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**The Mind-Body Connection:
Not Just a Theory Anymore**

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Abstract

The field of psychoneuroimmunology has witnessed an explosion of empirical findings during the last two decades. Research has documented the mechanisms through which stressful emotions alter white blood cell function. Stress diminishes white blood cell response to viral infected cells and to cancer cells. Moreover, vaccination is less effective in those who are stressed and wounds heal less readily in those who are stressed. While stress decreases the activity of some white blood cells, stress does not compromise the function of all types of white blood cells. Indeed, some types of autoimmune disease, which involve particular subsets of white blood cells, are exacerbated by stress.

The literature documents the efficacy of talk-therapy interventions in altering immune system parameters and enhancing the body's ability to combat disease. The literature also documents the impact of the chronic stress of poverty on immune system function.

**The Mind-Body Connection:
Not Just a Theory Anymore**

The field of psychoneuroimmunology was born when developmental psychologist, Robert Ader, showed that the immune system can be conditioned. Patients suffering from autoimmune diseases such as Lupus (Systemic Lupus Erythemia) had long been given immunosuppressive drugs to contain the attack of their white blood cells on their own cells. Ader showed that setting up placebo drug administration could ‘trick’ the immune system into suppression, in a process analogous to the bell eliciting salivation in Pavlov’s dogs (see review in Ader & Cohen, 2001). Since those initial observations, the field of psychoneuroimmunology has witnessed an explosion of empirical findings. Although the mind and the body might once have been seen as separate domains, the proliferating results demand an appreciation of the interconnection between mental states and physical processes.

This article will present an overview into the impact of how the mind can influence the disease processes in viral infections, cancer, autoimmune disease, wound healing, and vaccination. The empirical work investigating the mind’s influence on the body encompasses studies examining work status, subjugation and oppression, stressful events, as well as how personality factors and social support can buffer life events and circumstances. While an extensive literature exists exploring personality factors (coping mechanisms, stress reactivity, behavioral inhibition, optimism, hostility, mechanisms for coping with anger, etc.), this review primarily will focus on how environmental factors (stressful life-events or life circumstances)

impact the body, although seminal findings regarding personality variables will be mentioned.

With regard to the concept of stress, across studies stress has been operationalized in various ways. In some studies, subjects are asked about the occurrence of major life events (loss of job, death of spouse, daily hassles). In other studies, subjects are assessed during periods which many would regard as stressful (e.g., medical students during major examinations as opposed to summer vacation, basic training at West Point, caring for a spouse with Alzheimer's disease). Of course, individuals vary in terms of whether they will exhibit a stress response to events that induce a stress response (increased sympathetic nervous system activity and activation of the hypothalamic-adrenal gland axis) in the majority of individuals. Some studies have investigated those personality traits or resources that distinguish those who fail to exhibit a stress response while undergoing what is, for most people, a stress-response inducing situation. However, the most of the studies discussed in this review, compared the group mean on an immune system parameter in persons experiencing a given event which induces a stress response in the modal person to the group mean of an immune system parameter of persons who were not undergoing a stressful event. Despite the loss of power attributable to averaging over persons who were and were not exhibiting a stress response to an event likely to induce a stress response in most persons, researchers have observed statistically significant differences in comparison with groups who were not experiencing stressful events.

The reviewed literature offers implications for social work interventions. While much of the research reviewed here may initially seem arcane and off-the-track for social work, in fact, it is imperative that social workers be conversant with this information. For micro-practice social work, the findings emerging here attest to the importance of emotional factors on hard medical

outcomes (immune system parameters, morbidity, mortality). Studies are available that testify to the importance of group interventions for impacting health outcomes. Childhood poverty and adversities experienced during childhood have been shown to have consequences for immune system function in adulthood. This, in turn, underscores the imperative of creating a more just society in which the gap between the rich and the poor is minimized, the very fielding which our macro-practice colleagues labor.

This review will examine the impact of psychological factors in combating viral pathogens, in fighting cancer, in autoimmune disease processes, in response to vaccination, and in wound healing. Because the immune system plays a major role in most physiological processes and because stress heavily impacts the immune system, psychological processes cannot be ignored in any area of health, but the particulars are important.

