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Interrelations between psychopathology, psychosocial functioning, and physical health: An integrative perspective[1]

Jeremy W. Pettit[2] *(University of Houston, USA),*
Kelly E. Grover *(University of Houston, USA),*
and Peter M. Lewinsohn *(Oregon Research Institute, USA)*

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ABSTRACT. The field of psychosomatics is large and thriving, with diverse findings from multiple levels of analysis. As a result of the growth in psychosomatics over the past several decades, integration of empirical findings on the overlap of mental and physical health represents a daunting challenge. Moreover, much of the research in psychosomatics has involved examination of isolated variables in the absence of a guiding theoretical framework. The current paper presents a theoretical model delineating the pathways between psychopathology, physical health, and associated psychosocial constructs that may be used to (a) assimilate the large body of research findings in the field and (b) guide future research endeavors on the relations between mental and physical health. An example of model's relevance for explaining the relations between mental and physical health constructs is provided with a review of literature on depression and coronary heart disease. We hope that this model will provide a theoretical framework for understanding mental-physical health overlap, and that future research will test and refine paths in this model.

KEYWORDS. Psychosomatics. Depression. Coronary heart disease. Theoretical study.

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[2] Correspondence: Department of Psychology. 126 Heyne Building. University of Houston. Houston, Texas 77204-5022 (USA). E-Mail: jpettit@uh.edu

RESUMEN. El campo de los trastornos psicosomáticos es amplio y floreciente, abarcando diversos resultados desde múltiples niveles de análisis. Como resultado de su crecimiento durante las últimas décadas, la integración de hallazgos empíricos referidos tanto a la salud mental como a la física supone todo un reto. Además, la mayoría de las investigaciones psicosomáticas han examinado variables aisladas, sin guiarse por un marco teórico. Este artículo presenta un modelo teórico que delimita las relaciones entre la psicopatología, la salud física y constructos psicosociales asociados, cuyo uso permitiría: a) asimilar la gran cantidad de conclusiones de las investigaciones en este campo, y b) guiar investigaciones futuras sobre las asociaciones entre la salud mental y física. Se presenta un ejemplo de la relevancia de este paradigma para explicar las relaciones entre salud mental y física con una revisión de la literatura en depresión y la enfermedad cardiaca coronaria. Esperamos que este modelo proporcione una perspectiva teórica para la comprensión del solapamiento entre salud mental-física, y que la investigación futura pruebe y refine las asociaciones postuladas desde el modelo.

PALABRAS CLAVE. Psicosomática. Depresión. Enfermedad cardiaca coronaria. Estudio teórico.

RESUMO. O campo da psicossomática é amplo e florescente, abarcando diversos resultados a partir de múltiplos níveis de análise. Como resultado do seu crescimento durante as últimas décadas, a integração descobertas empíricas referidas tanto na saúde mental como na saúde física coloca um desafio. Além disso, a maioria das investigações psicossomáticas têm analisado variáveis isoladas, sem se orientarem por um marco teórico. Este artigo apresenta um modelo teórico que delimita as relações entre a psicopatologia, a saúde física e construtos psicossociais associados, cujo uso permitiria: a) assimilar grande quantidade de conclusões das investigações neste campo, e b) orientar investigações futuras sobre as associações entre saúde mental e física. Apresenta-se um exemplo da relevância deste paradigma para explicar as relações entre saúde mental e física com uma revisão da literatura em depressão e doença coronária. Esperamos que este modelo proporcione uma perspectiva teórica para a compreensão da sobreposição entre saúde mental-física, e que a investigação futura prove e aperfeiçoe as associações postuladas a partir do modelo.

PALAVRAS CHAVE. Psicossomática. Depressão. Doença coronária. Estudo teórico.

Introduction

The overlap between mental and physical health has long been a subject of theoretical and empirical investigation. Numerous models of mental-physical health links have been proposed and tested, typically falling within one of three areas of health psychology and behavioral medicine: health behavior and prevention; psychosomatics (*i.e.,* the impact of stress and emotion on the development of disease); and psychosocial aspects of acute and chronic illness and care (Smith and Ruiz, 2004). The majority of work has focused on psychosomatics, including the relations of physical health with stress, personality, social functioning, and to a lesser extent, psychopathology.

Perhaps the most active area of research in psychosomatics has been on the links between stress and physical well-being. Beginning with Selye's (1936, 1950) groundbreaking work on the general adaptation syndrome, researchers have systematically uncovered and refined associations between various manifestations of stress and physical health. Although research in this area is extensive and complex, models generally suggest that prolonged exposure to stress may directly suppress immune system functioning, which in turn enhances risk for disease (Kiecolt-Glaser, McGuire, Robles, and Glaser, 2002; Robles, Glaser, and Kiecolt-Glaser, 2005). Others have expanded this general approach to incorporate the roles of cognitive processes and behavioral responses. As an example, Cohen (1996) proposed that the perception of stress precipitates negative emotional responses. Negative emotions, in turn, impact immune system functioning both directly via physiological processes and indirectly via changes in health-related behaviors (*e.g.,* smoking). Cohen and his colleagues *(e.g.,* Manuck, Cohen, Rabin, Muldoon, and Bachen, 1991; Marsland, Bachen, Cohen, Rabin, and Manuck, 2002; Rabin, Cohen, Ganguli, Lyle, and Cunnick, 1989) have carried out an impressive line of empirical studies to provide support for this model. A related model (Pennebaker, 1992) suggests that stress, when followed by the inhibition of negative emotions, increases risk for adverse health outcomes via chronic hyper-arousal of the nervous system. According to Pennebaker (1997), inhibition of emotions may be viewed as a long-term low-level stressor that may trigger the onset or exacerbation of health problems for three reasons: (a) it is physiologically taxing because it leads to increased autonomic nervous system activity; (b) it hinders automatic cognitive processes that are thought to preserve health; and (c) it produces increased cognitive processing related to the stressor, which restricts the cognitive resources available for other tasks. Models such as Cohen's and Pennebaker's highlight the role of stress in deteriorating physical health while incorporating psychological constructs of negative affectivity, attributions or perceptions, and emotional inhibition.

