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# TRAUMA AND HEALTH Why Trauma Makes People Sick: Inflammation, Heart Disease and Diabetes in Trauma Survivors

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Trauma survivors have higher than average rates of serious illness including heart disease, diabetes and metabolic syndrome, the precursor to type 2 diabetes (Batten et al., 2004; Felitti et al., 2001; Kendall-Tackett & Marshall, 1999). The intriguing question is why this is so. One possible explanation is the connection between disease and inflammation—specifically, elevated levels of proinflammatory cytokines. Cytokines are proteins that regulate immune response and proinflammatory cytokines help the body heal wounds and fight infection. But there can be too much of a good thing; chronic inflammation is a likely cause of a wide range of illnesses including heart disease, diabetes, Alzheimer's disease, and even cancer (Batten et al., 2004; Robles et al., 2005; Suarez, 2006).

So why would proinflammatory cytokines be elevated in trauma survivors? Low levels of cortisol, which are common in trauma survivors, can allow inflammation to go unchecked since cortisol generally regulates the inflammatory response. Another possibility is that cytokines increase in the wake of two common trauma sequelae—depression and hostility. Depression and hostility act as stressors, and increase inflammation and subsequent risk of disease. These can affect survivors' health long after the trauma has ended.

## Depression, Inflammation and Health

Depression is one of the most commonly occurring sequela of trauma (Kendall-Tackett, 2003). But it's one we tend to think of it as an outcome—an endpoint we measure in the wake of traumatic events. Yet depression can also be a mechanism that leads to poor health. The negative impact of depression is well known in the cardiovascular literature. Patients who become depressed after a heart attack are two to three times more likely to have another one and are three to four times more likely to die (deJong et al., 2006; Lesperance & Frasure-Smith, 2000). And inflammation is the likely culprit (Kiecolt-Glaser et al., 2007).

In depressed people, there are several biomarkers of increased inflammation including acute-phase proteins, such as C-reactive protein (CRP; Kop & Gottdiener, 2005; Robles, Glaser, & Kiecolt-Glaser, 2005), and proinflammatory cytokines. The proinflammatory cytokines that have been identified in most studies of depressed people are interleukin-1 $\beta$  (IL-1 $\beta$ ), interleukin-6 (IL-6), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) and more recently, interferon- $\gamma$  (IFN- $\gamma$ ; Kiecolt-Glaser et al., 2007; Robles et al., 2005). Researchers hypothesize that chronic inflammation increases the risk of heart disease by damaging blood vessels, reducing the stability of plaque, and increasing the risk of acute episodes (e.g., Kop & Gottdiener, 2005).

In summary, depression raises inflammation and is generally bad for people's health. It alone could explain many

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of the health effects of trauma. But unfortunately, depression is not the only mental state that increases the risk of disease. Hostility is another common sequela of trauma that leads to poor health. Its effects are described below.

## Hostility and Trauma

For people with a hostile world view, life is not benign. People high in trait hostility don't trust others, are suspicious and cynical about human nature, and tend to interpret the actions of others as aggressive (Smith, 1992). And hostility is a common response among trauma survivors. In a sample from primary care, 52% of female sexual abuse survivors indicated that they could not trust others compared with 17% of the non-abused women (Hulme, 2000). In a community sample, approximately half of sexual abuse survivors indicated that their views of themselves and others were very negative. And in a sample of 90 women veterans (Butterfield, Forneris, Feldman, & Beckham, 2000), women with PTSD reported significantly higher levels of hostility and had poorer health than women without PTSD.

## The Health Effects of Hostility

Hostility is a reaction that may have been adaptive at one point, and served to protect the survivor from further danger. However, hostility has a well-documented negative impact on health. Hostility increases physiological arousal because of the way hostile people interpret the world (Kiecolt-Glaser & Newton, 2001). This reaction increases both the risk of cardiovascular disease and diabetes. In their review, Smith and Ruiz (2002) noted that people who are high in trait hostility are more prone to ischemia and constriction of the coronary arteries during mental stress. Trait hostility predicted new coronary events in previously healthy people. And for patients who already have coronary heart disease, hostility sped-up progression of the disease.