Because of the constraints of space, the considerable topic of the impact of psychological processes on cardiovascular disease will not be examined here.

Viral Infection

The human species has been living with viruses since the beginning of creation. In fact, much of the DNA in the human genome was probably contributed by retroviruses, i.e., viruses such as HIV that can insert themselves into host DNA, which infected our ancestors in the past but which lost the ability to escape from the cell. These prior infections have conferred many benefits on modern humans, the progeny of the initially infected. In fact, the ability of the white blood cells to respond in a specific manner to novel pathogens is probably a contribution of infection with retroviruses of the past (Lewin, 2000).

In responding to a virus, which are parasitic particles that can only replicate inside a host

cell, leukocytes that can destroy the infected cell provide the primary defense. Two types of white blood cells kill cells infected with a virus: Natural Killer Cells and CD8+ lymphocytes. Natural killer (NK) cells recognize an infected cell, as opposed to a healthy cell, through a complicated process, but NK cells are not specific for any particular virus. Any NK cell is capable of recognizing infection by a diverse array of microorganisms. NK cells are the first type of leukocyte on the scene to combat viral infections. If the organism is to survive, the NK cells initially stifle a virus before the arrival of the more specific response provided by the CD8+ cells. In animal models, stress decreases NK cell cytotoxicity (Welsh et al., 2004).

Unlike NK cells, CD8+ are very particular about the cells they will target for destruction. Each CD8+ cell only has one type of receptor for recognizing infection. Thus, any particular CD8+ cells will recognize only one type of protein belonging to a particular microorganism. A response from a CD8+ cell to a virus never before seen by the body occurs later in infection, typically three to ten days after the body becomes infected with a virus which the host has never seen before. After an initial infection, easily activated memory CD8+ cells reside in the host. Given a second infection, the memory CD8+ cells can mount a rapid response to a second infection. The CD8+ cells memory cells (as well as some other types of memory cells) constitute the basis for the efficacy of vaccinations. In stressed animals, the CD8+ cells, which are supposed to rapidly divide given provocation by a pathogen, display an apathetic response. They divide more slowly and thus will offer lesser control of an infection (Herbet & Cohen, 1993)

Colds

Cohen, Tyrell, & Smith (1991) and Cohen, Doyle, Skoner, Rabin, & Gwaltney, (1997) have been innovative in controlling for exposure to pathogens by inoculating his subjects with

cold viruses. Cohen et al. measured subjects on their levels of social support and stressor exposure. The dependent variable was degree of symptomatology (swelling in the nasal passage, degree of mucous production) after viral inoculation. Those with less social support and more stressors exhibited more extreme symptoms, although stressors associated with greater cold symptoms were interpersonal or work related and chronic (Cohen et al., 1998). In a more recent study of this type, lack of positive emotion (e.g., happy, cheerful, full of pep) predicted whether the virus once inoculated was able to establish a productive infection (Cohen, Doyle, Turner, Alper, & Skoner, 2003). (For more information regarding studies of this type, the reader should consult Cohen & Miller, 2001).

Herpes Viruses

Herpes Simplex Virus-1 & 2 The herpes viruses family are unique among virus families in that they can establish latency in a host cell. Distinguished from retroviruses (of which HIV is a member), herpes viruses do not integrate into host DNA, but rather live in the body of the host cell. HSV-1 and HSV-2 are viruses with very similar genomes. Both of the Herpes Simplex 1 and 2 infect neurons. They can hang out in the neuronal bodies in the spinal cord without replicating for years, but can periodically reactivate. Given reactivation, new copies of infectious particles (called virions) are produced. Healthy cells in a host can be infected and the virions can be shed into the environment to establish infection in new hosts. Both physical stress, as occurs given a cut, or infection with a cold virus, or fever, and emotional stress can reactivate Herpes Simplex viruses. When this occurs, lesions, usually on the lip for HSV-1, and usually on the genitals for HSV-2, become apparent. Garcia-Linares, Sanchez-Lorente, Coe, & Martinez (2004) examined the ability of the antibodies in saliva and other protective proteins from subjects to

control Herpes Virus Infection of cells in a Petrie dish. The saliva of women who were victims of partner abuse exhibited the least ability to control the spread of the virus infection to cells in the Petrie dish.

