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Health Benefits:

Meta-Analytically Determining the Impact of Well-Being on Objective Health Outcomes

Ryan T. Howell
San Francisco State University

Margaret L. Kern and Sonja Lyubomirsky
University of California, Riverside

Send comments to: Ryan Howell, Ph.D.
Department of Psychology
1600 Holloway Avenue
San Francisco, CA 94132
rhowell@sfsu.edu

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Abstract

This research synthesis integrates findings from 150 experimental, ambulatory, and longitudinal studies that tested the impact of well-being on objective health outcomes. Results demonstrated that well-being positively impacts health outcomes (r = .14). Well-being was found to be positively related to short-term health outcomes (r = .15), long-term health outcomes (r = .11), and disease or symptom control (r = .13). Results from the experimental studies demonstrated that inductions of well-being lead to healthy functioning and inductions of ill-being lead to compromised health at similar magnitudes. Thus, the effect of subjective well-being on health is not solely due to ill-being having a detrimental impact on health, but also to well-being having a salutary impact on health. Additionally, the impact of well-being on improving health was stronger for immune system response and pain tolerance, whereas well-being was not significantly related to increases in cardiovascular and physiological reactivity. These findings point to potential biological pathways, such that well-being can directly bolster immune functioning and buffer the impact of stress.

KEY WORDS: Objective Health Outcomes, Physical Functioning, Subjective Well-Being, Positive Affect, Health Processes, Meta-Analysis

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Increasing evidence suggests that happiness not only makes people feel good, but helps them accrue numerous advantages and rewards across multiple life domains, including work (Boehm & Lyubomirsky, in press), marriage (e.g., Marks & Fleming, 1999), and coping (e.g., Scheier et al., 1989). One of the most critical domains to explore is health. Indeed, two recent literature reviews summarized evidence that increased well-being is associated with improved health outcomes and lower morbidity (Lyubomirsky, King, & Diener, 2005; Pressman & Cohen, 2005). For example, happy individuals report having superior health and experiencing fewer unpleasant physical symptoms (Lyubomirsky, Tkach, & DiMatteo, 2006; Mroczek & Spiro, 2005); and higher levels of trait positive affect are associated with better quality of life for cancer patients (Ostir, Markides, Black, & Goodwin, 2000). In addition, a robust negative relation has been found between positive affect and morbidity (Pressman & Cohen, 2005).

Yet, in considering directional influences, the relation between well-being and health is undoubtedly complex. Being healthy can make people happy and being happy can bolster health. Fortunately, experimental, ambulatory, and longitudinal studies that focus on the possible impact of well-being on objective health outcomes can help disentangle causal influences. Specifically, experimental studies determine the effects of induced positive and negative transient moods and emotions on concurrent objective health outcomes. Ambulatory studies use experience sampling methodology across several days or weeks to examine how changes in daily mood relate to health outcomes. Longitudinal studies explore whether previous levels of happiness predict future levels of physical health across more extended periods. However, researchers have not, to our knowledge, explored the nature of the well-being–health link, beyond simply reporting its

magnitude. To this end, the primary goal of our meta-analysis is to synthesize the literature that investigates the possible effects of well-being on objective health status, with a focus on the moderators of this link.

Defining and Measuring Well-Being and Health

Defining well-being. The independent and predictor variables considered in this meta-analysis comprise what most researchers call "subjective well-being" (SWB, the technical term for "happiness" or simply "well-being") or, alternatively, "life satisfaction" or "positive affect." Happiness, life satisfaction, and positive affect are considered separable yet highly correlated constructs, and typically yield a single higher-order factor (e.g., Sheldon & Lyubomirsky, 2006; Stones & Kozma, 1980). Although these constructs are fairly heterogeneous, they are strongly related, both theoretically and empirically; thus, high ratings on life satisfaction scales and positive affect scales both indicate high well-being. For example, Watson and Clark (1994) document high correlations between average daily mood reports of positive and negative affect and trait versions of these scales (*r*s from .48 to .66). Furthermore, several studies have reported that the intercorrelations typically found between various measures of trait SWB are quite large (*r*s from .44 to .72; Kim, 1998; Lyubomirsky & Lepper, 1999; Suhail & Chaudhry, 2004).

Accordingly, SWB is employed here as an overarching term that comprises several related phenomena, including emotional responses (i.e., the experience of frequent positive and infrequent negative moods and emotions) and global judgments of life satisfaction (Diener, 2000; Diener, Suh, Lucas, & Smith, 1999). Furthermore, because we focus in this meta-analysis on the effects of well-being on objective health outcomes, we use the term *well-being* for positive psychological constructs that are measured (e.g., positive affect, life satisfaction, optimism) or manipulated (e.g., as part of a positive emotion induction). The term well-being is

then contrasted to the term *ill-being*, which we use to refer to negative psychological constructs that are measured (e.g., negative moods, stress, anger, depression) or manipulated (e.g., as part of a negative emotion induction). The research cited used a variety of measures of the different components of well-being.

Measuring well-being. Given this article's focus on well-being's impact on health, we have included studies that use measures of both trait levels of well-being (in the longitudinal research) and transient (state level) emotions and moods (in the ambulatory and experimental research). Measures of these constructs employ self-report methods, which appropriately allow the final judge of happiness and satisfaction to be "whoever lives inside a person's skin" (Myers & Diener, 1995, p. 11; see also Diener, 1994). However, the fact that self-reports are subjective does not mean that they are unrelated to relatively more "objective" variables (for a review, see Diener, 1994). For example, research reveals significant convergence of self-reported well-being with informant reports (e.g., Lyubomirsky & Lepper, 1999; Sandvik, Diener, & Seidlitz, 1993), recall of positive and negative events (e.g., Seidlitz, Wyer, & Diener, 1997), unobtrusive observations of nonverbal (i.e., smiling) expressions (e.g., Harker & Keltner, 2001), and physiological responses (e.g., Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005).

Typically, longitudinal and ambulatory studies assess well-being via self-report. The longitudinal studies described here included several different measures of global well-being, including, but not limited to, the Satisfaction With Life Scale (SWLS; Diener, Emmons, Larsen, & Griffin, 1985), the Memorial University of Newfoundland Scale of Happiness (Kozma & Stones, 1980), and various single-item scales (e.g., "How satisfied are you with your life?"). We also included longitudinal studies that employed more indirect indicators of well-being, such as measures of optimism (e.g., the Life Orientation Test [LOT]; Scheier & Carver, 1985). Optimism

has been found to be related to positive affectivity and thus serves as a defensible proxy for well-being (Lucas, Diener, & Suh, 1996). For example, Lyubomirsky and her colleagues (2005) found a very high correlation (r = .60) between optimism and happiness. However, some specific non-hedonic quality-of-life (QOL) measures were not included, as these measures focus primarily on physical symptoms, health problems, and medical issues (e.g., QLQ-30, Aaronson et al., 1993; FACT, Cella et al., 1993; see Gotay, 2006, for a review of studies linking these types of QOL to survival) and not on emotional responses or global judgments of life satisfaction.

The ambulatory (and some experimental) studies described here typically included self-reported measures of emotions and moods, such as the Affect Balance Scale (Bradburn, 1969), variants of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), and the Profile of Mood States (Curran, Andrykowski, & Studts, 1995). Such measures are appropriate to use in ambulatory studies, whose purpose is to track small changes in affect over time; and they are the only ones available to researchers interested in measuring affect in experimental studies, as no investigations to date, to our knowledge, have tested the effects of induced long-term happiness on health. Although transient mood is not equivalent to long-term happiness, it has notably been shown to be the very *hallmark*, or basic constituent, of happiness. Indeed, happiness has been defined as the experience of frequent positive emotions (Diener, Sandvik, & Pavot, 1991; Lyubomirsky et al., 2005). Hence, we expected the physical health outcomes of short-term positive moods to be parallel to those for the concomitants of global, long-term well-being (see Lyubomirsky et al., 2005, for a similar approach).

In the experimental research reported here, a variety of manipulations were used to induce transient emotions, including films, imagery, music, and the Velten induction task (Velten, 1968), among others (see Coan & Allen, 2007, for an overview). However, many

researchers induce global positive affect or positive emotions and do not discriminate among specific emotions (e.g., happiness, elation, or arousal) or moods. Notably, positive moods are not the opposite of negative moods. These two types of affect show moderate inverse relations across individuals, sometimes correlate with different variables, and appear to be rooted in distinct biological systems (Bradburn & Caplovitz, 1965; Cacioppo, Gardner, & Berntson, l999; Diener & Emmons, 1984; Diener, Smith, & Fujita, 1995).

Defining health. Health is a multi-dimensional construct that evades simple classification (Cacioppo & Berntson, 2007; Gochman, 1997). It can be conceptualized as two distinct categories – as a state or as a process (Carver, 2007; Kaplan, 1994, 2003) – but is usually defined as a state, extending from the traditional biomedical model. Accordingly, health is characterized by a lack of illness or disease (e.g., lack of fever, vomiting, chronic conditions, disability), maintaining normal function (ability to function well with minimal medical care), and positive self-assessments of health at the time of measurement (Breslow, 1972; Idler & Kasl, 1991). Health can be operationalized in a variety of ways, ranging from subjective single-item judgments of overall health to specific physiological measures such as concentrations of hormones and substances in the bloodstream.

In contrast, several theorists have suggested that health be defined as a lifelong process (Aldwin, Spiro, Levenson, & Cupertino, 2001; Baltes, Staudinger, & Lindenberger, 1999; Clipp, Pavalko, & Elder, 1992; Schultz & Heckhausen, 1996); that is, health involves regulation over time, such that the autonomic, neuroendocrine, and immune systems work together to maintain balance within the body (Cacioppo & Berntson, 2007). If this balance is threatened for an extended period, these systems can break down and lead to physical decline (McEwen, 1998;

McEwen & Stellar, 1993). In this meta-analysis, we define health according to this second, more holistic framework.

Measuring health. If health is considered as a process, a measurement at any single assessment reflects the individual's state within this broader process. For healthy individuals, maintaining a state of normal functioning and preventing disease are important goals, whereas for individuals with chronic illnesses, maintaining well-being and controlling symptoms are important goals (Westmas, Gil-Rivas, & Cohen Silver, 2007). Accordingly, how health is operationalized and measured depends on the person's position on the continuum between optimal functioning and clinical illness. Further, measures of health depend on whether researchers are interested in short-term markers of system activity (e.g., heart rate, blood pressure, cortisol levels) or long-term markers of overall health (e.g., cardiovascular fitness, survival). One goal of this meta-analysis is to integrate multiple levels of health; therefore, we included studies assessing both markers of physiological functioning and markers of overall functioning. Specifically, at the molecular level, health is marked by normal responses to stress and rapid recovery to baseline levels (Kemeny, 2007). At the molar level, for healthy individuals, well-being should maintain or increase normal functioning, and decrease risk of illness (such as colds and infections) and early mortality. For individuals with a chronic condition, well-being should decrease symptoms of illness (e.g., allergic reactions, asthmatic symptoms) and increase survival (longer life, despite the presence of one or more morbidities).

Finally, although health can be construed as a complex construct with multiple physical, cognitive, and affective dimensions (Fisher, 1995; Ryff & Singer, 1998; Ware, 1987), in the present analysis, we opted to operationalize health in terms of physiological measures and relatively objective physical outcomes rather than using subjective self-reports. This practice has

several advantages. Shared method variance between self-reported measures of health and well-being may be responsible for a strong association between these two constructs (Lyubomirsky et al., 2005; Pressman & Cohen, 2005). Additionally, objective measures are valuable from a public health perspective in which optimal health is achieved by extending life expectancy while compressing morbidity to the final years of life (Fries, 1990; Kaplan 2003).

Markers of normal functioning. In the literature on health and stress, several markers of hormonal responses and immune functioning are used to assess the effects of stress at the molecular level (Rabin, 1999; Segerstrom & Miller, 2004). For example, in the immune system, markers of normal immune responses include increases in lymphocytes (e.g., t cell counts on markers such as D4, CD8+, and CD16+), leukocytes (such as natural killer cells, macrophages, and interleukin cells), and immunoglobin (such as sIgA) after being exposed to an invading substance (Dayyani et al., 2004; Dreher, 1995; Linnemeyer, 1993; Perera, Sabin, Nelson, & Lowe, 1998; Rabin, 1999; Saleh et al., 1995). In the autonomic nervous system (ANS), stress activates the system, evidenced by the release of specific hormones (e.g., cortisol, adrenaline) and increases in heart rate, blood pressure, finger temperature, and skin conductance (Cacioppo & Tassinary, 1990; Pickering, 1999). This response should then taper back to baseline levels. Non-normal responses may represent some sort of dysregulation within the system (Kemeny, 2007).

At the molar level, health depends on a person's status. For healthy individuals, measures of health reflect normal or optimal functioning. For example, cardiovascular strength offers a marker of the organism's level of fitness, and is commonly assessed by increased power output and flow rates (Koehler, 1996). General health indicates overall functioning, and can be measured by indicators such as a healthy cholesterol ratio and weight (Kivimäki et al., 2005;

Pollard & Schwartz, 2003). Finally, longevity is a reliable and objective health outcome that is arguably the endpoint in a long causal chain of interrelated events. Longevity is determined by length of life in years, and is generally verified from vital records or familial report (e.g., Brown, Butow, Culjak, Coates, & Dunn, 2000; Friedman et al., 1993; Wingard, Berkman, & Brand, 1994).

