

The Role of Meat in the Diet: Nutrition Misinformation and Misbelief

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Nutrition Misinformation/Misbelief

Misinformation. Defined by *Webster's Dictionary* as “untrue or misleading information”; or, what people think is true but is not. Misinformation differs from *ignorance* (i.e., being unaware or uninformed) and is not *stupidity* (i.e., slow-mindedness).

Misbelief. When deeply-held, health misinformation becomes the misbeliefs that underlie the human tragedies seen in connection with quackery. Misbelief is involved in the fallacious behavior of a smaller proportion of the population, but makes up a subgroup that sustains the problem in society. Misbelief manifests itself in health-related ideologies (aka, “hygienic religions”) that are often utopian ideas about super-nutrition as a means of achieving super health, performance, or life extension. Experience has shown that many health scientists and educators harbor strong ideological biases regarding lifestyles and often color their reports accordingly. Excessive optimism also plays an important role because it sells products.

The prevalence of misinformation assures that misconceptions will be persistently reinforced.

Studies find over and over that nutrition-related misinformation is widespread. In the 1960s, following four national congresses on medical quackery that showed the fundamental role food faddism (i.e., an exaggerated belief in the role of diet in health and disease), a major study funded by several government agencies dug deeply into the American psyche. It found that nutrition misinformation & misbelief abound in epidemic proportions. Such misinformation related to food production, processing, shipping, storage: “It is clear that half or more of the sample are conditioned to doubt the healthfulness of food grown or processed with

‘modern methods.’ Acceptance of the ‘soil depletion myth’ is particularly strong, and so is predisposition to doubt the efficacy of man-made vitamins, which by extension could mean doubt about the value of fortified foods. Less than half believed that chemical fertilizers produce food as healthful as do natural fertilizers.¹

Table 1. Examples of nutrition misinformation and misbelief.

Statement	% Agree
1. The chemicals added to our manufactured food take away much of its value for health. (false)	48*
2. Man-made vitamins are just as good as natural vitamins. (true)	35
3. Much of our food has been so processed and refined that it has lost its value for health. (false)	60
4. Chemical sprays that farmers use make our food a danger to health even if they are used carefully. (false)	57
5. There is no difference in food value between food grown in poor, worn-out soil and food grown in rich soil (true)	15
6. Many foods lose a lot of their value for health because they are shipped so far and stored so long. (false)	73
7. Food grown with chemical fertilizers is just as healthful as food grown with natural fertilizers. (true)	44

*The percentage of consumers fearful of food additives grew to, and leveled off at about 57% in the late 1970s.²

¹A four-phase study of “the nature and prevalence of fallacious or questionable health beliefs and practices, and susceptibility to them” among American adults funded by seven government agencies. Phase I: A questionnaire was developed using interviews of 38 people suspected to have experienced quackery or expressed false beliefs, and 250 randomly selected respondents. Phase II: Depth interviews with 37 identified as being susceptible to misinformation or having engaged in questionable practices. Previous research suggested that feelings of hopelessness and powerlessness might be involved in susceptibility to fallacious health practices; feelings of insecurity or mild paranoia, fatalistic beliefs, and optimism may make quick and easy solutions seem more attractive. Six traits were tested for: *purposelessness & anomia, insecurity, pessimism, fatalistic thinking, belief in intervention by God*. These were analyzed by a skilled psychologist at National Analysts. Phase III: A questionnaire of over 200 items was administered to 2839 adults in a national area probability sample during the summer of 1969. No one respondent was asked all of the questions. The researchers attest that the study can be assumed to be a valid representation of the U.S. population. Phase IV: Follow-up depth interviews were done with 8 groups consisting of at least 8 subjects per group, and 9 individual depth interviews. This is probably the highest quality study ever conducted on this topic. It continues to serve as the baseline for FDA consumer behavior studies.

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It appears that a majority of the population over stresses the relationship between health and diet or nutrition.

1. Many people seem to believe that actual observed cases of poor health are more often due to “not eating right” than any other cause.” (In the survey, 75% agreed with a statement to this effect.)
2. Very large numbers of people believe that almost everyone can gain noticeable improvements in vigor and energy by improving his diet or using supplements.
3. Thus, many people are convinced that one can “fine tune” his health by simply improving his diet.
4. Health and nutrition courses in school may contribute to these misconceptions. Since these courses concentrate on things the individual himself can do for his health, they emphasize such factors as diet and cleanliness, rather than such causes as bacteria and virus infection, long-term tissue degeneration, and congenital factors.³

Transmitters of Nutrition Misinformation

The AMA Council on Foods and Nutrition of the American Medical Association identified the following as Transmitters of nutrition misinformation.⁴

Accidental

- Scientists with a bias
- Scientists interpreting the work of others
- Misinformed educators
- Government agencies
- Health care personnel
- Friends, relatives and associates

Intentional

- Commercial Communication bent on distorting information
- Health food operators
- Advertisers and food promoters
- Undiscriminating publishers and broadcasters

Women’s Magazines

Veteran writer, Marilyn Larkin, points out that women’s magazines are more beholden to advertisers than readers. Gloria Steinem is quoted as stating that “85% of women’s magazine copy is really ‘unmarked advertorial’ (i.e. combined advertising and editorial). The purpose of women’s magazines is to provide a nice environment of advertisers. The world according to women’s magazine editors is shaped primarily by two considerations: (1) providing a nice environment for advertisers, and (2) making sure that readers are not challenged by anything more than simple tips for healthy living.⁵

Americans’ #1 Nutrition Misconception

The idea that foods can be judged as “good” or “bad” was determined to be the most prevalent nutrition misconception in America.⁶ When asked if, nutritionally speaking, there were good foods and bad foods, most of the respon-

dents (77%) answered “yes.” This was deemed to be a wrong response because what counts is how a food fits into the total diet. “Good/bad” foods are commonly referred to as “health food” versus “junk food.”

Ambiguity of Allegedly “Good/Bad” Foods.

Food faddists of one type or another have condemned or extolled nearly every category of food or dietary practice.⁷

Grain: Extolled: Wheat is “the staff of life”; Wheaties is the “Breakfast of champions”; whole grains have positive image.

