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New Developments in Understanding Cardiovascular Disease and the Implications for Social Work

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Abstract

Cardiovascular disease is now viewed as an inflammatory disease. An index of chronic inflammation (viz., C-Reactive Protein) is as good a predictor of heart attacks as are fats in blood. The data suggest that stressful events are so closely associated with chronic inflammatory states, that the body's stress response can be viewed as an inflammatory state. This paper summarizes and explains the link between stress and cardiovascular disease. Negative health outcomes, particularly for cardiovascular diseases, are higher among those of lower socio-economic status. Differential stress among socio-economic tiers is considered as an explanation for the disparities. The literature linking cardiovascular risk factors to the stressors of workplace unfairness and lack of control over working conditions is reviewed. The role of the stressor of racism in explaining the higher rates of cardiovascular mortality in African Americans is discussed. Finally, for societies with wider gaps in income between the rich and the poor, increased stress is explored as a possible explanation for the diminished health outcomes found across all socio-economic tiers. The implications for social work direct practice and macro-practice are considered.

New Developments in Understanding Cardiovascular Disease and the Implications for Social Work

Cardiovascular disease is the cause of 50% of deaths in modern western society (Ross, 1999). In approximately half of the cases, the first indication of disease is a heart attack (Blumenthal et al., 1997). A decade ago, those who were concerned about development of cardiovascular disease checked cholesterol and triglyceride levels and watched people's diets. While high levels of fat particles are still important risk factors, the new awareness is that cardiovascular disease is as much about the immune system and inflammation as it is about fat metabolism. Currently, levels of C-Reactive Protein (CRP), an indicator of an on-going inflammatory response somewhere in the body, are viewed as equally important prognostic indicators of cardiovascular disease (Ridker, Cushman, Stampfer, Tracy, & Henneckens, 1998; Ridker, Hennekens, Buring, & Rifai, 2000; Ridker, Rifai, Stampfer, & Hennekens, 2000).

Heart attacks (diminished blood flow to the heart muscle such that the muscle is damaged) are most likely to occur in a person whose arteries have been narrowed by the deposition of plaque on the wall of the artery. The narrowed lumen of the artery impedes blood flow. The process of narrowing the lumen of the blood vessel (called arteriosclerosis or atheroslerosis) is now better understood. Activated white blood cells, whose activation occurs as part of an inflammatory process, play a primary role in the development or arteriosclerosis. Normally, the job of white blood cells is to attack foreign particles in the body. Unaltered fat particles are not seen as foreign to the white blood cells. However, fat particles can be modified. Sometimes they get studded with sugars, as occurs in diabetic patients who have higher levels of

glucose in their blood. Sometimes, fat particles get oxidized such that they become targets for white blood cells. When a white cell detects a foreign appearing fat particle, it responds as it would to any foreigner in the blood stream. It engulfs and incorporates the fat particle and, in the process, becomes a foam cell. Foam cells comprise the plaque that clogs the artery (Black, 2002; Libby, Ridker, & Maseri, 2002).

While psychological stress can dampen some arms of the immune system, it greatly enhances inflammatory responses (Herbert & Cohen, 1993). When an animal is stressed, e.g., by forced restraint or foot-shock, interleukin 6 is released by white blood cells and possibly by neurons. IL-6 will induce the liver to release inflammatory mediators (e.g., C-reactive protein) (Dhabhar, Miller, McEwen, & Spencer, 1996; Herbert & Cohen, 1993). Indeed, Maier and Watkins (1998) argue that during evolutionary development, the immune response designed to fight external pathogens was recruited to respond to stressors outside the body. Much empirical support exists for Maier and Watkins' view of the stress response being equivalent to an inflammatory response. Animals exposed to restraint stress or uncontrollable shock display enhanced plasma levels of many inflammatory mediators (Dhabhar et al., 1996; Takaki, Huang, Somogyvari-Vigh, & Arimura, 1994; Zhou, Kusnecov, Shurin, DePaoli, & Rabin, 1996). In response to psychic stress, humans exhibit fever and increases in IL-6, a response equivalent to the response made to a bacterium (Wachulec, Peloso, & Satinoff, 1997). Higher levels of IL-6 distinguish individuals under stress such as Alzheimer patient caregivers (Kiecolt-Glaser et al., 2003). Among the obese, major depression is associated with higher levels of IL-6 and C-reactive protein (Ladwig, Marten-Mittag, Löwell, Döring, & Koenig, 2003). Interleukin-6, is positively correlated with depression, anger, and hostility among those who do not use

multivitamins (Suarez, 2003).