For individuals with one or more chronic conditions, healthy functioning is marked by symptom control. For example, allergic reactions and asthma attacks indicate functional decline and dysregulation. Health is evident when a normal level of functioning can be maintained. Allergic reactions are typically measured by skin tests, in which allergens are introduced percutaneously and flare or wheel sizes are measured (Zachariae, Jorgensen, Egekvist, & Bjerring, 2001). Decreased respiratory functioning may signal respiratory failure (Quanjer et al., 1997; Quanjer, Lebowitz, Gregg, Miller, & Pedersen, 1997; Rosenow, 2005) and poor symptom control. Respiratory system functioning is commonly assessed by expiratory volume (a measure of how much air a person exhales during a forced breath), peak expiratory volume (the maximal amount of flow expelled in a forced breath), and oxygen saturation (the percentage of oxygen the red cells carry). Finally, terminal illnesses (e.g., cancer, HIV) progress through a series of stages and are marked by whether the individual declines (a lack of symptom control), maintains a stable level of functioning, or evidences some degree of recovery. Survival indicates how long a person stays alive despite having one or more chronic illness (Pressman & Cohen, 2005).

Although health investigators typically study a single illness or physiological marker of normal functioning, it is important to consider bodily systems as a whole, despite the complexity of the relations involved. For example, if well-being is indeed beneficial, then it should benefit health across systems and levels, regardless of the specific mechanisms and pathways involved

(Rabin, Kusnecov, Shurin, Zhou, & Rasnick, 1994). Thus, the present meta-analysis empirically examines the potential benefits of well-being across multiple markers of normal functioning, including (a) specific, short-term outcomes, (b) general, long-term markers of physical well-being and functioning, and (c) symptom control during stages of chronic conditions. Although limited empirical support exists in human studies on the complete process connecting short-term and long-term health outcomes (Keller, Shiflett, Schleifer, & Bartlett, 1994), physiological responses may indeed extend to clinical disease outcomes over time (Keller et al., 1994; Kemeny, 2007), and by combining the two in a single analysis, we can examine the macro-micro level linkages as a whole (Mroczek, Almeida, Spiro, & Pafford, 2006; Nesselroade, 1988, 1991).

Understanding the Potential Impact of Well-Being on Health

Linking emotion and health. Much of the theoretical and empirical work linking psychological and physical well-being comes from studies on stress and health. Comprehensive models relating stress to health outcomes have been elaborated (e.g. Carver, 2007; Keller et al., 1994; Rabin, 1999). For example, cortisol is often used as a marker of stress. An increase in cortisol is an adaptive response to a stressor, but when prolonged over time, it can negatively impact immune system functioning (Cohen & Williamson, 1991; Dickerson & Kemeny, 2004; Herbert & Cohen, 1993; Segerstrom & Miller, 2004). Further, multiple studies and reviews indicate that stress can negatively affect the cardiovascular system (e.g., Krantz & McCeney, 2002; Kubzansky & Kawachi, 2000), neuroendocrine activity, and negative disease outcomes (Carver, 2007).

In a very basic model, when a physical or emotional stressor is encountered, distress occurs (Keller et al., 1994). This may, in turn, activate the central nervous system, triggering a fight-or-flight response, characterized by physiological changes such as increased blood sugar

levels, heart rate, and blood pressure, and the release of stress hormones (such as cortisol and epinephrine; Cannon, 1932; Selye, 1956). This response may directly and indirectly influence immune functioning (Glaser & Kiecolt-Glaser, 1994; Keller et al., 1994). Immune response dysregulation in turn may continue to activate the central nervous system, leading to chronic strain and increased susceptibility to illness (Bowen, 2001; Dickerson & Kemeny, 2004). Thus, the cardiovascular, neuroendocrine, and immune systems work together and influence one another (Cacioppo & Berntson, 2007). Some evidence for this complete model comes from animal studies with mice and non-human primates (Laudenslager & Fleshner, 1994; Maynahan et al., 1994), which suggest that such a process of dysregulation over time can potentially lead to clinical illness. Unfortunately, empirical studies that test direct and indirect pathways are mostly lacking in human research (Keller et al., 1994), although the few studies that exist offer some support (see Cohen, 1994 and Keller et al., 1994, for reviews). We note that this is a simplistic description, and the system is undoubtedly much more complex (Friedman, 2007).

Although elements within the cardiovascular, endocrine, and immunological systems play various roles within the stress-disease process, if each system is considered as a whole, then multiple markers can be combined in an informative manner. For example, participants are often subjected to a stressor, and heart rate, blood pressure, saliva cortisol, and plasma concentrations of epinephrine are measured to determine the extent of their reactivity (e.g., Bachen et al., 1992; Kiecolt-Glaser, Malarkey, Cacioppo, & Glaser, 1994; Manuck, Cohen, Rabin, Muldoon, & Bachen, 1991). Similarly, multiple markers of immune response are often measured in response to stress. Segerstrom and Miller (2004) offer an excellent overview of the immune system, with evidence on how stress links to natural and specific immune responses. Although cost and participant constraints limit what physiological aspects a researcher considers (Keller et al,

1994), multiple markers may be telling a parallel story, which can be informative on how the body functions as a whole. Hence, in the present meta-analysis, we considered health in this more holistic fashion, combining multiple markers of function and dysregulation within each main system in the body and in overall functioning.

How well-being may influence health. If stress and negative emotions potentially foster detrimental health outcomes, can positive emotions and moods foster improved health? That is, whereas stress activates the sympathetic nervous system, an opposite reaction may decrease sympathetic system activity (Rabin et al., 1994) and promote optimal functioning. Empirical support for this notion is evident in personality research, which has demonstrated that negative traits such as neuroticism and hostility relate to increased mortality risk and poor health outcomes (e.g., Booth-Kewley & Friedman, 1987; Smith, 2006; Smith, Glazer, Ruiz, & Gallo, 2004; Smith & Williams, 1992; Suls & Bunde, 2005; Watson & Pennebaker, 1989), whereas positive traits such optimism, extraversion, agreeableness, and conscientiousness relate to decreased mortality risk and better health (Friedman et al., 1993; Hampson, Goldberg, Vogt, & Dubanoski, 2006). Because of the strong correlation between personality traits and SWB, similar mechanisms may characterize the relations between well-being and health (Pressman & Cohen, 2005; Ryff & Singer, 1998).

Specifically, Pressman and Cohen (2005) detailed two models linking positive affect and disease. In the *direct effects model*, positive affect may directly affect health practices, decrease autonomic nervous system activity, regulate the release of stress hormones, influence the opioid system and immune responses, and affect social networks; these in turn impact health and disease outcomes. In the *stress-buffering model*, positive affect may ameliorate the effects of stressful events by increasing resiliency and enhancing coping responses. Accordingly, well-

being may affect health by enhancing short-term responses (e.g., increasing immune response and pain tolerance) and long-term functioning (e.g., better cardiovascular fitness and longer life) or by buffering the effects of short term stressors (marked by high-level stress responses and heart reactivity) and long-term illness (e.g., slowing disease progression and increasing survival). Most likely, a combination of these two mechanisms operate, depending on the individual and the situation (Friedman, 2007). In turn, health status influences well-being and quality of life.

Possible Moderators of the Health-SWB Relation

Based on the theoretical literature and empirical studies outlined above, a primary goal of this meta-analysis was not only to establish *whether* well-being influences health outcomes, but also *under what conditions* well-being may exert its salutary effects. Accordingly, in addition to the overall relation of well-being to health, we examined several potential moderators of these relations.

Categories of health outcomes. First, we combined short-term and long-term outcomes using both general and specific markers of health. Specifically, at a broad level, we identified three types of health outcomes according to how health can be conceptualized, based on both length of follow-up and initial health status: short-term outcomes, long term outcomes, and disease and symptom control for chronically ill samples. Across these three categories, we examined the effects of 12 groups of health outcomes. Specifically, short term-outcomes included (a) immune system response, (b) cardiovascular reactivity, (c) endocrine system functioning and response, (d) physiological response, and (e) pain tolerance. Long-term outcomes included (a) general health outcomes, (b) cardiovascular functioning, (c) respiratory functioning, and (d) longevity. Finally, disease and symptom control included (a) measures of respiratory control (in conditions such as allergies and asthma), (b) disease progression, and (c)

survival despite having one or more terminal conditions. In turn, each of these 12 health outcomes was comprised of various specific markers.

Health outcome as a moderator. Our moderator predictions were based on theories from the stress and health literature (e.g., Rabin, 1999; Segerstrom & Miller, 2004) and the pathway models enumerated by Pressman and Cohen (2005), as well as other relevant work. We expected well-being to relate positively to *and increase* health-related functioning (i.e., longevity, survival, and pain tolerance), improve autonomic nervous system response (i.e., cardiovascular and respiratory functioning), and improve immune system functioning. In contrast, we expected well-being to relate negatively to *and decrease* cardiovascular reactivity (e.g., heart rate, blood pressure), endocrine response (e.g. measures of cortisol), physiological response (e.g., finger temperature), symptom response in chronic conditions, and disease progression. These negative relations were predicted because well-being is expected to buffer the system from negative outcomes. Furthermore, we predicted that well-being would affect short-term outcomes more than long-term outcomes. When stress occurs, the autonomic nervous system is immediately activated; if well-being interrupts this response (either through buffering stress or engendering a more rapid recovery), then its effects will be evident fairly quickly (Rabin et al., 1994). For long-term outcomes, a vast array of variables can moderate and intercede in this relation (Hall, Anderson, & O'Grady, 1994), ranging from psychosocial factors such as social support, health habits, and natural physiological changes that occur with age, to measurement unreliability; hence, any effects on long-term outcomes will be weaker, although still significant in a practical sense (Rosenthal, 1991; Smith, 2006).

Baseline health as a moderator. For healthy individuals, bodily systems naturally fluctuate with transient stress; thus, seemingly abnormal levels of one marker may actually be a

normal response to fluctuation in another system (Rabin et al., 1994). In contrast, for unhealthy individuals, the system as a whole is dysregulated, and abnormal values indicate further stress on the system, adding to the overall allostatic load (McEwen, 1998). Due to its differential role in defining and understand health outcomes, baseline health is important to consider. We expected well-being to have a greater effect for unhealthy samples than for healthy samples.

Operationalizations of well-being. Although study methodology typically dictates the operational definition of well-being in any particular design (i.e., transient emotions are typically measured in ambulatory studies and manipulated in experimental research, whereas trait levels of well-being are typically measured in longitudinal data), we expected the relation between well-being and objective health to vary as a function of state versus trait operationalizations. Specifically, we hypothesized that short-term health outcomes would be more strongly associated with state manipulations of well-being and that long-term outcomes would be more strongly associated with trait measures of well-being.

When stress occurs and the ANS is activated (releasing cortisol, increasing heart rate and blood pressure, etc.), transient positive emotions can have relatively immediate effects – for example, potentially moderating the stress response or enabling a quicker return to baseline, indirectly protecting other systems (such as the immune system) from the stressor. Thus, much like a stressor provokes a short-term response from the ANS, positive emotions may have a short-term counteracting influence on the stressor. In contrast, long-term health outcomes represent a process of accumulated regulation or dysregulation over time. Thus, with respect to long-term outcomes, well-being that is stable over time (i.e., trait well-being) can aid individuals to maintain stability, both internally and externally, thus avoiding system dysregulation and decreasing susceptibility to illness.

Age as a moderator. In both humans and animals, the immune system changes with advancing age (Bilder, 1975; Makinodan et al., 1991; Weksler & Hausman, 1982). Specifically a general decline in immune response may occur with age, which increases susceptibility to infections and disease (see Solomon & Benton, 1994, for a review). Notably, some factors moderate this decline. Studies with elderly individuals demonstrate that successful agers have stronger indices of immune function than normal and declining elderly individuals (Solomon et al., 1988; Thomas, Goodwin, & Goodwin, 1985), suggesting that factors other than age itself are important. Well-being may be one such factor (Kiecolt-Glaser et al., 1994). Thus, sample age is important to consider as a factor in the well-being and health relation. When there is a greater possibility of health decline, changes in health outcomes will be more evident (Solomon & Benton, 1994; Rowe & Kahn, 1987); therefore, we expected well-being to have stronger effects on health outcomes for older samples.

Gender as a moderator. Because males and females differ physiologically, gender differences may impact the role that well-being plays. For example, females typically live longer than males; yet males who reach older age are often both physically healthier than females and suffer from fewer psychological problems, such as anxiety and depression (Guralnik & Kaplan, 1989; Roos & Havens, 1991; Strawbridge, Cohen, Shema, & Kaplan, 1996). Thus, if those males who experience longevity also report less negative affect and more happiness, then it is possible that well-being plays a role in the observed gender differences in health outcomes for elderly samples. We predicted that well-being will be more important for males, acting as a buffer against decline.

Previous Research Syntheses Examining the Health-SWB Relation

To our knowledge, only two literature reviews to date have scrutinized the link between well-being and health; both were published in 2005. The first was a meta-analytic review of the relation of happiness and positive affect to a variety of indicators of "success," including health (Lyubomirsky et al., 2005); and the second was a qualitative review that focused on the link between positive affect (PA) and health (Pressman & Cohen, 2005).

