

Oncology Nutrition Connection

Oncology Nutrition

a dietetic practice group of the
Academy of Nutrition
and Dietetics



A publication of the ON DPG
ON DPG Website
www.oncologynutrition.org

Volume 23, Number 1, 2015
ISSN 1545-9896

Table of Contents

- Message from the Chair
page 1
- Case Study: Adult Gastric
Cancer Patient—Surgery and
Chemoradiation Therapy
page 2
- Pediatric Oncology Nutrition
Corner: Low Microbial Diet
in the Oncology Population
page 10
- Congratulations to Your
New ON DPG Executive
Committee Members
page 12
- CPE Articles: Soy and
Breast Cancer
page 13
- An Innovative Student Project:
Impact of Diet on the Risk of
Developing Stomach Cancer
page 18

Message from the Chair

I hope everyone is enjoying the beginning of summer and looking forward to some summer fun. I know I am looking forward to beginning the new year with ON DPG. As I'm diving into my DPG-related activities, I want to give a special thanks to the many hard-working people who have shared their time and expertise with the ON DPG Executive Committee (EC).

More than two dozen dedicated individuals are working hard to ensure that the many benefits of being an ON DPG member are provided for you. From our quarterly newsletter to our bi-weekly eBlasts, electronic mailing list (EML), recently updated website, professional alliances, FREE webinars with CPEs, and more, we have these volunteers to thank. When you see what we have in store for ON DPG this year, you'll know why we truly appreciate these talented RDs.

One of our biggest and most exciting projects is the collaboration of the ON DPG with the Institute of Medicine on a workshop to address access to nutritional care in cancer centers. This is something our Executive Committee feels very passionately about and they are committed to making this workshop happen this year. Our hope is that every patient will have access to a dietitian when they enter a cancer center. They deserve this expertise in care and we want to see that happen.

In addition to the Benchmarking Project, we've planned some great sessions for you at FNCE, taking place October 3-6, 2015 in Nashville, TN. We have our breakfast reception for members and our Spotlight Session: "Marijuana: Is It Medicine Yet for Cancer Symptom Management?" on Sunday October 4th. You do not want to miss these cutting-edge topics. We are also looking forward to an incredible Symposium in the spring of 2016 in Glendale, AZ.

These are just a few of the highlights, so be sure to check out our eBlasts, the website, the EML, and future newsletters for more information on upcoming ON DPG events, activities, and new resources. We are here for our members, and offer our sincerest thank you for your continued support and interest!

Warmly,
Tricia Cox, MS, RD, CSO, LD, CNSC



Oncology Nutrition Connection

A publication of Oncology Nutrition (ON), a dietetic practice group of the Academy of Nutrition and Dietetics. ISSN 1545-9896.

Visit the ON DPG website at www.oncologynutrition.org

Editor:

Suzanne Dixon, MPH, MS, RDN
sdixon@umich.edu

Associate Editors:

Robin Brannon, MS, RD, CSO
robin.brannon@gmail.com

Jodie Greear, MS, RD, LDN
jodie.greear@gmail.com

Maureen Leser, MS, RD, CSO, LD
mgoreleser@gmail.com

Jocelyne O'Brien, MPH, RDN, CSO, LDN
jocelynenasser@yahoo.com

Oncology Nutrition Connection (ONC) ISSN 1545-9896, is the official newsletter of the Oncology Nutrition Dietetic Practice Group (ON DPG), a practice group of the Academy of Nutrition and Dietetics, and is published quarterly. All issues of ONC are distributed to members in electronic format only.

Articles published in *ONC* highlight specific diseases or areas of practice in oncology nutrition. Viewpoints and statements in each newsletter do not necessarily reflect the policies and/or positions of the Academy of Nutrition and Dietetics or ON DPG.

Oncology Nutrition Connection is indexed in the Cumulative Index to Nursing and Allied Health Literature. For inquiries regarding copyright, single-issue sales and past issues, contact the editor. Individuals interested in submitting a manuscript to *ONC* should contact the editor or check the ON website for author guidelines. Individuals who are ineligible for membership in the Academy of Nutrition and Dietetics can order yearly subscriptions to *ONC* for \$35.00 (domestic fee) and \$40.00 (International fee), payable to the Academy of Nutrition and Dietetics/ON DPG. Institutions can subscribe to *ONC* for \$50.00 (domestic yearly fee) and \$65.00 (International yearly fee). ON DPG members have access to archived back issues in pdf format. Non-members can order printed copies of back issues (contact editor for availability) at a cost of \$10.00 each if mailed domestically and \$20.00 each if mailed internationally. Send requests for subscriptions or back issues to the editor. All ON DPG member mailing address changes and email address changes should be sent to the Academy using the address change card in the Journal of the Academy of Nutrition and Dietetics or at eatright.org in the members-only section.

©2015. Oncology Nutrition Dietetic Practice Group. All rights reserved.

CASE STUDY of Adult Gastric Cancer Patient status/post Surgery and Receiving Chemoradiation Therapy

By Nichole Giller, RD, CSO, LD

Background

Medical Diagnosis:

Gastric carcinoma is a type of gastric cancer that grows within the stomach wall as individual scattered cells, rather than forming a single mass or tumor (1). It is invasive, consistent with cancers that grow into normal, healthy tissues.

The patient, FG, was diagnosed with a poorly differentiated gastric cancer with histopathologic grade 3 and stage IIIC (2). The TNM cancer staging system is based on the size and/or extent (reach) of the primary tumor (T), whether cancer cells have spread to nearby (regional) lymph nodes (N), and whether metastasis (M), or the spread of the cancer to other parts of the body, has occurred (3). The specifics of FG's stage 2 diagnosis include:

- T4a – The tumor (T) has grown through the stomach wall into the serosa, but the cancer has not grown into any of the nearby organs or structures.
- N3b – The cancer has spread to 16 or more nearby lymph nodes (N).
- M0 – There is no distant metastasis (M); (i.e., the cancer has not spread to distant organs or sites, such as the liver, lungs, or brain).
- Stage IIIC - The cancer has grown completely through all the layers of the stomach wall into the serosa, but it has not grown into nearby organs or tissues (T4a). It has spread to 7 or more nearby lymph nodes (N3), but it has not spread to distant sites (M0).

Incidence and Survival:

The National Cancer Institute (NCI) predicted 22,220 new cases of gastric cancer and 10,990 deaths from gastric cancer in the United States (U.S.) in 2014 (4).

The survival rate of gastric cancer depends on the specific type, stage, and presence of metastasis. When diagnosed at stage 1, gastric cancer is associated with a 70% cure rate; that rate falls to 4% when diagnosed at stage IV (5). The majority of patients have either regional or distant metastasis when diagnosed, which is associated with an overall five-year survival rate of 29% (6).

Usual medical treatment:

Surgery with concurrent chemoradiation is commonly used to treat those patients diagnosed at advanced stages of gastric cancer.

Usual nutrition needs for patients diagnosed with gastric cancer (7):

Energy: 30-40 kcals/kg (for stable patients who are malnourished / in need of nutritional repletion)

Protein: 1.2-1.5 g/kg (assuming normal renal and hepatic function)

- With concurrent kidney disease: 0.5-0.6 g/kg (unstressed), 1.0 g/kg (with stress and hemodialysis)
- With concurrent encephalopathy: 0.6-0.8 g/kg (with end stage liver disease), 1.0-1.2 g/kg (with cirrhosis)

Fluids: 1ml/kcal

An Anti-dumping diet is often needed while recovering from gastric surgery to prevent or alleviate symptoms of dumping syndrome.

Case Study

Introduction:

38 y/o female (FG) with history of invasive, poorly differentiated diffuse gastric carcinoma (found in the lesser curve of the antrum of stomach), stage T4aN3bM0 (stage IIIC) was admitted for chemoradiation treatment. FG is s/p laparoscopic subtotal gastrectomy (Roux-en-Y surgery 9/10/2013), with liver wedge biopsy (negative) and scheduled for three sets of post-operative outpatient chemotherapy (three cycles per set) and one set of post-operative radiation.

Baseline Demographics:

- Age: 38 y/o
- Gender: Female
- Language: English speaking
- Korean descent
- Nonsmoker with no history of alcohol or drug use
- Employment: worked as a high school social worker prior to her diagnosis and treatment
- Adopted
- Married with 2 children (10 y/o and 8 y/o)
- Many friends and family involved in care

Baseline Nutrition Assessment:

- Height: 62 inches
- Weight: usual adult weight 148 lbs; pre-operative weight (9/5/2013) 145 lbs; post-op weight (and weight at start of first chemotherapy treatment) 128 lbs.
- Body Mass Index (BMI) for pre-op weight = 26.5 (overweight range); BMI at start of first chemotherapy treatment = 23.3 (normal range)
- Good appetite and intake when diagnosed
- Normal diet with acceptable variety of food when diagnosed
- FG did report heartburn and abdominal pain prior to surgical consult
- After surgery the inpatient RDN met with FG once to provide post-gastrectomy diet education (anti-dumping diet) and to give FG samples of high protein foods and medical nutrition beverages

Planned Treatment

FG was scheduled to receive three sets of outpatient chemotherapy treatments, with each set involving three cycles of epirubicin,

Table 1. Common Side Effects and Nutrition Impact Symptoms (NIS) of Planned Treatment (8)

Medication/Treatment	Nutrition Impact Symptoms / Side Effects
Epirubicin	Nausea, vomiting, diarrhea, mucositis, myelosuppression
Oxaliplatin	Nausea, vomiting, diarrhea, myelosuppression, hepatic toxicity, neurotoxicity, myelosuppression
Fluorouracil (5-FU)	Nausea, vomiting, diarrhea, myelosuppression, neurotoxicity
Radiation to Stomach and Abdomen	Diarrhea, malabsorption, enteritis, fatigue, nausea & vomiting, skin changes (e.g., erythema), urinary & bladder changes (e.g., cystitis)

Table 2. Common Nutrition Interventions for Nutrition Impact Symptoms (NIS) Associated with Treatment (9–11)

Nutrition Impact Symptom	Recommended Nutrition Interventions
Nausea	Eat 5-6 small meals/day; limit exposure to food odors; consider eating cool, light foods with little odor; avoid greasy & high fat foods; rest with head elevated for 30 minutes after eating; take anti-nausea medications as directed; consider use of evidence-based complementary therapies, such as standardized ginger dietary supplements and referral for acupuncture, if available
Vomiting	Eat 5-6 small meals/day; limit exposure to food odors; consider eating cool, light foods with little odor; avoid greasy & high fat foods; rest with head elevated for 30 minutes after eating; take anti-nausea medications as directed; consider use of evidence-based complementary therapies such as standardized ginger dietary supplements
Diarrhea	Identify problem foods or eating habits via detailed diet & symptom history; encourage low fat, low fiber, low insoluble and/or low lactose diet; avoid gas producing foods and alcohol; encourage small, frequent meals; consider bulking agents, pectin, and foods high in soluble fiber; avoid sorbitol and other sugar-alcohol containing products; consider multivitamin and mineral supplements
Mucositis	Use "Magic Mouthwash" as needed; use a soft toothbrush; practice good oral hygiene; use a baking soda + salt solution to swish and spit daily; use spoons and straws to direct food around sores; avoid extreme food temperatures
Anorexia*	Encourage small, frequent meals; use medical nutrition beverages; use foods that are easy to prepare and serve; eat by the clock rather than waiting for appetite or hunger cues; consume liquids between meals rather than with meals
Fatigue	Encourage use of easy-to-prepare meals, snacks, prepared foods, energy dense foods, and medical nutrition beverages; advise on use of non-perishable snacks at bedside; eat small, frequent meals and snacks; encourage energy-saving lifestyle habits

* Even when anorexia is not a direct side effect of treatment, it can result from other NIS (e.g., nausea).

oxaliplatin and 5-FU (EOF), followed by radiation therapy.

History During the First Set of Chemotherapy Treatments (began 10/16/2013, one-month post-surgery):

Cycle 1: EOF (epirubicin, oxaliplatin) on Day 1 with continuous infusion 5-FU Days 1-21

Cycle 2: EOF was stopped secondary to diarrhea

(Continued on next page)

Cycle 3: 5-FU was not provided during cycle 3 due to grade 3 diarrhea* as well as grade 3 Palmar-Plantar Erythrodysesthesia (PPE) (i.e., severe blisters and hyperkeratosis on hands and feet), (12).

*Per the Common Terminology Criteria of Adverse Events version 4.0, Grade 3 diarrhea is considered severe and reflects ≥ 7 stools per day over baseline as well as incontinence; hospitalization indicated; severe increase in ostomy output compared to baseline. Symptoms limit self-care of activities of daily living (ADL) (12).

The M.D. requested an outpatient RDN consult for nutrition assessment during first series of chemotherapy for post-gastrectomy symptom management, but patient was not seen until completion of the first set of treatments.

During the first set of treatments, the surgeon and nurse practitioner provided nutrition advice, with the reported goal of maximizing calorie and protein intake. They recommended a minimum intake of 850 calories with 50 grams protein and 48 ounces of fluid per day. Evidence-based energy needs for a stressed cancer patient in need of nutrition repletion are 30-35 kcal/kg, equal to 1740-2030 kcal for FG's pre-chemotherapy weight of 58 kg (7). The surgeon's recommended intake goal of 850 kcal represents 42-49% of FG's estimated energy needs and is inadequate for maintaining nutrition status. FG experienced difficulty eating due to mucositis, diarrhea, constipation, and nausea. The surgeon considered placing a peripherally inserted central catheter (PICC) to allow for parenteral nutrition (PN), but FG refused and committed to increasing her intake. FG remained on a regular diet, supplemented with one-half to one can Ensure[®] per day.

Per the medical record, FG's daily intake during the first set of treatments was approximately 500 kcal, and less than 16 ounces of fluids. In addition, the physician noted that FG was eating some "healthy" foods and some "energy-dense" foods such as flavored corn chips, onion dip, and regular cola. Unfortunately, the RDN, the oncology team member with the

knowledge, skills, experience, and expertise to complete a comprehensive dietary intake and analysis, was not consulted until after completion of the first set of chemotherapy.

During the first set of treatments, FG lost 17 pounds, or 12% (severe) of beginning weight, which meets criteria for malnutrition established by the Academy of Nutrition and Dietetics and the American Society of Parenteral and Enteral Nutrition (13).

After the first set of treatments was completed, the physician noted in the medical record that FG was drinking "protein drinks" and consuming a liquid diet with > 1000 kcal, > 60 g protein, and > 300 ml fluids per day. The physician ordered a pureed diet for two weeks, because the physician felt FG would tolerate pureed foods better than solid foods, and the physician wanted FG to take in more than liquid "protein drinks" for nutrition.

