

**The Relationship between Consumption of Animal Products (Beef,  
Pork, Poultry, Eggs, Fish and Dairy Products) and Risk of Chronic  
Diseases: A Critical Review**

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## BACKGROUND

Diet and nutrition have long been recognized to play an important role in human health, but specific dietary factors in relation to the development of chronic diseases including coronary heart disease, stroke, diabetes, and various cancers have not been clearly defined. Much controversy exists about associations between consumption of animal products such as red meat (beef, pork, lamb), white meat (poultry, fish), eggs, and dairy products (milk, cheese) and risk of chronic diseases. The controversy, in part, stems from the scarcity of data on the effects of these foods because, traditionally, nutritional epidemiologic studies have largely focused on nutrients rather than individual foods. Another reason for the controversy is that the same data, albeit limited, are given different weight by different scientists. For example, correlational or ecological analyses, which correlate per capita food consumption data with incidence or mortality rates of disease among different populations or cultures, are widely considered as a hypothesis generating process because of uncontrolled confounding by other aspects of diet and lifestyle. However, some put substantially more weight on findings from those studies. Randomized clinical endpoint trials are typically considered as “gold standard” in the evaluation of diet-disease relationships. However, in most circumstances, dietary trials are infeasible because of practical issues, including potential lack of compliance in the long run. Case-control studies, which compare the diets of diseased subjects and those of non-diseased controls, are able to examine the relation between diet and disease in more detail than correlational studies.

In such studies, however, the diet is assessed retrospectively and the results are thus susceptible to recall bias. Prospective cohort studies, in which diet is assessed prior to the occurrence of disease, are generally considered as the strongest nonrandomized design because they are less susceptible to bias that arise from the retrospective reporting of diet. For this reason, prospective studies with refined dietary assessment are usually given more weight in the assessment of scientific evidence. Over 30 large prospective studies of diet and chronic diseases have been established and data from some of these studies have become available during the last several years.<sup>1</sup> Nonetheless, no single study or study design can provide absolute proof. The strongest evidence comes from a combination of different types of epidemiological investigations, with support from animal studies, metabolic, and mechanistic data.

## INTRODUCTION

Red meat from beef and white meat from poultry are a central part in most diets in developed societies. These products are main sources of saturated fat and cholesterol in the diet. But they are also main sources of protein and various vitamins and minerals. Poultry fat contains a lower proportion of saturated fatty acids and a higher proportion of polyunsaturated fatty acids as compared with red meat. Fish contains much less saturated fat, and oily fish are rich in omega-3 fatty acids. Eggs are high in cholesterol, but also contain substantial amount of unsaturated fats, protein, folate, and B vitamins. Milk and dairy products are high in saturated fat content (unless the fat has been separated), but are also good sources of protein, vitamin D and calcium.

Because each product has different nutrient profile, it is conceivable that they may have different effects on human health. Also, because each food contains many nutrients, which may have opposite effects on disease risk, it is difficult to predict the effect of each product on disease risk with certainty based on the nutrient profile. Since no single biochemical or physiological measurement can simultaneously represent those effects of the various constituent nutrients, it is important to study the direct relationships between consumption of these products and actual disease incidence in humans. In this report, we review evidence regarding the relation of animal products with risk of coronary disease, stroke, diabetes, and various cancers.

## CORONARY HEART DISEASE

Consumption of animal products, especially red meat, is widely believed to be responsible for the development of coronary heart disease (CHD). This belief is largely based on ecological studies relating dietary intake of saturated fat and cholesterol to rates of CHD in different countries. In the Seven Country study directed by Dr. Keys,<sup>2</sup> intake of saturated fat as a percentage of calories had strong correlation with coronary death rates across 16 defined populations in seven countries ( $r=0.84$ ), whereas the percentage of energy from total fat had little relationship with CHD incidence or mortality. The effect of saturated fat, however, is considerably stronger in this and other ecological studies than would be expected by the effect of saturated fat on serum cholesterol, probably due to confounding effects of other variables including other aspects of diet, physical activity, smoking, obesity, and economic development. Indeed, the observed association between saturated fat and risk of CHD in subsequent

prospective cohort studies was substantially weaker, ranging from null to small (see a review by Willett). In the Nurses' Health Study<sup>3</sup> the largest and most detailed study of diet and CHD, each 5% increase of energy from saturated fat was associated with 17% increase in CHD (P-value =0.10). In metabolic studies, replacing carbohydrates with saturated fat increases not only plasma LDL cholesterol, but also HDL cholesterol.<sup>4</sup> The observed small effect in prospective studies is consistent with the possibility that the proportional increase in plasma HDL concentration produced by saturated fatty acids somewhat compensates for its adverse effect on LDL level. Notably, the amount of saturated fat in two glasses of whole milk per day would be predicted, on the basis of metabolic studies, to increase serum total cholesterol sufficiently to raise CHD risk by at most about 4%. Such an increase is far below what could be detected by any epidemiologic study or randomized trial. Thus, a small adverse effect on CHD risk may be considered possible or probable on the basis of indirect evidence.

The association of dietary cholesterol with risk of CHD observed in prospective studies is also considerably smaller than suggested by ecological studies. A significant positive association for dietary cholesterol was observed in some studies<sup>5,6</sup> but not in others.<sup>7-10</sup> In a pooled analysis of four studies<sup>5,6,11,12</sup> an increase of 200 mg dietary cholesterol per 1000 Kcal (about 2 eggs per day for an average person) was associated with a 30% increase in risk of CHD.<sup>13</sup> In the Nurses' Health Study<sup>3</sup>, an increase of 200 mg dietary cholesterol per 1000 Kcal was associated with only 12% increase in CHD risk. The relatively small risk associated with a large increase in dietary cholesterol intake is not unexpected; metabolic studies have

shown that the effect of dietary cholesterol on serum cholesterol and LDL-cholesterol levels in humans is relatively weak.<sup>14</sup>

The positive relationships between intakes of saturated fat and cholesterol and CHD, albeit weak, suggest that consumption of foods high in saturated fat and/or cholesterol such as red meat and eggs may increase risk of CHD. However, this cannot be concluded with certainty because red meat and eggs also contain many other nutrients besides saturated fat and cholesterol. In the Nurses' Health Study<sup>3</sup>, total animal fat, compared with the same energy intake from carbohydrate, was not significantly associated with CHD, probably due to the opposing effects of saturated and unsaturated fats, which are contained in approximately similar amounts in most animal fats. Few studies have examined the direct relationship between red meat consumption and risk of CHD. In a study of California Seventh-Day Adventists<sup>15</sup>, higher beef consumption was significantly associated with increased risk of fatal ischemic heart disease in men, but not in women. In a case-control study conducted in Italy<sup>16</sup>, meat and butter consumption was associated with increased risk of myocardial infarction in women.