The Lyubomirsky et al. review used three classes of evidence – cross-sectional, longitudinal, and experimental – to examine the extent to which various indicators of well-being were associated with successful outcomes (e.g., income and marriage), and with behaviors and attributes paralleling success (e.g., prosocial behavior, sociability, and creativity). However, computation of effect sizes between SWB and health outcomes constituted only a portion of the analyses conducted for this review. Furthermore, Lyubomirsky and her colleagues' analyses diverged from those of the current study in two critical ways. First, they did not examine any moderators of the well-being–health relation (nor of any of the well-being–success links they described). Nor did they distinguish between the different types of health outcomes (e.g., immune functioning vs. cardiovascular reactivity vs. survival) or whether those outcomes were short-term or long-term. Their goal was simply to test whether a positive association was present between well-being and success. Second, unlike the present study, their analyses of health outcomes did not separate objective indicators and subjective reports of health.

By contrast, the Pressman and Cohen (2005) review was a qualitative synthesis of the PA-health literature. The authors concluded that positive affect was related to many objective health outcomes, including lower morbidity, decreased symptoms, and diminished reported pain, among others. Whereas they provide great detail about the association between PA and health,

our review differs from theirs in three important ways. First, we expanded our analyses to include all positive psychological constructs in an attempt to determine how well-being in general (and not only positive affect) influences objective health outcomes. We believe this to be important, as many studies in the area of health psychology assess the cognitive component of well-being and would have otherwise been excluded. Second, we aimed to examine the differential effects of positive and negative psychological constructs on health outcomes. To this end, we included studies that simultaneously measured or manipulated both positive and negative psychological constructs. Although the present meta-analysis does not provide a complete review of the effects of ill-being on health, it does afford an opportunity to compare the effect sizes for the two constructs of well-being and ill-being. Such a comparison may illuminate unique relations and pathways between health and these two constructs, as well as the mechanisms and moderators underlying them. Third, in contrast to Pressman and Cohen's qualitative review, our meta-analytic review is able to estimate the size of the effect between well-being and objective health, as well as to test quantitatively for moderators of the well-being–health link.

Objectives of the Present Meta-Analysis

Given that numerous studies and reviews have considered the effect of negative emotions (or more generally *ill-being*) on compromised health functioning and increased illness (e.g., Friedman & Booth-Kewley, 1987; Herbert & Cohen, 1993; Segerstrom & Miller, 2004), the current meta-analysis focused on the effect of positive psychological constructs (or more generally *well-being*) on objective health outcomes. Furthermore, although several authors have recently suggested that health psychologists need to move beyond the medical model and consider health more broadly (e.g., Grzywacz & Keyes, 2004; Kaplan, 2003; Ryff & Singer,

1998; Smith & Spiro, 2002), we argue that any review of the literature must separately consider specific components of health. Thus, for the purpose of this research synthesis, we concentrated on measures of objective health outcomes using traditional biomedical markers. To these ends, our search strategy included seeking out literature examining positive emotions and positive traits as predictors of objective measures of physical and physiological health outcomes. Although the focus of the meta-analysis was on positive psychological constructs, if an included study reported the relation between well-being and health *as well as* the relation between ill-being and health, the size of the effect between ill-being and health was computed separately. This procedure allowed us to compare the average ill-being–health effect size with the average well-being–health effect size for those studies that examined both linkages. Such studies were expected to have similar effect sizes to those that examined only one of the links.

Because our meta-analysis was specifically concerned with assessing the potential impact of well-being on objective health, only studies that used experimental, ambulatory, and longitudinal methods were included. The copious cross-sectional literature has been reviewed in other sources (Lyubomirsky et al., 2005; Pressman & Cohen, 2005), yet some of the studies included in those reviews suffer from multiple limitations. First, correlational and cross-sectional studies provide little information regarding directionality, as these methodologies cannot test the possible impact of well-being on health. Second, due to their common reliance on self-reports of both well-being and health, these studies generally contain too much shared method variance to determine whether well-being has any tangible impact on important health outcomes. In contrast, particular sections of our Results pay special attention to studies that use experimental methodology, as true experiments allow us to estimate the causal effects of well-being on objective physical and physiological outcomes.

In sum, the primary aims of this meta-analysis were to (a) determine the average effect size between well-being and objective health; (b) compare this effect size to the average effect size for ill-being and health; (c) establish which particular health outcomes are most strongly associated with well-being; and (d) explore possible sample-specific moderators of the well-being–health relation.

Method

Literature Search Procedures

The present meta-analysis used several search techniques to retrieve all applicable studies for inclusion. Our primary search procedure extended work from the two recent reviews of health outcomes associated with positive affect and SWB (Lyubomirsky et al., 2005; Pressman & Cohen, 2005). First, each empirical article considered in these two reviews that addressed well-being and health was located; the total number of unique empirical articles examined by Lyubomirsky et al. or Pressman and Cohen was 240. Second, all literature reviews and theoretical articles cited in the two 2005 reviews was located, resulting in an additional 30 papers (for a total of 270) to be used for additional search techniques (i.e., forward and backward searching).

Each of these 270 titles was then submitted to the PsycINFO and Web of Science online databases, using both forward and backward search procedures to identify other potentially relevant articles. Specifically, the reference section of each of the 270 articles was examined for germane titles and abstracts, identifying previous studies relevant to well-being and health that were not included in the two 2005 reviews (backward search); and more recent articles were identified that cited the original 270 studies (forward search). These search procedures identified an additional 90 empirical studies to be examined for inclusion into the meta-analysis; thus, the

number of empirical articles cited in these reviews or located as a result of searches using these reviews was 330.

As a final check, a database search of PsycINFO and Web of Science was conducted, combining five terms reflecting well-being (positive affect, subjective well-being, happiness, life satisfaction, and hedonic quality of life measures) and three terms reflecting health (physical health, physical well-being, and physical functioning). Only four additional studies were identified in this final search, suggesting that we successfully located most applicable published studies in the field. All potentially relevant studies published or posted through June 1, 2006 were evaluated for inclusion. Thus, the three search strategies yielded an original set of 334 empirical articles that were examined using our inclusion and exclusion criteria.

Inclusion and Exclusion Criteria for Studies

Included studies. Potentially relevant studies were coded and included in the meta-analysis only if they met all six established criteria. To be included, a study had to (a) be written in English; (b) be an empirical study (rather than a literature review, meta-analysis, or theoretical paper); (c) include, as an independent variable, a subjective measure of well-being (e.g., positive affect, life satisfaction, happiness, optimism) or a positive mood or emotion manipulation (e.g., humorous films, imagining pleasant circumstances, etc.); (d) include, as the dependent variable, an objective measure of physical health (e.g., mortality/survival, respiratory functioning, endocrine and immune system functioning, pain tolerance, physical functioning) or illness (e.g., disease progression, heart disease, cancer, HIV symptoms); (e) state the specific sample group (e.g., cancer patients, healthy students, asthmatics); and (f) use experimental, ambulatory, and longitudinal methodology.[1] For studies that met these criteria, the effect size between well-being and health had to be either provided or computable from summary tables, descriptive statistics,

or inferential statistics (*t*-statistics, *F*-ratios, odds-ratios, or Chi-square statistics). For studies that only reported multiple regression or probit analyses, *r* equivalent effect sizes (see Rosenthal & Rubin, 2003) were computed from exact *p*-values (if available) or conservative *p* cut-offs (e.g. .01, .05).

Excluded studies. Because we were interested in the effects of well-being on health, studies were excluded if *only* ill-being constructs (e.g., depression, hostility, negative affect, anger) were measured or manipulated. Studies were also excluded if they (a) assessed only the contemporaneous correlation between well-being and health; (b) examined only cross-sectional mean differences between healthy and unhealthy samples; (c) measured health using only a self-report measure; or (d) examined the impact of physical health on well-being (rather than well-being on health).

Altogether, 120 of the 240 unique empirical studies analyzed by Lyubomirsky et al. (2005) and Pressman and Cohen (2005) were excluded based on the above criteria. A majority of these exclusions were due to the outcome being self-reported health or a health coping variable, the methodology used in the study being cross-sectional or correlational, or the study being focused on a proxy of health or a health behavior (e.g., physical exercise). Of the 94 additional empirical studies identified from our three primary search techniques, 64 studies were excluded for many of the same reasons above. As a result, 150 studies were included and coded in this meta-analysis, with 9 of these studies having been uniquely considered by Lyubomirsky et al. (2005), 92 by Pressman and Cohen (2005), and 19 by both studies.

Computing Effect Sizes

Effect sizes. The specific type of effect size used in this meta-analysis was the correlation coefficient or *r* index. An *r* effect size was computed for all relations between well-being and

health for each included study using the computer program *Comprehensive Meta-Analysis 2.0* (Borenstein, Hedges, Higgins, & Rothstein, 2005). If a study induced both positive and negative affect to compare post-induction health outcomes with participants' baseline health assessments, then effect sizes for positive affect on health and negative affect on health were computed separately. Conversely, if a study compared difference scores of a positive mood manipulation group with a negative mood manipulation group for a specified health outcome, then a single effect size was computed that estimates the size of the differential impact on health for positive moods compared to negative moods. Table 1 presents all studies included in the meta-analysis along with three important aggregated effect sizes. We expected well-being to relate positively to *and increase* health-related functioning, cardiovascular functioning, and immune system response. In contrast, we expected well-being to relate negatively to *and decrease* cardiovascular reactivity, endocrine response, physiological response, symptom response in chronic conditions, and disease progression. We anticipated the opposite for the effects of ill-being on these health outcomes. Thus, in Table 1 (and when effect sizes were entered into *Comprehensive Meta-Analysis 2.0;* Borenstein et al., 2005), positive values indicate that the relation was in the predicted direction and negative values indicate that the relation was opposite to our predictions.

Unit of analysis. Our primary unit of analysis was the independent sample(s) within each study. Every independent sample was included and coded separately within each investigation. For example, many studies reported descriptive and inferential statistics separately for males vs. females, unhealthy (e.g., asthmatics) vs. healthy control groups, or respondents experiencing different levels of the independent variable (e.g., high vs. low positive affect). Further, individual effect sizes were calculated within each independent sample for all (measured or manipulated) well-being constructs and for all measured health outcomes. For example, if a study manipulated

positive emotions to determine their effects on heart rate and blood pressure, we computed two separate effect sizes.

Then, for all estimates of central tendency and all tests of homogeneity, these multiple effect sizes were aggregated to derive a single effect size for each independent sample; consequently, each independent sample contributed only one *r* effect size for these analyses. However, coding for all possible well-being–health relations had implications for the types of moderator questions that this meta-analysis could address. First, for all of the sample-specific moderators we coded (see below), average effect sizes could be compared across all levels of the moderator – for example, effect sizes for healthy samples could be compared to effect sizes for unhealthy samples. Second, when examining the aggregate effect sizes by different health outcomes (both general and specific), because independent samples typically measured multiple health outcomes, we could not statistically compare the effect sizes across different health outcomes. For example, we could not test whether the effect of well-being on immune functioning was statistically stronger or weaker than its effect on cardiovascular functioning, because the effect sizes we used (e.g., those generated from multiple health outcomes measured on the same sample) were not all independent from each other. As a result, average effect sizes were not comparable across different health outcomes, but were instead compared *within* each health outcome to determine whether the relation between well-being and health was significantly stronger than zero.

Coding Sample Moderators

All independent samples that met the inclusion criteria were coded for possible moderators. Specifically, two classes of moderators were coded: (a) variable characteristics

(health and/or illness; state or trait well-being); and (b) sample characteristics (health status, age, gender).

Objective health outcome characteristics. Because the third goal of our meta-analysis was to compare effect sizes for the link between well-being and health across different health outcomes, we coded three general categories of health and 12 specific health outcome variables. First, for short-term outcomes (health outcomes measured at the molecular level), we coded the following: immune system response (e.g., sIgA concentration, NKCA), endocrine system response (e.g., cortisol, epinephrine, norepinephrine), cardiovascular system reactivity (e.g., blood pressure, heart rate), physiological response (e.g., finger temperature, skin conductance), and pain tolerance (e.g., time in cold pressure task). Second, for long-term outcomes (health outcomes measured at the molar level, for normal functioning) we coded the following: general health (e.g., accumulated mucus weight during infections, cholesterol), cardiovascular system functioning (e.g., high and low frequency power, ischemic episodes), respiratory functioning (e.g., inspiration volume, inspiratory volume), and longevity (mortality for participants without a chronic condition). Third, for chronic conditions (health outcomes measured at the molar level, for those with chronic illness), we coded respiratory diseases/conditions (e.g., flare reactions, wheel reactions, forced expiratory volume, bronchial responsiveness), disease progression (e.g., complete recovery from disease, viral load), and survival (staying alive despite having a chronic condition).

State and trait measures of well-being. We recorded the exact SWB construct measured or manipulated in each study. Each of these constructs was then coded as representing either a state variable (momentary positive and negative affect, induced hope, relaxation, etc.) or a trait

variable (global life satisfaction, happiness, optimism, and trait levels of positive and negative affect) of well-being or ill-being.

Sample characteristics. We predicted that the effects of well-being on objective health outcomes would be moderated by characteristics of the independent samples. Two sample-level categorical moderators were coded: (a) method of study (experiment, ambulatory, or longitudinal) and (b) health status of the sample at baseline (healthy or unhealthy). In addition, we recorded two continuous sample-level moderators: (a) mean participant age at baseline and (b) gender composition (percent male respondents). Coding of the sample characteristics was based on information gleaned from the Method section of each article.

