To:	**Denise Soffel, Ph.D.** **Executive Director, Senate Committee on Health**
Via Email:	**denise@tomduane.com**
Re:	**Testimony of the American Cancer Society, Eastern Division (NY and NJ)** **In Support of Requiring Calorie Labeling in certain chain restaurants**
Date:	**January 22, 2010**

Chairman Duane, members of the Health Committee, thank you for the opportunity to testify today on food policy. My name is Kris Kim, and I am the Chief Operating Officer of the American Cancer Society of New York and New Jersey. I will focus my comments today in support of calorie labeling.

As you point out, obesity is a major epidemic with serious implications for the health and economic status of New York and our nation. While most know that excess pounds raise the risk of heart disease, hypertension, diabetes, stroke, few are aware of the link between obesity and cancer. It is currently estimated that 14% of cancer deaths among males and 20% of deaths among females are attributed to obesity (Calle et al., 2003).

National health care expenditures are estimated at $70 to $100 billion per year and are expected to grow with the increasing rates of overweight and obesity (Olshansky, 2005). Healthcare costs are 56% higher for an obese person compared to a normal weight person. This puts significant financial pressure on the Medicaid program and the state budget since obesity is approximately twice as high in low-income populations compared to higher income groups (Willet and Domolky, 2003). The Centers for Disease Control and Prevention's Pediatric Nutrition Surveillance Study of 2002 found that New York State has the 3rd highest rate of low-income overweight children in the country.

Lessons learned in Tobacco Control can help with the Obesity Epidemic

Comprehensive tobacco control has helped states like New York realize significant declines in the rates of smoking and tobacco-related illness and death. If we don't take similar steps to control obesity, it could well replace smoking as the deadliest cause of cancer. We have learned

the substantial benefits of taking an aggressive policy-based approach from the fight against tobacco. Policies that make it easier to pursue healthier behaviors while creating barriers to unhealthy practices have been very successful. In the early years of tobacco control, states like California and Massachusetts implemented a variety of interventions before the effectiveness was clear. When these initial "real-world" efforts were evaluated and proven successful, best practices began to be utilized in other states. Utilizing lessons learned in tobacco, strategies such as calorie labeling, or taxing sugared beverages, should be part of a comprehensive approach to address obesity and the many factors contributing to the problem, drawing on previous success. Clearly, sugared soda is a major contributing factor to the obesity problem. Our history with tobacco taxes indicates that higher prices do drive down demand, but it is not so easy to simply substitute experiences here. With respect to the sugar beverage tax, the state should proceed cautiously and evaluate the impact carefully, perhaps also exploring different ways to reduce consumption of sugary beverages. As part of a comprehensive approach, New York should also consider policies that increase access to physical activity and healthful foods in schools, worksites, and communities, for example improving the school lunch program and enhancing physical activity opportunities for youth in schools.

Support for Calorie Labeling

The American Cancer Society promotes initiatives that empower individuals to make healthier choices, and therefore strongly supports initiatives like calorie labeling. This proposal will help create the environmental changes needed to impact the current trend in obesity.

Like other voluntary health organizations, ACS disseminates nutrition guidelines in order to empower individuals with information to make informed decisions about their health. However, in order for an individual to make an informed decision about what they eat, it is important that nutritional information be readily available *when purchase decisions are being made*. People have grown accustomed to having nutrition information on packaged foods in supermarkets (3/4 of people report using labels) and they want and deserve to have it on menus as well. A recent, industry-sponsored poll showed that 83% of Americans want restaurants to provide nutrition information.

Success in Other Localities

Menu labeling legislation has been introduced in dozens of states and cities across the country, as well as in the U.S. Congress. California was the first state to implement menu labeling, though it won't go into effect until 2011. A few days ago, New Jersey became the 4th state to take this step; Nassau and Suffolk Counties on Long Island, and Albany County have already adopted menu-labeling proposals, though they too have yet to take effect. Westchester and Ulster counties have this provision in effect, though no data is available yet, and New York City has had menu-labeling provisions in effect since July 2008.