Condemned: Grasses as a group have been condemned as unfit for human food, apparently because of gluten intolerance, allergies to pollen; corn allergies; white flour is commonly condemned. Curiously, vegetarians will extol whole grain products yet will eat highly refined gluten products as meat substitutes.

Fruit: Extolled: “An apple a day keeps the doctor away”; Maizel⁸ the Diamonds “the most important food,” and Robbins¹⁰ extol fruit because it is 70% water, just as is the planet earth.

Condemned: Fruit thought bad because overindulgence causes diarrhea, and diarrhea was associated with deadly diseases.¹¹

*Vegetable:*Extolled: Vegetarians extol vegetables as the preferred foods; primary on the food chain;

Condemned: Low CHO diet advocates extol the “Cave Man diet” which they allege was mostly meat¹²

Meat: Extolled: Raw meats, particularly organ meats, have been called “super foods”¹³

Condemned: Vegetarians blame meat eating for aggressiveness, uncontrolled sexual passion, ill-health, and more.¹⁴

Milk Extolled: raw milk has been extolled as perfect food (nature’s only intended food) - yogurt is a health food. Colostrum confers immunity to some infectious agents.

Condemned: Milk condemned by vegans and some “nature doctors” as for calves only; lactose intolerance and reactions by some infants to cow’s milk generalized as evidence that milk is “bad food.”

Epidemiology

Health scientists and educators rely heavily upon the science of epidemiology for information on health and disease. Epidemiology literally means “study of that which is upon the people” (*epi* = upon; *demos* = the people; *logos* =

logic/study). Modern epidemiology began in 1849 when John Snow (1813-1858) mapped the cases of cholera; found that the victims all obtained their water from the same water company – the only one below the point where sewage effluence entered the Thames River. He removed the handle of the Broad Street pump in London in an attempt to stop a cholera epidemic.

Epidemiology employs a classical model that examines interactions of host, environmental, agent factors. The main purpose is to identify populations which are at risk for specific diseases and to plan and execute intervention strategies that will reduce the incidence, prevalence, morbidity or mortality of the disease. Epidemiology's most recent triumph has been the association found between tobacco and diseases such as cancer, emphysema, and heart disease. Although epidemiology lacks the power to prove cause and effect, it has changed consensus which has shifted the burden of proof to those who hold that tobacco is innocent. Higher quality experimental research has verified the role of tobacco in disease causation.

Modern epidemiology has extended to studying healthy populations to determine associations between theoretical factors and desirable health outcomes. Certain religious groups, healthy oldsters (e.g. veterans, athletes, school alumni) have provided data for further study. A major problem with applying a model for studying disease to health is that diseases are specific and causal factors may be assigned to them, but health is nonspecific and is the absence of disease.

Defining and measuring "health."

The World Health Organization (WHO) defines "health" as "...a state of complete physical, mental and social well-being and not merely the absence of disease and infirmity." Most accept this definition uncritically; but it has significant limitations. The WHO definition is more "a statement of aims and principles" than a definition.¹⁵ Its purpose is to expand the popular view of health to include quality of life, by addressing factors that underlie poor health (e.g. poverty, ignorance, superstition, insanitation, lack of rights for women and children, population control, oppression, and human rights). WHO does not define health as well as do standard dictionary definitions.

Epidemiologist Richard Doll said of the WHO definition: "This is a fine and inspiring concept and its pursuit guarantees health professionals unlimited opportunities for work in the future, but it is not of much practical use for specialists in public health medicine who need to compare the states of health in different communities and at different times and who consequently need to give them numerical values."¹⁶ The "numerical value" used to compare the health of societies is life expectancy (LE)^b US LE is 77.6 yrs (women 80.1, men 74.8) which are record highs.¹⁷ This is best for nations over 200 million.

Life expectancy v. causes of death

Some point to the higher death rates from so-called "degenerative diseases" (i.e. cancer, heart disease, stroke, etc.) found in "industrialized" countries, and decry the fact that increases in such diseases occur when traditional cultures adopt "Western" lifestyles. Population pyramids reveal the fallacy of such thinking. Such are actually the diseases of aging and merely reflect the increased life expectancies of such countries.

Life expectancy v. life span

Life expectancy is a statistic that reflects what is happening at the reported time, but life span refers to the potential for longevity inherent of a species. It may be expressed in terms of maximums or averages. The average human life span is 85-years with a standard deviation of 10-years.¹⁸ 409/1,000 white girls born in 1989 can expect reach 85-years of age. Only half as many boys can expect to do so (224/1,000). For nonwhites: 348/1,000 girls; 174/1,000 boys.¹⁹

Strengths and limitations of epidemiological (epi) research.

Epi enables the study of agents which could not be assigned experimentally (e.g. smoking, drug abuse, diseases). Epi's greatest strength is that it operates in the real world in which all other variables are active rather than sterile, highly-controlled laboratory settings. Epi also studies large numbers of people. These same attributes also underlie its weaknesses which are that in the real world it is impossible to control many confounding variables, and utilizing large numbers of subjects generally limits it to superficial methods of measurement.

Epidemiological research includes "case-control" designs in which patients with a particular disease are contrasted with similar individuals who do not have their disease. Case-control reports on Ca and diet are useful, but not true controlled studies. They are analytical and subject to confounding. They are retrospective and depend upon memory; vulnerable to social expectation effects that cause people to overestimate the "good" things that they do and underestimate the "bad" practices, e.g., SDAs reporting meat and vegetable intakes are likely to under report the former, and over report the latter; further, researchers may fail to measure the right thing(s) – including the interaction of multiple factors, or may not have allowed enough time for disorder to develop in "controls."

Epidemiologists have also been criticized for over-generalizing results, and for putting insufficient emphasis upon the limitations imposed by its methods of observation. There has been a strong tendency to engage in *Post hoc* (i.e. after the fact) reasoning in which perfectly logical thinking results in theoretical errors.

