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Optimism Versus Pessimism as Predictors of Physical Health:

A Comprehensive Reanalysis of Dispositional Optimism Research

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(Abstract)

Prior research has related dispositional optimism to physical health. Traditionally, dispositional optimism is treated as a bipolar construct, anchored at one end by optimism and the other by pessimism. Optimism and pessimism, however, may not be diametrically opposed, but rather may reflect two independent, but related dimensions. This paper reports a reanalysis of data from previously published studies on dispositional optimism. The reanalysis was designed to evaluate whether the presence of optimism or the absence of pessimism predicted positive physical health more strongly. Relevant literatures were screened for studies relating dispositional optimism to physical health. Authors of relevant studies were asked to join a consortium, the purpose of which was to re-analyze previously published data sets separating optimism and pessimism into distinguishable components. Ultimately, data were received from 61 separate samples (N = 221,133). Meta-analytic analysis of data in which optimism and pessimism were combined into an overall index (the typical procedure) revealed a significant positive association with an aggregated measure of physical health outcomes (r = .026, p < .001), as did meta-analytic analyses with the absence of pessimism (r = .029, p < .001) and the presence of optimism (r = .011, p < .034) separately. The effect size for pessimism was significantly larger than the effect size for optimism (Z = -2.403, p < .02). Thus, the absence of pessimism was more strongly related to positive health outcomes than was the presence of optimism. Implications of the findings for future research and clinical interventions are discussed.

Keywords: Optimism, pessimism, physical health, meta-analytic methodology

Public Significance Statement: Prior research on dispositional optimism typically combines the presence of optimism and the absence of pessimism into an overall index. Prior research using this combined index suggests that dispositional optimism is associated with better physical health. The present reanalysis of existing data breaks apart the two components of dispositional optimism and suggests that the absence of pessimism is more strongly related to good physical health than is the presence of optimism.

Folk wisdom has long held that differences among people in optimism and pessimism are important to many aspects of daily living. In this case, folk wisdom seems to be right. Optimists have been documented to differ from pessimists in many important ways. They differ in how they approach and cope with the problems they confront (Nes & Segerstrom, 2006), the number and quality of relationships they form (Assad et al., 2007; Brissette, Scheier, & Carver, 2002), and the quality of life they experience (Duffy et al., 2013; Segerstrom, Carver, & Scheier, 2017).

Optimists and pessimists also differ in their physical health. For the past 3 decades, research on dispositional optimism and physical health has flourished. A *Google Scholar* search for *"dispositional optimism" and "physical health"* yields over 5,000 hits. Dispositional optimism predicts a number of short-term and long-term health outcomes, including rehospitalization after surgery (Scheier, Matthews, Owens, et al., 1999; Tindle, Belnap, Hum, et al., 2012), incident cardiovascular disease (Tindle, Chang, Kuller, et al., 2009), incident stroke (Kim, Park, & Peterson, 2011), and mortality (Kim, Hagan, Grodstein, et al., 2016; Tindle et al., 2009). It is also related to a number of biological markers tied to disease endpoints, including ambulatory blood pressure (Räikkönen, Matthews, Flory, et al., 1999), cortisol secretion (Jobin, Wrosch, & Scheier, 2013), as well as levels of lipids (Boehm, Williams, Rimm, et al., 2013a) and anti-oxidants (Boehm, Williams, Rimm, et al., 2013b).

Although links between dispositional optimism and physical health now seem well-established (for a general quantitative review see, Rasmussen, Scheier, & Greenhouse, 2009; for a general qualitative review see, Boehm & Kubzanksy, 2012), how best to construe the construct of optimism has proven more controversial. Most of the research that has been conducted on dispositional optimism treats the variable as bipolar in nature, anchored at one end by optimism (the generalized expectancy that favorable outcomes will occur in the future) and at the other end by pessimism (the generalized expectancy that unfavorable outcomes will occur in the future). According to this view, as someone moves away from optimism that person necessarily moves more toward pessimism.

This prevailing view has emerged in part because of the way in which the scales used to

measure dispositional optimism are scored. The two most widely used scales to measure dispositional optimism are the Life Orientation Test (LOT, Scheier & Carver, 1985) and the Life Orientation Test—Revised (LOT-R, Scheier, Carver, & Bridges, 1994). Each of these scales contains two sets of items. Items from one set are framed in a positive way (assessing the affirmation of optimism or not), and items from the second set are framed in a negative way (assessing the affirmation of pessimism or not). Typically, the negatively framed items are reverse coded and then added to the positively framed items to produce on overall scale score.

Some researchers have questioned the validity of this "bipolar" point of view. Interest in the question arose after several factor analyses suggested that a 2-factor model of the items on the LOT and LOT-R fit the data better than did a model with a 1-factor solution (e.g., Chang & McBride-Chang, 1996; Hjelle, Belongia, & Nesser, 1996). In these analyses, items assessing expectations for positive outcomes loaded on one factor (an "optimism" factor reflecting the affirmation of optimism or not), whereas items assessing expectations for negative outcomes loaded on a second factor (a "pessimism" factor, reflecting the affirmation of pessimism or not). Consistent with the factor analytic results, correlations between the optimism and pessimism subscales are modest (Mens, Scheier, & Carver, 2016).

Conceptually, it makes sense that optimism and pessimism are somewhat distinct. Clearly, someone who is not pessimistic is not necessarily optimistic. It only means that there is an absence of pessimism. Similarly, someone who is not optimistic is not necessarily pessimistic. It just means that there is an absence of optimism. People can be neither optimistic nor pessimistic. This is one reason why the two factors are thought to reflect the presence or absence of the characteristic in question. Consistent with this construal, the same terminology is used throughout this paper to refer to the two ends of the optimism and pessimism dimensions.

There are differences in opinion about what to make of the factor-analytic studies. Monzani, Steca, and Greco (2014) believe that the two factors are due to response style and that optimism should still be conceptualized as a single dimension. Others have argued that optimism and pessimism are distinct properties that may have differential effects on various aspects of

physical health (e.g., Kubzansky et al., 2004). Several attempts have used item response theory to resolve the issue. This research suggests that a single dimension may fit the LOT-R better (e.g. Steca, Monzani, Greco, Chiesi, & Primi, 2014). However, the issue is far from resolved psychometrically.

Research from the field of behavior genetics offers further support for the idea that optimism and pessimism are distinguishable. A variety of studies now support the idea that there is a genetic basis for differences in dispositional optimism (e.g., Caprara et al., 2009; Plomin et al., 1992). More importantly, there is also some evidence that the genetic origins of optimism and pessimism might be slightly different. For example, Plomin et al., (1992) have shown that shared environment is more important for optimism than pessimism. Using more complex modeling techniques, Bates (2015) has shown that optimism and pessimism contain genetic variation that separates them from both the Big 5 personality factors and from each other.

Recent research in health psychology also contributes to the discussion of dimensionality by documenting that optimism and pessimism can be related to physical health differentially. For example, research suggests that it is pessimism that produces associations with inflammation, not optimism (Roy et al., 2010, Ikeda et al., 2011; O'Donovan et al., 2009). Pessimism was also found to be a stronger predictor than optimism of in vitro fertilization success (Bleil et al., 2012). In contrast, Kim et al. (2011) showed that optimism, but not pessimism, predicted incidence of stroke. Although only a handful of studies speak directly to this issue, the available evidence tends to suggest that the absence of pessimism might be a more important contributor to associations with physical health than the presence of optimism. Clearly, however, more research is needed on this issue.

In this regard, an organization called the Optimism/Pessimism Meta-Analytic Consortium (OPMAC) was formed to pool data from across studies to examine more systematically the effects of optimism and pessimism on physical health. Each member of the consortium has reanalyzed data from a previously published study in such a way that the effects of optimism and pessimism can be separated and compared. The purpose of this paper is to present the results of

the reanalyses that were conducted on the novel data that OPMAC members provided. Given the trend of the few available studies published prior to the present reanalyses, the absence of pessimism was expected to be a stronger predictor of positive physical health than was the presence of optimism.

Method

Literature Search Strategy and Inclusion/Exclusion Criteria

To identify relevant researchers to contact, literature searches were performed on the MedLINE and PsycINFO databases for relevant studies published in English-language peer-reviewed journals up until December 31st 2016 using combinations of the following keywords: *optimism, pessimism, Life Orientation Test , LOT, Life Orientation Test—Revised, LOT-R, immune, inflammation, HIV or AIDS, arthritis, osteoarthritis, lupus, autoimmune, multiple sclerosis, pregnancy, infertility, cancer or neoplasms, cortisol, blood pressure, atherosclerosis, cardiovascular, coronary, heart, infarction, stroke, diabetes, glycemic, anemia, respiratory, tuberculosis, dementia, asthma, Huntington's, renal, kidney, influenza, pneumonia, cold, ulcer, sleep, survival, death, mortality, body mass index, wound, surgery,* and *metabolic*. The search terms used to identify studies were largely derived from a meta-analysis of the same area conducted by Rasmussen et al. (2009), with extra terms added to capture biomarkers more fully. Review papers and references from relevant articles were used to identify additional studies of interest. After an initial prescreening for potentially relevant articles, based on the study title and abstract, a total of 549 full-length manuscripts were downloaded for further evaluation based on our inclusion and exclusion criteria.

Manuscripts were included for consideration if they met two inclusion criteria. First, the study utilized the LOT (Scheier & Carver, 1985), the LOT-R (Scheier et al., 1994), or a validated translation or adaptation of either the LOT or LOT-R (e.g. the Parent-rated Life Orientation Test of children, Lemola et al., 2010). This criterion was enacted because the objective of the present set of reanalyses was to evaluate the differential effects of optimism and pessimism on physical health. The LOT and LOT-R are the only scales available that allow for overall/combined

optimism to be deconstructed into its underlying components. The LOT-R was created in order to remove two coping items that had been included in the original LOT. The LOT and the LOT-R correlate in the low .90's (Scheier et al., 1994). The psychometric properties of the LOT and LOT-R are well established (for a review, see Carver & Scheier, 2019), and they are used widely in the literature in health psychology. An example of a positively worded item is, "In uncertain times, I usually expect the best." An example of a negatively worded item is, "I hardly ever expect things to go my way." All items are answered along a 5-point Likert scale, ranging from "strongly disagree" at one end to "strongly agree" at the other.

Second, the study included an objective measure of physical health. Objective measures of physical health included biomarkers of various types (e.g., systolic and diastolic blood pressure, cortisol reactivity), disease incidence (e.g., stroke, acute myocardial infarction, diabetes, cancer), hospital stay or rehospitalization, and survival or mortality. Review papers, unpublished data, dissertations, and conference abstracts were not included.