These and other models of the connections between stress and compromised physical functioning heralded the examination of relations between numerous psychosocial constructs and physical health. Indeed, many of the constructs included in psychosomatic research *(e.g*., social functioning, psychopathology, coping skills) may be conceptualized as varied forms of stressors, precipitants of stressors, or moderators or mediators of stress-physical health relations. For example, certain personality variables (*e.g.,* hostility, neuroticism) that are frequently incorporated in psychosomatic research predict higher levels of environmental adversity and stress (*e.g.,* Kendler, Gardner, and Prescott, 2003; Suls, 2001). Unsurprisingly, these same personality variables are associated with increased risk of physical health impairments (*e.g.,* Duberstein et al., 2003; Friedman and Booth-Kewley, 1987; Goodwin and Engstrom, 2002; Smith, 1992). In contrast to the health risks conferred by personality variables like hostility and neuroticism, some evidence indicates that personality features such as high extraversion, and possibly high conscientiousness, may represent protective factors[3] for physical health (Cohen, Doyle,

[3] Although vulnerability and protective factors are often conceptualized as falling at opposite ends of a single continuum, we distinguish them here because variables that increase the likelihood of a given outcome may differ from those that decrease the likelihood of that outcome. In this paper, vulnerability and protective factors are conceptualized as variables that may influence outcomes in either an additive (*i.e.,* main effect) or interactive (*i.e.,* moderator) fashion.

Turner, Alper, and Skoner, 2003; Duberstein et al., 2003; Goodwin and Engstrom, 2002; Hampson, Goldberg, Vogt, and Dubanoski, 2006). Potential mechanisms by which personality impacts physical health, either positively or negatively, include physiological reactivity to stressors, levels of conflict-laden and stressful environments, frequencies of health risk and health maintenance behaviors, or shared biological etiologies (Smith, 2003; Wilson *et al.,* 2005).

In addition to intrapsychic processes such as perceptions of stress and personality dimensions, the role of interpersonal skills and social resources in physical health has also received substantial attention. While proposed links between interpersonal functioning and health have existed since antiquity (*e.g.,* Solomon wrote in approximately 1000 B.C. that "Pleasant words are a honeycomb, sweet to the soul and healing to the bones"; Proverbs 16:24), research on the construct of "social support" in relation to health burgeoned in the 1970s. A landmark finding from this research came from Berkman and Syme (1979), who reported that the status and extent of social relationships predicted mortality over a 9-year follow-up even after controlling for numerous sociodemographic, physical health, and health behavior variables. Subsequent research (*e.g.,* Berkman and Glass, 2000; Blazer, 1982; House, Robbins, and Metzner, 1982; Seeman, 2000) provided additional support for this finding and identified numerous conditions that moderate associations between interpersonal functioning and physical health outcomes (*e.g.,* Ben-Shlomo, Smith, Shipley, and Marmot, 1993; Ebrahim, Wannamethee, MacCullum, Walker, and Sharper, 1995).

Using such epidemiological findings to generate hypotheses about individual differences, researchers have focused on specific links between interpersonal functioning, biological processes, and disease (*e.g.,* Bovard, 1985; Cohen, 1988, 2004; Uchino, 2004). Here again, impaired social functioning characterized by negative interactions may represent a form of stress (Cohen, 2004) which impacts physical health indirectly via emotional experiences (Kiecolt-Glaser et al., 2002) and directly through physiological pathways, such as hypothalamic-pituitary-adrenal cortex (HPA) axis functioning (Bovard, 1985). In a comprehensive model of how one indicator of social functioning, loneliness, influences short- and long-term physical health, Cacioppo and colleagues (*e.g.,* Cacioppo, Hawkley, and Berntson, 2003; Hawkley and Cacioppo, 2003) proposed multiple potential pathways from loneliness to poor health outcomes. Lonely individuals may perceive higher levels of stress, react more negatively to stress, and benefit less from social interactions (Cacioppo *et al.,* 2002; Hawkley, Burleson, Berntson, and Cacioppo, 2003). It also appears that lonely individuals experience less efficient and effective sleep that may interfere with the restorative processes of sleep (Cacioppo *et al.,* 2002). Finally, evidence suggests that loneliness may impact medical care and medical decision making: Physicians reported that they provide better medical care to patients with well-established social networks as compared to patients who appear socially isolated (Cacioppo *et al.,* 2003).

