



The independent newsletter that reports vitamin, mineral, and food therapies

Opinion: Time to Boycott "Addiction, Inc."

If there's one outfit you ought to boycott, it's Philip Morris, the company I call "Addiction, Inc."

Philip Morris, of course, is the huge tobacco and cigarette company that's been selling "cancer sticks," denying the harm of cigarettes, and killing people for profit for decades.

These days, trying hard to look socially responsible, Philip Morris supports fine art exhibitions, helps abused women, and provides humanitarian aid to victims of natural disasters.

Yet, in my opinion, it's all "dirty money." The company's business model seems to be built mostly around addictive substances.

When Philip Morris decided earlier this year to buy Nabisco, one of the biggest makers of junk food, a newspaper cartoonist depicted a couple of children commenting on how addictive Oreo cookies were. It was funny, but it also appeared all too true.

So Philip Morris isn't trying to hook you on just tobacco. It's also doing the same with sugar.

That's not all. Philip Morris also owns Miller beer. And so, the company doesn't just try to addict you to cigarettes and sugar. It's trying to do the same with booze.

Philip Morris owns Kraft as well, one of the largest makers of processed and refined foods—basically, foods of dubious nutritional value. The Kraft brands include Oscar Mayer, Minute Maid, Kool Aid, Philadelpha cream cheese, Velveeta, Miracle Whip, Post cereals, Shake'N Bake, Jello-O, and Maxwell House.

Trying to show its social conscience, Philip Morris has been running full-page newspaper ads touting how it helps feed the hungry. Over the past 10 years, the company donated \$350 million in money and food—"including many of our brands like Kraft Macaroni & Cheese, Post Cereals, and Jell-O—to feed the hungry."

Just what poor, hungry people need—Jell-O and junk foods.

Fifty years ago, the world put Nazis on trial for atrocities described as crimes against humanity. In the 1990s, the tobacco companies were dragged in front of Congress and, in the courts, fined big time for knowingly selling cancer-causing products. It's my wish that, someday, executives of the Philip Morris family of companies—and all the other junk food and fast food companies—are held responsible for how their products damage the health of millions of people. I believe that kind of profiteering is also a crime against humanity.

In the mean time, I urge you to do what I have done: stop buying products from the Philip Morris family of companies.

More Encouraging News About Vitamin E Reducing Heart Disease Risk

In September, *The Nutrition Reporter*TM related growing evidence describing the antiinflammatory properties of vitamin E. Inflammation, of course, underscores many diseases, such as infections and rheumatoid arthritis. Increasingly, researchers believe that runaway inflammation also sets the stage for coronary artery disease, the leading cause of death in the Western world.

Inflammation results from activated immune cells attacking the body's own tissues as well as infectious viruses and bacteria. This immune response generates cell-damaging free radicals, and the free radicals further promote the inflammatory process.

The latest research, by Ishwarlal "Kenny" Jialal, MD, and Sridevi Devaraj, PhD, of the University of Texas Southwestern Medical Center, Dallas, clearly demonstrates that natural vitamin E supplements can signficantly reduce levels of C-reactive protein, a marker of inflammation. Elevated C-reactive protein levels are associated with a 4.5 greater risk of having a heart attack.

Jialal and Deveraj studied 72 subjects, which included adult-onset diabetics with cardiovascular disease, diabetics without cardiovascular disease, and healthy control subjects. After taking initial measurements of their C-reactive protein and interleukin-6 levels, the researchers asked all of the subjects to take

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1,200 IU of natural vitamin E (d-alpha tocopherol) daily for three months.

C-reactive protein is a byproduct of interleukin-6, a proinflammatory hormone-like compound called a cytokine. C-reactive protein is made from interleuken-6 in both the liver and in fat cells.

At the start of the study, the diabetic patients – especially those with cardiovascular disease – had increased levels of C-reactive protein. The vitamin E reduced C-reactive protein levels by 30 percent and interleukin-6 levels by about 50 percent in both diabetic groups and among the healthy subjects.

A normal level of C-reactive protein is less than 1.1 mg per deciliter of blood (the lower the better). Levels above 2 mg and especially above 4 mg indicate a greater risk of heart disease.

In a separate study, Jialal and Devaraj, asked 50 kidney dialysis patients to take 800 IU of natural vitamin E or a placebo daily for 12 weeks. The patients had chronic kidney failure and were on either hemodialysis or peritoneal dialysis. Blood was drawn from the patients and its low-density lipoprotein (LDL) content was separated and exposed to free radicals. Free-radical oxidized LDL is an early step in the development of heart disease.

LDL from patients taking vitamin E became significantly more resistant to oxidation. The supplements reduced LDL oxidation in patients undergoing both types of dialysis, but those on peritoneal dialysis benefited more. In effect, vitamin E may slow the progression of heart disease in patients with kidney failure.

References: Devaraj S, Jialal I. Alpha tocopherol supplementation decreases serum C-reactive protein and monocyte interleukin-6 levels in normal volunteers and type 2 diabetic patients. Free Radical Biology & Medicine, 2000; 29:790-792. Islam KN, O'Byrne D, Devaraj S, et al. Alpha-tocopherol supplementation decreases the oxidative susceptibility of LDL in renal failure patients on dialysis therapy. Atherosclerosis, 2000;150:217-224.

Low Levels of CoQ10 Levels May Be **Related to Breast Cancer Risk**

Coenzyme Q10, a vitamin-like nutrient, plays a key role in energy production, as well as an antioxidant, in cells. In the 1990s, several reports noted that supplements (390 mg daily) prevented the recurrence of breast cancer.

In the latest study, Turkish researchers analyzed CoQ10 levels in cancerous breast tissue and in noncancerous surrounding tissue in 21 patients. They found that CoQ10 levels were "significantly decreased" in cancerous tissues.

Although the researchers attributed the low

CoQ10 levels to free radical stresses, they may actually be indicative of defects in cellular energy production.

Reference: Portakal O, Ozkaya O, Inal ME, et al. Coenzyme Q10 concentrations and antioxidant status in tissues of breast cancer patients. Clinical Biochemistry, 2000;33:279-284.

Human Study Find that Herbal PC-SPES Beneficial in Prostate Cancer

Herbal PC-SPES is an over-the-counter supplement used in the treatment of prostate cancer. It's composed of eight Chinese herbs that cell studies have found to enhance immune function and inhibit cancer cell growth.

In a recent study, American and French researchers gave PC-SPES supplements (320 mg three times daily) to 69 men with prostate cancer. After two months, levels of prostate specific antigen (PSA), a marker of prostate disease, decreased in 82 percent of the men. After one year, 88 percent of the men had reduced levels of PSA.

Reference: de la Taille A, Buttyan R, Hayek O, et al. Herbal therapy PC-SPES: in vitro effects and evaluation of its efficacy in 69 patients with prostate cancer. Journal of Urology, 2000;164:1229-1234.

Diabetes Skyrockets in 1990s

The overall incidence of adult-onset diabetes grew by 33 percent between 1990 and 1998, and by 70 percent among Americans in their 30s.

According to the study, about 3.7 percent of people ages 30-39 had diabetes in 1998, compared with only 2.1 percent at the start of the decade.

The incidence of diabetes almost doubled in Minnesota and increased by 87 percent in Illinois. It increased by more than 64 percent in California, Colorado, and Michigan.

Reference: Mokdad AH, Ford ES, Bowman BA. Diabetes Trends in the U.S.: 1990-1998. Diabetes Care, 2000;23:1278-1283.

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