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How Stress Influences Disease: Study Reveals Inflammation as the Culprit

ScienceDaily (Apr. 2, 2012) — Stress wreaks havoc on the mind and body. For example, psychological stress is associated with greater risk for depression, heart disease and infectious diseases. But, until now, it has not been clear exactly how stress influences disease and health.

A research team led by Carnegie Mellon University's Sheldon Cohen has found that chronic psychological stress is associated with the body losing its ability to regulate the inflammatory response. Published in the Proceedings of the National Academy of Sciences, the research shows for the first time that the effects of psychological stress on the body's ability to regulate inflammation can promote the development and progression of disease.

"Inflammation is partly regulated by the hormone cortisol and when cortisol is not allowed to serve this function, inflammation can get out of control," said Cohen, the Robert E. Doherty Professor of Psychology within CMU's Dietrich College of Humanities and Social Sciences.

Cohen argued that prolonged stress alters the effectiveness of cortisol to regulate the inflammatory response because it decreases tissue sensitivity to the hormone. Specifically, immune cells become insensitive to cortisol's regulatory effect. In turn, runaway inflammation is thought to promote the development and progression of many diseases.

Cohen, whose groundbreaking early work showed that people suffering from psychological stress are more susceptible to developing common colds, used the common cold as the model for testing his theory. With the common cold, symptoms are not caused by the virus -- they are instead a "side effect" of the inflammatory response that is triggered as part of the body's effort to fight infection. The greater the body's inflammatory response to the virus, the greater is the likelihood of experiencing the symptoms of a cold.

In Cohen's first study, after completing an intensive stress interview, 276 healthy adults were exposed to a virus that causes the common cold and monitored in quarantine for five days for signs of infection and illness. Here, Cohen found that experiencing a prolonged stressful event was associated with the inability of immune cells to respond to hormonal signals that normally regulate inflammation. In turn, those with the inability to regulate the inflammatory response were more likely to develop colds when exposed to the virus.

In the second study, 79 healthy participants were assessed for their ability to regulate the inflammatory response and then exposed to a cold virus and monitored for the production of pro-inflammatory cytokines, the chemical messengers that trigger inflammation. He found that those who were less able to regulate the inflammatory response as assessed before being exposed to the virus produced more of these inflammation-inducing chemical messengers when they were infected.

"The immune system's ability to regulate inflammation predicts who will develop a cold, but more importantly it provides an explanation of how stress can promote disease," Cohen said. "When under stress, cells of the immune system are unable to respond to hormonal control, and consequently, produce levels of inflammation that promote disease. Because inflammation plays a role in many diseases such as cardiovascular, asthma and autoimmune disorders, this model suggests why stress impacts them as well."

He added, "Knowing this is important for identifying which diseases may be influenced by stress and for preventing disease in chronically stressed people."

In addition to Cohen, the research team included CMU's Denise Janicki-Deverts, research psychologist; Children's Hospital of Pittsburgh's William J. Doyle; University of British Columbia's Gregory E. Miller; University of Pittsburgh School of Medicine's Bruce S. Rabin and Ellen Frank; and the University of Virginia Health Sciences Center's Ronald B. Turner.

The National Center for Complementary and Alternative Medicine, National Institute of Mental Health, National Heart, Lung and Blood Institute and the MacArthur Foundation Research Network on Socioeconomic Status and Health funded this research.

Story Source:

The above story is reprinted from materials provided by **Carnegie Mellon University**.

Journal Reference:

1. Sheldon Cohen, Denise Janicki-Deverts, William J. Doyle, Gregory E. Miller, Ellen Frank, Bruce S. Rabin, and Ronald B. Turner. **Chronic stress, glucocorticoid receptor resistance, inflammation, and disease risk**. *PNAS*, April 2, 2012 DOI: 10.1073/pnas.1118355109