Because of their high cholesterol content, eggs are widely considered as an unhealthy food. Surprisingly, there is little direct evidence linking higher egg consumption and increased risk of coronary disease. In the Framingham study, Dawber and colleagues<sup>17</sup> found no association between egg consumption and incidence of CHD. In an earlier analysis of Seventh-day Adventists study,<sup>18</sup> higher egg consumption appeared to be associated with increased risk of fatal CHD, but this association was not present in a more recent analysis.<sup>15</sup> In a case-control study conducted in Italy,<sup>16</sup> egg consumption was not significantly associated with risk of CHD in women. The null association between egg consumption and risk of CHD is not surprising,

because one egg contains about 200 mg cholesterol, but also substantial amount of protein, unsaturated fats, folate, B vitamins, and minerals. It is conceivable that the small adverse effect caused by cholesterol is counterbalanced by potential beneficial effects of other nutrients.

White meat from chicken and fish contains substantially less saturated fat and cholesterol than red meat. In the California Seventh-day Adventist study, higher consumption of poultry was not associated with risk of CHD.<sup>15</sup> Studies on fish consumption in relation to risk of CHD, although not entirely consistent, have generally suggested that moderate fish intake may protect against coronary death. Kromhout and co-workers<sup>19</sup> found that Dutch men consuming more than 30 g of fish per day had only about half the risk of fatal CHD compared with men who consumed none. In a recent study of 20,551 US male physicians, Albert et al.<sup>20</sup> found that consuming fish at least once per week reduced risk of sudden death by half. The protective effect of fish consumption against CHD may be due to the antiarrhythmic effect of omega-3 fatty acids.

## CEREBROVASCULAR DISEASE

The mortality rates of cerebrovascular disease (stroke) are much higher in some Asian countries including Japan and China, as compared with North American and European countries. It has been speculated that some aspects of Asian diet, such as very low animal fat and protein and relatively high salt, may be responsible for high rates of stroke, especially hemorrhagic stroke, in some Asian countries.<sup>21</sup> In the past decades, incidence of stroke has declined substantially in Japan, which has been attributed to increased consumption of animal products, including meats, eggs, and dairy products.<sup>22</sup> A similar decreasing trend in stroke rate,

accompanied by increased consumption of animal products, has also been observed in China.<sup>23</sup> In a prospective study of Japanese men living in Hawaii,<sup>24</sup> usual Western diet opposed to Oriental diet was associated with increased risk of CHD (relative risk = 1.2) and severe atherosclerosis at autopsy (relative risk = 1.6), but reduced risk of cerebral hemorrhage (relative risk = 0.7). Recently, Gillman et al.<sup>25</sup> reported that low total and saturated fat consumption was associated with increased risk of ischemic stroke in the Framingham study. In a large study of U.S. men, low serum cholesterol level was associated with increased risk of hemorrhagic stroke.<sup>26</sup> Laboratory studies have suggested that very low serum cholesterol levels increase fragility of endothelium cell membranes of intracerebral arteries, which can result in hemorrhage, especially in the presence of hypertension.<sup>27</sup> Also, in a study of three Chinese population samples, Zhou et al.<sup>28</sup> found that lower intake of animal protein, and lower levels of urinary sulphate and certain serum and urinary amino acids from metabolized protein metabolism were associated with high blood pressure (one of the most important risk factors for stroke). These data suggest that very low intake of animal fat and protein and high intake of carbohydrate, as consumed by large populations in Asia, may not be optimal in regard to the risk of hemorrhagic stroke.

## DIABETES

Non-insulin-dependent diabetes mellitus (NIDDM) has become a highly prevalent disease in industrialized countries. The incidence of NIDDM has also increased substantially in developing countries including China.<sup>29</sup> Genetic factors, as well as age, obesity, diet and sedentary lifestyle are the major determinants of NIDDM. Dietary factors have been hypothesized to influence the

development of NIDDM through effects on insulin resistant or insulin demands. However, few data are available on the direct relationship between animal product consumption and risk of NIDDM. In the Nurses' Health study, neither animal fat nor saturated fat consumption were associated with increased risk of diabetes.<sup>30</sup> In contrast, diets with low cereal fiber content and high contents of refined carbohydrates were associated with significantly increased risk of diabetes. Similar results were observed among 42,759 men in the Health Professionals Follow-up Study.<sup>31</sup> Feskens and Kromhout<sup>32</sup> found no significant associations between intakes of foods and risk of diabetes in a follow-up study of 814 men, which included only 58 new cases of NIDDM. In the California Seventh-Day Adventist study, Snowden and Philips<sup>33</sup> found that after controlling for age and percent desirable body weight, frequency of meat consumption was related to increased risk of NIDDM in men, but not in women. In a prospective study of 1462 Swedish women, Lundgren et al.<sup>34</sup> found that women who developed NIDDM consumed less milk and less fruit, but the differences were not statistically different.

