EAT BEANS FOR GOOD HEALTH

Maurice Bennink
Food Science and Human Nutrition
Michigan State University

There are two major areas of health problem that could be significantly reduced by simply eating more beans. One area is chronic diseases and the other is malnutrition. The potential for beans to mitigate chronic diseases will be addressed first.

Beans and chronic diseases Chronic diseases (certain types of cancer, Type II diabetes, heart disease, and other diseases of the blood system) typically take many years (10 to 30 years) to develop. Chronic diseases are the most common causes of death in industrialized countries and they significantly lower the quality of life for millions. The single most important factor in the etiology of chronic diseases is the perpetual over-consumption of food (energy). Excess consumption coupled with inadequate physical activity results in a positive energy balance and eventually obesity. Obesity is a common etiologic factor in the development of chronic diseases. Other central components that lead to the development of chronic diseases are chronic elevated concentrations of blood glucose (hyperglycemia) and blood insulin (hyperinsulinemia). Excess body fat leads to hyperglycemia and hyperinsulinemia and vice versa. Hyperglycemia and hyperinsulinemia are hallmark features of Type II diabetes and Type II diabetes is a major contributor to the development of heart disease and other diseases of the blood system (cardiovascular diseases). In addition, recent epidemiological studies suggest that hyperglycemia and hyperinsulinemia contribute to the development of certain cancers.

The type of carbohydrate we eat has a strong influence on food intake, maintenance of normal blood glucose and insulin concentrations, and the occurrence of chronic diseases. Foods with a high glycemic index cause a more rapid and greater rise in blood glucose and insulin than foods with a low glycemic index even though the amount of carbohydrate consumed is equal. Eating foods that have a high glycemic index for a long period of time can lead to hyperinsulinemia, insulin resistance and Type II diabetes mellitus. Also, eating high glycemic index foods stimulates people to eat sooner after their last meal than if they ate low glycemic index foods (1, 2). Moreover, eating a high glycemic index meal produces the tendency to select high glycemic foods for a snack or for the next meal. This sets up a vicious cycle that leads to a greater caloric intake and greater blood glucose and insulin concentrations (3). With time, obesity and Type II diabetes develop. On the other hand when low glycemic foods are consumed, there is greater satiety and people don't feel hungry as quickly. Also the tendency to select high glycemic index foods for snacks or the next meal is reduced. Therefore, the likelihood of excessive calorie consumption is reduced and so is the likelihood of becoming obese and a Type II diabetic. Compared to other carbohydrate sources, beans have a low glycemic index, varying from 26-42 % relative to glucose (4). Beans are also high in fiber (typically 18% dietary fiber) and low in fat. While eating beans will not magically make you thin or make you loose weight, substituting beans for highly-refined cereal products, foods or beverages with a high sugar content, or any high glycemic index food will help curb caloric intake and help maintain a leaner physique.

Excess body fat increases the risk of developing heart disease, strokes, Type II diabetes mellitus, and some types of cancer (5). There has been a steady increase in the percentage of overweight and obese individuals in North America and Western Europe. The increase in obesity is considered to be of epidemic proportions in the U.S. (6) and in most developed countries (5-9). For example, on a worldwide basis, more than one billion adults are overweight and more than 300 million are obese (5,9). In the U.S. more than 60% of the adult population is overweight or obese (7). Obesity and overweight account for approximately 300,000 deaths per year in North America (10,11) and the cost associated with excess fatness is estimated to be greater than 117 billion dollars per year (12). Most of the costs associated with excess fatness are related to Type II diabetes, heart disease, and high blood pressure (13). Perhaps even more disturbing is the great increase in overweight and obese children and adolescents (8). Accompanying the rise in excess fatness is the increased incidence of Type II diabetics in children and adolescents. Soon we will experience a tremendous increase in morbidity and mortality resulting from complications of diabetes and obesity in children and adolescents.