Researchers do know some of the reasons why stress is associated with Herpes Virus reactivations. Given a stressor, the body produces more cortisol, the primary stress hormone. Herpes viruses have glucocorticoid response elements such that elevations in cortisol are sufficient to reactivate the virus (Glaser, Pearl, Kiecolt-Glaser, Malarkey, 1994; Glaser et al., 2005). CD8+ cells produce IFN-gamma which is a very important hormone for keeping Herpes viruses in a latent state. As previously mentioned, CD8+ cell function is compromised by stressors. Moreover, given viral reactivation, NK cells are important players in reducing the duration of the period of reactivation. NK cells are signaled to respond by local hormones released by injured tissue: Interferon alpha and Interferon beta. Cortisol, the stress hormone, suppresses the level of Interferon alpha and beta released by injured tissue, so that NK cells are less effectively recruited to respond. The extent and duration of the herpes lesions are enhanced (Ortiz, Sheridan, & Marucha, 2003).

Stress not only influences the course of herpes infection in the host, but also influences transmission of virus from the mother to infants. During birth, mothers with activated genital herpes can transmit the virus to their infants. Between 1/3000 to 1/7000 infants are infected with HSV during vaginal delivery (Kohl, 1997). During gestation, the mother's antibodies are transferred across the placenta so that the infant will have some protection against pathogens. (Infants have an immature immune system and they aren't able to produce antibodies until about the age of 10 months.) In stressed primate mothers, the level of antibody transmitted to infants

through breast milk or across the placenta is lower than that transmitted by non-stressed mothers. This is not only because stress decrease levels of antibody, but stress also decreases the transfer of antibodies across the placental barrier (Coe, Kemnitz, & Schneider, 1993; Sobrian, Vaughn, Bloch, Burton, 1992).

Researchers have examined the effect of stress on reactivation of other herpes viruses as well. Epstein Barr virus infects white blood cells and can reside in a dormant state inside memory leukocytes. Epstein Barr virus causes mononucleosis, and Burkitt' s lymphoma, a type of cancer, is associated with Epstein Barr virus infection. Again, stress plays a big role in determining the course of infection with Epstein Barr virus. Glaser et al. (1991) and Glaser et al. (1994) followed students at West Point as well as medical students. Examination stress at West Point was associated with reactivation of Epstein Barr virus, although the physical stress of basic training did not increase the reactivation of virus (Glaser et al. 1991, 1994). Medical students during final examinations, especially those students distinguished by high scores on a loneliness scale, were more likely to exhibit reactivation of Epstein Barr virus (Glaser, 2005). Men undergoing stressful divorces are less able to control Epstein Barr Virus (Kiecolt-Glaser et al., 1988).

HIV

Research attest to the negative impact of stress on the outcomes of those infected with HIV. Persons infected with HIV who experience a greater number of stressful events, who lack social support, and who rely on denial as a coping mechanism, exhibit a shortened trajectory to the progression to AIDS (Leserman et al., 1999; 2000; 2005). Bereavement predicts more rapid decline in CD4+ cell count (Kemeny & Dean, 1995). Those who are more socially inhibited

enter treatment with higher initial viral loads. They respond less vigorously to HAART (Highly Active Antiretroviral Therapy) in terms of decrease in viral loads and in terms of CD4+ cell count recovery (Cole, Kemeny, Fahey, Zack, & Naliboff, 2003). Symptoms of depression predict speed of progression to AIDS in those who are HIV+ (Ickovics et al., 2001; Leserman et al., 2000), and rapidity of CD4+ count decline (Antoni et al., 2006). Moreover, viral load and depressive symptoms are correlated (Evans et al., 2002; Antoni et al., 2006). Even when persons are being treated with the advent of Highly Active Antiretroviral Treatment, the life stressors, depression, avoidant coping, and hopelessness continue to predict speed of decline in CD4+ cells and increase in viral load (Ironson et al., 2005).

