Health Care Can Change From Within

continued from p. 11

Case—Part 2. The clinic KC visited recently implemented the Change from Within model. Following the new clinic protocol, the nurse asked KC about IPV, and KC responded affirmatively. The physician then obtained a more detailed violence history. KC described how, during a recent family gathering, her partner became angry, punched her, knocked her down, jumped on top of her and began to strangle her, causing her to fear for her life. Although he was arrested, she continued to feel unsafe and experience insomnia. The physician evaluated her physical injuries, educated her about strangulation injury, conducted initial safety planning, documented his findings, and offered her referral to one of the clinic's health care advocates, a clinical psychologist, for further assistance. The physician introduced the psychologist to KC at the time of the initial medical encounter, and she agreed to an appointment. Initial assessment identified the following symptom picture: sleep onset and interruption insomnia, headaches, nightmares, unpredictable crying spells, worry, difficulty concentrating on work, intrusive thoughts, and decreased appetite. The agreed-upon treatment plan included relaxation to manage intense physical arousal, sleep hygiene strategies, return to previously enjoyable life experiences, supportive counseling, and referral to a legal advocate at the local women's center. During psychological intervention, KC frequently set up appointments with her family physician on the same day. With her consent, the physician and psychologist consulted regularly about new developments, both positive and problematic, in her care. KC saw the psychologist for a total of 6 weekly sessions and one follow-up visit. During her last visit, she reported a nearly complete cessation of nightmares, insomnia, intrusive thoughts and headaches. Further, she returned to work and resumed her exercise program. She felt she had re-claimed her life and was prepared to proceed as a witness in criminal litigation in her partner's case. At her request, records of her medical and psychological treatment were provided to prosecution to aid in the litigation.

References

Ambuel, B., Hamberger, L. K., & Lahti, J. L. (1997). The family peace project: A model for training health care professionals to identify, treat and prevent partner violence. In L. K. Hamberger, S. Burge, A Graham, & A. Costa (Eds.), *Violence issues for health care educators and providers* (pp. 55–81). Binghamton, NY: The Haworth Press, .

Hamberger, L. K., & Phelan, M. B. (2004). *Domestic violence screening and intervention in medical and mental health settings: Research and practice*. New York: Springer.

McLeer, S.V., Anwar, R., Herman, S., & Maquiling, K. (1989). Education is not enough: A systems failure in protecting battered women. *Annals of Emergency Medicine, 18*, 651–653.

Minsky-Kelly, D, Hamberger, L. K., Pape, D. A., & Wolff, M. (2005). We've had training, now what? Qualitative analysis of barriers to domestic violence screening and referral in a health care setting. *Journal of Interpersonal Violence, 20*, 1288–1309.

World Health Organization. (1996). *WHO Global Consultation on Violence and Health. Violence: A public health priority*. Geneva, World Health Organization, Document WHO/EHA/SPI.POA.2.s

Inflammation and Traumatic Stress: A Likely Mechanism for Chronic Illness in Trauma Survivors

Kathleen Kendall-Tackett, PhD

Over the past decade, researchers in the field of psychoneuroimmunology (PNI) have made remarkable discoveries about the etiology of common chronic diseases, such as heart disease, diabetes, multiple sclerosis, and Alzheimer's. Each of these is due to increased levels of systemic inflammation (Kiecolt-Glaser et al., 2007; Pace et al., 2007; Robles et al., 2005). These findings have particular relevance to trauma survivors in that the human stress response activates inflammation. Sadly, trauma survivors have higher than average rates of many chronic diseases and often die prematurely, as the studies described below have found. An intriguing possibility is that some of this vulnerability to disease may be due to systemic inflammation.