The exact reason why consumption of high glycemic index foods leads to an increased risk for Type II diabetes is not known but may be due to an increase in insulin demand (3,14-16). High glycemic index foods are known to cause rapid elevations in blood glucose and insulin following a meal. Chronic consumption of high glycemic index diets may in turn lead to down-regulation or desensitization of receptors for insulin, eventually contributing to insulin resistance (15). The body initially adjusts to higher circulating glucose by increasing insulin secretion from the pancreas. However, in susceptible individuals over time insulin resistance combined with exhaustion of insulin producing cells will eventually lead to Type II diabetes (3,16). Current research (17, 18) also suggests that hyperglycemia and hyperinsulinemia stimulate fat cells and possibly cells that line blood vessels (endothelial cells) to secrete pro-inflammatory cytokines such as tumor necrosis factor alpha (TNF-) and interleukin-6 (IL-6). These cytokines promote insulin resistance and other clinical and biochemical symptoms associated with Type II diabetes. In addition, these cytokines are predictive of risk for cardiovascular disease.

Of the chronic diseases, CVD is generally the leading cause of death in North America and Western Europe. Factors that increase one's risk of developing CVD include high levels of total cholesterol and LDL cholesterol ("bad cholesterol"), low levels of HDL cholesterol ("good cholesterol"), obesity, diabetes, smoking, and high blood pressure. Only one epidemiological study has directly examined the frequency of legume consumption and risk of coronary heart disease in US men and women. After adjusting for confounding risk factors, individuals consuming legumes at least 4 times per week were found to have a 22% lower risk of heart disease than individuals consuming legumes less than once per week (19). In epidemiological studies where legumes are consumed as part of a healthier diet plan, consistent reductions in heart disease risk have also been observed. In the Health Professionals Follow-up Study, men that adhered to a more "prudent diet" which included greater consumption of whole grains, legumes, fish, and poultry had a 30% lower risk of having heart disease. Conversely, individuals following a more "Western" diet, characterized by increased consumption of red meat, refined grains, sweets, French fries, and high fat desserts had a higher risk of heart disease (20). Similar trends were seen in the Nurses Health Study (21). The relative risk of coronary heart disease in the 20% of women that followed the "prudent" dietary pattern more closely was 0.76 compared to 1.46 for women eating a "Western" type pattern (21). Thus, those that most consistently ate

the "prudent" type of diet had one half the risk of developing heart disease compared to those that most often ate the "Western" type of diet.

Data from several human intervention trials indicate that consumption of canned (22-24) and dry beans (22,25-28) reduce serum cholesterol. Generally, in carefully controlled clinical studies where the macronutrient intake was matched and the fiber content in the bean fed group was at least twice that of the control diet, significant reductions in both total and LDL cholesterol occurred (22,29). A 1% reduction in total cholesterol corresponds to about a 2% decrease in the risk of developing heart disease (30). Beans are a good source of soluble dietary fiber, containing approximately 4 g per cup of cooked beans (31). Soluble fiber has been shown to reduce blood cholesterol in epidemiological (32), clinical (21, 22, 25, 33), and animal (34, 35) studies. The consumption of dietary fiber in the US is only 12-13 g/day, well below the recommended 25-35 g/day. Incorporating one cup of cooked beans into the diet would add 12 g of total fiber and 4 g of soluble fiber per day. This increase in fiber intake would be expected to modestly lower serum cholesterol and risk of heart disease, especially in hyperlipidemic individuals.

Correa (36) examined data from 41 countries and found a significant inverse relationship between bean consumption and morbidity due to breast, prostate, and colon cancer. Two animal studies have shown that bean consumption reduces colon cancer (37, 38). Hughes et al. (37) fed rats either pinto beans or casein (milk protein) and found that feeding pinto beans reduced the number of rats with colon cancer by 50% compared to casein-fed rats. Moreover, in rats that did develop tumors, rats fed pinto beans had only 1 tumor while rats fed milk protein had and average of 2.5 tumors. In a similar study, Hangen and Bennink (38) fed rats a casein-based diet, a diet containing black beans, or a diet containing navy beans. They reported that feeding either black beans or navy beans reduced the number of animals that had colon cancer by over 50%. Similar to Hughes et al. (37), the number of tumors per rat was 50% less in bean fed rats. Hangen and Bennink (38) noted that rats fed beans were significantly leaner compared to control animals. These two animal studies corroborate the study by Correa (36) showing that bean consumption reduces colon cancer.

