

Oncology Nutrition Connection

Oncology Nutrition

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Editor's Note

As we entered 2014, the future of oncology nutrition is looking brighter than ever!

A highlight of this issue is our Continuing Professional Education (CPE) article on Optimizing Outcomes in Oncology Patients: Case studies using Updated Guidelines. This topic was presented at the Houston 2013 Food & Nutrition Conference & Expo™ (FNCE), and generated much excitement and interest.

We are pleased to have our expert and ON DPG member, Colleen Spees, PhD, MEd, RD, LD, answer five fascinating Expert Questions on the emerging topic of Nutrition Genomics. This article brings light and elevates the role of the Registered Dietitians Nutritionists (RDN). This issue also includes an update on Alcohol Consumption and Breast Cancer Risk, a topic that was featured in *Oncology Nutrition Connection (ONC)* back in 2010.

A literature review on the prophylactic use of feeding tubes in head and neck cancer patients was compiled with interesting findings.

You will also be able to discover the in-depth benefits of a cancer-fighting winter vegetable, the cauliflower, featured in our Eat Right to Fight Cancer series.

Hope you enjoy this issue, and please do not hesitate to contact me if you have any questions, ideas or comments regarding upcoming *ONC* issues. Always love to hear from you!

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Alcohol Consumption and Breast Cancer Risk

Sarah Stadtmiller, MS, RD and Suzanne Neubauer, PhD, RD, CNSC

Introduction

Breast cancer is the leading cause of cancer-related mortality in women aged 20–50 years world-wide (1). There are a variety of factors that have been shown to increase a woman's risk of developing the disease. Family history, gender, genetics, null parity, early menarche, and age are well-established risk factors. Other lifestyle-associated risk factors include body weight, physical activity, and smoking habits. One area of interest in current breast cancer research is the role that alcohol consumption plays in the development of the disease. The exact mechanism by which alcohol consumption may increase risk of breast cancer is not known. It has been proposed that alcohol intake increases circulating estrogen levels, possibly promoting the development of hormone-receptor-positive breast cancer. The metabolism of alcohol may also produce reactive oxygen species, causing DNA damage that contributes to breast cancer (2).

Volume of Alcohol Consumption

A number of studies have examined the association between volume of alcohol consumption and breast cancer risk. For most Americans, the current recommendation for moderate alcohol intake is up to one drink a day for women. One drink is typically defined as 12 ounces (oz) of beer, 4 oz of wine, or 1.5 oz of liquor or spirits (3). In a study by McCarty et al. (2), 1,041 cases of breast cancer and 1,070 controls completed a self-administered food frequency questionnaire (FFQ) that assessed their intake of alcohol over the previous 12 months. Subjects who reported drinking three or more servings of alcohol per day had twice the odds of developing breast cancer compared with non-drinkers (Hazard Ratio (HR)=2.00, 95% Confidence Interval (CI), 1.11-3.61). Even those subjects with a modest daily intake of alcohol (0-0.99 servings) had a 31% increased likelihood of breast cancer compared with non-drinkers. Though the case-control study methodology cannot prove cause and effect, this study and others support a connection between alcohol consumption and breast cancer risk.

In the Nurses' Health Study, Chen and colleagues (4) followed 105,986 women, among whom 7,690 cases of breast cancer were documented, between 1980 to 2008. Alcohol intake was assessed at baseline with a FFQ, and then again at eight different points during the study. Alcohol intake was averaged over the study period. There was a significant association between increasing volumes of alcohol consumption and breast cancer risk, and subjects who consumed ≥ 30 grams (g) alcohol/day (≥ 2 drinks/day) had the greatest risk of developing the disease compared with non-drinkers (Relative Risk (RR)=1.51, 95% CI, 1.35-1.70).

Additional studies have found a significant association between alcohol consumption and breast cancer incidence. Ferraroni and colleagues (5) assessed breast cancer risk and alcohol intake in 2,569 female cases and 2,588 female controls in Italy, where higher amounts of alcohol consumption in women are more common than in North America. There was a significant association between increased alcohol consumption and breast cancer risk, with the highest risk found in those subjects who reported

drinking > 27.60 g alcohol/day (~2 drinks/day) (Odds Ratio (OR) =1.41). A study by Levi and colleagues (6) included 230 cases and 507 controls in Switzerland. There was a significant association between breast cancer risk and number of alcoholic beverages consumed per day, with > 4 drinks/day presenting the greatest odds of developing breast cancer (OR=2.7). However, there were only 18 cases and 15 controls who reported drinking that amount daily, resulting in a wide confidence interval (95% CI, 1.3-5.8). Similarly, Li et al. (7) found a significant association between breast cancer risk and increased alcohol consumption as compared with nondrinkers in their study of 2,829 breast cancer cases. Also, when stratified by hormone receptor status, women with estrogen receptor positive tumors had a greater risk of breast cancer compared to non-drinkers when drinking 1-2 drinks/day and ≥ 3 drinks/day (RR=1.4 and 1.7, respectively). No relationship was found between breast cancer risk and alcohol consumption in women with estrogen receptor negative tumors. This is important to note, since it is proposed that alcohol may increase the levels of circulating estrogen in the body.

Not all reviewed studies found a relationship between volume of alcohol consumed and breast cancer risk. Terry and colleagues (8) evaluated this relationship in 1,508 cases and 1,556 controls through in-person interviews. When comparing lifetime alcohol intake and adjusting for current alcohol intake, there was no association between increasing alcohol intake and breast cancer risk. However, family history of breast cancer was not controlled for and this may have impacted the results. Bessaoud and Daurés (9) studied 437 breast cancer cases and 922 matched controls in Southern France. There was no dose-response relationship between alcohol consumption and breast cancer risk, though individuals who drank between 10 and 15 g of alcohol/day had a significantly lower risk than non-drinkers (OR=0.21, 95% CI, 0.10-0.91). These results may indicate a possible threshold effect, below which

breast cancer risk decreases or remains unchanged. However, this study also lacked the statistical power to examine moderate to heavy drinking, as those levels were rare in the study population.

Results of the majority of these studies suggest a relationship between breast cancer risk and alcohol volume, even at levels as low as 3 to 6 drinks/week. However, one limiting factor in each study was the use of structured questionnaires to collect data. Alcohol consumption is not always reported truthfully. The two studies that did not find a dose-response relationship used in-person interviews, which may have further influenced the subjects' truthfulness when questioned about their drinking habits.

Drinking Pattern

Frequency: In the previously mentioned studies, volume of alcohol intake was determined by averaging subjects' reported consumption over the course of the week. However, this does not take different drinking patterns into consideration. A number of studies have assessed the effects that these patterns have on breast cancer risk. Gao et al. (10) investigated the frequency of drinking in 1,351 Chinese women (669 cases) using an in-person interview. Alcohol drinkers were defined as subjects who reported drinking alcoholic beverages at least once a week for ≥ 6 months. There was a significant increase in breast cancer risk in current/ever drinkers compared with non-drinkers (OR 1.86, 95% CI, 1.02-3.39). However, the aforementioned study by Bessaoud and Daurés (9) found no association between breast cancer risk and subjects categorized as never, sporadic, or frequent drinkers.

In a larger study, Horn-Ross et al. (11) assessed the relationship between daily versus sporadic alcohol consumption and breast cancer risk in 103,460 cohort members (1,742 cases) from the California Teachers Study (CTS). Sporadic drinkers were classified as those who consumed alcohol ≤ 4 days/week, while daily drinkers

were classified as those who drank ≥ 5 days/week. Looking only at post-menopausal women (sample size (n) =819), daily heavy drinkers (≥ 20 g alcohol/day) had a significantly greater risk of developing breast cancer compared with nondrinkers (RR=1.34, 95% CI, 1.07-1.67). However, there was no relationship found between daily moderate drinking (≤ 20 g alcohol/day) or moderate sporadic drinking and breast cancer risk. There were insufficient data to determine if heavy sporadic drinkers had an increased or decreased risk of developing breast cancer. Moderate and heavy sporadic drinking was not explicitly defined in this study.

Chen and colleagues (4) reported the number of days study participants consumed alcohol in a typical week. Subjects who drank alcohol 5-7 days per week had a 20% greater risk of developing breast cancer compared with subjects who did not drink (RR=1.20, 95% CI, 1.11-1.30). However, when the authors controlled for subjects' cumulative alcohol intake over the course of the study, no relationship was found.

Binge Drinking: A number of studies also assessed whether heavy binge drinking plays a role in breast cancer risk. Mørch and colleagues (12) followed 17,647 nurses enrolled in the Danish Nurse Cohort Study from 1993-2001, 457 of whom were diagnosed with breast cancer during follow-up. A mailed questionnaire was used to assess subjects' drinking patterns on the latest weekday and previous weekend (Friday through Saturday). In this study, binge drinking was defined as consuming ≥ 4 alcoholic beverages a day. Subjects who drank 10-15 alcoholic drinks on a weekend had a 49% increased risk of developing breast cancer compared with subjects who consumed only 1-3 drinks (RR=1.49, 95% CI, 1.04-2.13). Similarly, subjects who drank 4-5 drinks on one weekday had a 55% increased risk of developing breast cancer compared with subjects who consumed only one drink.

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Chen et al. (4) asked subjects to report the number of drinks they consumed in one day in a typical month. There was a positive association between the largest number of alcoholic drinks consumed in one day and risk of breast cancer ($P < 0.001$). Subjects who binge drank (defined here as ≥ 6 drinks in one day) had a 33% greater risk of developing breast cancer compared with non-drinkers (RR=1.33, 95% CI, 1.11-1.59). When cumulative alcohol consumption over the course of the study was controlled for, the association between binge drinking and breast cancer risk remained statistically significant ($P = 0.04$).

Based on the findings from these studies, the evidence suggesting a relationship between breast cancer risk and pattern of alcohol consumption is mixed. Frequency did not appear to play a definitive role in the risk of breast cancer development, though both studies analyzing binge drinking showed it to be significantly associated with risk. This suggests that average volume of intake at one time may be a bigger risk factor than drinking pattern.

Duration of Consumption

Several studies have investigated whether the duration of alcohol consumption affected the risk of breast cancer development. Levi and colleagues (6) categorized subjects into one of three groups (< 20 years, 20-29 years, and ≥ 30 years). When controlling for age at starting alcohol use, there was no relationship found between breast cancer risk and duration of consumption. Ferraroni et al. (5) also found no association between increased breast cancer risk and increased duration of alcohol consumption in the study participants.