In contrast to impaired social functioning, the perception that others are available to provide assistance and emotional support appears to buffer the negative effects of stress on health. That is, the presence of positive social support reduces the likelihood of negative health outcomes in the wake of stressful events (Rosengren, Orth-Gomer,

Wedel, and Wilhelmsen, 1993). Similarly, the degree to which individuals actively participate in social activities and hold numerous social roles prospectively predicts health outcomes (Cohen, Doyle, Skoner, Rabin, and Gwaltney, 1997).

Although research on psychosomatics has traditionally placed greater emphasis on isolated cognitive and social variables than on the amalgam of features that compose psychopathological syndromes (*e.g.,* depression), findings nonetheless suggest connections between psychopathology and physical health. Frequently, cognitive, behavioral, and biological theories of stress have been posited as mechanisms by which psychopathology may impact physical health (Thompson and van Loon, 2002). That is, stress may be viewed as a precipitant, associated feature, and consequence of psychopathology. Hammen's (1991) novel and provocative findings suggested that individuals with major depressive disorder (MDD) engage in behaviors that create stressful environments, such as dysfunctional mate selection (Hammen, 1999; Pettit and Joiner, 2006) and poorer interpersonal functioning (Davila, Bradbury, Cohan, and Tochluk, 1997; Potthoff, Holahan, and Joiner, 1995). Of note, experiences related to physical illness may also induce stress, such as financial strain, physical impairment or pain (Given *et al.,* 1993), and critical reactions from others (Manne, Taylor, Dougherty, and Kemeny, 1997). Once generated, stress may negatively impact physical health through biological pathways (*e.g*., via chronic activation of the HPA axis and the sympathoadrenal system) and behavioral pathways (*e.g.,* via maladaptive coping strategies such as the use of cigarette smoking as a stress reduction strategy).

Clearly, the field of psychosomatics is large and thriving, with important contributions from epidemiology, psychology, psychiatry, immunology, and endocrinology. The quantity of findings regarding specific pathways between mental health, psychosocial functioning, and physical health provides health professionals with a wealth of information, yet integration of these findings presents a daunting task. Much of the extant literature addresses differing levels of analyses (which, in our view, has strengthened the field) with differing theoretical frameworks, and at times in the absence of theory[4]. In general, most models within psychosomatics maintain that some behavior or characteristic promotes the experience of stress and negative emotionality, which in turn has a direct influence on health via physiological reactions and an indirect influence on health via its associations with other cognitive, behavioral, or social constructs. The purpose of this theoretical study (Montero and León, 2005) is to provide a broader conceptual framework for integrating diverse findings on the relations between these psychosocial constructs, psychopathology, and physical disease. We do not presume to supplant existing models and theories within psychosomatics; rather, we present a broader theoretical perspective that may be used to integrate various theories and findings at lower levels of analyses.

In the remainder of this paper, we describe our model and review selected research relevant to it. The vast literature on psychosomatics is far too extensive to review in

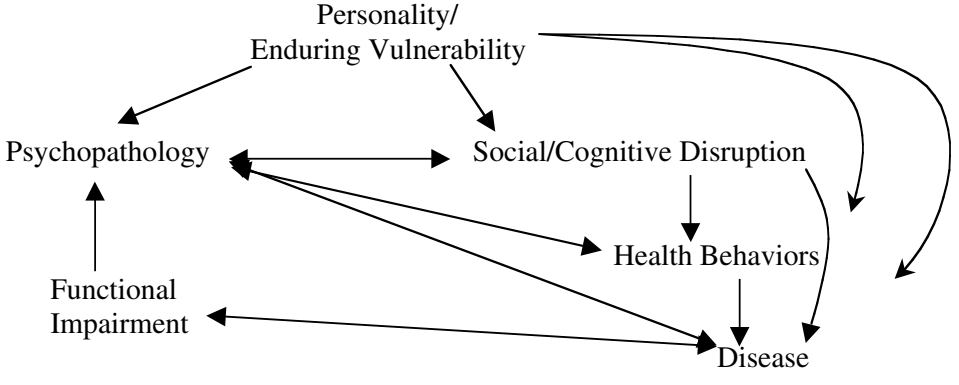
[4] Noteable exceptions to this are found in early psychoanalytic formulations of psychosomatic disorders (*e.g.,* Alexander, 1943; Dunbar, 1935; Menninger, 1949), which emphasized conversion of underlying neurotic conflict into organic disease.

any one paper. As such, we place particular emphasis on literature regarding the associations between depression, psychosocial functioning, health behaviors, and coronary heart disease (CHD). (This, too, is a large literature and our review will therefore be selective). Although we limit our focus to depression and CHD, we believe that this model may hold explanatory power for other manifestations of psychopathology and physical health, and hope that it may be used to guide future research endeavors.

