

# Anxiety and Depression as Bidirectional Risk Factors for One Another: A Meta-Analysis of Longitudinal Studies

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Not only do anxiety and depression diagnoses tend to co-occur, but their symptoms are highly correlated. Although a plethora of research has examined longitudinal associations between anxiety and depression, these data have not yet been effectively synthesized. To address this need, the current study undertook a systematic review and meta-analysis of 66 studies involving 88,336 persons examining the prospective relationship between anxiety and depression at both symptom and disorder levels. Using mixed-effect models, results suggested that all types of anxiety symptoms predicted later depressive symptoms ( $r = .34$ ), and all types of depressive symptoms predicted later anxiety symptoms ( $r = .31$ ). Although anxiety symptoms more strongly predicted depressive symptoms than vice versa, the difference in effect size for this analysis was very small and likely not clinically meaningful. Additionally, all types of diagnosed anxiety disorders predicted all types of later depressive disorders ( $OR = 2.77$ ), and all depressive disorders predicted later anxiety disorders ( $OR = 2.73$ ). Most anxiety and depressive disorders predicted each other with similar degrees of strength, but depressive disorders more strongly predicted social anxiety disorder ( $OR = 6.05$ ) and specific phobia ( $OR = 2.93$ ) than vice versa. Contrary to conclusions of prior reviews, our findings suggest that depressive disorders may be prodromes for social and specific phobia, whereas other anxiety and depressive disorders are bidirectional risk factors for one another.

### Public Significance Statement

This meta-analysis suggests that anxiety and depression symptoms bidirectionally prospectively predict one another with moderate and similar strength. These prospective bidirectional relationships are stronger over shorter time periods and weaken over longer time periods. Most anxiety and depressive disorders significantly bidirectionally prospectively predicted one another with strong nonsignificantly different strength. However, depressive disorders more strongly predicted social and specific phobia than vice versa, suggesting that depressive disorders may be prodromes for social and specific phobia.

**Keywords:** anxiety, depression, mood, risk factor, prodrome

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Approximately one fifth of the population suffers from an anxiety or depressive disorder in any given year based on a recent meta-analysis evaluating global prevalence rates (Steel et al., 2014). Moreover, within their lifetimes, 49% to 81% of persons with a depressive disorder have met diagnostic criteria for an anxiety disorder, and 47% to 88% of those with an anxiety disorder have met criteria for a depressive disorder (de Graaf, Bijl, Spijker, Beekman, & Vollebergh, 2003; Hek et al., 2011; Lamers et al., 2011; Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998). Compared with pure diagnoses, developing a depressive disorder after

an anxiety disorder is associated with lower quality social relationships (Clancy, Noyes, Hoenk, & Slymen, 1978), greater depression severity (Breier, Charney, & Heninger, 1984; Clancy et al., 1978; Kessler et al., 1998; Lesser et al., 1988), chronicity (Geller, Chestnut, Miller, Price, & Yates, 1985), interference (Kessler, Stang, Wittchen, Stein, & Walters, 1999), impairment (Kessler et al., 1999), recurrence (Kessler et al., 1999), and persistence (Kessler et al., 2008; Lesser et al., 1988). Given the impact of such sequential comorbidity, it is important to better understand the longitudinal relationship between anxious and depressive disorders.

With the advent of factor analysis (a statistical technique aimed at reducing the number of constructs), cross-sectional studies began to find that anxiety and depression both loaded onto a single higher order factor (e.g., onto a “social adaptability” factor; Fiske, 1949). This led to the suggestion that anxiety and depression were part of the same underlying construct. Although these models may collapse longitudinal relationships between these constructs into a single factor (Allport & Odbert, 1936), such cross-sectional factor

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analytic findings have been largely emphasized and continue to represent a major theoretical conceptualization of anxiety and depression to date (e.g., see discussions of neuroticism, negative affect, and hierarchical models of anxiety and depression; Clark & Watson, 1991; McCrae & John, 1992; Mineka, Watson, & Clark, 1998; Watson, 2005, 2009). For example, the tripartite model suggests that although anxiety is uniquely characterized by high arousal and depression is uniquely characterized by low positive affect, anxiety and depression are both components of negative affect (Brown, Chorpita, & Barlow, 1998; Clark & Watson, 1991). Subsequent studies examined the validity of combining anxiety and depression into the same construct compared to modeling anxiety and depression as separate constructs.<sup>1</sup> Some research has supported hierarchical models (Brown, Chorpita, & Barlow, 1998; Chorpita, 2002; Henry & Crawford, 2005; Norton, Cosco, Doyle, Done, & Sacker, 2013). Other research has found that such hierarchical models did not fit the data well or did not fit it as well as models specifying anxiety and depression as distinct constructs (Boschen & Oei, 2006; Buckby, Cotton, Cosgrave, Killackey, & Yung, 2008; Clara, Cox, & Enns, 2001; Crawford & Henry, 2003; den Hollander-Gijsman, de Beurs, van der Wee, van Rood, & Zitman, 2010; Jacobson, 2016; Lovibond & Lovibond, 1995; Ollendick, Seligman, Goza, Byrd, & Singh, 2003; Yufik & Simms, 2010). Although there is somewhat mixed support for whether anxiety and depression load onto a higher-order factor within cross-sectional data, this literature does tend to show that there appears to be high associations between anxiety and depression.

There have been several early clinical observations and theories about the longitudinal relationship between anxiety and depression. For example, Darwin recounted how, following a major stressor, anxiety produced a state of helplessness followed by depression (Darwin, 1873). This laid the foundation for a later model that perceived helplessness is likely to develop into a state of hopelessness because of the expectation of powerlessness to solve internal, stable, and global deficits. The model further posits that hopelessness leads to depressive symptoms as a result of despair and perceived loss (Alloy, Kelly, Mineka, & Clements, 1990). Likewise, Freud observed that high anxiety led to higher generalized inhibition, later producing depression (Freud, 1936/1961). Such a view is consistent with findings that reactivity to anxiety via avoidance might lead to depression (Jacobson & Newman, 2014). Similarly, Jung (1916) described a patient who, following a state of great fear, began to punish herself, developing feelings of guilt and leading to a state of depression. This is similar to later views that depression often results from a demoralization reaction to broad impairments caused by anxiety (Belzer & Schneier, 2004; Cloninger, Martin, Guze, & Clayton, 1990; Denollet, Strik, Lousberg, & Honig, 2006; Frank, 1974). Klein (1948) theorized that anxiety often leads to depression via aggression turned inward toward the self. Internalized aggression has been more recently linked to generalized anxiety disorder, posttraumatic stress disorder, and major depression (Deschênes, Dugas, Fracalanza, & Koerner, 2012; Riley, Treiber, & Woods, 1989). At the same time, others have theorized that depression results in later anxiety. Schmideberg (1935) noted how depressive guilt, through internalized aggression, transforms into anxiety over time. Beard (1969) described how depression leads to the later development of anxiety as the depression forces one to confront one's fear of imminent death. Fear of death has been shown to uniquely predict

later anxiety, and not later depression or demoralization (Vehling et al., 2011).

One of the leading overarching theories, the prodromal theory (Clark & Watson, 1991), encompasses all of the unidirectional models described above. Consistent with others (Kessler et al., 2008; Perugi et al., 1998), we define a prodrome as a prior manifestation of a construct preceding and predicting its onset with greater strength than the construct predicts its prior manifestation (Perez-Edgar & Guyer, 2014). Also, a prodrome includes both a prior manifestation contributing to the first onset as well as later symptom elevation contributing to relapse (e.g., Bauer et al., 2006; Birchwood & Spencer, 2001; Herz & Melville, 1980; Lauriello, Horan, & Bustillo, 2002; Malla & Norman, 1994; Morriss, 2004; Ryu, Song, Ha, Ha, & Cho, 2012). Importantly, the prodromal model suggests that anxiety is a unidirectional predictor of later depression. Because a prodromal theory of anxiety and depression requires a stronger unidirectional relationship of one construct predicting another, this theory would not be supported if anxiety and depression both significantly predicted one another at later times and with similar magnitudes. In this case, they would be bidirectional risk factors for one another.

Differentiating between whether a prodromal or a bidirectional risk factor relationship better captures the longitudinal relationship between anxiety and depression is important. Prodromal theories would suggest that anxiety and depression are essentially one underlying construct (i.e., part of the same underlying disease process; Kuerbis, Hagman, & Morgenstern, 2013; Larson, Walker, & Compton, 2010). In contrast, if anxiety and depression are bidirectional risk factors for one another, this would suggest either (a) anxiety and depression arise from a single disease process predicting aspects of itself over time, or (b) anxiety and depression have distinctions from one another and can be meaningfully inter-related over time.

Examining prodromal and bidirectional theories also has important implications for practical prevention efforts. If anxiety were a prodrome for depression, it would suggest that prevention efforts should attempt to monitor and treat early manifestations of anxiety, rather than waiting for depression to develop. Moreover, efforts to prevent early manifestations of anxiety might effectively also prevent manifestations of depression. In contrast, if anxiety and depression were bidirectional risk factors for one another, primary prevention strategies should target both anxiety and depression, whichever one comes first or both, as either alone would increase the risk of the other disorder occurring at a later time.

<sup>1</sup> The current review evaluates factor analyses based on research that reports practical fit indices, which are essential to determining whether models examining hierarchical structure of affect fit the data well, based on cutoff values associated with simulation studies (Hu & Bentler, 1998). Note that requiring these fit indices resulted in the exclusion of some papers that claimed to show support for hierarchical models of anxiety and depression (Krueger, 1999; Slade & Watson, 2006; Watson, 2005), and some that claimed to support specificity between anxiety and depression (Antony, Bieling, Cox, Enns, & Swinson, 1998; Bedford, Lukic, Allershand, & Deary, 2011; Brown, Chorpita, Korotitsch, & Barlow, 1997). This also includes papers that have claimed to find support for hierarchical models (e.g., Caspi et al., 2014), but fit indices presented within the results section suggested that these models actually provided poor fit to the data (Hu & Bentler, 1998).

Many individual studies have tested whether this relationship was unidirectional or bidirectional. Some concluded that anxiety was a unidirectional risk factor for depression (i.e., prodrome; e.g., Hettema, Kuhn, Prescott, & Kendler, 2006; Merikangas et al., 2003; Rice, van den Bree, & Thapar, 2004), whereas others concluded that depression also predicted later anxiety (i.e., bidirectional risk factors; e.g., Fichter, Quadflieg, Fischer, & Kohlboeck, 2010; Silberg, Rutter, & Eaves, 2001). Thus, findings have been mixed in this regard.

Seven qualitative nonsystematic review papers to date have synthesized such evidence, and each review concluded that anxiety was a prodrome for later depression (Alloy et al., 1990; Andover, Izzo, & Kelly, 2011; Fava & Tossani, 2007; Horn & Wuyek, 2010; Moras & Barlow, 1992; Schleider, Krause, & Gillham, 2014; Wittchen, Beesdo, Bittner, & Goodwin, 2003). Perhaps because of entrenched beliefs about anxiety unidirectionally predicting later depression, no review has evaluated the evidence of depression predicting later anxiety. As a test of the prodromal theory requires an examination of the bidirectional relationship between anxiety and depression, these reviews were fundamentally unable to test this theory.

Additionally, prior reviews of this literature only included a small subset of the studies that exist currently. For example, one of the most cited review papers is from 1990 (Alloy et al., 1990), but most studies on this topic have been published within the past two decades. Other review papers only included five (Moras & Barlow, 1992), six (Fava & Tossani, 2007; Wittchen, Beesdo, et al., 2003), 11 (Horn & Wuyek, 2010), 12 (Schleider et al., 2014), and 16 (Andover et al., 2011) studies examining the naturalistic longitudinal relationship between anxiety and depression.