Data Analysis: Fixed vs. Random Effects

Both fixed and random effects methods for meta-analysis have advantages. The fixed effects model provides a more precise and reliable estimate of the population effect size (Cooper, 1998), whereas the random effects model allows for relatively more generalizable conclusions. Random effects models also specify the amount of variance accounted for by between-studies differences and variance accounted for by within-study differences. Because each has advantages and disadvantages, aggregate *r* effect sizes were computed with *Comprehensive Meta-Analysis 2.0* (Borenstein et al., 2005) using both fixed and random models. When possible, we focused on the results from the random effect models. However, some moderator tests (e.g., meta-regressions) can only be estimated using fixed effects models. In these cases, all null hypotheses, fixed effects models, and post hoc comparisons followed steps outlined by Hedges (1994). For both models, homogeneity tests were used to determine whether variance in the effect sizes was explained by the proposed moderators. When the *r* effect sizes were aggregated using a fixed effects model, measures of central tendency were calculated by averaging weighted *r* effect

sizes (inverse variance weights) across all independent samples. When categorical groups were independent, and if a categorical moderator explained significant variance in the effect sizes (i.e., $p < .05$ for Q_{BET}), then post hoc contrasts were performed to determine which groups were statistically different. For continuous moderators, meta-regression analyses were used to determine whether variation in the effect sizes was explained by the moderator.

Results

Description of the Literature Included

Publication statistics. Our search techniques identified a total of 150 studies that met the established inclusion criteria. From these studies, 212 independent samples were identified, from 17 (mostly Western) nations, which measured or manipulated a well-being construct and measured a physical health outcome. From these 212 samples, 439 distinct effect sizes were computed between well-being and physical health; an additional 310 effect sizes were computed between ill-being and health from the 79 studies that measured both well-being and ill-being. The number of independent samples coded per study ranged from 1 to 6, with 107 studies (71.3%) using a single independent sample, and 33 studies (22.0%) reporting two independent samples (see Table 1 for a detailed description of each study). The number of effect sizes of the link between well-being and physical health per study ranged from 1 to 20 ($M = 2.93$, $SD = 2.44$), with 70.7% of the studies reporting 3 or fewer effect sizes. The typical study surveyed 50 respondents ($Mdn = 50.50$); however, the data with respect to sample size were positively skewed ($M = 294$, $SD = 867$).

Characteristics of the independent samples. The 150 studies recruited a total of 44,159 respondents. One-hundred fourteen studies ($N = 35{,}863$) reported participant age ($M = 37.91$ years, $SD = 20.23$). Of the 143 studies that reported gender composition, 30.8% had nearly equal

numbers of males and females (45% to 55% male), with the majority of the rest (61.6%) reporting a higher percentage of females (at least 56%). Table 2 summarizes in detail several other sample characteristics. For example, a majority of the studies were published during the last 15 years (76.0%), employed an experimental design (59.3%), included either a student or community sample (82.7%), received funding from academic or government sources (60.7%), and were conducted in the United States (56.7%).

Meta-Analyzing the Samples

What is the overall effect of well-being on health? As shown in Table 3, from the 212 independent samples, the mean unweighted *r* effect size for the well-being–health relation was .135 (*95% CI* = .110 - .160) from the random effects analysis, and .115 from the fixed effects analysis. Both *r* effect sizes are significantly different from zero ($Z = 9.99$ and 23.69, respectively). In addition, the second goal of this meta-analysis was to compare the effect of well-being on health to that of ill-being on health. Using the 99 independent samples that measured the impact of both well-being *and* ill-being on health, we found the ill-being effect size to be significant, negative, and of approximately the same magnitude ($r_{random} = -.155$; $r_{fixed} = -.099$, both ps < .001) as the well-being effect.

Notably, the average well-being–health effect sizes were not consistent across all sample characteristics (see *Q*-values in Table 3). Omnibus homogeneity tests demonstrated substantial within-group variation across the 212 independent samples that measured well-being (Q_w [211] = 903.88, $p < .001$). Hence, we examined average effect sizes separately for each of the three study designs (experimental, ambulatory, and longitudinal). Even with the less powerful random effects model, the average effect sizes varied by study design (Q_{BET} [2, $k = 212$] = 10.50, $p = .005$). Studies that used ambulatory procedures ($r_{random} = .029$, *ns*) reported significantly lower

effect sizes than both longitudinal and experimental studies. With the fixed effects model, the average effect of well-being on health was smaller for longitudinal designs (r_{fixed} = .113) than for experimental designs (r_{fixed} = .166). Because study design proved to be a moderator of the well-being–health effect sizes, and experimental procedures provide the only direct test of causal pathways, many of the subsequent analyses were performed with the three study designs combined and with the experimental studies alone.

Does well-being differentially impact health outcomes? The second goal of this meta-analysis was to determine which health outcomes were most strongly associated with well-being. First, we examined how well-being (and ill-being) was related to our general health categories (short-term outcomes, long-term outcomes, and disease/symptom control). As shown in Table 4, an analysis with 141 samples demonstrated that increases in well-being were positively associated with short-term outcomes (r_{random} = .148; p <.001). Notably, this effect was slightly stronger for the 123 studies that used experimental designs (r_{random} = .172, p <.001). Also, the associations between well-being and long-term outcomes (r_{random} = .112; p <.001) and between well-being and disease/symptom control (r_{random} = .127; p <.001) were both positive, suggesting that well-being promoted healthy functioning and symptom control. However, when examining these associations with only experimental studies, the well-being—long-term outcomes average effect size was smaller and nonsignificant (K = 15; r_{random} = .089, p = .11), and the well-being—disease/symptom control average effect was nearly identical to that for the nonexperimental studies but nonsignificant (K = 12; r_{random} = .122, p = .21).

Does ill-being differentially impact health outcomes? As expected, ill-being was *negatively* related to each category of health outcomes (see Table 4). Comparisons of the effects of ill-being vs. well-being revealed that ill-being has a slightly stronger effect on short-term

outcomes (r_{random} = -.166 vs. r_{random} = .148) and disease/symptom control (r_{random} = -.180 vs. r_{random} = .127), whereas well-being has a slightly stronger effect on long-term outcomes (r_{random} = .112 vs. r_{random} = -.081). Thus, in general the effect sizes for both well-being and ill-being were rather similar (though in opposite directions). These relations held for the experimental studies. Specifically, the average effect size for the short-term outcomes assessed from the 58 samples that manipulated ill-being (r_{random} = -.171) was nearly identical to the effect size from the 123 samples that manipulated well-being (r_{random} = .172). This finding demonstrates that inductions of well-being lead to healthy functioning and inductions of ill-being lead to compromised health at similar magnitudes.

Does well-being differentially impact specific types of health outcomes? The third goal of this meta-analysis was to determine which types of health outcomes were most strongly associated with well-being. As displayed in Table 5, we examined the effects of well-being on 12 specific health outcomes. Focusing on the random effects models for the five short-term health outcomes, we observe that the specific health outcome explained a significant amount of the heterogeneity (Q_{BET} [5, k = 141] = 131.509, p < .001). Well-being was strongly associated with improved immune functioning (r_{random} = .332) and higher pain tolerance (r_{random} =.320). As expected, well-being was also associated with a decreased endocrine system response (r_{random} = -.101), although this relation was much weaker and only marginally significant, when compared to immune and pain outcomes. Finally, well-being was not associated with cardiovascular response/reactivity (r_{random} = .026) nor physiological response (r_{random} = -.031).

Average effect sizes were more homogenous for long-term outcomes and disease/symptom control. For long-term outcomes, well-being was most strongly associated with increased longevity (r_{random} = .137). Well-being also predicted improved general health (r_{random} =

.110) and cardiovascular functioning (r_{random} = .119), and was marginally related to better respiratory functioning (r_{random} = .071; $p < .10$). For disease/symptom control, well-being was associated with slower disease progression (r_{random} = -.150) and longer survival from chronic illness (r_{random} = .097). Although well-being was not significantly related to reduced respiratory conditions (p = .20 from the random effects model), the effect size was in the predicted direction (r_{random} = -.105)

All of the above results – for the short-term, long-term, and disease control categories – were minimally altered when the ambulatory and longitudinal studies were removed, with the exceptions that the positive impact of well-being on improved immune functioning (r_{random} = .371) and better general health (r_{random} = .283) both became stronger, while the positive impact of well-being on long-term cardiovascular functioning (r_{random} = .016) and long-term respiratory functioning became weaker (r_{random} = .018) and nonsignificant.

Sample Specific Moderators of the Well-Being—Health Associations

As demonstrated by the *Q*-values in Tables 4 and 5, tests of heterogeneity indicated that some of the aggregate effect sizes may be moderated by sample level characteristics, such as health status of the sample at baseline, average age of the respondents, and exact health outcome measured. For example, although well-being had a positive effect on all three types of health outcomes (see Table 4), significant heterogeneity was observed within each group. A closer examination by specific health outcome (see Table 5) indicates that heterogeneity is minimal for some outcomes (such as pain tolerance) and much more significant for other outcomes (such as immune function and cardiovascular reactivity). Thus, based on both our general and specific categories and the heterogeneity statistics, we defined five specific groups and examined potential moderators: (a) short-term immune system functioning; (b) short-term endocrine

response; (c) cardiovascular reactivity and physiological response; (d) long-term promotion of healthy functioning (including cardiovascular functioning, general health, and mortality); and (e) enhanced symptom control and survival in chronic conditions.

To explore what underlies this heterogeneity, we focused on five moderators (three categorical and two continuous). The categorical moderators were (a) health status of the sample at baseline (healthy or unhealthy), (b) exact type of health outcome measured (e.g., sIgA antibody production in immune system response; heart rate and blood pressure in cardiovascular reactivity), and (c) the operational definition of well-being (as a state or trait variable). To illustrate the moderating effect of these categorical variables, we reported the descriptive and inferential statistics for each level of the specified moderator. Although we reported both random and fixed effects, we focused here on the more generalizable random effect models. The continuous moderator variables were (a) average age of the sample and (b) percent male respondents. Continuous moderator effects were examined using meta-regression analyses (a necessarily fixed effects models), focusing on the slope (β_1) of the meta-regression line, which indicates whether the effect sizes were associated with changes in the continuous moderators. In each case, we only tested for moderators if the outcome was reported in 10 or more samples. For the exact health outcomes, when effect sizes could not be grouped from 10 independent studies, we documented the exact health outcome most commonly reported.

Moderators of well-being and short-term immune system functioning. Several characteristics of the study sample moderated the effect of well-being on short-term immune system functioning. First, although the average effect sizes for healthy and unhealthy samples were both positive (e.g., increases in well-being were associated with improved immunity), the average effect size was significant for healthy (r_{random} = .360) but not unhealthy (r_{random} = .147)

samples (see top section of Table 6). Further, this positive effect of well-being was magnified in studies measuring sIgA antibody production. For these studies, the average impact of well-being on short-term immune system functioning was stronger (r_{random} = .370) than that in studies that measured other markers of normal immune responses (e.g., increased t cell counts on markers such as CD4, CD8+, and CD16+; r_{random} = .257). Additionally, studies that manipulated or measured state well-being variables reported higher average effect sizes (r_{random} = .338) than studies that determined the relation between well-being and immune system functioning using trait measures of well-being (r_{random} = .164, *ns*). Finally, the gender composition of the sample also moderated the relation between well-being and short-term immune system functioning (see top section of Table 7). The slope of the meta-regression line is significantly negative (β_1 = -.267, p = .0028), which indicates that samples with a higher proportion of female respondents reported larger effect sizes on average.[2]

Moderators of well-being and short-term endocrine response. Several characteristics of the study sample also moderated the effect of well-being on short-term endocrine response (see the second section of Tables 6 and 7). It should be noted again that a negative relation between well-being and endocrine response should be interpreted as promoting healthy functioning, because the increase in endocrine response from negative affect is typically interpreted as compromising health (especially when stress hormones are measured). With respect to the categorical variables, the effect of well-being on decreased endocrine response was not significant in the healthy sample group (r_{random} = -.075, p = .16). The effect size for the unhealthy sample group was quite a bit larger (r_{random} = -.343); however, the unhealthy sample group consisted of only a single small study (N = 26). Thus, neither of these effect sizes was significant. As was observed in the relation between well-being and immune system functioning,

the negative effect of well-being was strongest (and significant) when studies assessed the most commonly measured hormone associated with stress – levels of cortisol. For these studies, the average impact of well-being on short-term endocrine response was stronger (r_{random} = -.109, p = .04) than that for studies that measured other stress hormones (r_{random} = -.043, *ns*). Finally, neither the sample's gender composition nor the average age of the respondent moderated the relation between well-being and short-term endocrine response (see the second section of Table 7). This indicates that the marginally significant drop in stress hormones as a result of well-being is constant across gender and age.

Moderators of well-being and short-term cardiovascular and physiological reactivity. Fewer characteristics of the study sample moderated the effect of well-being on cardiovascular and physiological reactivity (see the third section of Tables 6 and 7). For example, the null association was consistent regardless of whether the sample (a) was healthy or unhealthy, (b) measured heart rate or skin conductance, or (c) manipulated state well-being.