An integrative model

Our proposed model of the links between psychopathology, psychosocial functioning, health behaviors, and disease is presented in Figure 1. Our model[5] draws from existing theories and research on psychosomatics, and was particularly influenced by Lewinsohn, Hoberman, Teri, and Hautzinger's (1985) integrative model of depression and Schulz, Martire, Beach, and Scheier's (2000) cascade model of mortality. Our model proposes reciprocal and bidirectional associations between psychopathology, social and cognitive disruptions, health behaviors, and disease. Psychopathology has direct and indirect paths to disease, through social/cognitive disruptions and health behaviors. Disease feeds back into psychopathology through direct biological pathways and indirectly via functional impairment. Social/cognitive disruption, health behaviors, and psychopathology exert reciprocal influences on each other, as indicated by the bi-directional arrows linking them. Personality and temperamental features represent enduring vulnerabilities with unidirectional links to other constructs in the model.

FIGURE 1. An integrative model of pathways connecting mental and physical health.



[5] This model does not specifically address the biological pathways by which psychosocial constructs may impact physical health. While biological processes ultimately mediate the effects of psychosocial factors on physical health, our model emphasizes molar processes by which psychosocial factors mutually influence physical health.

As an example of the model's application to mental-physical health overlap, consider the hypothetical case of Joe, a man who meets diagnostic criteria for MDD. During his depressive episode (Psychopathology), Joe experiences symptoms of hyperphagia, hypersomnia, and fatigue – all of which are criteria symptoms of MDD. He also ruminates, or broods, about his depressed mood (Cognitive Disruption; *e.g.,* Nolen-Hoeksema, 2000) and experiences a reduction in the quality and quantity of his social interactions (Social Disruption; *e.g.,* Pettit and Joiner, 2006). The combined effects of fatigue, brooding, and impaired social relationships lead to a reduction in his activity levels, including a cessation of regular exercise (Health Behavior; *e.g.,* Allgower, Wardle, and Steptoe, 2001). Moreover, his increased appetite and inactivity promote the establishment of poor eating habits. Over time, Joe gains approximately 20 pounds and develops Type 2 diabetes (Disease). Joe's Type 2 diabetes and obesity precipitate new difficulties in his life (Functional Impairment), including decreased work productivity, consequent lowered earnings (Lavigne, Phelps, Mushlin, and Lednar, 2003), and sexual dysfunction (De Berardis *et al.,* 2005). These difficulties exacerbate his depressive symptoms and re-initiate the mental-physical health cycle.

As illustrated in the preceding example, this model has potential utility in explaining the links between a variety of constructs relevant for mental and physical health. In the following section, we review selected research findings on depression and CHD that bear upon the proposed pathways in our model. As stated earlier, this review will necessarily be selective. Our goal is not to provide an exhaustive overview of mental-physical health associations; rather, we provide a brief review to serve as one example of how our model may be used to explain overlap between mental and physical health.

Specific application: Depression and CHD

Reciprocal paths between depression and CHD

An overwhelming amount of empirical data have accumulated to support the notion that depression increases the risk of CHD, as evidenced by the many papers, chapters, and books that extensively review this area (*e.g.,* Booth-Kewley and Friedman, 1987; Ford and Mead, 1998; Hayward, 1995; Rozanski, Blumenthal, and Kaplan, 1999; Smith and Ruiz, 2002). How might depression increase the risk of CHD? First, evidence suggests that depression may have direct physiological effects – likely on the autonomic nervous system, platelet functioning, and HPA axis — that increase risk of CHD (*e.g.,* Carney *et al.,* 1995; Evans *et al.,* 2005; Hughes and Stoney, 2000; Rozanski *et al.,* 1999; Stansfeld and Fuhrer, 2002b; Stein *et al.,* 2000). In addition to direct physiological paths from depression to CHD, it is likely that depression increases the risk of CHD indirectly through increased exposure to psychosocial stressors and through health behaviors (Smith and Ruiz, 2002; Stansfeld and Fuhrer, 2002a), paths which will be addressed in later sections of this paper.

Reflecting the reciprocal nature of depression-CHD paths, evidence also indicates that individuals who receive a diagnosis of heart disease are at prospective risk of developing (or exacerbating) depression over follow-ups of up to eight years (Hance, Carney, Freedland, and Skala, 1996; Havik and Maeland, 1990; Polsky *et al.,* 2005;

Schleifer *et al.,* 1989). Nevertheless, it is worth noting that the path from existing CHD to depression has received less empirical attention than the path from depression to CHD, and little is known about mechanisms by which CHD may promote depression. One possible explanatory pathway is that physical illness such as CHD may impact brain physiology to induce secondary depression (Lyness and Caine, 2000). As we will argue later, CHD may also influence depression via the introduction of functional impairments (*e.g.,* limitations in activities of daily living).

From the wealth of evidence linking these conditions, it can be concluded that the pathway from depression to CHD is firmly established. Less research has been conducted on the path from CHD to subsequent depression, although existing findings tend to support the notion that CHD confers risk for depression. In conjunction, these findings tentatively bear out the bidirectional path between psychopathology and disease in our model. In the following sections, we review evidence germane to specific cognitive and behavioral pathways that may link depression to CHD.