Elucidating the pathways through which inflammation contributes to arteriosclerosis has shed light on why obesity is a risk factor for diabetes and cardiovascular disease. It turns out that obesity is essentially an inflammatory state. Many of the cells in adipose tissue around the center of the body (omental fat) are white blood cells. Most cells in adipose tissue are activated and spewing out inflammatory mediators such as IL-6, and TNF-alpha (Fried, Bunkin, & Greenberg, 1998; Hotamisligil, Arner, Caro, Atkinson, & Spiegelman, 1995). TNF-alpha creates insulin resistance (González, Minium, Rote, & Kirwan, 2006). Thus, the development of non-insulin dependent, Type II diabetes is increased. The IL-6, which induces production of CRP from the liver, explains the increased risk for arteriosclerosis.

The fact that stress is an inflammatory process suggests that stress might play a role in arteriosclerosis. In fact, over 40% of individuals exhibiting cardiovascular disease do not exhibit any of the lipid risk factors and stress may constitute the sole risk factor (Black, 2002). In prospective studies, depression is a predictor of subsequent coronary artery disease (Anda, Williamson, & Jones, 1993; Musselman, Evans, & Nemeroff, 1998). Women in distressed marriages exhibit more calcium deposition on blood vessel walls, a measure of the progression of arteriosclerosis (Gallo et al., 2003). In the primate literature, social disruptions results in development of arteriosclerosis (Kaplan et al., 1996).

In this paper, the term stress has been used to refer to events (e.g., foot shock, caring for a relative with Alzheimer's disease, marital disputes) that create physiological disturbance in most organisms and are subjectively regarded as unpleasant. Of course, particular events that cause physiological disturbance and subjective discomfort in most persons fail to create disturbance in

some people. In between the event and the physiological and psychological responses is the person's appraisal of the event and the person's coping mechanisms. Personality encompasses how a person appraises the stressor, the emotional response the person is likely to have to the stressor (e.g., anger, sadness, fear), and how the individual copes. Persons with particular personality styles are also more likely to experience stressors in their lives.

It has long been recognized that particular personality traits increase the risk of cardiovascular disease. Hostility and high levels of anger, whether expressed or suppressed, is a risk factor for cardiovascular disease (Speilberger, 1999). D-personality, characterized by distressed affect and social inhibition, also increases the risk for cardiovascular disease (Denollet, Vaes, & Brutsaert, 2000). Major depression is a risk factor for cardiovascular disease (Frasure-Smith & Lesperance, 2005). The recognition that stress-induced emotions (anger, distress, frustration) are associated with higher levels of inflammatory mediators explains the connection.

The Link between Stress and Heart Attacks in Persons with Arteriosclerosis

While constricted vasculature can diminish oxygen supply to critical areas, heart attacks and strokes are usually caused by a second process. The fatty depositions in the wall of the vasculature can build up scar tissue over the top but under this scar tissue is a fatty base which is vulnerable to rupture. If the fatty plaque ruptures, a tear in the vasculature is produced. Platelets (cellular fragments that produce clots) rush in to repair the rent in the wall of the vasculature. The platelets can gather around so rapidly that a clot develops that further prevents the flow of blood through the vessel. The size of the clot that develops depends upon the concentrations of various blood clotting factors and clot dissolving factors in the blood (Toffler & Kelly, 2000).

Negative emotions are associated with higher levels of pro-coagulation proteins and

lower levels of proteins that dissolve clots (von Känel, Dimsdale, Patterson, & Grant, 2003). This may explain why negative emotions and stressful episodes at work often precede heart attacks (Mittleman et al., 1995; Möller, Theorell, de Faire, Ahlbom, & Hallqvist, 2005). Moreover, in those with diagnosed cardiovascular disease, major depression is a risk factor for heart attacks (Carney, Freeland, Veith, 2005). In exploring the molecular mediators connecting negative emotion and acute cardiac events, Strike et al. (2006) found that after stress provocation in the laboratory, clotting factors and platelet clot formation were elevated to a greater degree for those whose heart attacks had been precipitated by emotional events.