Hostility also increased levels of proinflammatory cytokines (IL-1 $\alpha$ , IL-1 $\beta$ , IL-8 and TNF- $\alpha$ ) in a study of 44 healthy, non-smoking, premenopausal women (Suarez et al., 2004). The combination of depression and hostility was especially deleterious, and there was a dose-responsive effect: the more severe the depression and hostility, the greater the production of cytokines. A study with men had similar results (Suarez, 2003). The author noted that increased levels of IL-6 predicted both future risk of cardiac events and all-cause mortality, and hypothesized that IL-6 may mediate the relationship between hostility and these health problems.

Hostility also increases the risk of metabolic syndrome. In a three-year follow-up of 134 white and African American teens, hostility at Time 1 predicted risk factors for metabolic syndrome at Time 2 (Raikkonen, Matthews, & Salomon, 2003). These risk factors were at the 75<sup>th</sup> percentile for age, gender and race and included BMI, insulin resistance, ratio of triglycerides to HDL cholesterol, and mean arterial blood pressure.

More recently, Suarez (2006) studied 135 healthy patients (75 men, 60 women) with no symptoms of diabetes. He found that women with higher levels of depression and

hostility, and who had a propensity to express anger, had higher levels of fasting insulin, glucose, and insulin resistance. These findings were not true for men and they were independent of other risk factors for metabolic syndrome including BMI, age, fasting triglycerides, exercise regularity, or ethnicity. The author indicated that these findings were significant since pre-study glucose levels were in the non-diabetic range. The author noted that inflammation, particularly elevated IL-6 and C-reactive protein, may mediate the relationship between depression and hostility, and risk of type 2 diabetes and cardiovascular disease, possibly because they increase insulin resistance.

## Anti-Inflammatory Treatment Approaches

The studies cited above indicate that two common trauma sequelae—depression and hostility—appear to increase inflammation and impair health. The inflammation-health connection raises at least the possibility that reducing inflammation may help lessen the severity of symptoms. The depression literature already indicates that many of the effective treatments for depression are also anti-inflammatory, and this may be another mechanism for their efficacy. For example, the selective serotonin reuptake inhibitor (SSRI) class of antidepressants have been found to lower levels of C-reactive protein in cardiac patients with major depression (O'Brien et al., 2006). This anti-inflammatory effect was independent of whether depression resolved in these patients.

Even cognitive therapy, a treatment with well-established efficacy, is arguably anti-inflammatory (Rupke et al., 2006). Two recent studies have demonstrated that negative beliefs, such as hostility, can increase the levels of proinflammatory cytokines—especially IL-6 (Kiecolt-Glaser et al., 2005; Suarez et al., 2004). The primary goal of cognitive therapy is to reduce negative cognitions. Since negative cognitions increase inflammation, reducing their occurrence should reduce inflammation.

## Omega-3 Fatty Acids, Inflammation and Health

In my view, some of the more promising work, with potential application to trauma survivors, is research on the health effects of long-chain Omega-3 fatty acids: EPA and DHA. EPA and DHA are anti-inflammatory and lower levels of proinflammatory cytokines. A recent large population study found that people with high blood levels EPA and DHA had low levels of IL-6, IL-1, TNF- $\alpha$  and lower levels of C-reactive protein. The opposite was true for people with low EPA/DHA in their blood (Ferrucci et al., 2006). Another study of older adults found that the combination of depressive symptoms and low blood levels of Omega-3s enhanced production of IL-6 and TNF- $\alpha$  (Kiecolt-Glaser et al., 2007). These are the same cytokines that are high in depression and hostility and that likely have a relation to heart disease and diabetes.

EPA and DHA may also protect mental health. High levels of EPA and DHA increased resilience to laboratory-induced psychological stressors in college students and attenuated the proinflammatory response (Maes et al., 2000). In population studies, populations with higher levels of EPA and DHA in their diets (usually from eating fatty fish) had lower levels of major depression (Tanskanen et al., 2001), postpartum depression (Hibbeln, 2002), bipolar disorder (Noagliul &

Hibbeln, 2003), and even future suicide risk (Sublette et al., 2006).