Two additional exclusion criteria were also used. First, studies were excluded if neither optimism/pessimism nor physical health were the primary focus of the study (the vast majority of these studies had simply included optimism or physical health as part of a wider set of covariates). The decision to exclude these studies was made largely on the basis of expected utility. That is, to provide useful data for the present reanalyses authors were required to reanalyze the data from their studies, breaking optimism and pessimism down into separate factors (the norm for published studies is to combine these components into an overall score). If the primary theoretical frameworks of authors were related to neither optimism/pessimism nor physical health, it seemed unlikely that they would put the needed effort into providing data for the reanalyses. For this reason, they were not pursued further.

Studies of primary interest in this analysis were those conducted in the field, often over prolonged periods of time. Consequently, studies were also excluded if they represented experimental laboratory studies that consisted of a single session, in which participants were randomly assigned to conditions. These studies were excluded because they were thought to be

too dissimilar to the larger set of field studies of primary interest. Including them would have made interpretation of results difficult.

Upon evaluating the 549 downloaded manuscripts, 189 relevant studies were identified that met our inclusion and exclusion criteria. Of these 189 studies, 16 were removed because they provided duplicate data across time. These papers tended to provide interim reports of ongoing longitudinal studies. The rule for serial publication of results was to take the longest follow-up time available. An additional 10 studies were removed because no email was provided for the original authors. Four studies were removed because the measure of optimism was assessed after the measure of physical health. The corresponding authors of the remaining 159 manuscripts were contacted and asked if they had an interest in joining the consortium.

Of the authors contacted, 44 did not respond to our request, and 50 reported that the date were no longer available. In addition, there were 2 cases (Ai, Seymore, & Tice, 2009; Lai, Evans, & Ng, 2005) for which incorrect analyses had been requested. Because the error was discovered late in the process of data analysis, these authors were not asked to provide corrected data. Finally, one study (Bennett et a., 2015) was excluded because the researchers only collected data on the optimism subscale, and data from both subscales were needed to conduct analyses.

Ultimately, the data from 62 papers were available for inclusion in the present reanalyses. Two pairs of these studies (Pänkäläinen et al., 2015 and Pänkäläinen et al., 2016, and Ruiz et al., 2003 and Scheier et al., 1999) reported on the same sample, but included different outcomes from one paper to the next. These two pairs of studies were included in the analyses, but the data from the pairs of studies were considered to be dependent for purpose of analysis. That is, they were treated as providing multiple outcomes from the same sample. Another study (Konkoly-Thege et al., 2015) provided separate, independent samples in the same paper (one comprised of healthy controls and one comprised of patients). These samples were treated as independent in the analyses. Thus, a total of 61 independent samples was ultimately available for inclusion in the present reanalyses (see Figure 1 for a graphic display of the study selection process).

Data Collection

Initial contact. Potential consortium members were contacted by email, informed of the purpose of the present project, told what additional analyses needed to be performed, and invited to join the effort. If no response was received, a second email was sent with the same information 2 to 4 weeks later. If no response was received to the second email, a third email was sent 2 to 4 weeks later. If no response was received to the third email, recipients were identified as non-responders. Recipients were also identified as non-responders if, after a corresponding author expressed interest in contributing their data, at least two months had passed without receiving the requested data and no response was given to a follow-up email regarding the status of their analyses.

The data collection process began on August 11, 2016. All data were received by May 31, 2017. Recipients who participated were given $200 as a token of appreciation for their effort and were entered into the Optimism/Pessimism Meta-Analytic Consortium (OPMAC). Consortium members are listed in Supplemental Online Table 1.

Requested analyses. Each consortium member was asked to conduct three separate analyses, one using the overall/combined optimism score as the predictor variable, one using the pessimism subscale as the predictor variable, and one using the optimism subscale as the predictor variable. All analyses treated optimism and pessimism as continuous variables. Items were recoded so that a high score indicated high optimism (for the overall/combined scale and the optimism subscale) or low pessimism (for the pessimism subscale). Effect sizes were coded such that a positive effect size indicated better health. Thus, the overall/combined scale, the optimism subscale, and the pessimism subscale should all be related in a positive manner with the health outcomes assessed.

If a published study contained physical health outcomes in the primary outcomes reported, those same physical health measures were requested as outcomes in the re-analysis. If a published study contained physical health outcomes, but did not report them as primary outcomes, all relevant physical health measures included in the study were requested as outcomes in the re-analysis. Relevant physical health measures were defined as those which had

been used as a primary outcome in at least one other study in the pool of studies in the analysis. This strategy was employed in order to avoid including an abundance of studies with idiosyncratic outcomes (i.e., outcomes that were not of established interest to the research literature on optimism and health more widely). Supplemental Online Table 2 lists the outcome measures obtained for each of the studies in the analyses.

When requesting covariates for the re-analyses, consortium participants were asked to use the same set of covariates that was used in the published paper. Some of the studies had an extraordinarily large number of covariates. Consequently, the number of covariates requested for inclusion in the re-analyses was capped at 20. Major classes of covariates included demographic variables (e.g., gender, education level), psychosocial variables (e.g., depressive symptoms, negative affectivity), or factors related to the study design (e.g., length of follow up from baseline to final assessment).

Several categories of covariates were explicitly excluded from the re-analyses. These included measures of coping styles and strategies, social support, situational expectations for the health context studied, biomarkers and preclinical indicators of disease (e.g., C-reactive protein and body mass index, respectively), and health behaviors (e.g., smoking, physical activity). These classes of covariates were excluded because existing data has shown that these variables are predicted by optimism (for a review, see Scheier & Carver, 2018). Because of this covariation, these variables could reflect underlying mechanisms whereby the impact of optimism on downstream health outcomes is mediated. Correcting for potential mediators could artificially reduce the effect size estimating the association between optimism and health by eliminating the contribution of indirect pathways (Gallo & Matthews, 2003). For this reason, potential mediators were excluded as covariates when re-analyses were conducted. The covariates included for each of the studies in the analyses can also be found in Supplemental Online Table 2.

When possible, consortium members were asked to re-analyze their data in the same way they analyzed their data in the original study. If the original study did not conduct an analysis

using optimism as a predictor and physical health as an outcome, consortium members were requested to conduct either a linear or logistic regression, depending on whether the physical health outcome was continuous or dichotomous. As previously noted, all of the predictors (the combined overall scale, the pessimism subscale, and the optimism subscale) were treated as continuous variables. In addition to the requested effect sizes, researchers were also asked to provide the internal consistency reliability for the overall/combined optimism scale, the pessimism subscale, and the optimism subscale, as well as the correlation between the optimism and pessimism subscales.

Abstracted data. In addition to effect size data, pertinent data from the original manuscript and from the requested re-analysis were abstracted. Abstracted data included year study was published, scale used to assess optimism, information about the number of participants in the study, the mean age of participants, the percent of the sample that was female, the percent of the sample that was white, the type of sample studied (i.e., clinical or nonclinical), the country from which the sample was drawn, the optimism measure used, the number and type of covariates included (e.g., demographic, psychosocial), and the study design (e.g., prospective or cross-sectional). Finally, the aim of the original study was also coded to distinguish between original studies that were explicitly focused on both optimism and physical health (and the relationship between the two of them), and studies that were primarily interested either in optimism or in physical health (but not explicitly with the association between the two).

For the purpose of this analysis, studies were coded as being prospective in design if they met one of the following two conditions: (1) the requested re-analyses controlled for the physical health outcome at baseline; (2) the study examined either mortality or disease incidence and screened out all participants with prior or current illness such that the sample was assumed to be physically healthy at baseline. Longitudinal studies were those that assessed optimism/pessimism measures at baseline and then documented health outcomes at a later point in time. Unlike prospective studies, however, health outcomes were not controlled for in some fashion at baseline. Cross-sectional studies were those that assessed predictor and outcome at the same

point in time. These distinctions are consistent with the description of study design differences presented by Cohen et al. (1986).

Abstracted data were double-entered. Discrepancies in coding were resolved through discussion of the coders. Coder reliability across entries averaged 84%, ranging from 52% to 99%, with the most disagreement occurring for the coding of study design.

Statistical Analyses

General considerations. Before the questions of primary interest could be answered, the data from the different samples needed to be harmonized, aggregated, and summarized. The analytic approach is based on methods used in meta-analysis for combining information from similar studies. Random effects models (which assume that samples are drawn from different populations and allow for both random variance and variance due to true population differences) were used for all analyses conducted. Given the different contexts represented across studies, random effects models were assumed to provide a more accurate estimate of confidence intervals than fixed effects models (see e.g., Schmidt, Oh, & Hayes, 2009).

Calculation of effect sizes. For outcomes that were treated as continuous variables, consortium members provided standardized beta coefficients from their analyses. For dichotomized outcomes, consortium members provided odds ratios or hazard ratios, depending upon the specific analysis conducted. The data received were than transformed into Fisher Z (Z') scores, following the guidelines offered by Borenstein, Hedges, Higgins, and Rothstein (2009). These converted Z' scores were then used in the meta-analytic analyses that were conducted, as well as in tables and figures that are presented. Z' estimates were transformed into *r* estimates for purposes of data presentation in text.

Heterogeneity. Heterogeneity of the variances in the effect sizes from the primary analyses were evaluated using the I^2 index, which is an indicator of the proportion of variance explained by heterogeneity. An I^2 index above 50% suggest a heterogeneous effect size distribution, which warrants additional moderator analyses (Higgins & Thompson, 2002).

Analytic strategy. Multiple outcomes within a study were treated as dependent, as the

outcomes assessed were likely to be correlated to a greater or lesser extent. Therefore, the robust variance estimation (RVE) method was used to account for dependency among samples with multiple effect sizes because it allows one to specify the within-study correlation among effects. We utilized the default within-study correlation value of .80 in our analyses. Further, the small sample adjustment was applied to correct for bias in p-values (Tanner-Smith, Tipton & Polanin, 2016).

Importantly, more traditional meta-analytic techniques were used to compute estimates for which the empirically calculated degrees of freedom fell below four. For these estimates, an average effect size for that study was computed and used in the relevant analysis. This strategy was selected given that the estimated p-values can be inaccurate when the empirically calculated degrees of freedom fall below four (Tanner-Smith, Tipton & Polanin, 2016).

For the RVE analyses, we tested the basic meta-regression model to estimate the mean effect size (i.e., intercept only, no predictors). Subsequently, moderator analyses were conducted by adding the respective moderator variable as a predictor to the meta-regression model. For continuous moderators, the coefficients can be interpreted as the estimated amount of change in strength of the association (i.e., mean effect size) given a one unit increase in the moderator. For categorical moderators, dummy codes were used and can be interpreted as the mean effect size difference between the relevant groups.

Prior to analyses, the following two sets of variables were identified as potential moderators, depending upon whether the variable was categorical or continuous in nature. Continuous moderators included year the study was published, average age of sample, percent of sample that was female, percent of sample that was white, number of psychosocial covariates used in the analyses, and total number of covariates included in the analyses. Categorical moderators included study objective (whether the focus of the study was on optimism, physical health, or both), study design (whether the study was cross-sectional, longitudinal, or prospective), participant status (healthy versus patient), scale used to assess optimism (LOT versus LOT-R), and the country of origin for the study (United States versus elsewhere).