^bLE is determined by calculating the Age-Specific Death Rate for each group for a given year. The formula is too elaborate to show. If you want to see it, see Arriaga. "Measuring and explaining the changes in life expectancies," *Demography* 1984;21:83-96.

The risk factor concept and its limitations.

A *risk factor* is “A measure that is correlated^c with the risk of developing a disease. Its presence does not guarantee that the disease will occur, nor does its absence guarantee that the disease will not occur in a given individual.” Risk factors can only predict likely health outcomes for groups, not individuals.

Expressions of Risk

Relative risk standardizes the average risk at 1.0 (e.g. 0.5 = half the risk; 1.75 = 75% greater risk; 3.0 - three times the risk). Caution: The risk may be infinitesimal in absolute terms.

Absolute risk states this risk in term of probability, or number of years of life expectancy lost.

Chances of dying this year: everyone = 1 in 118; 35-44 yrs of age = 1 in 437; policeman on the job = 1 in 4,500; giving birth 1 in 9,100; air crash = 1 in 167,000; lightning strike = 1 in 2 million; from asbestos in schools = 1 in 11 million.

Ultimate risk refers to the reality that everyone dies of something at sometime. The best measure of the health of a society is its life expectancy, not its causes of death per se.

Risk markers are behaviors that merely correlate with contracting or not contracting a disease.²⁰

Interpreting epidemiologic research

The search for subtle links between diet, lifestyle, or environmental factors and disease is an unending source of fear, but often yields little certainty. Michael Thun, director of analytic epidemiology for the American Cancer Society stated, “With epidemiology you can tell a little thing from a big thing. What’s very hard to do is to tell a little thing from nothing at all.” Ken Rothman, editor of the journal *Epidemiology* agrees and says, “We’re pushing the edge of what can be done with epidemiology.” Most epidemiologists interviewed by *Science* said they would not take seriously a *single* study reporting a new potential cause of cancer unless exposure to the agent in question increased a person’s risk by at least a factor of three.^d Even then, skepticism is in order unless the study was very large and extremely well-done, and biological data support the link. Few entries in the following list of potential cancer risks, reported in journals and picked up by the popular press over the past 8 years have come close to fulfilling those criteria.²¹

Table 2. Potential cancer risks as communicated by the popular press

Douching once a week and cervical cancer	4
Regular use of high alcohol mouthwash and oral cancer	1.5
Weighing 3.6 kilos or more at birth and breast cancer	1.3
Vasectomy and prostate cancer	1.6
Pesticide exposure indicated by high blood residues and breast cancer (Contradicted one year later by a larger study by same authors)	4
Drinking >3 liters fluid (particular chlorinated tap water) daily and bladder cancer	2.4
Experiencing psychological stress in workplace and colorectal cancer	5.5
Diet high in saturated fat and lung cancer in nonsmoking women	6
Eating more than 20 grams of processed meats (i.e. bologna) and colon cancer	1.72
Eating red meat 5+ times per week and colon cancer	2.5
Occupational exposure to electromagnetic fields and breast cancer	1.38
Smoking two packs of cigarettes a day and fatal breast cancer	1.74
Eating red meat twice a day and breast cancer	2
Regular cigarette smoking and pancreatic cancer	1.7
Ever having used a sun lamp and melanoma	1.3
Obesity in men (heaviest 25% in study) and esophageal cancer	3
Consuming olive oil once a day or less and breast cancer.	1.25

Diet & Cancer

Distortions of Doll & Peto diet/cancer report. Highly regarded epidemiologists, Doll and Peto published the most significant report on diet and cancer to date entitled, “The causes of cancer: quantitative estimates of avoidable risks of cancer in the United States today.”²² Doll & Peto stated that “By far the largest reliably known percentage is the 30% of current U.S. cancer deaths that are due to tobacco, although it is *possible* (emphasis added) that some nutritional factor(s) may eventually be found to be of comparable importance” (p.1194). Regarding these *possible* nutritional factors they stated:

Diet may also prove to have a material effect on the incidence of cancers of the breast and pancreas and, perhaps through the anti-carcinogenic effects of various micronutrients, on the incidence of cancers in other tissues. If this is so, it may be possible to reduce U.S. cancer death rates by practicable dietary means by as much as 35% (“guesstimated” as stomach and large bowel, 90%; endometrium, gallbladder, pancreas, and breast, 50%; lung, larynx, bladder, cervix, mouth, pharynx, and esophagus, 20%; other types, 10%). Although the figure of 35% is a plausible total, the parts that contribute to it are uncertain in the extreme, so the degree of uncer-

^cCorrelation cannot establish a cause-effect relationship.

^dThe speaker opines that the source and quality of the raw data are critical. For instance, Finland is noteworthy for its high quality data due to its well-organized health care system; data from France is questionable because people without health expertise can fill out death certificates.

tainty should be obvious, and we make no pretense of its reliability. (p.1235)

Despite powerful disclaimers of “most reliably known” for tobacco versus “no pretense of its reliability,” the National Center for Health Information published a table showing the risk of cancer from diet as 35% and tobacco as 30% without adjusting these numbers according to their differing degrees of reliability. The result has been that more emphasis has been given to the theoretical relationship of diet to cancer than the much more important known risk of tobacco to cancer.

Why diet is suspect in cancer etiology.

Dietary carcinogens are known to exist, but most dietary carcinogens are natural. For example, Aflatoxin B, a metabolite of *Aspergillus flavus* a mold that grows on untreated stored rice and legumes – especially peanut meal, is the most potent dietary carcinogen to date. Also, when people migrate, their cancer incidence changes – resembling their new places of residence more than their former places of residence.

Table 3. Japanese in Japan v. Hawaii. Age-standardized mortality rates for GI cancers ages 0-74

	Japan		Hawaii					
	Japanese		Foreign born		US Born		Caucasian	
	M	F	M	F	M	F	M	F
Stomach	58.4	30.6	29.9	13.0	11.7	11.3	8.0	4.0
Colon	1.9	2.1	6.1	7.0	6.3	10.4	7.9	8.3
Rectum	3.3	2.8	4.0	4.0	3.1	2.0	4.2	2.8
Total GI Cancer	63.6	65.8	40.0	24.0	21.1	23.7	20.1	15.1

Note: The net effect was that American-born Japanese men had 1/3 the risk for these GI cancers; women’s’ risk was cut by 50%. Addendum: Colo-rectal cancer rates for Japanese men and women now exceed those of the white population.²³

Why it is said that 90% of cancers are due to environmental factors?