Kathleen Kendall-Tackett, PhD

How Trauma Impacts Health

Using data from the National Comorbidity Study, researchers found that a history of childhood abuse increased the risk of cardiovascular disease. Interestingly, the link between child maltreatment and cardiovascular disease was especially strong for women, with maltreated women having a nine-fold increase in cardiovascular disease compared to non-maltreated women. The authors did not find a link between depression and cardiovascular disease; however, once child maltreatment was added to the analysis, the effect of depression disappeared. Indeed, it was trauma history, rather than depression, that accounted for the variance in cardiovascular disease (Batten et al., 2004).

Several other recent studies have found that health problems, disability, and suicidal behavior were more common in men and women with PTSD than their counterparts without PTSD. For example, analyzing data from the Canadian Community Health Survey (N = 36,984), 1% (N = 478) had a formal diagnosis of PTSD from a health care provider (Sareen et al., 2007). Even after adjusting for demographic factors and other mental illnesses, participants with PTSD had significantly higher levels of cardiovascular disease (hypertension and heart disease), respiratory

diseases (asthma and chronic obstructive pulmonary disease), chronic pain syndromes (fibromyalgia, arthritis, migraine), gastrointestinal illnesses (ulcerative colitis and ulcers), and cancer. PTSD was strongly associated with chronic fatigue syndrome and multiple-chemical sensitivity. There was no significant difference in rates of diabetes. PTSD was also associated with suicide attempts, poor quality of life, and short- and long-term disability. The authors concluded that these effects were above and beyond the effects of depression or other mental disorders and were the unique contribution of PTSD.

PTSD following a man-made disaster showed similar health effects (Dirkzwager et al., 2007). In this study, 896 survivors of a man-made disaster were surveyed at 3 weeks and 18 months after the disaster. These data were combined with health data one year before the disaster and four years after. (The disaster was an explosion of a fireworks depot that killed 23 people, injured 1,000, and forced the evacuation of 1,200 people). The authors found that PTSD was associated with physician-reported vascular, musculoskeletal, and dermatological problems. PTSD also increased risk of new vascular problems. These problems appeared even after controlling for previous health problem, smoking and demographic characteristics.

Not surprisingly, given the above-cited findings, people with PTSD use more health care services. In a study of women seeking health care at VA facilities (N = 2,578), 33% (N = 858) screened positive for PTSD (Dobie et al., 2006). The women with PTSD had more outpatient visits to the emergency department, primary care, medical or surgery subspecialities, ancillary services, and diagnostic tests. They had higher rates of hospitalizations and surgical procedures. The mean number of days in the hospital for a year was 43.4 for women with PTSD and 17 for women without PTSD. Women with PTSD were significantly younger than women without, were significantly less likely to be married, more likely to have a service-related disability, more likely to have chronic pain (e.g., irritable bowel syndrome and fibromyalgia), and more likely to be obese. They were also more likely to smoke, abuse alcohol and be depressed. Indeed, 75% of women with PTSD also screened positive for depression.

According to PNI research, many of these illnesses are due to trauma-related changes in the stress response. Severe or overwhelming stress alters and dysregulates the key systems that are designed to protect our lives. To understand these findings, it's helpful to review the three systems that respond to a perceived threat. These are described below.

How Humans Respond to a Perceived Threat

Human bodies have a number of interdependent mechanisms in place designed to preserve our lives when we perceive danger. The human stress response is highly complex. But in a simplified form, it can described as having three components: catecholamine, HPA Axis, and immune response.

The sympathetic nervous system responds first by releasing catecholamines (norepinephrine, epinephrine, and dopamine). This is the fight-or-flight response. The hypothalamic-pituitary-adrenal (HPA) axis responds with a chemical cascade: the hypothalamus releases corticotrophin releasing hormone (CRH), which causes the pituitary to release adrenocorticotropin hormone (ACTH), which causes the adrenal cortex to release cortisol, a glucocorticoid.

A third part of the process is the immune response. One way the immune system responds to threat is by increasing inflammation through the production of proinflammatory cytokines. Cytokines are proteins that regulate immune response. Proinflammatory cytokines increase inflammation and serve the adaptive purpose of helping the body heal wounds and fight infection. A key finding of PNI research is that both physical and psychological stress can trigger the inflammatory response. In these studies, researchers generally measure three markers of inflammation in the plasma: proinflammatory cytokines, C-reactive protein, or fibrinogen.

