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Is Positive Emotionality a Specific Risk Factor for Depression? A Meta-Analysis of Longitudinal Studies

Gabriela Kattan Khazanov and **Ayelet Meron Ruscio**

Department of Psychology, University of Pennsylvania

Abstract

Depression is well known to share a negative cross-sectional relationship with personality constructs defined by positive emotion (positive affect, extraversion, behavioral activation). These Positive Emotionality (PE) constructs have been proposed to represent stable temperamental risk factors for depression, not merely current mood state. These constructs have also been proposed to increase risk specifically for depression, relative to anxiety. We performed a meta-analysis of longitudinal studies to examine the relationship of PE to depression (59 effect sizes) and anxiety (26 effect sizes). In cross-sectional analyses, PE constructs were negatively associated with depression ($r = -.34$) and anxiety ($r = -.24$). PE constructs also prospectively predicted depression ($r = -.26$) and anxiety ($r = -.19$). These relationships remained statistically significant, but were markedly attenuated, when baseline levels of depression ($\beta = -.08$) and anxiety ($\beta = -.06$) were controlled. Moreover, depression and anxiety were equally strong predictors of subsequent changes in PE ($\beta = -.07$ and $-.09$, respectively). These findings are consistent with theoretical accounts of low PE as a temperamental vulnerability for depression, but suggest that the prospective relationship of PE to depression may be weaker and less specific than previously assumed.

Keywords

anxiety; depression; extraversion; longitudinal; positive affect

Major depressive disorder is one of the most common mental disorders in the U.S. (Kessler, Chiu, Demler, Merikangas, & Walters, 2005) and among the most burdensome diseases in the world (World Health Organization, 2002). A core symptom of depression is anhedonia, a lack of interest or pleasure in usual activities (American Psychiatric Association, 2013). Many depressed individuals exhibit a diminished tendency to experience positive emotions, even in the presence of normally appetitive stimuli (Berenbaum & Oltmanns, 1992; Kaviani et al., 2004; D. F. Klein, 1974; McFarland & Klein, 2009; Sloan, Strauss, & Wisner, 2001). This contrasts with the experience of individuals high in Positive Emotionality (PE), who exhibit elevations in positive mood states such as happiness, interest, energy, and confidence (Mineka, Watson, & Clark, 1998; Watson & Naragon-Gainey, 2010, 2014). In fact, PE is generally regarded not only as a marker of psychological health (Gilbert, 2012; Ozer &

Correspondence concerning this article should be addressed to Gabriela Kattan Khazanov, Department of Psychology, University of Pennsylvania, 3720 Walnut Street, Philadelphia, PA 19104. kattang@sas.upenn.edu.

Benet-Martinez, 2006; Tugade & Fredrickson, 2004; though see Gruber, Johnson, Oveis, & Keltner, 2008) but as an evolutionarily adaptive trait that increases psychological flexibility and strengthens physical, intellectual, and social resources (Fredrickson, 1998).

Several prominent theoretical models suggest that, in addition to reflecting current mood state, PE is a stable and heritable personality trait (Krueger, McGue, & Iacono, 2001) that, at low levels, increases risk for depression and exacerbates the course of the disorder (Clark, 2005; Clark & Watson, 1991, 1999; Davidson, 1998; Depue & Iacono, 1989; Gray, 1994; Watson, Stasik, Ellickson-Larew, & Stanton, 2015). For example, theories concerning the closely related behavioral activation and inhibition systems (Gray, 1994), approach and withdrawal systems (Davidson, 1998), and behavioral facilitation systems (Depue & Iacono, 1989; Fowles, 1994) all posit a causal role for low PE in depression. Broadly, these biobehavioral theories describe an approach system that controls goal-directed behavior, is activated in response to positive stimuli, and generates positive emotional experiences. Depression is thought to result from an underactive approach system (Shankman & Klein, 2003). Similarly, the influential tripartite model (Clark & Watson, 1991), later reformulated into the integrative hierarchical (Mineka et al., 1998) and quadripartite (Watson, 2009) models, identifies low PE as a core component and potential risk factor for depression (Clark, Watson, & Mineka, 1994). Unlike negative emotionality, which is viewed as a factor common to depression and anxiety, PE is thought to relate more specifically to depression. Although PE has also been associated with social phobia and agoraphobia (Bienvenu & Stein, 2003; Kashdan, 2007), it is generally more strongly and consistently correlated with measures of depression than anxiety (Watson, 2009). The PE personality trait in the Five-Factor Model, extraversion, is also theorized to constitute a risk factor for the development of depression (Clark et al., 1994; McCrae & Costa, 1987; Watson, Wiese, Vaidya, & Tellegen, 1999). Importantly, although PE is labeled differently in different theories, PE constructs are conceptually similar and correlations among them are high.[1]

Spurred in part by these models, numerous studies have investigated the relationship of PE to depression, with research efforts increasing sharply in recent years (Naragon-Gainey, Watson, & Markon, 2009; Proceedings, 2011; Watson, Clark, & Stasik, 2011; Watson, Gamez, & Simms, 2005; Watson et al., 2015). Many, though not all, of these studies have provided evidence for a cross-sectional relationship, paralleling experimental evidence for attenuated reactivity to positive stimuli in depression across self-report, behavioral, physiological, and neural measures (Bylsma, Morris, & Rottenberg, 2008; Dichter, 2010; Dichter, Damiano, & Allen, 2012; Dillon et al., 2014). Building on these findings, two meta-analyses have directly examined the cross-sectional relationship between PE (restricted to extraversion) and depression. One meta-analysis found that while mental disorders as a group were associated with low PE, mood disorders were associated with significantly lower PE than all other disorders (Malouff, Thorsteinsson, & Schutte, 2005). The second meta-

[1]Correlations among the PE constructs of positive affect, extraversion, and behavioral activation have been reported in the range of .48 to .66 (Naragon-Gainey et al., 2013). Following the lead of other researchers, we defined PE as including all three of these constructs (Brown, 2007; Clark et al., 1994; Naragon-Gainey et al., 2013; Naragon-Gainey et al., 2009). Initially, we also planned to include the related construct of reward dependence from the Tridimensional Personality Model (Cloninger, Przybeck, & Svrakic, 1991; Enns & Cox, 1997), but as we found few longitudinal studies of reward dependence and depression, we excluded this construct from our analysis.

analysis found that PE was most strongly associated with dysthymic disorder (a chronic form of depression) and social phobia (Kotov, Gamez, Schmidt, & Watson, 2010). Unexpectedly, PE shared weak associations with major depression and relatively strong associations with other anxiety disorders. This surprising pattern may have resulted from the presence of comorbid disorders in the diagnostic groups that were studied (Naragon-Gainey, Gallagher, & Brown, 2013), although it leaves open the possibility that low PE may be less specific to depression than has previously been suggested.

These cross-sectional studies, though informative, have been unable to test the fundamental assertion that PE increases vulnerability to depression. Demonstrating that low PE precedes and predicts subsequent increases in depression is essential for establishing PE as a risk factor, rather than an epiphenomenon or a consequence, of depression (Kraemer et al., 1997). Characterizing the temporal association between PE and depression is also important for adjudicating among explanations for their relationship. Explanations that have been proposed for the relationship between personality traits and psychopathology include: (a) the predisposition model, which posits that personality influences the development of psychopathology; (b) the pathoplasty model, which posits that personality affects the course of psychopathology; (c) the scar model, which asserts that psychopathology leads to permanent changes in personality; and (d) the complication model, which asserts that psychopathology leads to temporary changes in personality that last until the disorder remits (Clark et al., 1994; Kotov et al., 2010). Discovering that PE prospectively predicts depression would lend support to the predisposition and pathoplasty models, whereas evidence that depression predicts PE would provide support for the scar and complication models. While these pathways are not mutually exclusive, the degree to which PE predicts depression versus the reverse can shed light on the relative applicability of these explanatory models to the PE-depression relationship.

In line with views of PE as a vulnerability factor, some longitudinal studies have found that low PE predicts later heightened levels of depression (e.g., Geerts & Bouhuys, 1998; Kasch, Rottenberg, Arnow, & Gotlib, 2002; Naragon-Gainey et al., 2013). Further support comes from studies showing that initial levels of anhedonia predict increased odds of depression two years later (Wardenaar, Giltay, van Veen, Zitman, & Penninx, 2012) and a more chronic course of the disorder over ten years (Moos & Cronkite, 1999). In adolescents, diminished seeking of positive stimuli has been found to predict later depressive symptoms (Forbes, Shaw, & Dahl, 2007; Rawal, Collishaw, Thapar, & Rice, 2013). Similarly, heightened reactivity to positive stimuli has been found to predict recovery from depression in adults (Rottenberg, Kasch, Gross, & Gotlib, 2002).

Not all studies have found that PE predicts subsequent levels of depression, however (e.g., Brown, 2007; Weiss et al., 2009). These conflicting accounts mirror the findings from family and twin studies, which do not consistently identify PE as a risk factor for depression (D. N. Klein, Kotov, & Bufferd, 2011). Several narrative reviews have evaluated the longitudinal relationship between PE and depression and have come to different conclusions, suggesting that PE probably does predict depression (Clark et al., 1994; Morris, Bylsma, & Rottenberg, 2009; Watson & Naragon-Gainey, 2010), probably does not (Shankman & Klein, 2003), or that results are inconclusive (Enns & Cox, 1997). These conclusions are complicated by the

fact that few longitudinal studies have tested whether PE predicts *change* in depression by controlling for initial depression symptoms. Controlling for initial symptoms is critical to ensure that prospective effects of PE are not due to the cross-sectional relationship between PE and depression and the stability of depression over time (Finkel, 1995).

The present study is the first to examine the longitudinal relationship between PE and depression using meta-analysis, thereby overcoming several important limitations of past reviews (Rosenthal & DiMatteo, 2001). Meta-analysis does not rely on each study's report of statistical significance to determine the presence of a relationship, pools studies together to overcome the problem of low statistical power that is common in psychopathology research, and quantifies effect sizes to determine the strength of relationships. Importantly, the technique used in the present meta-analysis also allows for the control of initial symptom levels regardless of whether the study itself controlled for this factor (Sowislo & Orth, 2013), thereby providing a more rigorous test of PE as a risk factor for depression.

According to extant theoretical models, PE is a risk factor for depression in particular (Hasler, Drevets, Manji, & Charney, 2004). This suggests that PE should exhibit stronger and more consistent relationships with depression than with other emotional disorders (i.e., anxiety disorders; Mineka et al., 1998; Watson & Naragon-Gainey, 2010; Watson et al., 2015). In fact, low PE is one of very few risk factors that are thought to distinguish depression from anxiety (see Ruscio & Khazanov, 2016, for a review). The specificity of low PE to depression, however, has been examined only in cross-sectional meta-analyses (Kotov et al., 2010; Malouff et al., 2005), whose conflicting results have been described above. Previous narrative reviews of this topic have been similarly inconclusive (Clark et al., 1994; Shankman & Klein, 2003; Watson & Naragon-Gainey, 2010). To address this gap, the present study examined PE as a risk factor for anxiety as well as depression. We focused on general (rather than syndromal) measures of anxiety because we wanted to test the claim that PE is more strongly associated with depression than with anxiety conceptualized in general terms (Watson & Naragon-Gainey, 2010), and because there were not enough studies examining the longitudinal relationships between PE and specific anxiety syndromes (e.g., panic, social anxiety) to include in our analysis.[2]

Gaining a better understanding of the relation of PE to depression and anxiety may help reduce the steep societal and personal costs of these conditions. If PE is indeed a risk factor for depression, low PE levels could be used to identify vulnerable individuals for prevention efforts before the onset of depression (Kotov et al., 2010). Targeting prevention interventions toward individuals at high risk for depression is crucial to limit the cost and increase the feasibility of these interventions (Smit, Beekman, Cuijpers, de Graaf, & Vollebergh, 2004). Furthermore, PE measures could be included in initial clinical assessments to recommend treatment strategies that target this particular vulnerability (e.g., behavioral activation; Hopko, Lejuez, Ruggiero, & Eifert, 2003) or to provide therapists with information helpful for other aspects of treatment planning, such as promoting a strong therapeutic alliance and

[2]As PE has also been studied in relation to schizophrenia/schizotypy (Horan et al., 2008; Watson & Naragon-Gainey, 2010), we initially planned to include this relationship as a further test of specificity. Due to the very small number of longitudinal studies, however, we were unable to include schizophrenia/schizotypy in the analysis.

compliance with homework assignments (Zinbarg, Uliaszek, & Adler, 2008). Patient outcomes could improve if treatment choices were made based on characteristics that predict treatment response (DeRubeis et al., 2014), and personality traits have been found to predict response to particular types of treatments (Simon & Perlis, 2010). Evidence that PE is a specific risk factor for depression relative to anxiety would allow for more precise predictions regarding the onset and course of particular symptoms, further enhancing prevention and treatment efforts.