Bowlin and colleagues (13) also looked at the duration of alcohol consumption in 1,214 cases and an equal number of controls in Long Island, NY from 1984-1986. Those who reported drinking for between 20 and 40 years, regardless of menopause status, had a significantly greater risk of developing breast cancer compared with

nondrinkers (OR=1.48, 95% CI, 1.13-1.93). However, there was no relationship between breast cancer risk and alcohol consumption for subjects who reported drinking for < 20 years or > 40 years.

There is limited evidence to support a relationship between duration of alcohol consumption and breast cancer risk.

Type of Alcoholic Beverage

The results of the previous studies have been based on generalized alcohol consumption, without regard to the specific type of beverage consumed. To determine if the type of alcohol impacted breast cancer risk, a number of studies looked at beer, wine, and spirits/liquor independently. Zhang and colleagues (14) investigated whether the type of alcoholic beverage consumed by an individual affected breast cancer risk in 38,454 women (1,484 cases) enrolled in the Women's Healthy Study over the course of 10 years. While women who reported drinking beer had a slightly higher risk of developing breast cancer compared with nondrinkers (RR=1.14, 95% CI, 1.02-1.28), women who consumed red wine, white wine, and liquor were not at increased risk of developing breast cancer compared with nondrinkers. Similarly, a meta-analysis of 13 studies assessing the relationship between alcohol type and breast cancer risk found that, after controlling for the amount of alcohol consumed, risk did not differ among women consuming beer, wine or spirits when compared to non-drinkers of the same beverage (15). Bowlin et al. (13) also found the likelihood of developing breast cancer did not differ among women who reported consuming beer, wine, or liquor in their case-control study of 1,214 breast cancer cases ($n = 2,428$). There was, however, a significant increase in the odds of developing breast cancer for those subjects who reported drinking a combination of all three alcohol types (OR=1.56, 95% CI, 1.19-2.04).

Bissonauth and colleagues (16) conducted a case-control study of French-Canadian women ($n = 560$; 280 cases) who did not test

positive for the BRCA (Breast Cancer), a genetic mutation that puts men and women at increased risk for breast cancer and women at increased risk of ovarian cancer. Based on their responses to an interviewer-administered FFQ, subjects were placed into tertiles based on alcohol consumption. Breast cancer odds increased significantly in the highest tertile (≥ 2 drinks/week) in subjects who reported drinking beer, wine, or spirits (OR=1.34, 1.16, and 1.09, respectively) compared with the lowest tertiles of 0 bottles of beer/week, ≤ 5 oz wine/week, and ≤ 3 oz spirits/week.

In the study by Li and colleagues (7), wine, liquor, and beer were not found to be independently associated with breast cancer risk. When subdivided into categories, there was no relationship between type of wine and risk. Other studies also found that beer, liquor, or wine had no independent effect on breast cancer risk in subjects who consumed alcohol (17-19). Bessaoud and Daurés (9) added the category of aperitifs to their study, and also found no relationship between the four types of alcohol and breast cancer risk.

Additional studies focused on wine as the primary alcohol source. Allen et al. (20) compared the breast cancer risk of women who drank wine only with those who drank other alcoholic beverages (only beer and/or spirits, or a mixture of alcoholic beverages). Relative risks were calculated per 10 g/d increase in intake, and there were no differences found between the two groups. Additional studies found no difference in the type of wine consumed (red, white, and/or rose) and breast cancer risk (21, 22).

Evidence does not support one type of alcoholic beverage being more protective to breast cancer risk. Any type of alcoholic beverage appears to increase breast cancer risk.

Additional Factors that May Influence Risk

The following studies examined the potential effects of menopausal status and family history of breast cancer on the

relationship between alcohol consumption and breast cancer risk.

Menopausal Status: Colditz and Rosner (23) assessed whether menopausal status influenced the association between breast cancer risk and alcohol consumption in a large cohort study over the course of 14 years (n=58,520; 1,761 cases). A significant risk was noted in pre-menopausal drinkers. Women in this category who reported drinking 12 g alcohol/day had a 7% increased risk of developing breast cancer compared with nondrinkers. Ferraroni and colleagues (5) reported similar findings. There was a positive and significant dose-response relationship noted in pre-menopausal women who consumed alcohol (P=0.0002). No such association was found in post-menopausal women. However, other studies found no differences between pre- and post-menopausal women in terms of alcohol intake and breast cancer risk (8, 15, 24), indicating that additional studies are needed to clarify the impact, if any, of menopausal status on risk of breast cancer among regular alcohol consumers.

Family History: A family history of breast cancer is one of the most important risk factors for breast cancer (1). To determine if family history modified the association between alcohol intake and breast cancer risk, Suzuki and colleagues (25) assessed 51,847 women (1,188 cases) in the Swedish Mammography Cohort over the course of 10 years. Subjects self-reported data on alcohol consumption and family history of breast cancer. Results showed no interaction of alcohol intake and family history on breast cancer risk.

Berkey et al. (26) looked at a younger group of subjects (n=6,741; 67 cases). Girls aged 9-15 years, all of whom were daughters of Nurses' Health Study II participants, were included at the start of the cohort study in 1996. The girls were followed until 2007, and reported their alcohol consumption at three different points throughout the study. Subjects who had a family history of breast cancer, and who were also in the highest quartile of alcohol consumption for their

age, had a significantly greater risk of benign breast disease (BBD), compared to subjects with no family history and who abstained from drinking (OR=2.27). However, it is unknown whether the instances of BBD in this study were proliferative or non-proliferative BBD.

Levi and colleagues (6) found that subjects with a family history of breast cancer were not at an increased or decreased risk of developing breast cancer when considering alcohol intake. However, subjects without a family history of breast cancer who reported drinking ≥ 1 drink/day had a significantly higher risk of breast cancer compared to non-drinkers (OR=1.9, 95% CI, 1.3-2.9). However, these results may have been due to the small number of subjects with a family history of breast cancer, compared with women with no family history (n=22 and 208, respectively).

It remains unclear whether menopause status or family history plays a role in the association between alcohol and breast cancer risk, as the studies were mixed in their results.

Alcohol and Breast Cancer Recurrence

While many women in the general population may give little thought to how alcohol consumption affects breast cancer risk, breast cancer survivors often are keenly interested in this possible risk factor and its relationship to cancer recurrence and survivorship. A study by Kwan et al. (27) examined the association between alcohol consumption and recurrence and mortality in 1,897 early-stage breast cancer survivors. Women who consumed ≥ 6 g of alcohol per day had an increased risk of breast cancer recurrence and death from breast cancer compared with nondrinkers (HR=1.35 and 1.51, respectively). A meta-analysis of 25 cohort studies found an association between consumption of > 20 g alcohol/day and increased risk of breast cancer mortality, though there was no association with increased breast cancer recurrence (28). Harris and colleagues (29) found no association between alcohol intake and

breast cancer-specific mortality in their study of 3,146 women diagnosed with invasive breast cancer. They did however find that women who consumed 3.4-9.9 g alcohol/day had a lower risk of non-breast cancer-related death compared with nondrinkers (HR=0.67, 95% CI, 0.50-0.90). It may therefore be important for breast cancer survivors to weigh the potential risks of drinking and breast cancer recurrence against the potential benefits of low to moderate consumption, especially in terms of cardiovascular health.

Conclusion

The studies presented here support a larger body of research investigating the relationship between breast cancer and alcohol intake. Consumption of alcohol, even at low to moderate levels, has been shown to increase risk regardless of beverage type (30). This is consistent with the 2007 expert report by the American Institute for Cancer Research (AICR), which indicated that alcohol in any amount increases breast cancer risk (31). This suggests that it is ethanol itself that contributes to risk, and there is strong evidence to support a dose-response relationship between increased alcohol consumption and risk of breast cancer development. Risk may also increase with episodic binge drinking, though duration of consumption and frequency were not conclusively associated with risk. There is also limited evidence to suggest that risk differs for pre- or post-menopausal women, or those with a family history of breast cancer.

Despite these findings, other factors must be taken into consideration when determining the most appropriate recommendation. For example, heart disease is the leading cause of death in women, and modest alcohol consumption has been associated with a reduced risk of heart disease (32). Also, recent evidence has shown that moderate alcohol consumption pre- and post-diagnosis may be associated with better all-cause survival for women diagnosed with breast cancer (33).

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Based on the studies reviewed, a recommendation of < 1 drink/day should be encouraged to minimize the risk of breast cancer development in women. However, it is important for each individual to consider their medical and family history and weigh the potential risks and benefits associated with drinking alcohol. To prevent overconsumption, information surrounding proper portion sizes of beer, liquor, and wine should be included in nutrition education. Finally, Registered Dietitians should encourage women to practice proper screening techniques such as self-breast exams and mammograms to ensure early detection of breast cancer if it does indeed develop. Taking these steps may help to decrease a woman's risk of breast cancer development.