The molecular mechanisms through which distress influences HIV status have been explored. Norepinephrine and Substance P, a stress hormone released by sympathetic neurons enervating major organs and blood vessels, will increase the replication of HIV and will increase the expression of receptors to which HIV can attach to gain entry into cells (Chowdhury et al., 1993; Cole, Jamieson, & Zack, 1999; Cole, Korin, Fahey, Zack, 1998; Lai et al., 2001). Although cortisol, the stress hormone, can suppress immune system response, and seemed like the obvious suspect in mediating the deleterious effect of stress on disease progression, the findings on how cortisol relates to HIV disease progression have been inconsistent (Leserman et al., 2000). Studies documenting the impact of distress on progression to AIDS once a person has become infected with the virus, and on later disease progression, are firm. The real questions are around further illuminating the biological pathways through which distress can exert this effect on disease outcome.

Response to Vaccination

A large number of investigators have examined the efficacy of vaccination on persons who are distressed. The bottom line is that in stressed individuals antibody concentrations after vaccination are much lower. This has been observed in Alzheimer caregivers vaccinated with influenza antigen (Kiecolt-Glaser, Marucha, Malarkey, Mercado, & Glaser, 1995), in response to a DNA hepatitis B vaccine (Jabaaij et al., 1996), in medical students reporting stress vaccinated against hepatitis B (Glaser et al., 1992) and in depressed, elderly in nursing homes vaccinated against influenza (Trzonkowski et al., 2004). These findings are particularly relevant for infant mortality. Infants rely upon maternal antibodies transmitted across the placenta or through breast milk. Stressed mothers producing fewer antibodies are unable to provide the barrier against disease to their infants. This has been demonstrated in squirrel monkeys (Coe & Crispen, 2000) and in human mothers (Yorty & Bonneau, 2004).

Some caveats can be introduced here. The previously cited research findings have examined the impact of fairly high levels of stress on the immune systems ability to control infections. Ironically, low levels of stress can enhance the function of the immune system (Zautra, Hamilton, Potter, & Smith, 1999). Indeed, a recent vaccination study found that the experience of minor stressors immediately prior to vaccination actually enhanced the response to the vaccination (Edwards et al., 2006). Further, there can be differences in the way the many cells of the immune system respond to stressors. Thus, each disease entity (viral infections, cancer, particular autoimmune diseases, cardiovascular disease, wound healing) must be investigated separately.

Cancer

Although cancer develops when a cell develops mutations on proteins involved in

regulating proliferation, a growing literature suggests that stress also influences the process of cancer development. While stress probably may not affect the probability that a DNA will be exposed to Ultraviolet light or carcinogens which can damage DNA, stress will influence whether a cell can repair damaged DNA. DNA repair enzymes are less numerous in stressed mice and depressed individuals display attenuated repair of damaged DNA (Glaser, 2005). Once a cell divides despite the damage in critical genes regulating growth, CD8+ cells and NK cells play a role in recognizing and killing these cells. In animal studies, lesser NK cell activity is a predictor of greater spread of cancer cells throughout the body (Wiltrout et al., 1985; Barlozzari, Leonhardt, Wiltrout, Herberman, & Reynolds, 1985). In humans as well, measures of NK cell function assessed at the time of tumor removal is an independent predictor of tumor recurrence (Cook & Lewis, 1987; Takeuchi et al., 2001; Taketomi et al., 1998). Stress does compromise ability of NK cells to provide protection (Andersen et al., 1998; Herbert & Cohen, 1983; Levy, Herberman, Lippman, & d'Angelo 1987). These forces help to explain the associations between depression and the development of cancer. Depression is a predictor of lung cancer diagnosis among those who are smokers (Jung & Irwin, 1999; Kiecolt-Glaser & Glaser, 1999). Stressful events predict cervical neoplasia as do negative coping skills (Coker, Bond, Madeleine, Luchok, & Pirisi, 2003; Antoni and Goodkin, 1988). Number of adverse events (exposure to family violence, alcoholic or substance abuse of parent, parental mental illness, incarceration of parent, abuse to child) experienced during childhood raises the odds of developing cancer by 1.9 (Felitti et al., 1998).