By contrast, evidence that PE increases risk similarly for depression and anxiety would prompt reevaluation of its scope as a vulnerability factor and reformulation of its role in emotional disturbance. For example, a lack of specificity could suggest that PE, like negative emotionality (Lahey, 2009; Mineka et al., 1998), contributes to the close relationship and frequent co-occurrence of depression with anxiety. Similarly, evidence that PE follows, as well as precedes, depression would challenge the unidirectional causal pathway that is widely assumed in the literature (e.g., Brown & Barlow, 2009; Clark, 2005). Bidirectional effects would have implications not only for etiological models of depression, but for the broader debate over the nature of the relationship between personality and psychopathology (Akiskal, Hirschfeld, & Yerevanian, 1983; Clark et al., 1994; Krueger & Tackett, 2003). These findings would call into question the conceptualization of personality traits as primarily contributing to, rather than influenced by, the development of clinical disorders (Clark, 2005).

The Present Study

The present meta-analysis sought to establish whether PE is a specific risk factor for depression. To answer this question, four goals were pursued. The first goal was to examine three effect sizes quantifying the relationship between PE and depression: the cross-sectional relationship; the longitudinal relationship between PE and depression; and the longitudinal relationship between PE and depression, controlling for initial symptoms of depression. The longitudinal, controlled relationship was considered the most rigorous test of PE as a risk factor for depression. However, examining all three relationships allowed us to compare the meta-analytic results with patterns observed in previous research, much of which has focused on cross-sectional and uncontrolled relationships.

The second goal was to investigate whether depression predicts PE. To do this, we examined the longitudinal relationship between depression and PE, with and without controls for initial levels of PE. Together with the earlier tests of PE predicting depression, these analyses allowed us to compare the extent of support for the scar/complication models versus the predisposition/pathoplasty models.

The third goal was to evaluate the specificity of PE as a predictor and outcome of depression. This goal was pursued by recalculating the effect sizes described above for the relationship between PE and anxiety. We compared the effect sizes to determine whether the influence of PE is limited to depression or extends to other forms of emotional disturbance.

As the relationships of PE with depression and anxiety have been studied over widely varying time intervals, we recalculated the longitudinal, controlled relationships for relatively short (up to one year) and long (more than one year) inter-assessment intervals. These analyses allowed us to examine the magnitude and stability of the primary effect sizes across more homogenous time lags. They also allowed us to check that effect sizes were not disproportionally affected by studies with very long time lags.

The fourth goal was to examine moderators that may explain variability in the relationship between PE and depression. Given our primary interest in the longitudinal relationship between PE and depression, controlling for initial depression levels, we examined moderators of this relationship. Previous research prompted us to include several categories of potential moderators: the operationalization of PE, characteristics of the sample, and features of the study measures.

Operationalization of PE

While the constructs of positive affect, extraversion, and behavioral activation are generally grouped under the umbrella of PE, these constructs might differ in the magnitude of their association with depression. Specifically, positive affect may relate more strongly to depression than the broader construct of extraversion, which includes elements of sociability, ascendance, and fun seeking in addition to positive affect (Naragon-Gainey & Watson, 2014; Naragon-Gainey et al., 2009; Watson et al., 2015). Behavioral activation has also been suggested to relate more strongly to depression than extraversion (Shankman & Klein, 2003). To explore potential differences in the magnitude of these relationships, the type of PE construct was examined as a moderator.

Additionally, the time span covered by the PE measure might influence the strength of its relationship to depression (Watson & Clark, 1994). PE measures can be administered with state instructions (i.e., asking respondents to rate PE over a particular span of time) or trait instructions (i.e., asking respondents to rate their general levels of PE). Whereas state measures may relate more strongly to current ratings of psychopathology (Watson & Clark, 1994), trait measures may predict future depression more strongly (Brown, 2007). We therefore examined state versus trait instructions of PE measures as a moderator.

Demographic and other characteristics of the sample

Some, but not all, studies have suggested that the relationship between PE and depression may differ by age. Specifically, PE may be less consistently related to depression in children and adolescents than in adults (for a review, see De Bolle & De Fruyt, 2010). To examine this possibility more closely, we evaluated sample age as a moderator. Although few studies have investigated sex differences in the relationship between PE and depression, indications that this relationship may be stronger for males than females (Rorsman, Grasbeck, Hagnell, Isberg, & Otterbeck, 1993) prompted us to test sex as a moderator as well.

Given past reviews that have documented stronger relationships between personality characteristics and mental disorders in clinical than nonclinical samples (Kashdan, 2007; Kotov et al., 2010; Ruiz, Pincus, & Schinka, 2008), we examined sample type as a moderator. To understand more fully how sample characteristics influence the relationship of

PE to depression, we also examined the following sample characteristics as moderators: whether study eligibility criteria (e.g., including only individuals with major depression) likely yielded a restricted range of symptom levels, whether the sample experienced a defined event between assessments (e.g., loss of a loved one, medical treatment) that would be expected to produce change in symptom levels, and whether the sample consisted of a special population (e.g., individuals with a physical illness) that differed from the general population in important ways.

Study measures

Past studies generally have not found differences in the PE-depression relationship based on the type of reporter (e.g., self-report versus clinician-report; Watson et al., 2015). However, as discrepancies between self- and other-reports are not uncommon in research on psychopathology (Dozier & Lee, 1995), we examined type of reporter as a moderator. Also, as continuous disorder measures are generally more stable and reliable than dichotomous measures (Chmielewski, Clark, Bagby, & Watson, 2015; Watson, 2009), we examined whether the type of depression measure moderated the relationship between PE and depression.

Hypotheses

We hypothesized that PE would be related to depression in cross-sectional and longitudinal analyses and would predict depression even when initial symptoms were controlled. We further hypothesized that PE would predict depression to a greater extent than depression would predict PE, thereby obtaining stronger support for the predisposition and pathoplasty models than for the scar and complication models. In line with extant theoretical models, we hypothesized that PE would serve as a specific risk factor for depression, as evidenced by a stronger prospective, controlled relationship between PE and depression than between PE and anxiety. We also expected that PE's prospective, controlled relationships with both depression and anxiety would be stronger over shorter time intervals. Finally, based on previous research, we expected the PE-depression relationship to be moderated by type of PE construct assessed, state versus trait measurement of PE, sample age, and sample type (clinical versus nonclinical).

Method

Literature Search

Relevant studies were identified through a search on PsycINFO and Medline databases through November, 2014 using combinations of the keywords (1) positive affect*, positive emotion*, behavioral activation, BAS, extr*version, and reward dependence, paired with (2) depress*, dysthym*, dysphor*, anx*, phob*, fear, panic, post traumatic stress disorder, post-traumatic stress disorder, posttraumatic stress disorder, PTSD, acute stress disorder, obsessive compulsive disorder, obsessive-compulsive disorder, OCD, GAD, and schizo*, and (3) longitudinal, risk factor, vulnerability, prospective, retrospective, and antecedent. The asterisk allowed for the inclusion of alternate word endings. For example, anx* resulted in articles containing anxiety, anxious, and so forth. In an effort to reduce publication bias

(the “file drawer problem”), no restrictions were placed on publication type. Eight doctoral dissertations and one conference paper met our eligibility criteria and were included in the analyses. Additionally, the reference sections of all eligible papers, including papers without sufficient effect size data for which authors were contacted, were hand-searched for relevant studies. Finally, papers included in the two meta-analyses that examined the relationships between personality traits and clinical disorders (Kotov et al., 2010; Malouff et al., 2005) were screened.

Eligibility criteria—The following eligibility criteria were applied to select studies for the meta-analysis:

Study design: The study design was longitudinal and included measures of PE and depression/anxiety at one time-point and at least one of these measures at the following time-point (to allow for the control of symptoms or PE levels at baseline). Given these restrictions, retrospective studies with only one assessment occasion were excluded. Studies using ecological momentary assessment (EMA) were excluded unless the study also included a long-term followup (e.g., if a study measured PE using EMA at Time 1 and depression at Time 1 and Time 2, this study was eligible for inclusion). Due to difficulties with correlated data points in twin studies (Carlin, Gurrin, Sterne, Morley, & Dwyer, 2005), these types of studies were excluded. Finally, only reports in English were included.

Measures: Eligible PE measures included measures of positive affect, positive emotionality, behavioral activation, and extraversion. Given variability in the content of positive emotionality measures, these measures ($n = 5$) were categorized as positive affect measures if they focused exclusively on positive affect, and as extraversion measures if they also assessed sociability.

As stated previously, we included measures that used either state or trait instructions. To ensure that measures of PE reflected participants’ report of their natural state over a period of at least a few days, studies with instructions to rate PE “in the present moment” were excluded, as were studies using a measure of PE to assess changes in mood state following an experimental manipulation. EMA studies utilizing momentary assessments of PE that compiled these ratings over a period of at least a few days were included.

Symptom measures were required to include an assessment of symptoms of depression or anxiety. Measures of depressed affect that did not include assessment of other symptoms of depression were excluded. Anxiety measures focusing on fear (e.g., Beck Anxiety Inventory; Beck & Steer, 1993) and anxiety (e.g., State-Trait Anxiety Inventory; Spielberger, 1989) were included. As noted, only measures assessing general levels of anxiety were included. Measures assessing specific anxiety disorders (e.g., social anxiety disorder) or anxiety in particular situations (e.g., test anxiety) were excluded. Measures were also required to assess depression and anxiety as separate constructs. Measures assessing “internalizing symptoms” or “emotional disorders” were excluded. Both continuous and dichotomous symptom measures were included.

Self-report measures and measures rated by others (i.e., expert clinical interviewers, trained lay interviewers, parents) were included. Behavioral measures were excluded. Single measures assessing multiple constructs of interest (e.g., positive affect and depression measured on the same scale, as in the Center for Epidemiologic Studies Depression Scale; Radloff, 1977) were included if there was no content overlap.

Population and life events: All types of populations were included, except those for whom there were concerns about measurement validity due to potential cognitive impairment. Therefore, studies focusing on patients with dementia, stroke, multiple sclerosis, or traumatic brain injury were excluded. Participants who were bereaved, serving as caregivers, or currently or previously physically ill were included and identified as special populations. Given differences between participants with unipolar and bipolar depression on measures of PE in previous research (Watson & Naragon-Gainey, 2014; Watson et al., 2015) and our focus on unipolar depression, samples including participants with bipolar disorder were excluded.

Samples experiencing a defined life event between assessments (e.g., loss of a loved one, military deployment, birth of a child) were included. Treatment studies of medical treatments were included. Treatment studies of psychological or psychiatric treatments were excluded. If a study was not a psychological treatment study, but a portion of participants likely received treatment (i.e., studies recruiting from outpatient clinics), the study was included. Follow-up studies in which the first assessment took place after receipt of treatment were included. These criteria allowed us to be as inclusive as possible, while restricting the analyses to studies that tracked the progression of depression/anxiety without direct attempts to change symptom levels.

Independence of effect sizes: To ensure independence of effect sizes, when multiple papers presented data from the same study, one paper was selected for inclusion based on the following criteria (in order of importance): 1) if one paper presented data on both depression and anxiety and another presented data on only one of these constructs, the paper with more information was selected; 2) the paper with the most complete data to calculate effect sizes was selected; 3) the paper that most directly addressed the present research question was selected; and 4) the most recently published paper was selected.

Contact criteria—Authors of studies providing the majority of data needed to calculate effect sizes (i.e., only one correlation was missing) were contacted. Additionally, authors of studies including PE and depression/anxiety as central constructs, but not including the data necessary to calculate effect sizes, were contacted. Authors of studies that did not provide the majority of data needed to calculate effect sizes and that did not include PE and depression/anxiety as central constructs were not contacted.

When authors were contacted, all relevant measures included in the paper were requested. As the majority of eligible studies used continuous symptom measures, and as continuous effect sizes were used in the present analysis, continuous symptom measures were requested (e.g., a self-report measure or a measure of the number of symptoms endorsed in a clinical interview). As the majority of eligible studies used self-report measures, self-report

measures were requested. These requests were intended to minimize the heterogeneity between studies. When the paper presented data from up to three time points, data from all time points were requested. When the paper presented data from four or more time points (k = 10), three time points were requested: the baseline assessment, the time point closest to one year after baseline, and the time point closest to five years after baseline. This strategy allowed us to conduct additional analyses examining effect sizes for studies with more homogeneous time points.