History Between First and Second Set of Treatments:

The treating physician scheduled a one-month break in between the first and second sets of treatments, in order to allow FG to regain strength. The physician advised FG "to gain 10 pounds" via a regular diet. On 12/4/2013 the physician ordered a nutrition consult with an RDN, due to FG's continued poor oral intake and ongoing diarrhea. The RDN counseled FG on symptom management strategies for diarrhea, nausea, and vomiting; a food pattern that would prevent and/or reduce risk of dumping syndrome events; and high energy food and beverage choices that were likely to be well-tolerated, were consistent with other dietary modifications, and which could be used to increase her energy and protein intake. The RDN requested FG's husband submit a one-week food diary for his wife, but the food diary was not submitted, nor was any further mention of it recorded in the medical record. RDN recommended that the medical team consider Enteral Nutrition (EN) or Parenteral Nutrition (PN) if FG did not consume at least 500 calories (an amount equal to 25-29% of estimated energy needs) and 40-50 grams protein per day, a recommendation

provided by a dietitian seen in a previous consultation, prior to referral to the Certified Specialist in Oncology Nutrition (CSO).

To manage micronutrient losses secondary to diarrhea, FG received a saline solution containing sugar, multivitamins, folate, and thiamine (referred to as a "Banana Bag" in our institution) three times per week during the treatment break, along with the maximum allowable doses of loperamide and lomotil (diphenoxylate and atropine). Per the medical oncologist, FG would need to keep weight above 100 pounds, and if unable to do so, enteral or parenteral nutrition would be provided to improve nutritional intake. FG's husband stated she had a good appetite and had been eating well at meal times during the break; however, FG was unable to gain the physician-requested goal of 10 pounds. Contradictory to the husband's report, FG's friend observed she was "just not eating or even taking in the shakes." FG remained on a regular diet as tolerated. She lost an additional 15 pounds during her treatment break, confirming an inadequate intake contributing to further weight loss and malnutrition.

FG's 45 pound weight loss over four months prompted the treating physician to consult the CSO/RDN, who recommended nutrition support, optimally to begin before the second set of treatments commenced. The CSO/RDN discussed enteral nutrition (J-tube), peripheral parenteral nutrition (PPN), and Central Parenteral Nutrition (CPN) options with the physician, and CPN was recommended because of the risk for radiation enteritis and severe mucositis. FG already had single mediport placed for 5-FU delivery, however, a peripherally inserted central catheter (PICC) was chosen over double lumen for CPN administration, because Interventional Radiology noted the mediport is smaller and often becomes clogged.

Second Set of Chemotherapy/ Radiation Treatments (began 1/20/2014):

The second set of treatments included 25 radiation sessions and 5-FU (150 mg/m²/day x 5 days via single mediport). Because of

Table 3. Weight History

Date	Weight	Comments
Usual adult body weight	148 lb	x 1 year prior to gastric surgery consult
9/5/13	145 lb	Pre-operative weight
9/23/13	128 lb	17 lb weight loss since 9/5/2013 pre-op weight = 12% (severe) loss over 18 days immediately post-surgery
10/16/13	123 lb	22 lb weight loss since 9/5/2013 pre-op weight = 15% (severe) loss over 40 days post-surgery Nausea uncontrolled with Zofran TF consult ordered First set of treatments begin 10/16/2013
11/6/13	106 lb	39 lb weight loss since 9/5/2013 pre-op weight = 27% (severe) loss over 62 days post-surgery Poor symptom control with current regimen; nausea, diarrhea and mucositis continuing PO intake estimated ~ 500 kcal FG receiving loperamide and dexamethasone for diarrhea; additional symptom management strategies also discussed
12/18/13	104 lb	FG reports having difficulty balancing her intake – sometimes eating too much at meals, which causes abdominal pain/diarrhea (consistent with dumping syndrome)
1/23/14	90 lb	55 lb weight loss (severe) since 9/4/2014 = 38% (severe) loss over 79 days post-surgery FG begins CPN via PICC. Psychiatry consult ordered Second set of treatments begin 1/20/2014; CPN initiated 1/23/2014
2/12/14	97 lb	Physician states FG only able to eat a “bit;” endoscopy ordered to rule out stricture, ulcer, or other mechanical contributor to GI symptoms. No N/V at this time and FG reports motivation to eat more
2/26/14	98 lb	Surgeon notes inability to eat is psychological, but FG has not seen a psychiatrist and refuses follow up on recommended psychiatry referral. Surgeon recommends nutrition needs are: 1200 kcals and 50 g protein with goal weight of 110 lb., with daily post-gastrectomy vitamin/mineral supplement. However, dietitian adjusted energy and protein needs to: 1800 kcals and 67 g protein to meet estimated nutrition needs. FG indicates taste fatigue with protein drinks and is not drinking them at this time. RDN plans to cycle CPN down from 20 hours to allow more freedom with PO intake. FG has gained 8 lbs since beginning CPN
3/28/14	100 lb	FG reports “big” appetite and eating several small meals per day due to feeling full quickly. No nausea, vomiting, diarrhea, abdominal pain at this time. FG has yet to see psychiatrist. CPN cycled down to 14 hours – medical team tried to cycle CPN at 12 hours, however FG complained of nausea at this rate Third set of treatments begin 4/1/2014
4/11/14	98 lb	Mucositis resolved. FG reports being hungry all the time and eating “a lot”. CPN running 14 hours per day
4/22/14	99 lb	FG receiving first of three final chemotherapy cycles with 5-FU + oxaliplatin. Day 4 to 5 of chemotherapy, physician reports FG “not eating well, biting cheeks, and losing weight.” CPN running 14 hours/day. Per the physician, FG’s PO intake improving slowly. Lipids (for CPN) decreased to 3 times per week instead of daily, secondary to elevated liver function tests (LFTs). Per infusion company, fat calories will be replaced with glucose. FG indicates trying to eat protein bars in between meals. Physician advised FG to meet with CSO/RDN once chemotherapy is completed to focus on eating well. Per physician, will continue CPN until FG reaches goal weight of 110 lbs
5/13/14	101 lb	Physician states FG is eating well but slowly. CPN cycled down to 12 hours per FG’s request for more flexibility with schedule
6/3/14	101 lb	FG tolerating 12 hour cycle of CPN
Summary		Maximum weight loss = 55 lbs (= 38% of pre-op weight). After CPN was initiated, FG regained 7 lbs within 3 weeks and 11 lbs within 5 months (on CPN) Weight stabilized at 101 lbs / BMI 17.9 (underweight)

previous side effects, the dose of 5-FU was reduced by 25% for the second set of treatments. Chemoradiation treatment was paused for 1 week due to mucositis and diarrhea, and resumed at reduced rates. At one point during treatment, FG received 12.5% of the originally planned

chemotherapy dose. CPN was initiated on 1/23/2014. Because of the risk of refeeding syndrome associated with pre-existing malnutrition, and the extended period of poor intake, CPN was initiated slowly and cycled at 20 hours. Electrolytes (e.g. ,phosphorus and potassium) were monitored

during the initial three weeks on CPN to confirm that refeeding was not occurring (6). Once refeeding was ruled out, CPN was cycled down to 14 hours and provided 1.3 L, 3-1 custom bag providing 60 grams amino acids (AA), 200 grams Dextrose, and 25 grams

(Continued on page 7)

Table 4. Medications Influencing Nutrition Status

Medication	Dose	Purpose	Nutrition Impact Symptoms	Comments
Lexapro	20 mg PO qd	Antidepressant/ antianxiety	Increase weight and appetite, dry mouth, nausea	FG referred for psych consult, but FG does not follow up
Ativan	1 mg q6H prn	Antianxiety	Weight loss, increase appetite, fatigue	
Bactrim SS	PO qd	Antibiotic	Anorexia	
Emend (fosaprepitant)	150 mg	Antiemetic/ anti-nauseant (during chemotherapy)	Anorexia, abdominal pain, diarrhea, constipation, increase AST/ALT, BUN levels	
Dexamethasone (corticosteroid)	4 mg	Antinausea (up until treatment completion)	Increased weight, increased appetite, edema, Na/K retention, hypertension (HTN)	FG reported Zofran did not work for nausea
Optisource	qid	Post Bariatric Surgery Formula Chewable Vitamin & Mineral Supplement Tablet; chosen for best patient tolerance and to maximize absorption	100% DV for 22 vitamins and minerals from four doses	Per surgery dietitian, FG not always taking vitamin/mineral supplement due to upset stomach

Table 5. Biochemical Data

DATE:								
(2013-2014)	Reference Values (CU and SI)	9/12	10/25	11/20	12/18	2/1	4/22	5/13
Hct	0.42-0.52 x 100 (%)	26.7% ↓	30.8 ↓	34.9 ↓	35.1 ↓	34.6 ↓	32.0 ↓	31.7 ↓
Hgb	Female: CU: 12 - 15.6 g/dL SI: 120 - 156 g/L *conversion factor: 10	8.0 g/dL ↓	9.3 ↓	10.8 ↓	11.0 ↓	10.7 ↓	10.4 ↓	10.5 ↓
RBC	Female: 4.2 to 5.4 10e6/mcL	3.42 x 10e6/mcL ↓	3.90 ↓	4.13 ↓	3.91 ↓	3.49 ↓	3.36 ↓	3.30 ↓
WBC	4.5-10 x 10e3/mcL	13.52 x 10e3/mcL	0.8 ↓	2.0 ↓	3.1 ↓	2.1 ↓	2.2 ↓	2.7 ↓
BUN	CU: 6 - 20 mg/dL SI: 2.14-7.14 mmol/L *conversion factor 0.3571	3 mg/dL ↓	6	7	5 ↓	12	15	15
Creat, serum	Female: CU: 0.6 to 1.1 mg/dL SI: 53.04-97.24 μmol/L *conversion factor: 88.4	0.4 mg/dL ↓	0.49 ↓	0.54 ↓	0.53 ↓	0.37 ↓	0.41 ↓	0.41 ↓
Glucose	SI:3.8-5.5 mmol/L CU: mg/dL: 70-100 mg/dL *conversion factor 0.0555	78 mg/dL	100	97	87	106 ↑	89	91
Ca	SI: 2.12-2.55 mmol/L CU: 8.5 to 10.2 mg/dL *conversion factor 0.25	7.4 mg/dL ↓	9.3	10.1	9.7	9.7	8.6	8.5
K	SI:3.5-5.0 mmol/L CU: 3.5-5.0 mEq/L *conversion factor 1.0	4.2 mmol/L	3.9	5.1	4.2	4.4	4.0	4.4
Na	SI:136-145 mmol/L CU: 136-145 mEq/L *conversion factor 1.0	132 mmol/L ↓	144	146	147 ↑	141	141	141
ALT	SI: 25-65 U/L (only one unit used to measure ALT)		32 IU/L	15	21	23	57	39
AST	SI: 10-37 (only one unit is used to measure AST)	26 IU/L	22	34	34	62 ↑	28	

(Continued on next page)

Table 5. Biochemical Data continued

DATE: (2014)	Reference Values	1/20	2/10	2/17	3/12	3/28	4/1	4/15	5/27
PO4	SI:0.74-1.39 mmol/L CU: 2.29-4.3 mg/dL *conversion factor 0.323	4.3 mg/dL	4.3	4.5	3.9	3.9	2.9	3.9	4.0
Mg	SI:0.65-1.05 mmol/L CU: 1.3-2.1 mEq/L or 1.47-3.38 mg/dL *conversion factor 0.5 for mEq and 0.441 for mg/dL	1.8 mEq/L	2.0	2.0	1.8	1.9	1.7	1.8	1.9
DATE (2013- 2014):	Reference Values	10/25	11/20	1/20	2/10	3/28	4/18	5/27	
ANC	* Normal value: 1500 cells/mm ³ . Δ Mild neutropenia: 1000<1500/mm ³ . Ω Moderate neutropenia: 500 <1000/mm ³ . β Severe neutropenia: < 500/mm ³ .	35 β	800Ω	1900*	1300Δ	2600*	3200*	1500*	
DATE (2014):	Reference Value	2/17	3/12	3/28	4/1	4/15	4/29		
TG	Normal: <150 mg/dL SI: 1.7 mmol/L	88	60	82	52	64	82		
DATE (2013- 2014):	Reference Value	10/25	1/20	2/10	3/12	4/1	4/29	5/27	
PAB	18-38 mg/dL SI: 180-360 mg/L	16 mg/dL ↓	7 ↓	10 ↓	9 ↓	12 ↓	21	14 ↓	

intravenous fat emulsion (IVFE), with electrolytes and trace minerals. CPN provided 1170 calories and 60 grams protein (96% estimated energy needs and 100% of estimated protein needs); some additional energy and protein expected from oral intake. FG was instructed to remain on anti-dumping diet, and consume food as tolerated. The CSO/RDN provided recommendations for vitamin D supplementation via IV or CPN bag because FG was unable to tolerate an oral or sublingual supplement option. Per the surgeon's note (2/26/14), FG was eating fast food chicken and drinking fluids, without issue, while continuing on CPN, however this assessment was not confirmed by a CSO/RDN. FG weighed 90 lbs. when CPN was initiated; the goal was to continue CPN until FG's weight stabilized at 110 pounds.

History During the Third Set of Chemotherapy Treatments (began 4/1/2014):

The third and final set of treatments provided chemotherapy with 5-FU + oxaliplatin. Cycle 1 of the third set of treatments began with a 50% dose reduction for both chemotherapy agents. Cycle 2 began with a 50% dose

reduction of 5-FU and 25% dose reduction for oxaliplatin. The physician noted FG experienced fatigue, was biting her cheeks, was experiencing fingernail avulsion (loss of nails), and was not eating well. FG developed diarrhea and mouth sores during cycle 3. The maximum allowed dose of lomotil was ordered to control diarrhea. The PICC was removed due to rash and CPN was cycled for 12 hours per day via mediport. Though recommended at every follow-up visit, FG did not consult with a psychiatrist regarding psychological issues affecting eating, and other psychosocial issues.

Nutrition Diagnoses:

1. Malnutrition (10) related to (r/t) cancer treatment as evidenced by (AEB) nutrition impact symptoms of chemotherapy (e.g., mucositis and diarrhea), inadequate intake, and severe weight loss; >5% of weight loss within 1 month, and <75% of energy intake in >/_ 1 month
2. Inadequate intake r/t chemotherapy and radiation therapy AEB stated oral intake significantly below estimated needs; 38% weight loss x 3 ½ months

3. Altered GI function r/t surgery, 5-FU and XRT AEB diarrhea
4. Probable micronutrient insufficiency/ deficiency r/t diarrhea, AEB required use of "banana bag" 3 x/week to normalize electrolytes, and increased need for vitamin D supplementation

Nutrition Care Plan

1. PO diet as tolerated and modified to limit/avoid risk of dumping syndrome (associated with post-gastrectomy status) and to address treatment-related nutrition impact symptoms including diarrhea and mucositis.
2. CPN (started on 1/23/14 when FG weighed 90 lbs/41 kg); initiated at low rate due to risk of refeeding syndrome, and gradually increased to goal once refeeding had been ruled out (11).
3. CPN provided via central line in the chest. Infusion company RDN and Pharmacist managed CPN (i.e., recommended macronutrients, micronutrients, fluid needs; monitored electrolytes; adjusted CPN prescription as indicated per labs, weight, fluid

(Continued on next page)

status, and medical status). Goal included initial 20 hour/day infusion, cycled down to 14 hours/day, then cycled down to 12 hours/day. CPN via PICC provided 1.3 L, 3-1 custom bag (60 gm AA, 200 gm Dextrose, IVFE 25 gm), with electrolytes and trace minerals. CPN provided 1170 calories and 60 grams protein.