Some of the mechanisms through which stress influences NK cell function have been examined. Stress is associated with disruptions of the autonomic nervous system and disruption

of daily fluctuations in secretion of hormones including cortisol and melatonin. Indeed loss of circadian rhythm in hormone secretion is a predictor of cancer as well as cardiovascular disease, stroke, and Type II diabetes (Sephton & Spiegel, 2003; Rosmond & Bjorntorp, 2000). Sephton and Spiegel (2003) found that women who had lost the circadian pattern associated with cortisol release were lower on measures of NK cell cytotoxicity, had fewer NK cells in circulation, and had tumors which spread to more distal tissues rather than adjacent tissues. The Sephton and Spiegel findings were consistent with basic research. Filipski et al. (2002) had ablated the suprachiasmatic nucleus (SCN), the nucleus in the brain which responds to ambient light and sets the fluctuations on release of hormones, before inoculating animals with cancer cells. Those animals without a SCN, who lacked a mechanism for regulated hormonal release, demonstrated faster cancer progression. Central variations in hormonal release entrains the cyclical pattern of growth in tissues (Balsalobre et al., 2000). With no restraining influence on growth, tumor growth becomes more aggressive. Consistent with the notion that a normal pattern of circadian rhythms protects against cancer, is the finding that night shift workers have higher rates of cancer (Bovbjerg, 2003).

Not all of the research examining stress factors on the course of cancer has been correlational. Particular experimental studies also support stress factors as strong determinants of the control of cancer cell. Stefanski and Ben Eliyahu (1996) first exposed rats to an intruder and then inoculated cancer cells into the rats. Those rats subjected to intrusion exhibited increased lung retention of the cancer cells. More advanced tumors were particularly pronounced in those rats who appeared to be defeated by the intruder.

A rather large literature attests to the case that stress, lack of social support, an absence of

“fighting spirit” does influence death rates from cancer after diagnosis. Level of distress predicts cancer survival 10 years after diagnosis (Brown, Levy, Rosberger, & Edgar, 2003; Greer, 1991), although Spiegel and Giese-Davis (2003) have recognized the inconsistency in results across studies and caution that the direction of causation in the findings is ambiguous (i.e., poorer health status may cause depression rather than depression compromising ability to control cancer progression). Temoshok (2000) identifies the C-personality, associated with lack of assertion and self-sacrifice, as associated with faster progression.

Autoimmune Disease and Allergies

As a new student in the field of psychoneuroimmunology, I supposed that since severe stress impairs the function of the immune system, that stress would exert a beneficial impact on those suffering from autoimmune diseases, i.e., conditions in which white blood cells attack one's own body. Additionally, my expectation was that those with atopic conditions such as allergies in which a strong immune cell response is mounted against essentially harmless foreign particles like dust mites would also be improved by stress. Empirical research has dashed these expectations. The mechanistic arms of the immune system producing disease vary across autoimmune diseases and allergies. Inconsistencies across particular autoimmune diseases are found in how various degrees of stress impact outcome. A few representative examples follow.

Multiple Sclerosis (MS) is a disease in which white blood cells attack the fatty coating surrounding neurons in the Central Nervous System. In the first decade, MS is characterized by abrupt exacerbation followed by remission often without residual debilitation. Family and work related stressors are associated with more exacerbations. Persons with greater social support experience fewer exacerbations as do those who rely on instrumental coping and distraction

(Mohr & Pelletier, 2006). In order for MS to progress, leukocytes must enter the brain. Ordinarily, the blood brain barrier will prevent the entry of white blood cells into the brain. Given stress, mast cells in the dura mater covering of the brain will release TNF-alpha, histamine, serotonin, and CRH, which will compromise the Blood Brain Barrier which ordinarily protects the brain from the entry of white blood cells, allowing the influx of leukocytes (Esposito, et al., 2002).

