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Nutrition 24 (2008) 393–398

NUTRITION

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Book review

Food, Nutrition, Physical Activity and the Prevention of Cancer: A Global Perspective

World Cancer Research Fund/American Institute for Cancer Research. American Institute for Cancer Research, Washington, D.C., 2007. ISBN: 978-0-9722522-2-5.

During the late 20th and early 21st century, research interest in the relations among food, nutrition, lifestyle, and cancer prevention or predisposition has blossomed. In 2001, the World Cancer Research Fund, in collaboration with the American Institute for Cancer Research, launched a 5-y effort to identify and evaluate the global scientific literature describing these relations. The goal of this effort was to produce recommendations based on unbiased expert judgments of systematic reviews of the world literature that also are in harmony with nutritional and lifestyle recommendations designed to decrease the incidence of other disease conditions and promote general good health and well-being.

Most significantly, the primary goal was to accomplish a systematic review of *all* the relevant research findings, implementing a unique process that considered evidence provided by classic ecologic studies, including prospective cohort and case-control studies; intervention studies using foods, diets, or food components in various forms; and laboratory studies in humans and, to a lesser extent, animals to generate a comprehensive series of recommendations on food, nutrition, and physical activity, designed to reduce the risk of cancer and be suitable for all societies. Organized into three overlapping stages, the process was designed to maximize objectivity and transparency, separating the collection of evidence from its assessment and judgment.

First, an expert task force developed a method for the systematic review of the voluminous scientific literature. Second, research teams implemented this methodology in the collection and summarization of the literature. Third, an expert panel assessed this evidence, exploring the extent to which food, nutrition, physical activity, and body composition modifies the risk of cancer, a potentially preventable disease, and developed consensus recommendations that are based on solid evidence. The result of this 5-y effort is an authoritative summary of existing knowledge, an unbiased assessment of that knowledge base, and guidance for future scientific research, cancer prevention programs, and health policy around the world. It provides the solid evidence base required by policymakers, health professionals, and anyone else interested in this most timely topic.

The report itself is divided into three sections. The introductory section details the impact of urbanization and industrialization on changing patterns of cancer throughout the world and outlines the current understanding of the biology of the cancer process. Of particular interest is the evidence that patterns of cancer prevalence change as populations migrate from underdeveloped to developed regions, that non-genetic factors are the most important mediators of cancer initiation and progression, and that cancer incidence is expected to increase in parallel to continued migration to more carcinogenic environments. This section closes with a description of the types of evidence that were examined by the developers of the report and the rationale behind the decision to not limit science to simply placebo-controlled intervention human clinical trials (the position that reliable judgments concerning the causation or prevention of disease can be based on the assessment of a variety of well-designed epidemiologic and experimental studies is expanded in the companion text, *Systematic Literature Review Specification Manual*).

The second section of the report presents the findings of the systematic reviews of the literature upon which the report's conclusions and recommendations depend. These conclusions are summarized in three-dimensional illustrations that provide the foundation for each recommendation. This section of the report focuses on foods, beverages, specific dietary constituents, patterns of eating behavior, physical activity, body composition, growth and development, weight gain and excessive body weight, and the impact of diet and lifestyle on cancer survivability and recurrence. All conclusions and recommendations are presented in the context of the concurrent prevention of nutrition-responsive chronic diseases, including deficiency syndromes and predispositions to infectious disease, and are harmonized with other widely accepted prevention guidelines.

The third section of the report presents public health goals and personal recommendations that are driven by the existing scientific knowledge base. These are proposed as the basis for public policies and for personal choices that are expected to reduce the incidence of cancer for individuals, families, and communities. Eight general and two special goals and recommendations are detailed. In each case a general recommendation is followed by public health goals and personal recommendations, together with a summary of the evidence, justification of the goals and recommendations, and guidance on how they can be achieved. In addi-

tion, the dietary patterns most likely to protect against cancer are discussed.