A recent study supported by the Robert Wood Johnson Foundation (RWJF) reviewed the impact of NYC's menu labeling provisions. The study determined that after menu-labeling policies were implemented, 56 percent of customers reported viewing the calorie information, and customers made lower-calorie food purchases at 9 of the 13 fast-food and coffee chains included in the study. After only little more than a year in effect, these are very promising results.

While an early study by NYU and Yale University found increased awareness of calorie postings, but no real change in behavior of consumers at several fast food restaurants in lower income neighborhoods*, this more recent study determined that customers who used the calorie information consumed an average of 106 fewer calories than those who did not. (*Report Suggests New York City Menu Labeling Law is Effective at Promoting Health Changes*, 10/26/09, http://www.rwjf.org).

** (Both public health groups and the NYC Department of Health and Mental Hygiene point out drawbacks of the NYU/Yale study. They note that the research was done weeks after implementation of the regulation and may not have accurately captured behavior change. They further note that it is more difficult to change behavior in low-income neighborhoods where individuals are often more influenced by cost than calories. The more recent RWJF study may be more representative of the impact of the new law because it included more consumers over a longer period of time and was not limited to outlets in lower-income neighborhoods.)*

Other Implications of the Policy

In addition to providing consumers with information to help them make informed decisions, menu labeling provides an incentive for restaurants to add new menu items and reformulate existing options to reduce the calories. We saw this happen when Nutrition Facts labels went on

packaged foods in 1994 and we see it now with companies lowering or eliminating trans fats in response to the FDA requiring trans fat labeling. The food industry may think twice about selling a quad burger (4 beef patties, 4 slices of cheese, and 8 slices of bacon), as a leading fast food company does, if they have to show the 1,000 calorie price tag that goes along with it.

Even Trained Experts Need Calorie Information

Individuals need calorie labeling information because it is difficult to estimate the calories in restaurant meals. A study conducted by the Center for Science in the Public Interest and New York University found that even well-trained nutrition professionals couldn't estimate the calorie content of typical restaurant meals. They consistently underestimated calorie amounts and the underestimates were substantial – by 200 to 600 calories. For example, when shown a display of a typical dinner, hamburger and onion rings, the dietitians estimated that it had 865 calories, when it actually contained about 1,500 calories. If trained nutrition professionals can't estimate the calories in restaurant meals, the average consumer doesn't stand a chance.

With approximately half of the food dollar now being spent away from home (almost doubling since 1970), it is appropriate to make caloric information visible on menus, especially in establishments where foods are typically higher in fat, calories, and larger portion sizes prevail (Finkelstein et al, 2004). This is critically important for our youth, as one study found that children who ate fast food obtained 29 - 38 percent of their total energy intake from that source and ate more total fat, more saturated fat, more total carbohydrate, more added sugars, more sweetened beverages, less fluid milk, and fewer fruits and non-starchy vegetables than those who did not. The same study estimated that on a typical day nearly one third of children in the U.S. eat fast food (adolescents visit a fast-food outlet twice per week on average) and that these extra calories pack on an extra six pounds per child per year. Parents especially deserve to have more easily viewed caloric information to compare menu items and inform their family food purchases outside the home.

The current voluntary provision of nutrition information by many restaurants, although inconsistently offered, does show that providing food composition data is feasible, practical, affordable, and wanted by the consumer. Although having the information on a website or somewhere behind the counter is a good start, it is inadequate. Consumers should be able to at

least see the information when ordering their food and drinks. A patron should not be expected to request the information or go searching to view the caloric content of the food somewhere else like a poster on the wall with tiny print.