These analyses show only one significant moderating characteristic of the sample – as the average age of the respondents increases, well-being is associated with decreases in cardiovascular reactivity and physiological response. Again, as was true of endocrine system response, a negative relation between well-being and cardiovascular reactivity and physiological response is interpreted as promoting healthy functioning. Thus, as the average age of the sample increased, well-being was associated with promoting healthy cardiovascular reactivity and physiological response. However, three marginally significant findings are of interest. First, studies that measured blood pressure in response to increases in well-being demonstrated that well-being was associated with a marginally significant *increase* in blood pressure. Second, one study measured trait levels of well-being in a follow-up analysis and found these to predict lower

levels of cardiovascular reactivity and physiological response. Third, the marginally significant positive slope from the meta-regression examining gender composition ($\beta_1 = .122$, $p = .07$) indicated that samples with more females reported stronger *negative* relations between well-being and cardiovascular reactivity and physiological response.

Moderators of well-being and long-term healthy functioning. Several characteristics of the study sample moderated the effect of well-being on long-term healthy functioning. First, although the average effect sizes for both healthy and unhealthy samples were both positive (e.g., increases in well-being were associated with long-term healthy functioning), the average effect size was significant for healthy ($r_{random} = .113$) but not unhealthy ($r_{random} = .086$) samples (see Table 6). Interestingly, the different types of long-term health outcomes did not differ widely. For example, coronary risk factors ($r_{random} = -.125$), longevity ($r_{random} = .137$), and other general health outcomes ($r_{random} = .114$) all had similar average effect sizes. Additionally, studies that measured state well-being reported lower average effect sizes ($r_{random} = .075$) than studies that examined the relation between well-being and long-term optimal and healthy functioning using trait measures of well-being ($r_{random} = .132$). Finally, although average age of the respondents was not a significant moderator, the gender composition of a sample did moderate this relation (see the fourth section of Table 7). The slope of the meta-regression line is significantly positive ($\beta_1 = .073$, $p < .001$), which suggests that the average effect size for a sample with all females was lower ($r_{random} = .089$) than the average effect size for a sample with all males ($r_{random} = .162$).

Moderators of well-being and symptom control during chronic conditions. Several characteristics of the study sample moderated the effects of well-being on symptom control during chronic conditions. First, three small studies ($N \leq 20$) measured symptoms of chronic conditions (asthma and allergy symptoms) on healthy samples (see Table 6) in comparison to

unhealthy samples. These studies reported effect sizes that were on average lower (r_{random} = .095) than those samples that measured chronic condition on exclusively unhealthy samples (r_{random} = .120). Also, the type of health outcome measured significantly affected the average effect size. For example, well-being had a stronger relation with recovery from disease (r_{random} = .145) than with the reduction of asthma symptoms (r_{random} = -.077, p = .43). Additionally, studies that measured state well-being revealed a lower, nonsignificant average effect size (r_{random} = .109) than studies that measured trait well-being (r_{random} = .134). Finally, neither the samples' gender composition nor average age was found to moderate this relation (see the last section of Table 7). This indicates that the significant increase in symptom control and survival that is linked to well-being is constant across gender and age.

Discussion

This meta-analysis examined the unidirectional effect of well-being on objective physical and physiological health outcomes. Pooling the results of 150 experimental, ambulatory, and longitudinal studies, we found an average overall *r* effect size of .14 between well-being and objective health. The aggregated r-effect size for the 123 experimental studies that induced positive emotion was .17. Effect sizes can best be conceptualized in practical terms using a binomial effect size display (BESD; see Rosenthal, 1991, 1994, for a full explanation of the BESD procedure and rationale). The BESD is most easily understood when the outcome is dichotomous, as it is for survival. As a case in point, consider the interpretation of the aggregate *r* effect size between well-being and longevity (r_{random} = .14; see Table 5). Using the BESD to interpret this *r* effect size (see Table 8), we expect the probability of living longer increases by 14% for individuals with high well-being compared to those with low well-being. Also, we find

that the survival rate increases 10% for individuals with a chronic illness who have high versus low well-being (BESD not pictured).

Furthermore, not only can *r* effect sizes be converted into a BESD, but the BESD itself can then be translated into two other effect sizes often reported in biomedical research – namely, relative risk (RR) and odds ratio (OR; see Rosenthal & DiMatteo, 2001, for the steps to convert BESD information into other effect size indices). Using the BESD presented in Table 8, our overall effect size can be interpreted as the odds of survival (OR = 1.75) and as the relative risk of mortality (RR = .75). In each case, survival is more likely in the high well-being group and mortality is more likely in the low well-being group. When balancing the costs of improving well-being against the benefits of saving lives, these are very significant differences (Rosenthal, 1991, 1994).

In addition, we compared the differential impact of well-being and ill-being on health outcomes. Effect sizes were similar (though, as expected, in opposite directions), with higher levels of well-being more likely to result in enhanced functioning and higher levels of ill-being more likely to result in compromised functioning. The similar magnitude of these effects sizes was consistent across both experimental and longitudinal study designs. Further, the magnitude of the well-being and ill-being relations was relatively consistent across all three general health outcomes – that is, short-term outcomes, long-term outcomes, and disease/symptom control. Thus, these results demonstrate that the effect of SWB on health is not solely due to ill-being having a detrimental impact on health, but also to well-being having a salutary impact on health. Future research should seek to extend these findings on the differential impact of ill-being and well-being, with a focus on the fundamental underlying mechanisms involved.

Influential Moderators

An important benefit of a meta-analytic study is that its method of synthesizing primary results allows for statistical testing of moderators to determine the factors that affect the relations under investigation (Rosenthal & DiMatteo, 2001). In the present analysis, we examined the impact of several factors, including study design, health outcome, the effect of state vs. trait well-being, and sample characteristics.

Study design. First, our analyses clearly showed that study design was an important moderator, with experimental studies showing the strongest effects, as expected. This result corroborates the pattern of findings documented by Lyubomirsky et al. (2005), who reported the highest *r*s for the effects of experimentally induced positive affect on a variety of outcomes. To confirm that this finding was not simply a function of including similar studies as Lyubomirsky et al., we conducted a follow-up analysis in which we considered only the studies not originally included in that review. This follow-up analysis, which examined 80 new experiments, confirmed the larger effect sizes for experimental studies. There may be several reasons that experimental manipulations of affect produce the biggest effects on health, the most likely being the control that experimental studies have over extraneous variables. In longitudinal and ambulatory studies, other factors, such as psychosocial attributes and measurement differences, are likely to play a relatively greater role (Nesselroade, 1988).

Ambulatory studies offer an interesting cross between short-term processes (observed in experimental manipulations) that are embedded within more long-term periods (Little, Bovaird, & Slegers, 2006). Across the different well-being predictors and outcomes, studies that used ambulatory procedures had significantly lower effect sizes (most of which were not significantly different from zero) than those that used either longitudinal or experimental procedures. We

considered the possibility that the ambulatory studies all focused on a single health outcome by examining the effect sizes of the ambulatory studies for the different health outcomes. Ambulatory studies examined eight different health outcomes and only one health outcome (respiratory diseases/conditions) was significantly associated with well-being (r_{random} = -.166.). All other health outcomes were nonsignificant and near zero. Further, the effect sizes within each health outcome typically varied when experimental and ambulatory studies were directly compared. For example, experimental studies that assessed immune functioning reported a large positive association with well-being (r_{random} = .371), whereas ambulatory studies reported the same association to be near zero (r_{random} = .021).

Thus, it may be that the transient emotions in day-to-day life that are typically measured in ambulatory studies are simply more readily influenced by other variables (such as the weather, time of day, or daily hassles) that attenuate associations between such emotions and health. Alternatively, to date many fewer studies have used ambulatory methodology, and thus researchers are still establishing the best well-being measures to use and appropriate statistical techniques to analyze such data (Little et al., 2006; Mroczek et al., 2006). It will be important in the future to determine how health outcomes should be conceptualized and measured, how much our measures of health outcomes can be extended across lab and field settings and across short and long-term measurement occasions, and the appropriate statistical techniques that should be used in considering such relations (Mroczek et al., 2006).

Health outcomes. One of the main goals of this meta-analysis was to examine the impact of well-being on specific health outcomes. We expected differential effects depending on whether health was defined in terms of short-term states or as long-term processes, predicting that well-being would most strongly impact short-term outcomes. The data support this

hypothesis; however, the stronger short-term well-being–health relations were due to strong associations between well-being and immune functioning and pain tolerance. Short-term effects may be more directly observable, as fewer intervening variables impact the effects that researchers observe; over time, multiple, complex factors potentially moderate long-term health outcomes (Hall et al., 1994; Friedman, 2007). Further, it has been suggested that, at the molecular level, well-being may improve health more directly both by enhancing immune system response and buffering the system from negative effects of stress (Pressman & Cohen, 2005; Smith, 2006).

This hypothesis can be examined by comparing the average effect sizes observed between well-being and increased immune functioning and pain tolerance with the average effect sizes beween well-being and endocrine system response, cardiovascular reactivity, and physiological response. The average effect size for the 69 samples that measured immune functioning and pain tolerance was dramatically higher (r_{random} = .316) than the 80 studies that measured endocrine system response, cardiovascular reactivity, and physiological response (r_{random} = -.009). These results suggest that rather than buffering from cardiovascular and endocrine system response, well-being may be more likely to lead to a rapid recovery to baseline after a stressor is experienced. As an increase in cardiovascular and endocrine activity is a normal response to stress (e.g., Kemeny, 2007), well-being may counter chronic system activation rather than interrupt normal functioning. This finding corroborates the findings of Fredrickson and her colleagues (see Fredrickson & Levenson, 1998; Fredrickson, Mancuso, Branigan, & Tugade, 2000), who have found that people's cardiovascular activation (after a stressful situation) returns more quickly to their baseline levels after watching positive emotion-

inducing films. Further, it appears that well-being not only aids in deregulation of the ANS but also increases immune response; thus, well-being may affect multiple biological processes.

In addition, some of the most commonly assessed physiological markers had the strongest associations with well-being. For example, the relation between transient positive emotions and sIgA antibody production was the single strongest well-being–health effect size in the meta-analysis. This strong relation may be due to the ease at which sIgA antibody production is measured (through saliva), but future work should aim to determine why positive emotions exert such a strong influence on this immune response. Similarly, positive emotions produce a significant drop in cortisol, but a nonsignificant drop in all other stress hormones. The stronger effect may be due to cortisol being the stress hormone that is most susceptible to emotional triggers (Dickerson & Kemeny, 2004), whereas other stress hormones, such as epinephrine and norepinephrine, are activated by other types of triggers (such as physical forms of stress). Further, the results of the 32 studies that measured blood pressure revealed a marginally significant, positive association with well-being. A secondary analysis of these data demonstrated that whereas blood pressure increased as a result of increased positive emotions (r_{random} = .153), blood pressure increased more in the presence of negative emotions. Thus, while positive emotions do result in increases in blood pressure, these increases are smaller than the increases observed for negative emotions.

Operationalizations of well-being. Furthermore, we examined which health outcomes were most strongly associated with state and trait measures of well-being. We expected that short-term health outcomes would be more strongly associated with state manipulations of well-being, whereas long-term health outcomes would be more strongly associated with trait measures of well-being. With the exception of the relations between well-being and endocrine response

and cardiovascular/physiological reactivity, this prediction was supported. That transient emotions have stronger relations with short-term outcomes (especially immune functioning and pain tolerance) and trait levels of well-being have stronger relations with long-term outcomes is informative to future investigations. Researchers interested in altering short-term health outcomes (such as infections or immune system response) may need to focus on increasing transient emotions, whereas researchers interested in modifying long-term health outcomes (such as cardiovascular outcomes or survival) may need to focus on improving more general cognitive assessments of well-being.

Sample specific moderators. In addition, we examined specific sample characteristics, including initial health status, age, and gender composition of the samples. We expected well-being to have a greater impact where dysregulation is typically more evident, such as in unhealthy individuals and older individuals (Solomon & Benton, 1994). Results offered mixed support for these hypotheses. For initial health status, well-being had a greater impact for both short and long-term outcomes in healthy samples; however, well-being more strongly impacted unhealthy samples in controlling disease and increasing survival. This suggests that for healthy samples, well-being may enhance functioning at both molecular and molar levels, whereas for unhealthy samples, well-being may buffer from subsequent decline. This is important because it suggests that promoting well-being may indeed help bring about better physical functioning (Rowe, 1988), especially for healthy individuals, and may improve symptom control for unhealthy individuals. Considering that happiness interventions have demonstrated that individuals can increase their well-being by triggering "upward spirals" through the practice of specific daily behaviors (cf. Lyubomirsky, Sheldon, & Schkade, 2005; Seligman, Steen, Park, & Peterson, 2005), the most successful positive psychological interventions may be those that

ultimately increase the health of physically well individuals, and decrease the disease progression of already physically ill individuals.

