



Psychological interventions and coronary heart disease

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ABSTRACT. Epidemiological studies have shown psychological ill-being to be a strong and independent risk factor for both the development and worsening prognosis of coronary heart disease. Psychological intervention trials have attempted to decrease psychological ill-being in order to improve cardiovascular outcomes. Results from these studies, however, are mixed. Evidence from recent epidemiological studies suggests that psychological health assets (also known as positive psychological factors) independently promote cardiovascular health. Despite these promising findings, intervention studies have yet to examine how enhancing these psychological health assets might contribute to increased cardiovascular well-being. Therefore, we argue for an expanded focus on psychological health assets in our conceptualization of the relationship between psychological functioning and cardiovascular health. We examine this expanded focus by reviewing past psychological intervention trials (aimed at reducing psychological ill-being), recent epidemiological studies prospectively linking psychological health assets to cardiovascular health, and psychological health asset interventions. Further exploring this knowledge gap is important because it may eventually lead to innovative avenues of prevention and intervention.

KEY WORDS. Behavioral cardiology. Positive psychology. Health psychology. Coronary heart disease. Theoretical study.

RESUMEN. Los estudios epidemiológicos han demostrado que el malestar psicológico es un fuerte e independiente factor de riesgo para el desarrollo y el empeoramiento del pronóstico de la enfermedad coronaria del corazón. Los ensayos de las intervenciones psicológicas han pretendido disminuir el malestar psicológico para mejorar los resul-

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tados cardiovasculares. Los resultados de esos estudios, sin embargo, son mixtos. Evidencia de los estudios epidemiológicos recientes sugiere que los recursos de salud psicológica (también conocidos como factores psicológicos positivos) promueven independientemente la salud cardiovascular. A pesar de estos hallazgos prometedores, los estudios de intervención deben todavía examinar cómo la mejora de estos recursos de salud psicológica puede contribuir al aumento del bienestar cardiovascular. Por ello, abogamos por una mayor atención en los recursos de salud psicológica en la conceptualización de la relación entre el funcionamiento psicológico y la salud cardiovascular. Examinamos esta mayor focalización revisando los ensayos de intervenciones psicológicas (cuyos objetivos han sido reducir el malestar psicológico), los estudios epidemiológicos prospectivos recientes relacionan los recursos de salud psicológica con la salud cardiovascular y las intervenciones con recursos de salud psicológica. Mayor exploración de esta laguna en el conocimiento es importante, ya que finalmente puede llevar a innovar los modos de prevención e intervención.

PALABRAS CLAVE. Cardiología conductual. Psicología positiva. Psicología de la salud. Enfermedad coronaria del corazón. Estudio teórico.

Coronary heart disease (CHD) is the leading cause of death worldwide, killing more than 7 million individuals annually (Mackay, Mensah, Mendis, and Greenlund, 2004). Once thought to be a disease of industrialized nations, CHD is rapidly increasing in developing countries as well. As the average age of the world population rises, it is estimated that the deaths attributable to CHD will only increase (Braunwald, 1997).

Because CHD imposes psychological, social, and financial burdens on society, there is great interest in finding ways to reduce its occurrence. Therefore, a great deal of research has aimed to identify and reduce risk factors. Some of the risk factors have been linked to specific mechanisms, others not. The best-established risk factors for CHD are demographic, biological, and behavioral, including older age, male gender, family history of cardiovascular disease, smoking, high blood cholesterol, high blood pressure, physical inactivity, being overweight, diabetes mellitus, and excessive alcohol use (Lloyd-Jones *et al.*, 2010). A growing body of research has established that negative psychological factors (*e.g.*, anger/hostility, anxiety, and depression) are linked with an increased risk for cardiovascular events (Chida and Steptoe, 2009; Moussavi *et al.*, 2007; Pettit, Grover, and Lewinsohn, 2007; Lett *et al.*, 2004; Richards and Solanas, 2008; Rosengren *et al.*, 2004; Rozanski, Blumenthal, Davidson, Saab, and Kubzansky, 2005).