The National Academies' Institute of Medicine recommends that restaurant chains "provide calorie content and other key nutrition information on menus and packaging that is prominently visible at point of choice and use" (2006). The Food and Drug Administration, Surgeon General, U.S. Department of Health and Human Services, and the 2007 President's Cancer Panel also recommend providing point of purchase nutrition information at restaurants as a strategy to reduce caloric intake and help combat the worsening obesity crisis.

The American Cancer Society supports calorie labeling as part of a comprehensive approach to addressing obesity, and we believe it is likely to promote reductions in obesity and cancer. Thank you for this opportunity, and your commitment to the fight against cancer.

State of the Science Overview:
Nutrition, Physical Activity and Obesity Link to Cancer

Overweight & Obesity

Overweight and obesity contribute to 14 to 20 percent of all cancer deaths and have been associated with increased risk for several common cancers, including colon, esophagus, kidney, endometrial and breast cancer in postmenopausal women.[1] The biological link between overweight and obesity to cancer is believed to be related to multiple effects on fat and sugar metabolism, immune function, hormone levels and proteins that affect hormone levels, and other factors related to cell proliferation and growth.[2]

Colorectal cancer is the third leading cause of cancer death among both men and women in the U.S.[3] Obese men are more at risk for colon cancer.[4] Obese women who are premenopausal or postmenopausal and are taking hormone therapy have an increased risk of colon cancer, in similar magnitude as obese men.[5] In particular, abdominal body fatness appears to increase the risk of colon cancer.[6] In addition, obese individuals are two times more likely than healthy weight individuals to develop cancer in the lower esophagus and at the junction of the esophagus and stomach.[7] This is likely a result of epithelial damage, metaplasia, and dysplasia associated with gastric reflux.

Breast cancer is the most common cancer diagnosed among American women and is second only to lung cancer as a major cause of cancer deaths among women.[8] There is consistent evidence that increased body weight and weight gain during adulthood are associated with increased risk for breast cancer among postmenopausal women.[9] This increased risk is likely due to the higher levels of estrogens produced by extra adipose tissue after menopause; the adverse effect of weight gain is not seen as readily among women taking postmenopausal hormone therapy, since it may be masked by higher levels of estrogen taken as part of the therapy.[10] Obese, postmenopausal women are also more at risk of dying from breast cancer compared to healthy weight women because obese women have increased levels of estrogen and are more likely to be diagnosed at a later stage.[11]

Obese women have a two to four times great risk of developing type I endometrial cancer, and even overweight women have an increased risk.[12] Type I is low grade and the most common type of endometrial cancer, associated with hormone function. In premenopausal women, the increased risk of endometrial cancer is attributed to insulin resistance, elevation in ovarian androgens, anovulation, and chronic progesterone deficiency associated with being overweight.[13] In postmenopausal women, the increased risk is attributed to higher circulating concentrations of bioavailable estrogens produced by extra adipose tissues.[14]

There is also suggestive evidence that overweight and obesity are associated with increased risk for other cancers, including the pancreas, aggressive prostate, gall bladder, thyroid, ovary, cervix, and for multiple myeloma and Hodgkin's disease.[15] Abnormal glucose metabolism as a result of obesity and physical inactivity appear to be associated with pancreatic cancer.[16] Excessive weight has been associated with a worse prognosis after diagnosis and treatment for prostate cancer.[17] Body fatness may also be associated with cancer of the gall bladder.[18]

Weight loss

The science linking weight loss and cancer risk is currently more limited, but emerging evidence suggests that weight loss may be beneficial. Overweight and obese individuals who intentionally lose

weight have reduced levels of circulating glucose, insulin, and bioavailable estrogens and androgens – all associated with cell and tumor growth. Recent studies exploring intentional weight loss indicate that losing weight may reduce the risk of breast cancer.[19] Surgery to treat morbid obesity and short-term intentional weight loss have been shown to improve insulin sensitivity and biochemical measures of hormone metabolism, which may contribute to the relationship between obesity and cancer.[20]