Contrary to our predictions, the effects of well-being were fairly constant across age and gender. However, it is interesting to note where these characteristics did make a difference. Age moderated the link between well-being and cardiovascular and physiological reactivity. As individuals age, the risk of cardiovascular-related incidents significantly increases (Siegler, Bosworth, & Elias, 2003; Siegler, Poon, Madden, & Dilworth-Anderon, 2004). Also, chronic stress on the cardiovascular system over time may increase strain on the heart and lead to heart-related problems (Cacioppo & Berntson, 2007). Although the exact mechanisms are unclear, that well-being did decrease reactivity implies that well-being may indeed act as a buffer from strain on the system (McEwen, 1998) for individuals as they age. Further, males showed a stronger effect in long-term functioning outcomes. Through both direct and indirect pathways (Cacioppo & Berntson, 2007; Pressman & Cohen, 2005), well-being may compensate for other vulnerabilities that have led to greater mortality risk for males. Thus, well-being may play a more important role for males. Future research should include these characteristics, as age and gender differences may help us better understand the mechanisms linking well-being and health outcomes.

Limitations

Any research synthesis is only as good as the current research available to be meta-analytically combined. Thus, this meta-analysis is limited by the specific variables that were not manipulated, measured, or reported. First, future researchers may want to examine those specific health outcomes that have been studied with relative infrequency. For example, only three relatively small experimental studies have investigated the impact of well-being on decreased

allergy symptoms, and only eight studies assessed the effect of well-being on the rate of disease progression.

Second, most of the studies included in our meta-analysis focused on healthy populations – either students or healthy community members. This was especially true for the health outcomes related to immune functioning, endocrine functioning, and cardiovascular and physiological reactivity (all of which are likely important outcomes for any unhealthy sample). Although evidence is mounting that higher level of quality of life in is predictive of survival for unhealthy samples (e.g., cancer patients; see Gotay, 2006), the quality of life measures that have been used with these unhealthy samples have typically focused on physical symptoms and health problems rather than on emotional responses or global judgments of life satisfaction. Thus, future research needs to focus on examining the impact of hedonic well-being on health for unhealthy participants with a variety of conditions.

Third, adolescent samples were scarce in the reported research, and, as a result, any current conclusions about the effects of well-being on health for children and adolescents would be tenuous at this time. Finally, many studies did not report information regarding the ethnicity and marital status of their participants. Therefore, these analyses could not be conducted. Given that these variables may moderate many of the relations between well-being and health, we encourage researchers to report these descriptive statistics with greater frequency.

Conclusions

The purpose of this meta-analysis was to examine the effect of well-being on health outcomes. Much of the previous literature has focused either on the strong relation between ill-being and health or on how health influences well-being. Our findings compliment these other studies. Not only can health impact well-being, as has been established in many other

investigations, but well-being can also impact health. Furthermore, extending earlier research, our analyses highlight the complex interrelations between well-being and health. Notably, our findings point to potential biological pathways, such that well-being can directly bolster immune functioning and buffer the impact of stress.

That well-being can affect short term and long term health outcomes and buffer decline in disease is informative for potential medical and psychological interventions. Health has been a primary concern throughout history (Ryff & Singer, 1998), and our findings suggest that a prime area for health promotion involves boosting happiness and increasing the frequency of positive emotions. Indeed, health may be only one of many life domains – albeit a critical one – that is impacted when people actively enhance their own well-being (Lyubomirsky et al., 2005). Furthermore, from a public health standpoint, mortality and morbidity are important (Fries, 1990; Kaplan 2003). As morbidity increases, health care utilization increases, which in turn escalates health care costs. This escalation is a problem that pervades the U.S. health care system (Friedman 1991; Kaplan, 2003; Ryff & Singer, 1998). Thus, to address the question, "What are the benefits of well-being?" we conclude that the benefits extend from individuals' health to a society's health care costs. Accordingly, the problem of how to increase and sustain happiness should continue to be pursued by positive psychologists and health psychologists alike.

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Author Notes

Ryan T. Howell, Department of Psychology, San Francisco State University. Margaret L. Kern and Sonja Lyubomirsky, Department of Psychology, University of California, Riverside.

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Correspondence concerning this article should be addressed to Ryan Howell, Department of Psychology, 1600 Holloway Avenue, San Francisco, CA 94132; email: rhowell@sfsu.edu.

Endnotes

[1]For the studies included in the meta-analysis, experimental investigations typically followed a similar paradigm. Well-being and physiological variables were measured at baseline, mood or emotion was manipulated, and the physiological variables were measured one or more times; mood/emotion was again assessed immediately following as a manipulation check, and the physiological variables were again assessed. In some experiments, subjects acted as their own control, experiencing each mood condition; their reactivity in each condition was compared across conditions and to their baseline level, using repeated measures analysis of variance or similar methods (e.g., Brosschot & Thaler, 2003; Clark, Iverson, & Goodwin, 2001; Codispoti et al., 2003).

Other studies randomly assigned participants to a single mood/emotion condition and compared between subjects, either controlling for baseline levels or using change scores (e.g., Gendolla & Krüsken, 2001a, 2001b). Some of the more recent studies have incorporated multi-level modeling methods to analyze within and between person changes (e.g., Polk, Cohen, Doyle, Skoner, & Kirschbaum, 2005). We do note that there is a lot of variation by study, depending on the outcome of interest, the size of the sample, and the methods used. For example, in one study, pictures were used to induce positive, negative, or neutral moods (Codispoti et al., 2003). Ten participants experienced each condition, one week apart, in counterbalanced order. Blood was drawn at baseline, 30 minutes after baseline, and after the manipulation, and one-way repeated multivariate analysis of variance was used to analyze the effect of picture valance on several neuroendocrine markers. In another study, 54 students were randomly assigned to a negative or positive mood and to an easy or difficult task (Gendolla & Krüsken, 2001a). Heart rate, blood pressure, and skin conductance were continually monitored. Change scores between

the average baseline function, during manipulation, and post manipulation were used to assess the effect of mood on physiological response.

[2]The most straightforward way to interpret the meta-regression statistics from Table 7 is to write out the regression equations from these tables. The basic equation would be as follows:

$$\hat{Y} = \beta_0 + \beta_1$$

where $\hat{Y}$ is the predicted effect size; β_0 is the intercept (when β_1 equals 0); and β_1 is the slope (the change in the predicted effect size with a unit change in the predictor). For example, if we consider the first significant slope from Table 7 (percentage of male respondents), we can use the meta-regression coefficients to predict the effect size between well-being and improved immune functioning for samples differing in gender composition. In this case, the predictor (gender composition) runs from .00 (a completely female sample) to 1.00 (a completely male sample). Thus, the regression equation for this example (see Table 7) would be

$$\hat{Y} \text{ (predicted effect size)} = .392 + -.267\ (X_1)$$

where X_1 is the proportion of the sample that is male. For example, if the proportion is .00 (completely female sample), the predicted effect size between well-being and improved immune functioning is .392. If the proportion is .50 (half female/half male), the predicted effect size is .258. If the proportion is 1.00 (completely male sample), the predicted effect size is .125. We observe that as the sample composition becomes more dominated by males, the strength of the

well-being–improved health effect size decreases. These data suggest that well-being may be more strongly related to improved immune functioning for females.

Table 1

Effect Size Estimates and Sample Characteristics for All Studies

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes			Health Status of Participants	Study Design
					Short-Term Outcomes	Long-Term Outcomes	Disease/ Symptom Control		
Affleck et al. (2000)	1	48	2.31	0.33	.	.	0.33	Medical patients	Ambulatory
Alden et al. (2001)	1	20	1.24	0.29	0.29	.	.	Healthy	Experimental
Apter et al. (1997)	1	21	0.32	0.07	.	.	0.07	Medical patients	Ambulatory
Avia & Kanfer (1980)	1	39	0.96	0.16	0.16	.	.	Healthy	Experimental
Bacon et al. (2004)	1	135	1.36	0.12	.	0.12	.	Medical patients	Ambulatory
Berg & Snyder (2006)	2	173	4.33	0.32	0.32	.	.	Healthy	Experimental
Berk et al. (1989)	1	10	0.91	0.33	0.33	.	.	Healthy	Experimental
Berk et al. (2001)	5	52	4.55	0.63	0.69	.	.	Healthy	Experimental
Boiten (1996)	1	32	-0.37	-0.10	-0.10	-0.10	.	Healthy	Experimental
Brosschot & Thayer, (2003)	1	33	0.33	0.06	.	0.06	.	Healthy	Ambulatory

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Brown, (1993)	2	26	0.00	0.00	0.00	.	.	Healthy	Experimental
Brown et al. (2000)	1	335	-0.86	-0.05	.	.	-0.05	Medical patients	Longitudinal
Bruehl, (1993)	2	80	2.00	0.23	0.23	.	.	Healthy	Experimental
Buchanon et al.(1999)	1	30	2.59	0.46	0.46	.	.	Healthy	Experimental
Carson et al. (1988)	1	32	1.00	0.27	0.33	0.23	.	Medical patients	Experimental
Carter et al. (2002)	1	20	0.03	0.01	0.01	.	.	Healthy	Experimental
Cassileth et al. (1985)	2	359	0.89	0.05	.	.	0.05	Medical patients	Longitudinal
Christie & Friedman (2004)	1	68	-0.19	-0.03	-0.04	0.00	.	Healthy	Experimental
Clark et al. (2001)	1	22	1.55	0.34	0.34	.	.	Healthy	Experimental
Codispoti et al (2003).	1	10	0.53	0.20	0.20	.	.	Healthy	Experimental
Cogan et al. (1987)	2	36	3.26	0.53	0.53	.	.	Healthy	Experimental
Cohen et al. (2003)	2	334	5.27	0.28	.	0.28	.	Healthy	Experimental

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Long-Term Outcomes	Disease/ Symptom Control	Health Status of Participants	Study Design
Danner et al. (2001)	1	180	4.24	0.31	.	0.31	.	Healthy	Longitudinal
Davidson et al. (2003)	1	35	1.92	0.33	0.33	.	.	Healthy	Experimental
Deeg & Zonneveld (1989)	4	2645	6.25	0.12	.	0.12	.	Mix	Longitudinal
Derogatis et al. (1979)	1	35	1.89	0.32	.	.	0.32	Medical patients	Longitudinal
Devins et al. (1990)	1	97	-0.73	-0.08	.	-0.08	.	Medical patients	Longitudinal
Dillon et al. (1985)	1	10	1.94	0.62	0.62	.	.	Healthy	Experimental
Ekman et al. (1983)	1	16	-0.84	-0.23	-0.23	.	.	Healthy	Experimental
Evans et al. (1993)	1	12	-0.42	-0.14	-0.14	.	.	Healthy	Ambulatory
Florin et al. (1985)	2	72	-0.89	-0.16	-0.06	-0.04	-0.44	Mixed	Experimental
Foster et al. (2003)	3	23	-3.97	-0.79	-0.79	.	.	Healthy	Experimental
Frazier et al. (2004)	1	56	1.37	0.19	0.19	.	.	Healthy	Experimental
Fredricksons & Levenson (1998)	2	60	3.08	0.40	0.40	.	.	Healthy	Experimental

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Fredricksons el al. (2000)	6	522	1.91	0.08	0.08	.	.	Healthy	Experimental
Friedman et al. (1993)	2	1178	-3.25	-0.09	.	-0.09	.	Healthy	Longitudinal
Futterman et al. (1992)	1	5	0.16	0.12	0.12	.	.	Healthy	Experimental
Futterman et al. (1994)	1	16	3.57	0.76	0.76	.	.	Healthy	Experimental
Gellman et al. (1990)	1	50	-0.52	-0.08	-0.08	.	.	Unhealthy Community	Ambulatory
Gendolla & Krusken (2001a)	2	112	3.70	0.34	0.34	.	.	Healthy	Experimental
Gendolla & Krusken (2001b)	1	60	1.69	0.22	0.22	.	.	Healthy	Experimental
Gendolla & Krusken (2002)	2	92	0.61	0.07	0.07	.	.	Healthy	Experimental
Gendolla et al. (2001)	2	42	0.12	0.02	0.02	.	.	Healthy	Experimental
Giltay et al. (2004)	1	891	3.29	0.11	.	0.11	.	Mix	Longitudinal
Gomez (2005)	2	72	1.22	0.23	0.14	0.37	.	Healthy	Experimental
Gullette et al. (1997)	1	58	1.20	0.16	.	.	0.16	Medical patients	Ambulatory

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Harrison et al. (2000)	1	30	0.42	0.08	0.08	.	.	Healthy	Experimental
Hertel & Hekmat (1994)	1	20	2.37	0.52	0.52	.	.	Healthy	Experimental
Hess et al. (1992)	1	27	0.40	0.08	0.08	.	.	Healthy	Experimental
Horan & Dellinger (1974)	2	24	2.00	0.44	0.44	.	.	Healthy	Experimental
Houghton et al. (2002)	3	20	0.48	0.14	0.14	.	.	Unhealthy Community	Experimental
Hubert & de Jong-Meyer (1990)	1	24	0.60	0.13	0.13	.	.	Mix	Experimental
Hubert & de Jong-Meyer (1991)	1	20	1.15	0.27	0.27	.	.	Healthy	Experimental
Hubert et al. (1993)	1	52	-4.25	-0.54	-0.54	.	.	Healthy	Experimental
Hucklebride et al. (2000)	2	43	0.55	0.09	0.09	.	.	Healthy	Experimental
Hudak et al. (1991)	1	31	1.96	0.36	0.36	.	.	Healthy	Experimental
Hyland (1990)	1	10	0.53	0.20	.	.	0.20	Medical patients	Ambulatory
Jacob et al. (1999)	1	69	-1.74	-0.21	-0.21	.	.	Healthy	Ambulatory

