Clinical Capsules

Fiber Not a Factor in Colorectal Cancer

Dietary fiber intake showed no effect on the risk of colorectal cancer in an analysis that pooled data from 13 prospective cohort studies, reported Dr. Yikyung Park of Harvard School of Public Health, Boston, and associates.

Each of the 13 studies that Dr. Park and associates identified for their analysis included at least 50 cases of colorectal cancer and adequately assessed dietary fiber intake. The pooled data yielded over 7,300,000 person-years of follow-up (6-20 years of follow-up across the studies), and included incident colorectal cancer cases in 2,776 men and 5,305 women. The analysis had a level of statistical power such that "a substantial effect of fiber is unlikely to have been missed," the investigators said (JAMA 2005;294:2849-57).

The pooled analysis was undertaken because the results of many epidemiologic studies and randomized clinical trials have been conflicting, and there is still much debate over whether dietary fiber decreases colorectal cancer risk.

The initial, age-adjusted analysis showed a significant link between fiber and reduced colorectal cancer risk, with the highest levels of intake associated with a 16% lower risk than the lowest fiber intakes. However, after the data were adjusted for potential confounders including nondietary factors, milk and red meat intake, and alcohol consumption, "only a nonsignificant weak inverse association was found," they said. In addition, dietary fiber showed no effect on cancer risk when the data were categorized by subjects' body mass index or by specific food sources of fiber.

COX-2 Inhibitors vs. NSAIDs for Safety

Cyclooxygenase-2 inhibitors were found to be no safer than nonselective NSAIDs in averting adverse gastrointestinal events in a large observational study conducted in the United Kingdom.

The finding is important "given that enhanced gastrointestinal safety has been one of the main justifications for these drugs," wrote Dr. Julia Hippisley-Cox and her associates at the University of Nottingham (England). The COX-2 inhibitors were developed to relieve pain without inducing the GI side effects common with NSAIDs, but data on their long-term safety are lacking.

The researchers calculated the risk of GI events in patients who took these prescription pain relievers between 2000 and 2004. After reviewing the medical records in a database of more than 7 million patients treated at general practices throughout England, Scotland, and Wales, the investigators identified 9,407 who had an adverse GI event and matched them with more than 88,000 controls who had no such events (BMJ 2005;331:1310-6).

Patients with GI events were more likely to have taken either prescription NSAIDs (odds ratio 1.69) or prescription COX-2 inhibitors (odds ratio 1.89) than were control subjects.

The researchers also found that the concomitant use of ulcer-healing drugs reduces the GI risks of COX-2 inhibitors, which suggests "that there is some risk to protect against." Taken together, these results indicate that COX-2 inhibitors "may not be as safe as originally thought," the authors said.

Insulin and Pancreatic Cancer Risk

Both high insulin concentrations and insulin resistance appear to be associated with increased risk of pancreatic cancer, reported Rachael Z. Stolzenberg-Solomon, Ph.D., of the National Cancer Institute, Rockville, Md., and her associates.

In a large study of pancreatic cancer in male smokers, men who self-reported that they had diabetes had a twofold increase in risk for the malignancy. The investigators used the same patient population to further examine the issue, assessing whether fasting serum insulin concentrations predicted development of pancreatic cancer.

The subjects included more than 29,000 Finnish men ages 50-69 years at study entry (1985-1988). Baseline serum insulin and glucose levels were analyzed in 169 men who developed pancreatic cancer during a mean of 16 years of follow-up and in 400 randomly selected participants without pancreatic cancer who served as controls.

After the data were adjusted for age, years of smoking, and body mass index, the risk for pancreatic cancer was found to rise as insulin levels increased and as the degree of insulin resistance worsened. Subjects with established diabetes and the highest insulin levels were at twice the risk for pancreatic cancer as controls, the investigators said (JAMA 2005;294:2872-8).

Although researchers have noted an association between diabetes and pancreatic cancer for some time, it is unclear whether diabetes somehow contributes to carcinogenesis in the pancreas or is instead a result of subclinical pancreatic malignancy. In this study, Dr. Stolzenberg-Solomon and her associates included only cases of pancreatic cancer that developed 5 years or more after serum samples were collected.