Lupus and Arthritis

Persons with Lupus exhibit greater subjective symptoms and higher levels of C3, C4, and anti-DNA antibodies (objective measures of disease severity) following stress (Peralta-Ramírez, Jimènes-Alonso, Godoy-García, & Perez-García, 2004). For Rheumatoid arthritis patients the relationship between stress and disease activity is complex. Interpersonal conflicts are associated with rise in prolactin and estrogen (two immune system enhancing hormones) and disease exacerbation (Zautra, Burleson, Matt, Roth, & Burrows, 1994.) Depressive symptoms are associated with elevations in IL-6, which might be anticipated to exacerbate disease (Zautra et al., 2004). However, when Potter and Zautra (1997) distinguished between major life stressor and daily hassles, the major life stressors were associated with less disease severity, whereas the hassles were associated with more disease severity. As with many diseases, social support can buffer the impact of stressors on disease exacerbation (Zautra et al., 1999).

Atopic Conditions

Atopic dermatitis, eczema, psoriasis, and urticaria are all skin conditions mediated by excessive activation of mast cells. Mast cells are white blood cells located around blood vessels in areas just beneath the surface of the skin or mucosal linings of the body. The sympathetic

nervous system, which is activated by stress, will release CRH (corticotropin releasing hormone) into areas below the skin where mast cells reside. CRH induces mast cells to release mediators of vascular permeability and vasodilation exacerbating symptomatology (Theoharides et al., 1998). Thus, stress through its impact on activation of mast cells generally can be expected to exacerbate atopic conditions.

Wound Healing

In several clever demonstrations, Glaser and colleagues have documented the negative impact of stress on healing. Kiecolt-Glaser et al. (1995) contrasted the ability to heal a cheek lesion in medical students during final examinations as opposed to during summer vacations. The results were dramatic. During final examination periods, medical students cheek wounds healed far less rapidly than during vacation (see Marchua, Sheridan, & Padgett, 2001, for review). In another stressed population, Kiecolt-Glaser et al. (1995) found that the wounds of Alzheimer patient caregivers healed more slowly compared to lesions of age-matched controls. Kiecolt-Glaser et al. (2005) have also examined the time required to heal after hostile marital interactions. The wounds inflicted immediately subsequent to hostility heal less rapidly.

So how is this relevant to Social Work?

Implications for micro-practice: Clinical interventions have been demonstrated to produce positive impact on disease outcome. Interventions to retard the progression of disease in persons with HIV have been evaluated. Cognitive-behavioral-stress management interventions have demonstrated efficacy in comparison to drugs alone in enhancing immune function (Antoni et al., 2002; Antoni et al., 1991), in decreasing cortisol levels (Antoni et al., 2000) and in decreasing viral load (Antoni et al., 2006). These findings have been replicated in samples who

are taking newer drug cocktails (Antoni et al., 2006). Those receiving Cognitive Behavioral Stress Management Therapy, increased their perceived social support, had higher coping self efficacy, and experienced less distress, which related to decreases in viral load (Antoni et al., 2006). Group based bereavement counseling proved efficacious in decreasing viral load relative to those in a control group (Goodwin et al., 2001). Allowing HIV+ men to write about emotional experiences has been shown to increase CD4+ counts and decrease viral load relative to the control group (Petrie, Fontanilla, Thomas, Booth, & Pennebaker, 2004).

Talk-therapy interventions have demonstrated efficacy for reducing disease from other viruses in addition to HIV. With regard to controlling the Herpes Virus, healthy elderly people who were living independently were trained in relaxation techniques and guided imagery. After one month, those receiving the relaxation training manifested enhanced NK cell function and better control of latent herpes simplex I virus relative to the control group (Kiecolt-Glaser et al., 1985). For boosting response to vaccination against influenza, an 8 week course in mindfulness-meditation boosted antibody levels after influenza vaccination relative to the control group (Davidson et al., 2003).