An individual's psychology figures into the success of any intervention (*e.g.*, by influencing motivation to seek help and adhere to a medical regime), but the focus in this review is on interventions that explicitly target psychological risk factors and psychological health assets. So, smoking cessation, exercise, and diet programs are not reviewed. Furthermore, no large-scale intervention trials have examined how anxiety and anger interventions impact the occurrence of cardiovascular events among patients (Harlapur, Abraham, and Shimbo, 2010). Only interventions targeting depression and similar constructs are able to be reviewed here. Using standardized guidelines, we also review emerging research linking psychological health assets with cardiovascular health

and discuss how this line of research may provide new insights (Fernández-Ríos and Buela-Casal, 2009)

Depression plays a crucial role in the etiology and prognosis of CHD. Among those who suffer from an acute myocardial infarction (MI), the prevalence of major depression is three times higher when compared to a healthy population (Thombs *et al.*, 2006). This statistic is distressing because two recent meta-analyses estimated that cardiovascular patients with major depression are at a 2 to 2.5 fold increased risk of having another cardiovascular event (Barth, Schumacher, and Herrmann-Lingen, 2004; van Melle *et al.*, 2004). The additional hardships that depression inflicts in cardiac patients may explain why depressed CHD patients accrue higher health care costs (Frasure-Smith *et al.*, 2000).

A recent review of the literature on depression and CHD confirmed the damaging effects of depression. Goldstone and Baillie (2008) found nine reviews examining the epidemiological link between depression and CHD. In total, 138,807 individuals, who were CHD-free at baseline, ranging in age from 18 to 103, were followed for three to 27 years. All nine reviews agreed that a strong link exists between depression and CHD. Furthermore, seven reviews examining the prognostic literature among those with depression and CHD were identified. In total, 23,005 individuals, ranging in age from 24 to 88, were followed for four months to 19 years. All the reviews agreed that depression appears to increase the risk of both cardiovascular events and all-cause mortality in cardiac patients.

A multidisciplinary consensus document was recently issued by an American Heart Association Scientific Advisory Committee and endorsed by the American Psychiatric Association (Lichtman *et al.*, 2008). The document stated that 15-20% of all MI patients meet criteria for major depression and that an even larger proportion of these patients display elevated depressive symptoms. Depression appears to deter recovery because depressed cardiac patients have a diminished ability to modify risk factors and relevant health behaviors (*e.g.*, lower adherence to medication and cardiac rehabilitation).

Overall, depression possesses risk gradients comparable to traditional risk factors such as elevated cholesterol or smoking (Dimsdale, 2008). The INTERHEART study assessed eight coronary risk factors (current smoker, diabetes, hypertension, abdominal obesity, fruit/vegetable intake, exercise, alcohol, ApoB/ApoA-1 Ratio) along with an index of psychological distress (depression, perceived stress at home or work, locus of control, financial stress, and adverse life events) (Yusuf *et al.*, 2004). The information was derived from an international sample of 15,152 acute post-MI patients and 14,820 matched controls from 52 countries. The risk attributable to psychological distress was comparable to traditional risk factors.

Review of behavioral intervention studies

Given the strong association between psychological distress and CHD, it is plausible that alleviating psychological distress among individuals with CHD might reduce future cardiovascular events and mortality. Indeed, several large-scale intervention studies based on this rationale have been conducted. However, results from these trials are

mixed (Rozanski *et al.*, 2005). Here we review the large-scale randomized controlled trials that used stand-alone psychological interventions to target psychological distress among cardiac patients.

To date, six large-scale studies have examined the impact of stand-alone psychological interventions in cardiac patients. The results of the interventions vary. The Ischemic Heart Disease Study employed a home-based stress reduction program and successfully reduced stress and cardiac deaths by nearly 50% (Frasure-Smith and Prince, 1985). Furthermore, an analysis of seven-year follow-up data showed that the recurrence of MI was significantly lower in the treatment group. The Recurrent Coronary Prevention Project Study used behavior therapy in a group therapy format. The intervention reduced both negative affect and Type A behavior. In turn, rates of cardiovascular mortality and nonfatal MI were both reduced (Friedman *et al.*, 1986).

Two other large trial interventions were unable to reduce psychological distress and as a consequence, cardiac events in the treatment group were not reduced (Frasure-Smith *et al.*, 1997; Jones and West, 1996). In fact, there was a somewhat higher mortality rate among women who received the intervention in the Montreal Heart Attack Readjustment Trial (M-HART). The Canadian Cardiac Randomized Evaluation of Antidepressant and Psychotherapy Efficacy (CREATE) was another study that did not support the effectiveness of psychological interventions. It was designed to examine the short-term efficacy of an SSRI and interpersonal psychotherapy (IPT) in reducing depressive symptoms among those with depression and coronary artery disease. CREATE was a randomized controlled trial with a 2 x 2 factorial design. Patients were provided with 12 weeks of citalopram or placebo. The other groups were provided interpersonal psychotherapy (IPT) plus clinical management or clinical management alone. Citalopram significantly reduced depressive symptoms, but IPT did not perform better than clinical management alone. Like some other studies however, CREATE was not powered to detect differences in adverse cardiovascular events. As a result, no such differences were detected.