Nutrition

Poor nutrition can increase an individual's cancer risk through the exclusion of vital nutrients in their diet and as a major contributor to overweight and obesity, which is associated with several cancers.[21] Consumption of calorie-dense foods, foods that are high in calories from added sugars, and diets high in overall calories and fat contribute to overweight and obesity. In addition, consumption of foods and drinks high in added sugars may contribute to insulin resistance, alterations in the amount and distribution of body fat, and increased concentrations of growth factors that may promote the growth of cancer.[22]

Diets rich in certain food groups have been associated with decreased cancer risk. For example, there is extensive scientific evidence establishing that the increased consumption of vegetables and fruits is associated with reduced cancer risk. In particular, greater vegetable and fruit consumption has been associated with decreased risk of lung, esophageal, stomach, and colorectal cancer.[23] Diets rich in non-starchy vegetables, such as green, leafy vegetables or broccoli, appear to reduce the risk of cancer of the mouth, pharynx, larynx, esophagus and stomach.[24] Vegetable and fiber intake may decrease the risk of endometrial cancer, and diets high in certain vegetables (including tomatoes, cruciferous vegetables, soy, beans) may decrease the risk of prostate cancer.[25]

Vegetable and fruit consumption is believed to reduce cancer risk when the body uses certain nutrients from vegetables and fruits to protect against tissue damage that occurs constantly as a result of normal metabolism (oxidation). These nutrients, called antioxidants, interact and stabilize the free radicals from oxidation and may prevent the free radicals from damaging the tissue DNA. Over time, that damage can become irreversible and can lead to disease, such as cancer.[26]

In contrast, consumption of red meats and processed meats can actually increase cancer risk. Consumption of red meats and processed meats are associated with an increased risk of colorectal and prostate cancer.[27] New studies are assessing the association of red meats and processed meats to pancreatic and endometrial cancer. In addition to being major sources of total fat, saturated fat and cholesterol, these meats can contain mutagens and carcinogens, iron, nitrates/nitrites and salt that can increase cancer risk.[28] Certain methods of cooking meats, such as charring, can also produce carcinogens.

The scientific study of nutrition and cancer is highly complex and it is not presently completely understood how single or combined nutrients, or foods or how energy imbalance, affect the risk of specific cancers. In particular, while there does appear to be biological plausibility for the use of supplements containing specific antioxidants to reduce cancer risk, no study thus far has demonstrated this reduced risk.[29] In fact, two studies of supplements of high doses of beta carotene and vitamin A showed an increase in lung cancer risk in smokers, highlighting the importance of more scientific research before any recommendation can be made regarding supplement use.[30]

Physical Activity

Regular and intentional physical activity helps maintain a healthy body weight by balancing caloric intake with energy expenditure. Regardless of actual weight, physical activity has been shown to decrease the risk of colon cancer and breast cancer.[31] Physical activity can be beneficial after diagnosis as well. In one study of patients with early- to late-stage colorectal cancer, physical activity decreased their likelihood of recurrence or death by 40 to 50 percent compared to those patients who were not physically active.[32]

---

[1] Cokkinides V, Bandi P, Siegel R, Ward EM, Thun MJ. *Cancer Prevention & Early Detection Facts and Figures 2009*. Atlanta, GA: American Cancer Society, 2009.

[2] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity and mortality from cancer in a prospectively studied cohort of US adults. N Engl J Med 2003; 348: 1625-1638. Berrington De Gonzalez A, Sweetland S, Spencer E. A meta-analysis of obesity and the risk of pancreatic cancer. Br J Cancer 2003; 89: 519-523. Patel AV, Rodrigez C, Bernstein L, et al. Obesity, recreational physical activity, and the risk of pancreatic cancer in a large US Cohort. Cancer Epidemiol Biomarkers Prev 2005;14: 549-466. Lindblad M, Rodriguez LA, Lagergren J. Body mass, tobacco and alcohol and risk of esophageal, gastric cardia, and gastric non-cardia adenocarcinoma among men and women in a nested case-control study. Cancer Causes Control 2005; 16: 285-294. Amling CL, Riffenburgh RH, Sun L, et al. Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. J Clin Oncol 2004; 22: 439-445. Freedland SJ, Terris MK, Platz EA, Presti JC Jr. Body mass index as a predictor of prostate cancer: development versus detection on biopsy. Urology 2005; 66: 108-113. Amling CL. Relationship between obesity and prostate cancer. Curr Opin Urol 2005; 15: 167-171.