The goals and recommendations are designed to be generally relevant worldwide, although they will be best used in combination with locally targeted recommendations designed to prevent chronic and other diseases. Although the main focus of the report is on nutritional and other biological and associated factors that modify the risk of cancer, that risk also is modified by social, cultural, economic, and ecologic factors. Consideration of such local influences must be integrated with the report's global conclusions and recommendations.

The report's conclusions and recommendations were presented at the 2007 annual World Cancer Research Fund/ American Institute for Cancer Research scientific conference ("Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective") held in Washington, D.C., on November 1 and 2, 2007. More than 500 attendees were provided the opportunity to examine and evaluate the summary findings, conclusions, and recommendations in the report. In an introductory presentation ("Development of the Second Expert Report"), Geoffrey Cannon, chief editor of World Cancer Research Fund International, asserted that even small decreases in individual risk can become extremely impactful when experienced by large populations. He also illustrated the report's conclusions that the available scientific evidence is convincing that risk for developing cancer is reduced or suggests that risk for developing cancer probably is reduced by the items listed in Table 1.

Table 1
Decreasing the risk for developing cancer

Increased consumption of	Sites of cancer
Dietary fiber	Colon and rectum
Non-starchy vegetables	Mouth, pharynx, larynx, esophagus, stomach
Allium spp. of vegetables	Stomach
Garlic	Colon and rectum
Fresh fruits	Mouth, pharynx, larynx, esophagus, stomach, lung
Folate	Pancreas
Lycopene	Prostate
Selenium	Prostate
Calcium	Colon and rectum
Decreased consumption of	
Aflatoxins	Liver
Red meat	Colon and rectum
Processed meat	Colon and rectum
Cantonese-style salted fish	Nasopharynx
Calcium	Prostate
Salt, salted, or salty foods	Stomach
Arsenic in drinking water	Lung, skin
Yerba mate tea	Esophagus
Alcoholic drinks	Mouth, pharynx, larynx, esophagus, liver, colon and rectum, breast (premenopausal and postmenopausal women)
β-carotene	Lung (life-long tobacco smokers)
Other factors	
Increased daily physical activity	Colon and rectum, endometrium, breast (postmenopausal women)
Increased body fatness	Breast (premenopausal women)
Decreased body fatness	Esophagus, pancreas, gallbladder, colon and rectum, breast (postmenopausal women), endometrium, kidney
Decreased abdominal fatness	Pancreas, colon and rectum, endometrium, breast (postmenopausal women)
Decreased weight gain during adulthood	Breast (postmenopausal women)
Decreased maximum adult height	Pancreas, colon and rectum, ovary, breast (premenopausal and postmenopausal women)
Decreased birth weight	Breast (premenopausal women)
Increased duration and frequency of lactation	Breast (premenopausal and postmenopausal women)

The report's recommendations were then explained by a series of presenters, most of whom had served on the panel of experts that assembled and wrote the final report. The eight general and two special recommendations and their accompanying public health goals and personal recommendations are listed in Table 2.

Walter C. Willett, M.D., Dr.P.H. (Harvard School of Public Health, Boston, Massachusetts, USA), introduced "Recommendation 1: Body Fatness: Be as Lean as Possible Within the Normal Range Of Body Weight." According to Dr. Willett, maintenance of a healthy weight throughout life may be one of the most important ways to protect against cancer. Being overweight or obese increases the risk of some cancers, in addition to dyslipidemia, hypertension and stroke, type 2 diabetes, and coronary heart disease. In particular, the evidence is clear that the risk of death from any cancer increases with increasing body mass index (BMI). In addition, the most compelling evidence is the demonstration that the risks for colorectal and postmenopausal breast cancer increase with increasing BMI. Furthermore, the risk for postmenopausal breast cancer increases with BMI even within the range of 21 to 25 kg/m^2, especially in women with estrogen receptor–positive breast tissue.