Given that review studies have been qualitative and nonsystematic, have only examined the unidirectional relationship between anxiety and depression, have not included the majority of existing longitudinal studies, and individual studies have arrived at contradictory conclusions, a quantitative review was needed to synthesize the large number of findings and to examine study differences that may have impacted outcomes. Thus, we sought to quantitatively synthesize the prospective research on associations between anxiety and depression.

The current meta-analysis included studies that measured both symptoms and disorders of anxiety and depression. Symptom measures were included because anxiety and depression have been shown to fall along a continuum (Kollman, Brown, Liverant, & Hofmann, 2006; Olatunji, Broman-Fulks, Bergman, Green, & Zlomke, 2010; Ruscio, Borkovec, & Ruscio, 2001; Ruscio & Ruscio, 2000, 2002; Ruscio, Ruscio, & Keane, 2004), and dimensional measures tend to have greater power (Bjelland et al., 2009). Clinical diagnoses were examined separately as they may be more directly meaningful to clinicians and better reflect substantial distress and impairment (Helzer, van den Brink, & Guth, 2006; Kamphuis & Noordhof, 2009). By including both symptoms and disorders, we attempted to capitalize on the strengths of each.

Given differing conclusions reached by prior studies (i.e., some studies suggesting unidirectionality, other studies suggesting bidirectionality; Fichter et al., 2010; Hettema et al., 2006; Merikangas et al., 2003; e.g., Rice et al., 2004; Silberg, Rutter, & Eaves, 2001), another major goal of this meta-analysis was to examine factors that moderated the prospective strength of anxiety and depression predicting one another. In particular, we considered demographic

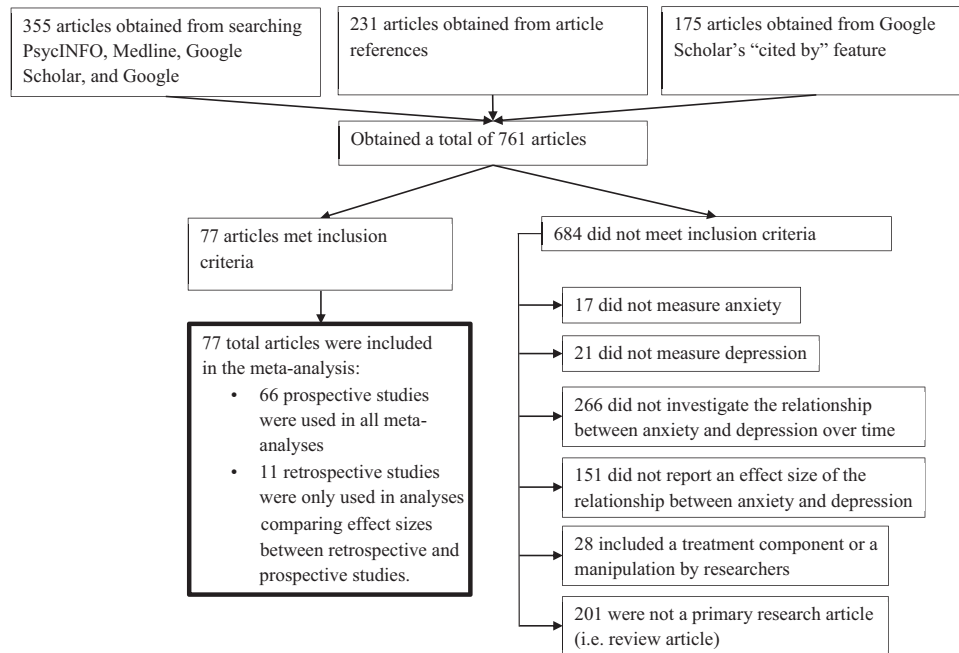
characteristics (i.e., gender, age, race, targeted population) to determine whether the strength of associations was influenced by the section of the population being studied, and, consequently, whether these models were specific to certain demographic populations or more widely applicable. Given their unique functional relationships (Nandi, Beard, & Galea, 2009), we also examined whether separate anxiety and depression constructs (e.g., worry vs. panic, generalized anxiety vs. obsessive-compulsive disorder) impacted the different associations. To determine the longevity of these associations, we examined whether lag time between measurements moderated the associations. Lastly, we examined other assessment features, such as type of measurement (e.g., self-report, independent observer report), recall period (e.g., six months, two years ago), and the diagnostic and statistical manual version.

Thus, this systematic meta-analysis analyzed prospective studies to determine (a) the strength of anxiety symptoms predicting later depressive symptoms, (b) the strength of depressive symptoms predicting later anxiety symptoms, (c) the strength of anxiety symptoms predicting later depressive symptoms compared to the strength of depressive symptoms predicting later anxiety symptoms, (d) the strength of anxiety disorders predicting later depressive disorders, (e) the strength of depressive disorders predicting later anxiety disorders, and (f) the strength of anxiety disorders predicting later depressive disorders compared to the strength of depressive disorders predicting later anxiety disorders. In addition, we examined moderators to each of these relationships.

## Method

### Obtaining and Selecting Studies

We searched four databases: PsycINFO, MEDLINE, Google, and Google Scholar. PsycINFO and MEDLINE are common databases for meta-analyses within psychology and were determined to be appropriate and useful sources (Rothstein, Sutton, & Borenstein, 2006). Additionally, Google Scholar was used to find articles typically thought to be outside the domain of psychology itself, but that contained data relevant to the current study. Moreover, Google was used to obtain both unpublished and published materials. In an attempt to be inclusive of unpublished material, we also incorporated unpublished doctoral theses. The following keyword search terms were used: *anxi\**, *posttraumatic stress*, *posttraumatic stress*, *phobia*, *obsessive-compulsive*, *depress\**, *dysphor\**, *dysthym\**, *temporal\**, *anteced\**, *sequen\**, *chronol\**, *predat\**, *antedat\**, and *preced\** (\* denotes a wildcard search). Note that all combinations of anxiety, depression, and temporal were used to create separate searches. Thus, we conducted a total of 105 searches within each database. Articles were selected based on their abstracts, and articles that mentioned a potential time relationship between anxiety and depression were selected. Using these search terms, we searched through 16,098 abstracts (5,763 from PsycINFO; 2,487 for Medline; 3,372 for Google Scholar; and 4,476 for Google). After obtaining these articles, we read each article and examined the reference section of each article in an attempt to find other related articles (see Figure 1). In addition, we used Google Scholar's "cited by" feature and examined all citations of the articles we obtained. Study searches continued until May, 2015.



*Figure 1.* This flow diagram depicts the source in which articles were selected, which met inclusion criteria, and which met exclusion criteria.

To qualify for the meta-analysis, the articles needed to meet the following inclusion requirements: (a) the study measured anxiety or anxiety disorders, (b) the study measured depression or depressive disorders, (c) the studies investigated the relationship between anxiety and depression over time (i.e., there was a time difference between the measurement of anxiety and depression either via prospective design or retrospective report; did not examine concurrent time periods),<sup>2</sup> (d) the study reported an effect size (or variables that could be converted to an effect size) of the relationship between anxiety and depression, (e) the study did not include a treatment component or any manipulation by researchers to affect the naturalistic course of anxiety or depression, and (f) the study was a primary research article (i.e., not a review article). See supplemental materials for a list of all obtained articles that were excluded and the reasons for their exclusion. This resulted in 77 studies (66 prospective studies used in all analyses and 11 retrospective studies used only in sensitivity analyses; 74 published; 3 unpublished [2 unpublished doctoral dissertations; and 1 unpublished manuscript]) being selected (see Figure 1).

## Coding

**Raters.** A team of eight persons coded the articles. Two or more raters coded all articles to establish interrater reliability. All conflicts between raters were resolved by all raters who had coded the article. See results section for interrater reliability.

**Study characteristics.** First, the research team coded sample size. Second, the team coded the targeted population, including (a) general population (i.e., not a clinical sample or did not measure clinical diagnoses), (b) clinical population (i.e., sampled from inpatients or outpatient services), (c) persons at risk for a specific disorder (i.e., subthreshold but elevated symptoms), (d) low risk

sample (i.e., a sample with low levels of symptoms and also did not meet criteria for a given disorder), (e) pregnant women, (f) mixed population (i.e., one or more other categories included), or (g) other population. The team also coded mean age, percentage of females, and percentage of Caucasians (based on an overview of the studies, it did not appear that examining the continuum of other racial groups would be possible due to too few articles examining them). We also coded the percentage of the sample with anxious and depressive disorders at baseline.<sup>3</sup>

In relation to anxiety symptoms, we coded the measurement focus, including (a) physiological aspects of anxiety (e.g., anxious arousal from the Mood and Anxiety Symptom Questionnaire), (b) cognitive aspects of anxiety (e.g., Brief Measure of Worry Severity; Penn State Worry Questionnaire), (c) non-disorder-specific anxiety (e.g., Spielberger State-Trait Anxiety Inventory; Anxiety Scale from the Depression Anxiety Stress Scale; Spence Children's Anxiety Scale), (d) generalized anxiety disorder symptoms (e.g., Generalized Anxiety Disorder Questionnaire—IV), (e) post-traumatic stress disorder symptoms (e.g., Child Posttraumatic Stress Disorder Checklist; Child PTSD Symptom Scale; Posttrau-

<sup>2</sup> Note that only prospective designs were used for the analyses, but we included retrospective methods to determine if these reports had substantially different effect sizes.

<sup>3</sup> We decided to utilize percentage of those with an anxiety or depressive disorder at baseline for several reasons. Although this measure reflects presence versus absence of clinical levels of psychopathology for a single individual, for an entire sample, using percentage with a given diagnosis, represents the degree of clinically significant anxiety and/or depression within that sample. This metric was important in determining whether samples with higher rates of an anxiety or depressive disorder had unique functional relationships between anxiety and later depression, compared with samples without any clinical diagnoses or lower rates.



matic Stress Disorder Checklist), (f) panic disorder symptoms (e.g., Structured Psychopathological Interview and Rating of the Social Consequences of Psychic Disturbances for Epidemiology), (g) social anxiety disorder symptoms (e.g., Liebowitz Social Anxiety Scale, The Social Anxiety Scale for Adolescents [SAS-A]), (h) specific phobia symptoms (e.g., Structured Psychopathological Interview and Rating of the Social Consequences of Psychic Disturbances for Epidemiology), or (i) other.<sup>4</sup> For depressive symptoms, coding included (a) general depression (e.g., Beck Depression Inventory, Center for Epidemiologic Studies Depression Scale), or (b) other.<sup>5</sup>

With respect to the diagnosis, we coded whether the study measured (a) mixed anxiety disorders, (b) obsessive–compulsive disorder (OCD), (c) generalized anxiety disorder (GAD), (d) post-traumatic stress disorder (PTSD), (e) panic disorder, (f) social phobia, (g) specific phobia, (h) mixed depressive disorders, (i) major depressive disorder (MDD), (j) dysthymia, or (k) other (i.e., overanxious anxiety disorder, separation anxiety disorder). Likewise, when diagnoses were included, we coded the diagnostic system used (i.e., *Diagnostic and Statistical Manual of Mental Disorders*, third edition [DSM–III], *Diagnostic and Statistical Manual of Mental Disorders*, third edition revised [DSM–III–R], *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition [DSM–IV]; American Psychiatric Association, 1980, 1987, 2000). For both anxiety and depressive disorders, we also coded the time reference period during which participants were asked to rate their anxiety or depressive symptoms (i.e., “Over the past year, have you had a period of at least 6 months when you experienced uncontrollable and excessive worry most of the day nearly every day?”), using the following categories (1) *less than 1 year*, (2) *1 to 2 years*, and (3) *greater than 2 years*. Note the time reference period should not be considered a proxy for chronicity, as these periods measured whether a person met diagnostic criteria at any point in time within this interval.