4. Per Home Solutions Pharmacist, FG was monitored for refeeding syndrome. When staff confirmed that refeeding was no longer a risk, CPN was gradually tapered to 14 hours/day (patient reported experiencing nausea with 12 hour/day infusion schedule). Due to elevated liver enzymes (3/28/14: AST 71, ALT 84), lipids in CPN were reduced (4/2014) from every day to three times per week. CPN via PICC provided 2-1 custom bag, 1.3 L (60 gm AA, 200 gm Dextrose), with electrolytes and trace minerals, for a 14 hour daily cycle. CPN provided 920 calories and 60 grams protein. IVFE at 25 gm.
5. PO Intake: Patient consuming PO diet primarily for pleasure and educated/ advised to consume, bland, low fiber, lean/low fat, high biological value protein foods.
6. Vitamin/mineral supplements: Patient receiving "banana bag" 3x/week in addition to micronutrients via CPN.
7. Because FG experienced significant anxiety r/t CPN, the RDN consulted with FG to explain the need for nutrition support during chemoradiation and reassure FG about CPN benefits.

Nutrition Recommendations: Based on body weight of 90 pounds (41 kg) when CPN was initiated; Adjusted IBW not used due to refeeding syndrome risk

1. Energy: 30-40 kcal/kg; 1225-1640 kcals
2. Protein: 1.2-1.5 g/kg; 49-62 g protein
3. Fluids: 1.5-1.8 Liter fluids/day (1 mL/ kcal)

Parenteral Nutrition Tolerance:

ALT, AST, T Bilirubin: Obtain baseline labs at initiation of CPN and continue to monitor

weekly in stable patients. Interventions for lab abnormalities: first investigate non-CPN-related reason for abnormalities; if CPN identified as cause, cycle PN, decrease dextrose, and limit fat to <1 gm/kg/day (14).

Nutrition Monitoring/Evaluation:

Indicator: CPN formula order

Criteria: CPN formula will meet current nutrition needs for macronutrients and micronutrients. Nutrient recommendations will be evidence-based and adequacy will be monitored via results of ongoing nutrition assessment including weight status.

Indicator: Refeeding syndrome

Criteria: To avoid refeeding syndrome, kcal intake will be gradually increased (14) and electrolytes will be maintained within reference ranges (i.e., maintain phosphorus, magnesium, and potassium levels). Electrolyte levels will be compared to institutional reference ranges. CPN will be adjusted, if indicated, to maintain electrolyte levels within reference ranges.

Indicator: PO intake adequacy

Criteria: PO intake will meet 60% energy/protein goals before FG is transitioned off of CPN.

Indicator: Body weight/composition

Criteria: Following initiation of CPN, goal is weight maintenance (due to higher risk of refeeding syndrome at CPN initiation), followed by gain; CPN was not ordered until FG experienced severe weight loss.

Final Outcome/Summary:

1. Weight stabilized after CPN was initiated and then gradually increased by 11 pounds
2. Creatinine level (below normal through 5/2014) suggests a loss of lean body mass secondary to severe weight loss. Recommended PO intake be consistently monitored when CPN is

tapered/discontinued, and continued medical nutrition intervention provided to address lingering symptoms and promote adequate PO intake.

3. Low prealbumin throughout treatment represented inflammatory status associated with treatment, and deemed a poor indicator of acute changes in nutrition status.
4. Electrolytes maintained within normal ranges once CPN began; refeeding avoided.
5. CPN appropriately adjusted per changing labs (e.g., lipid decreased when liver enzymes elevated).
6. Myelosuppression occurred secondary to treatment. CPN provided building blocks for repletion, which should occur when metabolic effects of treatments resolve.

Professional insight:

Barriers to providing effective nutrition care:

- Absence of systematic nutrition risk screening process prevented timely communication between outpatient center and inpatient RDN.
- Lack of dedicated CSO/RDN with specialized knowledge in oncology nutrition resulted in inadequate nutrition assessment, care, and follow-up.
- Nutrition Assessment by RDN was delayed until severe weight loss occurred and FG was malnourished, leaving FG in a "catch-up" situation. Proactive nutrition consult may have minimized nutrition impact symptoms and limited weight loss.
- During the first treatment cycle, nutrition recommendations were provided by non-CSO/RDN healthcare practitioners (i.e. Physician and Nurse Practitioner). These practitioners incorrectly assessed FG's intake, recommended energy and protein intake significantly below FG's nutrition needs, and did not develop a nutrition care plan, which resulted in inadequate intake, malnutrition, and the subsequent need for aggressive nutrition support.

- Initiation of CPN was delayed until severe malnutrition present, and refeeding became a risk factor for CPN.
- Ongoing weight loss and malnutrition resulted in dose reductions and treatment delays; total treatment provided was significantly less than planned.

Justification for CSO/RDN for Outpatient Cancer Center:

- CSO/RDNs understand the nutrition risks of each cancer type and its related therapies.
- CSO/RDNs are experts in oncology nutrition, and are knowledgeable about the nutrition needs of oncology patients and evidence-based interventions to address nutrition impact symptoms of oncology treatments.
- The majority of cancer patients now are receiving chemo- and radiation therapies as outpatients. As a result, inpatient RDNs who are not CSOs have limited experience with this population. If an outpatient CSO/RDN is not available, inpatient RDNs must find time to fit the outpatient into their schedule, and may need additional time to review current oncology nutrition literature to provide an effective intervention.
- Without a dedicated CSO/RDN present in the outpatient cancer center, staff often does not know how to communicate effectively with inpatient RDNs, thus delaying appropriate oncology nutrition care.
- Without having a dedicated CSO/RDN in an outpatient cancer center, physicians and other HCPs often make their own judgments and decisions regarding oncology nutrition care. Because they are not trained in this field, these decisions may be incomplete or inaccurate. For example, in FG's case, a dedicated CSO/RDN would have been invaluable, encouraging follow through and to reaffirm recommendations throughout treatment; consistent follow up would have identified problem areas and non-adherence to recommended nutrition interventions; this may have prevented severe weight loss and its associated sequelae, including reductions in treatment doses.
- A CSO/RDN dedicated to the oncology outpatient center would have the opportunity to educate physicians and the entire oncology center staff on oncology nutrition protocols to improve outcomes and quality of life for patients.
- A designated CSO/RDN could develop systematic protocols for various cancer types and provide appropriate oncology nutrition care for patients at low, moderate, and high risk of malnutrition.
- Absence of a dedicated CSO/RDN delayed initiation of nutrition support. There was no CSO/RDN available to routinely monitor FG's intake, and physician and nurse practitioner intake assessments were incomplete and inaccurate.
- A CSO/RDN would reinforce and encourage patients to consult with psychiatry (as recommended by physician), to address non-medically related eating behaviors that are a barrier to adequate intake. In FG's case, the CSO/RDN could have supported FG's positive eating behaviors and encouraged PO intake with effective and creative strategies to address ongoing symptom issues.

Nichole Giller, RD, CSO, LD is a clinical dietitian in Washington, DC.

References:

1. What you Need to Know About Stomach Cancer. NIH Publication No. 09-1554. U.S. Department of Health and Human Services. National Institute of Health, National Cancer Institute. Revised 2009, Accessed 7/30/2014: <http://www.cancer.gov/cancertopics/wyntk/stomach>.
2. How is stomach cancer staged? American Cancer Society. Last revised 5/20/2014. Accessed 7/30/2014: <http://www.cancer.org/cancer/stomachcancer/detailedguide/stomach-cancer-staging>.
3. Reference: The National Cancer Institute. *Cancer Staging*. Accessed: February 6, 2015: <http://www.cancer.gov/cancertopics/factsheet/detection/staging>

4. What are key statistics about stomach cancer? Stomach (Gastric) Cancer. American Cancer Society. Last revised 5/27/2014. Accessed 7/30/2014: <http://www.cancer.org/cancer/stomachcancer/detailedguide/stomach-cancer-key-statistics>.
5. American Cancer Society. Stomach Cancer. Accessed 7/30/2014: <http://www.cancer.org/cancer/stomachcancer/detailedguide/stomach-cancer-key-statistics>.
6. Survival rates for stomach cancer, by stage. American Cancer Society. Last revised 5/27/2014. Accessed 7/30/2014: <http://www.cancer.org/cancer/stomachcancer/detailedguide/stomach-cancer-survival-rates>.
7. Hamilton KK. Nutritional Needs of the Adult Oncology Patient. Chapter 4 in Leser ML, Trujillo E, Ledesma N, Bergerson S, eds. *Oncology Nutrition for Clinical Practice*. Oncology Nutrition DPG; 2013: pp 33–39.
8. Grant BG. Nutritional Effects of Cancer Treatment: Chemotherapy, Biotherapy, Hormone therapy and Radiation Therapy. Chapter 11 in Leser M, Trujillo E, Ledesma N, Bergerson S, eds. *Oncology Nutrition for Clinical Practice*. Oncology Nutrition DPG; 2013: pp 97–113.
9. Elliott L. Symptom Management of Cancer Therapies. Chapter 12 in Leser M, Trujillo E, Ledesma N, Bergerson S, eds. *Oncology Nutrition for Clinical Practice*. Oncology Nutrition DPG; 2013: pp 115–121.
10. Ryan JL, Heckler CE, Roscoe JA, Dakhil SR, Kirshner J, Flynn PJ, Hickok JT, Morrow GR, Ginger (Zingiber officinale) reduces acute chemotherapy-induced nausea: a URCC CCOP study of 576 patients. *Support Care Cancer*. 2012;20(7):1479–89.
11. Rithirangsiroj K, Manchana T, Akkayagorn L. Efficacy of acupuncture in prevention of delayed chemotherapy induced nausea and vomiting in gynecologic cancer patients. *Gynecol Oncol*. 2015;136(1):82–6.
12. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute. Common Terminology Criteria for Adverse Events (CTCAE). Version 4.0. Published May 28, 2009 (v r.03: June 2010). Accessed October 2, 2014: http://evs.nci.nih.gov/ftp1/CTCAE/CTCAE_4.03_2010-06-14_QuickReference_5x7.pdf
13. White J, Guenter P, Jensen G, Malone A, Schofield M. Consensus Statement: Academy of Nutrition and Dietetics and American Society of Enteral and Parenteral Nutrition: Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition). *J Acad Nutr Diet*. 2012;112(5):730–738.
14. Ryan A. Nutrition Support in the Oncology Setting. Chapter 13 in Leser M, Trujillo E, Ledesma N, Bergerson S, eds. *Oncology Nutrition for Clinical Practice*. Oncology Nutrition DPG; 2013: pp 123–133.

Low Microbial Diet in the Oncology Patient: What we know versus what we don't know

By Jennifer Caceres, MS, RD, LD

Introduction

Chemotherapy-induced neutropenia is a reality for oncology patients. Treatment for this population often places patients at higher risk for acquiring infections. Medical teams often recommend following a low bacteria diet during periods of acute neutropenia to reduce the risk of foodborne illness. While low bacteria diets have been around for more than five decades, for much of this time, research was not available to support this recommendation. Emerging research has not supported the efficacy of the diet in preventing foodborne illness either, and it has highlighted the marked variation of restrictions of a low bacteria diet within the oncology community (1).

From Sterile to Neutropenic

The use of the low bacteria diet in adult oncology patients can be traced back to the 1960s. During this time period, oncologists treated patients mainly on an inpatient basis. Medical teams tackled the high risk of infection by ensuring patients were treated in a clean and sterile environment. This not only focused on medical equipment and patient rooms (named "Life Islands"), but also addressed the risk of infection via nutritional intake. As part of the total protective environment the term "sterile diet" was born to refer to the food restrictions that oncology patients should follow. This specialized diet was far more restrictive than just limiting fresh fruits and vegetables (2). During this era of medicine and cancer treatment, foods were autoclaved and irradiated, often times leaving meals unpalatable and undesirable.

In an effort to increase quality of life and patient satisfaction, the National Cancer Institute performed a randomized trial of 21 patients with acute myelocytic leukemia (AML) in 1970. Participants were randomized to either the standard sterile diet, as described above, or a cooked food diet. The cooked food diet was designed by the Dietary and Environmental Sanitation Department of the National Institutes of

Health (NIH), and included most foods with the exception of uncooked foods and select dairy products. Standard practice at the time also included cleansing of the gastrointestinal tract with castor oil and soapsuds enemas, followed by sterilization of the gastrointestinal tract with antibiotics. Stools were cultured for aerobic bacterial, anaerobic bacterial, and fungal flora. The study analyzed the percentage of stools in which no organisms were cultured. There was no significant difference in the percentage of stools in which no organisms were cultured between the two groups, suggesting no benefit of a sterile diet over a cooked foods diet (3).

The first recommended standard for foods allowed as part of diets for neutropenic patients was published in a 1982 study. Pizzo et al examined the number of colony-forming units of bacteria found on various foods (4). Of 236 foods cultured, 66% grew less than 500 colony-forming units per gram of food. This became the upper-limit for foods acceptable for immunocompromised patients. 20% of processed meats and 30% of fresh fruits and vegetables cultured met this criterion. The diet which emerged from the results of this study is known today as the neutropenic diet. This diet is sometimes referred to as the low bacteria or low

microbial diet. Of note is that the diet was based simply on cultures grown from the surface of food, without consideration regarding pathogenicity of particular microbes to humans.

Inconsistencies Among Practice of Using the Neutropenic Diet

Criteria for implementation of the neutropenic diet, and the dietary restrictions imposed which constitute a low bacteria diet vary by institution, and also may vary amongst physicians within the same institution.

In 2000, a survey of 156 institutional members of the Association of Community Cancer Centers demonstrated 78% of institutions placed neutropenic patients on a low bacteria diet. Neutropenia was defined as absolute neutrophil count (ANC) less than 1,000/mm³ (μL) in 43% of the institutions, and ANC less than 500/mm³ (μL) in 46% of institutions, with the remainder of institutions indicating "other" for the threshold at which dietary restrictions were implemented. Of these, 83% of institutions enforced restrictions only while patients remained neutropenic, while the remaining 17% recommended a neutropenic diet throughout chemotherapy. The authors found that fresh fruits and vegetables, fruit juices, and raw eggs were the foods most often restricted (5).

In 2001, a smaller survey of seven hospitals in the United States and Canada found that most hospitals (5 out of 7) use low microbial diets for pediatric bone marrow transplant patients. Meal preparation methods for oncology patients varied by institution, with only two hospitals preparing meals in a separate kitchen. The survey revealed no consistency in the timing of initiating or discontinuing the diet (6).

A 2012 retrospective study examined implementation of the neutropenic diet (ND) versus a general hospital diet (GD) among hematopoietic stem cell transplant patients. Of the 726 consecutive patients considered, 50% received the ND, which restricted raw

fruits and vegetables, black pepper, raw/undercooked meats, fish and cheeses, unpasteurized dairy and miso products, raw grains, and brewer's yeast, and 50% were provided with the GD, which excluded undercooked meats, fish, and some unpasteurized dairy products. Patients on a ND remained on this diet until neutropenia resolved, at which time they were transitioned to a GD. Results indicate significantly fewer microbiological infections in the GD group ($P < 0.0272$), with diarrhea being more common in the ND group. Both groups had approximately the same length of stay. After neutropenia resolved, the ND group experienced a significantly higher rate of infection than the GD, specifically *C. difficile* and vancomycin-resistant *Enterococcus faecium* (VRE) infections (7).