—Mary Ann Moon

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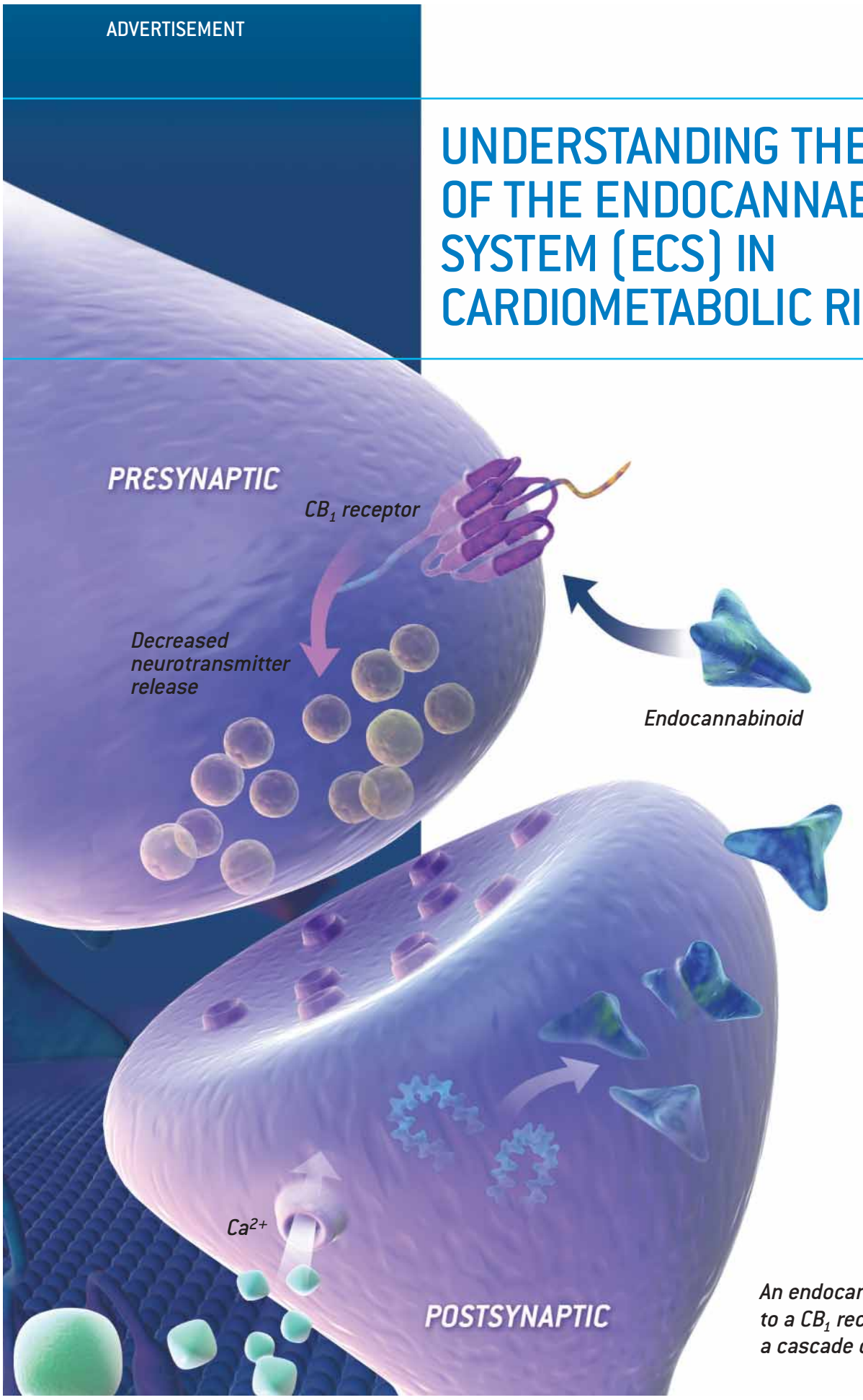
UNDERSTANDING THE ROLE OF THE ENDOCANNABINOID SYSTEM (ECS) IN CARDIOMETABOLIC RISK



An endocannabinoid binds to a CB_1 receptor, triggering a cascade of events.