decreased the likelihood of someone with depression at Wave 1 developing an anxiety disorder at Wave 2, while R-PSC had no impact.

3. Discussion

These findings highlight the importance of the social context in the sequential comorbidity of anxiety and depressive diagnoses. Results suggest PSC is an important moderator in the sequential comorbidity of anxiety and depression over a decade or more of time. The temporal relationships between Wave 1 and Wave 2 anxiety and depressive diagnoses were not apparent without considering PSC. It is somewhat surprising that these relationships were not statistically significant without the moderating effect of PSC. However, a meta-analysis did find that the strength of the sequential relationships between anxiety and depressive symptoms degraded as time between assessments increased (Jacobson and Newman, 2017). It may be that over longer periods of time direct relationships between anxiety and depression weaken and context variables become more important for predicting sequential comorbidity. It is essential then to consider context variables when investigating sequential comorbidity over longer periods of time.

A prior study utilizing the same data analyzed here found bidirectional sequential relationships between GAD and major depressive episode onset and persistence ten years later (Kessler et al., 2008). The lack of direct associations in the present study may be primarily due to differences in data analytic methods. The present study investigated latent variables composed of several anxiety or depressive diagnoses, which allowed us to determine paths between a general anxiety factor and general depressive factor, as opposed to between diagnoses. Analyzing latent variables may decrease inflation of relationships due to overlapping diagnostic criteria between GAD and mood disorders, such as sleep disturbance, appetite disturbance, restlessness, fatigue, and difficulty concentrating (Zbozinek et al., 2012). Investigating the long-term temporal relationships between specific diagnoses and symptom clusters as they are moderated by PSC is an avenue for future research.

Furthermore, these findings suggest that individuals with a history of anxiety who experienced higher levels of F-PSC or R-PSC were more at risk for meeting criteria for a depressive disorder over a decade later. It is difficult to say whether self-reported PSC is a true measure of a negative social environment or of biased perceptions of the social environment. It may be that higher PSC directly increases the risk of developing a depressive disorder (Burkhouse et al., 2012; Peterson and Smith, 2010; Peterson-Post et al., 2014). For example, frequency of PSC may be a proxy for a hostile social environment, which interacts with the diathesis underlying symptoms to produce more symptoms (Cohen et al., 2014; Hooley, 2004; Monroe and Simons, 1991). Alternatively, experiences of PSC may reflect negatively skewed perceptions of social cues, as is seen with rejection sensitivity (Feldman and Downey, 1994). Rejection sensitive individuals have been found to be more likely to perceive and internalize social criticism, leading to depression (Gilbert and Miles, 2000; Liu et al., 2014), or anxiety months after the criticism events (London et al., 2007). If criticism is perceived as hostile, it may activate and reinforce negative self-perceptions about the self, which may maintain or generate anxiety and/or depression symptoms (Sowislo and Orth, 2013). It is also plausible that the anxiety-

(*footnote continued*)
Wave 2 depression when controlling for Wave 1 depression (b = .13, 95% CI [.08, .17], SE = .03, t = 4.75, p < .001), but not the path from Wave 1 depression to Wave 2 anxiety when controlling for Wave 1 anxiety (b = .03, 95% CI [−.01, .07], SE = .02, t = 1.25, p = .211).

depression relationship is better captured as a moderated mediation model with rejection sensitivity as the mediator and PSC a moderator. Although this was not our focus, future research may investigate whether rejection sensitivity mediates the moderation we found here.

Given that PSC has been found to predict worse treatment outcomes for those with anxiety disorders, including persistently high anxiety and depression symptoms (Chambless et al., 2017; Chambless and Steketee, 1999; Renshaw et al., 2003; Steketee et al., 2007), and that interpersonal problems moderate the efficacy of treatment for anxiety, interventions that emphasize the social environment may play a role in preventing depressive disorders (Newman et al., 2017). In particular, evidence-based treatments that address interpersonal problems, biased information processing styles, and emotional processing avoidance may concurrently improve symptoms and the social environment resulting in maximum benefits (Newman et al., 2015; Newman et al., 2011). Finally, treatments may aim to decrease social criticism from others and increase social support or enhance relationship satisfaction; perhaps by involving friends and relatives in treatment.

These findings also suggest that those with a history of depression who experienced higher levels of F-PSC were at lower risk for meeting criteria for an anxiety disorder over a decade later. This relationship was not expected; however, it may be explained by the cognitive-affective crossfire theory. It is possible that those with depressive diagnoses found criticism cognitively validating, even if it was affectively unpleasant, because it confirmed pessimistic, global perceptions about the self (Swann et al., 1987). In fact, research on construal of interpersonal feedback found that positive feedback or compliments, increased anxiety in those with low self-esteem (Wood et al., 2005). Perhaps depressed individuals do not experience as much distress in response to PSC because critical feedback reifies their already negative schemas.