## BREAST CANCER

The principal dietary hypothesis in relation to breast cancer has been that higher dietary fat intake increases risk of breast cancer. This hypothesis was generated by striking correlations ( $r=0.7$  to  $0.9$ ) among countries between national per capita fat consumption and both incidence and mortality of breast cancer.<sup>35</sup> Such a strong association is unlikely to be causal because of intractable confounding by other risk factors of breast cancer such as reproductive factors, obesity, and sedentary life style, and by other many correlates of economic development.<sup>36</sup> Indeed, the association between total fat intake and breast cancer was substantially weaker

( $r=0.35$ ) in the China-Oxford-Cornell study conducted in 65 counties in rural China,<sup>37</sup> in which breast cancer rates were ascertained at the population level, but dietary data were collected at an individual level.

The associations of dietary fat with breast cancer observed in case-control and cohort studies, if exists at all, has been much weaker than suggested by the correlational studies. In the past decades, more than 20 case-control and 10 prospective studies have examined the relationship of fat consumption with risk of breast cancer. Howe et al.<sup>38</sup> conducted a meta-analysis of 12 case-controls of breast cancer, including 4,312 cases and 5,978 controls. They found a significant increase in risk for higher fat intake (odds ratio = 1.35 for a 100 g increase in daily fat intake). Clearly, this is an unrealistic estimate because a 100-g change in total fat intake per day is implausible in a free-living population. The largest prospective cohort study, the Nurses' Health Study, involved 1,439 incident cases of breast cancer.<sup>39</sup> The relative risks comparing extreme deciles of total fat intake were 1.0 for premenopausal breast cancer and 0.90 for postmenopausal breast cancer. No significant association was observed for either saturated fat or dietary cholesterol intake. However, there was a suggestion of inverse association between monounsaturated fat and risk of breast cancer. Hunter<sup>40</sup> conducted a pooled analysis of seven prospective studies, including 4,980 cases from 337,819 women. This is the most powerful analysis so far on this topic because of its large number of cases and the wide range of fat intake (as low as <20% total energy intake). The results from this analysis indicated no increased risk of breast cancer with higher intake of total fat or saturated fat. Based on these data, it can be reasonably concluded that fat intake during adult life is not an important cause of breast cancer.

Few data are available on the direct relationship between animal product consumption and breast cancer risk. Toniolo et al.<sup>41</sup> reported a significantly increased risk of breast cancer with higher consumption of red meat (relative risk for extreme quintile comparison = 1.9), although total fat or saturated fat intake was not significantly related to breast cancer. However, in the much larger Nurses' Health Study, red meat consumption was not significantly associated with risk of breast cancer.<sup>1</sup> The relative risk comparing women who consumed beef, pork, or lamb as a main dish daily or more with who consumed < 1/week was 1.05. The corresponding relative risk for beef, pork, or lamb as sandwich or mixed dish was 1.00. A case-control in China reported a statistically significant increased risk with meat intake over 80 grams per day, but the association became weaker and statistically nonsignificant after adjustment for energy intake.<sup>42</sup> A meta-analysis of seven case-control and cohort studies reported a significant increased risk for high meat intake (summary relative risk = 1.54).<sup>43</sup> In contrast, in the same meta-analysis, poultry intake was unrelated to breast cancer risk, with a summary relative risk of 0.94.<sup>43</sup> More recent cohort studies also found no material association between poultry consumption and risk of breast cancer<sup>41,44</sup>. Two prospective studies reported no association between fish consumption and risk of breast cancer<sup>39,41</sup> and one reported a weaker protective effect.<sup>45</sup> Most studies found no substantial association between consumption of milk and dairy products and risk of breast cancer<sup>45-47</sup>

## COLON CANCER

As with breast cancer, rates of colon cancer are strongly correlated with national per capita disappearance of animal fat and meat ( $r=0.8-0.9$ ) across countries.<sup>35</sup> A recent meta-

analysis of 13 case-control studies found a significant positive association between total energy intake and colon cancer, but saturated, monounsaturated and polyunsaturated fats were not associated with the risk independently of total energy.<sup>48</sup>

Earlier prospective cohort studies on diet and colon cancer have been inconsistent, probably due to small number of cases and crude diet assessment. Recent cohort studies have provided more consistent picture. In the Nurses' Health Study,<sup>49</sup> women in the highest quintile of animal fat intake had nearly twofold increase in risk of colon cancer compared with those in the lowest quintile. The relative risk of colon cancer in women who ate beef, pork, or lamb as main dish every day was 2.5, as compared with those reporting consumption less than once a month. Processed meats and liver were also significantly associated with increased risk, whereas fish and chicken were related to decreased risk. Interestingly, in a multivariate analysis of these data, which include red meat and animal fat intakes in the same model, red meat intake remained significantly predictive of colon cancer risk, whereas the association with animal fat was eliminated. In the Health Professionals Follow-up Study,<sup>50</sup> a cohort study of men, a direct association between red meat consumption and risk of colon cancer was also observed, but no association was seen for other sources of fat. In particular, no significant association existed between total or saturated fat despite a wide range of total fat (24-41% of energy from lowest to highest quintile) and saturated fat (7-14% energy). These data suggest that consumption of red meat, but not other sources of fat, is associated with increased risk of colon cancer. Non-lipid components of red meat, including carcinogens formed when red meat is cooked, may be responsible for the increased risk.

Several prospective studies have observed an inverse association between non-red meat protein sources and risk of colon cancer and colorectal adenoma (see a review by Giovannucci and Willett<sup>51</sup>). In the Nurses' Health Study,<sup>49</sup> the ratio of the intake of red meat to the intake of chicken and fish was particularly strongly associated with an increased risk of colon cancer, suggesting a potential protective effect against colon cancer if red meat is replaced by white meat in the diet.

The evidence on the association of egg consumption with risk of colon cancer is inconsistent. Some but not all case-control studies reported a positive association (see a review by Steinmetz and Potter<sup>52</sup>). In a case-control study conducted in China, reduced consumption of animal products including eggs and meat was actually associated increased risk of rectal cancer (but not colon cancer).<sup>53</sup> Only a few prospective studies have reported the relation of egg consumption to risk of colon cancer. In the California Seventh-day Adventist's study, higher egg consumption appeared to be associated with increased mortality from colon cancer, but not rectal cancer.<sup>54</sup> In the Iowa's women study, no significant association between frequency of egg consumption and the risk of either colon or rectal cancer was observed.<sup>55</sup>

The data on the association between milk and dairy product consumption are limited. Two prospective cohort studies<sup>50,56</sup> reported no association between any dairy foods and risk of colorectal cancer. One prospective study suggested an inverse association between dairy products and risk of colon cancer.<sup>57</sup> The findings from case-control studies are also inconsistent (see a review by Giovannucci and Willett<sup>51</sup>).