Most recently, a 2014 survey provided insight into the uniformity of use of the neutropenic diet among pediatric facilities. An 18-question self-administered electronic survey was delivered to 1,639 pediatric oncologists at 198 member institutions of the Children's Oncology Group (COG). Thirty-four percent of physicians responded, representing 87% of the COG member institutions. Being a HSCT center was the only significant factor associated with implementing a neutropenic diet. Among physicians implementing the diet, 72% did so based on ANC, while 84% did so in preparation for a HSCT. There was variability among ANC threshold for when to initiate the diet, with the majority (86%) initiating at ANC less than $500/\mu\text{L}$. The most commonly restricted foods included fruits that cannot be peeled, raw vegetables, herbs, sprouts, and unpasteurized dairy. When analyzing consistency among physicians at the same center, researchers found that there was moderate agreement on which patient population to place on a neutropenic diet, and fair agreement on the diet's use in HSCT patients. Within the same centers physicians were not consistent, however, on when to initiate/discontinue the diet, or on what foods to restrict (8).

What we need to know?

The evidence in support of the continued use of a neutropenic diet is lacking, especially among oncology patients receiving chemotherapy or radiation. This practice, for which there is little evidence of benefit, seems to be rooted in a "better safe than sorry" theory. The goal of minimizing exposure or ingestion of bacterial and fungal contaminants is often cited by clinicians recommending a neutropenic diet, though the 2012 review suggests there may be downsides to a neutropenic diet, including increased risk of diarrhea and infection during and after HSCT.

There has yet to be a standardized definition of the neutropenic diet. Research has consistently revealed a lack of consistency for implementation of the neutropenic diet in the hospital setting as well, and variability among the populations for which the diet is recommended. Future research should focus on standardizing guidelines for initiation, implementation, and discontinuation of the neutropenic diet. This will allow for more systematic study of the efficacy, or lack thereof, of a neutropenic diet for improving outcomes in specific oncology populations.

Dietitian's Role

Dietitians are aware that following a restricted diet can place patients at risk of nutritional deficiencies. Specific to the neutropenic diet, if only cooked fruits and vegetables are permitted, this may contribute to decreased nutritional intake, and to nutrient losses due to the cooking processes (high cooking temperatures).

Dietitians have the responsibility of monitoring and evaluating the adequacy of nutritional intake of pediatric oncology patients. Understanding the risk of nutrient deficiencies, and the risk of reduced dietary intake secondary to lack of palatability of the neutropenic diet can prepare the dietitian to provide appropriate nutritional intervention in a timely manner.

It is truly our responsibility, as the nutrition experts, to continue to educate our medical teams regarding the evidence, or lack thereof, behind current-day nutrition practices. In order to ensure the best possible patient outcomes, we must challenge nutrition practices that do not have strong evidence supporting their use.

For more information about, or to join the Pediatric Sub Unit of the ONC DPG, please contact Katie Badgett, MS, RDN, CSP, LDN (Katie.Badgett@STJUDE.ORG).

Jennifer Caceres, MS, RD, LD, is a clinical dietitian at Nicklaus Children's Hospital in Miami, Florida.

References

1. Moody K, Finlay J, Mancuso C, Charlson M. Feasibility and safety of a pilot randomized trial of infection rate: neutropenic diet versus standard food safety guidelines. *J Pediatr Hematol Oncol*. 2006;28(3):126–33.
2. Fox N, Freifeld AG. The neutropenic diet reviewed: moving toward a safe food handling approach. *Oncology (Williston Park)*. 2012;26(6):572–75, 580, 582 passim.
3. Preisler HD, Goldstein IM, Henderson ES. Gastrointestinal "sterilization" in the treatment of patients with acute leukemia. *Cancer*. 1970;26(5):1076–81.
4. Pizzo PA, Purvis DS, Waters C. Microbiologic evaluation of food items. *J Am Diet Assoc*. 1982;81(3):272–79.
5. Smith LH, Besser SG. Dietary restrictions for patients with neutropenia: a survey of institutional practices. *Oncology Nursing Forum*. 2000; 27(3):515–20.
6. French MR, Levy-Milne R, Zibrik D. A survey of the use of low microbial diets in pediatric bone marrow transplant programs. *J Am Diet Assoc*. 2001; 101(10): 1194–98.
7. Trifilio S, Helenowski I, Giel M, Gobel B, Pi J, Greenber D, Mehta J. Questioning the role of neutropenic diet following hematopoietic stem cell transplantation. *Biology of Blood and Marrow Transplantation*. 2012; 18(9): 1385–90.
8. Braun LE, Chen H, Frangoul H. Significant inconsistency among pediatric oncologists in the use of the neutropenic diet. *Pediatric Blood Cancer*. 2014;61:1806–10.

Meet Your Newly Elected ON DPG Executive Committee Members

Chair-Elect:

Kelay Trentham, MS, RDN, CSO, CD



Kelay Trentham is a Registered Dietitian Nutritionist and is Board Certified as a Specialist in Oncology Nutrition. She has worked in oncology for 11 of her 18 years as a dietitian, starting

with the Seattle Cancer Care Alliance, and is now with MultiCare Regional Cancer Center in Tacoma, WA. Kelay has authored two chapters on palliative care nutrition for oncology nutrition textbooks, most recently for *Oncology Nutrition for Clinical Practice*. She has lectured for the University of Washington's graduate nutrition program and is Past Chair of MultiCare's Biomedical Ethics Committee. She is a member of the Vegetarian Nutrition Dietetic Practice group, has actively served as a member of the Oncology Nutrition Dietetic Practice Group's Executive Committee for the past 5 years, and is currently ON DPG's Chair-elect. Kelay received her M.S. in Foods and Nutrition from the University of Georgia in Athens, and makes her home on Vashon Island, WA.

Treasurer:

Caitlin Benda, MS, RDN, CSO, LDN



Caitlin Benda is a Registered Dietitian Nutritionist and is Board Certified as a Specialist in Oncology Nutrition. Her love for oncology patients was fostered during her outpatient

oncology rotation while in her dietetic internship at Tufts Medical Center – Frances Stern Nutrition Center in Boston, MA. After completing her internship, she moved to Charlotte, NC, where her passion for the oncology population grew while working as an inpatient dietitian, primarily with GI and head and neck cancer patients at Carolinas Medical Center. Shortly thereafter, she

began working at the Levine Cancer Institute as an outpatient dietitian; in this role, Caitlin had a positive impact on bringing nutrition to a chronically underserved population located in more rural areas of North Carolina. She is currently working as a Clinical Nutrition Manager within Carolinas HealthCare System, and while she misses working with oncology patients on a day-to-day basis, she is thrilled to continue following her passion for oncology by joining the ON DPG Executive Committee.

Eastern Area Representative:

Dianne Piepenburg, MS, RD, CSO, LD



Dianne Piepenburg has over fifteen years' experience working in Clinical Nutrition, with greater than five of those years working exclusively in the field of oncology.

She has presented

to numerous community organizations as well as to dietetic and health professionals at both the state and national level. She has been a guest lecturer to undergraduate and master's level nutrition students, and has been a part of the ON-DPG for over 5 years. Dianne has been published in the *Oncology Nutrition Connection*, and recently co-authored a chapter on Nutrition and Cancer Survivorship. While a recent move across the country has modified her current professional focus, she is also in the process of developing her own Oncology Nutrition consulting program in efforts to meet the needs of cancer patients in her local area.

Central Area Representative:

Anita Vincent, RD, CSO, LDN



Anita Vincent, a Registered Dietitian Nutritionist, has been working in oncology nutrition for nearly 17 years. As part of her work, she has grown Nutrition Services from a

part-time to full-time position, and continues to work to acquire additional staff, hopefully soon! Anita has developed and presented classes and programs for patients and families, authored newsletters and written blogs. This includes a wellness program for patients, which she would like to extend to clinic staff. She has presented to the local nursing and dietetic societies, as well as the community at large, the faith community, and patients and staff on cancer prevention, and the role of nutrition during all phases of cancer care. Anita started an oncology rotation for local dietetic interns. She works closely and collaboratively with her nursing staff, and couldn't do her job without them! Anita was recently awarded "Friend of Nursing" at her institution, and she is very pleased to receive this recognition.

Western Area Representative:

Paula Charuhas Macris, MS, RD, CSO, FAND, CD



Paula Charuhas Macris is a nutrition education coordinator and pediatric nutrition specialist at the Seattle Cancer Care Alliance in Seattle, WA. Her area of practice has been

in hematopoietic cell transplantation and nutrition support with a concentration in pediatrics. Paula has presented at local, national, and international conferences and has an extensive publication list of review articles, research papers, and book chapters related to oncology nutrition and hematopoietic cell transplantation. Paula has served on the ON DPG Executive Committee as the Continuing Education Chair, and is a peer reviewer for the *Oncology Nutrition Connection* ON DPG newsletter. She has been a contributing author to practice group publications, and was an invited presenter at the ON DPG 2014 Symposium in Orlando, FL. Paula is a recipient of the Academy of Nutrition and Dietetics Excellence in Clinical Nutrition Practice Award.

CPE Article: Soyfoods in the Diets of Women With Breast Cancer

By Mark Messina, PhD, MS

Reprinted with permission from the United Soybean Board. This CPE article first appeared in the Winter 2015 - Vol 23, No 1 Issue of The Soy Connection (<http://www.soyconnection.com/newsletters/soy-connection/health-nutrition/soyfoods-diets-women-breast-cancer>)

Note from the Editor:

I first encountered this article at the 2014 American Institute for Cancer Research Conference. Dr. Messina was a guest at the United Soybean Board booth, and was sharing the article through this venue. He had written it very recently, in part to address questions raised by the publication of research by Shike et al., "The effects of soy supplementation on gene expression in breast cancer: a randomized placebo-controlled study." I had been considering how to respond to this research myself, because it had re-ignited the fear and anxiety many women affected by breast cancer have around soy. There were many things about the Shike, et al. study that concerned me, and being familiar with the long history of cell, animal, and human observational and interventional research, I knew the study results could easily be misread to mean that women with breast cancer should not eat soy foods at all. When I came across Dr. Messina's article, I realized it crystallized the key issues I'd been considering, and better yet, was a coherent synthesis of my less-organized thoughts on the topic! I just knew I had to share Dr. Messina's work with the ON DPG readership, because we all face these tough questions every day in our work. I can't think of another issue, save perhaps the perennially recurring "does sugar feed cancer?," that has such persistence as a nutritional concern for cancer patients, particularly those affected by breast cancer. With permission from the United Soybean Board, I am pleased to share this article and its short companion piece on cancer organizations' positions on the use of soy foods by women with breast cancer for this issue's CPE article.

The impact of soyfood intake on the prognosis of women with breast cancer has been the subject of rigorous investigation for nearly two decades (1) — almost as long as the role of soy in breast cancer prevention has been the focus of attention (2). In both cases the research has been undertaken primarily because of the phytoestrogens or isoflavones in soybeans.

Current evidence suggests that the consumption of soy must occur early in life, that is, in childhood or during the teenage years to reduce the risk of breast cancer (3,4). Animal and epidemiologic data are supportive of this hypothesis and there exist several mechanisms to explain the proposed protective effect. This notion that

early soy intake reduces breast cancer risk is starting to be embraced by the scientific community. However, clinical data (which may be impossible to generate) are not available, so this hypothesis will likely remain speculative (5,6). Nevertheless, it is reasonable to recommend that girls consume one serving of soyfoods per day because this amount has been associated with protective effects in epidemiologic studies.

Given the historically low breast cancer incidence rates in countries that consume soyfoods (7), and that in those countries, the prognosis of women with this disease is at least as good as women from Western countries (8), the concern that soyfoods

might be harmful to breast cancer patients has a less than obvious basis. On the other hand, soyfoods are uniquely-rich sources of phytoestrogens (9) and estrogen therapy *may* increase breast cancer risk and worsen the prognosis of patients with some types of breast cancer (10). Furthermore, despite a plethora of clinical studies showing isoflavones and estrogen affect a variety of endpoints differently, rodent work beginning in the late 1990s, shows consistently that isoflavones, and in particular, genistein (the predominant isoflavone in soybeans), stimulate the growth of existing estrogen-sensitive mammary tumors in mice implanted with estrogen receptor-positive (ER+) human breast cancer cells (11).

The mice in these studies lack an immune system, have had their ovaries surgically removed, develop mammary tumors outside of the mammary gland, and metabolize isoflavones differently than humans. Therefore, considerable grounds exist for questioning the utility of this model for predicting effects in women. Nevertheless, without clinical and/or epidemiologic data to the contrary, prudence dictates that at the very least, the findings of these rodent studies be strongly considered when making dietary recommendations. Consequently, until recently, many oncologists and oncology dietitians recommended that women with breast cancer avoid or at least severely limit soyfood consumption. However, this position is no longer consistent with the totality of the data. As discussed, clinical research shows neither soyfoods nor isoflavones adversely affect markers of breast cancer risk, and epidemiologic studies show that post-diagnosis soy intake *reduces* recurrence and breast cancer mortality.

Clinical Research

Not unexpectedly, no intervention studies have evaluated the impact of soyfood or isoflavone intake on the prognosis of breast cancer patients. Thus, definitive data are not available. However, an abundance of studies have evaluated the impact of soy on markers of breast cancer risk including reproductive

(Continued on next page)

hormone levels, mammographic density, and breast cell proliferation (12,13). These studies, which mostly intervene with isolated soy protein or soy extracts, consistently show that isoflavone exposure does not adversely affect breast tissue and breast cancer risk. These data provide reassurance about the safety of soyfoods, especially when considering the dose employed in many of these studies greatly exceeds typical Japanese isoflavone intake (~40 mg/d). The cell proliferation studies, which require taking biopsies before and after exposure to soy, are particularly revealing.

However, recent findings from a study published in the *Journal of the National Cancer Institute* (JNCI), which gained considerable media attention, led the authors to issue cautionary, although measured, statements about the use of soyfoods by breast cancer patients (14). But a close look at the results shows they are actually consistent with the existing clinical data. For approximately two weeks prior to surgical removal of their breast tumor, women consumed either 51.6 g of milk protein daily or a similar amount of soy protein that provided 103 mg of isoflavones, 62 mg of which were genistein. This amount of genistein is provided by approximately five servings of soyfoods.

Soy protein intake did not affect the primary outcomes of this study, which were breast cell proliferation and apoptosis (programmed cell death). However, in the 20% of women with the highest blood genistein levels, there were changes in the expression of genes that are associated with increased cell proliferation. The authors suggested cell proliferation was unaffected despite the gene expression changes, because the intervention period was too short. However, three longer-term intervention studies, which were three months (15), six months (16) and one year in duration (17), also failed to show proliferative effects. Thus, short duration is not an explanation for the lack of response. Two other studies (18,19) also found soy was without proliferative effects, although the duration of these was similar to that of the trial by Shike et al. published in the JNCI (14).