Finally, analyses were conducted to test for differences between optimism and pessimism. Preliminary analyses of the effect sizes for the pessimism and optimism subscales showed both distributions to be non-normal. As a result, a Wilcoxon signed rank test for paired samples was used to test the significance of the difference between the two subscales. For these comparisons, an average effect size for each study within each subscale was computed and used in the relevant analysis. Average ES's were used inasmuch as Robumeta does not provide ES estimates for individual studies.

Software. The RVE analyses were conducted in R (version 3.5.1) using Robumeta package (Fisher & Tipton, 2014) to estimate mean effect sizes and meta-regression models and clubSandwich package (Pustejovsky, 2015) to estimate the multiparameter F-tests. The standard meta-analysis estimates were obtained using Comprehensive Meta-Analysis Software Version 3 (BiostatTM, USA). Finally, all non-meta-analytic analyses were conducted using IBM SPSS Statistics Version 25.

Results

Study Characteristics

The number of participants in the studies reported here totaled 221,133. The participants averaged 63.71 years of age, were 91.44 percent female, and were 92.07 percent white (based on the 35 studies that reported the race of the participants). The majority of the studies were conducted in the United States, 90.12 percent. The high percentage of white women can be attributed largely to two studies, Kim et al. (2016) and Tindle et al. (2009), which were all women and largely white and contributed 167,274 to the participant count.

Cronbach's alpha for the overall/combined scale, the optimism subscale, and the pessimism subscale were 0.75, 0.72, and 0.75, respectively. A one-way repeated measures ANOVA was conducted to evaluate the significance of the differences between the alphas. This overall analysis was not significant, Wilk's Lambda = .904, F (2, 54) = 2.88, $p > .06$ (not all of the researchers provided alphas, which accounts for the fewer than 59 degrees of freedom). Because the significance level from this overall analysis approached significance, it was followed by pair-

wise comparisons using Bonferroni adjustments. None of the pair-wise comparisons was statistically significant, all *p*'s > .08. Thus, differences in the reliabilities of the three measures were unlikely to have caused any observed differences in effect size. The correlation between the pessimism subscale (with items reverse coded) and the optimism subscale was .33, $p < .02$. The standard deviation of the correlation between the scales was .20.

Primary Analyses

Primary analyses involved evaluating effect size estimates using all outcomes from all studies (see Row 1 of Table 1). The effect size for the overall/combined scale was significant ($k = 61$, $n = 201$, $r = .026$, 95%CI [.013 - .039], $p < .001$), as were the effect sizes for the pessimism subscale ($k = 61$, $n = 201$, $r = .029$, 95%CI [.018 - .041], $p < .001$) and the optimism subscale ($k = 61$, $n = 201$, $r = .011$, 95%CI [.002 - .019], $p < .034$). Optimism, as assessed via the overall/combined scale or the optimism subscale, and the absence of pessimism, as assessed by pessimism subscale, were all associated with better physical health. It is also clear, however, that the effect size associated with the pessimism subscale was considerably larger than the effect size associated with optimism subscale, just under 3 times as large. This difference in the magnitude of the effect sizes was statistically significant ($Z = -2.403$, $p < .02$). Thus, the absence of pessimism was a significantly better predictor of physical health than was the presence of optimism. Forest plots containing individual study effect sizes categorized according the manner in which optimism and pessimism was assessed can be found in Figure 2 (overall/combined scale), Figure 3 (pessimism subscale), and Figure 4 (optimism subscale).

Stratification by Outcome

In addition to the overall analyses, several subsidiary analyses were conducted. These analyses grouped outcomes a priori into several different categories, including biomarkers, disease prevalence/incidence/progression, survival/mortality, hospital stay or re-occurrence, cardiac-related, metabolic, immune function, pulmonary, and pregnancy/fertility. These categories were not mutually exclusive (e.g., systolic blood pressure was coded as both a biomarker and as cardiac-related). Additional groupings were identified, but not analyzed

because they contained less than 6 studies per group. The findings relevant to the outcomes examined can be found in the lower portion of Table 1.

These subgroup analyses generally paralleled the findings obtained for the primary analyses. The effect sizes associated with the pessimism subscale tended to be larger and were more likely to be significant than those associated with the optimism subscale. The effect sizes and significance levels of the overall/combined scale fell in between the two subscales. More specifically, except for outcomes dealing with disease prevalence/incidence/progression, survival/mortality, hospital stay/readmittance, and those that were cardiac-related, the effect sizes for the overall/combined scale were significantly different from zero. With respect to the optimism subscale, 6 effect sizes were not significantly different from zero: biomarkers, disease prevalence/incidence/progression, hospital stay/readmittance, cardiac-related, metabolic, and pulmonary. In contrast, only 1 of the 9 effect sizes (hospital stay/readmittance) was not significantly different from zero for the pessimism subscale. For three sets of outcomes (biomarkers, immune function, and pregnancy) the difference in magnitude of the effect sizes for the optimism and pessimism subscales was statistically significant (Z = -2.987, $p < .003$, Z = -2.293, $p < .022$, and Z = -2.028, $p < .043$, respectively). For all of these subsets, the absence of pessimism was a stronger predictor of specific health outcomes than was the presence of optimism.[1]

Sensitivity

In order to determine if effect size estimates were driven by a single study, “leave-one-out” analyses were conducted to determine how the significance level of the aggregated effect sizes would change as each study in turn was removed from the analysis (Greenhouse & Iyengar, 2009). For the primary analyses, the reported effect sizes for the overall/combined scale and the pessimism subscale were not dependent upon any single study or studies. Each study in the analysis could be removed one by one and the effect size estimate still remained significant. The reported effect size for the optimism subscale, however, was rendered statistically insignificant when 1 of 2 separate studies were removed (Price et al., 2016; Sutin, 2013).

Leave-one-out analyses were also conducted for the subgroup analyses. The removal of one study did sometimes make the effect size become nonsignificant, and this happened more frequently for subgroup analyses involving the optimism subscale (rather than the overall/combined scale and the pessimism subscale). These differences among the predictors are not surprising, inasmuch as the effects for the optimism subscale were often weaker to start with. Predictably, subgroup analyses that contained fewer studies were also more vulnerable to leave-one-out analyses. More details on sensitivity are presented in Supplemental Online Table 3.

Moderator Analyses

In the primary analyses, the amount of heterogeneity of variance associated with the effect sizes for the overall/combined scale and the pessimism subscale were quite large (I^2 = 62.62% and I^2 = 60.20%, respectively). The heterogeneity of variance in the effect sizes for the optimism subscale was considerably smaller (I^2 = 27.02%). Although the I^2 for the optimism subscale was below the suggested cut point identified by Higgins and Thompson (2002), moderator analyses were also conducted on the optimism subscale—both in order to be consistent across measures and because a set of potential moderator variables had been identified a priori.

The following moderators were evaluated: year published, study objective, study design, participant status, age, percent of sample that was female, percent of sample that was white, the country of origin for the study, scale used, the number of psychosocial covariates used in the analyses, and total number of covariates included in the analyses. No statistically significant moderator effects emerged for any of the three predictors used.

Publication Bias

Guidelines proposed by Rothstein, Sutton, and Borenstein (2005) were used to examine for the presence and magnitude of publication bias. First, as previously noted, sensitivity analyses were performed to see if obtained effects were dependent on one or two outlying studies. These sensitivity analyses for the primary analyses revealed two studies that, when removed, caused the effect size for the optimism scale to become nonsignificant. The effect sizes for the overall/combined scale and the pessimism subscale were not dependent upon any one study.[2]

Next, funnel plots for the overall/combined scale, the pessimism subscale, and the optimism subscale from the primary analyses were inspected for bias (see Supplemental Online Figure 1, Supplemental Online Figure 2, and Supplemental Online Figure 3, respectively). For all the plots, studies with larger standard errors and larger effect sizes were clustered at the bottom of the plot, less so for the optimism subscale.

Rank correlation and regression procedures were also used to evaluate publication bias. Kendall's Tau (corrected for continuity) was nonsignificant for the overall/combined scale and each of the two subscales (all p's > .55). Egger's regression was significant for the overall scale (Intercept = .47, SE = .21, p < .04) and for the pessimism subscale (Intercept = .53, SE = .20, p < .02), but not for the optimism subscale (Intercept = .11, SE = .13, p > .40). Taken together, these general set of findings suggested that some publication bias did exist.

Given the evidence for publication bias, Duval and Tweedie's (2000) trim-and-fill procedure was used to provide a bias-corrected effect size estimate. Use of this procedure left the primary analyses essentially unchanged. Effects that were significant before correction for bias remained significant after correction. The magnitude of the effects sizes was also similar. More detailed data on publication bias for the overall analyses (as well as the stratified analyses by outcome) can be found in Supplemental Online Table 3.

Discussion

The results of the present reanalyses confirm the findings from earlier quantitative and qualitative reviews. The presence of optimism combined with the absence of pessimism (as assessed by the overall/combined scale) is a reliable predictor of physical health. This was true for an analysis that pooled all of the outcomes together and also true for the majority of analyses that examined subgroups of outcomes separately. This replication of prior findings is noteworthy inasmuch as over 80 percent of the studies included in the present reanalyses were not included in the previous meta-analysis (Rasmussen et al., 2009).

The novel findings concern the relative strength of optimism and pessimism in contributing to associations with health. Although each was a significant predictor of physical health, the

effect sizes associated with the absence of pessimism were generally greater in size than those associated with the presence of optimism. The magnitude of these differences was great enough to be significantly different for the analysis aggregating across outcomes, as well as for several of the analyses that investigated subgroups of outcomes separately. Adjustment of the findings for publication bias did little to alter the basic nature of the primary findings.

Moderator analyses were conducted on the effect sizes from the overall/combined scale, as well as the two subscales. These analyses failed to identify any significant moderator. It is of interest that there were no significant differences in effect sizes as a function of the type of study employed. Cross-sectional studies are open to a number of methodological criticisms, most notably the issue of reverse causality. Longitudinal studies examine associations across time, but without provisions for equating the health of participants at baseline. As such, longitudinal studies are subject to many of the same criticisms as are cross-sectional studies. Prospective studies provide the gold standard, in that they offer an assessment of the change in the outcome variable overtime (or otherwise start with participants who can be assumed to be equivalent in health at baseline). Given these considerations, it is especially striking that the moderator analyses revealed that study design did not significantly impact the magnitude of the effect sizes that were obtained.