## PROSTATE CANCER

International comparisons also suggest a strong correlation between per capita fat consumption and mortality rate of prostate cancer.<sup>35</sup> Within regions in Italy, positive correlations were found between prostate cancer mortality and milk consumption ( $r=0.75$ ) and cheese consumption ( $r=0.69$ ).<sup>58</sup> Case-control studies have generally supported a positive association between saturated fat or major sources of animal fat, meats, and dairy products and prostate cancer risk (see review by Giovannucci<sup>59</sup>). A recent case-control study found positive associations between saturated fat and prostate cancer risk separately among blacks, whites, Chinese-Americans and Japanese-Americans.<sup>60</sup> The source of animal fat most strongly associated with high risk in case-control studies was from dairy products. Recent prospective cohort studies have provided stronger evidence for the positive association between animal fat intake and risk of prostate cancer. In the Health Professionals Follow-up study of 51,529 men in the U.S.,<sup>61</sup> animal fat consumption was significantly associated with increased risk of advanced prostate cancer (relative risk comparing extreme quintiles of intake was 1.6). Fat from red meat consumption was particularly strongly associated with advanced prostate cancer (relative risk = 2.6). However, fat from poultry, dairy, and fish was not significantly associated with risk of prostate cancer. In the Physician's Health Study,<sup>62</sup> men who consumed red meat at least five times per week had 2.5 times higher risk in prostate cancer, as compared with men who ate red meat less than once a week. In the California Seventh-day Adventist study,<sup>63</sup> consumption of meat, poultry, and fish was associated with risk of prostate cancer, but the study did not distinguish red meat from white meat. No material relationship was observed between egg consumption and risk of prostate cancer in prospective cohort studies, with relative risks ranging from 0.80-1.0.<sup>63-65</sup>

Several case-control studies have suggested that dairy products are associated with increased risk of prostate cancer<sup>66,67</sup>. But this positive association was not confirmed in prospective cohort studies<sup>61,63,64</sup>. In a more recent analysis of the Health Professionals Follow-up Study,<sup>68</sup> higher intake of calcium from both supplements and foods (mainly dairy) was associated with increased risk of prostate cancer.

## OTHER CANCERS

Cured and smoked meats have been consistently related to stomach cancer,<sup>69</sup> but consumption of fresh meat does not appear to increase the risk. Several other cancers, including esophageal cancer, oral and pharyngeal cancer have been also associated with cured and smoked foods.<sup>36</sup> Meat consumption has been associated with risk of pancreatic cancer in several studies,<sup>70</sup> but the data are not entirely consistent.<sup>71</sup>

## SUMMARY

The effects of animal products on risk of chronic diseases are an area of considerable controversy. Ecological studies tend to suggest positive associations between higher consumption of animal products and risk of heart disease and various cancers across different countries. However, international correlations between per capita food consumption and disease rates are seriously confounded by other lifestyle factors associated with economic affluence. Also, food disappearance data used in most of the calculations may be more indicative of food wastage within a country than actual consumption. Correlational studies conducted within a country can usually provide more credible data than international

comparisons because of relatively homogenous populations and the possibility of collecting data on potential confounding variables at individual level. One of the most comprehensive correlational studies conducted within a country is the China-Oxford-Cornell study,<sup>72</sup> a detailed study of geographic correlations between disease rates and numerous dietary and non-dietary factors across 65 counties in rural China. These correlations, although informative and valuable in many ways, cannot be used to establish causal relationships between dietary factors and disease risk. The limitations of geographical correlations were precisely stated by Drs Doll and Peto<sup>73</sup> :

*Trustworthy epidemiological evidence, it should be noted, always requires demonstration that a relationship holds for individuals (or perhaps small groups) within a large population as well as between large population groups. Correlation between the incidence of cancer in whole towns or whole countries and, for example, the consumption of particular items of food can, at most, provide hypotheses for investigation by other means. Attempts to separate the roles of causative and of confounding factors by statistical techniques of multiple regression analysis have been made often, but evidence obtained in this way is, at best, of only marginal value.*

Indeed, some of the correlations produced from the China-Oxford-Cornell study are peculiar and probably incorrect. For example, esophageal cancer had no clear association with smoking, and had a negative correlation with daily alcohol intake. These results are clearly contradictory to the well-established findings from studies of individuals that both smoking and alcohol use are strong risk factors for esophageal cancer. In addition, the study did not find a clear association between meat consumption and risk of heart disease or major cancers.<sup>72</sup>

Prospective cohort studies of individuals, in which diet is assessed prior to the occurrence of disease, are typically considered as the strongest nonrandomized design. Since most of prospective studies of diet and chronic diseases are conducted in developed countries, mostly in the U.S., this raises the question of generalizability of the findings to developing countries. Because there is no reason to believe that the basic human biology is different among various populations, it is not unreasonable that the associations of diet with disease risk observed in one population can be extrapolated to another. This is particularly true because large parts of the developing world are now urban, with more sedentary lifestyle. It is possible that the differences in social, cultural, and economical factors between developed and developing countries may exacerbate or mitigate the effects of animal product consumption, but there are no data to support this speculation. Thus, data from developing countries themselves would be highly desirable and establishing reliable prospective sources of information should be a priority.

The health effects of consuming a major source of food energy depend on the component of diet that they displace. But data on food substitutions are limited. We have generally assumed that animal products primarily displace carbohydrate source (especially starch, the main source of food energy in almost all diets), although in many studies the comparison is not clear. Since vegetables and fruits contain relatively small amount of energy, in most circumstances, it is unlikely that vegetables and fruits are replaced by animal products. In some situations, it is possible that nuts, soy products, and legumes (which may have positive benefits for cardiovascular disease) are partially replaced by animal products. This kind of substitution, especially by red meat, may be detrimental to health.