Furthermore, we coded type of measurement, including (a) self-report, (b) parent-report, (c) teacher-report, or (d) independent-observer report. For each effect size, we noted days between measurements to determine lag time (when time was reported in ranges, the average was taken, e.g., 4–5 weeks later would be coded as 31.5 days). Moreover, we coded whether each effect size was based on a prospective or retrospective design. Although primary study analyses used only prospective designs, retrospective designs were not an exclusionary criterion in determining whether prospective versus retrospective designs influenced the effect size. Lastly, we coded whether the study was published or unpublished. See Tables 1–4 for summaries of the studies included in the meta-analysis. See Table 5 for a summary of demographics.

**Odds ratio.** As recommended by Haddock, Rindskopf, and Shadish (1998) and similar to Liu, Huang, and Wang (2014), odds ratios were used to determine effect sizes for categorical measures of anxiety disorders predicting depressive disorders and vice versa. In this case, an odds ratio represented increased odds of being diagnosed with a depressive disorder when participants had a prior anxiety disorder, compared to those without a prior anxiety disorder (or vice versa for depressive disorders predicting anxiety disorders). An odds ratio greater than 1 indicated increased likelihood of being diagnosed with a depressive disorder when one had a prior anxiety disorder, whereas an odds ratio less than 1 indicated decreased likelihood of being diagnosed with a depressive disorder

when individuals had a prior anxiety disorder, compared with those without a prior anxiety disorder. Odds ratios have an asymmetrical distribution, and, as such, were converted into log odds ratios for analyses (Lipsey & Wilson, 2001). When a study controlled for baseline levels of either anxiety or depression, it was coded as a moderator variable. After results were obtained, data was converted back into odds ratios for interpretation.

**Correlation coefficient.** For predictions of temporal precedence of continuous measures of anxiety and depression, we used correlation coefficients and partial correlation coefficients to determine the primary effect size. When a study controlled for baseline levels of either anxiety or depressive symptoms, baseline levels were coded as a moderator variable. When baseline correlation coefficients were not reported, standardized and unstandardized regression coefficients were converted to correlation coefficients using a *t*-statistic estimator as recommended by Wolf (1986), using the following formula  $r = \sqrt{\frac{t^2}{t^2 + N - 1}}$ . Because correlation coefficients have a skewed distribution (Silver & Dunlap, 1987), Fisher’s *r*-to-*z* transformation was used to calculate primary effect sizes, using the following equation:  $z_r = \frac{1}{2} \ln\left(\frac{1+r}{1-r}\right)$  (Fisher, 1921). Following primary analyses, *z*s were converted back to correlations as the final output.

## Planned Analyses

Prior to primary analyses, agreement between raters was compared using Krippendorff’s alpha with both nominal and ratio data. Krippendorff’s alpha is optimal as it allows for incomplete data (important because all raters did not code every article, and most articles were coded by two or three raters; Hayes & Krippendorff, 2007; Krippendorff, 2004, 2012). Interrater reliability was conducted using the *R* package *irr* (Gamer, Lemon, Fellows, & Singh, 2007).

Effect size meta-analyses used the *R* package *Metafor* (Viechtbauer, 2010). All analyses used mixed-effect models, nesting each effect size within a study as a random effect. Mixed-effect models allow multiple measures of the same type to be utilized within the same study. This method was important as studies frequently included multiple effect sizes, such as when studies included the same measures being administered repeatedly over multiple time periods. This method also allows one to include all effect sizes for multiple publications using the same dataset. When this was the case, each study used different parts of the data (i.e., different time periods, different diagnostic groups, etc.). Similar to traditional multilevel models outside of the meta-analytic framework, random effects were used to account for lack of independence between effect size estimates (with the primary difference being the nesting of effect sizes within studies; see for Kalaian & Raudenbush, 1996 more information). Similar to Karelaia and Hogarth (2008) and Petersen and Hyde (2010), prior to analyzing the primary results, statistical tests of publication bias were examined, including Begg

<sup>4</sup> We also intended to code other constructs (i.e., obsessive–compulsive symptoms), but there were no qualified studies that actually examined the longitudinal relationship between anxiety and depression using these constructs.

<sup>5</sup> We hoped to include other depressive measures, such as dysthymia, but no studies measured these symptoms.

Table 1  
*Summaries of Anxiety Symptoms Predicting Depressive Symptoms*

Study	<i>N</i>	Time apart in measurements	<i>r</i>	Anxiety construct	Depression construct	Sample summary
Adams and Boscarino (2011)	1,681	1 year 2 years	.17 .03	panic symptoms panic symptoms	mixed depression mixed depression	Prospective and retrospective sample of adults living in New York City on 9/11/2001.
Alipour, Lamyian, and Hajizadeh (2012)	140	4.6 months 6.2 months	.35 .32	mixed anxiety mixed anxiety	mixed depression mixed depression	Prospective Iranian women 28–30 weeks pregnant
Austin, Tully, and Parker (2007)	575	5 months 5 months	.07 .10	mixed anxiety cognitive anxiety	mixed depression mixed depression	Prospective: women in their third trimester of pregnancy
Borelli and Prinstein (2006)	478	11 months	.46	social phobia	mixed depression	Prospective: adolescents in grades 6–8
Bromberger and Matthews (1996)	460	3 years	.37	mixed anxiety	mixed depression	Prospective: middle-aged women
Cole et al. (1998)	330	6 months 1 years 1.5 years 2 years 2.5 years	.62 .57 .49 .41 .29	mixed anxiety mixed anxiety mixed anxiety mixed anxiety mixed anxiety	mixed depression mixed depression mixed depression mixed depression mixed depression	Prospective: adolescents in grades 3–6
Fairbrother and Woody (2007)	127	1 month	.19	anxiety sensitivity	mixed depression	Prospective: women in their third trimester of pregnancy
Gazelle and Ladd (2003)	388	5 years	.40	anxious solitude	mixed depression	Prospective: kindergarteners
Grant et al. (2007)	102	1 year	.28	social phobia	mixed depression	Prospective sample of undergraduates
Grant, McMahon, et al. (2008)	100	7.5 months	.23	mixed anxiety	mixed depression	Prospective: pregnant women at risk for anxiety or depression.
Jacobson and Newman (2014)	6,504	13 years	.19	mixed anxiety	depression	Prospective sample of nationally representative adolescents
Jakobsen, Horwood, and Fergusson (2012)	948	15 years	.09	mixed anxiety	mixed depression	Prospective: New Zealand birth cohort
Leigh and Milgrom (2008)	367	1.5 months 5 months	.44 .13	mixed anxiety mixed anxiety	mixed depression mixed depression	Prospective: pregnant women
Mazza et al. (2009)	938	7 years	.18	mixed anxiety	mixed depression	Prospective: children in grades 1–2
Orcutt (2006)	182	9 months	.35	mixed anxiety	mixed depression	Prospective: female undergraduates
Rholes et al. (2011)	387	7.5 months–2 years	.24	attachment anxiety	mixed depression	Prospective: pregnant parents
Schindel-Allon et al. (2010)	147	2 weeks 2 months 2.5 months	.65 .68 .56	PTSD symptoms PTSD symptoms PTSD symptoms	mixed depression mixed depression mixed depression	Prospective: Israeli adults two weeks after single-event traumas
Sears and Armstrong (1998)	131	2 years	.17 .34	mixed anxiety	mixed depression	Prospective: adolescents from Nova Scotia
Silberg, Rutter, and Eaves (2001)	1,511	3 years 3 years 3 years	.11 .24 .22	specific phobia OAD separation anxiety	mixed depression mixed depression mixed depression	Prospective: monozygotic and dizygotic twins
Skouteris et al. (2009)	220	3 months 4 months 7 months	.51 .42 .44	mixed anxiety mixed anxiety mixed anxiety	mixed depression mixed depression mixed depression	Prospective: pregnant women
Snyder et al. (2009)	267	6 months 1 year	.17 .14	mixed anxiety mixed anxiety	mixed depression mixed depression	Prospective: children at age 5
Starr and Davila (2012b)	55	28 days 28 days	.40 .41	mixed anxiety physiological anxiety	mixed depression mixed depression	Prospective: persons with GAD
Starr and Davila (2012a)	55	1 day 2 days 3 days 4 days 1.5 days	.38 .56 .56 .41 .48	mixed anxiety mixed anxiety mixed anxiety mixed anxiety cognitive anxiety	mixed depression mixed depression mixed depression mixed depression mixed depression	Prospective: persons with GAD
Starr et al. (2014)	815	9 years 9 years	.10 .07	GAD social phobia	mixed depression mixed depression	Prospective: children with depressed mothers
Weems et al. (2007)	52	1.8 years 1.8 years 1.8 years	.31 .18 .09	mixed anxiety, GAD GAD symptoms PTSD symptoms	mixed depression mixed depression mixed depression	Prospective: adolescents before hurricane Katrina
Wetherell et al. (2001)	1,391	3 years 6 years	.48 .45	mixed anxiety mixed anxiety	mixed depression mixed depression	Prospective: elderly Swedish twins
Ying et al. (2012)	200	6 months 1 year	.58 .42	PTSD symptoms PTSD symptoms	mixed depression mixed depression	Prospective sample of adolescent survivors of the Wenchuan earthquake in China

(table continues)

Table 1 (continued)

Study	<i>N</i>	Time apart in measurements	<i>r</i>	Anxiety construct	Depression construct	Sample summary
Zaers et al. (2008)	47	7 months	.69	mixed anxiety	mixed depression	Prospective sample of pregnant women
Zavos et al. (2012)	2,603	2 years 2 years	.39 .33	mixed anxiety anxiety sensitivity	mixed depression mixed depression	Prospective sample of adolescent twins

Note. GAD = generalized anxiety disorder; OCD = obsessive compulsive disorder; PTSD = posttraumatic stress disorder; OAD = overanxious disorder.

and Mazumdar's (1994) test and Egger, Davey Smith, Schneider, and Minder's (1997) regression method. After examining publication bias, all results were tested for total effect (i.e., pooled effect size). Following the test of total effect, a *Q* test was used to determine whether there was substantial heterogeneity between studies (Cochran, 1954). This test of heterogeneity was important because, if studies were too homogeneous, it would make

examination of moderation across studies impossible. Following a significant *Q*-test to determine whether there were systematic differences between effect sizes of different studies, moderators were tested using metaregression. Post hoc tests were provided directly within metaregression estimates, wherein the referenced intercept value was changed such that each value estimated the simple slope and difference in slopes.