[3] American Cancer Society. *Cancer Facts and Figures 2008*. Atlanta, GA: American Cancer Society; 2008.

[4] Cann BJ, Coates AO, Slattery ML, et al. Body size and the risk of colon cancer in a large case-control study. International Journal of Obesity and Related Metabolic Disorders 1998; 22(2): 178-184. Giacosa A, Franceschi S, La Vecchia C, Favero A, Andreatta R. Energy intake, overweight, physical exercise and colorectal cancer risk. European Journal of Cancer Prevention 1999; 8 Suppl 1: S53-S60. Murphy TK, Calle EE, Rodriguez C, Kahn HS, Thun MJ. Body mass index and colon cancer mortality in a large prospective study. American Journal of Epidemiology 2000; 152(9): 847-854.

[5] Writing Group for the Women's Health Initiative Investigators. Risks and benefits of estrogen plus progestin in healthy postmenopausal women: Principal results from the Women's Health Initiative randomized controlled trial. Journal of the American Medical Association 2002; 288(3): 321-333. Slattery ML, Ballard-Barbash R, Edwards S, Caan BG, Potter JD. Body mass index and colon cancer: An evaluation of the modifying effects of estrogen (United States). Cancer Causes and Control 2003; 14(1): 75-84.

[6] Giacosa A, Franceschi S, La Vecchia C, Favero A, Andreatta R. Energy intake, overweight, physical exercise and colorectal cancer risk. European Journal of Cancer Prevention 1999; 8 Suppl 1: S53-S60. Murphy TK, Calle EE, Rodriguez C, Kahn HS, Thun MJ. Body mass index and colon cancer mortality in a large prospective study. American Journal of Epidemiology 2000; 152(9): 847-854. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[7] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. Brown LM, Swanson CA, Gridley G, et al. Adenocarcinoma of the esophagus: Role of obesity and diet. Journal of the National Cancer Institute 1995; 87(2): 104-109. Chow WH, Blot WJ, Vaughan TL, et al. Body mass index and risk of adenocarcinomas of the esophagus and gastric cardia. Journal of the National Cancer Institute 1998; 90(2): 150-155. Li SD, Mobarhan S. Association between body mass index and adenocarcinoma of the esophagus and gastric cardia. Nutrition Reviews 2000; 58(2 Pt1): 54-56. Lagergren J, Bergstrom R, Nyren O. Association between body mass and adenocarcinoma of the esophagus and gastric cardia. Annals of Internal Medicine 1999; 130(11): 883-890. Ji BT, Chow WH, Yang G, et al. Body mass index and the risk of cancers of the gastric cardia and distal stomach in Shanghai, China. Cancer Epidemiology, Biomarkers and Prevention 1997; 6(7): 481-485. Bianchini F, Kaaks R, Vainio H. Overweight, obesity, and cancer risk. The Lancet Oncology 2002; 3(9): 565-574.

[8] American Cancer Society. *Cancer Facts and Figures 2008*. Atlanta, GA: American Cancer Society; 2008.