Thus, all six intervention studies which examined in vivo breast cell proliferation have found soy and isoflavones are without effect.

Interestingly, this recent study in the *JNCI* is not the first clinical study to show isoflavones affect gene expression in a manner consistent with an estrogenic and mild proliferative effect, as the findings from two other intervention studies did likewise (16,18). But like the *JNCI* study, neither of these studies observed increases in cell proliferation. The expression of hundreds of genes is affected by isoflavones (and probably most biologically active compounds) but the vast majority (>90%), according to a recent study, are not associated with estrogen (20). Monitoring changes in gene expression is a useful endeavor, but clinical outcomes are ultimately the most relevant.

Prospective Epidemiologic Studies

Extensive prospective data have been published since 2009 showing that soy is not only safe for breast cancer patients, but actually beneficial. Because it is essentially impossible to completely control for all potentially confounding variables, findings from epidemiologic studies do not allow cause and effect relationships to be definitively established. On the other hand, epidemiologic findings provide much of the basis for the development of dietary guidelines.

Most relevant with respect to the prospective data is a pooled analysis of three studies (21), two from the United States (22,23) and one from China (Shanghai) (24). The 9,514 women with breast cancer participating in these studies were followed for an average of 7.4 years during which time there were 1,171 deaths from all causes, 881 breast cancer-specific deaths, and 1,348 recurrences. Approximately half of the women were premenopausal and half postmenopausal, and approximately half were Chinese and half Caucasian. In Shanghai, isoflavone intake occurs primarily via the consumption of soymilk and tofu and tofu products (24).

After adjustment for 17 variables, isoflavone intake was found to be associated with a marked improvement in prognosis. More specifically, when comparing women in the third isoflavone intake group with those in the first, risk of overall mortality, breast cancer-specific mortality and tumor recurrence were reduced by 13%, 17% and 25%, respectively, with the latter finding being statistically significant (21). Isoflavone intake was associated with similar benefits in Chinese and US women, ER+ and ER- patients, and in users and non-users of tamoxifen. In fact, these results suggested that recurrence was reduced more in users than non-users of this breast cancer drug. This finding, like the findings overall, are directly opposite to those reported in the previously discussed mouse model (25,26).

A meta-analysis involving over 11,000 breast cancer patients comprised of the three studies in the pooled analysis noted above plus two additional small Chinese prospective studies, reported similar benefits, although in this case breast cancer mortality was also statistically and significantly reduced (27). The authors of this analysis recommended that women with breast cancer consume soyfoods. Interestingly, one of the two additional Chinese studies found that soy intake was associated with an enhanced efficacy of anastrozole, an aromatase inhibitor used to treat breast cancer (28). As in the case of tamoxifen, this finding directly contradicts the findings in mice (29).

Mechanisms

Since the clinical studies show that isoflavone exposure has no effect on cell proliferation, apoptosis or other markers of risk such as mammographic density, there is no obvious mechanistic explanation for the beneficial effects of post-diagnosis soy intake reported in the prospective studies. It may be that protective effects occur via mechanisms that are not detected by changes in routinely used markers such as angiogenesis inhibition.

Noteworthy in this regard, is the ability of genistein to inhibit metastases in an animal

model (30). Also, a three-year clinical trial involving osteopenic postmenopausal women showed that BRCA1 and BRCA2 (genes that produce proteins that repair DNA damage) mRNA levels were unchanged in the group taking genistein (54 mg/d), whereas levels were decreased in the placebo group (31). Interestingly, there is some indication that at least among breast cancer patients, soy acts as an estrogen antagonist (32), although it would not appear that the benefits associated with isoflavone intake could be due entirely to an anti-estrogenic mechanism since, as mentioned in the pooled analysis, isoflavone intake was associated with benefits in both ER+ and ER- breast cancer.

Summary and Conclusions

For women with breast cancer, the clinical data support the safety of soyfoods and the epidemiologic data are supportive of benefit. Since even in the mouse studies, *whole* soyfoods do not stimulate tumor growth, there is essentially no meaningful evidence justifying a recommendation that breast cancer patients avoid traditional Asian soyfoods (33). Furthermore, since many women with breast cancer will live for many years without succumbing to their disease, focus needs to be not just on the impact of diet on breast cancer survival but on overall health (34). Cardiovascular disease (CVD) remains the number one killer among women and substantial clinical (35,36) and epidemiologic (37,38) evidence indicates that soyfoods reduce CVD risk. Thus, while the consumption of no single food is essential to health, there certainly are reasons for breast cancer patients to consume soyfoods. The consumption of two servings per day is consistent with both the clinical and epidemiologic findings.

References:

- Santell RC, Chang YC, Nair MG, Helferich WG. Dietary genistein exerts estrogenic effects upon the uterus, mammary gland and the hypothalamic/pituitary axis in rats. *J Nutr*. 1997;127(2):263–69.
- Messina M, Barnes S. The role of soy products in reducing risk of cancer. *J Natl Cancer Inst*. 1991;83(8):541–46.
- Messina M, Hilakivi-Clarke L. Early intake appears to be the key to the proposed protective effects of soy intake against breast cancer. *Nutr Cancer*. 2009;61(6):792–98.
- Messina M, Wu AH. Perspectives on the soy-breast cancer relation. *Am J Clin Nutr*. 2009;89(5):1673S–79S.
- Mahabir S. Association between diet during preadolescence and adolescence and risk for breast cancer during adulthood. *J Adolesc Health*. 2013;52(5 Suppl):S30–5.
- Gonzales JF, Barnard ND, Jenkins DJ, Lanou AJ, Davis B, Saxe G, Levin S. Applying the precautionary principle to nutrition and cancer. *J Am Coll Nutr*. 2014;33(3):239–46.
- Pisani P, Bray F, Parkin DM. Estimates of the world-wide prevalence of cancer for 25 sites in the adult population. *Int J Cancer*. 2002; 97(1):72–81.
- Yonemoto RH. Breast cancer in Japan and United States: epidemiology, hormone receptors, pathology, and survival. *Arch Surg*. 1980;115(9):1056–62.
- Franke AA, Custer LJ, Wang W, Shi CY. HPLC analysis of isoflavonoids and other phenolic agents from foods and from human fluids. *Proc Soc Exp Biol Med*. 1998;217(3):263–73.
- Eden JA. Why does oestrogen-only hormone therapy have such a small impact on breast cancer risk? A hypothesis. *Gynecol Endocrinol*. 2011;27(3):170–75.
- Helferich WG, Andrade JE, Hoagland MS. Phytoestrogens and breast cancer: a complex story. *Inflammopharmacology*. 2008;16(5):219–26.
- Messina M, Caan BJA, D.L., Hardy MM, G. It's time for clinicians to reconsider their proscription against the use of soyfoods by breast cancer patients. *Oncology*. 2013; 27(5):430–37.
- Fritz H, Seely D, Flower G, Skidmore B, Fernandes R, Vadeboncoeur, et al. Soy, red clover, and isoflavones and breast cancer: a systematic review. *PLoS One*. 2013; 8(11):e81968.
- Shike M, Doane AS, Russo L, Cabal R, Reis-Filho JS, Gerald W, et al. The effects of soy supplementation on gene expression in breast cancer: a randomized placebo-controlled study. *J Natl Cancer Inst*. 2014; 106(9). pii: dju 189.
- Cheng G, Wilczek B, Warner M, Gustafsson JA, Landgren BM. Isoflavone treatment for acute menopausal symptoms. *Menopause*. 2007;14(3 Pt 1):468–73.
- Khan SA, Chatterton RT, Michel N, Bryk M, Lee O, Ivancic D, et al. Soy isoflavone supplementation for breast cancer risk reduction: A randomized phase II trial. *Cancer Prev Res (Phila)*. 2012;5(2):309–19.
- Palomares MR, Hopper L, Goldstein L, Lehman CD, Storer BE, Gralow JR. Effect of soy isoflavones on breast proliferation in postmenopausal breast cancer survivors. *Breast Cancer Res Treatment*. 2004;88 (Suppl 1):4002 (Abstract).
- Hargreaves DF, Potten CS, Harding C, Shaw LE, Morton MS, Roberts SA, et al. Two-week dietary soy supplementation has an estrogenic effect on normal premenopausal breast. *J Clin Endocrinol Metab*. 1999; 84(11):4017–24.
- Sartippour MR, Rao JY, Apple S, Wu D, Henning S, Wang H, et al. A pilot clinical study of short-term isoflavone supplements in breast cancer patients. *Nutr Cancer*. 2004;49(1):59–65.
- van der Velpen V, Geelen A, Hollman P, Schouten EG, van't Veer P, Afman LA. Isoflavone supplement composition and equol producer status affect gene expression in adipose tissue: a double-blind, randomized, placebo-controlled crossover trial in postmenopausal women. *Am J Clin Nutr*. 2014;100(5):1269–77.
- Nechuta SJ, Caan BJ, Chen WY, Lu W, Chen Z, Swan ML, et al. Soy food intake after diagnosis of breast cancer and survival: an in-depth analysis of combined evidence from cohort studies of US and Chinese women. *Am J Clin Nutr*. 2012;96(1):123–32.
- Caan BJ, Natarajan L, Parker B, Gold EB, Thomson C, Newman V, et al. Soy food consumption and breast cancer prognosis. *Cancer Epidemiol Biomarkers Prev*. 2011; 20(5):854–58.
- Guha N, Kwan ML, Quesenberry CP, Jr., Weltzien EK, Castillo AL, Caan BJ. Soy isoflavones and risk of cancer recurrence in a cohort of breast cancer survivors: the Life After Cancer Epidemiology study. *Breast Cancer Res Treat*. 2009;118(2):395–405.
- Shu XO, Zheng Y, Cai H, Gu K, Chen Z, Zheng W, et al. Soy food intake and breast cancer survival. *JAMA*. 2009;302(22):2437–43.
- Du M, Yang X, Hartman JA, Cooke PS, Doerge DR, Ju YH, Helferich WG. Low-dose dietary genistein negates the therapeutic effect of tamoxifen in athymic nude mice. *Carcinogenesis*. 2012;33(4):895–901.
- Ju YH, Doerge DR, Allred KF, Allred CD, Helferich WG. Dietary genistein negates the inhibitory effect of tamoxifen on growth of estrogen-dependent human breast cancer (MCF-7) cells implanted in athymic mice. *Cancer Res*. 2002;62(9):2474–77.
- Chi F, Wu R, Zeng YC, Xing R, Liu Y, Xu ZG. Post-diagnosis soy food intake and breast cancer survival: A meta-analysis of cohort studies. *Asian Pac J Cancer Prev*. 2013; 14(4):2407–12.
- Kang X, Zhang Q, Wang S, Huang X, Jin S. Effect of soy isoflavones on breast cancer recurrence and death for patients receiving adjuvant endocrine therapy. *CMAJ*. 2010; 182(17):1857–62.
- Ju YH, Doerge DR, Woodling KA, Hartman JA, Kwak J, Helferich WG. Dietary genistein negates the inhibitory effect of letrozole on the growth of aromatase-expressing estrogen-dependent human breast cancer cells (MCF-7Ca) in vivo. *Carcinogenesis*. 2008;29(11):2162–68.
- Lakshman M, Xu L, Ananthanarayanan V, Cooper J, Takimoto CH, Helenowski I, et al. Dietary genistein inhibits metastasis of human prostate cancer in mice. *Cancer Res*. 2008;68(6):2024–32.

(Continued on next page)

31. Marini H, Bitto A, Altavilla D, Burnett BP, Polito F, Di Stefano V, et al. Breast safety and efficacy of genistein aglycone for postmenopausal bone loss: a follow-up study. *J Clin Endocrinol Metab*. 2008; 93(12):4787–96.
32. Dorjgochoo T, Gu K, Zheng Y, Kallianpur A, Chen Z, Zheng W, et al. Soy intake in association with menopausal symptoms during the first 6 and 36 months after breast cancer diagnosis. *Breast Cancer Res Treat*. 2011;130(3):879–89.
33. Allred CD, Allred KF, Ju YH, Goepfing TS, Doerge DR, Helferich WG. Soy processing influences growth of estrogen-dependent breast cancer tumors. *Carcinogenesis*. 2004;25(9):1649–57.
34. Izano MA, Fung TT, Chiuve SS, Hu FB, Holmes MD. Are diet quality scores after breast cancer diagnosis associated with improved breast cancer survival? *Nutr Cancer*. 2013; 65(6):820–6.
35. Li SH, Liu XX, Bai YY, Wang XJ, Sun K, Chen JZ, et al. Effect of oral isoflavone supplementation on vascular endothelial function in postmenopausal women: a meta-analysis of randomized placebo-controlled trials. *Am J Clin Nutr*. 2010;91(2):480–86.
36. Jenkins DJ, Mirrahimi A, Srichaikul K, Berryman CE, Wang L, Carleton A, et al. Soy protein reduces serum cholesterol by both intrinsic and food displacement mechanisms. *J Nutr*. 2010;140(12):2302S–11S.
37. Kokubo Y, Iso H, Ishihara J, Okada K, Inoue M, Tsugane S. Association of dietary intake of soy, beans, and isoflavones with risk of cerebral and myocardial infarctions in Japanese populations: the Japan Public Health Center-based (JPHC) study cohort I. *Circulation*. 2007;116(22):2553–62.
38. Zhang X, Shu XO, Gao YT, Yang G, Li Q, Li H, et al. Soy food consumption is associated with lower risk of coronary heart disease in Chinese women. *J Nutr*. 2003;133(9):2874–78.

CPE Article: Cancer Organizations Support the Use of Soyfoods by Women With Breast Cancer

By Mark Messina, PhD, MS

Reprinted with permission from the United Soybean Board. This CPE article first appeared in the Winter 2015 - Vol 23, No 1 Issue of The Soy Connection (<http://www.soyconnection.com/newsletters/soy-connection/health-nutrition/cancer-organizations-support-use-soyfoods-women-breast>).

Three analyses of the scientific literature by prestigious organizations have evaluated the impact of soy intake on the prognosis of women with breast cancer. On the basis of their assessments, the scientific consensus has gone from one urging breast cancer patients to be cautious about consuming soyfoods to cautiously concluding that doing so may be beneficial.

Since a controversy about the safety of soyfood use by breast cancer patients first erupted in the late 1990s, oncology dietitians and oncologists have been in the rather unenviable position of having to provide guidance about the use of such foods. This task was not easy given that for many years there were only very limited data upon which to base recommendations. Until fairly recently, no sanctioned health body had offered position statements on soy and the breast cancer patient. This void has now been filled.

In 2006, Doyle et al. (1), writing on behalf of the Nutrition, Physical Activity and Cancer Survivorship Advisory Committee of the American Cancer Society (ACS), concluded that “current epidemiologic and laboratory evidence suggests there are unlikely to be harmful effects when soy is provided in the diet consistent with amounts in a typical Asian diet ... This amount would be provided by as many as three servings per day of soy foods, such as tofu and soy milk.”

These conclusions provided guidance to those counseling patients, although the characterization of typical “Asian intake” by the ACS is a bit inaccurate. Even in Shanghai

(there is a wide range of intake among geographic regions in China) and Japan, which represent the highest soyfood-consuming populations, mean intake is no more than two servings per day, although women in the upper 20% of intake are consuming approximately three servings (2).