Table 2  
Summaries of Depression Symptoms Predicting Anxiety Symptoms

Study	<i>N</i>	Time apart in measurements	<i>r</i>	Anxiety construct	Depression construct	Sample summary
Aune and Stiles (2009)	1,439	1 year	.01	social anxiety	mixed depression	adolescents
Borelli and Prinstein (2006)	478	11 months	.37	social anxiety	mixed depression	adolescents
Britt et al. (2007)	1,685	6 months	.39	PTSD symptoms	mixed depression	US soldiers on a peacekeeping mission to Kosovo
Britton (2008)	296	1 month	.21	mixed anxiety	mixed depression	postnatal mothers
Cole et al. (1998)	330	6 months	.64	mixed anxiety	mixed depression	adolescents in grades 3–6
		1 year	.60	mixed anxiety	mixed depression	
		1.5 years	.52	mixed anxiety	mixed depression	
		2 years	.42	mixed anxiety	mixed depression	
		2.5 years	.34	mixed anxiety	mixed depression	
Grant et al. (2007)	102	1 year	.26	social anxiety	mixed depression	undergraduates
Grant, McMahon, et al. (2008)	100	7.5 months	.16	mixed anxiety	mixed depression	pregnant women at risk for later anxiety or depression.
Guglietti et al. (2010)	75	1 month	.25	PTSD symptoms	mixed depression	women with ovarian cancer
Orcutt (2006)	182	9 months	.48	mixed anxiety	mixed depression	female undergraduates
Schindel-Allon et al. (2010)	147	2 weeks	.77	PTSD symptoms	mixed depression	Israeli adult victims two weeks after single-event traumas
		1.8 months	.78	PTSD symptoms	mixed depression	
		2.3 months	.70	PTSD symptoms	mixed depression	
Sears and Armstrong (1998)	131	2 years	.32	mixed anxiety	mixed depression	adolescents from Nova Scotia
Silberg, Rutter, and Eaves (2001)	1,511	3 years	.01	specific phobia	mixed depression	monozygotic and dizygotic twins
		3 years	.11	overanxious disorder	mixed depression	
		3 years	.05	separation anxiety	mixed depression	
Skouteris et al. (2009)	220	3 months	.28	mixed anxiety	mixed depression	pregnant women
		4 months	.64	mixed anxiety	mixed depression	
		6 months	.38	mixed anxiety	mixed depression	
Snyder et al. (2009)	267	6 months	-.15	mixed anxiety	mixed depression	children at age 5
		1 year	-.06	mixed anxiety	mixed depression	
Whitehead et al. (2006)	135	3 months	.43	PTSD symptoms	mixed depression	persons with acute coronary syndromes
		9 months	.29	PTSD symptoms	mixed depression	
Wikman (2009)	452	6 months	.66	PTSD symptoms	mixed depression	patients with acute coronary syndrome
		1 year	.67	PTSD symptoms	mixed depression	
		3 years	.68	PTSD symptoms	mixed depression	
Ying et al. (2012)	200	6 months	.59	PTSD symptoms	mixed depression	adolescent earthquake survivors in China
		1 year	.51	PTSD symptoms	mixed depression	
Zavos et al. (2012)	2,603	2 years	.42	mixed anxiety	mixed depression	adolescent twins
		2 years	.35	anxiety sensitivity	mixed depression	

Note. GAD = generalized anxiety disorder; OCD = obsessive compulsive disorder; PTSD = posttraumatic stress disorder; OAD = overanxious disorder; PDD = premenstrual dysphoric disorder.

Table 3  
*Summaries of Anxiety Disorders Predicting Depressive Disorders*

Study	<i>N</i>	Time apart in measurements	<i>OR</i>	Anxiety construct	Depression construct	Sample summary
Alipour et al. (2012)	140	2 months	3.11	mixed anxiety	mixed depression	Prospective: women 28–30 weeks pregnant in Iran
		3.5 months	3.25	mixed anxiety	mixed depression	
		4.3 months	3.27	mixed anxiety	mixed depression	
		5.8 months	2.98	mixed anxiety	mixed depression	
Alloy et al. (2006)	347	1.5 months	2.27	mixed anxiety	major depression	Prospective: undergraduates with high or low risk for depression based on cognitive styles
		1.5 months	1.76	mixed anxiety	minor depression	
		1.5 months	2.32	mixed anxiety	hopeless depression	
Andrade et al. (2003)	37,000	Retrospective	81.60	GAD	major depression	Retrospective: 10 community samples across 10 countries worldwide
		Retrospective	52.70	OCD	major depression	
		Retrospective	27.03	panic disorder	major depression	
		Retrospective	9.40	PTSD	major depression	
		Retrospective	11.90	social phobia	major depression	
Austin et al. (2007)	575	5 months	10.70	specific phobia	major depression	Prospective: women during their third trimester of pregnancy
		3.11	mixed anxiety	mixed depression		
Biederman et al. (2007)	303	5 years	3.20	separation anxiety	mixed depression	Prospective: children of parents with panic disorder, major depression, or neither
Bittner et al. (2004); Friis et al. (2002); Stein et al. (2001)	2,548	4 years	3.5	social phobia	mixed depression	Prospective: adolescents and young adults
		4 years	1.73	mixed anxiety	major depression	
		4 years	3.90	GAD	major depression	
		4 years	2.74	panic disorder	major depression	
		4 years	1.80	specific phobia	major depression	
Bittner et al. (2007); Copeland et al. (2009); Costello et al. (2003)	1,420	4 years	2.30	social phobia	major depression	Prospective: children and adolescents
		1 year	3.0	mixed anxiety	mixed depression	
		4 years	1.2	GAD	mixed depression	
		4 years	2.3	social phobia	mixed depression	
		4 years	6.7	OAD	mixed depression	
		4 years	2.9	separation anxiety	mixed depression	
		5.5 years	2.4	OAD	mixed depression	
		5.5 years	7.4	GAD	mixed depression	
Breslau and Davis (1992)	861	1.2 years	2.14	mixed anxiety	mixed depression	Prospective: persons with a history of migraines
Coelho et al. (2011)	246	5 months	5.25	mixed anxiety	major depression	Prospective: women during their second trimester of pregnancy
		5 months	5.74	GAD	major depression	
		5 months	1.71	social phobia	major depression	
		7 months	4.57	mixed anxiety	major depression	
		7 months	5.19	GAD	major depression	
		7 months	2.24	social phobia	major depression	
		1.2 years	29.53	mixed anxiety	major depression	
		1.2 years	7.8	GAD	major depression	
		1.2 years	4.33	social phobia	major depression	
		1.6 years	2.72	mixed anxiety	major depression	
		1.6 years	6.58	GAD	major depression	
		1.6 years	1.66	social phobia	major depression	
		2.4 years	8.42	mixed anxiety	major depression	
		2.4 years	6.11	GAD	major depression	
		2.4 years	2.92	social phobia	major depression	
Fichter et al. (2010)	835	25 years	5.99	mixed anxiety	major depression	Prospective: persons from a rural Bavarian community
		25 years	3.84	mixed anxiety	dysthymia	
Gallerani (2008)	240	6 years later	1.12	mixed anxiety	mixed depression	Prospective: adolescents
Goodwin (2002)	15,703	1 year later	5.60	OCD	major depression	Prospective nationally representative sample
		1 year later	3.74	panic disorder	major depression	
		1 year later	2.30	specific phobia	major depression	
Goodwin et al. (2004); Jakobsen et al. (2012)	1,053	3 years	9.25	panic disorder	major depression	Prospective: young adults
		8 years	1.18	mixed anxiety	major depression	
Grant, McMahon, et al. (2008)	100	7.5 months	5.52	mixed anxiety	mixed depression	Prospective: pregnant women at risk for later anxiety or depression.
Heron et al. (2004)	8,323	4 months	2.1	mixed anxiety	major depression	Prospective: pregnant women
		7 months	1.73	mixed anxiety	major depression	
		9 months	2.17	mixed anxiety	major depression	
		1 year	1.72	mixed anxiety	major depression	

(table continues)





Table 4  
Summaries of Depressive Disorders Predicting Anxiety Disorders

Study	<i>N</i>	Time apart in measurements	<i>OR</i>	Anxiety construct	Depression construct	Sample summary
Acarturk et al. (2009)	7,076	1 year	7.31	social phobia	major depression	Prospective: Netherlands nationally representative sample
Alloy et al. (2006)	347	1 year	10.98	social phobia	dysthymia	Prospective: college students with high or low risk for depression
Breslau et al. (2008)	990	1.5 months	1.23	mixed anxiety	mixed depression	Prospective: young adults
Britton (2008)	296	4 years	2.38	PTSD	mixed depression	Prospective: postnatal mothers
Fergusson and Woodward (2002)	1,265	1 month	4.15	mixed anxiety	mixed depression	Prospective: adolescents
Fichter et al. (2010)	835	3.5 years	3.90	mixed anxiety	major depression	Prospective: Bavarian persons from a rural community
		25 years	2.59	mixed anxiety	mixed depression	
		25 years	.81	GAD	mixed depression	
		25 years	2.25	OCD	mixed depression	
		25 years	6.12	panic disorder	mixed depression	
		25 years	2.66	specific phobia	mixed depression	
Gallerani (2008)	240	6 years later	.59	mixed anxiety	mixed depression	Prospective sample of adolescents
Grant, McMahon, et al. (2008)	100	7.5 months	3.39	mixed anxiety	mixed depression	Prospective: pregnant women at risk for later anxiety or depression.
Hapke et al. (2006)	4,075	unknown	5.21	PTSD	mixed depression	Retrospective: German adults
Karsten et al. (2011)	1,167	2 years	2.81	mixed anxiety	mixed depression	Prospective: adults in the Netherlands
Kessler et al. (1998); Kessler et al. (2008)	8,098	10 years	1.2	GAD	major depression	Prospective and Retrospective nationally representative sample
		unknown	2.7	GAD	major depression	
		unknown	1.7	panic disorder	major depression	
Kessler et al. (2002)	20,189	unknown	3.1	GAD	major depression	Retrospective nationally representative sample
		unknown	2.7	GAD	dysthymia	
Neufeld et al. (1999)	3,481	13.5 years	2.8	mixed anxiety	mixed depression	Prospective: adults in Baltimore
Pine et al. (1998)	716	7 years	10.87	GAD	major depression	Prospective: adolescents
		7 years	2.05	social phobia	major depression	
		7 years	3.36	specific phobia	major depression	
		9 years	3.22	GAD	major depression	
		9 years	1.73	social phobia	major depression	
		9 years	2.79	specific phobia	major depression	
Prenoveau et al. (2013)	296	3 months–2 years	3.61	GAD	major depression	Prospective postnatal mothers
Skodol et al. (1996)	2,741	unknown	1.7	PTSD	mixed depression	Retrospective: Israeli men exposed to combat
Söderquist et al. (2009)	1,224	3–7 months	16.3	PTSD	mixed depression	Prospective: pregnant women
Sourander et al. (2005)	2,712	12.5 years	1.4	mixed anxiety	mixed depression	Prospective sample of Finnish boys
Vaananen et al. (2011)	1,843	2 years	6.60	social phobia	mixed depression	Prospective sample of 9th graders

*Note.* GAD = generalized anxiety disorder; OCD = obsessive compulsive disorder; PTSD = posttraumatic stress disorder; OAD = overanxious disorder; PDD = premenstrual dysphoric disorder.

## Results

### Interrater Reliability

Interrater reliability was high for all types of study information. Mean Krippendorff's alpha was 0.871 (range 0.722–1.000). Thus, all measures had at least adequate reliability with most items demonstrating excellent agreement (Krippendorff, 2012).

### Anxiety Symptoms Predicting Depressive Symptoms

The number of participants in studies of anxiety symptoms predicting depressive symptoms was 21,201. There was no evidence of publication bias based on the Begg test ( $\tau = 0.077$ ,  $p = .213$ ) or the Egger regression method ( $Z = 1.498$ ,  $p = .134$ ). Anxiety symptoms significantly predicted depressive symptoms ( $Z = 8.8017$ ,  $p < .001$ , see Table 6). There was also substantial

heterogeneity between studies, allowing moderation analyses to be conducted.