This informal estimate is attributed to Higginson in 1969.²⁴ It is based upon the variations in cancer incidence among various countries. Theorists hold that the country with the lowest incidence of a cancer represents its “natural level”; thus, countries with higher levels are due to environmental factors. The theory behind these comparisons assumes no racial-genetic differences in risk. Further, traditional countries have much lower life expectancies than industrialized nations, and aging per se is the strongest predictor of cancer. Thus, many people who would develop cancer later in life do not live long enough to do so (Table 4).

Beware of the implication that man-made carcinogens are the greatest problem. Most carcinogens are part of the natural environment (e.g. the sun, radon, nitrites, and naturally-occurring toxicants in foods) (Table 5). Natural carcinogens are so unavoidable that scientist Bruce Ames put it succinctly in Science magazine’s September, 1983 cover story: “Eat & Die,” noting that, unfortunately, we humans must do both.

The politicization of the diet/cancer question

In 1980, the National Academy of Sciences (NAS)^f was asked to review the scientific literature on the relationship of diet to health. NAS was unable to make recommendations as specific as some influential sources desired. Rather, its report, *Toward Healthful Diets*, found that no sound scientific basis existed for recommending dietary changes to reduce cancer. Rather, the report recommended nutrient

^eOncogenes and suppressor genes are essential in the etiology of cancer. Environmental promoters and suppressors and sufficient periods of time are also key ingredients.

^fThe National Academy of Sciences is an official U.S. government advisor chartered by Congress in 1863.

Table 4. Theorized “natural levels” versus “environmental influences” in cancer etiologies.²⁵

Cancer site	Low Risk Country	US White Male	US White Female	Relative Risk in High Risk Country			
				Male	Female	Male	Female
MORTALITY							
Lung	Portugal	3.6	2.4	Scotland	7.1	Scotland	4.2
Stomach	USA whites	N/A	N/A	Japan	7.8	Japan	7.8
Breast	Japan	N/A	5.5	N/A		Netherlands	6.6
Uterus	Israel	N/A	1.9	N/A		US non-white	4.7
Intestine	Japan	3.9	3.7	Scotland	4.3	Scotland	4.3
Rectum	Chile	2.4	1.6	Denmark	4.8	Denmark	3.3
Prostate	Japan	6.9	N/A	US nonwhite	12.4	N/A	
Leukemia	Japan	2.0	1.6	Denmark	2.1	Israel	1.9
INCIDENCE							
Colon	Rhodesia	50	44.0	Canada	60	US Hawaii	55
Rectum	Rhodesia	136	83.0	Canada	136	Canada	136
Lung	Nigeria	4.7	12.1	UK	72.1	New Zealand	37.7
Breast	Israel	N/A	7.9	US white	7.9		
Stomach	US Hawaii	3.3		Japan	30.4		
Stomach	Nigeria	9.5		Japan	79.4		

Table 5. The most common cancers worldwide and some of their suspected causes in 1999.²⁶

	Incidence per 100,000		Mortality		Main Suspect(s)
	Female	Male	Female	Male	
Mouth	2.9	6.6	1.4	3.1	Tobacco chewing, smoking
Esophagus	4.2	10.2	3.8	9.3	Tobacco+alcohol; hot mate'; nutr deficiencies+agent
Stomach	11.6	24.5	9.2	19.1	Dietary contamination; Helicobacter pylori
Colon/rectum	15.3	19.4	8.6	10.7	Speculative: likely environmental; presumably dietary
Liver	4.9	14.7	4.9	14.2	Hepatitis B&C virii; aflatoxin in foods
Lung	10.8	37.5	9.2	33.7	Tobacco smoking
Melanoma	2.2	2.3	.6.8		Sun exposure, white skin
Breast	33.0	n/a	12.9	n/a	Reproductive factors; Diet?
Cervix	15.4	n/a	8.0	n/a	Human papillomavirus
Prostate	n/a	19.8	n/a	8.2	Aging
Bladder	2.3	9.9	1.1	4.2	Tobacco smoking; occupational exposures
Non Hodgkin lymphoma	3.8	5.6	2.2	3.3	None given
Pancreas	3.1	4.4	3.1	4.4	None given

adequacy, variety (food groups), moderation (caloric input in balance with physical activity), reduce empty calories in inactive, and salt in moderation.

Outcries by people who wanted specific recommendations led to the impaneling of a second group whose goals were to:

1. Review...the state of knowledge and information pertinent to diet/nutrition and the incidence of cancer;
2. Develop a series of recommendations related to dietary components (nutrients and toxic contaminants); and,
3. Develop a series of research recommendations related to dietary components and nutritional factors and the incidence of cancer based upon the state-of-the-art appraisals and identification of gap areas.

The newly impaneled group's report, Diet, Nutrition and Cancer, recommended:

1. Reductions in both saturated and unsaturated fat;
2. Include fruits, vegetables and whole grain cereal products, especially citrus and carotene-rich and cabbage family vegetables, and to avoid high doses of supplements of individual nutrients;
3. Minimize consumption of cured, pickled and smoked⁸ foods;
4. Drink alcohol only in moderation.

Differences in the two reports were so great that Congress asked the General Accounting Office (GAO) to investigate. Its report found that:

Different scientists' philosophies about what scientific evidence is necessary as a basis for providing the public with dietary advice to reduce

the risk of cancer are a major factor in the reports' different conclusions and recommendations. Also, the reports are different because they were done for different purposes, on different topics, at different points in time by different groups.²⁷

The GAO found that the NAS "has no formal means to clarify scientists' disagreements for the public, and said that it should consider making reporting changes to aid public understanding."

The American Cancer Society (ACS).