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes			Health Status of Participants	Study Design
					Short-Term Outcomes	Long-Term Outcomes	Disease/ Symptom Control		
Kawamota & Doi, (2002)	1	2274	2.72	0.06	.	0.06	.	Healthy	Longitudinal
Kitmata (2004)	4	70	2.50	0.32	.	0.00	0.58	Mixed	Experimental
Kivimäki el al. (2005)	2	2852	0.53	0.01	.	0.01	.	Mix	Longitudinal
Knapp et al. (1992)	1	20	-0.35	-0.09	-0.09	.	.	Healthy	Experimental
Koivumaa-Honkanen et al. (2000)	2	7979	16.94	0.19	.	0.19	.	Healthy	Longitudinal
Krause et al. (1997)	1	345	2.93	0.16	.	.	0.16	Medical patients	Longitudinal
Kubzansky et al. (2002)	1	455	2.22	0.10	.	0.10	.	Healthy	Longitudinal
Kubzansky et al. 2001	1	875	6.79	0.23	.	0.23	.	Healthy	Longitudinal
Kugler & Kalveram (1987)	1	20	1.01	0.24	0.24	.	.	Healthy	Ambulatory
Laidlaw et al. (1994)	1	7	1.05	0.48	.	.	0.48	Healthy	Ambulatory
Laidlaw et al. (1996)	1	38	2.63	0.42	.	.	0.42	Medical patients	Experimental

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Lambert & Lambert (1995)	1	39	2.52	0.40	0.40	.	.	Healthy	Experimental
Lefcourt et al. (1990)	3	120	5.28	0.46	0.46	.	.	Healthy	Experimental
Levenson et al. (1990)	1	62	0.65	0.08	0.08	.	.	Healthy	Experimental
Levy et al. (1988)	1	36	3.14	0.50	.	.	0.50	Medical patients	Longitudinal
Levy et al. (2002)	1	660	6.55	0.25	.	0.25	.	Mix	Longitudinal
Liangas et al. (2003)	1	22	-2.69	-0.55	.	.	-0.55	Asthmatics	Ambulatory
Lutgendorf et al. (1999)	1	58	2.46	0.32	0.32	.	.	Mix	Longitudinal
Maier & Smith (1999)	1	513	6.87	0.30	.	0.30	.	Mix	Longitudinal
McClelland & Cheriff (1997)	3	109	4.66	0.44	0.44	.	.	Healthy	Experimental
McCraty et al. (1995)	1	12	-1.14	-0.36	-0.36	.	.	Healthy	Experimental
McCraty et al. (1996)	1	10	1.44	0.50	0.50	.	.	Healthy	Experimental
Meagher et al. (2001)	4	92	1.43	0.16	0.16	.	.	Healthy	Experimental

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Meininger et al. (2004)	1	371	-1.40	-0.07	-0.07	.	.	Healthy	Ambulatory
Milam et al. (2004)	1	412	1.01	0.05	0.05	.	.	HIV patients	Longitudinal
Miller & Wood, (1997)	1	48	0.92	0.20	0.33	.	0.06	Asthmatics	Experimental
Mittwoch-Jaffe et al. (1995)	1	123	3.10	0.28	0.28	.	.	Healthy	Experimental
Moskowitz, (2003)	1	407	3.72	0.18	.	.	0.18	Medical patients	Longitudinal
Neumann & Waldstein (2001)	2	42	-6.30	-0.78	-0.78	.	.	Healthy	Experimental
Njus et al. (1996)	2	50	0.38	0.06	0.06	.	.	Healthy	Experimental
O'Connor & Vallerand (1998)	1	128	1.87	0.17	.	0.17	.	Healthy	Longitudinal
Ong & Allaire (2005)	1	33	0.53	0.10	0.10	.	.	Healthy	Ambulatory
Ostir et al. (2000)	1	1196	7.83	0.22	.	0.22	.	Healthy	Longitudinal
Ostir et al. (2001)	2	2478	3.68	0.07	.	0.07	.	Healthy	Longitudinal
Ostir et al. (2004)	1	1558	3.88	0.10	.	0.10	.	Healthy	Longitudinal

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Ostir, et al (2002)	1	211	3.90	0.26	.	.	0.26	Unhealthy Community	Longitudinal
Palmore (1969)	1	265	2.26	0.14	.	0.14	.	Mix	Longitudinal
Parker et. al. (1992)	2	421	2.32	0.11	.	0.11	.	Healthy	Longitudinal
Perera et al. (1998)	1	15	2.71	0.65	0.65	.	.	Healthy	Experimental
Pitkala et al. (2004)	1	491	2.87	0.13	.	0.13	.	Healthy	Longitudinal
Polk et al. (2005)	1	334	2.19	0.12	0.12	.	.	Healthy	Longitudinal
Pollard & Schwartz (2003)	1	564	0.84	0.05	0.06	0.03	.	Healthy	Ambulatory
Prkachin et al. (1999)	1	31	1.71	0.31	0.31	.	.	Healthy	Experimental
Provost & Decarie (1979)	1	40	1.44	0.23	.	0.23	.	Healthy	Experimental
Reynolds & Nelson, (1981)	1	193	1.66	0.12	.	.	0.12	Medical patients	Longitudinal
Rhudy et al. (2005)	1	28	3.46	0.60	0.60	.	.	Healthy	Experimental
Richman et al. (2005)	1	1388	5.58	0.21	.	0.16	0.26	Medical patients	Longitudinal

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Ritz et al. (2000)	2	48	-0.93	-0.14	.	-0.13	-0.40	Asthmatics vs. Healthy	Experimental
Ritz et al. (2001)	2	40	-0.65	-0.11	.	.	-0.11	Mix	Experimental
Ritz et al. (2005)	2	60	0.10	0.01	.	0.07	-0.05	Mix	Experimental
Rosenbaum (1980)	2	40	1.95	0.32	0.32	.	.	Healthy	Experimental
Santibanez-H & Bloch (1986)	1	34	-1.97	-0.34	-0.34	.	.	Healthy	Experimental
Scheier et al. (1989)	1	51	1.76	0.25	.	.	0.25	Medical patients	Longitudinal
Schwartz et al. (1981)	1	32	-0.65	-0.12	-0.12	.	.	Healthy	Experimental
Schwartz et al. (1994)	1	246	-1.19	-0.08	-0.08	.	.	Healthy	Ambulatory
Scott & Barber (1977)	2	80	1.27	0.15	0.15	.	.	Healthy	Experimental
Shapiro et al. (2001)	1	203	0.01	0.00	0.00	.	.	Healthy	Ambulatory
Sinha et al. (1992)	1	54	-2.02	-0.39	-0.36	-0.18	.	Healthy	Experimental
Smyth et al. (1998)	1	120	0.87	0.08	0.08	.	.	Healthy	Ambulatory

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Sternbach (1962)	1	10	0.00	0.00	0.00	.	.	Healthy	Experimental
Steptoe & Holmes (1985)	2	14	-0.21	-0.07	.	-0.07	-0.08	Asthmatics vs. Healthy	Ambulatory
Steptow & Wardle (2005)	1	160	1.74	0.14	0.14	.	.	Healthy	Longitudinal
Stevens et al. (1989)	1	20	1.21	0.29	0.29	.	.	Healthy	Experimental
Stone et al. (1994)	1	96	0.46	0.05	0.05	.	.	Mix	Ambulatory
Stone et. al. (1987)	1	29	0.91	0.18	0.18	.	.	Healthy	Ambulatory
Stones et al. (1989)	1	156	-1.49	-0.12	.	.	-0.12	Institutional residents	Longitudinal
Szczepanski et al. (1997)	1	101	0.00	0.00	0.00	.	.	Healthy	Ambulatory
Uchiyama (1992)	1	6	-0.17	-0.10	-0.10	.	.	Healthy	Experimental
Uchiyama et al. (1990)	1	10	0.83	0.30	0.30	.	.	Healthy	Experimental
Van Domburg (2001)	2	354	1.71	0.09	.	.	0.09	Medical patients	Longitudinal
Van Eck et al. (1996)	1	86	0.00	0.00	0.00	.	.	Healthy	Ambulatory

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Effect Size *r* For Different Health Outcomes: Long-Term Outcomes	Effect Size *r* For Different Health Outcomes: Disease/ Symptom Control	Health Status of Participants	Study Design
Von Kanel et al. (2005)	1	27	0.00	0.00	0.00	.	.	Healthy	Experimental
Von Leupoldt & Dahme (2004)	1	20	-0.08	-0.02	.	-0.02	.	Healthy	Experimental
Von Leupoldt & Dahme (2005)	2	128	-0.92	-0.12	-0.17	0.02	-0.03	Mix	Experimental
Waldstein et al. (2000)	1	30	1.02	0.19	0.19	.	.	Healthy	Experimental
Weaver & Zillmann (1994)	2	48	1.39	0.21	0.21	.	.	Healthy	Experimental
Weid & Verbaten (2001)	1	43	1.57	0.24	0.24	.	.	Healthy	Experimental
Weisenberg et al. (1998)	1	86	2.34	0.25	0.25	.	.	Healthy	Experimental
Whorwell et al. (1992)	1	18	2.62	0.59	.	.	0.59	Medical patients	Experimental
Williams et al. (1993)	1	82	0.53	0.06	.	0.06	.	Healthy	Longitudinal
Wingard et al. (1994)	1	4725	1.79	0.03	.	0.03	.	Healthy	Longitudinal
Witvliet & Vrana (1995)	1	48	0.47	0.07	0.07	.	.	Healthy	Experimental
Worthington & Shumate (1981)	4	96	3.72	0.38	0.38	.	.	Healthy	Experimental

Table 1 (*continued)*

Study	No. of Samples	n	Z	Average r	Effect Size *r* For Different Health Outcomes: Short-Term Outcomes	Long-Term Outcomes	Disease/ Symptom Control	Health Status of Participants	Study Design
Yogo et al. (1995)	2	24	-5.08	-0.83	-0.83	.	.	Healthy	Experimental
Yoshino (1996)	2	57	1.40	0.19	0.19	.	.	Mix	Experimental
Zachariae et al. (1991)	1	12	0.76	0.25	0.25	.	.	Healthy	Experimental
Zachariae et al. (2001)	1	15	0.40	0.11	.	.	0.11	Healthy	Experimental
Zelman et al. (1991)	1	41	2.53	0.39	0.39	.	.	Healthy	Experimental
Zillmann et al. (1993)	2	40	2.55	0.41	0.41	.	.	Healthy	Experimental
Zillmann et al., (1996)	1	43	0.79	0.12	0.12	.	.	Healthy	Experimental
Zuckerman et al. (1984)	2	351	5.20	0.27	.	0.25	.	Mix	Longitudinal
Zweyer et al. (2004)	1	56	3.73	0.47	0.47	.	.	Healthy	Experimental
Total / Average	212	45,159	9.99	.14	.15	.11	.13		

Note. Each r-effect size represent the average unweighted effect size between well-being constructs and physical health outcomes within the category listed. Thus, effect sizes listed with positive values indicate enhanced health outcomes; effect sizes with negative values indicate compromised health outcomes. The sample size refers to the number of participants used to compute the effect size.

Table 2

General Characteristics of Included Studies

Characteristic	Number of Studies (%)	*r* Effect Size	Total *N*
Year of Report			
2001 – 2006	53 (35.3%)	.11	19,173
1991 – 2000	61 (40.7%)	.15	15,143
1981 – 1990	28 (18.7)	.15	9,310
1970 – 1980	6 (4.0%)	.27	258
Before 1970	2 (1.3%)	.07	275
Design of Study			
Experimental	89 (59.3%)	.16	4,683
Longitudinal	38 (25.3%)	.14	37,128
Ambulatory	23 (15.3%)	.04	2,348
Population Sampled			
Community	65 (43.3%)	.11	37,158
Students	59 (39.3%)	.15	3,162
Children / Adolescents	6 (4.0%)	-.05	1,730
Mixed / Specialized	20 (13.3%)	.25	2,109
Funding Source			
None	59 (39.3%)	.17	9,454
NIMH	9 (6.0%)	.18	1,620
NIA	8 (5.3%)	.18	8,183
NIH	6 (4.0%)	.09	5,713
Academic institution	6 (4.0%)	-.10	276
Grant – other	55 (36.7%)	.14	18,050
Most Common Journals			
Psychosomatic Medicine	20 (13.3%)	.12	4,891
Psychophysiology	10 (6.7%)	.11	727
Biological Psychology	5 (3.3%)	.14	199
Journal of Psychosomatic Research	5 (3.3%)	.02	444
Site of Study			
United States	85 (56.7%)	.16	22,165
Germany	14 (9.3%)	.03	1,249
England	12 (8.0%)	.16	956
Netherlands	7 (4.7%)	.07	4,084
Canada	6 (4.0%)	.16	970
Japan	6 (4.0%)	-.01	2,441

Note. Each *r*-effect size represents the average unweighted effect size between well-being constructs and physical health outcomes within the category listed.