References

- Walker MJ, Chiarelli AM, Knight JA, Mirea L, Glendon G, Ritvo P. Perceived risk and adherence to breast cancer screening guidelines among women with a familial history of breast cancer: A review of the literature. *Breast J*. 2013;15:1-10.
- McCarty CA, Reding DJ, Commins J, Williams C, Yeager M, Burmester JK, Schairer C, Ziegler RG. Alcohol, genetics and risk of breast cancer in the prostate, lung, colorectal and ovarian cancer (PLCO) screening trial. *Breast Cancer Res Treat*. 2012;133:785-792.
- "Frequently asked questions." *CDC.gov*. Division of Population Health, National Center for Chronic Disease Prevention and Health Promotion, Nov. 2012. Web. 12 June 2013. < <http://www.cdc.gov/alcohol/faqs.htm>>.
- Chen WY, Rosner B, Hankinson SE, Colditz GA, Willett WC. Moderate alcohol consumption during adult life, drinking patterns, and breast cancer risk. *J Am Med Assoc*. 2011;306:1884-1890.
- Ferraroni M, Decarli A, Franceschi S, La Vecchia C. Alcohol consumption and risk of breast cancer: A multicenter Italian case-control study. *Eur J Cancer*. 1998;34:1403-1409.
- Levi F, Pasche C, Lucchini F, La Vecchia C. Alcohol and breast cancer in the Swiss Canton of Vaud. *Eur J Cancer*. 1996;32:2108-2113.
- Li Y, Baer D, Friedman GD, Udaltsova N, Shim V, Klatsky AL. Wine, liquor, beer, and risk of breast cancer in a large population. *Eur J Cancer*. 2009;45:843-850.
- Terry MB, Zhang FF, Kabat G, Britton JA, Teitelbaum SL, Neugut AI, Gammon MD. Lifetime alcohol intake and breast cancer risk. *Ann Epidemiol*. 2006;16:230-240.
- Bessaoud F, Daurés JP. Patterns of alcohol (especially wine) consumption and breast cancer risk: A case-control study among a population in Southern France. *Ann Epidemiol*. 2008;18:467-475.
- Gao CM, Ding JH, Li SP, Liu YT, Qian Y, Chang J, Tang JH, Tajima K. Active and passive smoking, and alcohol drinking and breast cancer risk in Chinese women. *Asian Pac J Cancer Prev*. 2013;14:993-996.
- Horn-Ross PL, Canchola AJ, West DW, Stewart SL, Bernstein L, Deapen D, Pinder R, Ross RK, Anton-Culver H, Peel D, Ziogas A, Reynolds P, Wright W. Patterns of alcohol consumption and breast cancer risk in the California Teachers Study cohort. *Cancer Epidemiol Biomarkers Prev*. 2004;13:405-411.
- Mørch LS, Johansen D, Thygesen LC, Tjønneland A, Løkkegaard E, Stahlberg C, Grønbaek M. Alcohol drinking, consumption patterns and breast cancer among Danish nurses: A cohort study. *Eur J Public Health*. 2007;17:624-629.
- Bowlin SJ, Leske MC, Varma A, Nasca P, Weinstein A, Caplan L. Breast cancer risk and alcohol consumption: results from a large case-control study. *Int J Epidemiol*. 1997;26:915-923.
- Zhang SM, Lee I, Manson JE, Cook NR, Willett WC, Buring JE. Alcohol consumption and breast cancer risk in the Women's Health Study. *Am J Epidemiol*. 2007;165:667-676.
- Ellison RC, Zhang Y, McLennan CE, Rothman KJ. Exploring the relation of alcohol consumption to risk of breast cancer. *Am J Epidemiol*. 2001;154:740-747.
- Bissonauth V, Shatenstein B, Fafard E, Maugard C, Robidoux A, Narod S, Ghadirian P. Risk of breast cancer among French-Canadian women, noncarriers of more frequent BRCA1/2 mutations and consumption of total energy, coffee, and alcohol. *Breast J*. 2009;15:63-71.
- Hiatt RA, Klatsky AL, Armstrong MA. Alcohol consumption and risk of breast cancer in a prepaid health plan. *Cancer Res*. 1988;48:2284-2287.
- Smith SJ, Deacon JM, Chilvers CED. Alcohol, smoking, passive smoking, and caffeine in relationship to breast cancer risk in young women. *Br J Cancer*. 1994;70:112-119.
- Adami HO, Lund E, Bergström R, Meirik O. Cigarette smoking, alcohol consumption and risk of breast cancer in young women. *Br J Cancer*. 1988;58:832-837.
- Allen NE, Beral V, Casabonne D, Kan SW, Reeves GK, Brown A, Green J. Moderate alcohol intake and cancer incidence in women. *J Natl Cancer Inst*. 2009;101:296-305.
- Hirvonen T, Mennen LI, De Bree A, Castetbon K, Galan P, Bertrais S, Arnault N, Hercberg S. Consumption of antioxidant-rich beverages and risk for breast cancer in French women. *Ann Epidemiol*. 2006;16:503-508.
- Newcomb PA, Nichols HB, Beasley JM, Egan K, Titus-Ernstoff L, Hampton JM, Trentham-Dietz A. No difference between red wine or white wine consumption and breast cancer risk. *Cancer Epidemiol Biomarkers Prev*. 2009;18:1007-1010.
- Colditz GA, Rosner B. Cumulative risk of breast cancer to age 70 years according to risk factor status: data from the Nurses' Health Study. *Am J Epidemiol*. 2000;152:950-964.
- Beasley JM. Coronado GD, Livaudais J, Angeles-Llerenas A, Ortega-Olvera C, Romieu I, Lazcano-Ponce E, Torres-Mejia G. Alcohol and risk of breast cancer in Mexican women. *Cancer Causes Control*. 2010;21:863-870.
- Suzuki R, Ye W, Rylander-Rudqvist T, Saji S, Colditz GA, Wolk A. Alcohol and postmenopausal breast cancer risk defined by estrogen and progesterone receptor status: A prospective cohort study. *J Natl Cancer Inst*. 2005;97:1601-1608.
- Berkey CS, Tamimi RM, Rosner B, Frazier AL, Colditz GA. Young women with family history of breast cancer and their risk factors for benign breast disease. *Cancer*. 2012;118:2796-803.
- Kwan ML, Kushi LH, Weltzien E, Tam EK, Castillo A, Sweeney C, Caan BJ. Alcohol consumption and breast cancer recurrence and survival among women with early-stage breast cancer: the life after cancer epidemiology study. *J Clin Oncol*. 2010;28:4410-6.
- Gou YJ, Xie DX, Yang KH, Liu YL, Zhang JH, Li B, He XD. Alcohol Consumption and Breast Cancer Survival: A Meta-analysis of Cohort Studies. *Asian Pac J Cancer Prev*. 2013;14:4785-90.
- Harris HR, Bergkvist L, Wolk A. Alcohol intake and mortality among women with invasive breast cancer. *Br J Cancer*. 2012;106:592-5.
- Scocciati C, Lauby-Secretan B, Bello PY, Chajes V, Romieu I. Female breast cancer and alcohol consumption: a review of the literature. *Am J Prev Med*. 2014;46(3 Suppl 1):S16-25.
- 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.
- McDonald JA, Goyal A, Terry MB. Alcohol intake and breast cancer risk: weighing the overall evidence. *Curr Breast Cancer Rep*. 2015;3(3).
- Newcomb PA, Kampman E, Trentham-Dietz A, Egan KM, Titus LJ, Baron JA, Hampton JM, Passarelli MN, Willett WC. Alcohol consumption before and after breast cancer diagnosis: associations with survival from breast cancer, cardiovascular disease, and other causes. *J Clin Oncol*. 2013;31:1939-46.

New Oncology Nutrition Education Tools

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Past Chair of ON DPG

The ON DPG strives to offer value to its members, and has been working hard to meet this goal through its online presence at www.oncologynutrition.org. ON DPG has recently partnered with Abbott Nutrition to offer three great webinars created and presented by talented ON DPG members (Cyndi Thomson, PhD, RD, CSO, Jeannine Mills, MS, RD, CSO, LD and Rhone Levine, MEEd, RD, CSO, LD) on topics such as energy balance and cancer survivorship, tube feeding basics, and appropriate use of oral nutrition supplements for optimal outcomes.

ON DPG is now pleased to offer new oncology nutrition education tools designed to augment these three webinars.

The tools can be viewed at:

- *Energy Balance for Healthy Survivorship After Cancer*
http://dpg-storage.s3.amazonaws.com/ondpg/documents/8ba83a8a31dcd9c/Energy_Balance.pdf
- *Tube Feeding Basics*
http://dpg-storage.s3.amazonaws.com/ondpg/documents/6872d46082a7b18c/Tube_Feeding_basics.pdf
- *Appropriate Use of Oral Nutrition Supplements to Support Optimal Patient Outcomes*
http://dpg-storage.s3.amazonaws.com/ondpg/documents/76f7c0eff5c4bb1c/What_is_ONS.pdf

ON DPG continues to add resources to its website to help support and enhance the oncology nutrition practice of its members. Please be sure to check these out at <http://www.oncologynutrition.org/erfc/>.

Eat Right to Fight Cancer: Cauliflower

Maureen Leser, MS, RD, CSO, LD

Introduction

Cauliflower was a staple of diets in geographic areas near the Mediterranean Sea (now Iraq, Iran, and Cyprus) for centuries before being introduced to France around 1650 (1). Both Louis XIV and Louis XV considered cauliflower a delicacy, and chefs of the time added it to stews and served it with rich sauces. Today, cauliflower has worldwide appeal and the U.S. is a major producer. California is the center of U.S. cauliflower production (1-2).

Botanically, cauliflower is one of several cruciferous vegetables that are members of the family Brassicaceae. Only a few of the 3000 species of this family are edible; those include cauliflower, broccoli, cabbage, watercress, and Brussels sprouts (1-2). The petals of plants within this family have a cruciform arrangement, and they were originally referred to as “Criciferae or Cruciferae” vegetables before the moniker “Cruciferous” was adopted.

Cauliflower may lack the vivid colors considered a marker of the most healthful vegetables and fruits, but it is packed with nutrients, dietary fiber, and other bioactive food compounds, and has unique cancer-preventive benefits. Table 1 summarizes the macronutrient and micronutrient content of this nutrient-rich vegetable.

Cruciferous vegetables including cauliflower also are sources of other bioactive compounds and numerous phytochemicals (4), such as:

- Carotenoids
- Chlorophyll
- Fiber
- Flavonoids
- Indole-3-Carbinol
- Isothiocyanates
- Lignans
- Phytosterols

A Cancer-fighting Star

Cancer pathology involves abnormal and deregulated biological checkpoints, pathways, and processes that allow a cancer

to grow. For a cancer to grow, cells must proliferate at an unregulated pace; abnormal cells must evade apoptosis (cell death); and angiogenesis (formation of new blood vessels) must occur (5). Cancer prevention recommendations reduce exposure to carcinogens, inhibit cancer-promoting biological reactions, and favor cancer-prevention reactions.

Epidemiologic research supports an inverse relationship between several cancers and intake of plant foods. Research examining the associations between cancer risk and cruciferous vegetable intake is mixed (6). In recent decades, researchers have found that glucosinolates, naturally found in cruciferous vegetables, are converted to several isothiocyanates (ITCs), which strengthen cancer-preventive reactions (7-10). ITCs include phenethyl isothiocyanate (PEITC), benzyl isothiocyanate (BITC), and l-sulforaphane (d,l-sulforaphane (SFN) (10).

Research suggests that PEITC, BITC and SFN inhibit angiogenesis and promote apoptosis. Researchers are particularly excited about the potential ability of ITCs to selectively cause apoptosis in cancer cells while sparing healthy cells. The scientific explanation of these benefits is highly complex and researchers continue to work on unraveling its mechanisms. One area of exploration is the ability of PEITC to reduce the expression of vascular endothelial growth factor, a substance that activates genes that allow a cell to adapt to a low oxygen environment and promote angiogenesis (10). Inhibiting

(Continued on next page)

these processes limits angiogenesis and slows tumor development and growth.

Humans have an inborn ability to detoxify potentially harmful compounds in the environment, including ones that may be carried via medicines and food. Detoxification occurs in two main steps, commonly described as Phase I and Phase II reactions. Phase I reactions, which have been described as functionalization reactions, add a reactive site to a compound while Phase II detoxification reactions, also called conjugation reactions, convert compounds to forms that can be easily eliminated (7). Together, Phase I and Phase II reactions can transform a lipophilic compound to one that is water-soluble so the offending compound can be excreted in urine. ITCs induce enzymes important to Phase II reactions, thus helping the body remove harmful and potentially carcinogenic compounds (7-8).