In 1984, the ACS, approved a report *Nutrition and Cancer: Cause and Prevention*, which contained the disclaimer that "No concrete dietary advice can be given that will guarantee prevention of any specific human cancer. The American Cancer Society nonetheless believes that there is sufficient inferential information to make a series of interim recommendations that, in the judgment of experts, are likely to provide some measure of reducing cancer risk." Recommendations were to:

1. avoid obesity;
2. cut down on total fat intake;
3. eat more high fiber foods, such as whole grain cereals, fruits and vegetables;
4. include food rich in vitamins A and C in the daily diet;
5. include cruciferous vegetables such as broccoli,

⁸Fails to distinguish between naturally smoked foods and those flavored with liquid smoke.

The latter, which is used in commercial products (unless stated otherwise) is not carcinogenic – only natural smoke contains benzo(a)pyrene, the well-known carcinogen.

Health food stores sometimes advertise "naturally smoked" meats as if they are safer than commercially smoked--the opposite is true.

Brussels sprouts, kohlrabi and cauliflower in the diet^h;

6. be moderate in consumption of alcoholic beverages;
7. be moderate in consumption of salt-cured, smoked and nitrite-curedⁱ foods.²⁸

In 1996, the ACS issued a new report, *Guidelines on Diet, Nutrition, and Cancer Prevention: Reducing The Risk of Cancer with Healthy Food Choices and Physical Activity*. Couched within the report was the disclaimer that, "Although the committee recognizes that no diet can guarantee full protection against any disease, we believe that our recommendations offer the best nutrition information currently available to help Americans reduce their risk of cancer." The Guidelines were to:

1. Choose most of the foods you eat from plant sources. Eat 5 or more servings of fruits and vegetables each day. Eat other foods from plant sources such as breads, cereals, grain products, rice, pasta, or beans several times each day.
2. Limit your intake of high fat foods, particularly from animal sources. Choose foods low in fat. Limit consumption of meats, especially high-fat meats.
3. Be physically active; achieve and maintain a healthy weight. Be at least moderately active for 30 minutes or more on most days of the week. Stay within your healthy weight range.
4. Limit consumption of alcoholic beverages if you drink at all.³⁴

In 2001, the ACS published "Nutrition during and after cancer treatment: a guide for informed choices by cancer survivors."³⁵ Again, despite its optimistic tone, the truth was buried deep within the report: "*The state of scientific evidence regarding the effects of nutritional factors on the clinical outcomes among cancer survivors is not sufficient at this time to support a set of firm guidelines for cancer survivors.* p.155 (emphasis added). The article went on to say that "There is sufficient information to serve as a basis for a framework for reasonable informed choices." What followed was basically just sound nutritional advice. The ACS still seems unwilling to pop the public's bubble on the realities of diet and cancer.

In its most recent report (2006), the ACS still admits that "No diet or lifestyle pattern can guarantee full protection against any disease..." ACS goes on to note that its recommendations are consistent with guidelines established by other countries and those of the American Heart Association, the American Diabetes Association, and the 2003 Dietary Guidelines for Americans.³⁶ Its recommendations are consistent with those for general health, not protective for cancer.

Dietary fiber and colo-rectal cancer.

In 1999, Americans were shocked by the findings of the largest, best quality study yet done on dietary fiber and colon cancer. A 16-year prospective study of 88,757 health women 34-59 years old at the outset (there were 787 cases of colo-rectal cancer and 1,012 patients with adenomas among 27,530 who underwent endoscopy during the follow-up period) found no important protective effect of dietary fiber against colo-rectal cancer or adenoma after adjustment for age, established risk factors, and total energy intake.³⁷

In 2000, two intervention studies reported failure to find a connection between dietary factors and colo-rectal adenomas. The Polyp Prevention Trial Study Group randomly assigned 2,079 men and women 35-years-of-age or older who had one or more histologically confirmed colo-rectal adenomas to one of two groups. The treatment group received intensive counseling and was assigned to follow a diet low in fat (20%), high in fiber (18 g/1000 kcal), and fruits and vegetables. The controls received a standard brochure on healthy eating and assigned to follow their usual diet. After four years, 958 (39.7%) in the intervention group, and 947 (39.5%) in the control group had at least one recurrent adenoma.³⁸ The Phoenix Colon Cancer Prevention Physicians' Network randomly assigned 1,429 men and women, 40 to 80-years-of-age who had one or more histologically confirmed colorectal adenomas to a supervised program of dietary supplementation with either high or low of wheat bran fiber. Results: 338 (47%) in the high fiber group, and 299 (51.2%) of the low fiber group experienced at least one recurrent adenoma.³⁹ These studies were limited by the relatively brief period of time that they covered, and were delimited by their populations of individuals who may be presumed to be in the later stages of the evolution of colo-

^hThis recommendation is based upon a single case-control study that, according to Lorna Linda University cancer epidemiologist John Morgan, has never been replicated.

ⁱNitrosamines are naturally-occurring potential cancer-causing agents that are, like sunlight, unescapable because they are so widespread. The possibility that nitrosamines were a cause of cancer was debated extensively during the food additive hysteria of the 1970s. Although the issue was clarified by scientists, anticarnivorists never let go of issues that have alarmed the public about eating meat at some time in the past. Kushi stated in 1995, "N-nitroso compounds are abundant in certain meats, especially those which are processed."²⁹ What was left unsaid is that nitrates are abundant in common vegetables (e.g. beets, celery, carrots, spinach) and vegetable sources contribute about 86 mg daily per person. Cured meats supply an average of about 2.4 mg per day. Other variables such as not refrigerating cooked vegetables also increases their nitrite content.³⁰ Also, nitrosamines are formed by the action of microbial reduction when secondary amines (proteins) are added to normal human saliva.³¹ Another source says that the average daily ingestion of nitrite in the diet of an average US resident in 1972 has been estimated to be: from all vegetables, 198 mcg, 2,380 mcg from cured meats; and 8,620 from saliva.³² A leading nitrite fear-monger's reasoning was that eliminating cured meats would cut the total amount of intake. By the same reasoning, however, cutting down on certain vegetables would also reduce nitrite intake, but no one advocated reducing vegetable intakes. The nitrite issue has become even more meaningless since the meat industry has replaced nitrites with vitamin C and erythorbic acid which has reduced the residual nitrite levels in cured meats by 80%.³³

rectal adenomas. Although adenomas are a risk factor, most do not evolve into colorectal cancer. Observational studies around the world have found colo-rectal cancer to be lower among populations with high intakes of fruits and vegetables and that the risk changes on adoption of a different diet, but it is still not understood whether any single aspect of the diet, dietary practices (e.g. methods of cooking meat), or other factors account for these outcomes.⁴⁰