Not surprisingly, the ACS position has frequently been cited in support of the safety of soyfoods. The many co-authors of the ACS position paper are internationally recognized experts but their review of the literature was not as rigorous as one might assume because soy was simply one small part of a much larger review. Only one page of the 25-page document was devoted to soy and only four of the 244 references cited were soy-related.

In 2012, the ACS reaffirmed its previous conclusion stating that “for the breast cancer survivor, current evidence suggests no adverse effects on recurrence or survival from consuming soy and soy foods, and there is the potential for these foods to exert a positive synergistic effect with tamoxifen” (3). Although the scope of this review was similar to that in 2006, the more recent review had the advantage of

considering three prospective studies that evaluated the impact of post-diagnosis soy intake on breast cancer prognosis, all of which showed benefit.

Also in 2012, the American Institute for Cancer Research (AICR) concluded much the same as the ACS about soy and women with breast cancer (4). According to Karen Collins, MS, RD, CDN, a nutrition advisor to the AICR "... now we know it is safe — the evidence is so consistent." In its review of the data, the AICR noted: "Six recent human studies and one major meta-analysis have found that consuming moderate amounts of soy foods does not increase a breast cancer survivor's risk of recurrence or death." It further stated that "a small number of studies even suggest soy foods may be most protective for women who take tamoxifen or an aromatase inhibitor but more research is needed." Unfortunately, the AICR did not publish its findings in a peer-reviewed journal, but merely released short summaries online.

Finally, and most importantly, in 2014 the World Cancer Research Fund International (WCRFI) issued its perspective study on soy and the breast cancer patient as part of the Continuous Update Project (CUP), which analyzes global cancer prevention and survival research linked to diet, nutrition, physical activity and weight (5). The WCRFI represents the AICR and the World Cancer Research Fund in England, the Netherlands, and Japan. The CUP included 85 studies involving 206,988 deaths.

In regard to soy, the CUP identified three cohort studies evaluating the association between isoflavone intake, 12 months or more after a diagnosis of primary breast cancer and all-cause mortality, and two evaluating the association between soy protein intake and all-cause mortality. According to the WCRFI, "The evidence was sparse and generally consistent, and is suggestive of an inverse relationship between consumption of foods containing soy and all-cause mortality."

Importantly, however, the CUP's independent panel of scientists concluded that because of limitations in either the design or execution of much of the research that exists, the evidence is still not strong enough to make any specific recommendations for breast cancer survivors. Nevertheless, they noted that there are indications of links between better survival after breast cancer and maintaining a healthy body weight, being physically active, eating foods containing fiber, eating foods containing soy, and a lower intake of total fat (and, in particular, saturated fat).

Mark Messina, PhD, is the co-owner of Nutrition Matters, Inc., a nutrition consulting company, and is an adjunct professor at Loma Linda University, California. His research focuses on the health effects of soyfoods and soybean components. He is chairman of The Soy Connection Editorial Board and executive director of the Soy Nutrition Institute.

References:

1. Doyle C, Kushi LH, Byers T, Courneya KS, Demark-Wahnefried W, Grant B, et al. Nutrition and physical activity during and after cancer treatment: an American Cancer Society guide for informed choices. *CA Cancer J Clin.* 2006;56(6):323–53.
2. Messina M, Nagata C, Wu AH. Estimated Asian adult soy protein and isoflavone intakes. *Nutr Cancer.* 2006;55(1):1–12.
3. Rock CL, Doyle C, Demark-Wahnefried W, Meyerhardt J, Courneya KS, Schwartz AL, et al. Nutrition and physical activity guidelines for cancer survivors. *CA Cancer J Clin.* 2012;62(4):242–74.
4. American Institute for Cancer Research. Soy is safe for breast cancer survivors. http://www.aicr.org/cancer-research-update/november_21_2012/cru-soy-safehtml (accessed February 5, 2013) 2012.
5. World Cancer Research Fund International. Continuous Update Project Report: Diet, Nutrition, Physical Activity, and Breast Cancer Survivors. 2014. Available at: www.wcrf.org/sites/default/files/Breast-Cancer-Survivors-2014-Report.pdf.

FNCE 2015!

MARK YOUR CALENDARS FOR FNCE 2015!

Sunday, October 4, 2015

- **7:00-8:30 AM – Oncology DPG Breakfast Reception**
- **Place – Omni Hotel, Music Row 5**

Breakfast provided for *FREE* to all ON DPG members. We will send you a notification to RSVP soon via the EML, e-blast, etc. Join us as we network with other dietitians who specialize in oncology nutrition and earn CEUs. Topic during breakfast TBD.

Marijuana: Is it Medicine Yet for Cancer Symptom Management?

Featuring Dr. Donald Abrams and Kelay Trentham, MS, RD, CSO, CD

Sunday, October 4, 2015

- **1:30-3:00 PM – Oncology DPG Spotlight Session**
- **Place – Music City Center/ Room: Exhibit Hall D**

Join us as we learn the mechanisms through which cannabis exerts physiologic effects, discuss the potential use of cannabis in cancer symptom management, and review variable effects of different cannabis strains, cannabinoid constituents, and modes of delivery. By the end of this session, you will be empowered to apply principles of bioethics to your discussion with patients regarding medical cannabis use.

An Innovative Student Project: Impact of Diet on the Risk of Developing Stomach Cancer

A Year 12 Project

By Alanah Varricchio

Note from the Editor:

In early 2014, I was contacted by Alanah Varricchio, a student at Nazareth Catholic Community (NCC), which is located in South Australia. NCC is comprised of an R-12 College, Early Childhood Center and Integrated Services. R-12 refers to the school being a combined primary and secondary education center, covering “birth to year 12 education.” Alanah was a year 12 student, in her last year at NCC, when she reached out to me for a project she was completing on nutrition and stomach cancer. Our connection began with my reply to her email, “I received your inquiry about stomach cancer prevention diets through the ON DPG website. If you want to send me your questions, I’d be happy to help you out with your project.” I had no idea what a treat it would be to work with Alanah, even for our brief interactions. She enthusiastically dove into our email “conversation,” asking astute questions about what we do and do not know about the connection between nutrition and stomach cancer. She inquired about how dietitians manage the significant nutrition challenges faced by individuals affected by stomach cancer. At the completion of our emailing, when all of her questions were answered to her satisfaction, Alanah promised to send me a copy of her final project report. When I received it, I couldn’t believe the report had been written by a high school student! I have taught both undergraduate and graduate level nutrition coursework, and Alanah’s paper surpassed much of the work I have received from college-enrolled students. I am pleased to share this project with you, because it represents the incredible quality of work that can be produced by intelligent students who are held to a high learning standard.

I hope this article will give you ideas for projects for future students and interns, including the occasional high school student with whom you have the pleasure of working. As mentors and nutrition professionals, we should never underestimate our students and mentees. Many of them are much more capable than we even realize!

Please note original British/Australian spellings are maintained throughout the article.

Introduction

Cancer is estimated to be the leading cause of disease burden in Australia (1) with many possible risk factors including poor diets (2). Stomach cancer is the fifth most common cancer in the world. The 5-year survival rate for Australians with stomach cancer is 25% (3), making its prevention very important.

The stomach is a muscular organ that receives food from the oesophagus and commences digestion. Most stomach cancers develop in the mucosa, the primary layer of the stomach (3).

There are several risk factors that influence stomach cancer risk. This research project investigated the chemistry of certain foods and their impacts on stomach cancer risk. It

involved interviewing a smallgoods producer and five dietitians, each with different specialty areas, including nutrition for diagnosed stomach cancer patients. Various online articles and medical journals were accessed and provided professional knowledge about the risk factors of stomach cancer. A major component of this project was an experiment that tested nitrate quantities in a range of processed and home-cooked meats and raw vegetables. This material was utilized to create meal alternatives to meals that may increase risk of stomach cancer.

What impact does the consumption of plant-based foods have on the risk of developing stomach cancer?

Evidence shows that the benefits of plant-based foods are abundant, particularly

when referring to preventing diseases such as stomach cancer (5). Plant-based foods contain fibre which is essential for cleansing the body of toxins that can accumulate in the bowel and affect cells, potentially promoting stomach cancer development (6). According to the Australian Guide to Healthy Eating (AGHE), adults require 25g of fibre, 300g of fruit and 375g of vegetables (7) daily. These quantities could potentially reduce stomach cancer risk.

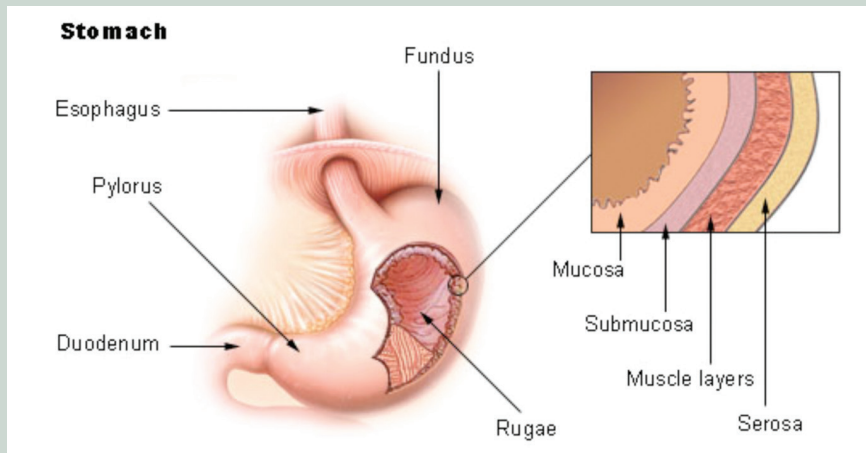
Some experts suggest adhering to a vegan diet in order to reduce cancer risk (8). Animal products lack fibre (9) and many antioxidants, and are therefore less effective in reducing stomach cancer risk.

Epidemiologist and Registered Dietician, Suzanne Dixon, MPH, MS, RDN, states that “The best foods for reducing stomach cancer risk are brightly coloured fresh fruit and vegetables” (10). The varying colours of fruits and vegetables represent the vast variety of phytonutrients they contain, including antioxidants and polyphenols. Antioxidants are compounds in foods that neutralise free radicals produced by oxidation in the human body (11). Free radicals are unstable molecules that form when oxygen metabolises during oxidation. They steal electrons from other molecules, causing DNA damage (12), thus increasing stomach cancer risk (13). Polyphenols also can act as antioxidants, and have additional properties that appear to contribute to cancer risk-reducing effects, including the ability to favourably regulate cell growth and replication (14).

Increasing fruit and vegetable consumption consequently increases antioxidant and phytonutrient intake, ensuring that free radicals are neutralised and unable to cause extended bodily damage. Allium vegetables such as garlic and onions boost levels of naturally occurring antioxidant enzymes and contain arginine, sulphur and flavonoids, that may be particularly protective of stomach cancer (16).

Lauren Stribley, Accredited Practising Dietitian and Nutritionist (APD), acknowledges the benefits of plant-based

Figure 1. The layers of the stomach wall (4)



foods but believes that “A vegan diet is impractical, unbalanced and can cause long-term health consequences like nutritional deficiencies” (17). Lauren emphasized that even protein-rich plant-based foods are not enough to meet long-term protein requirements and that moderate animal product consumption is required to ensure sufficient protein intake, though not all health experts agree with this position.

Such health experts include the Academy of Nutrition and Dietetics which hold firm to the position that “appropriately planned vegetarian diets, including total vegetarian or vegan diets, are healthful, nutritionally

adequate, and may provide health benefits in the prevention and treatment of certain diseases. Well-planned vegetarian diets are appropriate for individuals during all stages of the life cycle, including pregnancy, lactation, infancy, childhood, and adolescence, and for athletes” (18).

Further evidence suggests that most vegans take vitamin or mineral supplementations, indicating that their diets are nutritionally inadequate (19). However, vegans are less likely than omnivores to develop diseases such as cancer and heart disease due to a number of reasons. One reason is due to the fact that vegans do not consume the high

levels of saturated fat found in animal products (20) unlike omnivores. Other reasons include eating more phytonutrient and vitamin-rich diets and also having a lower body mass index as vegans tend to be leaner than omnivores (21).

This conflicting evidence demonstrates that the effectiveness of vegan diets on reducing stomach cancer risk is inconclusive.

How can the preparation of foods affect the risk of developing stomach cancer?

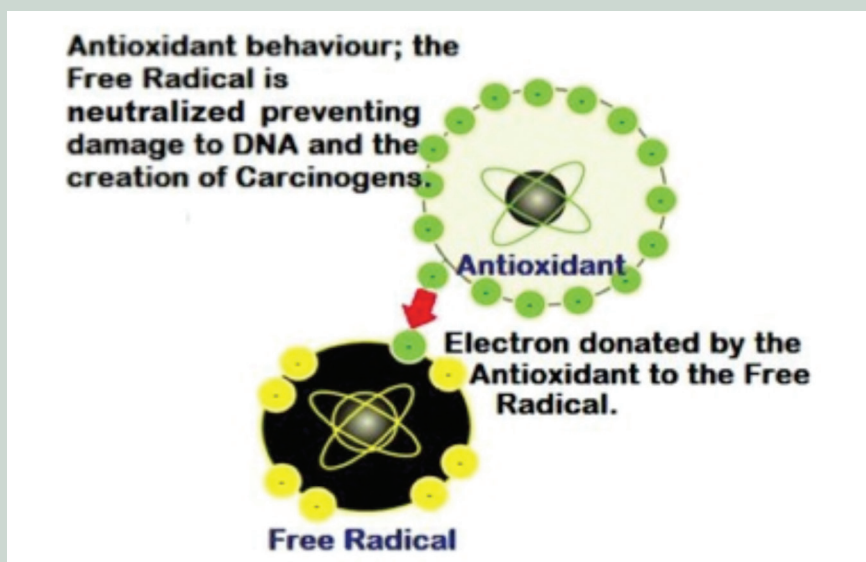
Research shows that preparation methods can influence how different foods affect stomach cancer risk. These include the ways in which foods are manufactured and preserved, the curing processes of deli meats and the cooking methods used to prepare fresh foods.

Excessive sodium consumption majorly contributes to increased stomach cancer risk, and sodium can induce inflammation of the mucosa (22). The maximum recommended daily intake (RDI) of sodium is 2,300mg for adults, but due to the increasing popularity of processed foods with high sodium contents, consumers’ intakes often exceed this recommendation (23). During manufacturing and refining processes, sodium is often added for flavour and texture enhancement. 4,800mg of sodium a day doubles the likelihood of developing stomach cancer (23) and much of this excess consumption likely results from consuming processed foods, without being aware of ‘hidden sodium’ content. High consumptions of smoked, salted or pickled foods, which extend shelf life with the addition of sodium, also contribute to excess sodium intake and is linked with increased stomach cancer risk (24,25). Clinical Dietician Erin Kennedy works with patients who have received a diagnosis of stomach cancer, and is aware of risk factors contributing to the incidence of this type of cancer. “The majority of patients that I have worked with ate diets low in fruits and vegetables whilst also consuming significant amounts of smoked and pickled foods” (26).