All authors were requested to send effect size data for the same sample of participants for all time points. This ensured that all effect size calculations were based on the same sample of participants. When eligible studies providing all data necessary for calculating effect sizes included data on different samples (i.e., baseline correlations included more participants than prospective correlations), authors were contacted and requested to send data for equivalent samples. Of 23 eligible studies, seven included data on different samples. The authors of these seven studies were contacted and revised data were sent for two papers. Of the remaining five papers, three provided data for samples that differed in size by less than 20% and the papers specified that no differences were found between these samples on measures of interest (PE and depression/anxiety measures). We conducted sensitivity analyses with the remaining two studies (Marques et al., 2011; Poon & Knight, 2013). As effect sizes were essentially unchanged whether these studies were included or excluded, analyses are reported with these studies included. For all studies, the percent of attrition from the first assessment to the assessment used for analyses was included as a moderator.

Selection of Studies

Our literature search led to the identification of 1,564 papers (see Figure 1 for schematic). Titles and abstracts were reviewed by the first author and a trained research assistant, and the full texts of all papers marked as potentially relevant by either reviewer (k = 869) were examined.

Of these papers, 23 met the eligibility criteria listed above and included all the information necessary to calculate effect sizes. Another 152 papers met the eligibility criteria, but did not include enough information to calculate effect sizes. Of these 152 papers, the authors of 66 papers were contacted based on the contact criteria listed above. Corresponding authors were contacted three times using up-to-date contact information found online or obtained from university administrators, coauthors, and former advisors. Data were obtained for 41 papers. Therefore, 64 papers were included in the analyses, with one paper providing effect sizes for two separate samples of participants (Langer, Weisman, Rodebaugh, Binder, & Lenze, 2014). As some articles (k = 20) provided effect sizes for both depression and anxiety, these 65 separate samples provided 59 effect sizes for depression and 26 effect sizes for anxiety.

Coding of Studies

Approach—As noted, we calculated three effect sizes for each set of analyses: (1) the cross-sectional correlation between the two variables (e.g., the correlation of PE at Time 1 with depression at Time 1); (2) the prospective correlation between the predictor variable

and the outcome variable (e.g., the correlation of PE at Time 1 with depression at Time 2); and (3) the cross-lagged regression between the predictor variable and the outcome variable, controlling for prior levels of the outcome variable (e.g., the correlation of PE at Time 1 with depression at Time 2, controlling for depression at Time 1). These effect sizes were calculated for four sets of analyses estimating the relationships between (1) PE at Time 1 and depression at Time 2; (2) depression at Time 1 and PE at Time 2; (3) PE at Time 1 and anxiety at Time 2; (4) anxiety at Time 1 and PE at Time 2.

Cross-lagged regressions were calculated for the third (controlled) effect size using zero-order correlations between variables in the following equation (Cohen, Cohen, West, & Aiken, 2003):

$$\beta_{Y1.2}=\frac{r_{Y1}-r_{Y2}r_{12}}{1-r^2_{12}}$$

$\beta_{Y1.2}$ is the standardized regression coefficient of X_1 predicting Y, adjusting for the effect of X_2 (e.g., the effect of PE at Time 1 on depression at Time 2, adjusting for depression at Time 1). The correlation between the predictor variable of interest [X_1] and the outcome variable [Y] is r_{Y1} (e.g., the correlation of PE at Time 1 with depression at Time 2). The correlation between the other predictor variable [X_2] and the outcome variable [Y] is r_{Y2} (e.g., the correlation of depression at Time 1 with depression at Time 2). The baseline correlation between the two predictors [X_1 and X_2] is r_{12} (e.g., the correlation of PE at Time 1 with depression at Time 1).

We used cross-lagged regressions, rather than cross-lagged correlations (e.g., comparing the correlation between PE at Time 1 and depression at Time 2 with the correlation between depression at Time 1 and PE at Time 2), because of criticisms that the latter are confounded by the stability of the outcome variable (Locascio, 1982; Rogosa, 1980; Sowislo & Orth, 2013). For example, a large cross-lagged correlation between PE and depression may merely reflect the high stability of depression from Time 1 to Time 2 if PE and depression are strongly correlated at Time 1. Cross-lagged regressions avoid this confound by controlling for the stability of the outcome variable.

Coding procedures—Studies were coded for effect sizes and moderators using a formal coding manual. The first author coded all studies and an advanced graduate student coded a random subset of 30 studies. Interrater reliability was calculated for effect sizes and continuous moderators using intraclass correlation coefficients and for categorical moderators using Cohen's kappa. Interrater agreement was high (ICC ≥ .99 and $\kappa \geq .91$ for continuous and categorical variables, respectively) and discrepant ratings were discussed until consensus was reached.

Coding decisions—When multiple symptom measures were available in one study, we coded the continuous measure and the self-report measure (PE measures were always continuous and self-report). These decisions were made to reduce the heterogeneity between studies, as the majority of symptom measures were continuous and self-report. Additionally, continuous measures were privileged because effect sizes were continuous. On the other

hand, when only dichotomous measures were available, they were included to avoid systematically biasing the sample by excluding studies in which major depression was measured via clinician diagnoses.

When more than one continuous, self-report depression or anxiety measure was available (k = 5), the effect sizes based on these measures were averaged for analysis. By contrast, we decided against combining multiple PE measures because we were interested in the moderating effect of PE constructs (positive affect vs. extraversion vs. behavioral activation) on the relationship of PE to depression and anxiety. In other words, combining a measure of extraversion with a measure of positive affect would have prevented us from examining differences between these constructs across studies. Instead, for studies in which more than one PE measure was available (k = 4), we chose the measure that had the strongest cross-sectional relationship with depression or anxiety at Time 1. This enabled us to represent the strongest available relationship between PE and symptoms, without basing selection on the primary effect size of interest (i.e., the prospective, controlled relationship). Sensitivity analyses using the weakest available relationship between PE and symptoms resulted in effect sizes that were essentially unchanged.

Coding of time lags—For prospective analyses, the effect size for the shortest time lag in each study was coded. Additionally, we grouped the cross-lagged effect sizes into two categories for separate analyses: (1) time lags up to and including one year and (2) time lags over one year. These categories were not mutually exclusive; studies reporting data for more than one time lag (k = 14), of which one was up to a year and one was over a year, were included in both groups. We chose this method of examining time lags because it allowed us to evaluate the stability of cross-lagged effect sizes across more homogenous intervals and to utilize multiple effect sizes from individual studies where available.

Coding of moderators—For each effect size, the following moderators were coded:

PE constructs: We coded the type of PE construct (positive affect, extraversion, or behavioral activation) and whether the measure used state instructions (i.e., asked participants to rate their PE over a particular span of time) or trait instructions (i.e., asked participants to rate their general levels of PE). All measures with state instructions asked participants to rate PE within the past year or less.

Demographic and other characteristics of the sample: As this meta-analysis utilized longitudinal data with varying time lags, it was not possible to code participants' exact age. Instead, the sample was coded as child/adolescent or adult, as few studies differentiated between children and adolescents.

As the proportion of male and female participants differed at different time lags and precise demographic information was not available for data requested from authors, we coded studies whose sample consisted primarily (over 70%) of one sex. Only one study in the depression analysis was predominantly male, whereas 15 studies were predominantly female. We therefore examined the use of a predominantly female sample as a moderator.

We coded sample type, including clinical samples (recruited from mental health inpatient or outpatient settings), physical health samples (recruited from physical health inpatient or outpatient settings), students, and community/representative samples. We tested clinical status as a moderator by examining differences (1) between clinical samples and all other samples, and (2) between clinical and physical health samples and all other samples.

To examine whether studies with a restricted range of symptom levels differed from other studies, we coded whether the study (1) included only participants with depression or anxiety, or (2) excluded participants with baseline symptoms of depression.

Samples that experienced a defined life event between assessments (e.g., loss of a loved one, military deployment, birth of a child), received medical treatment, or had a high probability of receiving psychological treatment were coded as such. As these were all samples in which change between assessments was more likely than in other samples, they were examined together as one moderator.

Finally, we coded whether the sample consisted of a special population, including participants who were bereaved, caretakers of others, or selected on the basis of a current or former physical illness.

Study measures: We distinguished studies in which all measures were self-reported from studies in which the depression measure was other-reported (PE measures were always self-reported). We also distinguished studies in which all measures were continuous from studies in which the depression measure was originally dichotomous and presented continuously by request (i.e., symptom count from a clinical interview). No dichotomous depression measures were included in the sample of studies examined for moderators.

Methodological considerations: The following indicators of study quality were coded: study location (study conducted in the U.S. or Western Europe versus anywhere else), sample size, year of publication, total number of study waves (not only those for which effect size data were available), and percent of attrition in the study sample from the first assessment to the assessment used for analyses. To ensure that there were no systematic differences between studies that included all the necessary data to calculate effect sizes in the report versus studies that required author contact, we included this variable as a moderator as well.

Meta-Analytic Procedure

Effect size and moderation analyses were conducted using Comprehensive Meta-Analysis version 3.3.070 (Borenstein, Hedges, Higgins, & Rothstein, 2014). As correlational effect sizes have problematic standard error formulations in their standard form (Alexander, Scozzaro, & Borodkin, 1989), all effect sizes were transformed using Fisher's Z_r-transform (Hedges & Olkin, 1985), defined as:

$$\mathrm{ES}_{Zr} = .5 log_e \left[(1+r) / (1-r) \right]$$

where r is the reported correlation, $\log_e$ is the natural logarithm, and ES_{Zr} is the Fisher's Z_r-transformed correlation.

As recommended by Hedges and Olkin (1985), each effect size was weighted by the inverse of its within-study variance plus the between-studies variance (tau-squared). The within-study variance (w_{zr}) was calculated using the following formula for Fisher's Z_r-transformed correlation coefficients:

$$W_{zr}=n-3$$

where n is the sample size. This weighting procedure gives greater weight to larger samples than smaller samples (Lipsey & Wilson, 2001). For ease of interpretation, Fisher's Z_r-transformed correlations were transformed back into the standard correlational form for the presentation of results. Following two previous meta-analyses utilizing cross-lagged regression coefficients as effect sizes (Kuykendall, Tay, & Ng, 2015; Sowislo & Orth, 2013), cross-lagged effect sizes were also transformed using Fisher's Z_r-transform and weighted as described above. According to current guidelines (Borenstein, Hedges, Higgins, & Rothstein, 2009), all analyses were conducted using a random effects model, with the "method of moments" utilized to estimate between-study variance.

We compared corresponding effect sizes across depression and anxiety using robust variance estimation (Hedges, Tipton, & Johnson, 2010), implemented in the statistical program R (version 3.1.1) with the "robumeta" package. This technique, utilized in numerous meta-analyses (De Vibe, Bjørndal, Tipton, Hammerstrøm, & Kowalski, 2012; Oswald, Mitchell, Blanton, Jaccard, & Tetlock, 2013; Tanner-Smith, Wilson, & Lipsey, 2013), permits comparison of effect sizes across studies in which multiple, dependent effect sizes are drawn from the same sample. We used this method as some studies ($k = 20$) provided effect sizes for both depression and anxiety. The method requires a parameter estimate for the correlation between dependent effect sizes. Given typically high correlations between measures of depression and anxiety (Ruscio & Khazanov, 2016), we initially set $\rho = .7$; however, sensitivity analyses recommended by the developers revealed that results remained the same across all values of ρ (0–1).

Outliers—For each effect size, the sample-adjusted meta-analytic deviance (SAMD) statistic was calculated to test for the presence of statistical outliers (Huffcutt & Arthur, 1995). The SAMD statistic values approximate a normal t distribution. A more conservative cutoff score of 2.58 was used to consider studies for exclusion in order to avoid removing outliers whose effects represented true population variability, as extreme values can result from true population variability or error (Beal, Corey, & Dunlap, 2002). The SAMDs were rank-ordered and the scree plots examined. When an effect size's SAMD value was greater than 2.58, but the scree plot indicated that it was continuous with the overall distribution, the study was retained. Only effect sizes that were clearly discontinuous with the overall distribution and that had SAMD values over 2.58 were excluded.

Tests for heterogeneity—Heterogeneity among effect sizes was examined using both τ^2 (an estimate of between-study variance) and the I^2 statistic. The I^2 index quantifies the

degree of heterogeneity by describing the percentage of the variance attributable to between-study variance. I^2 values of 25%, 50%, and 75% indicate low, moderate, and high heterogeneity, respectively (Higgins & Thompson, 2002). As the I^2 index is influenced by sample size and can be biased in meta-analyses with small samples (von Hippel, 2015), we included confidence intervals for each effect size as well.