The following table summarizes our assessment of the literature reviewed above. Higher red meat consumption probably increases risk of CHD, colon cancer, and prostate cancer, and possibly breast cancer. However, very low red meat consumption may increase risk of hemorrhagic stroke. Poultry consumption does not appear to be a risk factor for CHD or any forms of cancer. Substitution of poultry for red meat may lower risk of CHD and colon cancer. Fish consumption probably reduces risk of fatal CHD and sudden cardiac death. Substitution of fish for red meat may also lower risk of colon cancer. There is little evidence that egg consumption in the range of U.S. population (up to 1 egg/day) increases risk of CHD. However, it is not clear whether high egg consumption has adverse effect on cardiovascular disease in populations with very low background cholesterol intake (say, rural China). Several case-controlled studies have suggested that egg consumption may increase risk of colon cancer, but has not been confirmed in prospective cohort studies. Existing literature does not directly indicate a material association between dairy product consumption and risk of CHD, but higher consumption of dairy fat is likely to increase CHD rates mostly due to increase in blood cholesterol levels. On the other hand, there is some evidence that moderate dairy product consumption may reduce risk of hypertension and hemorrhagic stroke in places where these are important causes of death and overall consumption of animal products is low. Milk and dairy products possibly increase risk of prostate cancer, but the data are limited. The data on the relation of animal product consumption with diabetes are too scarce to draw any conclusions at this time, but the lack of association between saturated fat intake and risk of diabetes in large prospective studies suggest that there is not a strong association.

We do not address issues of specific deficiencies of micronutrients, such as iron or zinc deficiency, which may be common in parts of the developing world. Although red meat contains substantial amount of micronutrients, we do not regard it as a strong justification for increasing consumption of red meat in those countries because there are other cost-effective ways to improve iron or zinc status. We further recognize that effects on risk of chronic diseases are only part of decisions regarding the production of animal products.

**A qualitative assessment of associations of animal product consumption  
with risk of chronic diseases**

|                            | Red meat<br>(Beef, pork,<br>lamb) | Poultry                 | Fish                    | Egg*                               | Dairy<br>Products                  |
|----------------------------|-----------------------------------|-------------------------|-------------------------|------------------------------------|------------------------------------|
| Coronary heart<br>diseases | Probably ↑                        | Possibly ↓              | Probably ↓              | Probably no<br>increase in<br>risk | Probably<br>small ↑**              |
| Hemorrhagic<br>Stroke†     | Possibly ↓                        | Possibly ↓              | Uncertain §             | Probably no<br>increase in<br>risk | Possibly ↓                         |
| Breast cancer              | Possibly ↑                        | Probably no<br>relation | Probably no<br>relation | Probably no<br>relation            | Probably<br>no relation            |
| Colon cancer               | Probably ↑                        | Possibly ↓              | Possibly ↓              | Probably no<br>increase in<br>risk | Probably no<br>increase in<br>risk |
| Prostate cancer            | Probably ↑                        | Probably no<br>relation | Probably no<br>relation | Probably no<br>relation            | Probably ↑‡                        |

↓ increase in risk; ↑ decrease in risk

\* Up to 1 egg per day

\*\* It is possible that substitution of low-fat dairy (e.g., skim milk) for high-fat dairy products (e.g., whole milk) decreases risk of cardiovascular disease for individuals, but this is largely irrelevant for population disease rates because the dairy fat produced is almost inevitably consumed.

† The associations between animal products and risk of ischemic stroke, which is more common in the U.S., are probably similar to those for CHD.

‡ It is unclear whether it is due to fat or calcium.

§ Data are limited. It is possible that higher intake increases risk.

## CONCLUSIONS and IMPLICATIONS

The evidence reviewed in this report does not lead to an overall conclusion about the health effects of livestock products (beef, pork, and poultry), because different products can have different effects on the same disease, and the same product can have different effects on different diseases. The health effects of white meat from poultry and fish in epidemiologic

studies clearly differ from those of red meat from beef and pork. Diets containing substantial amount of red meat and products made from these meats probably increase risk of coronary disease and some forms of cancer. Substitution of red meat by white meat can have clear health benefits. In addition, consumption of animal products may have opposing effects on CHD and hemorrhagic stroke.

Based on the above review, and recognizing that data on diet and chronic disease are far from complete, we recommend that the following issues should be considered in making policies regarding investments in livestock production in developing countries:

1. Distinguish poultry and fish from beef and pork. As discussed above, white meat and red meat can have opposite health effects.

2. Distinguish eggs and dairy products from meats. As discussed above, there is little evidence that consumption of eggs has material adverse effects on chronic diseases, and moderate consumption of dairy products may have complex effects, including benefits and risks.

3. It is inappropriate to equate the health effects of livestock products to those of tobacco products. Tobacco use is an established cause of many chronic diseases and it has no counterbalancing beneficial effects. But animal products can provide many beneficial nutrients when consumed in small to moderate quantity.

4. In countries where hemorrhagic stroke rates are substantially elevated, very low consumption of animal products may not be optimal.

5. In many situations, the partial displacement of the carbohydrate staple source of energy with animal products may have neutral or beneficial health effect. However, the use of plant source of protein and fat, such as soy products, nuts, and vegetable oils, may provide even

greater health benefits and should therefore be considered simultaneously when considering investments in development.

6. Long term prospective studies of diet and health should be established in Asia, Latin America, and Africa to monitor the impact of policy and personal decisions regarding food choices on health and disease.