As length of time between measurements increased, effect sizes of anxiety symptoms predicting depressive symptoms showed degradation (see Figure 2; see Table 7 for a list of all omnibus moderation effects). When anxiety symptoms predicted depressive symptoms 1 month later, the average correlation was 0.609 CI [0.498, 0.720], 1 year later it was 0.326 CI [0.214, 0.438], and 3 years later it was 0.201 (CI = 0.089–0.312). The percentage of the sample with anxiety disorders at baseline significantly positively moderated the effect. In samples in which 0% of respondents were diagnosed with baseline anxiety disorders, the correlation was 0.310 CI [0.136, 0.465], and in samples with 100% of respondents diagnosed with anxiety disorders at baseline, the correlation was 0.585 CI [0.452, 0.693]. Likewise, percentage of the sample with depressive disorders at baseline significantly positively moderated the effect. In samples with 0% of respondents diagnosed with

Table 5  
Study Demographic at Baseline

Demographic variable	Anxiety disorders predicting depressive disorders		Depressive disorders predicting anxiety disorders		Anxiety symptoms predicting depression symptoms		Depression symptoms predicting anxiety symptoms	
	<i>M (SD)</i>	Range	<i>M (SD)</i>	Range	<i>M (SD)</i>	Range	<i>M (SD)</i>	Range
Percentage female	68.13 (27.44)	0–100	60.54 (21.47)	0–100	67.34 (23.41)	0–100	60.62 (24.05)	8–100
Percentage White	88.43 (3.83)	79–95	85.76 (6.34)	74–93	65.70 (14.92)	43–100	74.47 (12.49)	59–100
Mean age	23.41 (8.50)	6–38	24.73 (9.72)	12–38	21.36 (13.27)	5–61	18.58 (14.21)	5–61
Percentage anxiety disorders	18.82 (14.85)	7–61	19.36 (16.17)	7–61	25.00 (22.55)	0–98	38.09 (35.47)	15–100
Percentage depressive disorders	11.62 (6.29)	5–18	16.01 (9.03)	5–34	26.52 (30.00)	0–99	38.74 (27.12)	7–82

depressive disorders at baseline, the correlation was 0.295 CI [0.200, 0.383], and in samples with 100% of respondents diagnosed with depressive disorders at baseline, the correlation was 0.607 CI [0.539, 0.666]. No other moderators were significant.

### Depressive Symptoms Predicting Anxiety Symptoms

In studies examining depressive symptoms predicting anxiety symptoms, the number of participants was 7,750. There was no evidence of publication bias based on the Begg test ( $\tau = 0.014$ ,  $p = .900$ ) or Egger regression method ( $Z = 0.332$ ,  $p = .740$ ). The average correlation between depressive symptoms and later anxiety symptoms was significantly positive ( $Z = 5.407$ ,  $p < .001$ ). Additionally, there was significant heterogeneity in effect sizes across studies.

In terms of moderation, as length of time between assessments increased, the effect of depressive symptoms on subsequent anxiety symptoms decreased showing log-linear decay. When depressive symptoms predicted anxiety symptoms 1 month later, the average correlation was 0.609 CI [0.498, 0.721], 1 year later it was 0.255 CI [0.086, 0.424], and 3 years later it was 0.073 CI [−0.096, 0.241]. See Figure 2 for a graphical depiction of this result.

The percentage of the sample with anxiety disorders at baseline significantly positively moderated the effect. In samples in which 7% of respondents were diagnosed with anxiety disorders at baseline, the correlation coefficient was 0.213 CI [−0.096, 0.523], and in samples in which 100% of respondents were diagnosed with anxiety disorders at baseline, the correlation coefficient was 0.939 (CI [0.629–1.000]). Likewise, percentage of the sample with depressive disorders at baseline significantly positively moderated the effect. In samples in which 7% of respondents were diagnosed with depressive disorders at baseline, the correlation coefficient was 0.088 CI [−0.580, 0.234], and in samples in which 66% of respondents were diagnosed with depressive disorders at baseline, the correlation coefficient was 0.662 CI [0.516, 0.808].

Likewise, type of anxiety symptom being predicted was a significant moderator (see Table 8). Depressive symptoms only significantly predicted non-disorder-specific anxiety ( $SE = 0.095$ ,  $Z = 3.540$ ,  $p < .001$ ) and PTSD symptoms ( $SE = 0.104$ ,  $Z = 5.189$ ,  $p < .001$ ). Moreover, depressive symptoms predicted PTSD symptoms more strongly than specific phobia symptoms ( $Z = 2.135$ ,  $p = .037$ ).<sup>6</sup> Also, higher percentages of Caucasians were associated with lower effect sizes. Additionally, studies that did

not control for baseline had significantly larger effect sizes than studies that did. No other moderators were significant.

### Strength of Directionality of Anxious and Depressive Symptoms

Next, we tested the strength of directionality of anxiety symptoms predicting depressive symptoms compared to depressive symptoms predicting anxiety symptoms. Anxiety symptoms more strongly predicted later depressive symptoms, than depressive symptoms predicted later anxiety symptoms ( $\beta = 0.028$ ,  $SE = 0.012$ ,  $Z = 2.267$ ,  $p = .023$ ) across all anxiety constructs.

### Anxiety Disorders Predicting Depressive Disorders

The total number of participants in the studies obtained for anxiety disorders predicting depressive disorders was 38,902. There was no evidence of publication bias based on the Begg test ( $\tau = 0.134$ ,  $p = .086$ ) or Egger regression method ( $Z = 1.498$ ,  $p = .134$ ). Anxiety disorders significantly positively predicted depressive disorders ( $Z = 8.384$ ,  $p < .001$ ; see Table 6). Those with an anxiety disorder had a 154.8% greater probability of being diagnosed with a depressive disorder at the later assessment compared to those without a prior anxiety disorder. The heterogeneity test was significant, suggesting that there were between-study differences, allowing for the examination of moderation.

In the examination of moderators, anxiety disorder diagnosis also had a significant impact on anxiety disorders predicting later depressive disorders. Mixed anxiety disorder samples ( $SE = 0.130$ ,  $Z = 6.011$ ,  $p < .001$ ), OCD ( $SE = 0.155$ ,  $Z = 10.469$ ,  $p < .001$ ), GAD ( $SE = 0.202$ ,  $Z = 6.565$ ,  $p < .001$ ), PTSD ( $SE = 0.471$ ,  $Z = 2.776$ ,  $p = .006$ ), panic disorder ( $SE = 0.151$ ,  $Z = 7.997$ ,  $p < .001$ ), social phobia ( $SE = 0.170$ ,  $Z = 5.175$ ,  $p < .001$ ), specific phobia ( $SE = 0.150$ ,  $Z = 4.820$ ,  $p < .001$ ), and “other” anxiety disorder (e.g., overanxious anxiety disorder, separation anxiety disorder;  $SE = 0.215$ ,  $Z = 4.811$ ,  $p < .001$ ) significantly predicted later depression. Thus, all anxiety disorders significantly predicted later depressive disorders.

In comparing the different anxiety disorders, OCD was significantly stronger in predicting depressive disorders than mixed anxiety

<sup>6</sup> We were unable to examine depressive symptoms predicting OCD, GAD, and panic symptoms because no studies reported effect sizes using these symptoms.

Table 6  
 Summary of Effect Sizes for the Relationship Between Anxiety and Depression

Relationship	<i>k</i>	<i>N</i>	Weighted mean effect size	CI	Homogeneity ( <i>Q</i> )
Anxiety symptoms predicting depressive symptoms	29	21,201	$r = .338$	[.267–.406]	1429.618*
Depressive symptoms predicting anxiety symptoms	17	7,705	$r = .346$	[.226–.455]	1561.380*
Anxiety disorders predicting depressive disorders	38	38,902	$OR = 2.548$	[2.007–3.233]	2599.660*
Depressive disorders predicting anxiety disorders	17	30,686	$OR = 3.001$	[1.970–4.570]	114.720*

Note.  $N = 88,336$ .

\*  $p < .05$ .

disorder samples ( $Z = 5.237, p < .001$ ), panic disorder ( $Z = 10.253, p < .001$ ), social phobia ( $Z = 4.205, p < .001$ ), specific phobia ( $Z = 20.989, p < .001$ ), and other anxiety disorders ( $Z = 2.774, p = .006$ ). Likewise, GAD was a significantly stronger predictor of depressive disorders than mixed anxiety disorders ( $Z = 2.706, p = .007$ ), social phobia ( $Z = 2.207, p = .027$ ), and specific phobia ( $Z = 2.796, p = .005$ ). The effect of panic disorder was also stronger than mixed anxiety disorder samples ( $Z = 2.726, p = .006$ ) and specific phobia ( $Z = 17.455, p < .001$ ). See Table 9 for a summary.

The length of time of the depressive disorders reference period at outcome significantly moderated the relationship. Anxiety disorders significantly predicted depressive disorders across depressive disorder reference periods of less than one year ( $\beta = 4.951, CI [3.077, 7.967], SE = 0.243, Z = 6.591, p < .001, k = 3$ ), one to two years ( $\beta = 2.685, CI [1.933, 3.728], SE = 0.167, Z = 5.897, p < .001, k = 6$ ), and greater than two years ( $\beta = 2.104, CI [1.547, 2.861], SE = 0.157, Z = 4.743, p < .001, k = 8$ ). Nevertheless, studies that measured depressive disorders across less than one year had significantly larger effect sizes than studies that measured depression over one-two years ( $\Delta\beta = 2.267, SE = 0.295, Z = 2.076, p = .038$ ) or greater than two years ( $\Delta\beta = 2.847, SE = 0.289, Z = 2.961, p = .003$ ). There were no significant differences between depressive disorders measured across one to two years compared with those with greater than two years ( $\Delta\beta = 0.581, SE = 0.225, Z = 1.084, p = .278$ ). No other moderators were significant.

Note that all results described above only included prospective studies. Nevertheless, a sensitivity analysis to determine whether effects differed between retrospective and prospective studies found that retrospective studies tended to have marginally significantly larger effect sizes than prospective studies.

### Depressive Disorders Predicting Anxiety Disorders

In regard to depressive disorders predicting anxiety disorders, there were a total of 30,686 participants in the studies obtained. There was no evidence of publication bias based on the Begg test ( $\tau = 0.005, p = .984$ ) or the Egger regression method ( $Z = -0.057, p = .955$ ). Depressive disorders significantly predicted later anxiety disorders ( $Z = 5.119, p < .001$ , see Table 6). Those with a depressive disorder had a 200.1% greater probability of being diagnosed with an anxiety disorder compared with those without a prior depressive disorder. There was also significant heterogeneity in effect sizes for these studies, allowing for the examination of moderation.

Such moderation showed that type of anxiety measure impacted the relationship. Depressive disorders significantly predicted both self-reports ( $\beta = 6.903, CI [2.959, 16.105], SE = 0.432, Z = 4.470, p < .001, k = 3$ ) and independent observer reports ( $\beta = 2.431, CI [1.600, 3.701], SE = 0.215, Z = 4.141, p < .001, k = 12$ ) of anxiety disorders. However, self-report

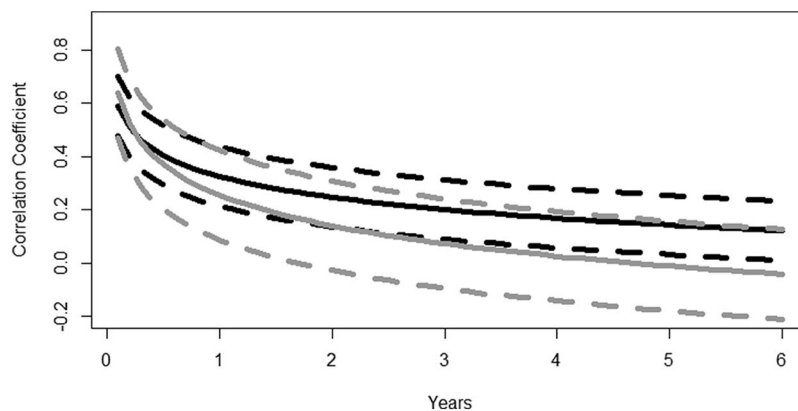


Figure 2. This figure depicts the relationship between anxiety symptoms predicting depressive symptoms over time, and depressive symptoms predicting anxiety symptoms over time. The black lines represent the correlation coefficients of anxiety symptoms on depressive symptoms, and the gray lines represent the correlation of depressive symptoms on anxiety symptoms. The solid lines represent the estimates, and the dashed lines represent the lower and upper confidence intervals.