Even though the benefits of fruits and vegetables are profuse, various cooking

(Continued on next page)

Figure 2. Antioxidant neutralising a free radical (15)



methods strip them of their cancer-fighting properties, rendering them less effective at reducing stomach cancer risk. Lauren states that “Fruits and vegetables are best when unpeeled” (27) as their colourful skins are densely packed with protective compounds and fibre that cannot always be found in their flesh (27). However, some fruits and vegetables require alterations in order to be consumed and these alterations can affect their properties.

Additionally, cooking meats at high temperatures can promote the formation of cancer-causing chemicals within the meat tissue (28) such as heterocyclic amines (HCAs) and polycyclic aromatic hydrocarbons (PAHs). These compounds can damage DNA and increase cancer risk (28), including stomach cancer risk. HCAs result from amino acids burning as meat cooks at very high temperatures and PAHs form when fat and juices from meat drip onto the open fire, causing flames and smoke (29).

Grilling any type of animal flesh until partly blackened or charred can increase stomach cancer risk. However, some evidence suggests marinating meat with herbs such as parsley and rosemary, which provide antioxidants to the meat, can decrease HCA and PAH formation (31), and thus decrease stomach cancer risk.

Some evidence suggests that processed deli meats may be even more of a risk factor than fresh meat cuts. This is due to the substances added to them during the curing processes that they undergo before reaching the deli counter (32). When deli meats are cured, preservatives are added to prolong their shelf lives and improve their colours and flavours. Nitrates are a preservative added to some processed meats for these purposes and are currently being condemned as cancer risk factors by the health industry (33).

The concern is the eventual formation of carcinogenic nitrosamines in the digestive tract, which may occur when people consume nitrates. When ingested, bacteria in saliva can reduce nitrates to nitrites (34). Nitrites can form bonds with amines found in meats and in acidic environments such as

Figure 3. How heat from cooking forms HCA compounds (30)

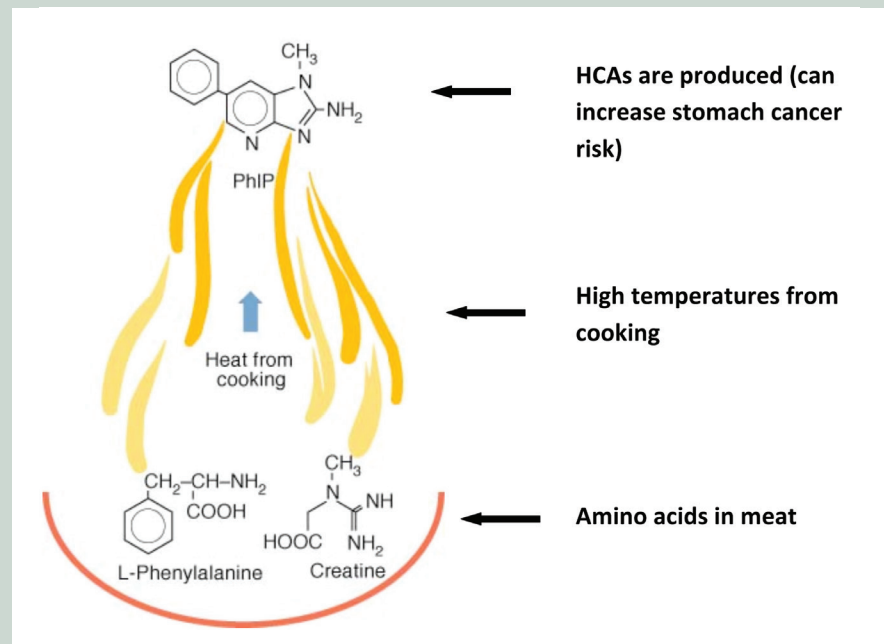


Table 1. Quantities of Nitrate in 20g Samples of Various Deli Meats, Home-Cooked Meats and Fresh Vegetables

Category	Substance tested	Nitrate (NO ₃ ⁻) Reading (mg/L)
Home-cooked	Roast Chicken	0.00
Home-cooked	Grilled Beef	0.00
Deli Meat	Smoked Shredded Ham	0.00
Deli Meat	Double Smoked Leg Ham	0.50
Deli Meat	Shortcut Rindless Bacon	40.0
Deli Meat	Smiley Fritz	40.0
Deli Meat	Roast Turkey	80.0
Deli Meat	Cocktail Frankfurts	160
Vegetable	Zucchini	200
Vegetable	Spinach	200

the human stomach, these bonds can lead to the formation of nitrosamines (33).

However, nitrates are found naturally in plants and therefore a normal component of the human diet (36). This adds to the speculation about whether nitrates are truly a risk factor of stomach cancer. Furthermore, unlike processed meats, fruits and vegetables do not contain amines (37) that can react with the nitrites. They also contain antioxidants which can neutralise the nitrites (38), and therefore lower stomach cancer risk. Nutrition Lecturer at the University of South Australia, Evangeline Mantzioris states

that “More research into this claim is required. There is not enough solid evidence to support these speculations. Processed meats cannot be dubbed a risk factor of stomach cancer because of their nitrate content as vegetables contain nitrates but pose no threat to human health” (39).

How do the nitrate contents of prepared meats and vegetables compare?

As part of this research project, an experiment was undertaken that compared nitrate quantities in 20g of various deli meats, home-cooked meats and raw vegetables.

The 20g samples of each food were pulverised and a nitrate test strip was inserted into each sample. These strips provided nitrate readings in the form of a colour change.

There were no nitrates present in the home-cooked meats, indicating that they will not contribute to nitrosamine formation and increase stomach cancer risk through this mechanism. However, most of the deli meats tested indicated the presence of various nitrate quantities depending on how they were cured. Cocktail Frankfurts have the highest nitrate quantity at 160mg/L followed by Roast Turkey Breast with 80.0mg/L.

However, the quantities found in Smoked Shredded Ham and Double Smoked Leg Ham were low. This is because of differences between the curing processes. Smoking these meats involved drying their exteriors and applying a smoke coat, making their surfaces more hostile for bacteria growth and extending their shelf lives (41). Smoking deems the addition of nitrates unnecessary, accounting for the low readings obtained in this experiment. Unfortunately, the smoking process involves the addition of salt (41) and exposure to high temperatures, both of which appear to be stomach cancer risk-increasing factors.

The results show that the vegetables tested have high nitrate quantities. This means that if nitrates are truly a risk factor of stomach cancer, vegetables and processed meats could similarly increase stomach cancer risk. Whilst plants do not contain amines, they can be eaten with meats containing amines, allowing for the formation of carcinogenic nitrosamines.

This experiment shows that high nitrate quantities are present in some deli meats but even higher quantities are present in cancer-protective vegetables. This gives reason to doubt the theory that nitrates are a stomach cancer risk factor.

This information about nitrates is unavailable to consumers when purchasing deli meats. Although further research is required to determine whether nitrates are a stomach cancer risk factor, consumers

still deserve the right to make informed decisions about their dietary choices. Therefore, this information should be available to consumers at the point of sale.

What would a stomach cancer risk reducing meal look like?

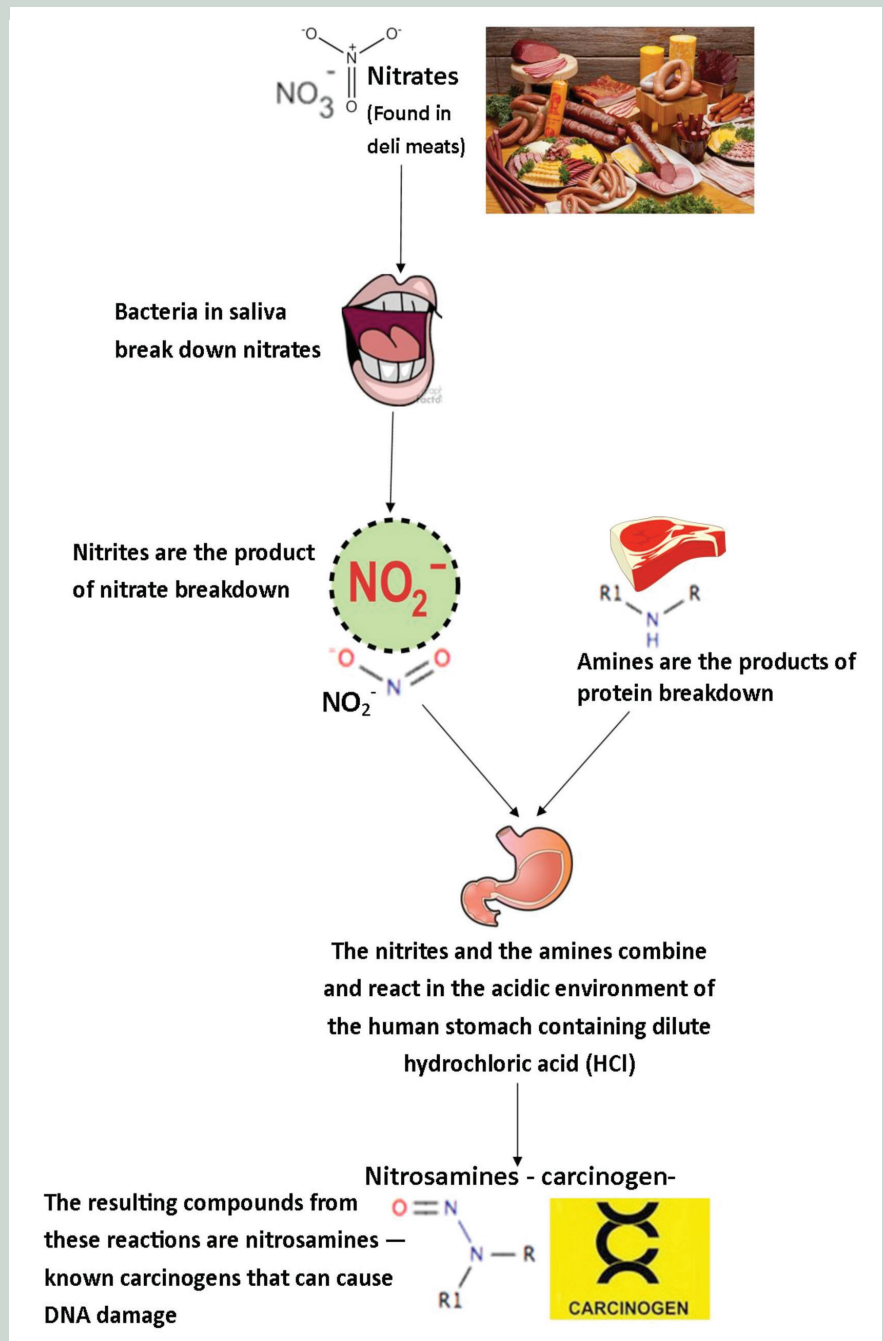
Using the evidence obtained from primary and secondary sources in this research project, potential stomach cancer risk-increasing meals were designed, and then

altered into stomach cancer risk-reducing meals. The altered meals included some of the foods in the original meals, but with replacements and altered cooking methods. An original meal with its alterations and approximate nutritional contents is shown below.

The potential stomach cancer risk-increasing meal is high in sodium, low in fibre and

(Continued on next page)

Figure 4. The Process of Nitrosamine Formation (35)



antioxidants and has been prepared in ways that may increase their content of carcinogenic substances.

No vegetables have been included, leaving this meal very low in fibre and antioxidants. The omission of vegetables in this meal denies the body of cancer-protective nutrients found in plant-based foods. Furthermore, there are high quantities of sodium and nitrate in the sausages and bacon and the baked beans also contain high amounts of sodium. The 3,321 mg of sodium is far beyond the daily allowance. Additionally, the animal products have been fried at high temperatures, increasing the likelihood of cancer-causing chemicals forming, thus increasing stomach cancer risk.

This meal can be altered by having a 2-egg omelette with various vegetables that contain fibre and multitudes of antioxidants. The alterations include a variety of vegetables, resulting in more antioxidants and 4.2g more fibre than the original. This altered meal also has six times less sodium due to the exclusion of sodium-laden processed ingredients. This can reduce the risk of an inflamed mucosa that is linked to increased stomach cancer risk.

Conclusion

This research has found that many factors may influence stomach cancer risk and it has been proven that diet can have a substantial impact. High sodium, low fibre, low antioxidant and high processed meat intakes have all been associated with an increased stomach cancer risk, as well as preparing foods at high temperatures.

There are indications that varying quantities of nitrates in processed meats may form carcinogenic nitrosamines but there is serious debate within the scientific community about this, and more research is required to reach a conclusion. Experimentation showed that nitrates were also present in vegetables at high quantities yet their consumption is not associated with increased stomach cancer risk. The main theory explaining this is that their antioxidants can neutralise the carcinogenic nitrosamines. Again, further research is required to confirm this. Information about

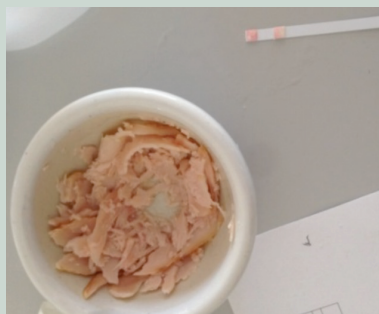
Figure 5. Roast Chicken (40)



Figure 6. Cocktail Frankfurts (40)



Figure 7. Roast Turkey Breast (40)



nitrate contents is not readily available to consumers, making it challenging for them to choose foods wisely in order to decrease stomach cancer risk.

Combining all of this acquired knowledge makes it possible to successfully synthesise stomach cancer risk-reducing meals and doing so could decrease disease burden in Australia.

References

1. Australian Government. Cancer in Australia statistics. Accessed December 3, 2013: <http://cancer australia.gov.au/affected-cancer/what-cancer/cancer-australia-statistics>.