The Enhanced Recovery in Coronary Heart Disease Patients (ENRICHD) study is the largest stand-alone psychological intervention trial to date. The study enrolled 2481 patients with a previous MI and depression and/or low perceived social support. The impact of psychological treatment on the primary end point (all-cause mortality and/or nonfatal MI) was tracked (The ENRICHD Investigators, 2003). Within a month of acute MI, patients were randomized into a treatment or usual care group. The treatment group consisted of individual cognitive behavioral therapy. For those with severe or unrelenting depression, group therapy and serotonin reuptake inhibitors were also offered—when possible. Follow-up analyses revealed that there was no difference between the treatment vs. usual care group in recurrent nonfatal MI, all-cause death, or cardiac death (The ENRICHD Investigators, 2003). Analyses also revealed that psychological functioning between the treatment and usual care group was only modestly different. During the experiment, researchers observed that usual care providers were also offering psychological therapies and cardiac rehabilitation programs. This overlap may have reduced treatment group differences.

The robust improvement in the usual care group has been attributed to the aggressive treatment provided (Rozanski *et al.*, 2005). In contrast, two randomized trials where cardiac participants were not chosen based on psychological criteria (or not told that they were chosen on the basis of psychological criteria), showed either no improvement, or worsening of anxiety and depression (Bishop *et al.*, 2005; Frasure-Smith *et al.*, 1997). Therefore, informing ENRICHD participants that they were selected for the study because they were depressed and/or socially isolated may have motivated patients in the usual care group to seek psychological treatment, helping them fight the trend of increasing anxiety or depression observed in the usual care groups of other studies (Saab *et al.*, 2009).

In a follow-up study, secondary analysis of the ENRICHD data revealed that those involved in group therapy plus individual therapy benefited from a 33% reduced risk of all-cause mortality and/or nonfatal MI compared to those in usual care (Saab *et al.*, 2009). The authors of the study cautioned that the subset of patients who partook in the group-therapy was not randomized into group training; therefore, causation cannot be inferred.

The Stockholm Women's Intervention Trial for Coronary Heart Disease (SWITCHD) is the sixth and most recent study to use a stand-alone psychological intervention (Orth-Gomér *et al.*, 2009). 237 women with CHD were recruited and randomized to either a group-based psychological intervention program or usual care. The intervention program began four months after hospitalization. Each group had four to eight women and met 20 times over the course of a year. At one year follow-up, participation in group therapy resulted in healthier levels of several inflammatory markers. Over a mean follow-up of seven years, participation in group therapy resulted in a three-fold reduction in mortality. This study is particularly important because previous studies indicated that psychological intervention programs were successful at reducing cardiovascular events or mortality only in men (Linden, Phillips, and Leclerc, 2007; Schneiderman *et al.*, 2004). In their discussion section of the M-HART (secondary analysis) outcomes, Cosette, Frasure-Smith, and Lesperance (2001) noted how women may have been less responsive to the type of treatment offered in the M-HART study. SWITCHD, however, demonstrated that women with CHD and psychological distress benefit from psychological interventions offered in a format tailored for women. The intervention in SWITCHD was based on observations made during a female coronary risk study (Orth-Gomér *et al.*, 2009).

Meta-analysis

The most recent meta-analysis to examine the impact of psychological treatment in cardiac patients identified 43 randomized trials, 23 of which reported mortality data (Linden *et al.*, 2007). The analyses revealed that psychological treatments were effective at reducing both mortality and recurrence of cardiovascular events. The benefit of psychological treatment, however, was moderated by gender, perceived distress, elapsed follow-up time, and timing of the treatment. The meta-analysis found that the psychological treatments only reduced mortality in men. However, only 10 studies allowed for gender analysis and the recent SWITCHD study (published after the meta-analysis) demonstrated

that women too benefit from psychological treatment. Furthermore, mortality reduction occurred only when distress was successfully reduced by the intervention. Moreover, reduction in the recurrence of cardiovascular events was only evident in long-term follow-up (more than two years) and not in short-term follow-up. Finally, only psychological treatments that began two months after the cardiac event significantly reduced mortality—mortality was reduced by 72% in this circumstance.