The human stress response has a number of checks and balances built in to ensure that various components do not become overactive. Unfortunately, in the case of severe or chronic stress, the normal checks and balances fail. When they do, humans become vulnerable to disease. McEwen (2003) noted that physiologic mediators of the stress response (e.g., catecholamines, glucocorticoids, and cytokines) have an important role in allostasis—maintaining homeostasis through change. However, these mediators create wear on the system—or allostatic load—when they are "on" long-term or when overused during a challenge. Indeed, overuse can damage tissues and organs. It is this failure that leads to a series of physiologic consequences, such as sleep disruptions; changes to brain structures, such as the hippocampus and prefrontal cortex; bone mineral loss; abdominal obesity; and increased risk of cardiovascular disease.

Inflammation in Trauma Survivors

There have been numerous studies during the past 10 years examining trauma's impact on two parts of the stress response: catecholamine and HPA axis. In both cases, researchers have found that trauma dysregulates these physiologic processes. Only recently have researchers examined the impact of trauma on the inflammatory response. Although a relatively new area of study, several researchers have found that traumatic events increase levels of proinflammatory cytokines in trauma survivors. The increase in inflammation likely mediates the relationship between trauma and health problems.

Childhood maltreatment was shown to affect clinically relevant levels of C-reactive protein when measured 20 years later in abuse survivors (Danese et al., 2007). The participants (N = 1,037) were part of the Dunedin Multidisciplinary Health and Development Study, a study of health behavior in a complete birth cohort in Dunedin, New Zealand. Participants were assessed every two to three years throughout childhood, and every five to six years through age 32. The effect of child maltreatment on inflammation was independent of co-occurring life stresses in adulthood, early life risks, or adult health or health behavior. Along these same lines, white blood cell count and fibrinogen were also elevated in those

continued on p. 14

continued from p. 13

who experienced childhood physical or sexual abuse. Severity of abuse was related, in a dose-response way, to severity of inflammation.

In a study of intimate partner violence (IPV), 62 women who had been in abusive relationships were compared with women who had not (Woods et al., 2005). The researchers found that interferon-γ (IFN-γ) levels, another inflammation marker, were significantly higher in abused versus non-abused women, and in women with current PTSD symptoms versus women without PTSD. Fifty-two percent of women in the IPV-group reported depression, and 39% had high levels of PTSD symptoms. The level of IFN-γ was mediated by PTSD symptoms in this sample, and was not related to other potential confounding variables. These findings also demonstrated the lingering health effects of intimate partner violence in women who experienced violence 8 to 11 years previously, yet were still manifesting significant physical symptomotology.

Immune parameters were also altered in a study of rape (Groër et al., 2006). In this study, 15 women who had been raped were compared with 16 women who had not been sexually assaulted on levels of immune markers. Women who had been raped were assessed 24 to 72 hours after their assault. The findings revealed that women who had been sexually assaulted had higher cytotoxic cells and proinflammatory biomarkers than the control group. Specifically, the sexually assaulted women had higher ACTH, C-reactive protein, IL-6, IL-10, IFN-γ than women in the control group. In addition, the assaulted women had lower B lymphocyte counts and decreased lymphocyte proliferation. The researchers interpreted their findings as indicating that sexual assault activated innate immunity and suppressed some aspects of adaptive immunity. If these long-term alterations persist, they could lead to health problems in rape survivors.