Rachel Ballard-Barbash, M.D., M.P.H. (National Cancer Institute of the National Institutes of Health, Bethesda, Maryland, USA; "Body Fatness, BMI and Cancer: Potential Mechanisms and Markers"), explained that as BMI increases, serum concentrations of sex hormone–binding globulin decrease, resulting in increased circulating concentrations of free estradiol and free estrogen. At the same time, hyperinsulinemia associated with low levels of routine daily physical activity decreases serum concentrations of sex hormone–binding globulin. Together, these interactions act to increase the exposure of estrogen receptor–positive breast tissue to antigenic stimuli. In contrast, at any BMI, increased routine daily physical activity stimulates insulin sensitivity, promotes increased serum concentrations of sex hormone–binding globulin, and reduces the risk for breast and colorectal cancers. One mechanism that might

Table 2
Recommendations and accompanying public health goals and personal recommendations

Recommendations	Public health goals	Personal recommendations
General		
1. Body fatness: be as lean as possible within the normal range of body weight.	Median adult BMI 21–23 kg/m^2, depending on normal range for different populations. The proportion of the population that is overweight or obese to be no more than the current level, or preferably lower, in 10 y.	Ensure that body weight through childhood and adolescent growth facilitates remaining within the lower end of the normal BMI range at age 21 y. Maintain body weight within the normal range from age 21 y. Avoid weight gain and increases in waist circumference throughout adulthood.
2. Physical activity: be physically active as part of everyday life.	The proportion of the population that is sedentary to be halved every 10 y. Average physical activity levels to be greater than sedentary.	Be moderately physically active, equivalent to brisk walking, for ≥30 min/d. As fitness improves, aim for ≥60 min/d of moderate activity or for ≥30 min/d of vigorous, physical activity. Limit sedentary habits such as watching television.
3. Food and drinks that promote weight gain: limit consumption of energy-dense foods; avoid sugary drinks.	Average energy density of diets to be lowered toward 125 kcal/100 g. Population average consumption of sugary drinks to be halved every 10 y.	Consume processed foods with energy densities >225 kcal/100 g sparingly. Avoid drinks with added sugars. Consume fruit juices in moderation.
4. Plant foods: eat mostly foods of plant origin.	Population average consumption of non-starchy vegetables and of fruits (of a variety of colors) to be ≥600 g (21 oz)/d. Relatively unprocessed cereal grains, pulses (legumes), and other foods high in dietary fiber to contribute to a population average of ≥25 g/d of non-starch polysaccharide.	Eat ≥5 servings (totaling ≥400 g or 14 oz)/d of a variety of non-starchy vegetables. Eat ≥5 servings (totaling ≥400 g or 14 oz)/d of a variety of fruits. Eat relatively unprocessed cereal grains, pulses (legumes), and other foods high in dietary fiber with every meal. Limit refined starchy foods. If starchy roots or tubers are traditional dietary staples, add non-starchy vegetables, fruits, and pulses (legumes) to the daily diet.
5. Animal foods: limit intake of red meat and avoid processed meat.	Population average consumption of red meats (from domesticated cattle, swine, sheep, or goats) to be ≤300 g (11 oz)/wk. Minimize consumption of smoked, cured, salted, or chemically preserved meats.	If red meats are consumed, limit to ≤500 g (18 oz)/wk. Avoid consumption of smoked, cured, salted, or chemically preserved meats.
6. Alcoholic drinks: limit alcoholic drinks.	Proportion of the population drinking >2 drinks/d (men) or >1 drink/d (women) to be reduced by one-third every 10 y (10–15 g ethanol/drink).	If alcoholic drinks are consumed, limit to ≤2 drinks/d (men) or ≤1 drink/d (women); 10–15 g ethanol/drink.
7. Preservation, processing, preparation: limit consumption of salt; avoid moldy cereal grains and pulses (legumes).	Population average consumption of salt from all sources to <5000 mg/d. Proportion of the population consuming >6000 mg/d of salt to be halved every 10 y. Minimize exposure to aflatoxins from moldy cereal grains and pulses (legumes).	Avoid salt-preserved, salted, or salty foods. If preserved foods are consumed, choose foods that have been preserved using refrigeration, freezing, drying, bottling, canning or fermentation. Limit consumption of foods with added salt. Do not eat moldy foods.
8. Dietary supplements: aim to meet nutritional needs through diet alone, if possible.	Maximize the proportion of the population achieving nutritional adequacy without the need for dietary supplements.	Dietary supplements are not recommended for cancer prevention (because of the increased risk of adversely high intakes).
Special		
1. Breast-feeding: mothers to breast-feed, children to be breast-fed.	Most mothers to breast-feed exclusively for 6 mo (human milk only; no other food or beverages).	Aim to breast-feed infants exclusively for up to 6 mo and continue with complementary feeding thereafter.
2. Cancer survivors: follow the general recommendations for cancer prevention.	All cancer survivors to receive nutritional care from an appropriately trained professional.	If able to do so, and unless otherwise advised, aim to follow the general recommendations for diet, healthy weight, and physical activity.