Moderators—Categorical moderators were tested using an analysis of variance (ANOVA) of mixed-effects models. Continuous moderators were tested using unrestricted maximum likelihood meta-regression. Given the large number of moderators tested, significant moderators were included in a single regression to test the predictive power of each, controlling for the effects of the others.

Publication bias—In order to reduce publication bias, we included unpublished dissertations and acquired effect size data from authors. Additionally, we tested for the presence of publication bias in several ways. First, we visually inspected a funnel plot, which plots the standard error for each study (determined by the study's sample size) against the study's effect size (Fisher's *Z*). In the absence of publication bias, the plot approximates a funnel shape, with large studies that provide a more reliable estimate of effect size clustered around the mean toward the top of the plot, and smaller studies that provide more variables estimates of effect size scattered more widely around the mean toward the bottom of the plot. In the presence of publication bias, the bottom of the plot appears asymmetrical. As the effect sizes in this analysis were expected to be negative, a pattern of more studies below than above the mean effect size would be the most likely indication of publication bias.

We also used Duval and Tweedie's (2000) trim-and-fill procedure to calculate the likely number of missing studies based on asymmetry in the funnel plot and to compute effect sizes adjusting for these missing studies. Additionally, we calculated the classic fail-safe *N* to determine the number of missing studies that would bring the *p* value above .05.

While the funnel plot and its associated tests are well-established methods of evaluating publication bias, these procedures rely on the assumption of homogeneity of effect sizes (Terrin, Schmid, Lau, & Olkin, 2003). As we expected to find heterogeneity among effect sizes, we also tested for publication bias by using publication type as a moderator, examining whether effect sizes reported in published studies differed significantly from effect sizes reported in unpublished studies (i.e., dissertations and the conference paper).

Results

Description of Studies

Studies providing data for PE and depression ($k = 59$; see Table 1) were published between 1994 and 2014, with a median publication year of 2010. Sample sizes ranged from 20 to 2,773 ($M = 415.31$, $SD = 542.74$, $Mdn = 223$). Time lags between assessments ranged from 1 to 228 months ($M = 18.41$, $SD = 40.46$, $Mdn = 7$). Only 73% of studies reported reliability coefficients (Cronbach's alpha) for measures within the study sample. Reliability

coefficients ranged from .68 to .95 (M = .86, SD = .07) for depression measures and from .60 to .96 (M = .81, SD = .09) for PE measures.

Studies providing data for PE and anxiety (k = 26; see Table 2) were also published between 1994 and 2014, with a median of 2010. Sample sizes ranged from 33 to 2,352 (M = 442.08, SD = 576.62, Mdn = 185.5). Time lags between assessments ranged from 1 to 60 months (M = 14.15, SD = 16.67, Mdn = 6). Reliability coefficients, reported by 81% of studies, ranged from .63 to .96 (M = .87, SD = .09) for anxiety measures and from .68 to .96 (M = .84, SD = .07) for PE measures. Of the 26 studies that assessed anxiety, six used measures that primarily (over 75% of items) assessed fear (e.g., racing heart), and three used measures that primarily assessed anxious distress (e.g., feeling worried or upset). As the remaining 17 studies used measures assessing fear and distress in more equal proportions, we treated anxiety as a unitary construct rather than examining fear and distress separately.

Preliminary Analyses

Preliminary analyses identified 1–2 outliers in 5 (out of 10) analyses (for details, see Tables 3 and 4). After original analyses were run without these outliers, sensitivity analyses were run with outliers included. All effect sizes with outliers included were within .01 of the original effect size values and remained statistically significant ($p < .05$).

The two main analyses evaluating the cross-lagged effect of PE at Time 1 on depression or anxiety at Time 2 showed no evidence of publication bias. The funnel plots appeared symmetrical (see Figures 2 and 3). Following Duval and Tweedie's (2000) trim-and-fill procedure, we used a random effects model to look for missing studies to the right of the mean. We found no evidence of missing studies in either of the two main analyses. Next, we calculated the classic fail-safe N. This test indicated that 1,370 studies and 190 studies would be required to reduce to nonsignificance the relationships of PE with depression and anxiety, respectively. Lastly, we evaluated publication type as a moderator of the relationships of PE with depression and anxiety. The effect sizes based on published and unpublished studies did not differ significantly for either depression ($Q(1) = .06$, $p = .812$) or anxiety ($Q(1) = .10$, $p = .753$).

Effect Size Analyses

PE as a predictor and outcome of depression—Table 3 displays the weighted mean effect sizes for the relationship of PE with depression. Consistent with our hypotheses, PE was related to depression in cross-sectional ($r = -.34$) and prospective, uncontrolled ($r = -.26$) analyses. These moderate effects diminished to a far smaller, though still reliable, association (−.08) in cross-lagged analyses that controlled for initial depression symptoms. Contrary to our hypotheses, depression predicted changes in PE to the same extent as PE predicted changes in depression: The uncontrolled (−.28 and −.26, respectively) and controlled (−.07 and −.08) effect sizes were nearly identical in both directions.[3]

[3]To rule out differences in study characteristics as an explanation for differences between effect sizes, we reran these analyses using a common set of studies for which all five effect sizes could be calculated (k = 31). The resulting effect sizes were very similar to those presented in the paper and the overall pattern of results was unchanged (see Supplementary Table 3).

Specificity to depression: Relationship between PE and anxiety—Table 4 displays parallel effect sizes for the relationship of PE with anxiety. In a pattern very similar to depression, PE was related to anxiety in cross-sectional ($r = -.24$) and prospective, uncontrolled ($r = -.19$) analyses. Controlling for initial anxiety symptoms diminished the cross-lagged relationship between PE and anxiety to −.06.[4] Importantly, none of the three effect sizes (cross-sectional, uncontrolled prospective, or controlled prospective) were significantly larger for depression than for anxiety, all $\beta < -.074$, all $p > .10$, based on robust variance estimation.[5] Additionally, the bidirectional paths observed between PE and depression were also evident for anxiety: Anxiety predicted, and was predicted by, PE to a very similar degree in both uncontrolled (−.19 and −.23, respectively) and controlled (−.06 and −.09, respectively) analyses.

Stability of controlled effect sizes across time lags—To examine the stability of the cross-lagged effects across different time lags, we recalculated the effects separately for inter-assessment intervals (a) up to and including one year and (b) more than one year, using the effect size closest to the time lag of interest (one year or five years). The prospective, controlled relationships between PE and depression were remarkably stable, regardless of the time frame that separated the assessments. Across all studies, the relationship (95% CI in brackets) between PE at Time 1 and depression at Time 2 was −.08 ([−.09, −.06], $k = 58$, *Mdn* time lag = 6.5 months). For studies with intervals up to one year, the effect size was −.08 ([−.10, −.06], $k = 48$, *Mdn* = 12 months) and for studies with intervals over one year, the effect size was −.08 ([−.10, −.05], $k = 24$, *Mdn* = 36 months). The relationship was equally stable in the opposite direction: Across all studies, the relationship between depression at Time 1 and PE at Time 2 was −.07 ([−.09, −.04], $k = 33$, *Mdn* = 6 months). For studies with intervals up to one year, the effect size was −.06 ([−.09, −.03, $k = 27$, *Mdn* = 12 months) and for studies with intervals over one year, the effect size was −.08 ([−.13, −.04], $k = 15$, *Mdn* = 36 months).

The prospective, controlled relationships between PE and anxiety were also quite stable over time, although results should be interpreted with caution given the small number of effect sizes available for some analyses. Across all studies, the relationship between PE at Time 1 and anxiety at Time 2 was −.06 ([−.09, −.04], $k = 26$, *Mdn* time lag = 6 months). For studies with intervals up to one year, the effect size was −.05 ([−.09, −.02], $k = 20$, *Mdn* = 6 months) and for studies with intervals over one year, the effect size was −.08 ([−.10, −.05], $k = 11$, *Mdn* = 38 months). Reversing the direction, the relationship between anxiety at Time 1 and PE at Time 2 was −.09 ([−.11, −.06], $k = 15$, *Mdn* = 5 months). For studies with intervals up to one year, the effect size was, −.04 ([−.07, −.002], $k = 12$, *Mdn* = 5 months) and for studies with intervals over one year, the effect size was −.13 ([−.19, −.08], $k = 7$, *Mdn* = 42 months).

[4]To ensure that Fisher's *Zr* transformation was not biasing the cross-lagged effect sizes, we recomputed them without first transforming each correlation. These untransformed effect sizes were essentially the same as those reported in the paper.

[5]We reran these analyses using only the studies that provided effect size data for both depression and anxiety. In this small sample (k ranged from 10–20, including just one-third of available depression studies), the effect sizes were very similar to those presented in the paper, although the cross-sectional and prospective, uncontrolled relationships between PE at Time 1 and depression at Time 2 were larger than the corresponding relationships between PE and anxiety when compared using robust variance estimation, both $\beta > -.09$, both $p < .01$ (see Supplementary Table 4).

Moderator Analyses

Tests of individual moderators—We tested moderators of the main relationship of interest: the cross-lagged relationship between PE at Time 1 and depression at T2. The amount of heterogeneity in this effect size estimate was small (28%), hinting that moderation effects were likely to be modest.

Consistent with this account, only two variables emerged as significant moderators of the cross-lagged relationship between PE and depression (see Table 5). The first was sample age: The link between PE and depression was stronger for adults (β = −.09 [−.11, −.07], p < .001, k = 42) than for children and adolescents (β = −.04 [−.07, −.01], p =.005, k = 15), $Q(1)$ = 8.45, p = .004.[6]

The second significant moderator was having an experience between assessments that posed a high likelihood of symptom change. Samples comprising individuals who experienced a defined life event, medical treatment, or the possibility of psychological treatment yielded larger effect sizes (β = −.11 [−.14, −.08], p < .001, k = 17) compared to other samples (β = −.06 [−.08, −.04], p < .001, k = 41), $Q(1)$ = 8.46, p = .004. Separate follow-up analyses found each type of inter-assessment change to be associated with a larger effect size compared to all other studies. These differences, however, were only statistically reliable when all types of change were analyzed together. Interestingly, the moderating effect of defined life events was similar whether the event was negative (e.g., military deployment) or positive (e.g., graduating from high school), $Q(2)$ = 4.10, p = .129.

We found no difference in the relationship between PE and depression depending on the operationalization of PE. This was the case even though we increased our power to detect a difference by increasing the number of behavioral activation effect sizes from 2 (in our main depression analyses) to 6, substituting a behavioral activation effect size for the 4 other studies that provided one (starred in Table 1). Similarly, moderation analyses revealed no significant differences by sample characteristics other than age, by study measures, or by any of the indicators of study quality evaluated. Importantly, effect sizes did not differ depending on whether data were published or requested from authors, supporting the decision to analyze them together.

Final model—As sample age and inter-assessment change both emerged as significant moderators, we included them together in a single regression analysis predicting the magnitude of the cross-lagged relationship between PE and depression. Each moderator remained statistically significant when levels of the other moderator were controlled, $Q(2)$ = 18.15, p < .001. The two moderators accounted for roughly equal proportions of the variance in the effect size, with slight changes depending on the order in which they were entered into the regression. Together, they accounted for 83% of the variance in the prospective, controlled relationship between PE and depression, leaving no unexplained heterogeneity, $Q(54)$ = 57.53, p = .346; I^2 for the final model = 6.14%. That is, the model with these two

[6]Zdanowicz et al. (2012) was excluded from this analysis, as the sample included both adolescents and adults.

predictors accounted for 83% of the unexplained variance (τ^2) in the model without these predictors.

Discussion

In a series of meta-analyses, we quantified the cross-sectional, prospective, and cross-lagged relationships between PE and depression and evaluated their specificity vis-à-vis anxiety. PE shared a moderate cross-sectional relationship and a small to moderate prospective relationship with depression. The prospective relationship was markedly attenuated, however, once initial levels of depression were controlled. Furthermore, PE predicted change in depression to the same extent that depression predicted change in PE. Finally, PE predicted change in depression to the same extent that it predicted change in anxiety. These results were consistent across shorter (up to one year) and longer (more than one year) assessment intervals and across differing operationalizations of PE. The PE-depression relationship was stronger for adults than for children and adolescents, and stronger for samples with a high probability of inter-assessment symptom change than for other samples. Despite these differences, in all samples lower levels of PE reliably, but weakly, predicted subsequent increases in depression.

