But is it food?

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The past year saw an acceleration of the trend towards products halfway between foods and pharmaceuticals. Sucrose polyester looks and tastes like fat, but it is not absorbed because it contains bonds between sucrose and fatty acids that cannot be hydrolysed by pancreatic lipases. Critics questioned whether the reduction in fat intake would make people lose weight, and worried about losses of fat-soluble vitamins (N Engl J Med 1996; 335: 669).

Another hitech food came from Finland. Miettinen et al found that converting the plant sterol sitosterol into sitostanol ester made it an effective cholesterol-lowering agent; a 10% suspension in margarine lowered LDL cholesterol by 13%. The margarine is a commercial success, but is it a food?

Vitamin supplements are another example of “nutraceuticals”. The CHAOS trial (Stephens) provided the first experimental evidence that vitamin E can reduce coronary disease (though mortality outcomes were still worrying). Carotene supplements fared less well. Carotene intake and plasma carotene levels are inversely associated with risk of cancer in epidemiological studies, but randomised trials of the effect of high-dose carotene supplements on risk of lung cancer in smokers yielded disappointing results: the CARET trial (Omenn) had to be stopped early because of a raised incidence of cancer, confirming earlier findings (Lancet 1996; 347: 249). In a low-risk population, total cancer incidence was unaffected by carotene supplements (N Engl J Med 1996; 334: 145).

The hypothesis that eating less fat will reduce the risk of breast cancer received a blow from a pooled analysis of seven prospective studies including almost 5000 cases; no association with dietary fat was seen over a wide range of intake (Hunter).

However, the association between high intakes of fruits and vegetables and reduced risk of cancer remains firm. Hollman et al defeated conventional wisdom by showing that human beings absorb flavonoid glycosides (Free Rad Biol Med 1996; 21: 73), but evidence that flavonoids protect against heart disease is still inconsistent (Ann Intern Med 1996; 125: 364, BMJ 1996; 312: 458). We should not ignore the potential of bioactive compounds to do harm, as testified by the adverse effects of coffee diterpenes on blood lipids and transaminases (Urgert). Both beneficial and adverse effects are modulated by nutrient gene interactions, and this will inevitably produce demands to tailor nutritional advice to genetic susceptibility. But should we know whether we have the proper allele of N-acetyltransferase before enjoying our barbecued steak (Lancet 1996; 347: 1372)? Genetic screening cannot substitute for prudent dietary habits.

One of the greatest recent advances is the finding that periconceptional intake of folic acid can prevent neural-tube defects. Increased consumption of folic acid might also reduce the risk for thrombosis, heart disease, and cancer. But again foods are at risk of losing out to pharmaceuticals because both the concentration and the bioavailability of folic acid in foods may be too low (Lancet 1996; 347: 657).

Plant breeders might be able to produce foods higher in available folic acid, but will consumers pay a premium for Super-Folic Broccoli?

Key references for 1996