Table 7  
Summary of Moderation Analyses

Moderator	k	N	Statistics
<b>Anxiety symptoms predicting depressive symptoms</b>			
Length of time between measurements	25	16,859	$\beta = -.114, SE = .014, Z = -7.930, p < .001^*$
Percentage of the sample with anxiety disorders at baseline	8	2,225	$\beta = .004, SE = .002, Z = 2.290, p = .022^*$
Percentage of the sample with depressive disorders at baseline	8	2,720	$\beta = .004, SE = .001, Z = 3.447, p = .001^*$
Type of anxiety construct	24	16,472	$Q(6) = 5.875, p = .437$
Type of depression construct	N/A	N/A	N/A
Type of anxiety measure	25	16,859	$Q(3) = 4.932, p = .177$
Type of depression measure	25	16,859	$Q(3) = 5.903, p = .116$
Gender	23	16,425	$\beta = -.002, SE = .002, Z = -.121, p = .903$
Age	21	13,455	$\beta = .001, SE = .003, Z = .270, p = .787$
Percentage of Whites	11	9,842	$\beta = -.003, SE = .003, Z = -.881, p = .378$
Targeted population	24	16,472	$Q(6) = 8.888, p = .180$
Controlling for baseline	25	16,859	$\beta = -.049, SE = .036, Z = -1.362, p = .173$
Unpublished studies	N/A	N/A	N/A
Prospective versus retrospective	N/A	N/A	N/A
<b>Depressive symptoms predicting anxiety symptoms</b>			
Length of time between measurements	15	7,052	$\beta = -.166, SE = .021, Z = -8.148, p < .001^*$
Percentage of the sample with anxiety disorders at baseline	6	2,168	$\beta = .008, SE = .003, Z = 2.269, p = .023^*$
Percentage of the sample with depressive disorders at baseline	6	2,389	$\beta = .008, SE = .002, Z = 4.623, p < .001^*$
Type of anxiety construct	15	7,052	$Q(4) = 10.991, p = .027^*$
Type of depression construct	N/A	N/A	N/A
Type of anxiety measure	15	7,052	$Q(3) = 4.322, p = .229$
Type of depression measure	15	7,052	$Q(3) = 2.294, p = .402$
Gender	15	7,052	$\beta = -.003, SE = .002, Z = -1.254, p = .210$
Age	15	7,052	$\beta = .003, SE = .004, Z = .663, p = .507$
Percentage of Whites	11	6,418	$\beta = -.009, SE = .004, Z = -2.690, p = .007^*$
Targeted population	15	7,052	$Q(5) = 10.586, p = .060$
Controlling for baseline	15	7,052	$\beta = .247, SE = .061, Z = 4.121, p < .001^*$
Unpublished studies	N/A	N/A	N/A
Prospective versus retrospective	N/A	N/A	N/A
<b>Anxiety disorders predicting depressive disorders</b>			
Length of time between measurements	19	30,122	$\beta = .109, SE = .077, Z = .428, p = .440$
Percentage of the sample with anxiety disorders at baseline	7	20,365	$\beta = -.022, SE = .005, Z = -1.134, p = .257$
Percentage of the sample with depressive disorders at baseline	5	3,278	$\beta = -.068, SE = .143, Z = -.277, p = .782$
Type of anxiety construct	19	30,122	$Q(7) = 542.388, p < .001$
Type of depression construct	19	30,122	$Q(3) = 1.861, p = .602$
Type of anxiety measure	19	30,122	$Q(3) = 4.470, p = .251$
Type of depression measure	19	30,122	$Q(3) = 4.010, p = .379$
Gender	14	9,258	$\beta = -.043, SE = .005, Z = -1.841, p = .066$
Age	12	7,310	$\beta = .030, SE = .016, Z = 1.492, p = .136$
Percentage of Whites	7	2,873	$\beta = .003, SE = .037, Z = 1.111, p = .267$
Targeted population	16	27,509	$Q(4) = 2.522, p = .641$
Controlling for baseline	19	30,122	$\beta = -.144, SE = .272, Z = -.205, p = .838$
Published versus unpublished studies	19	30,122	$\beta = -1.540, SE = .657, Z = -1.295, p = .195$
Anxiety disorder reference period	16	28,979	$Q(2) = .057, p = .972$
Depressive disorder reference period	15	28,683	$Q(2) = 8.999, p = .011^*$
DSM version	19	30,122	$Q(3) = .424, p = .935$
Prospective versus retrospective	31	107,830	$\beta = 1.085, SE = .317, Z = 1.747, p = .081$
<b>Depressive disorders predicting anxiety disorders</b>			
Length of time between measurements	15	28,034	$\beta = -.428, SE = .134, Z = -1.030, p = .303$
Percentage of the sample with anxiety disorders at baseline	6	14,457	$\beta = .012, SE = .011, Z = .505, p = .616$
Percentage of the sample with depressive disorders at baseline	6	4,398	$\beta = -.057, SE = .013, Z = -.838, p = .402$
Type of anxiety construct	15	28,034	$Q(6) = 3.040, p = .804$
Type of depression construct	15	28,034	$Q(2) = 1.498, p = .473$
Type of anxiety measure	15	28,034	$Q(1) = 4.679, p = .031^*$
Type of depression measure	15	28,034	$Q(1) = .704, p = .401$
Gender	12	22,414	$\beta = .013, SE = .010, Z = .672, p = .502$
Age	9	5,975	$\beta = .038, SE = .034, Z = 1.265, p = .206$
Percentage of Whites	6	2,749	$\beta = .002, SE = .054, Z = .809, p = .419$
Targeted population	13	25,895	$Q(3) = 2.324, p = .508$
Controlling for baseline	15	28,034	$\beta = -.213, SE = .286, Z = -.248, p = .804$

(table continues)

Table 7 (continued)

Moderator	<i>k</i>	<i>N</i>	Statistics
Published versus unpublished studies	15	28,034	$\beta = -1.993, SE = .961, Z = -1.612, p = .107$
Anxiety disorder reference period	14	27,044	$Q(2) = 7.419, p = .025^*$
Depressive disorder reference period	13	26,944	$Q(2) = 7.058, p = .029^*$
DSM version	15	28,034	$Q(3) = 2.719, p = .257$
Prospective versus retrospective	19	57,751	$\beta = 1.786, SE = .177, Z = 2.838, p = .005^*$

Note. This table presents all omnibus moderation effects (i.e. *Q* tests for categorical outcomes, and regression coefficients for continuous moderation).  
\*  $p < .05$ .

measures had significantly larger effects compared to independent observers ( $\Delta\beta = 4.472, SE = 0.483, Z = 2.163, p = .031$ ).

The length of the anxiety disorders reference period at outcome had a significant impact on the strength of depressive disorders predicting later anxiety disorders. Depressive disorders significantly predicted anxiety disorders when the reference time period was less than one year ( $\beta = 2.960, CI [1.352, 6.481], SE = 0.400, Z = 2.714, p = .007, k = 4$ ) and one to two years ( $\beta = 5.604, CI [3.032, 10.355], SE = 0.313, Z = 5.500, p < .001, k = 5$ ), but not over two years ( $\beta = 1.653, CI [0.882, 3.100], SE = 0.321, Z = 1.568, p = .117, k = 5$ ). Among the different anxiety disorder reference periods, measurements across one to two years in time had significantly larger effect sizes than studies over two years ( $\Delta\beta = 3.950, SE = 0.448, Z = 2.722, p = .007$ ). However, there were no significant differences between reference periods of less than one year compared to reference periods over two years ( $\Delta\beta = 1.306, SE = 0.513, Z = 1.136, p = .256$ ), or between reference periods of less than one year with reference periods from one to two years ( $\Delta\beta = 2.644, SE = 0.508, Z = 1.256, p = .209$ ).

Similarly, the length of the depressive disorder reference period also significantly impacted the effect of depressive disorders on later anxiety disorders. A depressive disorder reference period of less than one year ( $\beta = 2.865, CI [1.182, 6.947], SE = 0.452, Z = 2.329, p = .020, k = 3$ ) or from one to two years ( $\beta = 5.617, CI [2.984, 10.576], SE = 0.323, Z = 5.347, p < .001, k = 6$ ) significantly predicted later anxiety disorders. However, when reference periods were longer than

two years, depressive disorders did not significantly predict later anxiety disorders ( $\beta = 1.651, CI [0.865, 3.152], SE = 0.330, Z = 1.519, p = .129, k = 7$ ). Likewise, studies with reference periods from one to two years had significantly larger effect sizes than those with measurement periods longer than two years ( $\Delta\beta = 3.967, SE = 0.462, Z = 2.653, p = .008$ ), but there were no significant differences between reference periods of less than one year with reference periods longer than two years ( $\Delta\beta = 1.214, SE = 0.560, Z = 0.985, p = .325$ ), or between reference periods of less than one year with reference periods from one to two years ( $\Delta\beta = 2.752, SE = 0.555, Z = 1.212, p = .225$ ). No other moderators were significant.

Similar to anxiety disorders predicting depressive disorders, the above analyses only included prospective studies. Nevertheless, sensitivity analyses suggested that retrospective studies had significantly larger effect sizes than prospective studies.

### Strength of Directionality of Anxiety and Depressive Disorders

Next, we tested the strength of the directionality of anxiety disorders predicting depressive disorders compared to depressive disorders predicting anxiety disorders. The results showed that depressive disorders predicted social phobia significantly more strongly than social phobia predicted depressive disorders. Likewise, the results showed that depressive disorders predicted specific phobia significantly more strongly than specific phobia pre-

Table 8

Comparing Strength of Predicting Different Types of Anxiety Symptoms From Depression Symptoms

Type of anxiety symptoms	<i>r</i> (CI)	Comparisons between anxiety symptoms at outcome				
		Anxiety as a whole ( <i>k</i> = 6, <i>N</i> = 15,216) $\Delta\beta$ (SE)	Posttraumatic stress symptoms ( <i>k</i> = 5, <i>N</i> = 2,694) $\Delta\beta$ (SE)	Social phobia symptoms ( <i>k</i> = 3, <i>N</i> = 2,019) $\Delta\beta$ (SE)	Specific phobia symptoms ( <i>k</i> = 1, <i>N</i> = 1,511) $\Delta\beta$ (SE)	Other anxiety symptoms ( <i>k</i> = 1, <i>N</i> = 1,511) $\Delta\beta$ (SE)
Non-disorder-specific anxiety	.324 [.149, .479]*					
Posttraumatic stress symptoms	.496 [.326, .634]*	.20 (.14)				
Social phobia symptoms	.215 [-.049, .448]	-.11 (.16)	-.32 (.17)			
Specific phobia symptoms	.010 [-.409, .425]	-.32 (.24)	-.49 (.24)*	-.21 (.26)		
Other anxiety symptoms	.080 [-.347, .480]	-.25 (.25)	-.43 (.24)	-.14 (.26)	.07 (.03)	
Total	.346 [.226, .455]*					

Note.  $k = 15, N = 7,750$ . The "total" reflects the cumulative effects of depressive symptoms predicting anxiety symptoms *between* studies. Note that no studies reported effect sizes of obsessive-compulsive, generalized anxiety, and panic symptoms being predicted by depressive symptoms.