In a sample of 14 otherwise healthy patients with PTSD and a matched group on age and gender of 14 patients without PTSD, von Kanel and colleagues (2006) investigated blood coagulation. They noted that PTSD increases risk of cardiovascular disease and that the mechanism for this relationship is not well understood. In this study, they investigated whether PTSD was associated with coagulation activity by measuring various clotting factors (FVII:C, FVIII:C, FXII:C), fibrinogen, and D-dimer in the plasma. They found that FVIII:C was predicted by hyperarousal severity and overall PTSD severity. In patients with PTSD, hyperarousal and overall PTSD severity predicted fibrinogen. The more severe the PTSD, the greater the concentration of these coalgulation factors. They concluded that PTSD may elicit hypercoagulability, even at subthreshold levels, and this may increase risk for cardiovascular disease in trauma survivors.

Summary

Trauma has a demonstrated negative impact on health. PNI research indicates that one possible mechanism by which trauma increases risk of illness is its effects on systemic inflammation. Fortunately, inflammation can be specifically and easily addressed in health care settings, and a wide range of treatments specifically lower inflammation. With information from these studies, practitioners can address both the mental and physical health issues of trauma survivors—increasing their health and longevity in the process.

Kathleen Kendall-Tackett, PhD, is a Research Associate at the Family Research Laboratory, University of New Hampshire, and Division 56 Secretary. This article is excerpted from Kendall-Tackett, K. A. (Ed.). (in press). The psychoneuroimmunology of chronic disease. American Psychological Association.

References

Batten, S. V., Aslan, M., Maciejewski, P. K., & Mazure, C. M. (2004). Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. *Journal of Clinical Psychiatry, 65*, 249–254.

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study. *Proceedings of the National Academy of Sciences U S A, 104*(4), 1319–1324.

Dirkzwager, A. J., van der Velden, P. G., Grievink, L., & Yzermans, C. J. (2007). Disaster-related posttraumatic stress disorder and physical health. *Psychosomatic Medicine, 69*(5), 435–440.

Dobie, D. J., Maynard, C., Kivlahan, D. R., Johnson, K. M., Simpson, T., David, A. C., et al. (2006). Posttraumatic stress disorder screening status is associated with increased VA medical and surgical utilization in women. *Journal of General Internal Medicine, 21*(Suppl. 3), S58–S64.

Groer, M. W., Thomas, S. P., Evans, G. W., Helton, S., & Weldon, A. (2006). Inflammatory effects and immune system correlates of rape. *Violence and Victims, 21*(6), 796–808.

Kiecolt-Glaser, J. K., Belury, M. A., Porter, K., Beversdoft, D., Lemeshow, S., & Glaser, R. (2007). Depressive symptoms, omega-6: Omega-3 fatty acids, and inflammation in older adults. *Psychosomatic Medicine, 69*, 217–224.

McEwen, B. S. (2003). Mood disorders and allostatic load. *Biological Psychiatry, 54*, 200-207.

Pace, T. W., Hu, F., & Miller, A. H. (2007). Cytokine-effects on glucocorticoid receptor function: Relevance to glucocorticoid resistance and the pathophysiology and treatment of major depression. *Brain, Behavior and Immunity, 21*(1), 9–19.

Robles, T. F., Glaser, R., & Kiecolt-Glaser, J. K. (2005). Out of balance: A new look at chronic stress, depression, and immunity. *Current Directions in Psychological Science, 14*, 111–115.

Sareen, J., Cox, B. J., Stein, M. B., Afifi, T. O., Fleet, C., & Asmundson, G. J. (2007). Physical and mental comorbidity, disability, and suicidal behavior associated with posttraumatic stress disorder in a large community sample. *Psychosomatic Medicine, 69*(3), 242–248.

von Kanel, R., Hepp, U., Buddeberg, C., Keel, M., Mica, L., Aschbacher, K., et al. (2006). Altered blood coagulation in patients with posttraumatic stress disorder. *Psychosomatic Medicine, 68*(4), 598–604.

Woods, A. B., Page, G. G., O'Campo, P., Pugh, L. C., Ford, D., & Campbell, J. C. (2005). The mediation effect of posttraumatic stress disorder symptoms on the relationship of intimate partner violence and IFN-gamma levels. *American Journal of Community Psychology, 36*(1-2), 159–175.