Efficacious Interventions to Reduce Risk Factors

Interventions for reducing negative outcomes in those with cardiovascular disease have been studied. Some of these interventions are directed at the personality factors which exacerbate response to stressors. In patients with myocardial ischemia, those receiving psychotherapy not only displayed reduced distress and hostility, but also were less likely to experience a cardiac event during the 38 month follow-up period (Blumenthal et al., 1997). Group interventions for reducing hostility have been designed for those individuals scoring high on a hostility scale. An intervention was successful in reducing hostility and blood pressure relative to those in a control group (Gidron, Davidson, & Bata, 1999). Other interventions may influence an individual's psychological and physiological response to a stressful event. Regular exercise can not only reduce depression in cardiovascular patients but also improves coronary artery disease outcomes as well (Lett et al., 2004). Statin medications reduce both CRP and Low Density Lipoproteins (Mora & Ridker, 2006).

Does Stress Explain the Increased Risk of

Cardiovascular Disease among

Those of Lower Socioeconomic Status?

Mortality rates for all causes are higher in persons of lower socioeconomic status (SES). In fact, mortality rates are linearly related to socioeconomic status such that every increment in status is associated with commensurate increase in more salubrious health outcomes. These differences are found in countries with universal health care and in countries with less ethnic and racial diversity than the U. S. (Marmot, 2004).

The differences among social classes in mortality are most pronounced for cardiovascular disease and Type II diabetes (Steptoe & Marmot, 2002). Researchers have attempted to parse out the reasons for this association. Life style risk factors (diets low in vegetables and high in fats, less exercise, more cigarette smoking, being overweight) are known to be more pronounced in the poor (Lynch, Kaplan, & Salonen, 1997). However, these variables account for only a small amount of the disparity among socioeconomic classes in cardiovascular mortality (Marmot, 2002, p. 44) and all cause mortality (Lantz, House, Lepkowski, Williams, Mero, Chen, 1998). In fact, smoking, obesity, diabetes, atherogenic lipid profiles, hypertension, and sedentary life style can account for only 20-35% of the association between SES and coronary heart disease (Rose & Marmot, 1981).

In the beginning of this paper some of the chemical mediators involved in causing arteriosclerosis were discussed. Poor individuals are more likely to exhibit higher levels of risk factors for cardiovascular disease (higher C-reactive protein and coagulation proteins) (Steptoe & Marmot, 2002). Seeman et al. (2004) found that after controlling for a composite measure encompassing many physiological measures including CRP and coagulation factors, the

relationship between SES and mortality was no longer significant. Thus, SES may affect cardiovascular disease outcomes through inflammatory mediators and coagulation factors, both of which are influenced by stress.

Poor individuals do more frequently experience many types of stressors including unemployment, low levels of social support, financial strains, neighborhood crime (Hemingway and Marmot, 1999). These stressors reduce sense of control over daily hassles (e.g., having an unreliable automobile for transport to work, not having the money to pay for unanticipitated household repairs, etc.). Thus, differential exposure to chronic stressors may in part account for health outcome disparities between the affluent and the poor. Stressful events, not in themselves economic, can be more burdensome for the poor. For example, Brummett et al. (2005) found that among caregivers of those with dementia, those living in neighborhoods with high crime rates had less glucose control than those caregivers from safer neighborhoods (as evidenced by higher levels of glycosylated hemoglobin which is a risk factor for cardiovascular disease).