Reciprocal paths between depression and social or cognitive disruption

An abundance of empirical evidence demonstrates bi-directional relationships between depression and disruptions in social or cognitive functioning. Indeed, leading psychological models of depression place social and/or cognitive disruptions as the primary causal agents of depression. For example, Lewinsohn *et al.*'s (1985) model holds that depression is initiated and maintained by the co-occurrence of interrelated environmental and interpersonal risks that increase negative experiences and decrease positive experiences. Beck's (1967) cognitive theory of depression asserts that stressful life events activate dysfunctional automatic thoughts, irrational beliefs, and self-schemas to eventuate in depression (Ingram, Miranda, and Segal, 2006). Rehm's (1977) self-control theory of depression maintains that depression can be characterized as a series of specific deficits in self-management behavior. Under adverse circumstances, individuals with poor self-control skills (*e.g.,* stringent self-evaluation; self-punishment) are more likely to develop depression. Joiner, Coyne, and others (Joiner and Coyne, 1999; Pettit and Joiner, 2006; Segrin, 2001) have presented compelling evidence that interpersonal and social deficits both precipitate and maintain depression. In each of these models, existing depressive symptoms serve as a feedback element that sustains the cycle. That is, depression and its attendant behavioral features increase the occurrence of unpleasant life events, particularly negative interpersonal events (Hammen, 1991), which are in turned viewed through a dysfunctional cognitive lens to further strengthen depressive symptoms.

In contrast to the reciprocal risks of maladaptive psychosocial functioning and depression, evidence suggests that adaptive social and cognitive features may decrease risk of depression or promote recovery from depression. For example, numerous studies indicate that the presence of positive social support networks decreases risk of depression, although recent findings have been less supportive of the stress-buffering effect of social support (Bisschop, Kriegsman, Beekman, and Deeg, 2004; Burton, Stice, and Seeley, 2004; Stroebe, Zech, Stroebe, and Abakoumkin, 2005). Similarly, it appears that optimism and cognitive distraction from negatively-valenced stimuli may represent cognitive features that protect against depression (Giltay, Zitman, and Kromhout, 2006; McCabe and Gotlib, 1995; Nolen-Hoeksema, Morrow, and Fredrickson, 1993).

In summary, substantial empirical work indicates that social and cognitive disruptions may be viewed as causes, correlates, and consequences of depression, while positive cognitive styles and social interactions reduce the risk of depression. The combination of these findings supports a bidirectional path between psychopathology and social/cognitive disruption.

Reciprocal paths between depression and health behaviors

In addition to relations with social and cognitive disruptions, depression is also associated with both the absence of health maintenance behaviors and the presence of health risk behaviors. These likely represent indirect pathways by which depression increases the risk of CHD. For example, depressed individuals display higher rates of health risk behaviors such as cigarette smoking (Black, Zimmerman, and Coryell, 1999; Breslau, Kilbey, and Andreski, 1993; Brown, Lewinsohn, Seeley, and Wagner, 1996; Glassman *et al.,* 1990; Niles, Mori, Lamber, and Wolf, 2005), substance abuse and dependence (Hirschfeld, Hasin, Keller, Endicott, and Wunder, 1990; Lewinsohn, Rohde, Seeley, and Hops, 1991; Swendsen and Merikangas, 2000), and unhealthy dietary intake (Bonnet *et al.,* 2005; Golden *et al.,* 2004). Conversely, depressed individuals are less likely to engage in health promoting behaviors such as physical activity (Allgower *et al.,* 2001; Bonnet *et al.,* 2005; Farmer *et al.,* 1988; Hassmen, Koivula, and Uutela, 2000).

It also appears that health behaviors may influence subsequent depressive symptoms. Recent reviews (Sjosten and Kivela, 2006; Stathopoulou, Powers, Berry, Smits, and Otto, 2006) report that exercise interventions for existing depression produce strong positive effects (but also note that exercise may not protect from initial onset of depression; Cooper-Patrick, Ford, Mead, Chang, and Klag, 1997). Others (*e.g.,* Kahler *et al.,* 2002) have reported short- and long-term reductions in depressive symptoms among individuals who maintain smoking abstinence follow smoking cessation treatment. Numerous additional investigations confirm the reciprocal nature of the health behaviors-depression relation (*e.g.,* Breslau, Novak, and Kessler, 2004; Lampinen, Heikkinen, and Ruoppila, 2000; Swendsen and Merikangas, 2000).

Although most studies have found that depression continues to confer prospective risk for CHD after controlling for health behaviors, the predictive strength of depression weakens, suggesting that health behaviors account for at least some of the depression-CHD association. Furthermore, it should be noted that most studies have simultaneously assessed depressive symptoms and health behaviors. It may be that the mediating role of health behaviors in the depression-CHD link strengthens across longer temporal periods (*e.g.,* depression may gradually initiate health behavior patterns that become more entrenched over time and slowly increase the risk of CHD).

In summary, research to date on depression, health behaviors, and CHD supports bidirectional paths between psychopathology and health behaviors, as well as an indirect path from psychopathology to disease via health behaviors. While these findings are promising, additional longitudinal research is needed to further clarify the independent and overlapping effects of depression and health behaviors in the prediction of CHD.