It is notable that only PSC from friends moderated the relationship between depression and later anxiety in this way, suggesting a differential impact of criticism from friends and family. These results challenge research that suggests PSC only matters or is only predictive of psychological distress when it comes from individuals in the home, such as family members (Renshaw, 2007). There are many possibilities for explaining this finding that cannot be definitively supported by these analyses. For example, it may be that criticism from friends is more salient or important to depressed individuals than that from their relatives. Future research should aim to collect collateral information about the importance of relationships and how meaningful criticism is from particular individuals.

4. Limitations

The novel findings of this study should be understood within the context of the study's limitations. First, two different versions of the DSM were utilized from Wave 1 to Wave 2. Second, psychiatric diagnoses were assigned by non-clinician administrators who may have been less accurate than clinicians. Third, diagnoses queried at Wave 1 were presence versus absence of any lifetime diagnosis and at Wave 2 were presence versus absence of any 12-month diagnosis. It is possible that the use of two different time periods affected our results. Also, our methods cannot speak to whether any of the episodes captured were new episodes versus recurring or chronic episodes. It may be that our use of a middle-aged sample inadvertently captured chronicity or multiple prior occurrences which might also increase the likelihood of future episodes. We examined this question indirectly by rerunning all analyses controlling for age, and our findings did not change. Nonetheless, future research should examine whether chronicity or multiple prior episodes increase the likelihood of these disorders predicting one-another across time. Relatedly, the sample was not specifically recruited at ages that align with ages-of-onset for depression and anxiety (Kessler at al., 2005). Future research should explore the moderating effect of PSC in a sample that is developmentally aligned with typical first onsets of depression and anxiety.

Additionally, it is likely that non-measured individual and environmental factors occurred in between Wave 1 and Wave 2 that may also have impacted the sequential comorbidity of anxiety-depression, including trait vulnerabilities or strengths, various environmental or life factors, the quantity and quality of social relationships, the onset of other psychological disorders, and engagement with psychological treatment. Given the nature of secondary data analysis, we were unable to investigate all of these variables in this particular study. It is notable that despite these limitations, PSC significantly moderated these relationships over a decade (or more). However, these associative findings must not be taken as causal.

Furthermore, the PSC items were analyzed as single indicators and only assessed criticism from friends and relatives, respectively. The use of single indicators may limit the reliability of these findings and it is possible that single items do not fully capture PSC as a multifaceted construct (Robins et al., 2001). Future studies should continue to investigate the role of criticism in friendships and family relationships as well as in romantic relationships, given that spousal relationships may be associated with dynamics of anxiety and depression (Jacobson et al., 2017). Additionally, corroborating measures of PSC from relationship partners were not available here, so it is unclear whether the frequency of perceived criticism was due to true elevations in criticism, perhaps related to symptom manifestation, or whether it was due to sensitivity to criticism resulting in skewed perceptions of social cues. Similarly, it is unclear whether the absence of criticism indicates better relationships *or* a lack of social interaction generally, which is notable given the role of avoidance in the relationship between anxiety and depression (Jacobson and Newman, 2014). Future research may also include a measure of criticism from a significant other to corroborate participant reports or account for general levels of socializing. Finally, we analyzed PSC as a moderator; however, it is possible that PSC also functions as a mediator, which is an avenue of future research.

5. Conclusions

The present study identified PSC as an important moderator of the sequential comorbidity of anxiety and depressive disorders. In fact, the prospective relationships between Wave 1 and Wave 2 anxiety and depressive disorders were only significant in the context of PSC. This may suggest that interventions aiming to improve the social environment either by directly changing the social environment or by altering biased perceptions of social feedback, particularly for those diagnosed with anxiety disorders, may not only reduce anxiety but also reduce the likelihood of later depressive disorders. Future research should investigate these relationships over decades of time, and continue to attempt to isolate the unique substantive contributions of social context variables, such as the process of interpreting and responding to social criticism, in anxiety-depression sequential comorbidity.

Author statement contributors

K.A.L. and N.C.J. developed the study concept under the supervision of M.G.N. N.C.J., M.K.S., and K.A.L. performed the data analyses and interpretation under the supervision of M.G.N. K.A.L. drafted the paper, and N.C.J., M.K.S., and M.G.N. provided critical revisions. All authors approved the final version of the paper for submission.

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The data that support the findings of this study are available from the Inter-university Consortium for Political and Social Research at https://www.icpsr.umich.edu/icpsrweb/. These data were derived from the following resources available in the public domain: https://www.icpsr.umich.edu/icpsrweb/ICPSR/series/527.

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Declaration of Competing Interest

None.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jad.2020.05.030.

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