The foregoing discussion speaks to the statistical reliability of the effects that emerged. A few words also need to be said about the magnitude of the effects that emerged. The effects sizes reported here appear small. Several considerations should be borne in mind, however, when evaluating the effect sizes obtained. First, as just noted, the effect sizes reported are adjusted for a host of factors, including those related to demographics, study design, and other confounding psychosocial factors. Thus, the effect sizes reported are unique to optimism and pessimism. It is not surprising that the effect sizes are somewhat small, especially so inasmuch as shared variance with related psychosocial factors had been removed.

The second point to make is that statistical effects, even small ones, can be quite meaningful when applied to large numbers of people. Take for example, the effect size

characterizing the association between the pessimism subscale and mortality. The corresponding adjusted odds ratio for this effect in the present reanalysis is 1.074 [95% CI (1.024, 1.126)]. In terms of the number of people who lived and died in the United States in 2016 (the year the most recent study in these reanalyses was published), this odds ratio implies that a 1-point change in the pessimism direction of the pessimism subscale corresponds to an increase in 97,914 deaths from all causes [95% CI (32,540, 162,641)].

Finally, it is worth mentioning that the size of the effects obtained using the present meta-analytic techniques are quite comparable to effects reported in other meta-analyses of psychosocial factors and physical health when the studies are put on this same metric [see, e.g., Richardson et al. (2012) for a meta-analysis of perceived stress and incident coronary heart disease and Kivimäki et al., 2012 for a meta-analysis of job strain and coronary heart disease]. Taken together, these considerations suggest that from a public health standpoint the magnitude of the effects obtained in the present analysis are nontrivial and quite comparable to other findings in the literature.

The present set of reanalyses has several potential limitations that should be highlighted. First, search terms for the present analysis relied heavily on the framework used by Rasmussen et al. (2009). The scheme used here is only one of many that could be adopted. Different search terms could yield a different corpus of studies, and the findings obtained using those different studies could be somewhat different.

Second, the yield rate for relevant studies was 32%. It is difficult to evaluate this yield rate compared to other meta-analytic studies. This is the case because the data required for the present study could not be extracted from published studies. Rather, the analysis was contingent on authors of those published studies reanalyzing their data and forwarding on the results of those re-analyses. It is likely that this extra requirement lowered the yield rate to some extent.

The third limitation concerns the homogeneous nature of the gender and racial composition of the participants. Although these factors differed somewhat from study to study, over 90% of the overall sample were white and women. Additionally, over 90% of the studies were conducted

in the United States. More studies are clearly needed to determine if the effects reported here are replicable in more diverse populations.

Fourth, the conduct of the present research was a group effort. The analyses could not have been done if consortium members had not conducted the needed analyses and forwarded their findings to the primary authors for further meta-analytic processing. On the positive side, the project represents one of the best examples of collaborative science in the truest sense of the term. On the negative side, the more people involved, the more potential there is for error. This concern is mitigated by the fact that the researchers involved had already published peer reviewed papers with these same data, and as such had already demonstrated significant capability with these analyses.

Finally, the outcomes examined in the present study all involved physical health. It is unclear if similar findings would obtain if mental health outcomes were examined. Perhaps optimism and pessimism would be equally robust as predictors of psychological well-being. Perhaps optimism would be stronger. It is important not to extrapolate the findings obtained with the present set of outcomes to possible findings involving other outcomes. Future research on psychological well-being should report results for the optimism and pessimism subscales separately, in order to evaluate the relative strength of the two dimensions in predicting outcomes in that domain.

There is a more nuanced point to be made here than simply to acknowledge that the differential impact of optimism and pessimism on psychological well-being needs to be explored. That is, stress has been identified as one potentially important factor that might mediate the impact of optimism (and pessimism) on physical health (Scheier & Carver, 2018). How? The idea is that stress (and stress-related emotions) might modulate downstream biological systems that underlie health and disease.

Optimists cope with and psychologically react to adversity in a different way than do pessimists (Segerstrom et al., 2017). It would be interesting to see within this context if the presence or absence of optimism and the presence or absence of pessimism relate differentially

to the various emotions that arise in reaction to stressful circumstances. It would further be interesting to see if these potentially different emotions (that characterize the reactions of optimists and pessimists to stress) might themselves be more or less strongly related to physical health outcomes. Answering questions such as these could further in a significant way our understanding of why it might be that the absence of pessimism is more strongly related to physical health outcomes than is the presence of optimism.

Limitations aside, the present findings have at least three implications. First, future research should, as a matter of course, provide effect size information for the overall/combined scale and the two subscales separately—a suggestion that has been made previously (Scheier et al., 1994). Such a practice is even more important now that quantitative data exist documenting the differential associations of the two subscales with physical health. With the complete complement of effect sizes reported, future research could continue to evaluate the importance of the separate contributions of optimism versus pessimism without the need to establish consortiums.

The present findings also hold important implications for positive psychology (Peterson & Park, 2003; Seligman & Csikszentmihalyi, 2000). Positive psychology emphasizes those characteristics that enable people to experience full, industrious, and resilient lives. As such, it stands in contrast to traditional views that tend to focus on negative attributes, such as depression, anxiety, and other characteristics which undermine successful living. Dispositional optimism is often described as a good example of a variable falling within the positive psychology domain (e.g., Dunn, 2018). As the present data make clear, however, the presence of optimism does not provide the whole story. Optimism is important, but it does not appear to be as important as the absence of pessimism in predicting physical health.

In the future, researchers in positive psychology might benefit from taking these findings into account when planning and conducting research. Researchers should examine more closely the predictor variables they are using to see if negative and positive characteristics might be intermingled in the measures employed. If so, an effort should be made to tease apart the positive

and negative components of the measures to determine what is in fact responsible for doing the predicting. Ultimately, it may turn out that it is the positive aspects of the measures that are important, but it also possible that the negative features are the ones driving the observed associations. Only by explicitly evaluating these possibilities will we know for sure.

The final implication concerns interventions. Future efforts to design and adapt interventions to promote better health should keep in mind the differential links between optimism, pessimism, and physical health. In this regard, it is interesting that some cognitive behavior therapies seem to put a greater emphasis on lessening pessimism than they do on promoting optimism. One example of such an intervention concerns cognitive restructuring (Leahy & Rego, 2012), in which participants are trained to challenge the automatic thoughts, beliefs, and expectancies underlying negative feelings. Participants confront their automatic, negative thinking by systematically, and explicitly monitoring their moods and assessing in a more objective fashion the information in the ongoing context that either supports or challenges their negative thoughts. Perhaps existing interventions that focus more on lessening pessimism such as those involving cognitive restructuring will be more successful in promoting better health than will those that place a greater weight on promoting optimism, or even those that place an equal weight on both components. Note that it is not a matter of causing harm, but more a matter of targeting the component that offers the most gain.

It is also possible, however, that things are more complicated. Perhaps what works best will depend on the nature of the outcome of interest (e.g., health behaviors versus biological pathways). Intervention efforts with respect to optimism, pessimism, and physical health are still in their infancy. As research in the intervention domain continues to evolve, it would seem prudent to keep the distinction between optimism and pessimism in mind. Doing so may prove profitable both practically and theoretically.

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Footnotes

[1] As Table 1 shows, RVE could only be used for some of the analyses conducted because of constraints on degrees of freedom. As a supplement to these RVE analyses, traditional meta-analytic techniques were also used to replicate the findings produced using RVE. These supplemental analyses yielded largely the same effects as did the RVE method. All primary analysis ES's that were significant using one technique were significant using the other, and the ES's themselves were also quite similar. The biggest difference between the estimated ES's was for the optimism subscale. The RVE method produced a slightly larger ES estimate than did the analysis using one average ES per study (.011 versus .007, respectively). The subgroup analyses that were conducted were also similar, especially for the two subscales. Convergence of these two methodologies increases the confidence in the results that are reported.

[2] RVE was used to assess sensitivity whenever the empirically calculated degrees of freedom for the analysis was 4 or greater. Traditional meta-analytic methods, using an average outcome per study, were used to assess sensitivity when the degrees of freedom were less than 4. Traditional meta-analytic methods were also used for the remaining publication bias analyses that are reported.

Table 1. Effect size estimates for the overall/combined scale, the pessimism subscale, and the optimism subscale.

	Optimism/Pessimism Overall/Combined					Optimism Subscale Only					Pessimism Subscale Only					Subscale Difference[a]	
Outcomes	***k***	***n*[b]**	***Z'***	***95% CI***	***p* <**	***k***	***n*[b]**	***Z'***	***95% CI***	***p* <**	***k***	***n*[b]**	***Z'***	***95% CI***	***p* <**	***Z***	***p* <**
All	61	201	.026	.013, .040	.001	61	201	.011	.002, .019	.034	61	201	.029	.018, .041	.001	-2.403	.016
Biomarkers	39	140	.030	.015, .046	.001	39	140	.006	-.008, .020	.352	39	140	.046	.030, .062	.001	-2.749	.007
Disease Prevalence/ Incidence/ Progression	15	30	.012	-.009, .034	.189	15	30	.011	-.008, .031	.191	15	15[†]	.008	.003, .012	.001	-0.625	.532
Survival/ Mortality	9	15	.024	-.014, .061	.162	9	9[†]	.007	.002, .011	.006	9	9[†]	.020	.007, .033	.004	-0.980	.327
Hospital Stay/ Re-admit	7	11	.002	-.040, .045	.899	7	11	.018	-.011, .047	.161	7	11	-.002	-.062, .057	.921	-0.845	.398
Cardiac-Related	20	71	.016	-.006, .038	.121	20	71	.014	-.007, .034	.158	20	20[†]	.012	.007, .016	.001	-0.672	.502
Metabolic	13	29	.028	.000, .056	.049	13	29	.006	-.026, .038	.672	13	13[†]	.049	.035, .063	.001	-1.572	.116
Immune Function	10	10[†]	.011	.004, .018	.003	10	10[†]	.005	-.015, .025	.022	10	10[†]	.023	.000, .046	.050	-2.293	.022
Pulmonary	6	6[†]	.008	.001, .015	.032	6	6[†]	.008	.001, .015	.753	6	6[†]	.011	.004, .018	.004	0.314	.753
Pregnancy/ Fertility	7	7[†]	.042	.013, .071	.005	7	7[†]	.010	-.031, .051	.043	7	7[†]	.062	.034, .091	.001	-2.028	.043

Note: [a]As determined by the Wilcoxon Signed Ranks Test. [b]Number of effect sizes. [†]Estimated using one effect size per study given empirical degrees of freedom < 4.

Figure 1. Flow chart showing inclusion/exclusion of studies identified from intitial search.