Paths from social or cognitive disruption and from health behaviors to CHD

Substantial evidence indicates that impaired social functioning, interpersonal conflict, and social stressors increase the risk of CHD (Berkman, 1995; Knox *et al.,* 1998; Lett *et al.,* 2005; Smith and Ruiz, 2002; Stansfeld and Marmot, 2002), worsen the prognosis of existing CHD (Orth-Gomér *et al.,* 2000; Smith and Ruiz, 2002), and relate to associated risk factors for CHD (Horsten, Wamala, Vingerhoots, and Orth-Gomer, 1997; Horsten, Mittleman, Wamala, Schenk-Gustafsson, and Orth-Gomer, 1999; Lepore *et al.,* 2006). In addition to social impairment, other forms of stress such as job strain, chronic work stress, and high family demands increase the risk of CHD and its attendant features (Chandola, Brunner, and Marmot, 2006; Haynes and Feinleib, 1980; Karasek and Theorell, 1990; Vitaliano, Young, and Zhang, 2004). Similarly, fewer socioeconomic resources and lower educational attainment associate with greater risk of CHD in developed countries (*e.g.,* Hemingway, Shipley, McFarlane, and Marmot, 2000; Kaplan and Keil, 1993; Thurston, Kubzansky, Kawachi, and Berkman, 2006). Conversely, indicators of positive psychosocial functioning such as the perception of supportive social networks appear to protect against CHD (Erikson, 1994; Stansfeld and Fuhrer, 2002b).

Cognitive processes also have demonstrated links to CHD. To clarify, we are not referring to cognitive disorders (*e.g.,* dementia), but rather to more normative –yet at times maladaptive— ways of perceiving and interpreting oneself, others, and life circumstances. For example, the cognitive features of hopelessness and low perceptions of control have been shown to predict cardiac events (Barefoot *et al.,* 2000; Everson *et al.,* 1996; Johnson, Stewart, Hall, Fredlund, and Theorell, 1996; Kubzansky, Davidson, and Rozanski, 2005). In contrast, positive cognitive styles such as optimism and high internal locus of control have been connected to a decreased risk of developing CHD (Kubzansky, Sparrow, Vokonas, and Kawachi, 2001; Rosengren *et al.,* 2004; Sheppard, Maroto, and Pbert, 1996) and a better prognosis following cardiac surgery (Helgeson, 2003; Scheier *et al.,* 2003; Shen, McCreary, and Myers, 2004).

The accumulation of evidence, therefore, substantiates paths from social functioning and cognitive styles to CHD. This risk is likely conferred through direct physiological pathways and indirect behavioral pathways. Kop (1999) delineated a model of how these factors may increase risk of CHD via physiological pathways. Although the physiological mechanisms are likely complex, reduced cardiovascular reactivity and hypercortisolemia appear to be direct physiological effects of stress and social impairment that may promote CHD (Uchino, Cacioppo, and Kiecolt-Glaser 1996). In addition to direct physiological effects, it is likely that social and cognitive disturbances also have indirect paths to CHD via health behaviors (Steptoe and Willemsen, 2002). Indeed, it appears that indicators of social disruption (*e.g.,* life stress, impaired social functioning, and impaired academic or work functioning) may promote health risk behaviors such as smoking, fatty dietary intake, excessive alcohol consumption, and physical inactivity (Rozanski *et al.,* 1999; Treiber *et al.,* 1991; although exceptions exist: Cacioppo *et al.,* 2002), which in turn increase the risk of CHD. Likewise, lower socioeconomic status is associated with higher levels of health risk behaviors (Winkleby, Fortmann, and Barrett, 1990). In contrast, the presence of adaptive psychosocial and cognitive functioning (*e.g.,* available social support, high self-efficacy, perceived behavioral control) may

operate as a protective factor because it appears to facilitate health maintenance behaviors such as exercise and healthy dietary intake (Cohen, 1988; Johnston, Johnston, Pollard, Kinmonth, and Mant, 2004; Moore *et al.,* 2006; but for contradictory evidence, see Cacioppo et al., 2002). Conceivably, the higher incidence of health risk behaviors and the absence of health maintenance behaviors among individuals who experience psychosocial impairment may partially account for links between psychosocial functioning and CHD. Nonetheless, it is worth noting that psychosocial factors still predict CHD even after controlling for health risk behaviors like smoking (*e.g.,* Marmot, Shipley, and Rose, 1984).

Findings therefore support a direct path from social/cognitive disruption to disease and are generally supportive of an indirect path from social/cognitive disruption to disease via health behaviors. As is the case with the path from depression →health behaviors← CHD, additional research is necessary to more fully understand the circumstances under which health behaviors may mediate the long-term connections between psychosocial functioning and CHD.

Reciprocal paths between CHD and functional impairment

Thus far, we have reviewed evidence supporting bidirectional paths between (a) depression and CHD, (b) depression and social/cognitive disruption, and (c) depression and health behaviors. We have also reviewed evidence suggesting paths from psychosocial disruption and health behaviors to CHD. After CHD is established, how does it relate to other variables in the model? That is, aside from direct physiological paths from CHD to other psychosocial constructs, are there indirect paths by which CHD influences psychological well-being?