Group interventions have achieved salubrious results in cancer patients as well. Cognitive behavioral stress management, which was effective in helping patients to identify positive growth experiences from their breast cancer, was associated with lower cortisol levels (Antoni et al., 2001; Cruess et al., 2000). Persons with stage I and II malignant melanoma exhibited stronger NK cell activity after 6 weeks of cognitive behavioral therapy delivered in a group context relative to the control group. Moreover, at 6 year follow-up and again at 10 year follow-up, fewer of those in the cognitive behavioral therapy group had died (Fawzy et al.,

1990a; 1990b, 1993, 2003). Those in a one-year support intervention for breast cancer exhibited higher survival rates than those in the control condition (Spiegel, Bloom, Kraemer, & Gottheil, 1989).

Support groups, stress management groups, cognitive behavioral therapy groups, mindful meditation groups are certainly within the scope of clinical social work interventions. Such interventions can constitute an important supplement to medical interventions. Some of the interventions with demonstrated efficacy, e.g., Cognitive Behavioral Stress Management used by Antoni et al. (2006), are manualized. Manualized treatments might be incorporated into Social Work education.

The National Institute of Health in 2003 in announcing its program for studying social work practice in health has acknowledged the role that social workers have in medicine (Stoesen, 2006). Finger and Arnold (2002) have suggested that “mind-body” interventions be routinely incorporated into social work curriculum. While consensus that the mind does affect health is rapidly emerging, there is less consensus on whether talk-interventions can influence the mind in health promoting ways (Freedland, Miller, & Sheps, 2006). Outcome research is limited and has largely been conducted by a limited number of people. Social workers have been active in developing group interventions for diverse conditions such as diabetes (Alley & Brown, 2002; Claiborne & Massaro, 2000), cancer (Roberts, Piper, Denny, Cuddeback, 1997), and sickle cell disease (Comer, 2004). Hopefully, social work researchers can play an active role in further developing interventions, refining these interventions through empirical testing, isolating critical components, and advancing best practices. The case has been made that influencing the mind will impact health. Demonstrating that social work interventions can effectively influence the

mind constitute the work ahead.

Implications for macro-practice: Morbidity and mortality from all causes are higher among the poor than among the more affluent. The poor are at increased risk for morbidity and mortality from many diseases (Cohen, 1999). While in the United States, some of the health statistics might be attributable to racism, the association between poverty and health risk also obtains in countries with populations with less diversity in ethnicity and race (e.g., Finland). Indeed, the British Whitehall study comparing health outcomes and various predictors of cardiovascular health outcome limited the subject pool to white male civil servants. Thus, the major variable distinguishing participants was socioeconomic status (Marmot, 2004).

Not only does adult socioeconomic status relate to health outcomes, but one's childhood SES also is predictive for many health outcomes. Low socioeconomic status during childhood is correlated with higher mortality from cardiovascular disease, stroke, respiratory disease, stomach and lung cancer, and is also related to higher rates of chronic bronchitis and periodontal disease (Cohen, Doyle, Turner, Alper, & Skoner, 2004). SES at the time of entry into the convent predicted health status 50 years later among Nuns who had shared the same diet, health care, and housing throughout their adult lives (citation by Salpolsky, 2005). Cohen et al. (2004) showed that those individuals who were poor as youngsters, regardless of their adult status, were more susceptible to colds. Cohen et al. used the same strategy of bringing subjects into the laboratory and then inoculating them with cold virus. Those whose parents had owned their own home exhibited fewer symptoms.

Given the surprising generality and robustness of these findings, and the fact that the effects of early experiences (childhood socio-economic status) could have such long-term

consequences, these new findings offer medical grounds for a more just society, alongside the many moral and philosophical justifications with which we are familiar. This “dollar and cents” argument, might succeed where moral arguments have failed to persuade. These new facts offer further justification for the work of macropractice social workers trying to bring us closer to the ever-promised level playing field, along with the many new places for interventions by micropractice social workers in their task of lessening the impact of stress on people’s health.

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