A number of animal studies have demonstrated that ITCs help prevent chemically-induced cancers (4). An additional avenue of research is exploring the ability of ITCs to sensitize cancer cells to chemotherapy, with the potential to improve its efficacy (4). Research examining anti-cancer effects of ITCs has not yet moved to humans; however, regular intake of cruciferous vegetables increases levels of biomarkers associated with cancer prevention.

In cruciferous vegetables, ITCs are stored as glucosinolates, the sulfur-containing compounds that give these vegetables their pungent aroma and spicy flavor. Cutting and chewing these vegetables releases an enzyme (myrosinase) that converts glucosinolates to ITCs (10). Glucosinolate content of cruciferous vegetables ranges from 0.5 to 3 mg/gram; a 1-cup serving (approximately 100 grams) potentially provides a significant amount of glucosinolate (10). Plant genetics and the thioglucosidase activity of human intestinal microbial flora influence the exposure of the gastrointestinal tract to isothiocyanates and indoles, and post-harvest storage

Table 1. Energy, Macronutrient and Micronutrient Analysis of Cauliflower (3)

Nutrient	Nutrient Content in 1 cup, chopped (107 grams)	Daily Value (DV) per Nutrient	Percent Daily Value* Per 1 cup chopped Cauliflower
Energy	27	2000	1%
Protein	2	(see RDAs)	n/a
Fat	0.3	65 g	0%
Carbohydrate	5.3	(see RDAs)	n/a
Dietary Fiber	2.1	25 g	8%
Calcium	24 mg	1,000	2%
Iron	0.45 mg	18 mg	2.5%
Magnesium	16	400 mg	4%
Phosphorus	47	1,000	5%
Potassium	320	3500 mg	9%
Zinc	0.27	15 mg	2%
Folate	57	400 ug	14%
Vitamin C	48.2	60 mg	80%
Vitamin K	15.5 mcg	80 mcg	19%

* Foods providing 20% of more of the DV are considered to be high sources of a nutrient. Foods providing lower percentages of the DV also contribute to a healthful diet.

environment and cooking methods influence isothiocyanate content in food (1).

The European Prospective Investigation into Cancer and Nutrition (EPIC) Study followed cancer incidence in 521,000 subjects throughout Europe and has provided valuable information on the influence of diet on various cancer types. Data from this study also has provided information on cruciferous vegetable intake in Europe, reporting a mean intake of approximately 12 grams per day in Spain and 34.4 grams per day in the United Kingdom (1 cup of cauliflower = approximately 100 grams), most commonly in cooked form (1,11). In addition, mean consumption by male and female participants, respectively, was 20.8 grams/day and 21.4 grams/day, and cauliflower represented 25% of all cruciferous vegetables consumed. There is no specific recommendation for cruciferous vegetable intake, though cauliflower fits within the "other" vegetable group on My Plate, a nutrition education program that encourages an intake of 6 ½ to 7 cups per day of "other" vegetables (12). The Linus Pauling Institute recommends eating cruciferous vegetables five times per week (2).

Purchasing and storing cauliflower

The highest quality cauliflowers have a clean, creamy white, compact head (also called the curd) in which the bud clusters are not separated. Spotted or dull-colored cauliflower should be avoided, as well as those in which small flowers appear. Heads surrounded by many thick green leaves are better protected and will be fresher (12-14).

Cauliflower can remain fresh when stored up to a week in a paper or plastic bag in the refrigerator. To prevent moisture from developing in the floret clusters, store it with the stem side down. Pre-cut cauliflower florets rarely remain fresh longer than a few days. Cooking causes cauliflower to spoil more quickly, so cooked cauliflower should be consumed within two to three days of being stored in the refrigerator (13-14).

Incorporating Cauliflower in the Diet

Cauliflower can be eaten raw or cooked; can be incorporated in casseroles and stews; can be blended with other vegetables in purees; or can be roasted or steamed.

Following are a few additional cooking ideas:

- Serve as a crudité with a dip
- Steam, then puree or mash



- Steam, puree, and blend with other ingredients into a soup
- Add to chili, curries, omelets, and vegetable pies
- Roast with spices such as cumin
- Steamed and combined with pasta, tofu, and whole grains such as quinoa
- Incorporate in salads (either raw, steamed, or roasted)
- Slice, cook, and serve as a “steak”
- Use as the main ingredient in carbohydrate-free “risotto” or fried “rice”

Growing Cauliflower

Cruciferous vegetables are commonly grown in Australia, New Zealand, Eastern Europe, Latin America, the U.S., Canada, East and South-East Asia with smaller amounts grown in sub-Saharan Africa and the Near East. They are also becoming popular additions to home gardens.

Cauliflower is considered a cool-weather vegetable and among the fresh vegetables sold in supermarkets in winter months. Growing temperatures must remain consistently in the 60s; hot weather will produce multiple small button-size heads rather than one large head (13-14). To grow in a home garden:

- Plant in the spring and fall in a site with six or more hours of full sun
- Soil should be rich in organic matter with a pH between 6.5 and 6.8.
- Plant outside shortly before the average frost date in the spring and about six to eight weeks before the first fall frost (and when the temperature is below 75 degrees F). To harvest in the fall,

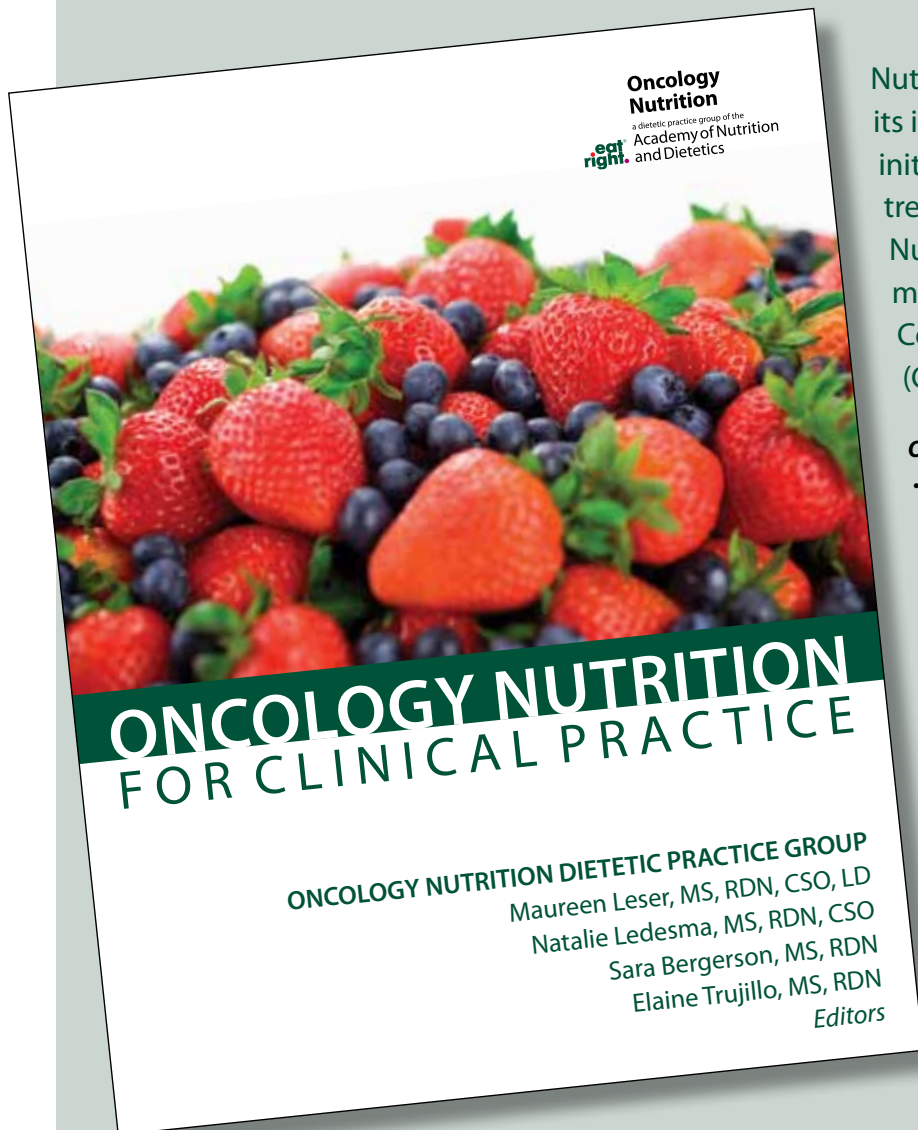
cauliflower should be planted mid-summer. Gardeners may need to cover plants in early spring (if there is an unexpected frost) and shade them in the fall (if an unexpected heat wave occurs). Many State Extension Services publish recommended dates for planting vegetables (including cauliflower) in home gardens.

- Home gardeners usually plant transplants rather than seeds.
- Cauliflower needs about 1 to 1.5 inches of water each week, which may not be provided by rainfall alone.
- Cauliflower plants needs adequate nitrogen, and may require fertilizer.

When the heads are slightly smaller than a doorknob, the leaves should be tied together over the heads (often called “blanching”). This helps protect the head from sunlight and allows cauliflower to develop its characteristic creamy white color. Cauliflower is usually mature about a week after the leaves are tied.

References

1. IARC Handbooks of Cancer Prevention. International Agency for Research on Cancer. World Health Organization. ch 1: Cruciferous vegetables. IARC Press, 2004,p 1-12. Oxford Press, UK.
2. Higdon J. Cruciferous Vegetables. Micronutrient Information Center. Linus Pauling Institute. 2005. <http://lpi.oregonstate.edu/infocenter/foods/cruciferous/> Accessed 2/4/14.
3. U.S. Department of Agriculture, Agricultural Research Service. 2013. USDA National Nutrient Database for Standard Reference, Release 26. Nutrient Data Laboratory Home Page, <http://www.ars.usda.gov/ba/bhnrc/ndl>. Accessed 2/4/14.
4. Talalay P, Fahey JW. Phytochemicals from cruciferous plants protect against cancer by modulating carcinogen metabolism. *J Nutr*. 2001;131(11): 30275-30335.
5. Kampman E, Arts IC, Hollman PC. Plant foods versus compounds in carcinogenesis; observational versus experimental human studies. *Int J Vitam Nutr Res*. 2003;73(2):70-78.
6. National Cancer Institute Fat Sheet. Cruciferous Vegetables and Cancer Prevention. Reviewed 6/07/12. <http://www.cancer.gov/cancertopics/factsheet/diet/cruciferous-vegetables>. Accessed 2/8/14.
7. Liska DJ. The Detoxification Enzyme Systems. *Alternative Medicine Review*. 1998;3(3):187-198.
8. Kirilin WG, Cai J, DeLong MJ, Patten EJ, Jones DP. Dietary compounds that induce cancer preventive phase 2 enzymes activate apoptosis at comparable doses in HT29 colon carcinoma cells. *J Nutr*. 1999;129(10):1827-1835.
9. Lee J, Khor TO, Shu L, Su Z-Y, Fuentes F, Kong AT. Dietary phytochemicals and cancer prevention: Nrf2 signaling, epigenetics, and cell death mechanisms in blocking cancer initiation and progression. *Pharmacology & Therapeutics*. 2013;127(2):153-171.
10. Singh SV, Singh K. Cancer chemoprevention with dietary isothiocyanates mature for clinical translational research. *Carcinogenesis*. 2012;33(10):1833-1842.
11. Agudo A, Ibanex R, Amiano, et al. Consumption of cruciferous vegetables and glucosinolates in a Spanish adult population. *Eur J Clin Nutr*. 2008;62(3):324-331.
12. Center for Nutrition, Diet and Health. Cauliflower. The University of the District of Columbia. <https://www.udc.edu/docs/causes/online/cauliflowersm.pdf>. Accessed 2/15/14.
13. Jett J. Growing Cauliflower. Cooperative Extension Service. West Virginia University Center for Extension and Continuing Education. Developed 1996. Accessed 2/15/14.
14. Smith KL. Growing broccoli and cauliflower in the home garden. Ohio State University Extension. Accessed 2/16/14.




