In the NEWS

Obesity Linked to Inflammation and Heart Disease

Human fat cells produce C-reactive protein (CRP), which is linked with both increased inflammation and elevated cardiovascular disease risk, according to researchers at the University of Texas M.D. Anderson Cancer Center and the University of Texas Health Science Center in Houston.

This finding helps to explain why overweight adults tend to have elevated CRP levels, and demonstrates that body fat may participate in the inflammatory process that can lead to cardiovascular disease. Researchers had previously found that CRP is produced mainly in liver tissue, and more recent findings indicate that blood vessel walls also produce this protein. However, these findings did not explain why individuals with metabolic disorders or obesity exhibit high CRP levels, which are associated with a greater risk of heart disease or stroke.

To study the relationship between fat cells and CRP, the investigators used human adipose tissue donated by plastic surgery patients. Fat cells were isolated, cultured, and stimulated under different conditions. The cultured fat cells produced cytokines that resulted in inflammation and triggered production of high levels of CRP. When exposed to resistin, a hormone associated with diabetes and insulin resistance, the fat cells also produced CRP. This is particularly interesting, as fat cells themselves produce resistin.

The investigators then sought to explore why certain drugs, including aspirin and cholesterol-lowering statins, can lower CRP. When fat cells producing high levels of CRP were exposed to these medications, CRP production declined.

"This study is the first to show how body fat participates in the inflammatory process that leads to cardiovascular disease, but also demonstrates that this process can be blocked by drugs now on the market," said study leader Edward T.H. Yeh, MD. "Inflammation is a very complicated process, but at least now we have a few more clues as to what it does and how the damage it produces can be prevented."

—Elizabeth Wagner, ND

References

APIGENIN INHIBITS PROSTATE CANCER GROWTH

Apigenin, a dietary flavonoid commonly found in celery, parsley, garlic, bell peppers, and guava, inhibits prostate cancer cell growth, according to a recent report. This new finding supports epidemiological evidence linking a diet rich in fruits and vegetables to a reduced risk of prostate cancer.

In this investigation, scientists transplanted an androgen-dependent human prostate cancer cell line into mice bred to serve as a model for tumor growth conditions. A liquid suspension containing either apigenin or placebo was administered to the mice via a gastric tube daily for eight weeks. Prostate cancer cells were inoculated into the mice either two weeks before or two weeks after apigenin administration commenced. Tumor growth was measured twice weekly following transplantation; tumors were then excised and weighed at the study's end. In parallel experiments, prostate cancer cells were cultured in the presence of apigenin, and cell viability was determined.

Administering apigenin to mice, either before or after inoculation, inhibited the volume of prostate cancer cells in a dose-dependent manner by as much as 50% and 53%, respectively. Together, these results suggest that apigenin partially interferes with the establishment of tumors and slows the growth of established tumors. Similarly, exposure of prostate cancer cells in culture to apigenin for as little as 48 hours resulted in growth inhibition of up to 67%. No adverse effects were associated with apigenin administration.

Nutritional strategies to help avert cancer may be especially important in late-onset, slow-growing tumors such as prostate cancer. Since Americans consume an average of only 13 mg per day of apigenin, or approximately three to four and one-half times less than the lowest comparable dose used in this study, increasing daily apigenin intake may be a prudent dietary strategy for protecting against prostate cancer.

—Linda M. Smith, RN

Reference