2. Government of South Australia. The risks of poor nutrition. Accessed April 3, 2014: <http://www.sahealth.sa.gov.au/wps/wcm/connect/public+content/sa+health+internet/healthy+living/is+your+health+at+risk/the+risks+of+poor+nutrition>.
3. GI Cancer Institute. What is Gastro-Intestinal Cancer? Accessed March 31, 2014: <http://gicancer.org.au/gi-cancer/what-is-gastro-intestinal-cancer/>.
4. Modern Cancer Hospital Guangzhou. What is stomach cancer? Accessed May 1, 2014: <http://www.moderncancerhospital.com/cancer/stomach-cancer/>.
5. Barnard ND, Reilly JK. The Cancer Survivor's Guide: Foods That Help You Fight Back. Healthy Living Publications, a division of Book Publishing Company. Summertown, TN; ISBN: 978-1-57067-225-5 ©2008 The Cancer Project.
6. Interview with *SimplyVibrant* Natural Therapist Lisa Fitzgerald on March 5, 2014.
7. Australian Government. Department of Health. What is a Serve of Vegetables? Accessed May 4, 2014: <http://www.eatforhealth.gov.au/food-essentials/how-much-do-we-need-each-day/serve-sizes>.
8. Physician's Committee for Responsible Medicine. Food for Life Cancer Project. Healthy Eating for Life. Accessed May 7, 2014: <http://pcrm.org/health/cancer-resources/pubs/healthy-eating-for-life>.
9. The Houston Chronicle. Live Healthy. A List of Foods for No Fiber Diets. Accessed May 7, 2014: <http://livehealthy.chron.com/list-foods-fiber-diets-2397.html>.
10. Interview with Suzanne Dixon; Epidemiologist/Registered Dietician, Past Chair of the Oncology Nutrition Dietetic Practice Group, The Academy of Nutrition and Dietetics. March 20, 2014.
11. State Government of Victoria. Better Health Channel. Antioxidants. Accessed January 6, 2014: <http://www.betterhealth.vic.gov.au/bhcv2/bhcarticles.nsf/pages/Antioxidants>.
12. MedicineNet.com. Antioxidants and Cancer Prevention. Accessed January 1, 2014: <http://www.medicinenet.com/script/main/art.asp?articlekey=47814>.
13. Cancer Research UK. Genes, DNA and cancer. Accessed: April 1, 2014: <http://www.cancerresearchuk.org/about-cancer/what-is-cancer/genes-dna-and-cancer>
14. Link A, Balaguer F, Goel A. Cancer chemoprevention by dietary polyphenols: promising role for epigenetics. *Biochem Pharmacol.* 2010;80(12):1771–92.
15. Wall JEM. Figure 5.1 Antioxidants neutralize free radicals. In: K Hefferon, *Let thy Food Be Thy Medicine: Plants and Modern Medicine*. Oxford University Press, Inc; New York, New York ©2008.
16. Escott-Stump S. Cancer, pp. 734-38. In: *Nutrition and Diagnosis – Related Care*; 7th Edition. Lippencott Williams & Wilkens; Baltimore, Maryland ©2012.
17. Interview with Lauren Stribley, Accredited Practising Dietician at North East Modbury Medical & Dental Centre on March 20, 2014.
18. Position of the American Dietetic Association: Vegetarian Diets. *J Am Diet Assoc.* 2009;109(7):1266–82.
19. Perkins L. The Good, The Bad, and The Vegan. Accessed April 15, 2014: http://www.vanderbilt.edu/AnS/psychology/health_psychology/vegan.htm.
20. People for the Ethical Treatment of Animals. Top 10 Reasons to Go Vegan in the New Year. Accessed April 15, 2014: <http://www.peta.org/living/food/top-10-reasons-go-vegan-new-year/>.

Figure 8. Smoked Shredded Ham (40)



Figure 9. Zucchini being prepared for testing (40)



Figure 10. Baby Spinach (40)



BREAKFAST

Potential Stomach Cancer Risk Increasing Meals



Figure 11 (40)

- 2 fried eggs
- 100g fried bacon
- 3 20g sausages
- 2 40g slices of toasted white bread
- ¼ cup canned baked beans (for each slice = ½ cup beans total)

Nutrition Facts (42)

Total Energy: 3,619kj
Protein: 67.6g
Carbohydrate Total: 49g
Sugar: 7.1g
Fibre: 7g
Fat Total: 43.2g
Saturated Fat: 14.1g
Sodium: 3,321mg

Potential Stomach Cancer Risk Decreasing Meal

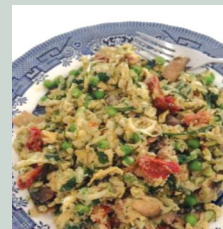


Figure 12 (40)

- 2 egg messy omelette with 30g sundried tomatoes,
- 1 cup baby spinach leaves,
- 1 cup mushrooms
- ½ cup ricotta cheese

Nutritional Facts (42)

Total Energy: 2,264kj
Protein: 34.5g
Carbohydrate Total: 18.2g
Sugar: 16.7g
Fibre: 11.2g
Fat Total: 35.5g
Saturated Fat: 14.4g
Sodium: 501mg

21. Interview with Suzanne Dixon; Epidemiologist/Registered Dietician, Past Chair of the Oncology Nutrition Dietetic Practice Group, The Academy of Nutrition and Dietetics. June 10, 2014.
22. World Cancer Research Fund & American Investigation of Cancer Research. *Food, Nutrition and the Prevention of Cancer: a Global Perspective*, BANTA Book Group, Menasha WI, USA (1997).
23. Utton T. How a salt-heavy diet can double cancer risk. The Daily Mail, Dailymail.com. Accessed December 31, 2013: <http://www.dailymail.co.uk/health/article-205378/How-salt-heavy-diet-double-cancer-risk.html>.
24. Hohmann A (contributor). Lowimpact.org. Food smoking: introduction. Accessed April 2, 2014: http://www.lowimpact.org/factsheet_food_smoking.html.
25. Garden-Robinson J, Hegerfeld-Baker J. South Dakota State University Extension Service. Food Preservation. Making Pickled Products. Accessed April 2, 2014: <http://www.ag.ndsu.edu/pubs/yf/foods/fn189.pdf>.
26. Interview with Erin Kennedy, Clinical Dietician, Nutrition and Speech Pathology Department at Peter MacCallum Cancer Centre. February 25, 2014.
27. Interview with Lauren Stribley, Accredited Practising Dietician at Royal Park Medical and Dental Centre. Thursday, 20th March 20, 2014.
28. AICR eNews. Cooking for Cancer Prevention. Maximizing cancer prevention in the kitchen. Accessed February 9, 2014: <http://www.aicr.org/enews/2013/march-2013/Cooking-for-cancer-prevention.html>.
29. Sass C. Eating Well. Does Grilling Cause Cancer? Accessed February 9, 2014: http://www.eatingwell.com/nutrition_health/nutrition_news_information/does_grilling_cause_cancer.
30. Andrews R. Precision Nutrition. All About Cooking & Carcinogens. Accessed April 9, 2014: <http://www.precisionnutrition.com/all-about-cooking-carcinogens>.
31. Stuyck K. Reducing Cancer-Causing Chemicals in Outdoor Cooking. Tips for healthy grilling. OncoLog. 2012; 57(9). Accessed February 9, 2014: <http://www2.mdanderson.org/depts/Oncolog/articles/12/9-sep/9-12-hc.html>.
32. World Cancer Research Fund & American Institute of Cancer Research. *Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective* (2007). Accessed April 7, 2014: <http://www.dietandcancerreport.org/>.
33. Collins D. Underground Health Reporter. Fact or Myth: Do Nitrites/Nitrates in Food Cause Cancer. Accessed January 26, 2014: <http://undergroundhealthreporter.com/sodium-nitrates-in-food#axzz2rUB0GkVY>.
34. Chiado A. Food Additives. Sodium Nitrate. Accessed January 26, 2014: <http://flipper.diff.org/app/items/info/4138>
35. Varricchio A. Image created by author.
36. Suddoo AG. Processed meat: sodium nitrate versus sodium nitrite. Accessed January 26, 2014: <http://www.examiner.com/article/processed-meat-sodium-nitrate-versus-sodium-nitrite>.
37. Kirjoitettu E. Nitrates are beneficial. Where did I get it wrong? 2010. Accessed May 2, 2014: <http://www.pronutritionist.net/nitrates-are-beneficial-where-did-i-get-it-wrong/>.
38. Healthy Child Healthy World. How to Avoid Added Nitrates and Nitrites in Your Food. April 2013. Accessed May 2, 2015: <http://healthychild.org/easy-steps/avoid-nitrates-and-nitrites-in-food/>.
39. Interview with Evangelina Mantzioris, Lecturer in Nutrition: School of Pharmacy and Medical Sciences University of South Australia. May 1, 2014.
40. Varricchio A. Author photo taken March 19, 2014.
41. Interview with Franz Knoll, Managing Director of Standom Smallgoods Pty Ltd. Adelaide, SA. April 18, 2014.
42. Values for each ingredient provided by CalorieKing App. Mathematical processes done by author. Accessed May 3, 2014.

2015-2016 Oncology Nutrition DPG Officers and Committee Chairs

(* Voting member)

Chair*

Tricia Cox, MS, RD, CSO, LD, CNSC
Email: tricia.melhart@baylorhealth.edu

Chair-elect*

Kelay Trentham, MS, RDN, CSO, CD
Email: kelayt@gmail.com

Secretary*

Katie Badgett, MS, RDN, CSP, LDN
Email: Katie.badgett@stjude.org

Treasurer*

Caitlin Benda, MS, RD, CSO, LDN
Email: caitlin.benda@gmail.com

Past Chair*

Andreea Nguyen, MS, RD, CSO, LD, CNSC
Email: andreea.nguyen@baylorhealth.edu

Nominating Committee Chair*

Heidi Scarsella, RDN, CSO, LDN
Email: heidiscarsella@gmail.com

Western Area Representative

(CA, TX, AZ, NM, WA, OR, NV, WY, ND, SD, HI, AK, ID, MT, UT, CO, Asia, NZ, AU)
Paula Charuhas-Macris, MS, RD, CSO, FAND, CD
Email: pcharuha@seattlecca.org

Eastern Area Representative

(VA, GA, PA, DE, NH, RI, NC, SC, NY, FL, NJ, MD, VT, ME, CT, MA, DC, PR, and Europe)
Dianne Phippenburg, MS, RD/N, CSO
Email: Dianne.Phippenburg@va.gov (primary) or
pkippenburgmom@yahoo.com (secondary)

Central Area Representative

(MI, IN, AR, AL, IA, KS, OH, KY, MO, MS, MN, OK, WV, TN, LA, IL, WI, NE and Canada)
Anita Vincent, RDN, CSO, LDN
Email: avincent@westclinic.com

Development Coordinator

Janet Mildrew, RD
Email: janet.mildrew@rivhs.com

Account Manager

Alliance Coordinator

Rhone M. Levin, MEd, RD, CSO, LD
Email: uwgirl@aol.com or levinr@slhs.org

Electronic Mailing List (EML) Administrator

Maureen Gardner, MA, RD, CSO, LDN
Email: maureen.gardner@moffitt.org

Membership Chair

Michelle Bratton, RD, CSO
Email: michebratton@yahoo.com

Academy Representative to CoC

Kathryn Hamilton MA, RD, CSO, CDN, FAND
Email: kathryn.hamilton@verizon.net or kathryn.hamilton@atlantichealth.org

Awards Chair

Erin Gurd, RD
Email: erin.gurd@moffitt.org or erin.n.gurd@gmail.com

Oncology Nutrition Connection Newsletter Editor

Suzanne Dixon, MPH, MS, RD
Email: sdixon@umich.edu

Associate Editors

Robin Brannon, MS, RD, CSO
Email: robin.brannon@gmail.com

Jodie Greear, MS, RD, CSO, LDN
Email: jodie.greear@gmail.com

Maureen Leser, MS, RD, CSO, LD
Email: mgoreleser@gmail.com

Jocelyne O'Brien, MPH, RD, CSO, LDN
Email: jocelynenasser@yahoo.com

Continuing Education Chair

Tiffany Barrett, MS, RD, CSO, LD
Email: tiffany.barrett@emoryhealthcare.org

Policy & Advocacy Leader

Colleen Spees, PhD, MEd, RDN, FAND
Email: spees.11@osu.edu

House of Delegates ONDPG Delegate

Nicole Fox, RD, LMNT, CNSC, CSO
Email: nfox@nebraskamed.com

Website Administrator

Heather Bell-Temin MS, RD, CSO, LDN
Email: heather.bell_temim@hotmail.com

Public Content Manager – ONDPG Website

Erin Williams, RD, CNSC
Email: erinbrowe@gmail.com

Social Media Coordinator

Lindsay Sappah, RD, CSO, LDN, CNSC
Email: lindsay.kovacic@walgreens.com

EBlast Coordinator

Kristen Lange, MS, RD, CSO, LD/N
Email: kristen.lange@moffitt.org

Webinar Planning Committee

Chair: Amy Patton, RD, CSO, CNSD
Email: amypattonrd@hotmail.com

Bernadette Festa, MS, RD, CSO
Email: FestaB@sutterhealth.org

Abby Traul, RD, CSO, LD
Email: abigailt@portmed.org

Cheryl Tuttle, MHS, RD, CSO, LD
Email: inmydna@myway.com

Small Project Research Grant Chair

Heidi Ganzer, DCN, RD, CSO, LD
Email: hganzer@mchsi.com

Chair – Pediatric Sub-Unit

Rachel Hill, RD, LD, CNSC
Email: Rachel.Hill@cookchildrens.org

Leadership Committee Members – Pediatric Sub-Unit

Nancy Sacks, MS, RD, LD (Chair-Elect)
Email: sacksgregg@juno.com

Katie Badgett, MS, RDN, CSP, LDN (Secretary)
Email: Katie.Badgett@stjude.org

Jodie Greear, MS, RD, CSO, LDN (Cure4Kids Administrator)
Email: jodie.greear@gmail.com

Paula Charuhas Macris, MS, RD, CSO, CD, FAND (PNPG Liaison)
Email: pcharuha@seattlecca.org

Jennifer Caceres, MS, RD, LD (Newsletter/ Communication Coordinator)
Email: jennifer.caceres@mch.com

Chelsea Schulman MS, RD, LDN (Webpage Content Manager)
Email: SchulmanC@email.chop.edu

Special Project Chairs

Project Chair – Oncology Nutrition in Clinical Practice

Maureen Leser, MS, RD, CSO, LD
Email: mgoreleser@gmail.com

Project Coordinators – Oncology Nutrition Symposium (Spring 2016)

Heather Bell-Temin MS, RD, CSO, LDN
Email: heather.bell_temim@hotmail.com

Jeannine Mills, MS, RD, CSO, LD
29 Sweeney Rd

Email: jeannine.b.mills@hitchcock.org

Andreea Nguyen, MS, RD, CSO, LD, CNSC
Email: andreea.nguyen@baylorhealth.edu

Planning Committee – Oncology Nutrition Symposium (Spring 2016)

JJ Barten, RD, CSO
Email: jacklyn.barten@uch.edu

Katrina Claghorn, MS, RD, CSO, LDN
Email: katrina.claghorn@uphs.upenn.edu

Suzanne Dixon, MPH, MS, RD
Email: sdixon@umich.edu

Kristen Lange, MS, RD, CSO, LD/N
Email: kristen.lange@moffitt.org

Denise C Snyder, MS, RD, CSO, LDN
Email: denise.snyder@duke.edu

Kelay Trentham, MS, RDN, CSO, CD
Email: kelayt@gmail.com

Rhone M. Levin, MEd, RD, CSO, LD
Email: uwgirl@aol.com or levinr@slhs.org

Maria Petzel, RD, CSO, LD, CNSC, FAND
Email: mpetzel@mdanderson.org

Maureen Huhmann, DCN, RD, CSO
Email: Maureen.huhmann@us.nestle.com

Elaine Trujillo, MS, RDN
Email: trujille@mail.nih.gov

Project Chair – EAL Oncology Nutrition Guideline Revision Expert Workgroup Chair & ON Publications

Laura Elliott, MPH, RD, CSO, LD
Email: Elliott@mngmc.com

Project Chair – Research Award & SOP/SOPP

Jodie Greear, MS, RD, CSO, LDN
Email: jodie.greear@gmail.com

Project Chair – Benchmarking Project

Elaine Trujillo, MS, RDN
Email: trujille@mail.nih.gov

Academy DPG Relations Manager

Carrie Kiley
Email: ckiley@eatright.org