BMI, body mass index

link increased physical activity and reduced risk for non–hormone-sensitive cancers may be the effect of increased activity to stimulate weight loss; humans losing weight often exhibit less procarcinogenic DNA adduct formation and lower plasma concentrations of methylmalonic acid (a biomarker for systemic oxidative stress).

Juan A. Rivera, Ph.D. (Instituto Nacional de Salud Publica, Cuernavaca, Mexico), introduced "Recommendation 2: Physical Activity: Be Physically Active as Part of Everyday Life." According to Dr. Rivera, most existing human populations, especially those living in industrialized and urban settings, have habitual levels of activity that are lower than the

levels of routine energy expenditure to which the human body is adapted. Physical inactivity coupled with the ready availability of energy-dense foods promote widespread obesity in these settings. In contrast, all forms of physical activity are chemoprotective and decrease the risks for developing obesity-related chronic diseases and early mortality.

Henry J. Thompson, Ph.D. (Colorado State University, Fort Collins, Colorado, USA; "Physical Activity and Cancer: Energetics, Mechanisms and Markers"), presented the hypothesis that carcinogenesis reflects a defect in energy metabolism caused by mitochondrial dysfunction. This defect may be exacerbated by physical inactivity, whereas physical activity may reduce cancer risk independently of any effects on body fatness. Muscle cells that have been worked to fatigue secrete several cytokines, including a tumor-suppressive factor. These messengers appear to affect energy metabolism in non-muscle cells, including breast epithelial cells. In the absence of the tumor-suppressing factor, precancerous cells can develop resistance to muscle cell–derived "energy messengers" and express various manifestations of mitochondrial dysfunction that disrupt normal cell cycling.

Shiriki Kumanyika, Ph.D., M.P.H. (University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania, USA), introduced "Recommendation 3: Foods and Drinks That Promote Weight Gain: Limit the Consumption of Energy-Dense Foods and Avoid Sugary Drinks." According to Dr. Kumanyika, consumers can confuse the term *nutrient-dense* with *nutrient-rich*. Therefore, it is recommended that the terms *energy-dense* and *calorie-dense* be used to describe foods providing more than 225 kcal per 100 g or per 100 mL. Efforts to reduce calorie consumption are especially thwarted by energy-dense beverages; such beverages appear to exert little influence on total daily self-selected energy intakes and their habitual consumption can lead to rapid and sustained weight gain even in the face of restricted solid food intake.