Diet and Coronary Artery Disease

Coronary artery disease (CAD) aka, coronary heart disease and ischemic heart disease, involves the heart's blood supply, is one of four major types of heart disorder. The others are congenital defects, rheumatic fever and congestive heart failure. Statistically, CAD is the #1 cause of death in US & most other industrialized nations. The 2003 rate for all forms of heart disease was 232.1. The US CAD rate has been dropping since its peak in 1950 for unknown reasons. Deaths per 100,000: 1950=307.2; 1960=286.2; 1970=253.6; 1980=202.0; 1990=152.0; 1992=144.3. Curiously, this time period coincides with the wider use of trans-fats and a substantial increase in per capita meat consumption.

Coronary artery disease and some common misconceptions about it.

Myth: CAD is a 20th Century disease.

Reality: Mummies of Egypt's 19th-21st dynasties were found to have died of CAD (1400 BC--King Tut was 18th dynasty; Ramses 19th). A 2100 years dead lady from China's past was found to have died of CAD. Conclusion: CAD has been with us for a long, long time. Historically, it took a long time to figure out what was going on. Circulation of blood discovered by Harvey who published in 1628; angina pectoris recognized by Heberden in 1768; digitalis used for heart failure by Withering in 1785; stethoscope invented by Laennec in 1819; Herrick established the diagnosis and threat of coronary thrombosis in 1912.⁴¹

Myth: CAD involves plugging arteries like accumulations in a pipe.

Reality: Anatomy: *adventitia* (outer wall of artery); *media* (smooth muscle & elastic tissue); *intima* (lining membrane of flat cells); *lumen* (the opening through which blood passes); closing of lumen = *stenosis*. Mechanism of CAD involves injury to intima. The leading suspect of such injuries are inflammatory responses to infectious agents.

Risk factors identified by the Framingham study.

Researchers at the National Heart, Lung, and Blood Institute's Framingham Heart Study (the nation's longest running prospective epidemiological study of CAD) say that 1-in-2 men and 1-in-3 women ages 40 and younger will develop coronary heart disease. At age 70, 1-in-3 men and 1-in-4

women will develop the disease during their remaining years of life.⁴² Risk factors were said to be:

Age	Rises with increasing age, especially after 55 yrs.
Smoking	25% of CAD deaths attributable to cigarette smoking. (CO, vasoconstriction, and >clotting)
Hypertension	1/3 of the total risk.
High cholesterol	11% of total risk; particularly LDL cholesterol.
Obesity	Sudden death was not necessarily due to CAD. Overexertion can cause coronary insufficiency.
Diabetes	Control blood sugar; avoid early degeneration.
Gender	Being male. Estrogen is protective, but men object to side-effects.
Heredity	Family history of heart disease.
Inactivity	An important independent factor.
Combinations	Cigarette smoking, hypertension and high LDL account for 60-65% of total risk of CAD death. Combinations increase risk by more than simple addition. They multiply the risk. For instance, three risk factors increase the risk 10-fold, rather than just 3-fold (i.e. additive).

Genetic factors in the development of CAD.

Genetic factors are often played down because they are not modifiable. The result has been a distortion of perspective in the public mind as to the powerful role heredity plays. Simopoulos, et al, state that "it has been estimated that heredity accounts for 65 percent of the development of coronary heart disease. Of this, about 25 to 30 percent is attributed to single gene defects affecting the way our bodies metabolize fats, and the rest to multifactorial elements--numerous genes, many environmental factors, and the interactions among them."⁴³ Heredity is so powerful that 50% of heart attacks occur in only 5% of families.

Specific genetic factors identified include:

- Familial hypercholesterolemia afflicting 15% of those with heart attacks.
- Familial hypertriglyceridemia responsible for 5% of heart attacks.
- Familial combined hyperlipidemia responsible for about 10% of all heart attacks.
- Lipoprotein (a) in combination with the three above.

- Type III hyperlipoproteinemia in combination with obesity, diabetes or underactive thyroid. Apolipoprotein defects, a variety of which exist.
- Homocysteine, a normal amino acid when elevated promotes clotting everywhere in the arteries.
- Hemochromatosis, a genetic disorder that causes excessive body iron.

Controllable risk factors the authors list include:

- Cigarette smoking
- Hypertension (which also has powerful genetic components)
- Diabetes mellitus (ditto)
- Obesity (ditto)
- Stress (greatly exaggerated in the public mind)
- Lack of exercise
- Personality (Type A overrated, but anger and free floating hostility appear to be valid risk factors) High blood cholesterol (+240 mg/dl)
- Low HDL
- High triglycerides

1988 Behavior Risk Factor Surveillance System (BRFSS) and its findings.

The Office of Surveillance and Analysis and Cardiovascular Health Branch, Division of Chronic Disease Control and Community Intervention, Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention monitors risk factors for CAD. According to the 1988 BRFSS, sedentary lifestyle was the most prevalent (58%) modifiable risk factor for CAD reported, followed by cigarette smoking (25%), obesity (22%), hypertension (17%), and diabetes (5%). Based upon the 1976-1980 2nd National Health and Nutrition Examination Survey it is estimated that 31 % of persons 20-74 yrs-old have serum cholesterol levels greater than 200 mg/dl.⁴⁴

Genetics apparently trump lifestyle.

One hundred healthy people over 90 years of age (50 men and 50 women) were randomly recruited from two Amsterdam homes for the aged were interviewed regarding their lifestyles as middle-aged adults. The coronary risk profile of these people was determined to be "a mirror image of that of the contemporary coronary-prone middle-aged adult."⁴⁵

Living to Be 100.