## REFERENCES:

1. Willett WC. Nutritional Epidemiology, 2nd edition. . New York, NY: Oxford University Press; 1998.
2. Keys A. Seven Countries: A multivariate analysis of death and coronary heart disease. . Cambridge, MA: Harvard University Press; 1980.
3. Hu FB, Stampfer MJ, Manson JE, et al. Dietary fat intake and risk of coronary heart disease in women. *N Engl J Med.* 1997;337:1491-9.
4. Mensink RP, Katan MB. Effect of dietary fatty acids on serum lipids and lipoproteins: a meta-analysis of 27 trials. *Arterioscler and Thromb.* 1992;12:911-919.
5. McGee DL, Reed DM, Yano K, Kagan A, Tillotson J. Ten-year incidence of coronary heart disease in the Honolulu Heart Program: Relationship to nutrient intake. *Am J Epidemiol.* 1984;119:667-76.
6. Shekelle RB, Shryock AM, Paul O, et al. Diet, serum cholesterol, and death from coronary heart disease: The Western Electric Study. *N Engl J Med.* 1981;304:65-70.
7. Pietinen P, Ascherio A, Korhonen P, et al. Intake of fatty acids and risk of coronary heart disease in a cohort of Finnish men: The Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study. *Am J Epidemiol.* 1997;145:876-87.
8. Ascherio A, Rimm EB, Giovannucci EL, Spiegelman D, Stampfer MJ, Willett WC. Dietary fat and risk of coronary heart disease in men: cohort follow up study in the United States. *Br Med J.* 1996;313:84-90.

9. Posner BM, Cobb JL, Belanger AJ, Cupples A, D'Agostino RB, Stokes III J. Dietary lipid predictors of coronary heart disease in men. *Arch Intern Med.* 1991;151:1181-1187.
10. Esrey KL, Joseph L, Grover SA. Relationship between dietary intake and coronary heart disease mortality: Lipid research clinics prevalence follow-up study. *J Clin Epidemiol.* 1996;49:211-216.
11. Kushi LH, Lew RA, Stare FJ, et al. Diet and 20-year mortality from coronary heart disease: The Ireland-Boston Diet-Heart study. *N Engl J Med.* 1985;312:811-18.
12. Kromhout D, De Lezenne Coulander C. Diet, prevalence and 10-year mortality from coronary heart disease in 871 middle-aged men: the Zutphen Study. *Am J Epidemiol.* 1984;119:733-41.
13. Stamler J, Shekelle R. Dietary cholesterol and human coronary heart disease. *Arch Pathol Lab Med.* 1988;112:1032-1040.
14. Grundy SM, Barrett-Connor E, Rudel LL, Miettinen T, Spector AA. Workshop on the impact of dietary cholesterol on plasma lipoproteins and atherogenesis. *Arterioscler.* 1988;8:95-101.
15. Fraser GE. Diet and coronary heart disease: beyond dietary fats and low-density-lipoprotein cholesterol. *Am J Clin Nutr.* 1994;59 (suppl):117-1123.
16. Gramenzi A, Gentile A, Fasoli M, Negri E, Parazzini F, La Vecchia C. Association between certain foods and risk of acute myocardial infarction in women. *BMJ.* 1990;300:771-3.
17. Dawber TR, Nickerson RJ, Brand FN, Pool J. Eggs, serum cholesterol, and coronary heart disease. *Am J Clin Nutr.* 1982;36:617-625.

18. Snowdon DA, Phillips RL, Fraser GE. Meat consumption and fatal ischemic heart disease. *Preventive Med.* 1984;13:490-500.
19. Kromhout D, Bosscheiter EB, de Lezenne Coulander C. The inverse relation between fish consumption and 20-year mortality from coronary heart disease. *N Engl J Med.* 1985;312:1205-9.
20. Albert CM, Hennekens CH, O'Donnell CJ, et al. Fish consumption and risk of sudden cardiac death. *JAMA.* 1998;279:23-28.
21. Willett WC. Diet and health: What should we eat? *Science.* 1994;264:532-537.
22. Shimamoto T, Komachi Y, Inada H, Doi M, Iso H. Trends for coronary heart disease and stroke and their risk factors for Japan. *Circulation.* 1989;79:503-515.
23. Cheng X-M, Ziegler D, Lai Y-H, et al. Stroke in China, 1986 through 1990. *Stroke.* 1995;26:1990-4.
24. Reed DM. The paradox of high risk of stroke in populations with low risk of coronary heart disease. *Am J Epidemiol.* 1990;133:579-88.
25. Gillman MW, Cupples A, Millen BE, Ellison RC, Wolf PA. Inverse association of dietary fat with development of ischemic stroke in men. *JAMA.* 1997;278:2145-2150.
26. Iso H, Jacobs DR, Wentworth D, Neaton JD, Cohen JD, for the MRFIT Research Group. Serum cholesterol levels and six-year mortality from stroke in 350,977 men screened for the multiple risk factor intervention trial. *N Engl J Med.* 1989;320:904-10.

27. Masawa N, Yoshida Y, Yamada T, Joshita T, Sato S, Mihara B. Morphometry of structural preservation of tunica media in aged and hypertensive human intracerebral arteries. *Stroke*. 1994;25:122-7.
28. Zhou B, Zhang X, Zhu A, et al. The relationship of dietary animal protein and electrolytes to blood pressure: A study on three Chinese populations. *International Journal of Epidemiology*. 1994;23:716-722.
29. Pan XR, Yang WY, Li GW, Liu J. Prevalence of diabetes and its risk factors in China, 1994. National Diabetes Prevention and Control Cooperative Group. *Diabetes Care*. 1997;20:1664-9.
30. Salmeron J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *J Am Med Assoc*. 1997;277:472-477.
31. Salmeron J, Ascherio A, Rimm EB, et al. Dietary fiber, glycemic load, and risk of NIDDM in Men. *Diabetes Care*. 1997;20:545-550.
32. Feskens EJ, Kromhout D. Cardiovascular risk factors and the 25-years incidence of diabetes mellitus in middle-aged men. *Am J Epidemiol*. 1989;130:1101-1108.
33. Snowdon DA, Phillips RL. Does a vegetarian diet reduce the occurrence of diabetes? *Am J Public Health*. 1985;75:509-512.
34. Lundgren H, Bengtsson C, Blohme G, al. e. Dietary habits and incidence of noninsulin-dependent diabetes mellitus in a population study of women in Gothenberg, Sweden. *Am J Clin Nutr*. 1989;49:708-12.