June Stephens, Ph.D. (University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA; "Obesity Prevention"), discussed the difficulties of studying the prevention of obesity in humans. Because randomized, controlled trial designs are used more frequently to evaluate obesity prevention programs, careful attention must be paid to the selection of outcome measurements with high validity, the description of the intervention, the use of analyses that match the sampling design, and dealing with loss to follow-up. The use of preliminary or evidentiary research designed to develop and test intervention components and hypothesized mediators is recommended before fully powered, randomized, obesity prevention trials are attempted. Randomized and observational designs can be used in these preliminary studies. Attention to design issues will ultimately lead to more useful randomized trials that drive more rapid movement toward efficacious and effective obesity prevention programs.

John A. Milner, Ph.D. (National Cancer Institute of the National Institutes of Health), introduced "Recommendation 4: Plant Foods: Eat Mostly Foods of Plant Origin." According to Dr. Milner, most diets that are protective against cancer are characterized by large intakes of foods of plant origin and, indeed, several cancers are responsive to increased intakes of plant-based foods. However, most responses are non-linear, with the greatest benefits being realized by those with previously limited intakes of such foods. Therefore, it appears that there is a threshold of intake at which risk is rapidly reduced, although risk reduction reaches a plateau at intakes that are less than most recommendations. Consequently, individuals who are consuming five or more servings of non-starchy vegetables plus five or more servings of fruits may not realize any benefits from increasing their consumption further—their risk-benefit ratios may begin to increase as they begin to increase their risks for developing obesity and its associated chronic disease states. Avoiding this unintended consequence will require reliance on non-starchy vegetables such as green leafy vegetables, broccoli, okra, eggplant, and bok choy and non-starchy roots and tubers such as carrots, Jerusalem artichokes, celery root, rutabaga, and turnips, whereas potato, yam, sweet potato, and cassava are avoided. With that caveat, increasing plant-food intake will decrease the risk for developing cancer and increase the likelihood of remaining cancer free.

David M. Klurfeld, Ph.D. (Agricultural Research Service of the U.S. Department of Agriculture, Greenbelt, Maryland, USA; "Plant Foods: Are They Still What's for Dinner?"), explained that most research on plant foods has used laboratory animals, tissue and cell systems, or even cell-free systems; these systems require explicit description of the mechanistic linkages that could explain potential relevant human clinical responses. The criteria perhaps first delineated by A. B. Hill in 1965 (Proc R Soc Med 1965;58:295–300) should guide attempts to create such linkages: the strength of the apparent association, the consistency of the observed association, the specificity of the association, the temporal relation of the association, the potential existence of a gradient of association (changing response with changing exposure), the biological plausibility of the proposed association, and the coherence (consistency with other existing knowledge) of the association. Most human clinical trial databases have addressed the treatment of existing cancer, not the primary prevention of cancer, and have mistakenly equated reduction in the rate of tumor recurrence to "prevention." Unfortunately, the general absence of clinical trials examining diet, nutrition, and cancer forces an over-reliance on the findings of epidemiologic studies; these sources usually rely on relatively unreliable intake data and tend to ignore the variability in the nutrient contents of foods over time, geography, processing, and storage. Often, nutrients themselves are defined inconsistently or vaguely (e.g., "folate" and "folic acid").

In addition, the hormetic effect of nutrient intakes is recognized in the arena of deficiency disease prevention but not in the area of nutritional prevention of cancer. According to this model, a threshold of intake must be achieved

before a beneficial effect will become manifest; intakes below this threshold invariably will have no effect. However, at some level of intake a previously beneficial substance may begin to exert toxicity, direct or indirect. For example, exposure to a small amount of a pesticide may maximize the induction of chemoprotective phase II enzymes, whereas larger exposures may themselves overwhelm the maximum capacity of the phase II detoxification system and induce tumorigenic biochemical cascades. Perhaps requiring an identifiable dose-response relation should be changed to requiring an identifiable threshold-response relation. Furthermore, with a few notable exceptions, most studies of nutrition and cancer intentionally isolate an individual nutrient or food, suppressing any information that could be obtained from a consideration of interactions among exposures.

