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Psychosocial Factors in Coronary Heart Disease

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Cardiovascular disease represents the leading cause of death globally, which includes mortality due to stroke and coronary heart disease (CHD); of these two forms of cardiovascular disease, CHD accounts for more deaths annually (World Health Organization, 2015). The primary features of CHD include plaque development in the coronary arteries (atherosclerosis), heart attack (myocardial infarct), and acute chest pain (angina; Labarthe, 1998). The traditional risk factors for CHD include age, obesity, high cholesterol, high blood pressure, inactive lifestyle, smoking, excessive alcohol consumption, and family history of the disease (World Heart Federation, 2015). Epidemiologic evidence suggests that traditional risk factors of CHD may account for 58–75% of new cases (Beaglehole & Magnus, 2002). Other predictors of CHD may include stress-related psychosocial factors at a person level (e.g., dispositional hostility and depression) and/or environmental level (e.g., chronic work-related stress and lack of social support; Albus, 2010).

What Is a Psychosocial Risk Factor?

Simply stated, a psychosocial risk factor should be conceptualized as a psychological attribute that impacts social behaviors in a way that elevates likelihood of illness, such as emotion dispositions of anger that increase antagonistic social interactions, thereby leading to exacerbated stress levels and social isolation, eventually giving rise to disease onset. The impact of psychosocial risk factors on illness onset is often both direct (e.g., stress-induced elevations in markers of inflammation that hasten atherosclerotic plaque formation) and indirect (e.g., stress-induced changes in lifestyle behaviors that contribute to unhealthy diet, smoking, and excessive alcohol consumption as maladaptive means of coping). When studying psychosocial risk factors for CHD, researchers must measure and control for the presence of traditional risk factors in their

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statistical models to effectively be able to ascertain that the psychosocial risk factors in question are predictive of CHD above and beyond the traditional risk factors aforementioned.

Early Research on the Type A Behavior Pattern and Trait Hostility

In the 1950s, cardiologists Friedman and Rosenman began to take notice that a pattern of personality characteristics seemed to typify their CHD patients. These physicians termed the personality style as the Type A behavior (TAB) pattern, characterized by a strong sense of time urgency, hard-driving competitiveness, ambitiousness, hostility, and tendencies to experience anger and become aggressive. They conducted a wide-scale assessment of TAB entitled the Western Collaborative Group Study (WCGS) to determine if a reliable relationship exists between this set of personality traits and the severity of coronary dysfunction; 3,154 men aged 39–59 were administered the structured interview to assess TAB and then followed for 8.5 years (Rosenman et al., 1975). Results indicated that Type A individuals were more than twice as likely to subsequently develop clinically significant CHD symptoms than Type B (essentially calm demeanor, even tempered) counterparts.

However, the Multiple Risk Factor Intervention Trial (MRFIT) called the TAB construct into question by failing to replicate WCGS results. Reliable differences in the administration and coding of the structured interview to assess TAB have been reported between WCGS and MRFIT, which may account for these discrepant results (Sherwitz & Brand, 1990). Further, the multifaceted nature of the Type A construct may increase the probability of inconsistencies in the literature. Importantly, subsequent analyses of data from WCGS and MRFIT indicated that the structured interview-derived subscale score entitled "potential for hostility," an interactive style of expressive speech and experience of anger, was a better predictor of CHD in both datasets than the global Type A score (Dembroski, MacDougall, Costa, & Grandits, 1989; Hecker, Chesney, Black, & Frautschi, 1988). Therefore, the toxic elements of TAB appear to concern a particular interactive style separate from the qualities of ambitiousness, competition, and time urgency.

More recently, evidence suggests a reliable association between trait hostility/anger and CHD outcomes. For example, a meta-analytic report across 21 articles longitudinally examining 71,606 initially healthy participants and 18 articles longitudinally studying 8,120 CHD patients found that elevated ratings of anger/hostility were linked to an average 20% increase in disease risk, in terms of risk of eventual diagnosis amid initially healthy participants and risk of additional coronary events/CHD mortality for the patient samples (Chida & Steptoe, 2009). Interestingly, the risk profile was more pronounced amid the latter samples of participants already diagnosed with CHD, suggesting that the mere diagnosis and experience of CHD as a condition may promote bouts of anger and hostility in a way that contributes to poorer prognosis. Specifically, anger/hostility ratings predicted a 24% increased risk of additional coronary events/CHD mortality amid patients compared with a 19% increased risk of CHD diagnosis amid initially healthy participants.

Currently, the hostility construct remains a point of controversy as a risk factor for the development of CHD, yielding a mixture of results claiming significant and insignificant associations. A clear problem in the literature that may explain many of these discrepancies across studies concerns the multidimensionality of the hostility construct (cognitive mistrustfulness, emotional anger, and behavioral aggression). This problem is compounded by variability in measurement instruments: some assessment tools for measuring hostility are more reliably predictive of disease onset than others (e.g., cynical mistrustfulness and the experience of

anger versus anger expression through aggression), a quality that adds confusion to the medical science literature.