\*  $p < .05$ .

**Table 9**  
**Comparing Strength of Different Types Anxiety Disorders Predicting Depressive Disorders**

Disorder	Odds ratio (CI)	Comparisons between anxiety disorders predicting depressive disorders							
		Anxiety disorder ( <i>k</i> = 11, <i>N</i> = 20,223) Δ <i>B</i> ( <i>SE</i> )	Obsessive-compulsive disorder ( <i>k</i> = 2, <i>N</i> = 16,769) Δ <i>B</i> ( <i>SE</i> )	Generalized anxiety disorder ( <i>k</i> = 5, <i>N</i> = 5,576) Δ <i>B</i> ( <i>SE</i> )	Posttraumatic stress disorder ( <i>k</i> = 2, <i>N</i> = 2,317) Δ <i>B</i> ( <i>SE</i> )	Panic disorder ( <i>k</i> = 4, <i>N</i> = 20,370) Δ <i>B</i> ( <i>SE</i> )	Social phobia ( <i>k</i> = 5, <i>N</i> = 5,280) Δ <i>B</i> ( <i>SE</i> )	Specific phobia ( <i>k</i> = 5, <i>N</i> = 20,855) Δ <i>B</i> ( <i>SE</i> )	Other disorders ( <i>k</i> = 3, <i>N</i> = 2,499) Δ <i>B</i> ( <i>SE</i> )
Mixed anxiety disorders	2.183 [1.693, 2.816]*								
Obsessive-compulsive disorder	5.039 [3.723, 6.821]*	2.86 (.16)*							
Generalized anxiety disorder	3.773 [2.538, 5.608]*	1.59 (.20)*	-1.27 (.22)						
Posttraumatic stress disorder	3.693 [1.468, 9.289]*	1.51 (.47)	-08 (.50)						
Panic disorder	3.342 [2.487, 4.492]*	1.16 (.16)*	-1.70 (.04)*	-08 (.50)					
Social phobia	2.407 [1.726, 3.356]*	.22 (.16)	-2.63 (.18)*	-1.29 (.49)	-08 (.50)				
Specific phobia	2.062 [1.537, 2.768]*	-.12 (.16)	-2.98 (.04)*	-1.63 (.48)	-1.28 (.03)*	-.34 (.17)			
Other disorders	3.406 [2.364, 4.908]*	.63 (.22)	-2.23 (.20)*	-1.71 (.22)*	-1.63 (.48)	.41 (.23)	.75 (.21)		
Total	2.548 [2.007, 3.233]*								

Note. *k* = 19, *N* = 38,902. The "total" reflects the cumulative effects of anxiety disorders predicting depressed disorders *between* studies, whereas mixed anxiety disorders reflect the cumulative effects of anxiety disorders predicting depressed disorders *within* studies.  
\* *p* < .05.

dicted depressive disorders. No other anxiety disorder significantly predicted depressive disorders more strongly than depressive disorders predicted later anxiety disorders (see Table 10).<sup>7</sup>

### Discussion

We examined the longitudinal relationship between anxiety and depression using 66 prospective studies with a total sample size of 88,336. Results showed that across a wide variety of symptoms and disorders, anxiety and depression had bidirectional prospective relationships with one another. Unlike prior reviews (Alloy et al., 1990; Andover et al., 2011; Fava & Tossani, 2007; Horn & Wuyek, 2010; Moras & Barlow, 1992; Schleider et al., 2014; Wittchen, Beesdo, et al., 2003), these findings do not support the theory that anxiety disorders constitute a prodrome for depressive disorders. Concluding that anxiety is a prodrome requires that (a) anxiety disorders precede and predict the onset of later depressive disorders, and (b) anxiety disorders predict later depressive disorders with greater strength than depressive disorders predict later anxiety disorders. No anxiety disorders evidenced superior directional predictive power than depressive disorders predicting later anxiety disorders. Further, the strength of depressive disorders predicting social and specific phobia was greater than vice versa, in stark contrast to prior reviews (Alloy et al., 1990; Andover et al., 2011; Fava & Tossani, 2007; Horn & Wuyek, 2010; Moras & Barlow, 1992; Schleider et al., 2014; Wittchen, Beesdo, et al., 2003). This may mean that depressive disorders are prodromes for both social and specific phobia, however, more research should be conducted before drawing any definitive conclusions.

As opposed to the bidirectionality of anxiety and depressive disorders, anxiety symptoms predicted later depressive symptoms more strongly than vice versa. Although there were significant differences in effect sizes of this analysis, the magnitude of the difference may not be clinically meaningful (difference in estimated *r*s = .03 lower for depressive symptoms predicting anxiety symptoms). Nevertheless, contrasting symptom versus disorder findings may suggest that the effect of depressive symptoms on later anxiety symptoms is intensified at the disorder level (perhaps due to a substantial decrease in behavioral activation in depressive disorders).

There were other notable differences between disorder and symptom level results. In particular, depressive disorders significantly predicted later social and specific phobia diagnoses, but

<sup>7</sup> As a sensitivity analysis, to account for effect sizes that could have been influenced by the failure to control for baseline levels of the outcome measure, all analyses were repeated using a dummy-coded variable indicating whether studies controlled for baseline levels of the outcome of interest (i.e., controlling for baseline depressive symptoms when examining anxiety symptoms predicting depressive symptoms). With one exception, the significance of all results remained consistent with the present findings. The one exception was that percentage of baseline anxiety disorders no longer significantly moderated the relationship of depressive symptoms predicting later anxiety symptoms ( $\beta = 0.007, SE = 0.004, Z = 1.818, p = .069$ ). Note that even though this effect became marginally significant with this control in place, it still led to large effect sizes for estimated differences. Samples with 7% of respondents who were diagnosed with an anxiety disorder had an estimated correlation coefficient of 0.122 CI [-.310, 0.554], and samples with 100% of respondents meeting criteria for an anxiety disorder had an estimated correlation of 0.782 CI [0.351, 1.000]. Thus, although marginally significant, the effect appears to remain large and may still be clinically significant.

Table 10

*Comparisons Between the Strength of Anxiety Disorders Predicting Depressive Disorders Against the Strength of Depressive Disorders Predicting Anxiety Disorders*

Anxiety disorder	<i>k</i>	Odds ratio (CI) of anxiety disorder predicting depressive disorder	Odds ratio (CI) of depressive disorder predicting anxiety disorder	$\Delta\beta$ ( <i>SE</i> )
Generalized anxiety disorder	14	2.582 [1.811, 5.232]*	1.876 [1.259, 2.796]*	-.71 (.18)
Obsessive-compulsive disorder	3	5.599 [5.214, 6.014]*	2.255 [.203, 25.030]	-3.34 (1.23)
Panic disorder	5	4.597 [3.026, 6.982]*	6.126 [1.168, 32.119]*	1.53 (.87)
Posttraumatic stress disorder	4	4.651 [.755, 28.627]	5.745 [1.118, 29.540]*	1.09 (1.25)
Social phobia	7	2.606 [1.709, 3.973]*	6.045 [3.387, 10.789]*	3.44 (.35)*
Specific phobia	6	1.719 [1.314, 2.248]*	2.934 [1.791, 4.807]*	1.22 (.27)*
Total	24	2.770 [2.101, 3.653]*	2.725 [2.030, 3.657]*	-.05 (.11)

\*  $p < .05$ .

depressive symptoms did not show the same pattern. The eligible studies that examined depressed mood predicting social and specific phobia symptoms had lower means and standard deviations of baseline depressive symptoms (Borelli & Prinstein, 2006; Grant, McMahon, & Austin, 2008; Silberg, Rutter, & Eaves, 2001) compared with comparable validation samples (Angold & Costello, 1995; Boyce, Stubbs, & Todd, 1993; Finch, Saylor, & Edwards, 1985; Green, 1998; Simonoff et al., 1997). Thus, in these studies, there may have been insufficient variability and elevation in depressive symptoms to observe their impact on later social and specific phobia symptoms. At the same time, there were no representative studies available to examine whether depressive symptoms predicted OCD, GAD, or panic disorder symptoms. Taken together, this may be why the only anxiety disorder symptoms significantly predicted by depressed mood were PTSD and non-disorder-specific anxiety symptoms.

In an attempt to explain the findings of depressive disorders predicting social and specific phobia more strongly than most anxiety disorders, consider that this may be attributable to lack of behavioral activation in those with depressive disorders (Kasch, Rottenberg, Arnow, & Gotlib, 2002). Such diminished activity may lead to decreased exposure to new situations and experiences (Kasch et al., 2002) and increased fear and avoidance cycles (File & Hyde, 1978). There is also evidence that social and specific phobia are defined by discrete fears to a greater extent than other anxiety disorders (American Psychiatric Association, 2013). Thus, reduced activity leading to more discrete fears and increased avoidance may explain the unique relationship between depressive disorders and later social and specific phobia.

Another explanation for the relationship between depressive disorders and later social phobia may be that it is driven by the irritating and consequently negative interpersonal impact of depressive disorders (Coyne, 1976). Compared with those without depression, persons with depression are more likely to be evaluated negatively and rejected by people close to them (Burchill & Stiles, 1988). They also experience lower satisfaction in romantic relationships (Weissman, 1987) than nondepressed controls, suggesting that they may generate stressful interpersonal life events (Daley et al., 1997). Additionally, interpersonal rejection predicted later social anxiety symptoms (Siegel, La Greca, & Harrison, 2009). Thus, future research should examine behavioral inhibition and social rejection as potential mediators between depressive disorders and later social and specific phobia.

An alternative potential link between depressive disorders and later social phobia could be the influence of depressive disorders on self-esteem (Sowislo & Orth, 2013) and cognitions of worthlessness (Beck, Brown, Steer, Eidelson, & Riskind, 1987). Low self-esteem and worthless cognitions could directly increase depressed individuals' expectations of being rejected (Kang, Downey, Iida, & Rodriguez, 2009) and consequently promote interpersonal avoidance. Thus, further research should examine low self-esteem, worthless cognitions, and expectations of rejection as mediators between depressive disorders and later social phobia.

Depressive disorders leading to specific phobia may be explained by evolutionary mechanisms, which have emphasized the role of life history in activating both disorders to improve one's survival and chance of reproduction (Del Giudice, 2014; Jacobson, 2016). For example, according to evolutionary models of specific phobia (Coelho & Purkis, 2009), objects related to evolutionary threat can be more rapidly tied to fear learning compared to evolutionarily neutral stimuli (Ohman, Eriksson, & Olofsson, 1975). In parallel, depression has been theorized to be an evolutionarily adaptive coping response (Allen & Badcock, 2003). Thus, perhaps in response to substantial early environmental threats (Allen & Badcock, 2003), depression leads to a strategy uniquely tied to increasing one's likelihood of survival, wherein one decreases one's behavioral activation and subsequently avoids potentially threatening cues (Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002).