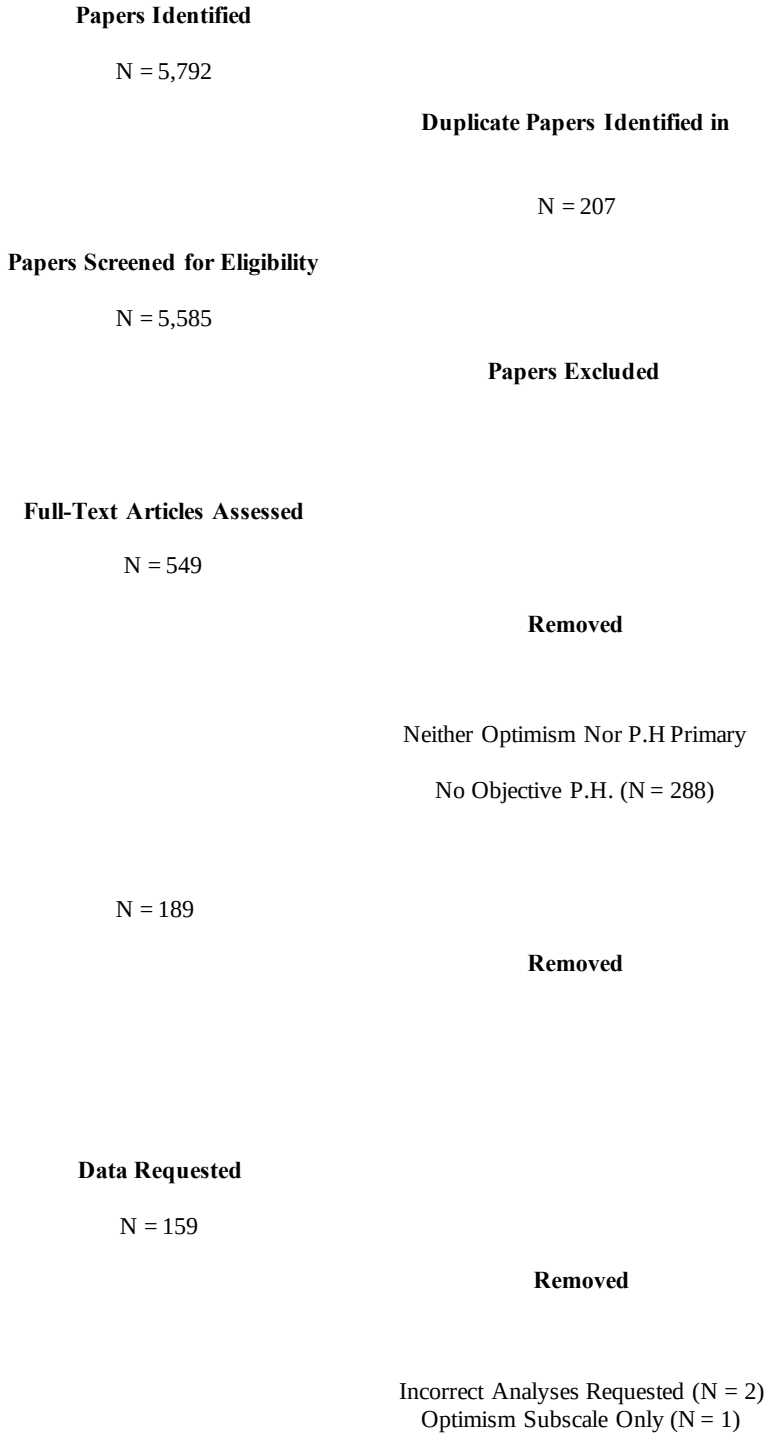
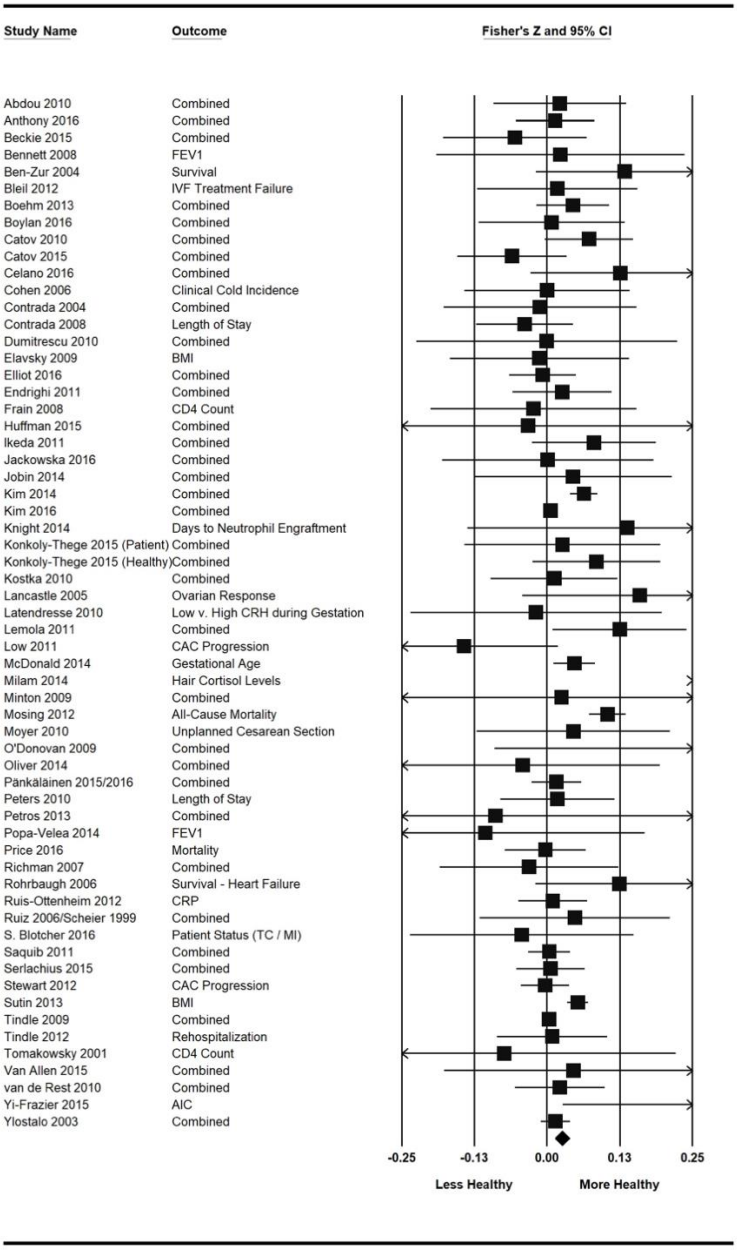
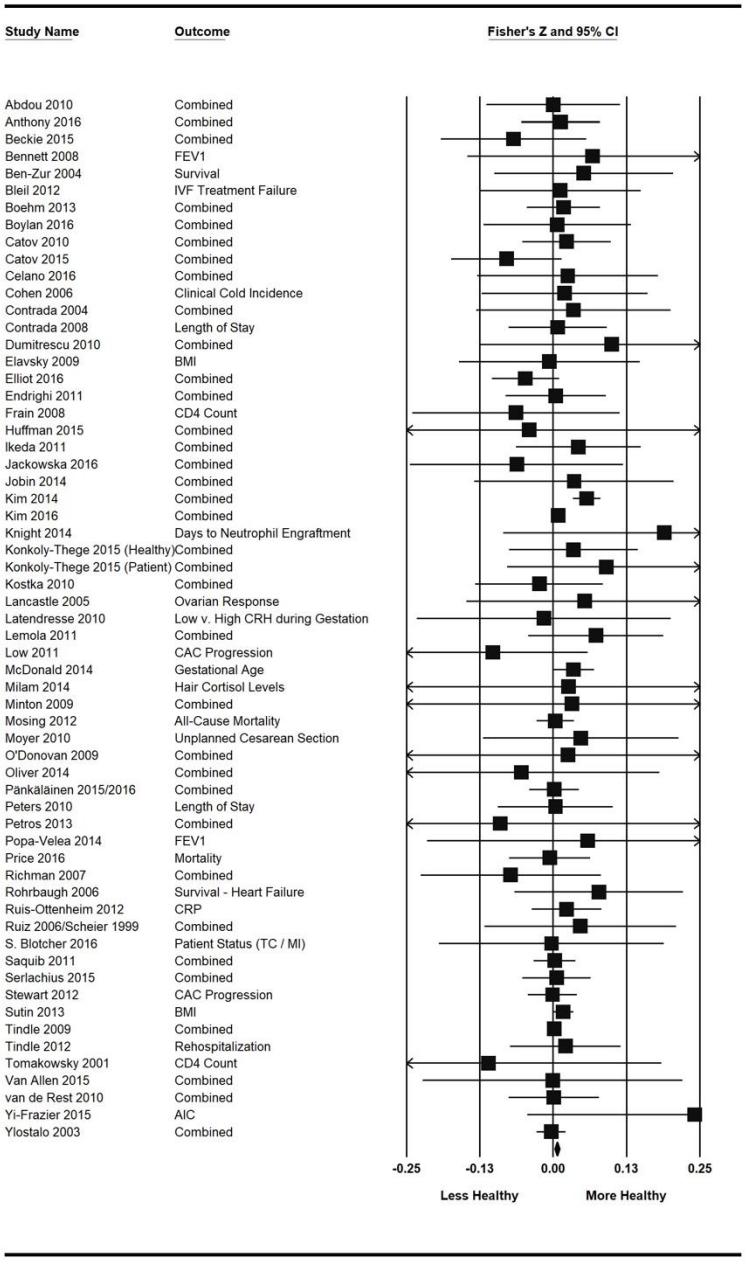


Figure 2. Forest plot of effect sizes associated with the overall/combined scale.



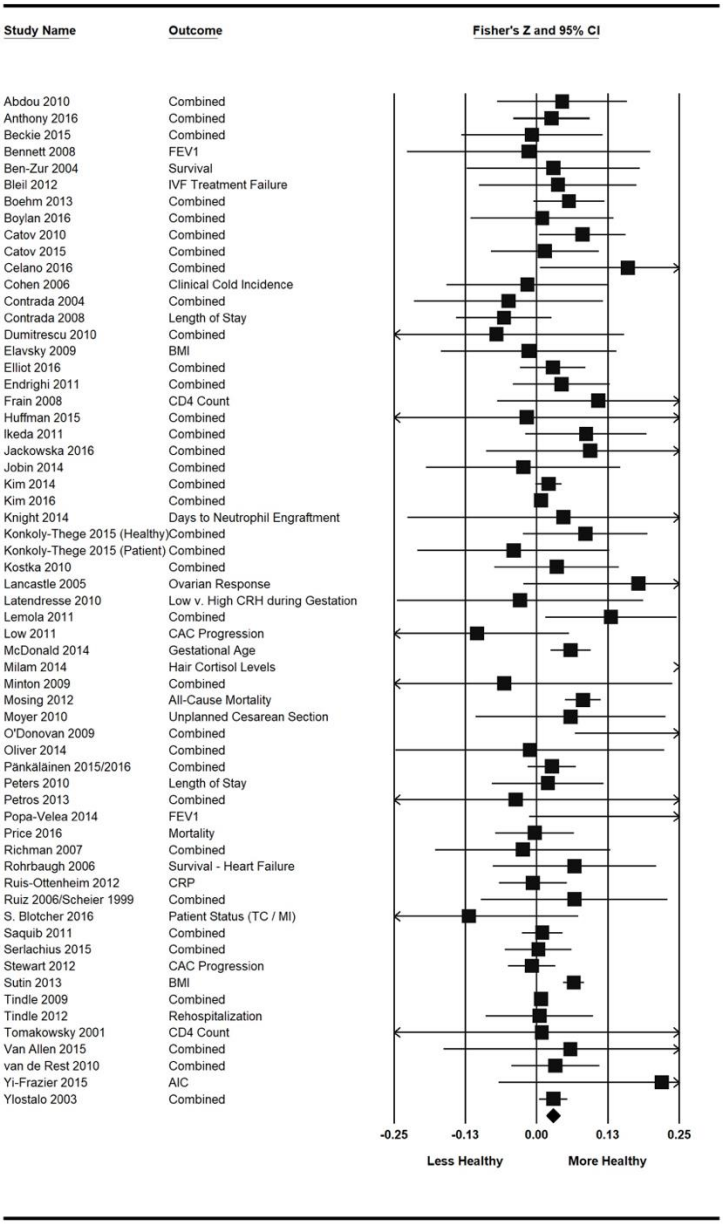
Note: Diamond symbol at bottom of forest plot reflects average effect size across studies.