Psychological treatments that began immediately after the cardiac event were ineffective at reducing mortality. Linden *et al.* (2007) speculated that early recruitment captures a subsample of patients who possess both the resources and resilience to autonomously recover without assigned psychological aid. When this subsample is recruited into both the treatment and control groups, they may distort the results. The authors of the meta-analysis went on to state that their hypothesis is consistent with Schrader, Cheok, Hordacre, and Guiver's (2004) observations; they found that depression in post-myocardial infarction patients can remain steady, worsen over time, or spontaneously improve. Furthermore, depression that is detected immediately after a cardiovascular event may remit on its own because the depression is a transient reaction to the medical instability induced by the cardiovascular event itself (Davidson *et al.*, 2006). One reason why psychological treatments appeared to reduce mortality only in men, according to this meta-analysis, may be that a large percentage of women included in the gender analysis came from the ENRICHD study, which randomized patients and initiated treatment within one month of a cardiac event.

Survey of psychological health assets and cardiovascular outcomes

Less understood is the possible protective role played by positive psychological factors, what can be termed psychological health assets, such as optimism, positive affect, emotional vitality, life satisfaction, happiness, and meaning and purpose in life. The vast majority of past research has focused on how psychological ill-being negatively impacts physical health. But a scientific discipline examining how psychological health assets impact the risk of disease onset and progression hardly exists. Exploring this knowledge gap is important because it may lead to innovative avenues of prevention and intervention (Pitt and Deldin, 2010; Seligman, 2008). Furthermore, the American Heart Association's Strategic Impact Goals Through 2020 challenged researchers to examine determinants of cardiovascular health above-and-beyond the mere absence of clinically manifest disease (Lloyd-Jones *et al.*, 2010). Recent research shows that psychological health assets uniquely enhance several domains of physical health including: health behaviors, biological processes, morbidity, and longevity (Boyle, Barnes, Buchman, and Bennett, 2009; Fredrickson, Mancuso, Branigan, and Tugade, 2000; Friedman, Hayney, Love, Singer, and Ryff, 2007; Peterson, 2006; Pressman and Cohen, 2005; Steptoe, Wardle, Marmot, and McEwen, 2005; Xu and Roberts, 2010). Such research bears directly on the possibility that positive psychological states promote cardiovascular health.

Investigating relations between psychological health assets and CHD is important because psychological health assets do not merely indicate the absence of negative

psychological factors, but may operate via distinct mechanisms (Ryff *et al.*, 2006). They may enhance cardiovascular health by either directly impacting the biological system or indirectly (*e.g.* facilitating better health behaviors). For example, psychological health assets have been uniquely linked with slowed atherosclerosis and calcification, higher heart rate variability (an indicator of cardiovascular health), and healthier levels of cardiac functioning (*e.g.*, blood pressure, heart rate), inflammation (*e.g.*, C-reactive protein, interleukin-6, fibrinogen), and metabolism (*e.g.*, glycemic control, body mass index) (Boehm and Kubzansky, 2010). Psychological health assets have also been associated with healthier behaviors such as less smoking, non-excessive alcohol consumption, and more physical activity. These findings demonstrate that an exclusive focus on psychological ill-being paints an incomplete view of the relations between psychological factors and cardiovascular health. Therefore, expanding the focus and making room for the inclusion of psychological health assets when conceptualizing the dialogue between psychological functioning and cardiovascular health, may lead to innovative insights.

Investigation of how psychological health assets impact physical health has already led to the emergence of a growing body of literature that longitudinally links several psychological health assets with better cardiovascular health including: optimism, positive affect, vitality, hope, purpose in life, and life satisfaction (Boehm and Kubzansky, 2010). In order to demonstrate that a psychological health asset has a unique effect, controlling for psychological illness and distress is crucial. To our knowledge, approximately twenty studies specifically examined how psychological health assets impact cardiovascular health while also controlling for negative affect (*e.g.*, anxiety, anger, and/or depression). A handful of them are outlined here. Tindle and colleagues (2009) tracked over 97,253 women for eight years and found evidence of an association between optimism and MI. After adjusting for cardiovascular risk factors and depressive symptoms, more optimistic women were less likely to die from CHD or CHD-related causes and lived approximately eight years longer. Kubzansky and Thurston (2007) followed 6913 individuals for an average of 15 years. After adjusting for depressive symptoms and traditional cardiovascular risk factors, individuals with high vitality had a reduced risk of CHD. Denollet and colleagues (2008) tracked 874 patients over a two year follow-up and studied the link between positive affect and outcomes following percutaneous coronary intervention by stent implantation. In multivariate analyses (controlling for depression, anxiety, and negative affect) individuals with low positive affect were more likely to experience MI six months post-intervention than those with high positive affect. Sone and colleagues (2008) followed 43,391 Japanese adults over a period of 7 years. After adjusting for mental stress, a one-item measure of *ikigai*—a deep-rooted of sense meaning in life—was linked with reduced CVD-related mortality. Davidson, Mostofsky, and Whang (2010) tracked 1,739 Canadian Adults in Nova Scotia for 10 years. Using a non-self report measure of positive affect, where coders rated video interviews of participants, the team found that positive affect was associated with a reduced risk of CHD. This association persisted in multivariate analyses that controlled for traditional risk factors along with depression, anxiety, and hostility. The key theme, the strand that weaves together all these findings, is a link between psychological health assets and cardiovascular health: psychological health assets buffer against negative cardiovascular events.