Depression

Clinical depression is diagnosed based upon criteria specified in the Diagnostic and Statistical Manual published by the American Psychological Association, including such symptoms as depressed mood, feelings of worthlessness, fatigue, changes in sleep patterns, and suicidal ideation. The relationship between clinical depression and CHD diagnosis/mortality is more reliable and robust than the associations apparent for hostility/TAB. For example, a recent meta-analysis across 30 prospective cohort studies including a total of 893,850 participants indicated that depression was associated with a 30% increased risk of CHD diagnosis (Gan et al., 2014). Similar to the hostility construct, the timeframe of depression onset has been found to be meaningfully related to CHD prognosis: patients who experienced recurrent–persistent depression pre-CHD diagnosis and maintained depression levels post-CHD diagnosis were 1.5–2 times more likely to be re-hospitalized for a coronary event or experience coronary mortality, respectively, than their non-depressed counterparts (Leung et al., 2012). These findings indicate that clinically depressed adults are at an elevated risk for CHD diagnosis and that they furthermore exhibit poorer CHD prognoses compounded by a higher mortality risk due to coronary complications.

Environmental Factors: Work Stress

A long-standing history of research has illustrated work-related stress to contribute to disease outcomes, ranging the full gamut from acute bouts of insomnia to chronic medical conditions, such as CHD (Ganster & Rosen, 2013). A variety of theoretical orientations have been offered to organize research assessments, including the job strain model, the effort–reward imbalance model, and the organizational injustice model (e.g., Kivimäki et al., 2006). The job strain model proposes the demand–control hypothesis that occupations characterized as high demand–low control represent a toxic combination in terms of giving rise to chronically elevated stress levels that degrade immune function and lead to adverse health outcomes. In short, this model stipulates that employees who are burdened with oftentimes unpredictable waves of high volume responsibilities (elevated demand) without appropriate decision latitude for managing the workload (poor control) are at risk for work-related illness and eventual burnout. Along similar lines, the effort–reward imbalance model stipulates that work environments whereby employees are disproportionately challenged relative to compensation and recognition are likewise at risk for stress-induced illness. Finally, the organizational injustice model argues that stress-induced illnesses may arise in workplaces whenever employees reliably report being treated unfairly by their supervisors. Each model grapples with different ways by which workers may experience heightened stress levels on a daily basis.

Evidence supports all three models of conceptualizing the work stress–CHD relationship, as indicated by a meta-analytic report of 14 prospective cohort studies involving 83,014 employees, whereby elevations in work stress predicted an average 50% increase in CHD diagnosis/mortality (Kivimäki et al., 2006). Although all three models were supported in this meta-analysis, the evidence was significantly stronger in support of the effort–reward imbalance and organizational injustice models relative to the job strain model, after controlling for

the variance pertaining to traditional CHD risk factors. Irrespective, there is a clear and strong pattern of evidence that elevated stress in the workplace predicts adverse coronary outcomes over time.

Environmental Factors: Social Support

Having the opportunity to discuss stressful life experiences with a trusted confidant can be helpful in reducing emotional strain and promoting problem-focused coping, envisioning potential resolutions to the circumstances underlying the challenge/threat in question. Social support resources elicit the tendency to perceive that a weight has been lifted from one's shoulders through self-disclosure, thereby enhancing the sensation of self-efficacy (ability to effectively handle the stressful situation) and reducing the maladaptive tendency of catastrophizing (rumination of difficulties that leads to perceived hopelessness—common when people feel overwhelmed by their life stressors and alone in their path of coping). If chronically elevated stress levels increases risk of CHD diagnosis/mortality, it stands to reason that stress-reducing social environments would confer protective benefit.

Sources of social support are typically defined in the health sciences literature in terms of being "functional" or "structural" in scope. Types of functional support include the following categories: emotional, informational (e.g., receiving advice), financial, appraisal (someone helping to evaluate a situation), and instrumental (e.g., receiving assistance in completing a project), whereas structural support pertains to the number of individuals in one's social network in terms of marital status, proximity of friends and family, community memberships, and the frequency of social contacts (e.g., number of times visiting with friends per month).

The pattern of evidence in both etiologic (initially healthy samples) and prognostic (samples of CHD patients) longitudinal studies has been significant for functional social support but insignificant for structural social support, whereby low functional support predicted elevated relative risk of heart attack amid initially healthy samples and, in particular, a 59% increased risk of mortality amid CHD patients after controlling for traditional risk factors in a meta-analysis (Barth, Schneider, & von Känel, 2010). These findings highlight the prominence of functional social support with regard to the quality of supportive social interactions, rather than the structural quantity of social interactions, when predicting health outcomes.