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Prophylactic Feeding Tube Placement and the Incidence of Malnutrition in the Head and Neck Population – A Review of the Literature

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The incidence of malnutrition in patients diagnosed with head and neck cancer (HNC) ranges from 30-50% (1). Malnutrition ($\geq 10\%$ weight loss in 6 months, or $\geq 5\%$ weight loss in 1 month) is associated with multiple morbidities including impaired wound healing, compromised immune function, and decreased tolerance to various modalities of cancer treatment (2,3). Tumor location and associated treatments also affect nutritional status due to alterations in normal metabolism and the side-effects they induce (4).

Feeding tube placement prior to treatment may reduce, or prevent, the identified morbidities in HNC patients. However, there are currently no standardized criteria regarding this practice (5). Therefore, the purpose of this literature review is to answer the question: in patients with HNC receiving chemotherapy, radiation therapy, or chemoradiation therapy, is the risk of malnutrition reduced with prophylactic feeding tube placement versus relying on oral food, fluid, and nutritional supplement intake in the short-term during treatment as well as the in the long-term?

Background

HNC include tumors of the oral cavity, pharynx, larynx, salivary glands, and sinus (6). Tumors invading areas involved with swallowing increase one's risk for malnutrition due to difficulties managing treatment-related side effects, a decrease in swallowing abilities, and the metabolic alterations that occur (4,6). Treatments include: surgery, chemotherapy, radiation therapy, or combinations thereof. Common treatment side-effects include: dysphagia, nausea/vomiting, anorexia, mucositis, xerostomia,odynophagia, and dysgeusia.

Tumor presence induces multiple alterations in metabolism. Glucose is the main energy source for tumors, which increases the demand for glucose production in the liver (6). Concurrently, there are also abnormal

elevations in the Cori cycle, which increases lactate production. The host also experiences increased gluconeogenesis and increased insulin insensitivity due to increased production of glucocorticoids and glucagon. Muscle wasting and increased hepatic protein synthesis occur due to increased proteolysis and increased nitrogen depletion as a result of Tumor Necrosis Factor (TNF). Cytokines enhance liver lipogenesis, and lipid-mobilizing factor induces lipolysis by increasing cyclic adenosine monophosphate (cAMP) production. Cytokines may also contribute to malnutrition by decreasing gastric motility and emptying, and by increasing corticotropin-releasing hormone, resulting in reduced food intake (6).

Literature Review

Most recent literature primarily focuses on the prevalence and risk-factors associated with malnutrition in HNC patients. However, Peerawong et al (7) recently published their findings of a retrospective study looking at the percentage of weight change in patients receiving standard chemoradiation (cisplatin 100mg/m² or carboplatin (6 AUC) every three weeks with concurrent radiotherapy) who had PPEG (prophylactic percutaneous enteral gastrostomy) (n=77) versus those without PPEG (non-PPEG) (n=142). Results indicated that, while both groups did lose weight, those with PPEG lost significantly less than those non-PPEG patients (9% from baseline versus 15.3%).

The prevalence of malnutrition in this population was the focus of both Jager-Wittenaar et al (2) and Givens et al (8). Jager-Wittenaar et al (2) identified that malnutrition occurred in 24% of 54 patients who received primary radiotherapy, surgery plus radiotherapy, or chemoradiation, while Givens et al found that 26% of their 104 patients experienced malnutrition. Twenty six point four percent of the 104 patients who received combined chemoradiation with either cisplatin or carboplatin/5-fluorouracil every three weeks; or weekly chemotherapy of cisplatin or carboplatin/paclitaxel required enteral feedings between the date of treatment completion and date of most recent follow-up; however, it was not indicated when the feeding tubes were placed in relation to their treatment (8). Logemann et al (3) also noted that malnutrition risk was increased during combined chemoradiation utilizing multiple chemotherapy regimens. Six percent were taking $\leq 50\%$ of nutrition orally at baseline, which rose at the 3 month assessment period to 23%. Of the 53 patients within this study, those requiring enteral nutrition also increased from 15% pre-treatment to 40% when evaluated at the 3 month interval.

In efforts to decrease the incidence of malnutrition and placement of feeding tubes, Lee et al (1) sought to determine if initiation of a free oral nutritional supplement program could reduce weight loss and decrease feeding tube placement in HNC patients receiving radiation therapy. Results indicated that weight loss was reduced from approximately 9.3% to 5.7% in patients with an existing feeding tube, and the need for percutaneous enteral gastrostomy (PEG) tube placement was reduced from 31% to 6% with use of the program. One of the limitations of this study is the difficulty for all medical oncology clinics to obtain free oral nutrition samples for their head and neck population.

As noted, treatment-related side effects, including mucositis, can increase a patient's risk for malnutrition due to the resulting significant decline in oral intake. Zahn et al (9) noted that in patients receiving radiation therapy, mucositis severity significantly decreases (n=40, $p=.010$) as protein intake

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increases (goal intake of 1.5gm/kg). In addition, Meirovitz et al (10) noted that while seven of the fifteen patients (46.7%) required placement of a feeding tube during combined chemoradiation (weekly cisplatin 40mg/m² or carboplatin AUC 2), all individuals who experienced grade IV mucositis (66.7%) required feeding tube placement.

Zahn et al (9) also noted that 5% were dependent on enteral nutrition before treatment. This percentage rose to 67.5% when pre-treatment and during-treatment feeding tube placement periods were combined. Weight loss was also reduced from 7.2% to 4.1% when comparing those who *did not* versus those who *did* meet caloric intake goals of 35kcal/kg (8). Interestingly, Cheng et al (10) noted that those with more significant disease (Stage III/IV), and individuals requiring combined chemoradiation therapy are more likely to have a feeding tube placed ($p=0.03$, $P < 0.001$).

Consistent with other findings (7,10) Jeffrey and colleagues (11) performed a retrospective study and found that a significant increase in need of feeding tube placement occurred with higher-risk tumor sites (87.5%). The need for ENS (enteral nutrition support) was also higher in patients requiring combined chemoradiation therapy. Weight loss was unaffected by the use of either ENS or oral nutrition support (ONS). In fact, those that received nutrition counseling *only* experienced less weight loss than the two other groups (Counseling 0.06% versus ONS 7.61% and ENS 8.94%). It is important to note that those within the counseling only group also experienced fewer nutrition impact symptoms than the other two groups.

Placement and use of an enteral feeding tube is not without its associated risks. These include: increased infection rates, leakage from the feeding tube site, and the potential for longer dependence on enteral nutrition due to atrophy of muscles involved with swallowing (3,4,8). Therefore, careful consideration to each patient's needs is necessary. Conversely, there are also benefits including: fewer, or no hospitalizations, decreased incidence of dehydration, fewer treatment breaks, improved wound healing, potentially-

reduced weight loss, and the ability to individualize nutrition prescriptions (3,4,5,8).

Conclusion/Areas for Future Research

Current literature indicates that weight loss and associated morbidities may be reduced with placement of a feeding tube. However, at present, there is insufficient evidence to determine whether prophylactic feeding tube placement results in a net benefit to the patient (5). It is also known that the sequelae from the treatments can last long past treatment completion (14). Theoretically, this may also impact the HNC patient's long-term nutritional status. For this reason, future research should "place high priority on conducting a full-scale, multi-center randomized clinical trial" regarding prophylactic feeding tube placement and the long-term incidence of malnutrition (5).