[9] Radimer KL, Ballard-Barbash R, Miller JS, et al. Weight change and the risk of late-onset breast cancer in the original Framingham cohort. Nutr Cancer 2004; 49:7-13. Trentham-Dietz A, Newcomb PA, Egan KM, et al. Weight change and risk of postmenopausal breast cancer (United States). Cancer Causes Control 2000; 11: 533-542. Carmichael AR, Bates T. Obesity and breast cancer: a review of the literature. Breast 2004; 13: 85-92. Stephenson GD, Rose DP. Breast cancer and

obesity: an update. Nutr Cancer 2003; 45: 1-16. Swerdlow AJ, De Stavola BL, Floderus B, et al. Risk factors for breast cancer at younge ages in twins: an international population-based study. J Natl Cancer Inst 2002; 94: 1238-1246. van den Brandt PA, Spiegelman D, Yaun SS, et al. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. Am J Epidemiol 2000; 152: 514-527. Morimoto LM, White E, Chen Z, et al. Obesity, body size, and risk of postmenopausal breast cancer: the Women's Health Initiative (United States). Cancer Causes Control 2002; 13: 741-751. Feigelson HS, Patel A, Teras LR, et al. Adult weight gain and histopathologic characteristics of breast cancer among postmenopausal women. Cancer 2006; 107: 12-21. Huang Z, Hankinson SE, Cloditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. Journal of the American Medical Association 1997; 278(17): 1407-1411. Trentham-Dietz A, Newcomb PA, Storer BE, et al. Body size and risk of breast cancer. American Journal of Epidemiology 1997; 145(11): 1011-1019. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[10] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. van den Brandt PA, Spiegelman D, Yuan SS, et al. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. American Journal of Epidemiology 2000; 152(6): 514-527. Huang Z, Hankinson SE, Cloditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. Journal of the American Medical Association 1997; 278(17): 1407-1411. Lahmann PH, Lissner L, Gullberg B, Olsson H, Berglund G. A prospective study of adiposity and postmenopausal breast cancer risk: The Malmo Diet and Cancer Study. International Journal of Cancer 2003; 103(2): 246-252. Yoo KY, Tajima K, Park S, et al. Postmenopausal obesity as a breast cancer risk factor according to estrogen and progesterone receptor status (Japan). Cancer Letters 2001; 167(1): 57-63.

[11] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. Huang Z, Hankinson SE, Cloditz GA, et al. Dual effects of weight and weight gain on breast cancer risk. Journal of the American Medical Association 1997; 278(17): 1407-1411. Tretli S. Height and weight in relation to breast cancer morbidity and mortality. A prospective study of 570,000 women in Norway. International Journal of Cancer 1989; 44(1): 23-30. Petrelli JM, Calle EE, Rodriguez C, Thun MJ. Body mass index, height, and postmenopausal breast cancer mortality in a prospective cohort of U.S. women. Cancer Causes and Control 2002; 13(4): 325-332. Cui Y, Whiteman MK, Flaws JA, et al. Body mass and stage of breast cancer at diagnosis. International Journal of Cancer 2002; 98(2): 279-283.

[12] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002.

[13] Amant F, Moerman P, Neven P, et al. Endometrial cancer. Lancet 2005; 366: 491-505.

[14] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002

[15] Cokkinides V, Bandi P, Siegel R, Ward EM, Thun MJ. *Cancer Prevention & Early Detection Facts and Figures 2008*. Atlanta, GA: American Cancer Society, 2007.

[16] Call EE, Murphy TK, Rodriguez C, et al. Diabetes mellitus and pancreatic cancer mortality in a prospective cohort of United States adults. Cancer Causes Control 1998; 9: 403-410. Michaud DS, Giovannucci E, Willett WC, et al. Physical activity, obesity, height and the risk of pancreatic cancer. JAMA 2001; 286: 921-929.