Figure 3. Forest plot of effect sizes associated with the optimism subscale.



Note: Diamond symbol at bottom of forest plot reflects average effect size across studies.

Figure 4. Forest plot of effect sizes associated with the pessimism subscale.



Note: Diamond symbol at bottom of forest plot reflects average effect size across studies.

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Supplemental Online Table 2. Characteristics of studies included in the meta-analysis, outcomes assessed, and covariates controlled.

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Abdou et al. 2010	N = 297 M_{age} = 31 % female = 100 % white = 77 USA	Systolic BP Diastolic BP	C	ethnicity, childhood SES, adult SES, marital status, depressive symptoms, perceived stress, state anxiety, self-esteem
Anthony, Kritz-Silverstein, & Barrett-Connor 2016	N = 876 M_{age} = 74 % female = 58 % white = USA	All-Cause Mortality CVD-Mortality CHD-Mortality Cancer-Mortality	L	Age, sex, medications
Beckie et al. 2015	N = 252 M_{age} = 63 % female = 100 % white = 82 USA	HDL-Cholesterol LDL-Cholesterol Triglycerides Fasting Glucose Body Mass Index Body Fat Percentage Systolic BP Diastolic BP Heart Rate	C	Age, sex, marital status, state-trait anxiety, depression, self-reported stress, anxiety, depression, hope
Ben-Zur, Rappaport, & Uretzky 2004	N = 168 M_{age} = 61 % female = 19 % white = NA Israel	Survival	L	Current anxiety
Bennett et al. 2008	N = 87 M_{age} = 13 % female = 56 % white = 87 USA	FEV1	C	Age, gender, SES, state anxiety, trait anxiety, depressive symptoms

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Bleil et al. 2012	N = 204 M_{age} = 35 % female = 100 % white = 77 USA	IVF Treatment Failure	P	Trait negative affect, age, SES, income, parity, duration of attempted pregnancy, history of oral medication use, history of injectable medication use, history of intrauterine insemination, number of infertility-related diagnoses.
Boehm et al. 2013	N = 982 M_{age} = 55 % female = 55 % white = 93 USA	trans-β-carotene 13-cis-β-carotene α-carotene β-cryptoxanthin lutein zeaxanthin lycopene α-tocopherol γ-tocopherol	C	Age, sex, race/ethnicity, education, household income, time between assessments
Boylan et al. 2016	N = 246 M_{age} = 32 % female = 0 % white = 44 USA	Systolic BP Reactivity Diastolic BP Reactivity Heart Rate Reactivity HF-HRV Reactivity Systolic BP Recovery Diastolic BP Recovery Heart Rate Recovery HF-HRV Recovery	P	Age, race, child SES, marital status, task demand, current SES
Catov & Markovic 2010	N = 667 M_{age} = 22 % female = 100 % white = 70 USA	Gestational Age Infant Birth Weight Centile	L	Trait affect, maternal age at delivery, education, marital status, race/ethnicity, receipt of public assistance, preeclampsia
Catov et al. 2015	N = 429 M_{age} = 25	log-C-reactive protein log-Interleukin-6	C	Trait anxiety, maternal race, gestational age at blood draw,

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
	% female = 100 % white = 73 USA	Gestational Age		Maternal age at delivery, education, receipt of public assistance, neighborhood levels
Celano et al. 2016	N = 164 M_{age} = 62 % female = 16 % white = 84 USA	C-reactive protein Interleukin 6 TNF-α sICAM-1 NT-proBNP Rehospitalization	P	Age, sex, gratitude, depression, anxiety, baseline biomarker
Cohen et al. 2006	N = 193 M_{age} = 37 % female = 51 % white = NA USA	Clinical Cold Incidence	P	Age, sex, education, race, virus type, season of exposure, mastery, self-esteem, life engagement, extraversion, positive emotional style, negative emotional style
Contrada et al. 2004	N = 142 M_{age} = 65 % female = 19 % white = 84 USA	Length of Stay Postoperative Complications	L	Age, sex, marital status, education, anesthesia time, comorbidity index, depressive symptoms, trait hostility, religiousness (attendance, prayer, beliefs)
Contrada et al. 2008	N = 550 M_{age} = 65 % female = 26 % white = 88 USA	Length of Stay	L	Age, ethnicity (non-White), trait anger, anxiety, depressive symptoms, religious involvement, history of atrial fibrillation, duration of surgery
Dumitrescu & Kawamura 2010	N = 79 M_{age} = 41 % female = 61 % white = NA Norway	Body Mass Index Total Remaining Teeth Plaque Index Calculus Index Bleeding on Probing Index Mean PD	C	Age, sex, type A, anxiety, depressive symptoms, emotional intelligence, stress, self-esteem, and satisfaction with life

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
		Mean CAL Number of Sites with PD > 6mm Number of Sites with CAL > 5mm Number of Teeth with PD > 6mm		
Elavsky & McAuley 2009	N = 164 M_{age} = 50 % female = 100 % white = 83 USA	Body Mass Index	L	Age, marital status, education, neuroticism, trait anxiety, baseline body mass index
Elliot & Chapman 2016	N = 1152 M_{age} = 57 % female = 57 % white = 80 USA	Interleukin 6 C-reactive protein	L	Age, sex, race, SES, chronic disease burden, medications, negative affect, positive affect, adult stress exposure, childhood stress exposure, self-esteem, perceived control
Endrighi, Hamer, & Steptoe 2011	N = 527 M_{age} = 63 % female = 46 % white = NA UK	Cortisol Awakening Response Total Daily Cortisol Output Cortisol Diurnal Slope Total Task Cortisol Output Cortisol Reactivity Cortisol Recovery	L P	Age, sex, employment grade, depressive symptoms, time of awakening
Frain et al. 2008	N = 125 M_{age} = NA % female = 12 % white = NA USA	CD4+ T-cell Count	C	Age, years since diagnosis
Huffman et al. 2015	N = 22 M_{age} = 64 % female = 41 % white = 77 USA	Length of Stay Body Mass Index LVEF	C	Age, sex, race, marital status, anxiety, depressive symptoms, PANAS

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Ikeda et al. 2011	N = 340 M_{age} = 71 % female = 0 % white = NA USA	C-reactive protein ICAM-1 VCAM-1 Interleukin 6 TNF-RII	P	Baseline age, change in Age, educational attainment, brief symptom inventory depression
Jackowska et al. 2016	N = 119 M_{age} = 26 % female = 100 % white = 72 UK	Sleep Efficiency Sleep Latency Sleep Duration	C	Age, relationship status, ethnicity, life satisfaction, positive affect, negative affect, depressive symptoms
Jobin, Wrosch, & Scheier 2014	N = 135 M_{age} = 72 % female = 53 % white = 80 USA	Average Cortisol AUC Average Cortisol Awakening Level Average Cortisol Evening Level	P	Age, sex, education, income, subjective social status, average perception of stress across days, outcome at wave 2.
Kim et al. 2016	N = 70021 M_{age} = 70 % female = 100 % white = 98 USA	All-Cause Mortality Heart Disease Stroke Respiratory Disease Infection Total Cancer Lung Cancer Breast Cancer Colorectal Cancer Ovarian Cancer	L	Age, race, marital status, education level, husband's education level, father's education level, depression status
Kim et al. 2014	N = 6808 M_{age} = 70 % female = 59 % white = 71 USA	Stroke Incidence Incident Heart Failure	P	Stroke: Age, gender, race/ethnicity, marital status, education, positive affect, anxiety, cynical hostility, depression, negative affect, neuroticism

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
				Incident heart failure: Age, gender, race/ethnicity, marital status, education, total wealth, anxiety, cynical hostility, depression
Knight et al. 2014	N = 54 M_{age} = 47 % female = 48 % white = 86 USA	Days to Neutrophil Engraftment	L	Age, race, sex, conditioning regimen, stem cell source, anxiety
Konkoly-Thege et al. 2015 (Study 5)	N = 138 M_{age} = 65 % female = 49 % white = NA Hungary	Brachial Augmentation Index Aortic Augmentation Index Aortic Pulse Wave Velocity Aortic Systolic Blood Pressure Systolic Area Index Diastolic Area Index Systolic BP Diastolic BP Mean Arterial Pressure Forced Vital Capacity FEV1	C	Sex, age, education, life satisfaction, general well-being, meaning in life, sense of coherence
Konkoly-Thege et al. 2015 (Study 5a)	N = 321 M_{age} = 43 % female = 71 % white = NA Hungary	Brachial Augmentation Index Aortic Augmentation Index Aortic Pulse Wave Velocity Aortic Systolic Blood Pressure Systolic Area Index Diastolic Area Index Systolic BP Diastolic BP Mean Arterial Pressure Forced Vital Capacity FEV1	C	Sex, age, education, life satisfaction, general well-being, meaning in life, sense of coherence

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Kostka & Jachimowicz 2010	N = 324 M_{age} = 75 % female = 78 % white = NA Poland	Systolic BP Diastolic BP Hypertension Ischemic Heart Disease Post MI Diabetes	C	Age, sex, education, health locus of control, self-efficacy
Lancastle & Boivin 2005	N = 97 M_{age} = 33 % female = 100 % white = NA UK	Ovarian Response	L	Trait anxiety
Latendresse & Ruiz 2010	N = 85 M_{age} = 26 % female = 100 % white = 69 USA	Low v. High CRH during Gestation	C	Perceived inadequacy of income, depressive symptoms, perceived stress (measured with perceived stress scale)*
Lemola et al. 2010	N = 291 M_{age} = 8 % female = 51.55 % white = NA Finland	Sleep Latency Sleep Efficiency	L	Age, sex, parental level of education, parental optimism, self-esteem, social competence
Low et al. 2011	N = 149 M_{age} = 64 % female = 100 % white = NA USA	CAC Progression	P	Age, baseline CAC, time between assessments, psychological risk (depressive symptoms, perceived stress, cynicism, anger-in), mastery, self-esteem