References

- Lee HL, et al. Effect of oral nutritional supplementation on weight loss and percutaneous endoscopic gastrostomy tube rates in patients treated with radiotherapy for oropharyngeal carcinoma. *Supportive Care in Cancer*. 2008;16:285-289. <http://www.springerlink.com.ezproxy.rosalindfranklin.edu:2048/content/a811lg663v126712/>. Accessed October 30th, 2011.
- Jager-Wittenaar H, et al. Malnutrition and quality of life in patients treated for oral or oropharyngeal cancer. *Head and Neck*. June 2011;33(6):863-870. <http://onlinelibrary.wiley.com/doi/10.1002/hed.21545/full>. Accessed September 15th, 2011.
- Logermann JA, et al. Site of disease and treatment protocol as correlates of swallowing function in patients with head and neck cancer treated with chemoradiation. *Head and Neck*. January 2006;28(1):64-73. <http://onlinelibrary.wiley.com.ezproxy.rosalindfranklin.edu:2048/doi/10.1002/hed.20299/abstract;jsessionid=B7D662FFD480B2E13B70F6D4B8E70A2C.d04t04>. Accessed September 16th, 2011.
- Schattner M, Shike M. Nutrition Support of the Patient with Cancer. In: Shils, ME, Shike M, Ross AC, Caballero B, Cousins RJ. *Modern Nutrition in Health and Disease*. 10th Edition. New York: Lippincott, Williams, and Wilkins;1290-1313.
- Locher JL, et al. Prophylactic percutaneous endoscopic gastrostomy tube placement in treatment of head and neck cancer: A comprehensive review and call for evidence-based medicine. *JPEN*. May 2011;35(3):365-374. <http://sagepub/c.com/content/35/3/365>. Accessed September 15th, 2011.
- Elliott L, Molseed LL, McCallum PD, et al. *The Clinical Guide to Oncology Nutrition*. Second Edition. USA:American Dietetic Association;2006.
- Peerawong T, Phunggrassami T, Pruegsanusak K, Sangthong R. Comparison of treatment compliance and nutritional outcomes among patients with nasopharyngeal carcinoma with and without percutaneous endoscopic gastrostomy during chemoradiation. *Asian Pacific Journal of Cancer Prevention*. 2012;13(11):5805-5809. http://www.apocpcontrol.org/page/apjcp_issues_view.php?sid=Entrez:PubMed&id=pmid:23317260&key=2012.13.11.5805. Accessed October 18th, 2013.
- Givens DJ, et al. Adverse events associated with concurrent chemoradiation therapy in patients with head and neck cancer. *Archives of Otolaryngology – Head and Neck Surgery*. December 2009;135(12):1209-1217. <http://archotol.ama-assn.org.ezproxy.rosalindfranklin.edu:2048/cgi/content/full/135/12/1209>. Accessed September 15, 2011.
- Zahn KL, et al. Relationship of protein and calorie intake to the severity of oral mucositis in patients with head and neck cancer receiving radiation therapy. *Head and Neck*. June 2011. <http://onlinelibrary.wiley.com/doi/10.1002/hed.21795/full>. Accessed September 16th, 2011.
- Meirovitz, et al. Cytonikes levels, Severity of acute mucositis and the need for PEG tube installation during chemo-radiation for head and neck cancer – a prospective study. *Radiation Oncology*. February 25, 2010;5(16):1-7. <http://www.ncbi.nlm.nih.gov/pubmed/?term=cytokines+levels%2C+severity+of+acute+mucositis+and+the+need+of+PEG>. Accessed March 31, 2014.
- Cheng SS, et al. Variables associated with feeding tube placement in head and neck cancer. *Archives of Otolaryngology – Head and Neck Surgery*. 2006; 132:655-661. <http://archolo.ama-assn.org.ezproxy.rosalindfranklin.edu:2048/cgi/content/full/132/6/655>. Accessed September 16th, 2011.
- Jeffrey E, Sherriff J, Langdon C. A clinical audit of the nutritional status and need for nutrition support amongst head and neck cancer patients treated with radiotherapy. *Australasian Medical Journal*. 2012;5(1):8-13. <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3413925/pdf/AMJ-05-08.pdf>. Accessed October 18th, 2013.
- Wilson JA, Carding PN, Patterson JM. Dysphagia after nonsurgical head and neck cancer treatment: patients' perspectives. *Otolaryngology- Head and Neck Surgery*. November 2011;145(5):767-71. <http://oto.sagepub.com/content/145/5/767.abstract>. Accessed October 18th, 2013.
- Garden AS, Harris J, Trotti A, et al. Long-term results of concomitant boost radiation plus concurrent cisplatin for advanced head and neck carcinomas: a phase II trial of radiation therapy oncology group (RTOG 99-14). *Int J Radiat Oncol Biol Phys*. 2009;71:1351-2355.

EXPERT INTERVIEW: Five Questions on Nutritional Genomics

Guest Expert: Colleen Spees, PhD, MEd, RD, LD

Maureen Leser, MS, RD, CSO, LD

Nutritional genomics is an emerging area of nutritional science that will enable practitioners to personalize nutrition prescriptions. It helps explain why some people need more folate than others and why cruciferous vegetables may enhance cancer prevention. It explains how certain nutrients help prevent genetic damage which may ultimately decrease cancer risk and how specific nutrients and bioactive food components may have benefits for cancers with unique genetic signatures.

On Monday, October 21st, 2013, several hundred Registered Dietitian Nutritionists (RDNs) filled the Food & Nutrition Conference & Expo™ session “Nutrition and Cancer: From Genotype to Phenotype” to hear Ohio State University professors Colleen Spees, PhD, MEd, RD, LD and Steven Clinton, MD, PhD, share their knowledge and insights about nutrigenomics and cancer. By bringing the science of nutrigenomics to the forefront, Drs. Spees and Clinton excited the crowd and demonstrated how eating right is pivotal to cancer prevention. Dr. Spees is our guest expert of our FIVE QUESTIONS feature, answering five questions that will enhance the oncology RDNs knowledge of the expert’s specialty.

Question 1. What terms should RDNs need to be familiar with to better understand the field of nutrigenomics?

Term	Definition
Acetylation	Modification of histones by attachment of acetyl groups.
Acquired (or somatic) mutation	A change in the genetic structure that is neither inherited nor passed to offspring; occurs after conception.
Bioactive food components	Substances in foods which are not essential nutrients, but may have biologic effects. These include phytochemicals (plants), zoochemicals (animal), fungochemicals (mushrooms), bacterochemicals (gut bacteria).
Chromatin remodeling	Changes in chromatin structure that occur during regulatory processes and alter the nuclease sensitivity of a region of chromatin.
Deletion	A type of mutation in which genetic information has been lost.
Duplication	A type of mutation that involves the production of one or more copies of a gene or region of a chromosome.
Epigenetics	Modifications in DNA that affect gene expression and function without altering the nucleotide sequence. Examples of epigenetics include altering the proteins that control gene expression through methylation or acetylation, or regulation of microRNA's which can then regulate gene expression.
Epigenomics	The study of epigenetic changes in a cell or entire organism.
Genomewide association study (GWAS)	An approach that looks for associations between many specific genetic variations and particular diseases.
Haplotype	A group of gene variants that occur together.
Histone	A protein around which DNA is wrapped.
Histone modification	A variety of modifications (i.e. acetylation, methylation, phosphorylation, ADP-ribosylation) that occur on histones and may augment gene transcription and translation.

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Term	Definition
Inherited mutation	A change in the genetic structure that is inherited or passed to offspring.
Methylation	The addition of methyl (-CH ₃) groups to DNA. DNA methylation patterns can be inherited and impact patterns of gene expression.
MicroRNAs	Small fragments of cytosolic RNA, usually about 22 nucleotides in size, which bind to mRNA and function as post transcriptional regulators. Also called miRNA.
Noncoding RNAs	RNA that does not encode a protein but appears to play a role in both oncogenic and tumor suppressive pathways.
Nutritional genomics	The scientific investigation of the composite interactions between nutrients, bioactive food components and the genome as they impact host health and disease.
Oncogenes	A gene whose product is involved either in transforming cells in culture or inducing cancer in animals. Most are mutant forms of normal genes involved in control of cell growth or division.
Phenotype	The physical and observable properties, or traits, of an organism.
Point mutations	Change of a single nucleotide in DNA, especially in a region coding for protein.
Polymorphisms	The minor variations, among individuals, in the sequence of DNA bases in specific genes.
Translocation	Movement of a segment of a chromosome from its normal site to another chromosome.
Tumor suppressor gene	Genes that normally restrain cell growth but, when missing or inactivated by mutation, allow cells to grow uncontrolled.
Xenobiotics	Chemicals found in an organism but not produced by it, such as drugs or pollutants.

Question 2. Can you provide a specific example of current research in this field that oncology RDNs should know about?

One example of nutritional genomics related to cancer oncology concerns the methylenetetrahydrofolate reductase (MTHFR) gene. MTHFR encodes for 5,10-MTHFR, an enzyme required for the successful conversion of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate. This critical chemical reaction is required for successful folate metabolism and the conversion of homocysteine to methionine. Two common MTHFR variants result in lower bioavailable folate and higher homocysteine levels. This combination correlates with increased susceptibility to certain cancers (1,2,3), neural tube defects (4), mental health disorders (5), and stroke (6). Medical nutrition therapy for mild to severe MTHFR deficiency consists of dietary modifications and folate supplementation in an attempt

to restore enzymatic function to near normal levels (7). RDNs remain critical health care team members when identifying and managing gene-diet and gene-environment interactions.

Another fascinating area of research relates to epigenetics. Several bioactive food components have been associated with altering molecular mechanisms that alter gene expression and impact the carcinogenic cascade.

Indeed, exciting developments are occurring in the research field of nutritional genomics. However, until gene therapy is widely studied and proven to be efficacious, RDNs should provide high risk clients (predisposed to cancer secondary to genetic mutations) with evidence-based recommendations for modifiable lifestyle behaviors known to delay the onset of cancer, slow cancer progression, reduce the risk of cancer-related co-morbidities,

and improve quality of life. Such recommendations also provide most clients with some sense of control in an otherwise chaotic environment. The American Institute of Cancer Research (AICR) provides the most current evidence for practitioners and clients related to diet and physical activity guidelines for cancer prevention and survivorship.

Question 3. What is the future of nutrigenomics for the RDN?

Nutrigenomics offers the unique opportunity to conduct applied research that will improve our ability to provide personalized nutritional prescriptions for optimizing health and for the prevention and treatment of chronic diseases, including cancer. As the field of nutritional genomics continues to advance, it is our professional duty to ensure that RDNs are adequately trained and positioned to serve as the nutrition experts in both nutritional genomic research and education. The ON

DPG is in a unique position to lead this charge as the majority of Nutritional Genomics (NG) research is based on describing how cancer and bioactive foods impact carcinogenesis. In addition, the ON DPG has several members that are national experts in this field.

Question 4. Can you share how the Ohio State's Medical Dietetics Program is training future RDNs in this field?

In 2011, I began teaching a Nutritional Genomics course (MD 6900) after discovering that RDNs were not adequately exposed to this novel field of study. Introducing RDNs in training (or dietetics students) to NG will better prepare them to become leaders in the field and to serve as the nutrition expert on translational NG teams. Although my course continues to focus on the field of nutrition, it has grown to include students from other disciplines including pharmacy, food science, public health, nursing, exercise science, and others. In 2013, we began recording national NG webinars so our students could be exposed to the role of NG in other diseases. Kathy Camp, the late John Milner and other professionals have generously contributed to our webinar series. We hope to eventually expand our course offering to dietetics students and RDNs soon!

Question 5. What resources would you recommend for a "Nutrigenomics Toolkit"?

- Oncology Nutrition Dietetic Practice Group's newly published book *Oncology Nutrition for Clinical Practice* is one of the few books available on oncology nutrition that includes a chapter on Nutritional Genomics & Cancer.
- The Academy's recently published Position Paper on NG (<http://www.eatright.org/About/Content.aspx?id=6442479881>)
- AICR/WCRF's Resources for Health Professionals (<http://www.aicr.org/health-professionals/>)

- CDC's Genomic Health Updates (<http://www.cdc.gov/genomics/public/index.htm>)
- Basic resources for those new to the field of Nutrigenomics:
 - <http://learn.genetics.utah.edu/>
 - <http://ghr.nlm.nih.gov/>
 - <http://nutrigenomics.ucdavis.edu/>
 - http://www.nchpeg.org/nutrition/index.php?option=com_content&view=article&id=400&Itemid=563
 - <https://familyhealthlink.osumc.edu/Notice.aspx>

584-594, ISSN 0140-6736, [http://dx.doi.org/10.1016/S0140-6736\(11\)60872-6](http://dx.doi.org/10.1016/S0140-6736(11)60872-6). (<http://www.sciencedirect.com/science/article/pii/S0140673611608726>)

7. Ferguson LR, Barnett MPG. Research in nutrigenomics and potential applications to practice. *Nutrition & Dietetics*. 2012, 69: 198-202. doi: 10.1111/j.1747-0080.2012.01623.