Stressful conditions at work among those in blue collar jobs. Researchers have paid particular attention to how working conditions impact the development of cardiovascular disease. Greater job strain and greater effort-reward discrepancy are more often characteristic of lower status jobs. The concept of job strain captures a working situation in which job demands are higher but worker control is minimized. Across many studies, job strain has been associated with hypertension and cardiovascular disease (Schnall, Landsbergis, & Baker,1994). Job strain is associated with greater narrowing of arteries in males aged 24 to 39 after controlling for SES, drinking, smoking, exercising, cholesterol levels, social support, age, and body mass index (Hintsanen et al., 2005). The concept of effort-reward discrepancy is characterized by a

competitive, hard-working person in an insecure job situation offering little opportunity of recognition. Those persons whose work lives are characterized by higher levels of effort-reward discrepancy are more likely to develop cardiovascular disease (Bosma, Peter, Siegrist, Marmot, 1998). In a recent publication of the Whitehall Studies of British civil-servants, Kivirmäki et al. (2005) examined the role of supervisor unfairness in the development of cardiovascular disease (fatal heart attack, non-fatal heart attack or compromised blood supply to the heart). Those men who perceived their immediate supervisor as unfair, had a higher incidence of cardiovascular events. For those men who perceived their supervisors as fair, both job strain and effort-reward discrepancy were unrelated to cardiovascular disease.

Childhood income level impacts adult health. Not only does adult SES relate to cardiac outcomes, but childhood SES can affect adult outcomes as well (Kivirmäki et al., 2004; Lehman, Taylor, Kiefe, & Seeman, 2005). Of course, parents have an influence on those behavior patterns that are established during childhood that persist throughout adult life. Adult obesity, cigarette consumption, diets lacking in fruits and vegetables, and low levels of physical activity are more likely in persons whose parents were poor even after controlling for adult income (Lynch, Kaplan, & Salonen, 1997). Not only are behavioral practices learned during childhood, but so also are perspectives on life. Harper et al. (2002) found that parental income level during a subject's childhood predicts adult cynical hostility and hopelessness, and depression, all of which are risk factors for cardiovascular disease. Thus, relative economic deprivation during childhood is associated with the development of personality traits which might increase the frequencies of stressors and the selection of less salubrious coping responses for dealing with stressful events.

Cardiovascular Disease in African Americans

African Americans, both males and females, at every level of income have higher mortality rates than white persons in the same income brackets. Overall, the mortality rate is 30% higher in African Americans than in whites. In terms of types of diseases eventuating in mortality, African Americans have 5.75 times the risk of dying from HIV, 2.4 times the risk of dying from diabetes, 1.8 times the risk of dying from strokes, and 1.47 times the risk of dying from heart disease (Williams, 1999; 2005). African Americans also exhibit higher rates of hypertension which is associated with subsequent diagnosed cardiovascular disease (Jones et al., 2002).

Higher levels of stressors characterize the lives of African Americans living in areas of concentrated poverty (Collins & Williams, 1999). Of course, African Americans at all levels of income encounter a greater number of stressors in their lives than do their white counterparts. For middle class African Americans, the toll of caring for those family members who are not doing well contributes to stress (Williams, 2005). For middle class African Americans, Cole and Omari (2003) recognize that "disidentification, distancing, and alienation from one's community of origin" may contribute to stress as well. Clark, Anderson, Clark, and Williams (1999) argue that racism constitutes a unique stressor in the lives of African Americans and speculate upon its role in cardiovascular disease as well as other diseases. While findings regarding the association between perceived discrimination and higher blood pressure have been inconsistent (Brondolo, Rieppi, Kelly, & Gerin, 2003), perceived discrimination does relate to indicators of extant cardiovascular disease. Troxel, Matthews, Bromberger, and Sutton-Tyrrell (2003) found that perceived discrimination predicted the degree of narrowing of arteries in African American

women. Lewis et al. (2006) found that perceived racism predicted coronary artery calcification. Thus, the stressor of racism may account for some of the increased morbidity and mortality from cardiovascular disease observed among African Americans.

Racism is not only manifested in interpersonal discriminatory encounters, but also in exposure to pejorative racial stereotypes that exist in the culture. A phenomenon emerging from this type of racism is stereotype threat. Steele (1997) demonstrated that African Americans perform less well on intellectual measures when they must indicate their racial identity on the test form before taking the test than when they are racially anonymous. Steele coined the term "stereotype threat" to label the phenomenon. Blascovich, Spencer, Quinn, and Steele (2001) examined how stereotype threat impacts blood pressure. When the salience of the racial stereotype is raised, mean arterial pressure of African Americans is elevated in comparison to performing the same task when the salience of the stereotype is low. Thus, there is suggestive evidence that another manifestation of the stress of racism contributes to a risk factor for cardiovascular disease.

