EQUILIBRIUM Energy + Education
850 S. Wabash Ave., Suite 300 Chicago, IL 60605
(312) 786-1882 www.equilibrium-e3.com

Does Stress Feed Cancer?

A new study shows stress hormones make it easier for malignant tumors to grow and spread

By Katie Moisse -- April 13, 2010 , Scientific American

A little stress can do us good—it pushes us to compete and innovate. But chronic stress can increase the risk of diseases such as depression, heart disease and even cancer. Studies have shown that stress might promote cancer indirectly by weakening the immune system's anti-tumor defense or by encouraging new tumor-feeding blood vessels to form. But a new study published April 12 in *The Journal of Clinical Investigation* shows that stress hormones, such as adrenaline, can directly support tumor growth and spread.

For normal cells to thrive in the body, "they need to be attached to their neighbors and their surroundings," says the study's lead author Anil Sood from The University of Texas M. D, Anderson Cancer Center in Houston. Cells that detach from their environment undergo a form of programmed cell death called anoikis. "But cancer cells have come up with way to bypass this effect—they avoid anoikis," Sood says. This allows cancer cells to break off from tumors, spread throughout the body (in blood or other fluid) and form new tumors at distant sites—a process called metastasis. So Sood wondered: Could stress affect anoikis? "It surprised us that this biology hadn't been studied before," he notes. "Stress influences so many normal physiological processes. Why wouldn't it be involved in tumor progression?"

Sood and his team first studied the effects of stress hormones on human ovarian cancer cell anoikis in culture. Cells that were exposed to stress hormones were protected from self destruction—meaning they could survive without being anchored to their surroundings. The stress hormone treatment activated a protein called FAK (focal adhesion kinase), which is known to protect cells from anoikis. Inhibiting FAK reversed

But real tumors behave differently than cancer cells in vitro, so Sood and his team extended their exciting findings into a mouse model of cancer. After receiving a transplant of ovarian cancer cells, mice were restrained to cause stress. As such, their tumors grow more quickly. Isoproterenol (a drug similar to adrenaline) had the same accelerative effect. The tumor-feeding effects of behaviorally and pharmacologically induced stress, both of which were mediated by FAK, were inhibited by the adrenaline-blocking drug propranolol.

But how closely does the stress caused by restraining a lab animal mimic that experienced by human patients? Sood and colleagues looked at samples from 80 cases of human ovarian cancer grouped according to patient stress using the National Institutes of Health's Center for Epidemiological Studies Depression scale as a surrogate marker. Patient stress (according to the scale), along with elevated stress hormone activity were associated with higher levels of activated FAK, which was in turn linked to faster disease progression.

Ovaries contain high levels of stress hormones compared with other organs, but Sood plans to investigate whether the stressors could still be involved in other types of cancer. He hopes to identify ways to interfere with the tumor-feeding effects of stress hormones either behaviorally or pharmacologically. "Reducing the hormone levels may not be so easy," Sood says. "Blocking the receptors using drugs like beta-blockers or antidepressants may be a better strategy." Teaching patients to manage their stress using cognitive behavioral therapy might also be effective, he adds. "We're really trying to understand the biology. We hope it will help us identify better therapeutic strategies."

http://www.scientificamerican.com/article.cfm?id=does-stress-feed-cancer