[17] Amling CL, Riffenburgh RH, Sun L, et al. Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. J Clin Oncol 2004; 22: 439-445. Freedland SJ, Aronson WJ, Kane CJ, et al. Impact of obesity on biochemical control after radical prostatectomy for clinically localized prostate cancer: a report by the Shared Equal Access Regional Cancer Hospital database study group. J Clin Oncol 2004; 22: 446-453. Cerhan JR, Torer JC, Lynch CF, et al. Association of smoking, body mass, and physical activity with risk of prostate cancer in the Iowa 65+ Rural Health Study (United States). Cancer Causes and Control 1997; 8(2): 229-238. Putnam SD, Cerhan JR, Parker AS, et al. Lifestyle and anthropometric risk factors for prostate cancer in a cohort of Iowa men. Annals of Epidemiology 2000; 10(6): 361-369. Irani J, Lefebvre O, Murat F, Dahmani L,, Dore B. Obesity in relation to prostate cancer risk; comparison with a population having benign prostatic hyperplasia. BJU International 2003; 91(6): 482-484.

[18] Lowenfels AB, Maisonneuve P, Boyle P, Zatonski WA, Epidemiology of gallbladder cancer. Hepato-Gastroenterology 1999; 46(27): 1529-1532. Moerman CJ, Bueno-de-Mesquita HB. The epidemiology of gallbladder cancer: Lifestyle-realted risk factors and limited surgical possibilities for prevention. Hepato-Gastroenterology 1999: 46(27): 1533-1539. Hartz AJ, Rupley DC, Rimm AA. The association of girth measurements with disease in 32,856 women. American Journal of Epidemiology 1984; 119*1(: 71-80.

[19] Radimer KL, Ballard-Barbash R, Miller JS, et al. Weight change and the risk of late-onset breast cancer in the original Framingham cohort. Nutr Cancer 2004; 49: 7-13. Eng SM, Gammon MD, Terry MD, et al. Body size changes in relation to postmenopausal breast cancer among women on Long Island, New York. Am J Epidemiol 2005; 162: 229-237. Parker ED, Folsom AR. Intentional weight loss and incidence of obesity-related cancers: the Iowa Women's Health Study. Int J

Obes Relat Metab Disord 2003; 27: 1447-1452. Trentham-Dietz A, Newcomb PA, Egan KM, et al. Weight change and risk of postmenopausal breast cancer (United States). Cancer Causes Control 2000; 11: 533-542. Harvie M, Howell A, Vierkant RA, et al. Assocation of gain and loss of weight before and after menopause with risk of postmenopausal breast cancer in the Iowa women's health study. Cancer Epidemiol Biomarkers Prev 2005; 14: 656-661.

[20] Cokkinides V, Bandi P, Siegel R, Ward EM, Thun MJ. *Cancer Prevention & Early Detection Facts and Figures 2008*. Atlanta, GA: American Cancer Society, 2007.

[21] Cokkinides V, Bandi P, Siegel R, Ward EM, Thun MJ. *Cancer Prevention & Early Detection Facts and Figures 2008*. Atlanta, GA: American Cancer Society, 2007.

[22] Kushi LH, Byers T, Doyle C, Bandera EV, McCullough M, Gansler T, Andrews KS, Thun MJ, 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 Cancer J Clin 2006; 56: 254-281.

[23] Fruits and Vegetables, vol 8. Lyon, France: International Agency for Research on Cancer, World Health Organization; 2003.

[24] World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrtion, Physical Activity, and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007.

[25] Kushi LH, Byers T, Doyle C, Bandera EV, McCullough M, Gansler T, Andrews KS, Thun MJ, 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 Cancer J Clin 2006; 56: 254-281. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[26] NCI. Antioxidants and Cancer Prevention Factsheet. Updated 7/28.04. Downloaded 12/8/08. http://www.cancer.gov/cancertopics/factsheet/antioxidantsprevention