How beans slow cancer growth and which component(s) of beans have anticarcinogenic properties are not yet known. One potential mechanism whereby beans could inhibit cancer is related to regulation of blood glucose and insulin. Recent research findings suggest that high levels of blood insulin (39,40) and/or high levels of blood glucose (41) promote colon cancer. The Cancer Prevention Study by the American Cancer Society found that subjects with Type II diabetes have a higher propensity of developing colon cancer than individuals without diabetes (42). Type II diabetics typically have elevated blood glucose and insulin concentrations. Data from other large prospective studies also suggest that subjects with Type II diabetes have an increased risk of colon cancer (43, 44). Additional evidence supporting the relationship between hyperinsulinemia and promotion of colon cancer was provided by two studies that utilized animals exposed to a colon carcinogen and subsequent injections with insulin. Insulin injections promoted both the early stages of colon cancer (45) and growth of colon tumors (46). As discussed above, eating beans produce low blood glucose and insulin concentrations compared to most other sources of dietary carbohydrates. Taken together, these studies suggest that eating beans to keep blood insulin and glucose low may be one mechanism that slows colon carcinogenesis.

There are at least two other possible mechanisms whereby bean constituents may inhibit colon carcinogenesis. In experimentally induced colon cancer, feeding fiber often does not reduce colon cancer. However, if resistant starch is added along with fiber, fermentation in the colon is altered and colon cancer is reduced. Beans contain high amounts of resistant starch (38) as well as high amounts of soluble and insoluble fibers which leads to favorable fermentation and possibly explains why feeding beans inhibits colon cancer. Another possible mechanism of cancer inhibition is by phytonutrients. Beans contain phytonutrients such as anthocyanins, a variety of phenolic compounds, protease inhibitors, phytic acid, and saponins. Phytonutrients are not considered to be essential nutrients. However, research over the past 15 years clearly demonstrates that some phytonutrients do provide health benefits. Purified protease inhibitors, phytic acid, and saponins inhibit various aspects of carcinogenesis (47-49). But direct evidence that these phytonutrients in foods inhibit cancer is lacking. Therefore, how much of the anticancer activity associated with beans is due to phytonutrients remains to be determined.

Beans and malnutrition Protein energy undernutrition (PEU) remains a common problem in much of the developing world. More than one third of children less than five years of age in developing countries suffer from PEU and the proportion of children who are undernourished has changed very little during the past 20 years (50). Apart from PEU, deficiencies of iron and vitamin A are widespread and often severe. PEU and deficiences of vitamin A and iron account for more than 75% of the deaths of infants and young children in some developing countries (51). Greater consumption of beans by children in developing countries would significantly reduce morbidity and mortality in this age group.

PEU and micronutrient deficiencies begin during weaning and/or immediately thereafter as most food used for weaning do not provide adequate amounts of energy, protein and micronutrients. Traditional weaning foods are based on starchy staples such as maize, sorghum, finger millet, and rice or non-cereals such as cassava, potato, and plantains and these foods have been widely associated with nutrient deficiencies among pre-school age children (52,53). Beans are not typically fed to small children. However, appropriate combinations of beans and cereals, consumed in adequate amounts, will prevent PEU.

LITERATURE CITED

1. Leathwood, P. and Pollet, P. 1988.   Appetite 10: 1-11.
2. Ludwig, D. S. Majzoub, J. A. Al-Zahrani, A. Dallal, G. E. Blanco, I. and Roberts, S. B. 1999.
        (available at: http://www.pediatrics.org/cgi/content/full/103/3/e26).
3. Ludwig D.D.S. 2002. Journal of the American Medical Association. 287 (18): 2414-2423.
4. Foster-Powell, K. and Miller, J. B. 1995. Am J Clinical Nutrition 62: S871-S890.
5. WHO. 2002. The world health report: 2002: World Health Organization, Geneva.
6. U.S. Department of Health and Human Services. 2001. Rockville, MD.
7. Flegal, K. M. Carroll, M. D. Ogden, C. L. and Johnson, C. L. 2002. JAMA 288: 1723-1727.
8. Ogden, C. L. Flegal, K. M. Carroll, M. D. and Johnson, C. L. 2002. JAMA 288: 1728-1732.
9. WHO. 2000. World Health Organization, WHO Technical Report Series, No. 894, Geneva
10. McGinnis, J. M. and Foege, W. H. 1993. JAMA 270: 2207-2212.
11. Allison, D. B. et al. 1999. JAMA 282: 1530-1538.
12. Wolf, A. M. 1998. American Journal of Managed Care 4, S141-S145.