As displayed in the model, we argue that functional impairment resulting from CHD serves as one psychosocial mechanism linking CHD to depression. Evidence indicates that CHD and functional impairment influence each other in a bi-directional manner. For example, older adults with CHD experience greater functional impairment and greater difficulties with activities of daily living than their same age peers (Bild *et al.,* 1993; Burnette, Mui, and Zodikoff, 2004). Moreover, CHD prospectively predicts decreases in health-related quality of life, functional declines in activities of daily living, and decreased work productivity (Brenner, 1987; Brown *et al.,* 2000; Gregg *et al.,* 2002; Lamb *et al.,* 2006; Liu, Maniadakis, Gray, and Rayner, 2002; Spiers *et al.,* 2005; Wang, van Belle, Kukull, and Larson, 2002). Additionally, a recent study suggests that the metabolic syndrome, a potential precursor to CHD, represents a unique and strong predictor of declines in mobility (Blazer, Hybels, and Fillenbaum, 2006). Conversely, declines in mobility, functional impairment, and lowered activity levels increase the risk for subsequent CHD (Williams *et al.,* 2002).

Hence, the path from disease (*i.e.,* CHD) to functional impairment appears to be well-established, at least among older adults. The impact of CHD on functional status among younger and middle-aged adults is less clear. Moreover, although some evidence indicates that declines in mobility and activities enhance risk of CHD (*e.g.,* Williams *et al.,* 2002), additional research is needed to validate the path from functional impairment to disease.

Path from functional impairment to depression

Once functional impairments are present, they appear to increase the risk for the development or exacerbation of depression. For instance, Zeiss, Lewinsohn, Rohde, and Seeley (1996) found that declines in mobility and other areas of daily functioning enhanced the risk of depression (see also Lewinsohn, Seeley, Hibbard, Rohde, and Sack, 1996). Consistent with that finding, Steffens *et al.* (1999) reported that among older adults with a history of CHD, functional disabilities positively associated with depressive symptoms (see also Williams *et al.,* 2002). The directionality of the effect, however, was unclear. That is, it may be that (a) CHD patients who are depressed are more likely to develop functional impairments, that (b) CHD patients with greater functional impairments are more likely to become depressed, or (c) a combination of both (a) and (b). Therefore, while the path from functional impairment to psychopathology appears valid, it is not yet clear whether disease indirectly predicts depression via functional impairment. Further longitudinal research is needed to directly examine that question.

Personality as a higher-order risk factor

In our model, personality and temperament are framed as enduring dispositions that set the stage for the other components of the mental-physical health cycle. A large body of research supports relations between personality variables such as hostility, anger, and neuroticism/negative affectivity as predictors of CHD (for reviews, see Booth-Kewley and Friedman, 1987; Eysenck, 2000; Friedman and Booth-Kewley, 1987; Krantz and McCeney, 2002; Scheier and Bridges, 1995; Suls and Bunde, 2005). These personality dispositions may directly increase risk of CHD via exacerbation of biological and physiological processes underlying cardiopathogenesis, such as increased HPA activation and inflammation (Smith and Ruiz, 2002; Suarez, 2003; Suls and Bunde, 2005).

As reflected in our model, personality variables may also increase the risk of CHD via their influence on depression, social and cognitive functioning, and health behaviors. Evidence suggests that hostility and negative affectivity prospectively predict the development of depression (Brummett et al., 1998; Reinherz, Giaconia, Hauf, Wasserman, and Silverman, 1999; Steunenberg, Beekman, Deeg, and Kerkhof, 2006; Zuroff, Mongrain, and Santor, 2004). Individuals high in hostility and neuroticism also tend to experience higher levels of stressful life events and report lower levels of social support (*cf.* social disruption; Brummett *et al.,* 1998; Friedman, 2000; Kendler *et al.,* 2003; Smith and Frohm, 1985; Suls, 2001). Furthermore, individuals high in anger and negative affectivity are more likely to engage in health risk behaviors such as cigarette smoking (Kassel, Stroud, and Paronis, 2003; Scherwitz *et al.,* 1992) and less likely to engage in health maintenance behaviors such as exercise (De Moor, Beem, Stubbe, Boomsma, and De Geus, 2006; Hassmen *et al.,* 2000).

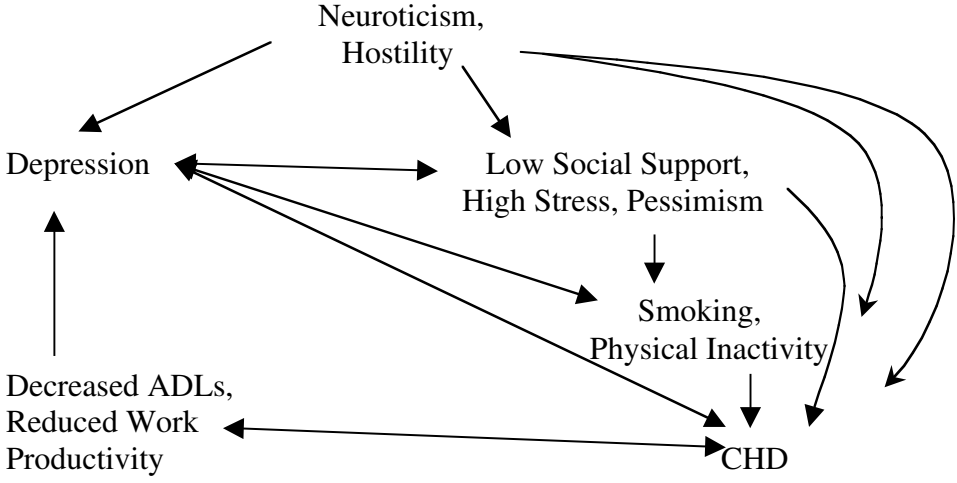
While it may be suggested that psychopathology and other psychosocial variables simply represent proximal mechanisms by which personality distally predicts health outcomes, it is important to note that the limited evidence to date is not consistent with that notion. To wit, psychosocial variables such as social integration remain significant predictors of health outcomes after controlling for personality variables such as extraversion

(Cohen *et al.,* 1997, 1998). More research incorporating other personality dimensions and psychosocial variables needs to be conducted before drawing firm conclusions, but it appears as though psychosocial variables and personality dimensions have both overlapping and independent effects on physical health outcomes (Uchino *et al.,* 1996).