If future investigations continue to show that psychological health assets contribute to cardiovascular health, research into interventions that increase positive psychological functioning, which may in turn enhance cardiovascular health, is needed. Past interventions that have attempted to reduce cardiovascular events by first reducing psychological distress have generated mixed results. Researchers should continue work in this important area, but also address the fact that intervention trials examining the impact of psychological health assets on cardiovascular health is lacking.

Psychological health asset interventions

One novel method of promoting cardiovascular health is tailoring existing interventions that bolster psychological health assets, and applying them to cardiac patients. Although specific interventions that target cardiovascular health do not currently exist, candidate interventions are on hand. A growing body of research, the best of it random assignment placebo controlled, demonstrates that interventions can reliably enhance various psychological health assets including positive affect (Cohn and Fredrickson, 2010; Fredrickson, Cohn, Coffey, Pek, and Finkel, 2008; Sheldon and Lyubomirsky, 2006), happiness (Lyubomirsky, Sheldon, and Schkade, 2005; Seligman, Steen, Park, and Peterson, 2005), psychological well-being (Fava and Tomba, 2009; Jacobs *et al.* (2011), life satisfaction (Lyubomirsky, Sousa, and Dickerhoof, 2006; Seligman, Rashid, and Parks, 2006), subjective well-being (Emmons and McCullough, 2003; King, 2008), resilience (Brunwasser, Gillham, and Kim, 2009), and optimism (Antoni *et al.*, 2001; Peterson and Bossio, 1991; Riskind, Sarampote, and Mercier, 1996; Seligman, Schulman, and Tryon, 2007). Just recently, randomized controlled interventions trials, with the aim of enhancing psychological health assets in cardiac patients has begun (Burton, Pakenham, and Brown, 2009; Charlson *et al.*, 2007). Such experimental studies will help us better grasp the nature of the relationship between psychological health assets and CHD.

Limitations

Several of the intervention studies described in this paper were not sufficiently powered to distinguish among cardiovascular outcomes; therefore, it is not surprising to find that their interventions showed no effects. Furthermore, existing intervention studies have traditionally targeted individuals who had already developed CHD. Studies have yet to determine if interventions among individuals free of CHD at baseline would benefit from interventions that reduce psychological distress. Finally, studies have yet to explore how interventions that reduce anxiety or anger may impact future cardiovascular events.

Future research directions

This review identifies several unresolved issues that require answers, answers that can be found with further research. One crucial but unexplored issue is whether randomized

controlled trials that increase psychological health assets protect against cardiovascular events. A recently deployed large-scale intervention may provide an exciting answer for this unresolved frontier. The United States Army is currently teaching every soldier a range of empirically tested methods that increase various psychological health assets (Cornum, Matthews, and Seligman, 2011). The program is called Comprehensive Soldier Fitness (CSF), and every soldier's psychological profile is being regularly assessed over time with a comprehensive psychological assessment called the Global Assessment Tool (Peterson, Park, and Castro, 2011). These psychological data points are being added to the wealth of existing physical health data that the U.S. Army already collects. As the program rolls out, CSF may serve as a natural trial to examine the impact that psychological health asset enhancing interventions have on cardiovascular health (Lester, McBride, Bliese, and Adler, 2011).

Our wish to raise awareness about the impact that psychological health assets have on cardiovascular health should not be interpreted as a call to deny, minimize, nor ignore the severity and negative impact that traditional psychological risk factors have on cardiovascular outcomes. In fact, we believe that more research on this important topic is needed. Rather, our hope is that the field of behavioral cardiology will expand its focus and make room for psychological health assets when conceptualizing how psychological factors impact cardiovascular health. By doing so, the field may become better equipped to combat the toll that CHD takes on society.

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