35. Armstrong B, Doll R. Environmental factors and cancer incidence and mortality in different countries, with special reference to dietary practices. *Int J Cancer*. 1975;15:617-31.
36. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition and the Prevention of Cancer: A Global Perspective. ; 1997.
37. Marshall JR, Yinsheng Q, Chen J, et al. Additional ecological evidence: Lipids and breast cancer mortality among women aged 55 and over in China. *Eur J Cancer*. 1992;28A:1720-1727.
38. Howe GR, Hirohata T, Hislop TG, et al. Dietary factors and risk of breast cancer: combined analysis of 12 case-control studies. *J Natl Cancer Inst*. 1990;82:561-569.
39. Willett WC, Hunter DJ, Stampfer MJ, et al. Dietary fat and fiber in relation to risk of breast cancer: An eight year follow-up. *J Am Med Assoc*. 1992;268:2037-2044.
40. Hunter DJ, Spiegelman D, Adami H-O, et al. Cohort studies of fat intake and risk of breast cancer: a pooled analysis. *N Eng J Med*. 1996;334:356-361.
41. Toniolo P, Riboli E, Shore RE, Pasternack BS. Consumption of meat, animal products, protein, and fat and risk of breast cancer - A prospective cohort study in New York. *Epidemiol*. 1994;5:391-397.
42. Qi X, Zhang A, Wu G, Pang W. The association between breast cancer and diet and other factors. *Asia Pac J Public Health*. 1994;7:98-104.
43. Boyd NF, Martin LJ, Noffel M, al. e. A metaanalysis of studies of dietary-fat and breast-cancer risk. *British Journal of Cancer*. 1993;68:627-636.

44. La Vecchia C, Negri E, Franceschi S, Decarli A, Giacosa A, Lipworth L. Olive oil, other dietary fats, and the risk of breast cancer (Italy). *Cancer Causes Control*. 1995;6:545-550.
45. Vatten LJ, Solvoll K, Loken EB. Frequency of meat and fish intake and risk of breast cancer in a prospective study of 14,500 Norwegian women. *Int J Cancer*. 1990;46:12-15.
46. Ursin G, Bjelke E, Heuch I, et al. Milk consumption and cancer incidence: a Norwegian prospective study. *Br J Cancer*. 1990;61:454-459.
47. Knekt P, Jarvinen R, Seppanen R, Pukkala E, Aromaa A. Intake of dairy products and risk of breast cancer. *Br J Cancer*. 1996;73:687-691.
48. Howe GR, Aronson KJ, Benito E, et al. e. The relationship between dietary fat intake and risk of colorectal cancer: evidence from the combined analysis of 13 case-control studies. *Cancer Causes Control*. 1997;8:215-228.
49. Willett WC, Stampfer MJ, Colditz GA, Rosner BA, Speizer FE. Relation of meat, fat, and fiber intake to the risk of colon cancer in a prospective study among women. *N Engl J Med*. 1990;323:1664-72.
50. Giovannucci E, Rimm EB, Stampfer MJ, Colditz GA, Ascherio A, Willett WC. Intake of fat, meat, and fiber in relation to risk of colon cancer in men. *Cancer Res*. 1994;54:2390-2397.
51. Giovannucci E, Willett WC. Dietary factors and risk of colon cancer. *Ann Med*. 1995;26:443-452.
52. Steinmetz K, Potter J. Egg consumption and cancer of the colon and rectum. *European Journal of Cancer Prevention*. 1994;3:237-45.

53. Hu J, Liu Y, Yu Y, Zhao T, Liu S, Wang Q. Diet and cancer of the colon and rectum: A case-control study in China. *International Journal of Epidemiology*. 1991;20:362-367.
54. Snowdon DA. Animal product consumption and mortality because of all causes combined, coronary heart disease, stroke, diabetes, and cancer in Seventh-day Adventists. *Am J Clin Nutr*. 1988;48:739-48.
55. Bostick RM, Potter JD, Kushi LH, et al. Sugar, meat, and fat intake, and non-dietary risk factors for colon cancer incidence in Iowa women (United States). *Cancer Causes Control*. 1994;5:38-52.
56. Phillips RL, Snowdon DA. Dietary relationships with fatal colorectal cancer among Seventh-Day Adventists. *JNCI*. 1985;74:307-317.
57. Bostick RM, Potter JD, Sellers TA, McKenszie DR, Kushi H, Folsom AR. Relation of calcium, vitamin D, and dairy food intake to incidence of colon cancer in older women. *Am J Epidemiol*. 1993;137:1302-17.
58. Decarli A, La Vecchia C. Environmental factors and cancer mortality in Italy: Correlational exercise. *Oncology*. 1986;43:116-126.
59. Giovannucci E. Diet and other extrinsic factors influencing prostate cancer risk. In: Fortner JG, Sharp PA, eds. *Accomplishments in cancer research 1996*: Lippincott-Raven Publishers; 1996:250-267.
60. Whittemore AS, Kolonel LN, Wu AH, et al. Prostate cancer in relation to diet, physical activity, and body size in blacks, whites, Asians in the United States and Canada. *J Natl Cancer Inst*. 1995;87:652-661.