Arthur Schatzkin, M.D., Dr.P.H. (National Cancer Institute of the National Institutes of Health), introduced "Recommendation 5: Animal Foods: Limit the Intake of Red Meat and Avoid Processed Meat." According to Dr. Schatzkin, despite a growing bias against animal foods within the "cancer community," an integrated approach to the evidence shows that many foods of animal origin are nourishing and healthy if consumed in modest amounts. Meat can be a valuable source of nutrients, in particular protein, iron, zinc, and vitamin B12. Although individuals who conform to some variant of a "vegetarian diet" are at low risk of some diseases including some cancers, this level of risk may reflect other often-concurrent lifestyle choices, such as avoidance of tobacco and alcohol products and regular vigorous physical activity. This recommendation should not be interpreted as an advisory against all foods of animal origin. However, red and processed meats are convincing or probable causes of some cancers. In addition, diets containing large amounts of animal fats are relatively high in energy, increasing the risk of weight gain.

Rashmi Sinha, Ph.D. (National Cancer Institute of the National Institutes of Health; "Meat Intake and Cancer Risk"), explained that the carcinogenicity of foods of animal origin usually stems from the production of *N*-nitroso compounds in the intestinal tract after the consumption of such foods. Human exposure to *N*-nitroso compounds directly reflects red meat intake but is independent of white meat intake. This difference in response may result simply from the much greater heme-iron content of red meats (that produces the red color). It is established that *N*-nitroso compound formation within the human gut is determined by the amount of heme iron that is consumed (but not by non–heme-iron intake). The salting of fish during processing or cooking also stimulates *N*-nitroso compound formation from the combination of nitrate and nitrite contaminants in the salt with the proteins in the meat of fish. Also of note is the formation of polycyclic aromatic hydrocarbons during the incomplete combustion of meat during "charring." The National Cancer Institute makes available free software ("Computerized Heterocyclic Amines Resource for Research in Epidemiology of Disease") that can be downloaded from http://charred.cancer.gov/software and used to calculate the amount of mutagens present in cooked meats.

W. Philip T. James, M.D., C.B.E., Dr.Sc., F.R.S.E., F.R.C.P. (International Obesity Task Force, London, United Kingdom), introduced "Recommendation 6: Alcoholic Drinks: Limit Alcoholic Drinks." According to Dr. James, ethanol disrupts folate and estrogen metabolism and increases the incidence of mammographic densities. In premenopausal women, ethanol stimulates estrogen secretion. In postmenopausal women, ethanol stimulates the conversion of testosterone to estrogen. Consequently, alcohol intake coupled with poor folate nutriture, especially in women with estrogen receptor–positive mammary tissue, increase the risk for developing breast cancer at any age. These (and other) biological effects of alcoholic beverages reflect their content of alcohol per se and are not attributable to congeners or contaminants.

Keith W. Singletary, Ph.D. (University of Illinois, Champaign, Illinois, USA; "Alcoholic Drinks and Cancer: Mechanistic Insights"), described several potential mechanisms through which ethanol may exert carcinogenicity. Ethanol undergoes a two-step sequential pathway of detoxification. First, ethanol in converted to acetaldehyde by alcohol dehydrogenase. In turn, acetaldehyde is converted to acetate by acetaldehyde dehydrogenase. The rate of acetaldehyde formation from ethanol is determined by the presence or absence of single nucleotide polymorphisms in the gene coding for alcohol dehydrogenase. Similarly, the persistence of acetaldehyde is determined by the presence or absence of polymorphisms in the gene coding for acetaldehyde dehydrogenase. Acetaldehyde forms stable DNA adducts and chromosomal aberrations, inhibits DNA repair enzymes and is carcinogenic in cells, animals and humans. Alternatively, ethanol can be metabolized by P450 enzymes, producing reactive oxygen species, and can induce the activity of CYP2E1, a cytochrome P450 enzyme that bioactivates procarcinogens.