ON DPG would like to sincerely thank Dr. Spees for sharing her expertise with our members.

References

1. Martha L, Slattery J, Potter D, Samowitz W, Schaffer D, Leppert M. Methylenetetrahydrofolate Reductase, Diet, and Risk of Colon Cancer. *Cancer Epidemiol Biomarkers Prev June 1999 8:513-518*
2. Curtin K, Bigler J, Slattery ML, Caan B, Potter JD, Ulrich CM. MTHFR C677T and A1298C Polymorphisms: Diet, Estrogen, and Risk of Colon Cancer. *Cancer Epidemiol Biomarkers Prev February 1, 2004 13:285-292; doi:10.1158/1055-9965.EPI-03-0083*
3. Krajcinovic M, Lamothe S, Labuda D, Lemieux-Blanchard E, Théorêt Y, Moghrabi A, Sinnett D. Role of MTHFR genetic polymorphisms in the susceptibility to childhood acute lymphoblastic leukemia *Blood 2004 103:252-257; published ahead of print September 4, 2003, doi:10.1182/blood-2003-06-1794*
4. Blom HJ, Smulders Y. Overview of homocysteine and folate metabolism. With special references to cardiovascular disease and neural tube defects. *Journal of Inherited Metabolic Disease*. February 2011, Volume 34, Issue 1, pp 75-81
5. Gilbody S, Lewis S, Lightfoot T. Human Genome Epidemiology (HuGE) Review: Methylenetetrahydrofolate Reductase (MTHFR) Genetic Polymorphisms and Psychiatric Disorders: A HuGE Review *Am. J. Epidemiol 2007. 165 (1): 1-13 first published online October 30, 2006 doi:10.1093/aje/kwj347*
6. Holmes MV, Newcombe P, Hubacek JA et al. Effect modification by population dietary folate on the association between MTHFR genotype, homocysteine, and stroke risk: a meta-analysis of genetic studies and randomised trials, *The Lancet*, Volume 378, Issue 9791, 13-19 August 2011, Pages

CPE Article: Optimizing Outcomes in Oncology Patients: Case Studies Using Updated Guidelines

Laura Elliott, MPH, RD, CSO, LD and Rhone Levin, MEd, RD, CSO, LD

The Oncology Update 2013 to the Evidence Analysis Library (EAL) is a valuable resource to the oncology Registered Dietitian Nutritionists (RDNs) practice. With limited availability to the public and full availability to the Academy of Nutrition and Dietetics' members at www.evidencelibrary.com, the library provides evidence based guidelines for nutrition care. In this age of easy access to nutrition information on the web, directing professionals as well as patients towards evidence based nutrition therapy is of utmost importance. The EAL uses a systematic process, beginning with development of relevant practice questions, critical review of scientific literature, with creation of answers which are then graded and translated into practice recommendations. The objective of this article is to provide a summary of the EAL Oncology update project. This project supports the practicing oncology RDN with evidence based practice recommendations, resources, and guidelines for oncology patient care. The guidelines include an executive summary, recommendations, algorithms, background information and references (1).

These guidelines may be used in various ways:

- To justify adequate RDN full-time equivalent (FTE) to provide evidence-based Medical Nutrition Therapy in the oncology setting.
- To implement nutrition screening and assessment, utilizing validated tools.
- To enhance rapid decision making in adult oncology patient nutrition care.
- To demonstrate to administration how nutrition care affects patient outcomes and the bottom line.
- To expand the scope and practice of the RDN learning and developing oncology specific skill sets.
- To justify frequent assessment by the RDN and the use of nutrition support in the head and neck population.
- To justify the use of modified food supplements.
- To provide evidence-based discussion points regarding use of honey, antioxidants, or fish oils.
- To justify discontinuing the use of 'neutropenic precautions' in diet orders.

- To provide evidence-based practice information to students and peers, and promote development of nutrition care related policies and procedures.
- To promote outcomes based research, and capture data on outcomes affected by the work of a RDN using Medical Nutrition Therapy.
- To generate recommendations for future research.

Use of the Academy's EAL facilitates RDNs' evidence-based practice.

The Oncology Update 2013 project yielded 16 conclusion statements, 22 recommendations and involved analysis of 95 articles. The primary focus of this update was strengthening evidence-based statements regarding the value of the RDNs in improving oncology patient outcomes. This was different from the approach used in the 2007 project, which focused primarily on disease state interventions. The current project yielded recommendations with greater value, of which almost 70% were of the highest

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grade, strong. See Table 1 for the definitions of each grade.

This update includes recommendations for each step of the nutrition care process and provides guidance regarding the following: nutrition screening and screening tools; oncology nutrition assessment criteria and validated assessment tools; assessment and intervention in cancer cachexia, including the use of fish oil; diagnosis of malnutrition in adult oncology patients; significance of weight change, loss of lean body mass, and sarcopenia; and recommended monitoring and evaluation.

In order to expand the scope of the RDN and avoid duplicating work already published by other professional organizations, external guideline grading systems were reviewed by the Evidence-Based Practice Committee and aligned with the Academy's EAL scoring systems. This allowed guidelines written by the American Society of Parenteral and Enteral Nutrition (A.S.P.E.N.), the Oncology Nursing Society (ONS) and the Clinical Oncological Society of Australia (COSA) to be included in the Academy's EAL guideline for the following topics: glutamine, neutropenic precautions, nutrition substances and chemotherapy-induced peripheral neuropathy.

The project also provided an opportunity to propose new terminology to be considered in the Nutrition Care Process. The new terminology gives the RDN caring for oncology patients more appropriate and accurate terms that characterize oncology

Table 1. Definition of Statement Grading

Statement Rating	Definition	Implication for Practice
Strong	A Strong recommendation means that the workgroup believes that the benefits of the recommended approach clearly exceed the harms (or that the harms clearly exceed the benefits in the case of a strong negative recommendation), and that the quality of the supporting evidence is excellent/good (grade I or II). In some clearly identified circumstances, strong recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Practitioners should follow a Strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Fair	A Fair recommendation means that the workgroup believes that the benefits exceed the harms (or that the harms clearly exceed the benefits in the case of a negative recommendation), but the quality of evidence is not as strong (grade II or III). In some clearly identified circumstances, recommendations may be made based on lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits outweigh the harms.	Practitioners should generally follow a Fair recommendation but remain alert to new information and be sensitive to patient preferences.
Weak	A Weak recommendation means that the quality of evidence that exists is suspect or that well-done studies (grade I, II, or III) show little clear advantage to one approach versus another.	Practitioners should be cautious in deciding whether to follow a recommendation classified as Weak, and should exercise judgment and be alert to emerging publications that report evidence. Patient preference should have a substantial influencing role.
Consensus	A Consensus recommendation means that Expert opinion (grade IV) supports the guideline recommendation even though the available scientific evidence did not present consistent results, or controlled trials were lacking.	Practitioners should be flexible in deciding whether to follow a recommendation classified as Consensus, although they may set boundaries on alternatives. Patient preference should have a substantial influencing role.
Insufficient Evidence	An Insufficient Evidence recommendation means that there is both a lack of pertinent evidence (grade V) and/or an unclear balance between benefits and harms.	Practitioners should feel little constraint in deciding whether to follow a recommendation labeled as Insufficient Evidence and should exercise judgment and be alert to emerging publications that report evidence that clarifies the balance of benefit versus harm. Patient preference should have a substantial influencing role.

patient care, ultimately leading to improved outcomes for data collection.

While we were pleased with the work accomplished, our workgroup acknowledges there are many more oncology nutrition questions yet to be answered. Periodic updates of the EAL are planned for the future. See table 2 for a summary of the recommendations and their grading.

One of the useful tools developed during the oncology update reviews the relationship between nutrition status and oncology related outcomes.

This information underscores the value of the oncology RDN and nutrition services

which may influence the following morbidity outcomes:

- Hospital admissions or re-admissions
- Hospital length of stay
- Quality of life
- Radiation treatment tolerance
- Chemotherapy treatment tolerance

The National Cancer Institute (NCI) at the National Institutes of Health defines morbidity as “a disease or the incidence of disease within a population. Morbidity also refers to adverse effects caused by a treatment.” In the case of cancer, examples of morbidity include, but are not limited to side effects of chemotherapy, radiation therapy treatment or surgery, infection, and hospitalization. Morbidity is classified and

reported using tools such as the NCI Common Toxicity Criteria (2). The relationship between nutrition status and mortality, as it relates to the cancer diagnosis was also reviewed.

For an overview of the relationship between nutrition status and outcomes, see outcomes chart.

References

1. Carson, JA. The EAL: A Valuable Tool for the Oncology Dietitian *ON Newsletter* 15:4, 2007/2008, p. 7-8.
2. Jaques, DP. Measuring morbidity. *Ann Surg.* 2004 Aug; 240 (2):214-5.

(Continued on page 20)

Table 2. Oncology Nutrition Update 2013 Recommendations

Recommendation	Grade
Nutrition Screening and Referral	
Screening for Malnutrition Risk and Referral of Adult Oncology Patients	Consensus
Referral of Adult Oncology Patients Identified at Malnutrition Risk to the RDN	Consensus
Malnutrition Screening Tools for Adult Oncology Patients	Strong
Medical Nutrition Therapy	
Medical Nutrition Therapy in Adult Oncology Patients Undergoing Chemotherapy and Radiation Treatment	Strong
MNT as a Part of Multi-modal Therapy in Adult Oncology Patients Undergoing Chemotherapy and Radiation Treatment	Fair
Nutrition Assessment	
Nutrition Assessment Tools for Adult Oncology Patients	Strong
Nutrition Assessment Criteria in Adult Oncology Patients	Consensus
Assessment of Food/Nutrition-Related History of Adult Oncology Patients	
Assessment of Anthropometric Measurements in Adult Oncology Patients	
Assessment of Biochemical Data, Medical Tests and Procedures of Adult Oncology Patients	
Assessment of Nutrition-focused Physical Findings and Client History of Adult Oncology Patients	
Nutrition Assessment for the Stages of Cancer Cachexia	Consensus
Nutrition Diagnosis	
Nutrition Diagnosis of Malnutrition in Adult Oncology Patients	Consensus
Nutrition Intervention	
Nutrition Intervention for Adult Oncology Patients with Cancer Cachexia	Consensus
Fish Oil, Weight and Lean body Mass in Adult Oncology Patients	
Dietary Supplements containing Fish Oil for the Adult Oncology Patient	Strong
Medical Food supplements Containing Fish Oil for the Adult Oncology Patient	Strong
Glutamine and Oral Mucositis in Adult Oncology Patients with Solid Tumors	Weak (ONS)
Parenteral Glutamine and Hematological Cell Transplant in Adult Oncology Patients	Fair (A.S.P.E.N.)
Nutrition Substances and Chemotherapy-Induced Peripheral Neuropathy in Adult Oncology Patients	Weak (ONS)
Neutropenic Dietary Precautions for Adult Oncology Patients	
Neutropenic Dietary Precautions for Adult Oncology Patients with Neutropenia (non-Bone Marrow Transplant)	Fair (A.S.P.E.N.)
Neutropenic Dietary Precautions for Adult Oncology Patients Undergoing Bone Marrow Transplant	Weak (A.S.P.E.N., ONS)
Monitoring and Evaluation	
Monitoring and Evaluation in Adult Oncology Patients	Consensus
Monitoring and Evaluating Adult Oncology Patients with Cancer Cachexia	Consensus
Outcomes Management	
Nutrition Status and Outcomes of Adult Oncology Patients	Strong

Recommendations from external organizations are noted in parentheses.