61. Giovannucci E, Rimm EB, Colditz GA, et al. A prospective study of dietary fat and risk of prostate cancer. *J Natl Cancer Inst.* 1993;85:1571-79.
62. Gann PH, Hennekens CH, Sacks FM, Grodstein F, Giovannucci E, Stampfer MJ. A prospective study of plasma fatty acids and risk of prostate cancer. *J Natl Cancer Inst.* 1994;86:281-286.
63. Mills PK, Beeson WL, Phillips RL, Fraser GE. Dietary habits and breast cancer incidence among Seventh-day Adventists. *Cancer.* 1989;64:582-90.
64. Hsing AW, McLaughlin JK, Schuman LM, et al. Diet, tobacco use, and fatal prostate cancer: results from the Lutheran Brotherhood Cohort Study. *Cancer Res.* 1990;50:6836-6840.
65. Le Marchand L, Kolonel LN, Wilkens LR, Myers BC, Hirohata T. Animal fat consumption and prostate cancer: a prospective study in Hawaii. *Epidemiology.* 1994;5:276-282.
66. La Vecchia C, Negri E, Parazzini F, et al. Height and cancer risk in a network of case-control studies from northern Italy. *Int J Cancer.* 1990;45:275-279.
67. Mettlin C, Selenskas S, Natarajan MS, Huben R. Beta-carotene and animal fats and their relationship to prostate cancer risk. *Cancer.* 1989;64:605-612.
68. Giovannucci E, Rimm EB, Wolk A, et al. Calcium and fructose intake in relation to risk of prostate cancer. *Cancer Research.* 1998;58:442-447.
69. Correa P. Human gastric carcinogenesis: a multistep and multifactorial process—first American Cancer Society award lecture on cancer epidemiology and prevention. *Cancer Research.* 1992;52:6735-40.

70. Howe GR, Burch JD. Nutrition and pancreatic cancer. *Cancer Causes and Control*. 1996;7:69-82.
71. Ji B-T, Chow W-H, Gridley G, et al. Dietary factors and risk of pancreatic cancer: A case-control study in Shanghai, China. *Cancer Epidemiology, Biomarkers & Prevention*. 1995;4:885-893.
72. Chen J, Campbell TC, Tunyao L, Peto R. Diet, lifestyle and mortality in China: a study of the characteristics of 65 Chinese counties. . Oxford, England: Oxford University Press; 1990.
73. Doll R, Peto R. The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today. *JNCI*. 1981;66:1191-308.

## APPENDIX (Annotation of terminology)

### **Types of studies (Source: Willett 1998 Nutritional Epidemiology, 2nd edition):**

Ecological or correlational studies: Comparison of disease rates in populations having different diets by assessing average intake of specific nutrients and determining disease incidence or mortality rates. The major limitation is that diet is only one of many variables that distinguish different populations. In addition, gathering even crude data on average nutrient intake across populations is difficult. This type of study is probably best used to generate hypotheses.

Case-control studies: Comparison of earlier diets reported by patients having a particular disease with diets reported by controls without the disease. The major limitations are selection and recall biases. Selection bias can occur if controls do not accurately represent the population from which cases arise; recall bias results when patients systematically differ from controls in ability to recall diets. In studies of rapidly fatal diseases, researchers must often rely on recall of proxy respondents such as spouses.

Prospective Cohort Studies: Comparison of disease rates in people whose diets and other potentially relevant traits are determined before follow-up begins. Selection and recall biases should not occur, but cohort studies must enroll thousands or even tens of thousands of people and monitor their health for many years to ascertain adequate number of cases for study.

Controlled Clinical Trials. Comparison of incidence of disease in two groups randomized to specific diet interventions or sometimes to no interventions. In this type of studies, compliance with substantial dietary change is difficult for many people; subjects cannot be easily blinded to their intervention status. Optimal dosages (e.g., of supplemental nutrients) and dose-response relationships can be difficult to ascertain; duration of intervention required is generally unknown but may be decades.

**Types of fatty acids (Source: American Heart Association):**

Saturated fatty acids have all the hydrogen the carbon atoms can hold. Saturated fats are usually solid at room temperature, and they are more stable -- that is, they do not combine readily with oxygen. The main sources of saturated fatty acids in the typical American diet are foods from animal products, but palm and coconut oils are large sources in some populations. Foods from animals that have high amounts of saturated fatty acids include beef, beef fat, veal, lamb, pork, lard, poultry fat, butter, cream, milk, cheeses, and other dairy products made from whole milk. These foods also contain dietary cholesterol. Most saturated fatty acids raise blood cholesterol levels.

Polyunsaturated and monounsaturated fatty acids make up the total of unsaturated fatty acids.

Unsaturated fatty acids have at least one unsaturated bond -- that is, at least one place that hydrogen can be added to the molecule. They are often found in liquid oils of vegetable origin.

Polyunsaturated oils are liquid at room temperature and in the refrigerator. Common sources of polyunsaturated fatty acids are safflower, sesame and sunflower seeds, corn and soybeans,

many nuts and seeds, and their oils. Monounsaturated oils are liquid at room temperature but start to solidify at refrigerator temperatures. Canola, olive and peanut oils and avocados are mainly comprised of monounsaturated fatty acids, but almost half the fat from meat and dairy sources is also monounsaturated. Both types of unsaturated fatty acids lower blood cholesterol level when used in place of saturated fatty acids in the diet.

Trans fatty acids: Naturally occurring liquid vegetable oils are mainly comprised of unsaturated fatty acids that have a bent shape. Around the turn of this century a process called partial hydrogenation was developed that adds hydrogen to these molecules and changes their shape from bent to straight. These new fats are called trans unsaturated fatty acids. The major sources of trans fatty acids in the U.S. diet include margarine (hard margarine in particular), vegetable shortening, commercially baked products, and commercial deep fried foods. Metabolic studies have conclusively shown that trans fatty acids increase LDL (the bad cholesterol) and decrease HDL (the good cholesterol), and raise triglyceride levels in the blood.

### **Blood Cholesterol Fractions (Source: American Heart Association):**

LDL (low density lipoprotein) is the major cholesterol carrier in the blood. When a person has too much LDL cholesterol circulating in the blood, it can slowly build up within the walls of the arteries feeding the heart and brain. Together with other substances it can form plaque, a thick, hard deposit that can clog those arteries. This condition is known as atherosclerosis. A high level of LDL cholesterol reflects an increased risk of heart disease. That is why LDL cholesterol is often called "bad" cholesterol.

HDL (High Density Lipoprotein) is known as "good" cholesterol because a high level of HDL protects against heart attack. About one-third to one-fourth of blood cholesterol is carried by high density lipoprotein or HDL. HDL tends to carry cholesterol away from the arteries and back to the liver, where it's passed from the body.



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