Seegerberg⁴⁶ wrote about 1200 people who lived to be 100. His data came mostly from interviews conducted by the Social Security Administration, while some was garnered from gerontological studies. No patterns emerged that can serve as guides, but there is an absence of healthful living zealots.

Attributions: Edward Ocker, Albany, NY: "I like fat. I always did like the fat meat. I guess I'd have no trouble up north eating what they call blubber. Most people don't eat the fat. I do. I eat fat."(p.143) Leslie Carpenter, Rochester, IN, gave as his reason for longevity: "I think because I eat a lot of fat pork. I love fat pork." A recent media reports on oldster, Mary Clark, 106, stated that she claims her daily dose of fries keeps her healthy.⁴⁷

*National Geographic*⁴⁸ proclaimed that it was going to reveal "the secrets of living longer" on the cover of its November, 2005, issue. However, a careful reading found no such revelations.¹ Rather, readers were treated to superficial descriptions of the lives of oldsters in Okinawa (Japan), Sardinia (Italy), and Loma Linda, California. In my opinion, the article would have been more useful if it had pointed out contradictions in the lifestyles of these populations. Adventists eschew pork while both the Okinawans and Sardinians savor the swine. According to a History Channel presentation on *Spam*, Okinawans cultivated a taste for the pork shoulder-ham product after contact with the U.S. military following the Occupation and are the highest per capita eaters of *Spam* in the world. Further, Adventist longevity is based upon an aggressive public relations effort by the Loma Linda University School of Public Health which compares the health and longevity of Adventists to the general population rather than a comparable group of nonsmoking, middle-class, well-educated, socially connected people with above average access to health care that does not follow the Adventist lifestyle. When asked why Adventists' lifestyle instead of Mormons have been so extensively studied when Mormons fared a bit better in earlier studies, Adventist Health Study project director David Snowden informed me that the reason was that the Adventist population is more diverse in its eating habits than Mormons who all eat a lot of meat.

More recent information comparing vegetarians and nonvegetarians.

Three prospective studies examined the mortality of vegetarians in Britain (European Prospective Investigation in Cancer and Nutrition-Oxford (n=56,000), the Health Food Shoppers Study (n=11,000), and the Oxford Vegetarian Study (n=11,000). Mortality for major causes of death was not significantly different between vegetarians and nonvegetarians. The only significant difference between vegetarians and non vegetarians was mortality from mental and neurological diseases, rr=2.46.⁴⁹

The most important heart disease intervention study that has been done.

In 1984 the National Heart, Lung, and Blood Institute completed a ten-year intervention study in which 3,806 men 35 to 59 years-old with cholesterol levels above 265 deciliters (i.e. hypercholesterolemic) were divided into two

¹Not smoking, maintaining close family ties, being physically active, and eating wisely is hardly news.

groups; half were put on 6-time daily doses of cholestyramine (a gritty, unpleasant, cholesterol lowering drug that works by binding with cholesterol in the bowels to reduce the amount reabsorbed) mixed with orange juice. The other half received a similarly gritty, unpleasant placebo. Diet was not controlled because of the difficulty of measuring the diets of so many men over such a long period of time. Compliance was understandably poor due to the unpleasant nature of the treatments, but among those who stuck it out, the experimental group achieved an average reduction in cholesterol levels 8.5% below that of controls and had suffered 19% fewer heart attacks, and 24% fewer deaths. Basil Rifkind, the project director, noted that for every 1% reduction in total cholesterol level, there was a 2% reduction of heart disease risk, and that this study had provided the evidence scientists had been waiting for, and that this study's findings was a turning point in heart disease research. When *Time* magazine (3/26/84) reported on this study, its cover pictured a plate of bacon and eggs; its story was titled "Hold the eggs and butter" And, although the study involved neither average people nor diet, *Time's* opening paragraph quoted Basil Rifkind as saying that "the more you lower cholesterol and fat in your diet, the more you reduce your risk of heart disease," the "you" ostensibly meaning the magazine's average reader.

As a result of the NIH study, in 1988 the National Cholesterol Education Program put together a set of guidelines urging that all people over 20 years of age have their cholesterol measured. Those below 200 mil/dec would be told to have another measurement in 5 years; those above 200 could be put on diet modification or drug therapy depending upon their cholesterol, LDL levels, and other risk factors. These guidelines would detect everyone who has even a remote chance of developing CAD; it would involve many who do not have heart attacks despite the fact they exhibit "risk factors." A large study found that 973/1000 men treated with cholesterol-lowering drugs survived 5 years without a heart attack while 959 of the placebo group did so. The cost per-year-of-life-saved was estimated to be \$990,000. Medical economists question whether this can be justified.

Casting doubt on the value of intervention.

A Finnish study began in 1974 randomly divided 1,222 business executives with one or more risk factors into intervention and control groups. Those in the intervention group were seen regularly and advised about diet, physical activity and smoking; and, were treated if hypertension or hyperlipidemia was present. Five years later, the predicted risk of CAD had fallen by half in the intervention group. Despite this, more non-fatal and fatal heart attacks had occurred in the intervention group. All subjects were followed up 10 years after the end of the study. All cause mortality, cardiac deaths and deaths associated with violence was still significantly higher in the intervention group 15 years after original allocation. There is no convincing explanation for these findings. No evidence of control group "contamination"; adverse effects of medication used to treat intervention

group seems unlikely (only 1/3 received medication, and follow-up among those treated found that subgroup had a marginally significant reduction in CAD death and all cause mortality); "rebound" after treatment ended deaths was not apparent upon examination of data; higher death associated with violence is consistent with several other cholesterol-lowering trials which remains unexplained.⁵⁰