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
McDonald et al. 2014	N = 3021 M_{age} = NA % female = 100 % white = 80 Canada	Gestational Age	P	Maternal age, education, household income, ethnicity, personal/family history of pre-term birth, reproductive history, mode of conception, pregnancy complications, poor prenatal care, perceived stress (measured with the perceived stress scale)*
Milam et al. 2014	N = 27 M_{age} = 15 % female = 70 % white = NA USA	Hair Cortisol Levels	C	Perceived stress, stressful life events, depressive symptoms
Minton et al. 2009	N = 47 M_{age} = 74 % female = 100 % white = 100 USA	Average Cortisol AUC Average Cortisol Awakening Level Average Cortisol Evening Level	P	Age, length of marriage, psychological stress, life satisfaction, spiritual well-being, baseline* outcome
Mosing et al. 2012	N = 3752 M_{age} = 61 % female = 69 % white = NA Australia	All-Cause Mortality	L	SES, age, sex, neuroticism, psychoticism, extraversion, social desirability
Moyer et al. 2010	N = 141 M_{age} = 30 % female = 100 % white = NA China	Unplanned Cesarean Section	P	Labor duration, birth complications, previous abortion, previous miscarriage, pregnancy complications, self-reported difficulty
O'Donovan et al. 2009	N = 36 M_{age} = 61	Telomere Length Interleukin 6	C	Age, caregiver status, perceived stress, neuroticism

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
	% female = 100 % white = 81 USA			
Oliver et al. 2014	N = 72 M_{age} = 19 % female = 50 % white = 97 USA	FEV1 Body Mass Index	C	Age, sex, total stigma, total distress
Pankalainen et al. 2016	N = 2719 M_{age} = NA % female = NA % white = NA Finland	CHD-Mortality	P	Age, sex, CHD at baseline
Pankalainen, Kerola, & Hintikka 2015	N = 1697 M_{age} = NA % female = NA % white = NA Finland	CHD Incidence	L	Age, sex
Peters et al. 2010	N = 401 M_{age} = 54 % female = 54 % white = NA Netherlands	Length of Stay	L	Age, sex, type of operation, anatomical region
Petros, Opacka-Juffry, & Huber 2013	N = 32 M_{age} = 29 % female = 63 % white = NA UK	DHEA-S Cortisol DHEA-S / Cortisol Ratio	C	Age, gender, self-efficacy, anxiety, depressive symptoms
Popa-Velea et al. 2014	N = 54 M_{age} = 58 % female = 48 % white = NA	FEV1	C	

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
	Romania			
Price et al. 2016	N = 798 M_{age} = 61 % female = 100 % white = NA Australia	Mortality	P	Depression, age at diagnosis, grade at diagnosis, time since diagnosis, current treatment, age, time post-diagnosis to study entry
Richman et al. 2007	N = 165 M_{age} = 34 % female = 45 % white = 43 USA	Systolic BP Reactivity Diastolic BP Reactivity Heart Rate Reactivity Systolic BP Recovery Diastolic BP Recovery Heart Rate Recovery	P	Baseline outcome (for reactivity outcomes but not recovery outcomes), age, gender, socioeconomic status, race, overall hostility, cynicism, perceived discrimination (in past year and in life).
Rohrbaugh, Shoham, & Coyne 2006	N = 189 M_{age} = 53 % female = 26 % white = 83 USA	Survival - Heart Failure	L	Sex, marital quality, self-efficacy, psychological distress, hostility, neuroticism
Ruis-Ottenheim et al. 2012	N = 1084 M_{age} = 71 % female = 36 % white = 100 Netherlands	C-reactive protein	C	Age, sex, marital status, history of cancer, history of cardiovascular disease, cohort (if appropriate)
Ruiz et al. 2006	N = 111 M_{age} = 61 % female = NA % white = NA USA	Number of Grafts Number of Vessels Occluded 50% Total Cholesterol Ejection Fraction < 40% Acute MI Angina	C L	Age, education, employment, neuroticism, depressive symptoms, relationship satisfaction

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Salmoirago-Blotcher et al. 2016	N = 107 M_{age} = 61 % female = 100 % white = 85 USA	Patient Status (TC / MI)	C	Age, ethnicity, education, income, psychological distress, perceived stress, hostility, type D personality
Saquib et al. 2011	N = 2967 M_{age} = 53 % female = 100 % white = NA USA	Breast Cancer All-Cause Mortality	P L	Age at randomization, race/ethnicity, menopausal status, initial tumor type, initial tumor stage, anti-estrogen use, clinical site, time between cancer diagnosis and study entry, hot flashes, randomization group, interaction between intervention group and hot flashes, marital status, education, hostility
Scheier et al. 1999	N = 284 M_{age} = 63 % female = 30 % white = 99 USA	Angina - Rehospitalization MI - Rehospitalization PTCA - Rehospitalization All-Cause Rehospitalization	L	Age, education, employment, neuroticism, depressive symptoms, relationship satisfaction
Serlachius et al. 2015	N = 1113 M_{age} = 32 % female = 58 % white = NA Finland	Total Cholesterol Body Mass Index Systolic BP Diastolic BP Fasting Glucose	P	Age, sex, ideal cardiovascular health at baseline, medication use at baseline, level of education, occupational status, depressive symptoms
Stewart et al. 2012	N = 2171 M_{age} = 40 % female = 58 % white = 57 USA	CAC Progression	P	Age, sex, race, education, depression

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Sutin 2013	N = 11207 M_{age} = 68 % female = 60 % white = 85 USA	Body Mass Index	C	Age, sex, ethnicity, education
Tindle et al. 2009	N = 97253 M_{age} = 63 % female = 100 % white = 92 USA	Incident MI Incident CHD All-Cause Mortality CHD-Mortality CVD-Mortality Cancer-Mortality	P	Age, race/ethnicity, education, income, depressive symptoms, and cynical hostility
Tindle et al. 2012	N = 430 M_{age} = 65 % female = 39 % white = 88 USA	Rehospitalization	L	Age, sex, education, marital status, hamilton depression rating scale
Tomakowsky et al. 2001	N = 47 M_{age} = 39 % female = 0 % white = 69 USA	CD4+ T-cell Count	P	Age, education, current employment status, years since HIV diagnosis, negative affectivity, baseline CD4+ T-cell count, duration until follow-up
Van Allen et al. 2015	N = 81 M_{age} = 14 % female = 48 % white = 89 USA	HbA1c Frequency of SMBG	P	Age, hope, baseline (Time 1) of the outcome variable
Van de Rest et al. 2010	N = 644 M_{age} = 69 % female = 22 % white = NA Netherlands	Body Mass Index Systolic BP Diastolic BP Glucose Total cholesterol HDL-Cholesterol	C	Age, living alone, education, depressive symptoms

Authors/Year	Sample Characteristics	Outcome	Design[1]	Covariates
Yi-Frazier et al. 2015	N = 50 M_{age} = 16 % female = 52 % white = 94 USA	HbA1c	C	Age, sex, race, education, income, self-esteem, diabetes-related distress, duration of diabetes
Ylostalo et al. 2003	N = 6033 M_{age} = 31 % female = 52 % white = NA Finland	Body Mass Index Total Cholesterol HDL-Cholesterol Triglycerides Tooth Loss	C	Life satisfaction, education, gender, income, marital status

Note: AUC = area under the curve; BP = blood pressure; CAC = coronary artery calcium; CAL = clinical attachment level; CHD = coronary heart disease; CRH = corticotropin-releasing hormone; CVD = cardiovascular disease; DHEA-S = dehydroepiandrosterone sulfate; FEV1 = forced expiratory volume in 1 second; HbA1c = glycated hemoglobin; HDL = high-density lipoprotein; HF-HRV = high-frequency heart rate variability; ICAM-1 = intercellular adhesion molecule 1; IVF = in-vitro fertilization; LDL = low-density lipoprotein; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NT-proBNP = N-terminal pro b-type natriuretic peptide; PANAS = Positive and Negative Affect Schedule; PD = probing density; PTCA = ; sICAM-1 = soluble intercellular adhesion molecule-1; SMBG = self-monitoring of blood glucose; SES = socioeconomic status; TC = takotsubo cardiomyopathy; TNF-α = tumor necrosis factor alpha; TNFR-2 = tumor necrosis factor receptor 2; VCAM-1 = vascular cell adhesion molecule 1

[1]Design of the study was coded as either cross-sectional (C), longitudinal (L), or prospective (P).

Supplemental Online Table 3. Sensitivity analyses, publication bias estimates, and adjusted effect sizes.