Table 3

The Effect of Well-Being and Ill-Being on Health Outcomes by Study Design

Study Design	Sample Size		*r* Effect Size[a]		95% CI Random Effects Model		Z-value		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Well-Being	42,928	212	.135	.115	.110	.160	9.99	23.688	903.88	<.001
Experimental	4,428	139	$.164_{A}$	$.166_{A}$	.126	.202	8.366	11.162	442.475	<.001
Ambulatory	2,066	24	$.029_{B}$	$-.005_{C}$	-.035	.102	.768	-.256	47.184	.012
Longitudinal	36,434	49	$.128_{A}$	$.113_{B}$	.090	.166	6.556	21.810	356.861	<.001
Ill-Being	8,187	99	-.155	-.099	-.113	-.196	7.166	11.171	341.214	<.001
Experimental	1,892	68	$-.166_{A}$	$-.159_{A}$	-.107	-.224	5.462	7.730	216.091	<.001
Ambulatory	1,707	18	$-.152_{A}$	$-.098_{b}$	-.064	-.238	3.368	7.277	54.315	<.001
Longitudinal	4,588	13	$-.133_{A}$	$-.071_{B}$	-.044	-.221	2.915	4.954	58.348	<.001

Note. Well-being includes life satisfaction, happiness, and positive emotions, whereas ill-being comprises such negative constructs as stress, depression, and anger. Effect sizes with different subscripts in each column differed significantly at $p < .05$. Within the well-being and ill-being sections, effect sizes are independent across study design, so experimental, longitudinal, and ambulatory effect sizes can be compared. Across the well-being and ill-being sections, effect sizes are not independent, so comparisons cannot be made (e.g., experimental to experimental). All effect sizes with Z-values greater than 1.96 are significant at $p < .05$.

[a] Effect sizes listed with positive values indicate enhanced health outcomes; effect sizes with negative values indicate compromised health outcomes.

Table 4

The Effect of Well-Being and Ill-Being on Three Types of Health Outcomes

General Categories of Physical Health Outcomes	Sample Size		*r* Effect Size [a]		95% CI Random Effects Model		Z-value		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Well-Being										
Short-Term Outcomes	6,430	141	.148	.084	.099	.197	5.830	7.159	489.937	< .001
Long- Term Outcomes	34,106	51	.112	.119	.087	.152	7.084	21.017	337.026	< .001
Disease/Symptom Control	3,623	33	.127	.140	.061	.192	3.748	8.849	99.206	< .001
Ill-Being										
Short- Term Outcomes	3,584	73	-.166	-.114	-.105	-.225	5.312	9.671	285.847	< .001
Long- Term Outcomes	3,564	18	-.081	-.054	-.018	-.144	2.515	3.522	43.321	< .001
Disease/Symptom Control	1,275	18	-.180	-.154	-.082	-.274	3.568	6.220	51.643	< .001

Note. Effect sizes within each category are not independent and cannot be compared. All effect sizes with Z-values greater than 1.96 are significant at $p < .05$.

[a] Effect sizes listed with positive values indicate enhanced health outcomes; effect sizes with negative values indicate compromised health outcomes.

Table 5

The Effect of Well-Being and Ill-Being on Specific Health Outcomes

Specific Measures of Physical Health Outcomes	Sample Size		*r* Effect Size[a]		95% CI Random Effects Model		Z-value[b]		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Short- Term outcomes										
Immune System Response	1,323	32	.332	.224	.228	.410	6.423	8.110	73.529	<.001
Pain Tolerance	1,096	37	.320	.320	.257	.380	9.467	10.505	41.504	.243
Endocrine System Response[c]	1,154	21	-.101	-.090	.001	-.201	1.939	2.968	40.607	.004
Cardiovascular System Reactivity[d]	3,181	60	.026	.018	-.045	.096	-.710	-1.144	221.769	<.001
Physiological Response	527	18	-.031	-.056	.098	-.156	.473	1.343	36.596	.004
Long- Term outcomes										
Cardiovascular Functioning	4,332	10	.119	.117	.056	.181	3.706	7.885	25.445	.003
General Health	5,124	7	.110	.057	.024	.195	2.511	4.110	33.072	<.001
Longevity[e]	24,869	24	.137	.128	.093	.181	5.989	20.435	263.246	<.001
Respiratory Functioning	672	12	.071	.071	-.002	.144	1.907	1.907	7.721	.738

Table 5 Continued

Specific Measures of Physical Health Outcomes	Sample Size		*r* Effect Size		95% CI Random Effects Model		Z-value		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Disease / Symptom Control										
Respiratory Conditions	353	16	-.105	-.129	.056	-.262	1.281	2.841	39.671	.001
Disease Progression	1,540	8	-.150	-.170	-.018	-.276	2.229	6.908	36.137	<.001
Survival[e]	2,065	10	.097	.093	.018	.175	2.394	4.420	28.330	.001

Note. Effect sizes from the categories of health are not independent and cannot be compared. All effect sizes with Z-values greater than 1.96 are significant at $p < .05$.

[a] Positive values indicate that well-being produces increased levels of the health category; negative values indicate that well-being produces decreased levels of the health category. [b] Positive Z-values indicate that results were in the expected directions. For example, we would expect well-being to produce less cardiovascular reactivity, but there is a non-significant increase, so the Z is a negative value. [c] Refers to stress hormones, such as cortisol and epinephrine. [d]Includes heart rate reactivity and blood pressure responses. [e] Longevity refers to overall length of life. Survival refers to staying alive despite having one or more chronic conditions.

Table 6

Categorical Moderators of Relations Between Well-Being and Grouped Health Outcomes

	Sample Size		*r* Effect Size[a]		95% CI Random Effects Model		Z-value[b]		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Well-Being → Short-Term Immune System Functioning										
Moderator										
Health Status										
Sample Healthy	715	27	.360	.335	.262	.451	6.795	9.048	46.483	.008
Sample Unhealthy	454	3	.147	.070	-.117	.393	1.090	1.470	2.527	.283
Exact Heath Outcome										
sIgA	514	16	.370	.327	.252	.478	5.792	7.646	29.439	.014
All Other	853	18	.257	.153	.113	.391	3.435	4.323	40.319	.001
State or Trait SWB										
State	853	30	.338	.301	.243	.427	6.612	8.82	54.439	.003
Trait	470	2	.164	.083	-.119	.422	1.139	1.797	3.844	.050
Well-Being → Short-Term Endocrine Response										
Health Status										
Sample Healthy	19	1,104	-.075	-.077	.030	-.178	1.398	2.493	36.273	.007
Sample Unhealthy	1	26	-.343	-.343	.138	-.693	1.449	1.716	-	-
Exact Heath Outcome										
Cortisol	21	1,154	-.109	-.092	-.003	-.212	2.020	3.044	43.488	.002
All Other	4	133	-.043	-.043	.135	-.217	.471	.471	.952	.813
State or Trait SWB										
State	19	660	-.097	-.059	.022	-.214	1.593	1.447	39.257	.003
Trait	2	494	-.131	-.127	.114	-.361	1.048	2.830	.058	.809

Table 6 Continued

	Sample Size		*r* Effect Size		95% CI Random Effects Model		Z-value		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Well-Being → Cardiovascular Reactivity and Physiological Response										
Moderator										
Health Status										
Sample Healthy	3,124	57	.014	.011	-.058	.085	-.373	-.731	215.567	<.001
Sample Unhealthy	140	5	-.011	.013	.252	-.272	.081	-.145	8.575	.073
Exact Heath Outcome										
Blood Pressure	2,218	32	.091	.064	-.005	.186	-1.868	-3.597	156.792	<.001
Heart Rate	1,841	43	.060	.019	-.034	.154	-1.251	-.884	155.314	<.001
Skin Conductance	396	16	.016	-.016	-.114	.145	-.236	.368	30.094	.012
State or Trait SWB										
State	3,128	62	.016	.017	-.054	.086	-.449	-.1.100	72.342	<.001
Trait	160	1	-.136	-.136	.019	-.285	1.717	1.717	-	-
Well-Being → Long-Term Healthy Functioning										
Health Status										
Sample Healthy	23	24,315	.113	.114	.066	.160	4.677	17.876	236.784	<.001
Sample Unhealthy	4	942	.086	.112	-.032	.203	1.428	3.836	8.387	.039
Exact Heath Outcome										
Coronary Risk Factors[c]	7	4,480	-.125	-.059	-.134	.190	3.693	8.224	17.113	.009
Other	10	4,897	.114	.063	.049	.179	3.397	5.048	39.876	<.001
State or Trait SWB										
State	7	567	.075	.075	.002	.147	2.014	2.043	6.077	.415
Trait	32	32,867	.132	.114	.095	.168	6.946	20.888	320.828	<.001

Table 6 Continued

	Sample Size		*r* Effect Size		95% CI Random Effects Model		Z-value		Test of Heterogeneity	
	N	K	Random	Fixed	Lower	Upper	Random	Fixed	Q-Value	Significance
Well-Being → Enhanced Symptom Control and Survival During Chronic Conditions										
Moderator										
Health Status										
Sample Healthy	42	3	.095	.078	-.266	.432	.612	.633	2.538	.281
Sample Unhealthy	3,543	29	.120	.138	.052	.187	3.445	8.633	93.261	<.001
Exact Heath Outcome										
Asthma Symptoms	380	16	-.077	-.125	.114	-.262	.787	2.725	52.998	.003
Recovery from Disease	1,919	10	.145	.126	.063	.224	3.463	5.520	24.925	.003
State or Trait SWB										
State	447	19	.109	.131	-.041	.225	1.424	3.118	49.760	<.001
Trait	3,176	14	.134	.142	.064	.203	3.721	8.285	49.388	<.001

Note. The following samples were not included in the analysis of healthy vs. unhealthy samples becaused they combined healthy and unhealthy samples: two samples that measured immune functioning, one sample that measured endocrine response, 12 samples that measured long-term optimal and healthy functioning, and one sample that measured enhanced symptom control. Effect sizes from the categories of health are not independent and cannot be compared. All effect sizes with Z-values greater than 1.96 are significant at $p < .05$.

[a] Positive values indicate that well-being produces higher levels of the health category; negative values indicate that well-being produces lower levels of the health category. [b] Positive Z-values indicate that results were in the expected directions. [c] We coded cholesterol ratio, HDL and LDL cholesterol level, hypertension, high and low frequency power, nonfatal MI, triglycerides levels as coronary risk factors for the purposes of this moderator test.

Table 7

Continuous Moderators of Relations Between Well-Being and Grouped Health Outcomes

				95% CI for β	
Parameter	Estimate	S*e*	Z-value	LL	UL
Well-Being → Short-Term Immune System Functioning					
Moderator: Average age of the sample (age centered on sample mean).					
$Q_{\text{Model}}(1, k = 23) = .738, p = .390$					
β_0	.199	.034	5.774	.131	.267
β_1	-.002	.002	-.859	-.006	.002
Moderator: Percentage of male respondents					
$Q_{\text{Model}}(1, k = 32) = 8.91, p = .003$					
β_0	.392	.089	6.345	.271	.513
β_1	-.267	.062	-2.986	-.092	-.441
Well-Being → Short-term Endocrine Response					
Moderator: Average age of the sample (age centered on sample mean).					
$Q_{\text{Model}}(1, k = 18) = .228, p = .633$					
β_0	-.073	.033	2.152	-.006	-.139
β_1	-.001	.003	.478	.004	-.007
Moderator: Percentage of male respondents					
$Q_{\text{Model}}(1, k = 20) = .556, p = .454$					
β_0	-.118	.056	2.104	-.007	-.229
β_1	.073	.097	.748	-.118	.264

Table 7 Continued

				95% CI for β	
Parameter	Estimate	S*e*	Z-value	LL	UL
Well-Being → Cardiovascular Reactivity and Physiological Response					
Moderator: Average age of the sample (age centered on sample mean).					
Q_{Model} (1, k = 39) = 10.093, p = 001.					
β_0	.025	.019	-1.300	-.012	.062
β_1	-.005	.001	3.176	-.002	-.007
Moderator: Percentage of male respondents					
Q_{Model} (1, k = 61) = 3.315, p = .068					
β_0	-.050	.037	1.376	.021	-.122
β_1	.122	.067	-1.82	-.009	.251
Well-Being → Long-Term Promotion of Healthy Functioning					
Moderator: Average age of the sample (age centered on sample mean).					
Q_{Model} (1, k = 35) = .450, p = .502					
β_0	.128	.006	21.721	.116	.140
β_1	.0002	.0003	.671	-.0004	.0008
Moderator: Percentage of male respondents					
Q_{Model} (1, k = 36) = 21.187, p <.001					
β_0	.089	.009	9.524	.071	.107
β_1	.073	.015	4.603	.042	.104

Table 7 Continued

Parameter	Estimate	S*e*	Z-value	95% CI for β	
				LL	UL
Well-Being → Enhanced Symptom Control and Survival During Chronic Conditions					
Moderator: Average age of the sample (age centered on sample mean).					
Q_{Model} (1, k = 28) = 2.608, p = .106					
β_0	.111	.021	5.122	.069	.154
β_1	.002	.001	1.615	-.0004	.0042
Moderator: Percentage of male respondents					
Q_{Model} (1, k = 32) = .803, p = .370					
β_0	.175	.037	4.688	.101	.248
β_1	-.048	.053	-.896	.057	-.153

Note. Z-value tests the null hypothesis that the parameter is zero in the population. Moderators that included fewer than 10 samples were not examined using the meta-regression analyses.

Table 8

Binomial Effect Size Display for the Average Impact of Well-Being on Longevity

Levels of variable	High Well-Being	Low Well-Being
Survival	57	43
Death	43	57

Note. The BESD is based on the average effect size (r = .14) for well-being and survival (see Table 5).