Relationship of PE to Depression

The moderate cross-sectional relationship observed here between PE and depression ($r = -.34$) was similar to the continuous effect sizes reported in a recent meta-analysis ($r = -.25$ to $-.29$, depending on type of depressive disorder; Kotov et al., 2010). Building on these results, we found that the prospective relationship of PE to depression was also robust ($r = -.26$). Demonstrating the temporal precedence of PE is an important precondition for establishing vulnerability, but it is not sufficient, given the cross-sectional relationship between PE and depression and the persistence of depression over time. In our sample of studies, for example, the correlation between depression at Time 1 and Time 2 was .56 ($k = 57$, 95% CI [.53, .59], $\tau^2 = 0.02$).

We undertook a more rigorous test of the vulnerability hypothesis by controlling for initial levels of depression, even when the original study did not include this control. That PE remained a significant predictor in this conservative analysis provides the most compelling psychometric evidence to date for its status as a risk factor for depression. At the same time, the large reduction in effect size that occurred when baseline symptoms were controlled suggests that the risk conferred by low PE may be much smaller than has previously been suggested. In addition, our finding that the cross-lagged relationship of PE to depression was nearly identical to the cross-lagged relationship of depression to PE suggests that PE is as likely to be a consequence as a cause of depression.

Explanation of Findings

What might account for the departure of our findings from extant theories of PE and depression? There are several possible explanations. First, it is rare in psychopathology for a single risk factor to have large and specific effects (Coie et al., 1993). This does not preclude the possibility that more potent risk factors than PE exist, and future research should be

directed in part toward identifying the strongest predictors of depression. Nevertheless, comprehensive models will almost certainly need to consider multiple risk factors and their interactions in order to powerfully predict symptom change (D. N. Klein et al., 2011; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001).

Second, it is possible that the constructs of PE and depression are overly broad and that stronger cross-lagged relationships exist between specific aspects of these constructs (D. N. Klein et al., 2011; Watson & Naragon-Gainey, 2010). Although we did not find stronger associations of depression with the narrower construct of positive affect than with the broader construct of extraversion, positive affect is itself composed of several facets that may relate differentially to depression. For example, the positive affect facet of Joviality is associated more strongly with depression than are Self-Assurance and Attentiveness (Stanton & Watson, 2014), and the positive emotion of pride is associated more strongly with depression than are happiness and amusement (Gruber, Oveis, Keltner, & Johnson, 2011). Additionally, we focused on global levels of PE and not on PE responses to specific stimuli, which can be divided into emotional responses during anticipation, reaction, and recall of a stimulus (Gilbert, 2012). Preliminary evidence that depression relates more strongly to some phases of positive emotional responding (e.g., anticipation) than others (e.g., reaction; D. Gard, M. Gard, Kring, & John, 2006) highlights the importance of examining each temporal aspect of PE separately. Similarly, heterogeneity in the expressed features, pathophysiology, and etiology of depression (Monroe & Anderson, 2015) limits the power of any individual risk factor to predict change in depression over time. Given this heterogeneity, progress may be accelerated by investigating associations of PE with more homogenous subsets of depression symptoms. This approach is supported by evidence that some depression symptoms (anhedonia, depressed mood, worthlessness, and lassitude) are more strongly related to PE than others (motor, sleep, and appetite disturbance; Watson & Naragon-Gainey, 2010).

Third, it is possible that behavioral measures of PE will be revealed to predict depression more powerfully than the self-report measures investigated here. Behavioral PE measures include tasks evaluating reward learning, effort exertion for rewards, and facial expressions in response to pleasant stimuli (Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008; Rottenberg et al., 2002; Treadway, Buckholtz, Schwartzman, Lambert, & Zald, 2009). While several studies have found that behavioral measures of PE prospectively predict depression (Pizzagalli, 2014; Rottenberg et al., 2002), substantial differences between these measures and uncertainty over their relationship to self-reported PE (Forbes & Dahl, 2005; Pizzagalli, 2014) impede a meaningful quantitative synthesis across studies. In addition, behavioral measures of PE might be expected to yield smaller effect sizes than self-report measures, simply because the latter share method variance with self-reported depression symptoms. That said, behavioral measures deserve more attention in view of advantages they offer over global self-report measures. In particular, they map more directly onto dimensions of reward processing that are disrupted in depression (Der-Avakian & Markou, 2012), such as motivation for, engagement with, and learning from positive stimuli (Proceedings, 2011; Treadway & Zald, 2011). These dimensions are conceptually very similar to PE, and clarifying their relationship to PE would help bridge the burgeoning literature on reward processing with the vast literatures on normal personality and emotion. In doing so,

behavioral measures could help illuminate behavioral and neurobiological mechanisms linking PE to psychopathology.

Fourth, prospective relationships between PE and depression may have been weakened by effects of current mood on PE levels. Although we controlled for *initial* symptoms of depression, we were unable to rule out the possibility that *concurrent* symptoms of depression biased participants' reports of PE (Clark, Vittengl, Kraft, & Jarrett, 2003). With the use of repeated assessments, a recent study applied a statistical method for isolating the influence of time-invariant (as opposed to transient, mood-state-dependent) aspects of personality traits on disorder symptoms (Naragon-Gainey et al., 2013). Isolating the time-invariant component of PE enhanced its ability to predict the longitudinal course of depression. Another study using this method found that the time-invariant component of PE also predicted the first onset of depressive disorders (Kendall et al., 2015). These results suggest that state variance may dilute the association of PE with depression, especially in samples with high levels of depression whose responses are influenced by extreme mood states. Our moderation analyses found no evidence of a weaker cross-lagged relationship between PE and depression for clinical relative to nonclinical samples, or for depressed/anxious samples relative to all other samples. However, we could not ascertain whether mood state was more variable and led to more extreme changes in PE depending on mood in clinical than nonclinical participants. It therefore remains possible that the influence of current mood state on reports of PE weakened PE's ability to predict depression.

The acknowledgment that PE has both trait and state components could help explain the discrepancy between our findings of the nonspecificity of PE and the many cross-sectional studies showing relative specificity of PE to depression vis-à-vis anxiety (Clark et al., 1994; Malouff et al., 2005; Mineka et al., 1998; Watson et al., 2005). As depression, unlike anxiety, includes symptoms reflecting low PE, it is possible that the state component of PE inflates its cross-sectional association with depression relative to anxiety. In longitudinal studies in which PE and symptoms are assessed months or even years apart, the influence of this state component on symptom reports is reduced.

Theoretical Implications

Perhaps the most striking departure from existing theories was not the magnitude of the observed effects, but their remarkable similarity in both temporal directions. Our results showed clearly that PE is as likely to be an outcome of depression (consistent with the scar and complication models) as a vulnerability factor (consistent with the predisposition and pathoplasty models). This suggests that theories conceptualizing PE largely as a risk factor for depression should be broadened to recognize and explain bidirectional influences between the two constructs. The nature and form of these influences, however, remain to be described. As our analysis focused on symptom, rather than diagnostic, measures of depression, we were unable to distinguish between the predisposition model (which asserts that PE increases risk of disorder onset) and the pathoplasty model (which asserts that PE influences the course and expression of the disorder). Past research suggests that both models are plausible (Kendall et al., 2015; D. N. Klein et al., 2011), and as they have

different implications for intervention, studies capable of discriminating between them are needed.

For similar reasons, our analysis cannot distinguish the scar model (in which declines in PE persist even after the disorder has resolved) from the complication model (in which declines in PE are temporary and last only while the illness is active). Still, our discovery that depression predicts subsequent reductions in PE over periods longer than a year hints that changes in PE are not entirely temporary. Further investigation of low PE as a sequela of depression would help clarify these and related issues, such as whether declines in PE resulting from previous depressive episodes increase risk for new depressive episodes, and whether the mechanisms involved are the same in each temporal direction.

Our findings also have implications for two other models of the personality-psychopathology relationship. The spectrum model suggests that disorders and traits are different manifestations of the same process; this model would be supported by high, specific correlations between PE and depression (Kotov et al., 2010). As even the cross-sectional correlation between PE and depression was moderate in the present analysis ($r = -.34$), our results do not support this model. On the other hand, our results are broadly compatible with the common cause model, which posits that disorders and traits are associated because of shared genetic or environmental vulnerabilities. Evidence for this model comes from twin studies showing that PE and depression share a modest genetic correlation (Kendler, Gatz, Gardner, & Pedersen, 2006; Kendler & Myers, 2010). Meta-analyzing longitudinal studies for other personality traits (e.g., negative emotionality), as well as other forms of psychopathology, would shed further light on the important and complex relationship between personality and psychopathology.

Moderators of the Pathway from PE to Depression

A complete theoretical account of PE as a risk factor for depression will need to address the moderating influences identified here. We provided quantitative evidence for prior suggestions that PE is a stronger predictor of depression in adults than in children and adolescents (De Bolle & De Fruyt, 2010). The weaker relationship between PE and depression in youth could have a number of explanations, including differing manifestations of depression in children and adolescents relative to adults (De Bolle & De Fruyt, 2010; Jacques & Mash, 2004), different causal factors for early-onset relative to late-onset depression (Shankman, Klein, Tenke, & Bruder, 2007), or decreased stability of PE in childhood (Roberts & DelVecchio, 2000). While behavioral measures of low PE like reduced pursuit of positive stimuli and lower observer ratings of positive emotional expression have also been shown to predict depressive symptoms in youth (Dougherty, Klein, Durbin, Hayden, & Olino, 2010; Forbes et al., 2007; Rawal et al., 2013), we do not yet know whether these measures are more robust predictors of depression than self-reported PE. If this is found to be the case, it may indicate poorer validity of self-report relative to behavioral measures of PE in children and adolescents.

We also found a stronger relationship between PE and depression in samples with an increased likelihood of inter-assessment change, including samples that experienced a defined life event, received medical treatment, or had a high probability of receiving

psychological treatment. This finding is consistent with research showing that the relationship between traits and mental disorders are stronger in the context of adverse life experiences (Clark, 2005; Geschwind et al., 2010). Life experiences, whether adverse or favorable (e.g., treatment), may amplify the relationship between PE and depression by increasing the need for adaptive information processing, coping strategies, and emotion regulation – all processes influenced by PE (Folkman & Moskowitz, 2000). Our moderation results may help explain inconsistencies across studies regarding the status of PE as a risk factor, as many, but not all, studies have relied on samples of individuals recruited in settings in which the possibility of treatment was high (Clark et al., 1994; Enns & Cox, 1997; Shankman & Klein, 2003; Watson & Naragon-Gainey, 2010). Unlike previous meta-analyses (Kashdan, 2007; Kotov et al., 2010), however, we did not find a reliably stronger relationship between PE and depression in clinical than nonclinical samples, perhaps because of insufficient statistical power to detect these differences or our focus on longitudinal, as opposed to cross-sectional, relationships.

Contrary to expectations, neither the type of PE construct (positive affect, extraversion, behavioral activation) nor the time span of PE measurement (trait, state) moderated the relationship between PE and depression. This contradicts several studies in which depression related more strongly to positive affect than to the broader trait of extraversion (Naragon-Gainey et al., 2009; Watson & Naragon-Gainey, 2014; Watson et al., 2015). A possible reason is that those studies distinguished between facets of extraversion, whereas our analysis focused on the far larger number of studies that used complete measures of positive affect and extraversion. The present results also run counter to suggestions that depression relates more strongly to behavioral activation than to extraversion (Shankman & Klein, 2003), although the relatively small number of prospective studies measuring behavioral activation left us with low power to detect these differences. Based on the available evidence, the consistency of effect sizes across constructs and time spans suggests that the relationship of PE to depression is quite stable regardless of how PE is measured. Further evidence of stability was provided by showing that the relationship does not vary by features of study measures or indicators of study quality.

Specificity of PE as a Risk Factor for Depression Relative to Anxiety

We found a cross-sectional relationship of PE to anxiety ($r = -.24$) that was not reliably different from the cross-sectional relationship of PE to depression ($r = -.34$). This aligns with the results of a previous meta-analysis (Kotov et al., 2010), which reported correlations of a similar magnitude for PE with depression ($r = -.25$ to $-.29$) and several anxiety disorders ($r = -.18$ to $-.37$, excluding specific phobia). In the current study, the same pattern held when we examined the prospective and the cross-lagged relationships between these constructs. In each analysis, the effect sizes for depression and anxiety were statistically indistinguishable. Contrary to our hypothesis and prominent theoretical models of emotional disorders, these findings challenge the notion that PE is a specific risk factor for depression relative to anxiety.