Tomio Hirohata, M.D., Dr.Sc.Hyg., Ph.D. (Kyushu University, Fukuoka City, Japan), introduced "Recommendation 7: Preservation, Processing, Preparation: Limit Consumption of Salt and Avoid Moldy Cereal Grains and Pulses (Legumes)." According to Dr. Hirohata, the strongest evidence on methods of food preservation, processing, and preparation shows that salt and salt-preserved foods are probably a cause of stomach cancer, and that foods contaminated with aflatoxins are a cause of liver cancer. Although salt is necessary for human health, typical levels of consumption are vastly excessive. In addition, the microbial contamination of foods, drinks, and water supplies remains a major public health problem worldwide. The contamination of cereal grains and pulses (legumes) with aflatoxins produced by some molds when such foods are stored for too long in warm temperatures is an important public health problem, and not only in tropical countries.

Robert J. Turesky, Ph.D. (Wadsworth Center of the New York State Department of Public Health, Albany, New York, USA; "Impact of Dietary and Environmental Chemicals in Cancer Risk"), reported that the dietary genotoxicants of most concern are the aflatoxins found in moldy cereals, spices, oilseed, and nuts; the heterocyclic aromatic amines found in cooked meats and tobacco smoke, and the *N*-nitroso compounds found in cooked meats and salted fish. The formation rates of these genotoxicants are proportional to storage and cooking times and temperatures. The food contents of these compounds can be maximized quickly; for example, the content of a known human colorectal carcinogen, 2-amino-1-methyl-6-phenylimidazo[4-5-b]pyridine, is maximized in meat in only 5 min when the meat is cooked at 165°C (329°F).

John A. Milner, Ph.D. (National Cancer Institute of the National Institutes of Health), introduced "Recommendation 8: Dietary Supplements: Aim to Meet Nutritional Needs Through Diet Alone." According to Dr. Milner, perhaps the greatest danger associated with the use of dietary supplements is the possibility that the consumption of supplements is serving as an alternative to dietary change (supplements as "magic bullets" that can overcome cancer-friendly dietary and lifestyle practices). Nonetheless, when used to truly "supplement" healthy diets, supplemental nutrients can be invaluable.

Ricardo Uauy, M.D., Ph.D. (Instituto de Nutrición y Tecnología de los Alimentos, Santiago, Chile), introduced "Special Recommendation 1: Breastfeeding: Mothers to Breastfeed; Children to be Breastfed." According to Dr. Uauy, the evidence on cancer and other diseases shows that sustained, exclusive breast-feeding is protective for the mother and the child. The extent of premenopausal breast cancer risk reduction reflects the total number of months of lifetime breast-feeding. Breast-feeding also protects against infections in infancy, protects the development of the immature immune system, protects against other childhood diseases, and is vital for the development of the bond between mother and child. Breast-feeding is especially vital in parts of the world where water supplies are not safe and where impoverished families cannot readily afford infant formula and other foods for infants and young children.

Steven H. Zeisel, M.D., Ph.D. (University of North Carolina at Chapel Hill), introduced "Special Recommendation 2: Cancer Survivors: Follow the Recommendations for Cancer Prevention." According to Dr. Zeisel, when appropriate, and unless advised otherwise by a qualified professional, the recommendations of this report also apply to cancer survivors. In addition, because the treatment of many cancers is increasingly successful and cancer survivors increasingly are living long enough to incur the risk of developing new primary cancers or other chronic diseases, following the recommendations in this report also should reduce the risk of those conditions among survivors of previous cancer.

The full 537-page report can be obtained electronically from http://www.dietandcancerreport.org without cost. Paperback or hardbound copies also may be purchased from the American Institute for Cancer Research through that Web address. Purchased copies include a CR-ROM that contains the report and the full text and references of each of the systematic literature reviews on which the report's conclusions and recommendations are based.

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doi:10.1016/j.nut.2007.12.019