[27] Sandhu MS, White IR, McPherson K. Systematic review of the prospective cohort studies on meat consumption and colorectal cancer risk: a meta-analytical approach. Cancer Epidemiol Biomarkers Prev 2001; 10: 439-446. Norat T, Riboli E. Meat consumption and colorectal cancer: a review of epidemiologic evidence. Nutr Rev 2001; 59: 37-47. Norat T, Lukanova A, Ferrari P, Riboli E. Meat consumption and colorectal cancer risk: dose-response meta-analysis of epidemiological studies. Int J Cancer 2002; 98: 241-256. Chao A, Thun MJ, Connell CJ, et al. Meat consumption and risk of colorectal cancer. JAMA 2005; 293: 172-182. Kolonel LN. Fat, meat, and prostate cancer. Epidemiol Rev 2001; 23: 72-81. Rodriguez C, McCullough ML, Mondul AM, et al. Meat consumption among Black and White men and risk of prostate cancer in the Cancer Prevention Study II Nutrition Cohort. Cancer Epidemiol Biomarkers Prev 2006; 15: 211-216. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[28] Kolonel LN. Fat, meat, and prostate cancer. Epidemiol Rev 2001; 23: 72-81. Cross AJ, Sinha R. Meat-realted mutagens/carcinogens in the etiology of colorectal cancer. Environ Mol Mutagen 2004; 44: 44-55. Gerrior S, Bente L, Hiza H. Nutrient Content of the US Food Supply, 1909-2000 (Home Economics Research Report No. 56). US Department of Agriculture, Center for Nutrition Policy and Promotion; 2004. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[29] NIH Consensus Development Program: State-of-the-Science Conference Statement: Multivitamin/mineral supplements and chronic disease prevention (draft statement). National Institutes of Health; 2006. Available at http://consensus.nih.gov/2006/MVMDRAFT051706.pdf. Morris CD, Carson S. Routine vitamin supplementation to prevent cardiovascular disease: a summary of the evidence for the US Preventive Services Task Force. Ann Intern Med 2003; 139: 56-70.

[30] Omenn GS. Chemoprevention of lung cancer: the rise and demise of beta-carotene. Annua Rev Public Health 1998; 19: 73-99. Albanes D. Beta-carotene and lung cancer: a case study. Am J Clin Cutr 1999; 69: 1345S-1350S. The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. N Engl J Med 1994; 330: 1029-1035. Omenn GS, Goodman GE, Thornquist MD, et al. Effects of a combination of Beta Carotene and vitamin A on lung cancer and cardiovascular disease. N Engl J Med 1996; 335: 1150-1155. Omenn GS, Goodman, G, Thornquist MD, et al. The beta-carotene and retinol efficacy trial (CARET) for chemoprevention of lung cancer in high risk populations: smokers and asbestos-exposed workers. Cancer Res 1994; 54: 2038a-2043s.

[31] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. Patel AV, Rodrigez C, Bernstein L, et al. Obesity, recreational physical activity, and the risk of pancreatic cancer in a large US Cohort. Cancer Epidemiol Biomarkers Prev 2005;14: 549-466. Patel AV, Calle EE, Bernstein L, et al. Recreational physical activity and risk of postmenopausal breast cancer in a large cohort of US women.

Cancer Causes Control 2003; 14: 519-529. Martinez ME, Giovannucci E, Spiegelman D, et al. Leisure-time physical activity, body size, and colon cancer in women. Nurses' Health Study Research Group. J Natl Cancer Inst 1997; 89: 948-955. Slattery ML, Edwards SL, Ma KN, et al. Physical activity and colon cancer: a public health perspective. Ann Epidemiol 1997; 7: 137-145. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.

[32] Vainio H, Bianchini F. Weight control and physical activity, vol. 6. Lyon, France: International Agency for Cancer Research Press; 2002. Martinez ME, Giovannucci E, Spiegelman D, et al. Leisure-time physical activity, body size, and colon cancer in women. Nurses' Health Study Research Group. J Natl Cancer Inst 1997; 89: 948-955. Slattery ML, Edwards SL, Ma KN, et al. Physical activity and colon cancer: a public health perspective. Ann Epidemiol 1997; 7: 137-145. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: AICR, 2007.