These longitudinal results extend prior claims that low PE is a feature of certain anxiety disorders (Bienvenu & Stein, 2003; Watson & Naragon-Gainey, 2010) to suggest that PE be

reframed as a general vulnerability factor for emotional disturbance rather than a specific vulnerability factor for depression. These results align with a growing recognition of dimensions of psychopathology (Insel, 2014) and associated maintaining processes (Harvey, 2004) that cut across disorder categories. To date, research on dimensions of temperament that increase vulnerability for both depression and anxiety has focused mainly on negative emotionality (Barlow, Ellard, Sauer-Zavala, Bullis, & Carl, 2014; Lahey, 2009). By contrast, deficits in PE have more commonly been studied in connection with depression and schizophrenia (Proceedings, 2011), although most studies have examined low PE as a feature of these disorders rather than a risk factor for them. Our results indicate that it will be important to extend research on PE to anxiety.

A further priority will be to identify variables that account for the development of each disorder in the presence of low PE. Due to the small number of available anxiety studies as well as our principal interest in the PE-depression relationship, we did not examine moderators of the PE-anxiety relationship here. As more longitudinal studies become available, it will be valuable to explore differences in moderators and mechanisms of the pathways connecting PE to depression, anxiety, and other conditions (schizophrenia, mania) in which PE has been implicated (Gruber et al., 2008; Horan, Blanchard, Clark, & Green, 2008; Watson & Naragon-Gainey, 2010).

Clinical Implications

The present findings have implications for the prevention and treatment of depression and anxiety. We found that PE levels were quite stable over time (the correlation between PE at Time 1 and Time 2 was .61, $k = 32$, 95% CI [.57, .65], $\tau^2 = 0.03$) despite studies with long times lags, samples experiencing defined life events, and effects based on state as well as trait measures included in the analysis. This stability, together with evidence that PE precedes and predicts increases in depression and anxiety, suggests that PE could be used to identify individuals at risk for depression. Despite PE's stability, studies have found that standard pharmacological and psychological treatments for depression can raise levels of PE (Du, Bakish, Ravindran, & Hrdina, 2002; Tang et al., 2009). These findings speak to the possibility of intervening to increase PE with the goal of preventing the onset or recurrence of depression and anxiety. In fact, recognition that conventional treatments, which focus mainly on reducing negative emotions, may not remedy deficits in positive emotions has spurred the development of interventions that target PE. These interventions seek to enhance and prolong PE by encouraging patients to savor and actively anticipate positive experiences and by removing barriers to PE expression. Preliminary evidence suggests that these types of interventions increase PE and decrease symptoms among currently depressed and anxious individuals (Carl, 2015; McMakin, Siegle, & Shirk, 2011). Although clinical trials are needed, these results highlight the potential of PE-focused interventions to alleviate and perhaps also prevent symptoms of depression and anxiety.

At the same time, the modest magnitude of the longitudinal, controlled relationship observed in this synthesis suggests that risk factors other than PE play a significant role. Previous research has identified numerous individual (e.g., negative emotionality, self-esteem) and environmental (e.g., early life experiences, social support) risk factors for depression.

Theoretical models and prevention programs will benefit from further investigation of how these risk factors operate with or through PE to increase vulnerability for emotional disorders (e.g., Gershuny & Sher, 1998; Joiner & Lonigan, 2000). This will allow prevention programs to be offered to individuals whose vulnerability is especially high, and will identify the set of modifiable risk factors that are most important for such programs to address.

Limitations

The present results are qualified by several important limitations. First, we cannot reach strong conclusions regarding causality because all studies used correlational designs. In the absence of experimental manipulation, it is possible that a third variable was responsible for these effects, especially in light of the modest reciprocal effects observed here. One particularly important third variable is negative emotionality. PE, depression, and anxiety all correlate with negative emotionality, and the extent to which negative emotionality accounts for relationships among these constructs is unclear (Kendall et al., 2015; Naragon-Gainey et al., 2013; Watson & Naragon-Gainey, 2014). Additional prospective studies assessing negative emotionality in conjunction with PE, depression, and anxiety would permit a more systematic evaluation of these relationships. Extending this work to consider other variables associated with both PE and depression, such as stress, substance use, and conscientiousness, would further clarify the contribution of third variables to these relationships. Although correlational findings cannot speak decisively to causality, our analysis of longitudinal studies enabled us to go a step beyond meta-analyses of cross-sectional data in evaluating PE as a risk factor and consequence of depression. Future research can more directly test low PE as a cause of symptoms through increased use of randomized controlled prevention or intervention trials, quasi-experimental designs, and statistical matching methods (Jaffee, Strait, & Odgers, 2012).

Second, we did not determine whether PE predicts change in depression more strongly than depression predicts change in PE when both paths are considered simultaneously. Our use of cross-lagged regression effect sizes allowed us to account for the stability of the outcome variable when estimating the relationship between each predictor and outcome. This approach did not, however, allow us to adjust for the predictor variable at Time 2 or to pit one cross-lagged effect size against the other in the same analysis. Although our finding of nearly identical cross-lagged effect sizes suggests that PE predicts change in depression to much the same extent as depression predicts change in PE, it is possible that simultaneous estimation of both paths would have revealed a stronger effect for one than the other.

A third set of limitations stems from our focus on symptoms of depression and anxiety rather than on diagnoses of clinical disorders. As noted earlier, our focus on sympom measures meant that we could not distinguish between competing theoretical models whose predictions are linked to the timing of disorder onset and remission. Additionally, although psychological scientists may seek to understand the full range of outcomes with which PE is associated, clinical interest in PE as a risk factor may depend on its prediction of clinically significant disorders. Finally, two prior meta-analyses found that relationships between personality and psychopathology were stronger when focusing on disorders versus

symptoms (Malouff et al., 2005; Ruiz et al., 2008). This hints that our effect sizes might have been larger had we evaluated the associations of PE with depressive and anxiety disorders rather than with symptoms.

A related limitation arises from our use of general, rather than specific, measures of anxiety. Although theoretical models posit that low PE is a risk factor for depression versus anxiety conceptualized generally (e.g., Watson & Naragon-Gainey, 2010), there is evidence that PE may share stronger associations with some anxiety disorders than others (Bienvenu & Stein, 2003; Kashdan, 2007). It is also possible that PE is related more strongly to disorders or symptoms characterized by distress (major depressive disorder, generalized anxiety disorder, posttraumatic stress disorder) than those characterized by fear (panic disorder, social anxiety disorder, specific phobia), although the available cross-sectional data do not support this assertion (Kotov et al., 2010). Our hope is that the current study helps stimulate interest in the relationship between PE and anxiety so that future meta-analyses can test whether PE better predicts some forms of anxiety than others.

Despite these limitations, a focus on general depression and anxiety symptoms has some significant advantages. Studies that test theories of PE as a risk factor for depression typically utilize symptom measures (Fowles, 1994; Gray, 1994; Watson, 2009; Watson et al., 2005); as a result, the number of effect sizes available for symptom measures far outnumber those available for diagnostic measures. Similarly, many more effect sizes are available for general, rather than specific, anxiety measures. Furthermore, assessments of symptoms are usually more reliable than dichotomous, disorder-based measures (Chmielewski et al., 2015; Naragon-Gainey et al., 2009). Importantly, our finding that PE is not specific to depression is consistent with a recent meta-analysis of cross-sectional studies that used diagnostic rather than symptom measures (Kotov et al., 2010). That meta-analysis found that nearly all anxiety disorders, as well as depressive disorders, were associated with decreased levels of PE, suggesting that the pattern of effects observed here may generalize to diagnostic measures and to clinical presentations characterized by distress or fear.

Fourth, the majority of studies in this meta-analysis used self-report measures. Although effect sizes did not differ for studies using self- versus other-report, it is possible that the preponderance of self-report measures inflated estimates of effect size due to shared method variance. This limitation is more applicable to the cross-sectional and uncontrolled prospective relationships, as shared method variance was reduced for cross-lagged effects by controlling for prior levels of the outcome variable (Sowislo & Orth, 2013). It is possible that this control for shared method variance was partly responsible for the attenuation in effect size between the prospective, uncontrolled relationship and the cross-lagged relationship of PE with depression.

Fifth, while we tested the relationship of PE to both depression and anxiety, it is important to keep in mind that there is significant content overlap in these measures (Watson et al., 1995). This likely limited our ability to detect differential relationships with PE. That said, these measures reflect the typical assessment of depression and anxiety in the literature, and they include assessment of non-overlapping as well as overlapping symptoms (Watson, 2009).

More widespread use of measures that better distinguish between depression and anxiety would enhance future efforts to evaluate specificity (Watson et al., 1995).

A sixth limitation concerns the relatively small number of studies included in the meta-analysis. This was especially a limitation of the anxiety analyses, but may have also limited our ability to detect more modest moderation effects and the influence of moderators that were represented by relatively few studies (e.g., behavioral activation measures of PE). Additionally, the cross-sectional effect sizes presented in this paper were derived only from studies including a longitudinal component and therefore do not represent an exhaustive review of the literature.

Finally, we did not test for mediators of the relationships of PE to depression and anxiety. Surprisingly few studies have examined mediators of these relationships, although this work is beginning to be done. For example, there is some evidence that the relationship between low PE and later depression is mediated by supportive relationships (Finch & Graziano, 2001; Wetter & Hankin, 2009). It will be important for future research to identify constructs that account for the link between PE and symptoms, as these constructs represent potential targets for treatment.

Even with consideration of these limitations, this meta-analysis is the first to quantify the prospective relationship between a broad personality dimension and symptoms of depression or anxiety. Strengths of this analysis included the examination of longitudinal as well as cross-sectional relationships between PE and symptoms, the rigorous evaluation of PE as a risk factor by controlling for initial symptom levels, the evaluation of directionality by testing symptoms as risk factors for PE, and the evaluation of specificity by testing relationships with anxiety as well as depression. Our results provide support for theoretical accounts that cast low PE as a vulnerability factor for depression. At the same time, these results highlight the need to identify additional vulnerability factors, understand the mechanisms responsible for bidirectional relationships between personality and symptoms, and extend this work to other forms of psychopathology in which PE plays a role.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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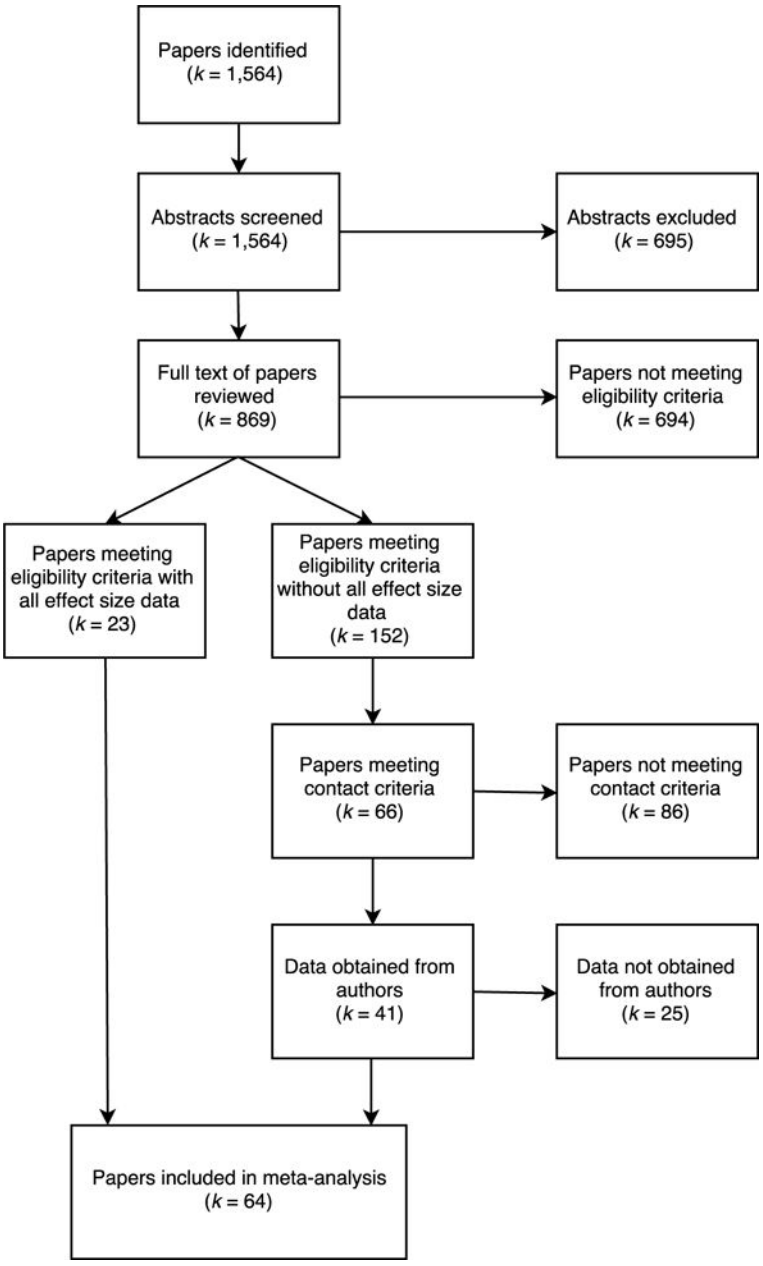

Figure 1.
Flow diagram for inclusion and exclusion in meta-analysis.