Summary and conclusion

As demonstrated from this brief and admittedly selective review of literature on the associations between depression and CHD, the integrative model of mental-physical health is largely consistent with existing research on specific pathways. To summarize how the extant literature on depression-CHD may fit within the framework of the model, Figure 2 provides examples of each of the model's constructs and the pathways linking them. The complete model displayed in Figure 2 has not yet been subjected to empirical testing, but the individual constructs and pathways are selected from existing findings that have been reviewed in this paper. Additional research will be necessary to further validate certain pathways, particularly those involving functional impairment and those involving mediation or moderation effects. It is also important to recognize the existence of contradictory findings –*i.e.,* those that do not support pathways proposed in our model. On the whole, however, the majority of research to date on depression-CHD links accords with our theoretical model.

FIGURE 2. Pathways connecting depression and coronary heart disease.



NOTE. ADLs: Activities of Daily Living.

We selected depression-CHD for our review because it is among the most thoroughly-researched areas in the field of psychosomatics. A consistent theme that emerges in the depression-CHD literature, and in the field of psychosomatics in general, is that psychosocial constructs related to the persistent experiences of negative emotions have detrimental physical consequences, both through direct physiological mechanisms and through their associations with other psychosocial variables and health behaviors. Whether they occur through personality variables – perhaps even a "disease-prone personality" (Friedman and Booth-Kewley, 1987), syndromal levels of psychopathology (Rozanski *et al.,* 1999), stressful events (*e.g.,* Selye, 1950), impaired interpersonal functioning (Uchino *et al.,* 1996), cognitive styles (Kubzansky *et al.,* 2001), or some other psychosocial construct, the common risk features appear to be chronic negative emotions and maladaptive health behavior patterns. The goal of the present paper has been to display how these behavior patterns and various psychosocial constructs that tap into negative emotions interrelate in a meaningful fashion to promote physical disease.

Although other areas of psychosomatics (*i.e.,* other forms of psychopathology and physical disease manifestations) have received less empirical attention than depression and CHD, we believe that the paradigm presented in this paper will provide a useful framework for integrating diverse findings on mental-physical health overlap. This model may also be used as a conceptual framework to guide research on the relations between psychopathology and physical health, and to identify areas in need of more extensive research. Indeed, two challenges facing the field of psychosomatics are to (a) explore paths depicted in this model in the context of a broad array of psychosocial and physical health constructs and (b) to integrate such findings in a comprehensive, meaningful way. We are hopeful that this model may provide direction in addressing these challenges and that accumulation of empirical findings on these pathways will be used to refine the model as needed.

Finally, we wish to highlight potential prevention and treatment implications of this model. We did not explicitly include treatment studies in our review of depression and CHD, but to the extent that psychosocial constructs and disease relate in reciprocal fashion, it may be possible to reduce the risk for physical disease by intervening at any of the pathways. Clearly, reducing health risk behaviors like smoking and increasing health maintenance behaviors like physical activity is expected to produce superior physical health outcomes. It is interesting to note, however, that such health behavior interventions are also likely to reduce psychopathology (*e.g.,* Kahler *et al.,* 2002; Stathopoulou *et al.,* 2006), and that interventions designed to reduce psychopathology may also improve health behaviors (*e.g.,* Buchanan, Gardenswartz, and Seligman, 1999; McFall *et al.,* 2005).

While interventions aimed at any one pathway will likely indirectly impact other pathways in the model, more comprehensive treatment approaches directly target multiple paths. Examples of comprehensive treatment approaches include combined cognitive-behavior therapy (CBT) for depression and smoking cessation (*e.g.,* Brown *et al.,* 2001), CBT plus nutritional and physical activity programs for obese patients with binge eating disorder (*e.g.,* Fossati *et al.,* 2004), and multidisciplinary pain programs aimed at improving physical, psychological, and social functioning (*e.g.,* Jensen, Turner,

and Romano, 1994). Such approaches may treat existing symptoms of psychopathology (*e.g.,* depression, anxiety) while also developing appropriate health behavior strategies (*e.g.,* increased exercise) and psychosocial coping strategies (*e.g.,* relaxation training). Combination of treatment approaches necessarily varies across individual cases; the emphasis, however, is to identify and target vulnerabilities that may be present at each of the constructs represented in the model. Continued empirical research will be necessary to validate such approaches, but consistent with the reciprocal paths in the model, we anticipate that improvement in any one domain of functioning would likely engender a cascade of improvements in other domains.

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