Environmental Factors: Socioeconomic Status

Typically assessed by annual income and/or years of education, socioeconomic status (SES) represents an environmental attribute worthy of consideration as a psychosocial risk factor for CHD. There are a variety of putative mechanisms that may connect low SES to CHD outcomes, such as indirect effects of inadequate resources to maintain a healthy lifestyle and direct effects of stress-induced influences on coronary dysfunction. For example, individuals of a lower class standing may consume a cheaper, highly processed diet conducive to the development of obesity, type 2 diabetes, hypertension, and elevated cholesterol. Likewise, the economic burden of poverty often compels individuals to work two to three jobs to make ends meet, thereby exacerbating perceptual stress levels as a function of workload and reducing time spent with family and friends—effectively degrading social support resources for stress management. Thus, lower SES tends to co-occur with elevated work stress and poor social support, eliciting potential synergistic effects on disease outcome, suggesting a need for

theoretical orientations designed to guide research investigations involving psychosocial risk factors for CHD morbidity and mortality.

Meta-analytic evidence involving 70 studies has indicated significant effects across income, occupation, and education whereby individuals in the lowest socioeconomic positions exhibited elevated risk of incident heart attack relative to their more affluent counterparts, with the effects most apparent in terms of income (a staggering 71% increase in relative risk of heart attack; Manrique-Garcia, Sidorchuk, Hallqvist, & Moraldi, 2011). Further, prospective evidence following 66,500 initially healthy adults across 8 years found low SES predicted CHD mortality after controlling for traditional risk factors. SES was measured in terms of three occupational classes (professional/managerial, skilled manual/non-manual workers, and semi-routine/unskilled workers) with each step down from professional/managerial associated with a 24% increase in CHD mortality (Lazzarino, Hamer, Stamatakis, & Steptoe, 2013). Moreover, a synergistic effect was observed whereby psychological distress augmented the adverse effects of low SES: the combination of low SES and high psychological distress was linked to a 33% increase in CHD mortality relative to high SES and low psychological distress counterparts (Lazzarino et al., 2013).

Piecing Together the Puzzle: Integrative Theoretical Frameworks

The evidence to date reveals that a variety of psychosocial risk factors predict elevated CHD morbidities and mortalities to a varying degree after controlling for the presence of traditional risk factors, including dispositional factors such as hostility and depression (e.g., Chida & Steptoe, 2009; Gan et al., 2014) and environmental factors such as occupational strain and low social support (e.g., Barth et al., 2010; Kivimäki et al., 2006). However, emergent findings increasingly show that these psychosocial risk factors do not exist in a vacuum, but rather dynamically interact with one another, thereby requiring more comprehensive theoretical models to guide future research. For example, the transactional model of hostility stipulates that the cynical mistrustfulness associated with this disposition serves as a driving force to create a social environment conducive to antagonistic behaviors that degrade social support resources and exacerbates emotional strain (e.g., Vella, Kamarck, Flory, & Manuck, 2012). Finally, the reserve capacity model was designed to explain associations between SES and health outcomes, whereby lower SES directly increases perceptual stress levels while simultaneously reducing social support resources for coping (the reserve capacity); in turn, both of these SES-induced developments of increased stress and decreased coping reserve are thought to directly elevate daily negative emotions/cognitions and reduce positive emotions/cognitions, collectively altering physiological processes that underlie CHD risk (Gallo & Matthews, 2003).

Numerous issues pervade this literature and are worthy of mention as limitations for consideration to improve subsequent research. First, whether dispositional or environmental in scope, all psychosocial risk factors reviewed here feature the same common denominator of elevated stress levels that may impact upon health status via direct and indirect pathways. As such, researchers should focus their attention on incorporating standardized measures of perceptual stress levels in concert with the psychosocial risk factors in question to disentangle the pathways leading to CHD outcome. Second, most of these psychosocial risk factors are multifaceted in scope, suggesting a need to concentrate efforts on measurement issues to reliably delineate the relative degrees to which psychosocial risk factor attributes predict the development and prognosis of CHD. Finally, just as it is known that functional social support may

improve one's ability to effectively cope with stressful life experiences, reducing risk of CHD morbidity and mortality, researchers should also counterbalance their investigations by incorporating other psychosocial "protective" factors that may confer significant health benefit as part of their theoretical models (e.g., frequency of positive emotions and trait optimism). Future research on psychosocial risk factors of CHD should make targeted efforts to ascertain interactive pathways that include traditional risk factors to maximally predict health outcomes and enhance scientific understanding of these provocative associations.

Author Biography

Dr. Elizabeth J. Vella is an associate professor of psychology at the University of Southern Maine. Her research interests include the link between psychosocial factors and cardiovascular risk and the physiological mechanisms that may explain these associations, as well as the implications for stress management interventions in improving quality of life and reducing physiological responses to stressors among at risk populations. She has authored numerous articles in scientific journals and presented her research at academic conferences.

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