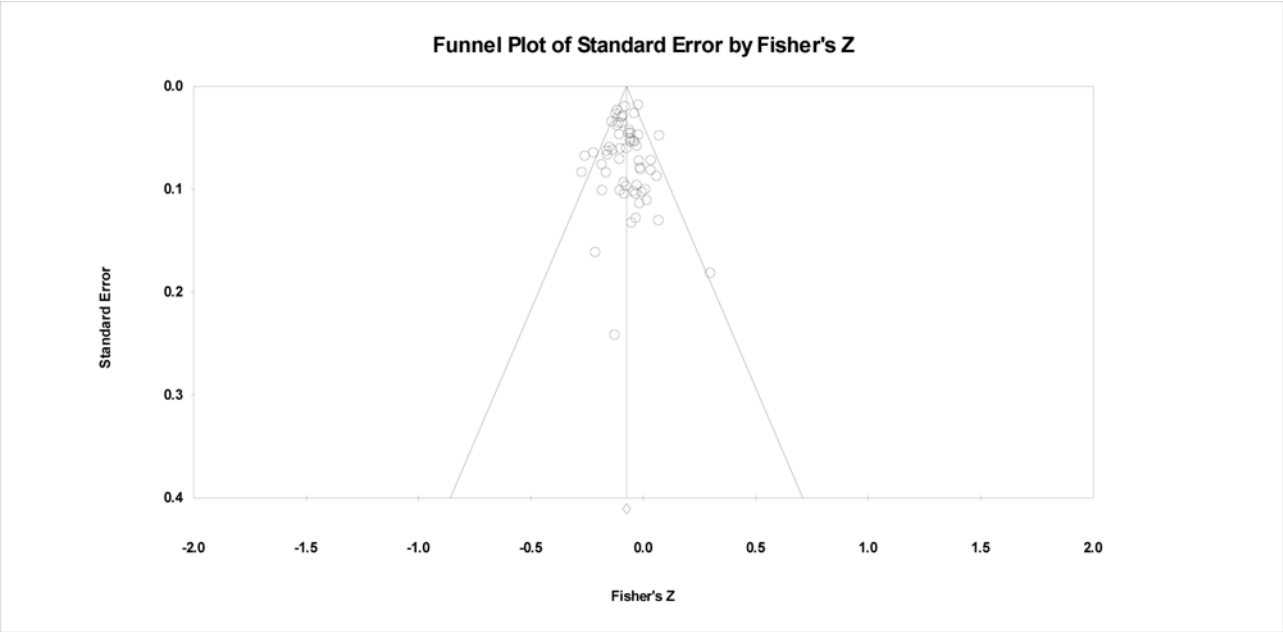

Figure 2.
Funnel plot for the relationship between PE and depression, controlling for initial symptoms of depression.

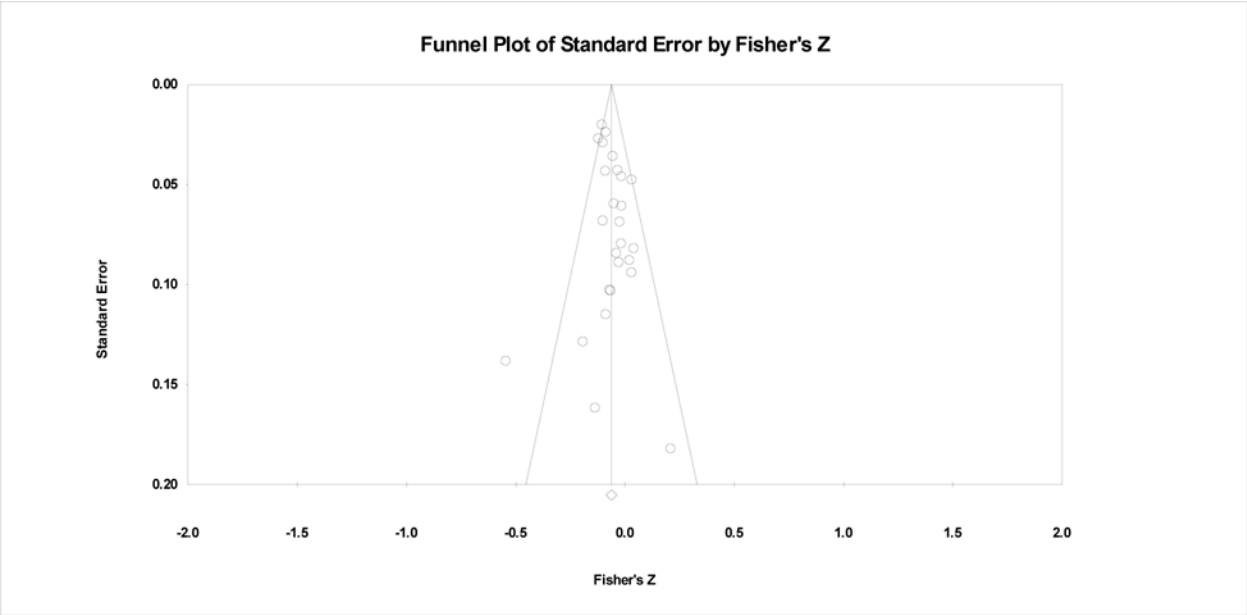

Figure 3.
Funnel plot for the relationship between PE and anxiety, controlling for initial symptoms of anxiety.

Table 1

Longitudinal Studies of the Relation Between Positive Emotionality and Depression

	Sample Characteristics							Effect Sizes				
Study	***N***	**Time lag in months**	**Age**	**Sample Type**	**Positive Emotionality Construct**	**Trait or State**	**Inter-assessment change**	**Cross-sectional[a]**	**PE ➔ D[a]**	**PE ➔ D, controlled[b]**	**D ➔PE[a]**	**D ➔ PE, controlled[b]**
Bardi & Ryff, 2007	280	8	Adult	Other	Extraversion	Trait	Yes	−.24	−.26	−.15		
Beran, 2009	79	6	Adult	Physical health	Extraversion	Trait	No	−.30	−.20	−.01		
Billstedt et al., 2014	274	288	Adult	Other	Extraversion	Trait	No	−.15			−.12	−.03
Bornstein, 2003	255	3	Adult	Other	Extraversion	Trait	No	−.46	−.37	−.13		
Brown, 2007[c]	197	12	Adult	Clinical	Positive Affect	Trait	Yes	−.46	−.35	−.10	−.35	−.03
Buffington, 2012[c]	236	2	Adult	Other	Positive Affect	Trait	No	−.41	−.42	−.21		
Chien et al., 2007	1,348	12	Adult	Other	Extraversion	Trait	No	−.19	−.14	−.03		
Christie, 2011	153	4	Adult	Physical health	Positive Affect	State	No	−.68	−.33	−.01	−.36	.10
Coleman & Neimeyer, 2010	99	12	Adult	Other	Positive Affect	State	No	−.30	−.35	−.18	−.33	−.15
De Bolle et al., 2011	150	12	Child/Ad	Clinical	Positive Affect	Trait	Yes	−.44	−.25	.04	−.23	−.05
Dill, 2005	1,146	12	Child/Ad	Other	Extraversion	State	Yes	−.38	−.23	−.09	−.22	−.05
Duberstein et al., 2008	223	60	Adult	Other	Extraversion	Trait	No	−.09	−.19	−.15		
Fox, 2010	131	12	Child/Ad	Other	Positive Affect	Trait	No	−.19	−.03	.06		
García-Peña et al., 2013	2,352	12	Adult	Other	Positive Affect	State	No	−.43	−.34	−.08	−.48	−.44
Gershuny & Sher, 1998	466	36	Adult	Other	Extraversion	Trait	No	−.08	−.08	−.05		
Gudiño et al., 2012	159	6	Child/Ad	Other	BAS	Trait	No	.16	.08	−.01		
Haase et al., 2012	752	12	Adult	Other	Positive Affect	State	Yes	−.37	−.26	−.10	−.30	−.17
Hart & Charles, 2013	59	6	Adult	Physical health	Positive Affect	State	Yes	−.49	−.34	−.05	−.17	.03
Hou et al., 2010	215	3	Adult	Physical health	Positive Affect	State	Yes	−.72	−.43	−.25	−.44	−.18
Joiner & Lonigan, 2000	33	2	Child/Ad	Clinical	Positive Affect	Trait	Yes	−.67	−.14	.29		
Joiner, 1994	96	1	Adult	Other	Positive Affect	State	No	−.53	−.24	−.00	−.28	−.05

	Sample Characteristics							Effect Sizes				
Study	***N***	**Time lag in months**	**Age**	**Sample Type**	**Positive Emotionality Construct**	**Trait or State**	**Inter-assessment change**	**Cross-sectional[a]**	**PE ➔ D[a]**	**PE ➔ D, controlled[b]**	**D ➔PE[a]**	**D ➔ PE, controlled[b]**
Joiner, 1995	97	1	Adult	Other	Positive Affect	State	No	−.31	−.18	−.04	−.16	−.03
Joiner, 1997	172	1	Adult	Other	Positive Affect	State	No	−.52	−.52	−.18	−.36	−.04
Jorm et al., 2000	433	43	Adult	Other	Extraversion	Trait	No	−.12	−.08	−.02		
Jylhä et al., 2012	142	6	Adult	Clinical	Extraversion	Trait	Yes	−.18	−.20	−.16	−.08	.05
Karademas & Tsaousis, 2014	115	3	Adult	Physical health	Extraversion	Trait	No	−.51	−.36	−.08		
Keyes et al., 2010	1,725	120	Adult	Other	Positive Affect	State	No	−.27	−.19	−.11	−.09	−.02
Klimstra et al., 2010	1,060	12	Child/Ad	Other	Extraversion	Trait	No	−.30	−.22	−.09	−.15	.02
Langer et al., 2014	93	6	Adult	Other	Positive Affect	State	No	−.11	−.12	−.08	−.09	−.02
Langer et al., 2014	336	6	Adult	Physical health	Positive Affect	State	No	−.04	−.07	−.05	−.12	−.10
Lengua, 2003	83	12	Child/Ad	Other	Positive Affect	Trait	No	−.07	−.01	.02	−.08	−.05
Loh et al., 2014	107	3	Adult	Other	Positive Affect	Trait	No	−.56	−.47	−.07	−.51	−.17
Lonigan et al., 2003	270	7	Child/Ad	Other	Positive Affect	Trait	No	−.38	−.35	−.10	−.32	−.09
Marques et al., 2011	382	6	Adult	Other	Positive Affect	State	Yes	−.35	−.22	−.06		
Mascaro & Rosen, 2005	191	2	Adult	Other	Extraversion	Trait	No	−.42	−.25	.04	−.29	.04
McIntosh et al., 2013	759	38	Adult	Other	Extraversion	Trait	No	−.42	−.36	−.09		
Meeks et al., 2012	441	6	Adult	Other	Positive Affect	State	No	−.43	−.34	−.10	−.30	−.11
Mezulis et al., 2011	423	24	Child/Ad	Other	Extraversion	Trait	No	−.07	.04	.07		
Naragon-Gainey et al., 2013[c]	810	6	Adult	Clinical	Positive Affect	Trait	Yes	−.49	−.40	−.13	−.44	−.13
O'Neill et al., 2004	101	2	Adult	Other	Positive Affect	State	No	−.29	−.13	.01		
Parrish et al., 2011	93	2	Adult	Other	Positive Affect	Trait	No	.03	−.01	−.03	.00	−.02
Poon & Knight, 2013	337	36	Adult	Other	Positive Affect	State	No	−.29	−.17	−.03	−.27	−.14