Similar findings have been noted in randomized clinical trials, where researchers have given either EPA/DHA supplements or a placebo to people currently receiving treatment for unipolar or bipolar depression. Two recent studies added EPA to patients' normal regimen of antidepressants and found that EPA made the antidepressants more effective in treating depression than the placebo (Nemets et al., 2002; Peet & Horrobin, 2002). Similarly, in a study of childhood depression, children who received EPA and DHA in addition to their medications had significantly improved depression compared with children who received their meds and a placebo (Nemets et al., 2006). And EPA also helped stabilize symptoms of bipolar disorder in a 12-week double-blind trial (Frangou et al., 2006).

Although these findings are preliminary, treatments that are anti-inflammatory show promise as primary or adjunct treatments in trauma survivors. Although cognitive therapy and antidepressants have been used successfully with trauma survivors (Kendall-Tackett, 2003), to my knowledge, EPA and DHA have not been tried. But this may prove to be an effective addition to our treatment regimens and would be a fruitful avenue to explore.

### Overall Summary

Depression and hostility are common sequelae of trauma and violence. In addition to their negative impact on day-to-day functioning, they can also act as chronic stressors in trauma survivors. Both of these can have a profound impact on health, in part, by raising levels of proinflammatory cytokines. Treatments that reduce inflammation show promise in alleviating depressive and trauma symptoms, and also in decreasing the risk of subsequent health problems.

*This paper is part of a Division 56 Symposium that will be presented at the 2007 APA Convention: Traumatic Stress, Cardiovascular Disease, Metabolic Syndrome and Neurodegenerative Disease. Session co-presenters include Kathleen Kendall-Tackett, PhD; Jeff Kibler, PhD; Mary Meagher, PhD; James Flatt, PhD; and Robert Geffner, PhD.*

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# Shifting the Paradigm: Trauma Psychologists' Role in Viewing Combat Stress Injuries as Opportunities for Mental Illness Prevention<sup>1</sup>

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As trauma psychologists we have an obligation to understand and help the traumatized using best practices. Thanks to Terry Keane, other members of Division 56, and a growing number of science practitioners, the paradigm for helping military combatants is shifting. This article talks about these changes in the context of understanding combat stress injuries, the role of the National Symposium and the associated book (Figley & Nash, 2006) in articulating these changes, and the major role trauma psychologists in bring about this new paradigm.

### Combat Stress Injuries

I have been investigating the immediate and long-term psychosocial consequences of combat for the combatants since 1971. Like other members of the Trauma Psychology Division, I was extremely skeptical about the decision to commit American forces to a war in Iraq. No matter what you think of today's wars being fought by the US military, these warfighters represent less than 1% of the US population.<sup>2</sup> They deserve our respect and our help. One way of helping is for the other 99% of the US to be more aware of what these men and women are going through and how best to help them during and following deployment. The 2nd National Symposium will increase awareness. At the same time we must all keep in mind that our efforts are for those who risked their lives for all of us; "... for those who bore the

battle. . ."<sup>3</sup>

The concept of combat stress injuries<sup>4</sup> is an important distinction. Mental health diagnostic labels can harm both warfighters and the military units they serve within. Navy Captain Bill Nash, MD, who is co-chair of the Symposium and co-editor of *Combat Stress Injuries*, makes the point in Chapter 3; that there are major problems associated with medicalizing and pathologizing operational stress problems. Stress injuries have been kept separate from the physical injuries or wounds. He points out that if given any label at all, they have been classified as having something benign like "battle fatigue," "exhaustion," or "combat stress reaction." The avoidance of labeling and a focus on normalization have also long been central to civilian crisis management efforts. The Israeli Defense Force has always used Combat Stress Reaction (CSR), for example, which is discussed by Zahava Solomon (1993) in her book by the same name.<sup>5</sup> But there are limitations for normalizing what might be acute dysfunction with long-term negative consequences unless the right action is taken, rather than simply returning the injured warfighters to battle or discharging them to fend for themselves as a civilian.

As with any injury, complications may set in. In the case of combat stress injuries, the complications may be a stress disorder, depression, substance abuse, family violence, homicide, and suicide. Further, Dr. Nash suggests that combat stress injuries can be divided into three categories depending upon the source of the stress: (1) stress fatigue, caused by the wear-and-tear of accumulated stress; (2) grief stress,