Outcomes Chart

Relationship Between Nutrition Status and Morbidity Outcomes and Mortality in Adult Oncology Patients

Studies	Morbidity					Mortality
	Hospital Admissions and Readmissions	Hospital Length of Stay	Quality of Life	Radiation Treatment Tolerance	Chemotherapy Treatment Tolerance	Mortality
Alexandre 2003					+	
Amaral 2008		+				
Antoun 2009		+				
Barlow 2011	NS	+				
Bauer 2005			+			
Braga 1998		+				
Capuano 2008	+			+	+	+
Carey 2011			+			
Correia 2007			+			
Dewys, 1980						+
Eriksson 1998					+	
Fearon 2006			+			+
Gioulbasanis 2011						+
Gupta 2010						+
Hammerlid 1998			NS			+
Hill 2011	+			+	+	
Horsley 2005		+				
Hyltander, 2005		NS	+			
Ionescu 2009		+				
Isenring 2003			+			
Iverson 2010			+			
Kathiresan, 2011	+					
Laky 2010		+	+			
Martin 2009						+
Martin 2010						+
Nourissat 2008			+			
Odelli, 2005				+		
Ollenschlager 1992			+			
Persson 1999						+
Phippen 2011					+	
Piquet 2002	+					
Pressoir 2010		+				+
Prado, 2007					+	
Prado, 2008						+
Prado, 2009					+	
Prado, 2011					+	
Ravasco, 2003			+	+		
Ravasco, 2005 (JCO)			+	+		
Ravasco, 2005 (H&N)			+	+		
Robinson 2008					+	+
Ross 2004					+	+
Shahmoradi 2009			+			
Sorenson 2008		+				+
Tan 2009						+
Yoon 2011						+

KEY
 NS = Nonsignificant effect on outcome.
 + = Positive effect on outcome.
 Blank = No negative effect on outcome.

The following case studies demonstrate the application of nutrition assessment and Medical Nutrition Therapy (MNT) in an oncology nutrition setting, and makes note of the application of practice recommended in the updated Oncology EAL.

Case Study 1: Pancreatic Cancer

- ▶ Patient is a 78-year-old female.
- ▶ **Medical diagnosis:** Locally advanced adenocarcinoma of pancreas, possible early metastasis to lung. New physical findings: diabetes, lower extremity edema.
- ▶ **Treatment:** Chemotherapy with Gemzar® (Gemcitabine) and Capecitabine® (Xeloda).
- ▶ **Medications:** Using Pancrelipase (Creon 24®), 2 tablets with meals (one before and one after), no diabetic medication, and no acid suppressors.
- ▶ **Anthropometrics:**
 - Height: 61 inches (154 cm)
 - Weight: 136 pounds (lb) (61.8 kg)
 - Weight Change: loss of 7.7 lb (3.5 kg) in 1 month (representing 5% of body weight)
 - Body Mass Index (BMI): 27.6 (calculated as kg/m²)
 - Adjusted Body Weight: 118.8 lb (54 kg)
- ▶ **Laboratory findings:**
 - August Ca 19-9*: 1300 U/ml
 - November Ca 19-9: 134 U/ml
 - January Ca 19-9: 58 U/ml

**Ca 19-9 radioimmunoassay test measures the concentration of tumor-associated antigens in the serum of persons with pancreatic cancer.*

Home monitoring blood fasting glucose ranging between 82 and 90 mg/dL, post meal excursion 150 mg/dL.

Patient is a vegetarian, following a strict diabetic diet despite controlled glucose range and decreased oral intake. Patient is choosing low fat, low carbohydrate foods due to “healthy lifestyle”. Patient does not want to use diabetic medications; she is actively restricting her diet to avoid any glucose excursions that would require medication.

Patient describes her bowel movements when using pancreatic enzymes as “mild constipation” and more “normal” appearance, the color is light tan if not using adequate enzymes.

- ▶ **Oral intake:** Approximately 1000 kcal, 40 g protein (pro), 1850 ml fluid per day.
- ▶ **Estimated needs:**
 - 1450 kcal/day (27 kcal/kg)
 - 65-75 g pro/day (1.0-1.2 g/kg)
 - 1850 ml fluid per day (30 ml/kg)
- ▶ **Nutrition impact symptoms:** Early satiety, gassiness, burping, fatigue, nausea, vomiting in evening if forcing food.

Nutrition diagnosis of malnutrition:

Significant weight loss, oral intake below estimated needs, LE edema, and loss of muscle led to 17 nutrition follow up visits. Examples of guideline based MNT interventions include the following:

- Change pattern of pancreatic enzyme use to maximize effectiveness: change to 1 tablet before meals and 1 in the first half of the meals (per package insert recommendations).
- Consider use of proton pump inhibitor (PPI) to increase enzyme effectiveness (per package insert recommendations).
- Consider use of anti-emetics and/or motility agents to address nausea, vomiting, and mild constipation respectively.
- Check C-reactive protein (CRP), if results are elevated consider fish oil supplementation to reduce inflammation (per guidelines, however: patient did not wish to use fish oil).
- Increase calorie intake: liberalize diet, include higher, calorie- dense ingredients, include carbohydrates at meals with option to use oral hypoglycemic medications if needed (per guidelines to achieve adequate calories to avoid lean body mass loss).
- Increase protein intake: foods, bar, powders, liquids taken in small volumes throughout the day. Patient preferred to try to eat and drink every 2 ½ hours in place of meal only schedule (per guidelines to achieve adequate protein to avoid lean body mass loss).

- Liquid supplement to replace evening meal to reduce evening emesis (per MNT).

Case Study 2: Breast Cancer

- ▶ Patient is a 65-year-old female.
- ▶ **Medical diagnosis:** metastatic ER-/PR-/HER2- breast cancer.
- ▶ **Treatment:** Surgery followed by chemotherapy Taxol® (Paclitaxel).
- ▶ **Medications and dietary supplements:** Narcotic pain medication, St. John’s Wort.
- ▶ **Anthropometrics:**
 - Height: 65.5 inches (167 cm)
 - Weight: 110 lb (50 kg)
 - Weight change: loss of 9.9 lb (4.5 kg) in 1 month (representing 8.3% of body weight)
 - BMI: 17.9
 - Ideal Body Weight (IBW): 128 lb (58 kg)
 - % Ideal Body Weight: 86%
 - Karnofsky Performance score on admission to oncology was 90%. Current Karnofsky Performance score: 60%.
- ▶ **Laboratory findings:**
 - Albumin: 2.9 g/dL
 - Blood Urea Nitrogen (BUN): 6 mg/dL
 - Absolute Neutrophil Count (ANC): 1,000 cells/ml (Grade II Neutropenia)

Patient follows a low cholesterol diet at home. Drinks grapefruit juice daily when her mouth is not sore. Her friend has recommended she avoid all fresh fruits and raw vegetables. She has fallen at home due to numbness in her feet and feeling weak. Patient has been instructed to use stool softener twice per day and laxative every other day but is only initiating bowel regimen medications on day 3 of no bowel movement.

- ▶ **Oral intake:** Approximately 1250 kcal, 40 g pro, 1200 ml fluid per day.

► **Estimated needs:**

1750-1850 kcal (25-27 kcal/kg actual weight plus 500 kcal/day for weight recovery)
60-80 g pro/day (1.0-1.4 g/kg IBW)

► **Nutrition Impact Symptoms:**

Fatigue (may be related to neutropenia), anorexia, early satiety, constipation, mucositis for several days after chemotherapy, painful tingling in her hands and feet.

► **Nutrition diagnosis of malnutrition:**

Significant weight loss, oral intake below needs, decrease in Karnofsky performance score and visible wasting/loss of muscle, which led to 6 nutrition follow up visits.

Examples of guideline based Medical Nutrition Therapy interventions include the following:

- Liberalize diet, discontinue cholesterol restrictions. Maximize nutrient density in foods. No food restrictions due to neutropenia, patient instructed on Food and Drug Administration (FDA) food safety recommendations.
- Use bowel medications as instructed by physician/pharmacist. Education provided regarding the necessity of bowel medications when using narcotics.
- Referred to physician, instructed to wean off of St. John's Wort which is contraindicated with paclitaxel. Discontinue use of grapefruit juice while under treatment with Taxol® (Paclitaxel).
- Consider fatty acid supplementation to help interfere with muscle wasting.
- Option to trial oral glutamine to address peripheral neuropathy, although there is no strong evidence for use.
- Referral to social worker to evaluate for Meals on Wheels and in home services. Strategies reviewed regarding energy conservation: convenience food items, activate family /friend network to assist with shopping and cooking, oral nutritional supplements if eating less than 50% of meals.
- Pain management for mucositis, avoid acidic/ spicy food and beverages, choose soft/moist foods and liquid nutrition when chewing is uncomfortable, use a straw to direct liquids away from ulcers, dip dry foods in soups or gravies.

This self-study program is available only to members of the Oncology Nutrition Practice Group through the ON DPG website. After reading the continuing professional education articles, access the test online by going to <http://www.oncologynutrition.org/> Click "Login" in the top right-hand corner, and sign in using your eatright.org credentials. Hover over "Member Benefits" and click "Quizzes" in the menu. Click the word "take" next to the name of the article that you just read. This activity has been approved for one and a half hour (level 1) of continuing professional education for RDNs by the Commission on Dietetic Registration. Suggested learning need codes include 3000, 5000, 9000.

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