References

1. National Analysts. *A Study of Health Practices and Opinions*. Wash, DC: National Technical Information Service, Dept of Commerce (distributor) PB 210 978, 340 pp., 1972.
2. Maracom Research Corp. *A Summary Report on U.S. Consumers' Knowledge, Attitudes and Practices About Nutrition-* 1980. Mpls: Gen Mills, Inc. 1980.
3. National Analysts, 1972; Depth interviews.
4. Council on Foods and Nutrition, *Resource Conference on Food Faddism and Cultism*. Chicago: Am Med Assoc, 10/26-27/71.
5. Larkin M, "Confessions of a former women's magazine writer," *Priorities*, Fall/Winter, 1993.
6. Good Housekeeping Institute/American Dietetic Association/Kraft General Foods survey, *Good Housekeeping*, Nov. 1991.
7. Jarvis WT. "Food and Nutrition Mythology," in: Philip L White and Nancy Selvey, ed. *Malnutrition: Determinants and Consequences, Proceedings of the Western Hemisphere Nutrition Congress VII*, Miami Beach, FL, Aug 7-11, 1983, 494pp, New York: Alan R Liss, Inc, 1984.
8. Maizel, J. *The Beverly Hills Diet*. Macmillan, 1981.
9. Diamond H, Diamond M. *Fit For Life* Warner Books, 1985, pA9.
10. Robbins A. *Unlimited Power*, Fawcett Columbine, 1986, pp.172-5.
11. Harington J. *The School of Solernum Regimen Sanitatus Salemi, 1611*, Ente Provinciale per Il Turismo-Salerno, undated.
12. *Dr. Atkins' Diet Revolution*. Bantam Books, 1972, p.5.
13. *Nutrition Applied Personally*. International College of Applied Nutrition, c.1974, pp.56-7.
14. Whorton J, "Tempest in a flesh-pot: the physiological rationale for vegetarianism," *J Hist Med*, 1977;[April]:115-39.
15. Hanlon JJ. *Principles of Public Health Administration, Fifth Edition*, St. Louis: CV Mosby Co., 1969, p.5.
16. Doll R. "Health and environment in the 1990s," *Am J Publ Hlth* 1992;82:933-41.
17. "American life expectancy hits record high" Associated Press, Feb 28, 2005.
18. Fries J. "Aging, natural death, and the compression of morbidity," *New Engl J Med* 1980;303:130-5.
19. *UC Berkeley Wellness Letter*, 10/91.
20. Nieto FJ. "Cardiovascular disease and risk factor epidemiology: a look back at the epidemic of 20th century," *Am J Publ Hlth* 1999;89:292-4.
21. Taubes G. "Epidemiology faces its limits," *Science*, 1995;269(July 14):164-9.
22. *J Nat Can Inst* 1981;66:1192-1251.
23. *Ca-Can J Clin*, 1999;49:51.
24. *Proceedings Canadian Cancer Conference* 1969;8:40-75.

25. Wynder RL, Gori GB. "Contribution of the environment to cancer incidence: an epidemiologic exercise," *J Natl Can Inst* 1977;58:825-832.
26. Parkin et al. "Global cancer statistics," *Ca-Can J Clin*, 1999;49:33-64.
27. National Academy of Sciences' Reports on Diet and Health--Are They Credible and Consistent (GAO/RCED-84-109).
28. *Ca-Can J Clin*, 1984;34(2):121-6.
29. Kushi LH, Lenart EB, Willett WC. "Health implications of Mediterranean diet in light of contemporary knowledge. 2. Meat, wine, fats, and oils." *Am J Clin Nutr*, 1995;61:1419S.
30. Jukes TH. "Current Concepts in Nutrition: Food Additives," *New Engl J Med*, 1977;297:427.
31. Tannenbaum SR, Archer MC, Wishnok JS, Bishop WW. "Nitrosamine in human saliva," *J Natl Cancer Inst*, 1978;60:251-3.
32. Hickey RJ, Clelland Re. "Hazardous food additives: nitrite and saliva," *New Engl J Med*, 1978;298:1036-7 (Cites White JW Jr. "Relative significance of dietary sources of nitrate and nitrite," *J Agric Food Chem*, 1975;23:866-91; 1976;24:202.
33. "The good news about cured meat products," *Environmental Nutrition*, December, 1997, p.8.
34. *Ca-Can J Clin*, 1996;46(6):325-41.
35. Brown J, Byers T, Thompson K, Eldridge B, Doyle C, Williams AM. *Ca-Can J Clin*, 2001;51(3):153-187.
36. Kushi L, et al. "American Cancer Society Guidelines on Nutrition and Physical Activity for Cancer Prevention: Reducing the risk of cancer with healthy food choices and physical activity," *Ca-Can J Clin*. 2006;56(5):254-281; p.255-256.
37. Fuchs, et al. "Dietary fiber and the risk of colorectal cancer and adenoma in women," *New Engl J Med*, 1999;340:169-76.
38. *N Engl J Med*, 2000;342:1149-55.
39. *N Engl J Med*, 2000;342:1156-62.
40. Editorial, *N Engl J Med*, 2000;342[16].
41. From White, PD. "The evolution of our knowledge of the heart and its diseases," in Hurst & Logue, *The Heart, 2nd Ed*, McGraw-Hill, 1970.
42. "Coronary heart disease; lifetime risk is high," *The Nation's Health*, March, 1999, p.28.
43. Simopoulos AP, Herbert V, Jacobson B. *Genetic Nutrition*. New York; MacMillan, 1993, pp. 86-87.
44. Centers for Disease Control, MMWR, 9/17/90.
45. Danner SA, De Beaumont M, Dunning AJ. "Cardiovascular health in the tenth decade," *Brit Med J*, 1978;277:663.
46. Segerberg O. *Living To Be 100; 1200 Who Did and How They Did it*, New York: Chas Scribner's Sons, 1982.
47. "Perspectives." *Newsweek*, May 14,2001, p.15.
48. Buettner D. "The secrets of long life," *Nat Geog*, November, 2005, pp.2-27.
49. Key TJ, et al. "Mortality in British vegetarians: review and preliminary results from EPIC-Oxford," *Am J Clin Nutr*, 2003;78(suppl):533S-8S.
50. Oliver MF. "Doubts about preventing coronary heart disease," *Brit Med J* 1992;304:393-4.