Importantly, those with OCD had larger odds of being diagnosed with a subsequent depressive disorder compared to those with most other anxiety disorders (including mixed anxiety disorders, panic disorder, social phobia, specific phobia, and other anxiety disorders [e.g., overanxious anxiety disorder, separation anxiety disorder]). This may imply that OCD has unique ties to later depressive disorders. Such ties may be explained partially by OCD's strong relationship with shame and perfectionism (Fergus, Valentiner, McGrath, & Jencius, 2010), relative to other anxiety disorders. For example, OCD may be associated with thoughts of harming one's children, which may bring about feelings of shame (Abramowitz, Moore, Carmin, Wiegartz, & Purdon, 2001), and shame has strong ties to depression (Kim, Thibodeau, & Jorgensen, 2011). Shame also cross-sectionally mediated the relationship between OCD and depressive symptoms (Weingarden & Renshaw, 2014). Likewise, OCD is characterized by high levels of perfectionism (Coles, Frost, Heimberg, & Rhéaume, 2003), and



perfectionistic dysfunctional attitudes are central to the diagnosis of depression (Mongrain & Blackburn, 2005).

A growing body of literature has examined potential mechanisms of anxiety leading to later depression. Avoidance mediated the relationship between anxiety and later depressive symptoms across a decade (Jacobson & Newman, 2014), perhaps related to diminished exposure to positive events. These pathways also applied to those who met diagnostic criteria for anxiety and depressive disorders (Jacobson & Newman, 2014). Additionally, perceptions of close and group relationships mediated anxiety and later depressive symptoms and anxiety and later depressive disorders, such that high anxiety symptoms predicted poorer perceptions of close and group relationships which in turn led to higher depressive symptoms (Jacobson & Newman, 2016). Similarly, sociability and interpersonal oversensitivity was a mediator of earlier anxiety disorders and later depressive disorders (Starr, Hammen, Connolly, & Brennan, 2014). Thus, although more research is needed, preliminary findings suggest that avoidance and interpersonal relationships mediate the relationship between earlier anxiety and later depression, and this applies to both symptoms and disorders.

Results regarding percentage of the sample with anxiety and depressive disorders at baseline as a moderator were particularly striking and may have important implications. Specifically, the percentage of the sample with anxiety or depressive disorders at baseline moderated the relationship between anxiety and depressive *symptoms* predicting one another. At the same time, this moderation was not observed with anxiety or depressive *disorders* predicting one another. Although the former pattern became marginally significant after controlling for baseline symptoms, this analysis still had strong effect sizes and therefore may still be clinically important. Perhaps anxiety and depressive disorders have unique impacts on the relationship between anxiety and depressive *symptoms*. This impact could be attributable to the presence of the disorders creating broad (as opposed to transient or specific) levels of avoidance behaviors and low behavioral activation. It is also possible that reaching the level or chronicity of symptoms required for a disorder diagnosis, majorly increases the likelihood of higher symptoms of the other disorder later.

There was also a striking difference between symptoms and disorders in impact of the time lag between measurements. The effect of anxiety symptoms on later depressive symptoms and depressive symptoms on later anxiety symptoms was depleted as lag time increased whereas time lag did not deplete the impact of disorders on one another. There are several potential methodological explanations for this difference. First, the minimum amount of time between symptom measures was 1 day whereas the minimum amount of time between disorder measures was 45 days. Therefore, symptom measures allowed for the examination of much smaller time lags when moods could affect one another. Second, disorder measures build a certain level of chronicity (did you feel  $x$  nearly every day for  $x$  time period) into their assessments, which makes them potentially less susceptible than symptoms to variability of their effects over time. Also, because symptoms did not require participants to meet a specific threshold and reflected the full continuum, they were likely more sensitive to detecting smaller changes over time compared to disorders.

It is also possible that the relationship between anxiety and depressive symptoms is best characterized by short-term fluctuations of symptoms bidirectionally predicting one another. Indeed, there is

some preliminary support that anxiety and depression symptoms may be maintained by across time periods as short as hours (Jacobson & Newman, 2012). Thus, anxiety and depressive disorders may be much more robust predictors/risk factors for one another. Once people reach a threshold in anxiety and depressive symptoms they may be continually at risk for the other respective disorder, but, until that point, the relationship between anxiety and depression may degrade over time.

It is also important to consider “third variables” that could explain the bidirectional relationship between anxiety and depression. One such variable is the common higher-order factor of neuroticism. Substantial evidence has accrued that neuroticism levels are high across anxiety and depressive disorders (see Kotov, Gamez, Schmidt, & Watson, 2010 for a meta-analysis). Additionally, based on cross-sectional research, some have tended to emphasize neuroticism as a joint risk factor for both anxiety and depression (Manee & Majreh, 2016; Merino, Senra, & Ferreira, 2016; Muris, Roelofs, Rassin, Franken, & Mayer, 2005; Roelofs, Huibers, Peeters, & Arntz, 2008). Lastly, neuroticism accounts for a large portion of prospective change patterns of anxiety and depression (Brown, 2007), and neuroticism interacts with chronic stress to predict change in depressive symptoms (Brown & Rossellini, 2011). Thus, this—on its face—suggests that neuroticism may confound and explain the current findings.

Nevertheless, suggesting that neuroticism confounds the current findings is problematic. Most principally, this argument is tautological as neuroticism itself combines face-valid anxiety, depression, and anger items (Costa & McCrae, 1992). Thus, using a combined measure of anxiety and depression items to predict anxiety and depression across time as separate constructs ignores the containment of anxiety and depression symptoms within measures of neuroticism.

A second problem of attempting to explain the current findings using neuroticism is that it assumes that the construct of neuroticism can be validly applied to within-person changes in anxiety and depression, disregarding a pervasive and widespread measurement issue. In particular, neuroticism was formed based on the *between*-person structure of correlations of negative emotionality (i.e., anxiety, depression, and anger) given to persons on *one measurement occasion* (Eysenck, 1947). Many would assume that constructs examined between persons also readily define the factor structure of the same items *within persons* across time. This presumes that the process is ergodic (a strict mathematical assumption that rarely holds in psychological research, for a review see Molenaar, 2004). However, when one analyzes neuroticism items within the same people across time (using a method known as dynamic factor analysis), the factor structure of these items does not well-define the within-person variance (Borkenau & Ostendorf, 1998; Molenaar & Campbell, 2009). Thus, the between-person factor structure of neuroticism does not reflect a fundamental building block of within-person variation of negative mood, suggesting that neuroticism, itself, may not be applicable to changes within persons.

In addition to tautological and psychometric issues, attempting to explain the current findings using a measure that lumps anxiety and depression items into the same construct disregards substantial experimental research that anxious and depressive moods can be induced independently of one another (Cryder, Lerner, Gross, & Dahl, 2008; Ferrer, Grenen, & Taber, 2015; Gray, Ishii, & Ambady, 2011; Innes-Ker, 2015; Kreibig, Wilhelm, Roth, & Gross,

2007; Nabi, 2002). Such experimental findings provide strong evidence that these moods do not represent the same construct. Thus, these issues may suggest that neuroticism does not represent a “third variable” that confounds the relationship between anxiety and depression.

Nevertheless, aspects of neuroticism that do not overlap directly with anxiety and depression symptoms may be important to explaining their comorbidity. In particular, neuroticism is often measured by items such as “moody,” “unstable,” and “stable” (reverse coded; John & Srivastava, 1999). Similarly, recent models have suggested that stress reactivity is a central feature of neuroticism (Barlow, Ellard, Sauer-Zavala, Bullis, & Carl, 2014), which has been supported by research examining the contribution of neuroticism to reactivity to stressors in daily life (Bolger & Schilling, 1991). Such unstable and varying moods may be a byproduct of previous anxiety and depression (Roberts & Gotlib, 1997). Thus, future research should examine whether neuroticism’s facet of stress reactivity helps to inform the longitudinal relationship between anxiety and depression at both symptom and disorder levels.

Another potential third variable not accounted for in the current meta-analysis is stress. Notably, stress can vary independently of anxiety and depression, and consequently it may not represent a confound. This has been supported by studies controlling for the influence of stressful life events or perceived stress, which have found (a) anxiety symptoms significantly predicted later depressive symptoms (Jacobson, Lord, & Newman, 2017), (b) depressive symptoms significantly predicted later anxiety symptoms (Britton, 2008; Jacobson et al., 2017), (c) anxiety disorders significantly predicted later depressive disorders (Friis, Wittchen, Pfister, & Lieb, 2002); and (d) depressive disorders significantly predicted later social phobia and mixed anxiety disorders (Acarturk et al., 2009; Britton, 2008). Nevertheless, stress may be important to examine in future studies, as it has been associated frequently with anxiety and depression (e.g., Andrews & Wilding, 2004; Paykel, 2003).

The current findings have important clinical implications. With regard to prevention efforts, as the effect of anxiety and depressive symptoms tends to degrade over time, but anxiety and depressive disorders appear to predict one another across decades, prevention strategies should be particularly focused on early identification of subthreshold elevation to prevent one from meeting criteria for an anxiety or depressive disorder. The current findings also may have implications about symptom change within psychotherapy. As anxiety and depressive disorders are mostly bidirectional risk factors for one another, this may suggest that transdiagnostic treatments aimed at either anxiety or depressive disorders would likely effectively impact both disorders (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). Nevertheless, exceptions to this may occur within domains of depressive disorders and subsequent social and specific phobia. For these disorders, symptom reduction may be more effective if depression is treated prior to social or specific phobia, given that depressive disorders may predict greater likelihood of development and/or resurgence than vice versa. However, it is also possible that transdiagnostic treatments would be just as effective in this regard.

The current meta-analysis had several strengths. For instance, we examined differential impacts of anxiety and depressive disorders on one another at both symptom and disorder levels, and results were largely consistent with one another. There was also substantial heterogeneity between studies, including sample ages,

populations, and time lag between measurements. As age, race, and gender did not moderate the findings, relationships between anxiety and depression may be generalizable across many different groups.

Although there were many strengths, there were also limitations. Most importantly, all studies included had no experimental manipulation, and consequently this research is unable to address the causal relationship between anxiety and depression. Nonetheless, the present data are important because they may be among the strongest form of evidence ethically possible, as experimentally examining the causal relationship between anxiety and depressive disorders would require experimenters to give participants an anxiety or depressive disorder. At the same time, the present research establishes the strength and temporal precedence between anxiety and depression. However, it does not eliminate the possibility of a co-occurring confounding variable, and, as such, use of the term “risk factor” over “cause” is more appropriate. Also, only about half of the studies we included controlled for baseline depression or anxiety symptoms. Given that these symptoms are known to co-occur concurrently, we attempted to offset this problem by rerunning all analyses controlling for baseline symptoms and disorders. We were also unable to test directly first onset versus recurrent prodromal relationships and controlling for baseline symptoms did not offset this limitation. Nevertheless, because age did not moderate the findings and we included studies within theorized developmental periods of anxiety and depressive diagnoses, this may suggest that there are not differential relationships between anxiety and depression for first onset compared with recurrent anxiety and depression. Further, because only one study examined bidirectional longitudinal relationships between anxiety and depression for both symptoms and disorders, we were unable to rule out the possibility that disorder versus symptom findings may have been influenced by methodological differences across studies. Additionally, most included studies examined Western cultures (only five studies used non-North American/non-European country samples), and, as such, future research should determine if these findings would be applicable to other countries. Moreover, we only examined longitudinal associations but not mechanisms between anxiety and depression. Little research has examined such longitudinal mechanisms (Jacobson, Lord, & Newman, 2017; Jacobson & Newman, 2014, 2016; Starr et al., 2014), and no research has examined mechanisms of depression on later anxiety. Thus, more research is needed to explore potential mediators of these relationships.

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