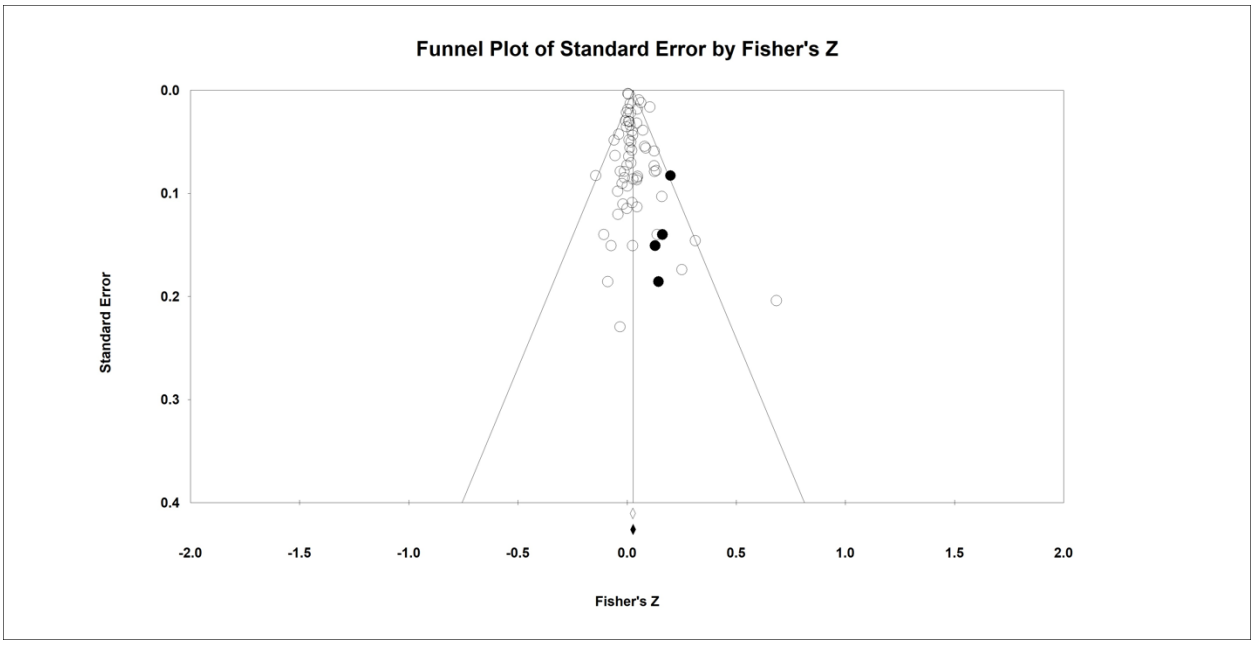
Overall/Combined Scale						
			Trim and Fill			
Outcome	***k***	**Number of individual studies removed that would make p > .05**	**Number Studies Trimmed/ Filled Left of Mean**	**Number Studies Trimmed/ Filled Right of Mean**	**Observed Point Estimate (95%CI)**	**Adjusted Point Estimate (95%CI)**
All Outcomes	61	0	0	4	.026 (.015 - .038)	.028 (.016 - .040)
Biomarkers	39	0	0	2	.032 (.017 - .046)	.033 (.018 - .047)
Disease Prevalence/ Incidence/ Progression	15	NA	0	3	.013 (.001 - .024)	.014 (.002 - .026)
Survival/ Mortality	9	NA	0	0	.019 (.006 - .033)	As Observed
Hospital Stay/ Re-admit	7	NA	0	0	.002 (-.042 - .046)	As Observed
Cardiac-Related	20	NA	0	2	.016 (.003 - .030)	.019 (.005 - .034)
Metabolic	13	8	0	2	.030 (.007 - .054)	.033 (.012 - .054)
Immune Function	10	2*	0	1	.011 (.004 - .018)	.011 (.004 - .018)
Pulmonary	6	1*	0	1	.008 (.001 - .015)	.008 (.001 - .016)
Pregnancy/ Fertility	7	1*	0	0	.042 (.013 - .071)	As Observed
Pessimism Subscale						
			Trim and Fill			

Outcome	***k***	**Number of individual studies removed that would make p > .05**	**Number Studies Trimmed/ Filled Left of Mean**	**Number Studies Trimmed/ Filled Right of Mean**	**Observed Point Estimate (95%CI)**	**Adjusted Point Estimate (95%CI)**
All Outcomes	61	0	0	2	.029 (.018 - .041)	.030 .019 -.042
Biomarkers	39	0	0	1	.045 (.029 - .063)	.047 (.036 - .062)
Disease Prevalence/ Incidence/ Progression	15	0*	0	0	.008 (.003 - .012)	As Observed
Survival/ Mortality	9	0*	0	0	.020 (.007 - .033)	As Observed
Hospital Stay/ Re-admit	7	NA	0	0	-.002 (-.046 - .042)	As Observed
Cardiac-Related	20	0*	0	0	.012 (.007 - .016)	As Observed
Metabolic	13	0*	0	3	.049 (.035 - .063)	.050 (.036 - .064)
Immune Function	10	7*	2	0	.023 (.000 - .046)	.020 (-.013 - .053)
Pulmonary	6	5*	0	1	.011 (.004 - .018)	.011 (.004 - .019)
Pregnancy/ Fertility	7	0*	0	1	.062 (.034 - .091)	.064 (.035 - .093)
Optimism Subscale						
			Trim and Fill			
Outcome	***k***	**Number of individual studies removed that would make p > .05**	**Number Studies Trimmed/ Filled Left of Mean**	**Number Studies Trimmed/ Filled Right of Mean**	**Observed Point Estimate (95%CI)**	**Adjusted Point Estimate (95%CI)**
All Outcomes	61	2	4	0	.007	.007

					(.003 - .011)	(.003 - .011)
Biomarkers	39	NA	1	0	.009 (-.003 - .020)	.009 (-.003 - .020)
Disease Prevalence/ Incidence/ Progression	15	NA	0	2	.011 (.000 - .020)	.012 (.001 - .022)
Survival/ Mortality	9	1*	2	0	.007 (.002 - .011)	.006 (.002 - .011)
Hospital Stay/ Re-admit	7	NA	0	1	.018 (-.026 - .062)	.020 (-.024 - .064)
Cardiac-Related	20	NA	0	1	.013 (.001 - .025)	.015 (.002- .028)
Metabolic	13	NA	0	3	.004 (-.026 - .035)	.013 (-.016 - .041)
Immune Function	10	8*	0	3	.005 (-.015 - .025)	.008 (-.017 - .032)
Pulmonary	6	NA*	0	1	.008 (.001 - .015)	.008 (.001 - .016)
Pregnancy/ Fertility	7	6*	1	0	.010 (-.031 - .051)	.011 (-.025 - .047)

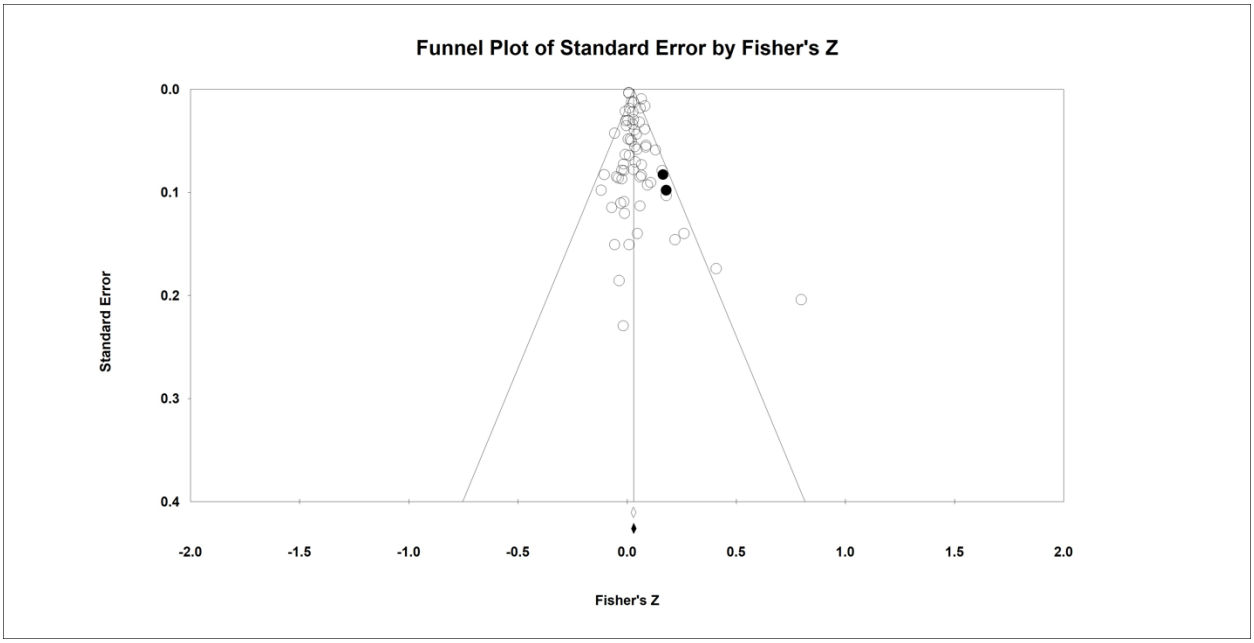
Note: NA = Not applicable because initial ES estimate was not significant. *Denotes leave one out analysis was based on traditional meta-analytic methodology using one average effect size per study. The remainder of the leave one out analyses were conducted using RVE. Number of studies trimmed and filled and adjusted effect sizes are based on traditional meta-analytic methodology using one average effect size per study.

Online Supplemental Figure 1. Funnel plot for effects involving the overall/combined scale.



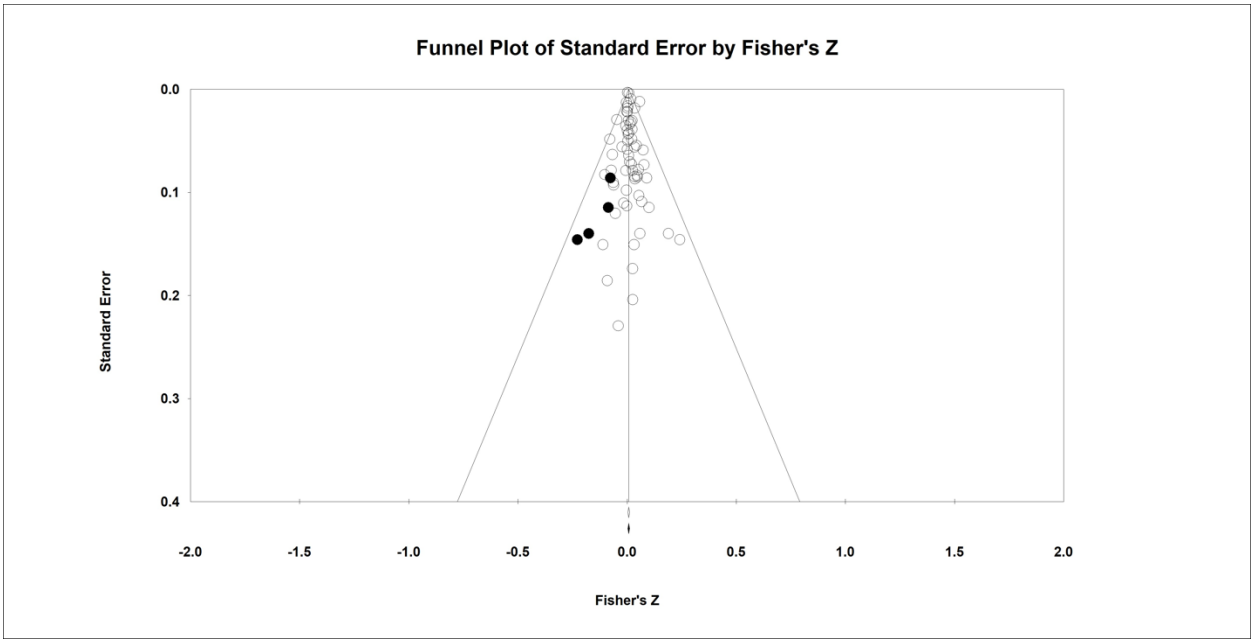
Note: Open circles represent observed values and filled circles represent values imputed in order to correct for potential publication bias.

Supplemental Online Figure 2. Funnel plot for effects involving the pessimism subscale.



Note: Open circles represent observed values and filled circles represent values imputed in order to correct for potential publication bias.

Supplemental Online Figure 3. Funnel plot for effects involving the optimism subscale.



Note: Open circles represent observed values and filled circles represent values imputed in order to correct for potential publication bias.