13. Wolf, A. M. and Colditz, G. A. 1998. Obesity Research 6: 97-106.
14. Jenkins D.J.A. et al. 1987. Am J Clin Nutr 46: 968-75.
15. Jenkins D.J.A. et al. 2000. British Journal of Nutrition. 83: S157-S163.
16. Augustin L.S. et al. 2002. European Journal of Clinical Nutrition. 56 (11): 1049-1071.
17. Esposito K. et al. 2002. Circulation. 106 (16): 2067-2072.
18. Soop M. et al. 2002. Am J Physiol-Endo Metab 282: E1276-E1285.
19. Bazzano, L. A. et al. 2001. Archives of Internal Medicine 161: 2573-2578.
20. Hu, F. B. et al. 2000. American Journal of Clinical Nutrition 72: 912-921.
21. Fung, T. T. et al. 2001. Archives of Internal Medicine 161: 1857-1862.
22. Anderson, J. W. 1987. American Journal of Cardiology 60: G17-G22.
23. Anderson, J. W. Smith, B. M. and Washnock, C. S. 1999. Am J Clin Nutr 70: 464S-474S.
24. Shutler, S. M. et al. 1989. British Journal of Nutrition 61: 257-265.
25. Anderson, J. W. et al. 1990. American Journal of Clinical Nutrition 51: 1013-1019.
26. Bingwen, L. et al. 1981. Chinese Medical Journal 94: 455-458.
27. Jenkins, D. J. A. et al. 1983. Diabetologia 24: 257-264.
28. Simpson, H. C. R. et al. 1981. Lancet 1: 1-4.
29. Anderson, J. W. et al. 1984. American Journal of Clinical Nutrition 40: 1146-1155.
30. Rifkind, B. M. 1984. Jama-Journal of the American Medical Association 251: 365-374.
31. Anderson, J. W. Smith, B. M. and Gustafson, N. J. 1994. Am J Clin Nutr 59: S1242-S1247.
32. Brown, L. Rosner, B. Willett, W. W. and Sacks, F. M. 1999. Am J Clin Nutr 69: 30-42.
33. Anderson, J. W. and Tietyenclark, J. 1986. Am J Gastroenterology 81: 907-919.
34. Rosa, C. O. B. et al. 1998. Archivos Latinoamericanos De Nutricion 48: 299-305.
35. Rosa, C. O. B. et al. 1998. Archivos Latinoamericanos De Nutricion 48: 306-310.
36. Correa, P. 1981. Cancer Research 41: 3685-3689.
37. Hughes, J. S. Ganthavorn, C. and Wilson-Sanders, S. 1997. J Nutr 127: 2328-2333.
38. Hangen, L. A. and Bennink, M. R. 2003. Nutrition and Cancer 44: 60-65.
39. Giovannucci, E. 1995. Cancer Causes & Control 6: 164-179.
40. Sandhu, M. S. Dunger, D. B. and Giovannucci, E. L. 2002. J Natl Cancer Inst 94: 972-980.
41. McKeown -Eyssen, G. 1994. Cancer Epidemiology Biomarkers & Prevention 3: 687-695.
42. Will, J. C. Galuska, D. A. Vinicor, F. and Calle, E. E. 1998. Am J Epidem 147: 816-825.
43. Fung, T. et al. 2003. Archives of Internal Medicine 163: 309-314.
44. Hu, F. B. et al. 1999. Journal of the National Cancer Institute 91: 542-547.
45. Corpet, D. E. Peiffer, G. and Tache, S. 1998. Nutrition and Cancer 32: 29-36.
46. Tran, T. T. et al. 1996. Cancer Epidem Biomarkers & Prev 5: 1013-1015.
47. Harland, B. F. and Morris, E. R. 1995. Nutrition Research 15: 733-754.
48. Kennedy, A. R. 1994. Cancer Research 54: S1999-S2005.
49. Koratkar, R. and Rao, A. V. 1997. Nutrition and Cancer 27: 206-209.
50. de Onís M. 1993. Bull. World Health Org. 71: 703 – 712.
51. Ou B. et al. 2002. J Agric Food Chem. 22;50 (11): 3122-8.
52. Seenappa M. October 1987. In: Alnwick, D, Moses, S & Schmidt, OG (Eds). Proc Workshop Household Level Food Technologies .....Southern Africa, Nairobi, Kenya.
53. Walker A.F. 1990. Nutr. Rev. Res. 3: 25 – 47.

Additional information on the nutritional value of beans can be found at:
http://www.michiganbean.org/research.html