	Sample Characteristics							Effect Sizes				
Study	***N***	**Time lag in months**	**Age**	**Sample Type**	**Positive Emotionality Construct**	**Trait or State**	**Inter-assessment change**	**Cross-sectional[a]**	**PE ➔ D[a]**	**PE ➔ D, controlled[b]**	**D ➔PE[a]**	**D ➔ PE, controlled[b]**
Poulin et al., 2009	532	5	Adult	Other	Positive Affect	State	No	−.47	−.35	−.06	−.38	−.09
Prenoveau, 2009	189	12	Child/Ad	Other	Extraversion	Trait	No	−.26	−.14	−.01	−.13	.06
Reddy, 2010	99	12	Adult	Other	Extraversion	Trait	Yes	−.31	−.22	−.10		
Robinson-Whelen et al., 2001	143	48	Adult	Other	Positive Affect	State	Yes	−.38	−.49	−.26	−.51	−.33
Robison et al., 2009[c]	61	6	Adult	Clinical	Extraversion	Trait	Yes	−.41	−.12	.07		
Smith et al., 2012	269	18	Adult	Other	Positive Affect	State	No	−.68	−.48	−.07	−.50	−.03
Spinhoven et al., 2011	1,322	12	Adult	Physical health	Extraversion	Trait	Yes	−.36	−.33	−.12		
Takahashi et al., 2012	109	1	Adult	Other	BAS	Trait	No	−.34	−.27	−.02	−.33	−.06
Vanhalst et al., 2012	290	12	Child/Ad	Other	Extraversion	Trait	No	−.12	−.09	−.02		
Verstraeten et al., 2009	249	12	Child/Ad	Other	Positive Affect	Trait	No	−.36	−.32	−.16		
Vliegen et al., 2013	41	42	Adult	Clinical	Positive Affect	Trait	Yes	−.53	−.38	−.21	−.38	−.07
Voelz et al., 2001	63	2	Adult	Other	Positive Affect	State	No	−.40	−.18	−.03	−.14	.15
Weinstein et al., 2007	466	6	Child/Ad	Other	Positive Affect	State	No	−.29	−.27	−.06	−.30	−.10
Weiss et al., 2009	659	12	Adult	Other	Extraversion	Trait	Yes	−.26	−.26	−.11		
Wetter & Hankin, 2009	345	5	Child/Ad	Other	Missing	Trait	No	−.47	−.35	−.04		
Yang et al., 2008	2,773	12	Child/Ad	Other	Extraversion	Trait	No	−.23	−.16	−.02		
Zdanowicz et al., 2012[d]	20	6		Other	Extraversion	Trait	No	.34	.07	−.12		

Note. Child/Ad = child/adolescent; PE = Positive Emotionality; D = depression; *N* = number of participants; Time lag in months = time between the first and second assessment; Age = age of sample; Sample type = the type of sample (Clinical = sample recruited from inpatient or outpatient mental health clinics; Physical health = sample recruited from inpatient or outpatient physical health clinics); Positive Emotionality Construct = type of Positive Emotionality construct assessed; Trait or State = whether Positive Emotionality was assessed using a trait or a state measure; Inter-assessment change = whether the sample likely experienced change between assessments.

[a]Correlation coefficient.

[b]Standardized regression coefficient.

[c] Study included a non-primary BAS measure that was utilized for the "Positive Emotionality construct" moderation analysis.

[d] Zdanowicz et al., 2012 included both adolescents and adults.

Table 2

Longitudinal Studies of the Relation Between Positive Emotionality and Anxiety

	Sample Characteristics							Effect Sizes				
Study	***N***	**Time lag in months**	**Age**	**Sample Type**	**Positive Emotionality Construct**	**Trait or State**	**Inter-assessment change**	**Cross-sectional[a]**	**PE → A[a]**	**PE → A, controlled[b]**	**A → PE[a]**	**A → PE, controlled[b]**
Avey et al., 2011	128	4	Adult	Other	Positive Affect	State	No	−.14	−.09	−.02	−.03	.04
Bardi & Ryff, 2007	280	8	Adult	Other	Extraversion	Trait	Yes	−.09	−.09	−.05		
Benyamini & Roziner, 2008	525	60	Adult	Other	Positive Affect	Trait	No	−.34	−.26	−.09	−.23	−.06
de Beurs et al., 2005	1,694	36	Adult	Other	Positive Affect	State	No	−.45	−.31	−.08	−.32	−.14
De Bolle et al., 2011	150	12	Child/Ad	Clinical	Positive Affect	Trait	Yes	−.34	−.18	.04	−.20	−.06
Dill, 2005	1,146	5	Child/Ad	Other	Extraversion	State	No	−.08	−.14	−.10	−.12	−.07
Festen et al., 2013	78	3	Child/Ad	Clinical	Extraversion	Trait	No	−.28	−.29	−.08		
Fox, 2010	131	12	Child/Ad	Other	Positive Affect	Trait	No	−.25	−.06	.02		
García-Peña et al., 2013	2,352	12	Adult	Other	Positive Affect	State	No	−.34	−.29	−.10	−.30	−.23
Gershuny & Sher, 1998	466	36	Adult	Other	Extraversion	Trait	No	−.04	−.03	−.01		
Gudiño et al., 2012	159	6	Child/Ad	Other	BAS	Trait	No	.21	.10	−.01		
Hou et al., 2010	215	3	Adult	Physical health	Positive Affect	State	Yes	−.54	−.29	−.10	−.33	−.09
Joiner & Lonigan, 2000	33	2	Child/Ad	Clinical	Positive Affect	Trait	Yes	−.39	.03	.21		
Joiner, 1994	96	1	Adult	Other	Positive Affect	State	No	−.14	−.13	−.06	−.14	−.08
Joiner, 1995	97	1	Adult	Other	Positive Affect	State	No	.07	−.04	−.07	−.04	−.07
Jorm et al., 2000	433	43	Adult	Other	Extraversion	Trait	No	−.00	.03	.04		
Jylhä et al., 2012	142	6	Adult	Clinical	Extraversion	Trait	Yes	−.14	−.11	−.04	−.05	.05
Karademas & Tsaousis, 2014	115	3	Adult	Physical health	Extraversion	Trait	No	−.46	−.23	.03		
Lonigan, et al., 2003	270	7	Child/Ad	Other	Positive Affect	Trait	No	−.30	−.22	−.01	−.32	−.14
McIntosh et al., 2013	759	38	Adult	Other	Extraversion	Trait	No	−.22	−.20	−.05		

	Sample Characteristics							Effect Sizes				
Study	***N***	**Time lag in months**	**Age**	**Sample Type**	**Positive Emotionality Construct**	**Trait or State**	**Inter-assessment change**	**Cross-sectional[a]**	**PE ➔ A[a]**	**PE ➔ A, controlled[b]**	**A ➔PE[a]**	**A ➔ PE, controlled[b]**
Poulin et al., 2009	532	5	Adult	Other	Positive Affect	State	No	−.33	−.23	−.03	−.25	−.04
Salsman et al., 2009	55	3	Adult	Physical health	Positive Affect	State	No	−.77	−.74	−.49	−.62	−.12
Spinhoven et al., 2011	1,322	12	Adult	Physical health	Extraversion	Trait	Yes	−.14	−.20	−.12		
Van Dijk et al., 2013	212	6	Adult	Other	Positive Affect	State	Yes	−.32	−.21	−.02	−.19	−.09
Vliegen et al., 2013	41	42	Adult	Clinical	Positive Affect	Trait	Yes	−.53	−.45	−.13	−.56	−.32
Voelz et al., 2001	63	2	Adult	Other	Positive Affect	State	No	−.08	−.23	−.19	−.01	.04

Note. Child/Ad = child/adolescent; PE = Positive Emotionality; A = anxiety; *N* = number of participants; Time lag in months = time between the first and second assessment; Age = age of sample; Sample type = the type of sample (Clinical = sample recruited from inpatient or outpatient mental health clinics; Physical health = sample recruited from inpatient or outpatient physical health clinics); Positive Emotionality Construct = type of Positive Emotionality construct assessed; Trait or State = whether Positive Emotionality was assessed using a trait or a state measure; Inter-assessment change = whether the sample likely experienced change between assessments. See Supplementary Table 2 for a machine-readable version of Table 2.

[a] Correlation coefficient.

[b] Standardized regression coefficient.

Table 3

Effect Sizes for the Relation Between Positive Emotionality (PE) and Depression (D)

Variable	*k*	*N*	Effect size	95% CI	τ^2	I^2
Cross-sectional[a]	59	24,503	−.34	[−.38, −.30]	0.03	91.03
Prospective, PE → D[a]	57[c]	23,806	−.26	[−.29, −.22]	0.01	82.45
Prospective, controlled PE → D[b]	58	24,229	−.08	[−.09, −.06]	0.00	28.01
Prospective, D → PE[a]	32[d]	9,185	−.28	[−.33, −.24]	0.01	79.69
Prospective, controlled D → PE[b]	33[e]	10,910	−.07	[−.09, −.04]	0.00	45.06

Note. Computations utilized a random effects model. *k* = number of studies; *N* = total number of participants in the *k* samples; CI = confidence interval; τ^2 = tau-squared estimate of between-study variance; I^2 = degree of heterogeneity. All effect sizes are significant at $p < .05$.

[a] Correlation coefficient.

[b] Standardized regression coefficient.

[c] 1 outlier (Mezulis et al., 2011).

[d] 2 outliers (García-Peña et al., 2013; Keyes et al., 2010).

[e] 1 outlier (García-Peña et al., 2013).

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Table 4

Effect Sizes for the Relation Between Positive Emotionality (PE) and Anxiety (A)

Variable	*k*	*N*	Effect size	95% CI	τ^2	I^2
Cross-sectional[a]	25[c]	9,800	−.24	[−.31, −.17]	0.03	90.77
Prospective, PE → A[a]	26	11,494	−.19	[−.23, −.14]	0.01	83.01
Prospective, controlled PE → A[b]	26	11,494	−.06	[−.09, −.04]	0.00	30.18
Prospective, A → PE[a]	16	7,718	−.23	[−.29, −.17]	0.01	81.22
Prospective, controlled A → PE[b]	15[d]	5,366	−.09	[−.11, −.06]	0.00	5.97

Note. Computations utilized a random effects model. *k* = number of studies; *N* = total number of participants in the *k* samples; CI = confidence interval; τ^2 = tau-squared estimate of between-study variance; I^2 = degree of heterogeneity. All effect sizes are significant at $p < .05$.

[a] Correlation coefficient.

[b] Standardized regression coefficient.

[c] 1 outlier (de Beurs et al., 2005).

[d] 1 outlier (García-Peña et al., 2013).

Table 5

Moderation Analyses for the Cross-lagged Regression Between PE at Time 1 and Depression at Time 2

Categorical moderators	**Category**	**β**	**Category**	**β**	**Category**	**β**	***Q***
Operationalization of PE							
Type of PE measure[b]	Extraversion (*k* = 21)	−.07*	Positive affect (*k* = 30)	−.08*	Behavioral activation (*k* = 6)	−.05	1.75
Time span of PE measure	State (*k* = 22)	−.09*	Trait (*k* = 36)	−.07*			1.43
Demographics and other sample characteristics							
Sample age[b]	Adults (*k* = 42)	−.09*	Children/Adolescents (*k* = 15)	−.04*			8.45*
Sample sex[b]	More than 70% female (*k* = 15)	−.09*	Less than 70% female (*k* = 42)	−.07*			0.33
Type of sample I	Clinical (*k* = 7)	−.09*	Nonclinical (*k* = 51)	−.07*			0.20
Type of sample II	Clinical and physical health (*k* = 14)	−.10*	Other samples (*k* = 44)	−.07*			1.45
Restricted range I	Depressed/anxious-only samples (*k* = 6)	−.12*	Other samples (*k* = 52)	−.07*			3.64
Restricted range II	Samples excluding baseline depression (*k* = 6)	−.13*	Other samples (*k* = 52)	−.07*			2.28
Likely inter-assessment change	Yes (*k* = 17)	−.11*	No (*k* = 41)	−.06*			8.46*
Special population	Yes (*k* = 11)	−.11*	No (*k* = 47)	−.07*			3.20
Study measures							
Depression measure report	Self-report (*k* = 52)	−.07*	Other-report (*k* = 6)	−.10*			0.55
Depression measure distribution	Continuous (*k* = 56)	−.07*	Originally dichotomous (*k* = 2)	−.12*			1.86
Methodological considerations							
Study location	U.S. and western Europe (*k* = 51)	−.08*	Other locations (*k* = 7)	−.05*			1.92
Data publication status	Data published (*k* = 21)	−.08*	Data requested (*k* = 37)	−.07*			0.02

Continuous moderators	β	*Q*
Sample size	<.001	0.27
Publication year	−.002	0.58
Total number of study waves	−.001	0.08
Percent attrition	.054	1.00

Note. Computations utilized a random effects model. β = standardized regression coefficient; *Q* = test of moderation.

[b] Analysis excluded one study that did not include enough information for coding moderators or did not fall into one of the moderator categories. Additional information can be found in Supplementary Table 1.

* $p < .01$. No p values were $< .05$.