Relative Disadvantage

Much of the research documenting cardiovascular-health-outcome-disparities between persons varying in socio-economic status has occurred in nations where nutrition is generally good for everyone, where there is universal health care. Even still, cardiovascular health disparities across income levels are observed. Examining the persistence of these disparities where the disparities can not be easily attributed to sanitation, health care, or exposure to infectious agents, Salpolsy (2005) emphasizes the psychological stressor of being poor in a country in which one is surrounded by others who have more. Wilkinson (2000, pp. 100-143)

finds that the poor suffer the worst health outcomes for many diseases when they live in societies where the gap between the rich and the poor is especially pronounced. Relative poverty rather than absolute deprivation seems to be a relevant parameter in influencing negative health outcomes.

Surprisingly, in societies in which the gap between the rich and poor is pronounced, the health outcomes of everyone in the society are diminished (Salposky, 2005; Kawachi & Kennedy, 2002; Wilkinson, 2005, pp. 100-143). For example, comparing a person in the U.S. with a person in Greece who is at an equivalent income level, the person in the U. S., the developed nation with the widest gap in incomes between the rich and the poor, the health outcomes of the American are worse (Marmot, 2004, p. 65). The findings of worse health outcomes in the high inequality society are robust for most income levels. However, the impact of inequality on the health of those at the very top remains controversial (see Kawachi & Kennedy, 2002, p. 107; Wilkinson, 2005, pp. 100-143).

The explanation for the poorer health outcomes in persons from societies in which there is a greater gap between the rich and the poor, may well be explained by the greater stress level of everyone in inequitable societies. It is known that the rate of violent crime increases as a function of increasing gaps between the rich and the poor within the locality (Hsiech & Puch, 1993). Probably other stressors (interpersonal hostilities, frequency of being pan-handled, etc.) increase as well. Increased stress is a likely source for the negative health outcomes for all members of the highly-stratified society.

Implications for Direct and Macro Practice Social Work

Many social work interventions might be developed to prevent negative outcomes of

stress. Psychotherapeutic interventions are available to reduce hostility and encourage salubrious coping styles. Additionally, those with stronger social ties exhibit lower CRP (Ford, Loucks, & Berkman, 2006). Increased social ties might function as a buffer against stress. Social worker interventions to strengthen coping mechanisms, reduce hostility, and increase social ties might be developed. These interventions might be added to social work interventions for assisting clients in making life style changes in diet, exercise, smoking cessation and increasing medication compliance to statins.

The findings of health-outcome disparities between the rich and the poor as well as differences among racial groups remind us of the imperative to work toward social justice. The new note in the findings is the documentation in cardiovascular health outcomes of the psychological stress created by assignment to a lesser status, created by having little control over one's environment, and created by being treated unfairly. Eliminating unfairness and racism would, of course, reduce health disparities. But, in the real world, individuals have to find a way to cope. A literature on coping mechanisms for dealing with unfairness and discrimination is emerging. However, this literature is in its infancy. Social worker researchers might empirically investigate useful attitudes and perspective for dealing with the reality of racism and unfairness. Whether anger and righteous indignation are salubrious responses for the vasculature could be investigated. Answers might offer direction for micro-practice social workers who assist clients in coping with the painful realities they confront.

The findings on better health outcomes for people in more equitable societies raise profound questions. Social workers have traditionally been concerned about the injustices visited upon the worst-off, most vulnerable in the society. Perhaps the recent findings on the outcomes

of highly stratified societies should be a rallying cry for a more egalitarian, less stratified society that might enhance the health of everyone in the country. At least it offers an objective point in its favor, suggesting an advantage for everyone. This, of course, raises questions regarding the American perspective. Should social workers be questioning the American ethic of working to excel and distinguish oneself, as opposed to the Eastern philosophy of shame in distinguishing oneself from one's peers? Should the debate about the inheritance tax be discussed in Policy classes to the same extent as welfare reform? Hopefully, this article will have alerted the social work community to these questions so that social workers and social work educators will be examining the implications of these new data.

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