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Beginning to Identify Trends among Dietitians Treating Eating Disorders: When Do You Make Referrals to Mental Health Care?

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INTRODUCTION

Registered Dietitians (RDs) apply their training in Medical Nutrition Therapy (MNT) together with their continuing education to provide nutrition counseling to patients. The Nutrition Care Process (NCP) provides structure for nutrition counseling, one facet of which is referrals to providers of other disciplines of care when a patient's needs fall outside of the RD Scope of Practice.[1,2]

Although RDs in any practice area may make a referral to mental health care (MHC), RDs with expertise in Eating Disorders (EDs) may be particularly aware of the frequently co-occurring psychiatric issues (anxiety, depression, addiction, obsessive-compulsive symptoms, post-traumatic stress and others) that can prevent or interfere with nutrition counseling and nutritional restoration.[3-7] While proper nutrition is essential for ED recovery, nutrition counseling alone will not repair the psychiatric and emotional components of EDs.[3,5-7] Concurrently, acute and chronic malnutrition exacerbate brain dysfunction and psychiatric symptoms, strengthening irrational thinking and maladaptive eating behaviors that maintain the ED cycle. Appropriate treatment of co-occurring disorders is necessary for stabilization and movement toward recovery.

In cases where the RD is the first professional to identify the symptoms of psychiatric or emotional distress, the RD may be responsible for making the referral to MHC, either directly or in consultation with the primary care professional.[8] No standard guidelines have been published regarding when, how, or under what circumstances an RD can or should make referrals to MHC, leaving the RD to rely on consultation with colleagues and/or his or her own clinical judgment. Experienced RDs have honed their expertise over time. Newer or less experienced RDs would likely benefit from at least general guidelines for triggers that indicate the need for such a referral.

Because of their special expertise in the mental health arena, ED RDs were selected as a population sample for this survey of referral practices. If trends can be identified in the clinical practice of experienced practitioners, they may provide a first step toward future development of profession-wide guidelines.[9]

METHODS

A 19-question survey about referral practices of RDs treating EDs was developed by the investigators based on published literature and clinical experience.[3-7] One hundred members of the International Federation of Eating Disorder Dietitians (IFEDD) who reside and practice in the United States were invited to anonymously

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complete the survey through a link to SurveyMonkey provided via email. The survey link was open for 5 months and closed once 40 responses were received. Participation in the survey was voluntary with no monetary compensation offered or provided.

Of the 19 questions, 15 allowed one response to multiple choices, three allowed multiple responses to multiple choices, and one question was open-ended with unlimited response length. Responses of quantitative data were compiled electronically into charts using the SurveyMonkey software. Open-ended responses were reviewed by hand, using qualitative methodology to identify trends.

To ensure that participants were in fact experienced in their area of practice, demographic questions requested respondent age, length of time as a dietitian, length of time treating individuals with EDs, professional credentials, work experience, and continuing education. Other questions surveyed familiarity with the RD Scope of Practice, preference for multidisciplinary team treatment of ED, and how the RD proceeds once a patient does not follow through on a mental health referral. The primary objective for the survey was to identify what triggers an experienced ED RD to refer a patient to MHC. This was assessed by the question, "How do you decide to make the recommendation that a patient meet with a mental health professional? (check all that apply)." Participants could select any or none of the 33 possible responses, and were offered an opportunity to add an unlimited number of additional reasons for referral. The response options were developed from published literature. Since the diagnosis of an ED is considered by many to be in itself a trigger for a mental health referral, individual signs included in the diagnostic criteria for ED, such as bingeing, purging and restrictive eating, were not included as potential responses in order to study the additional triggers for mental health care rather than the eating disorder itself.

RESULTS

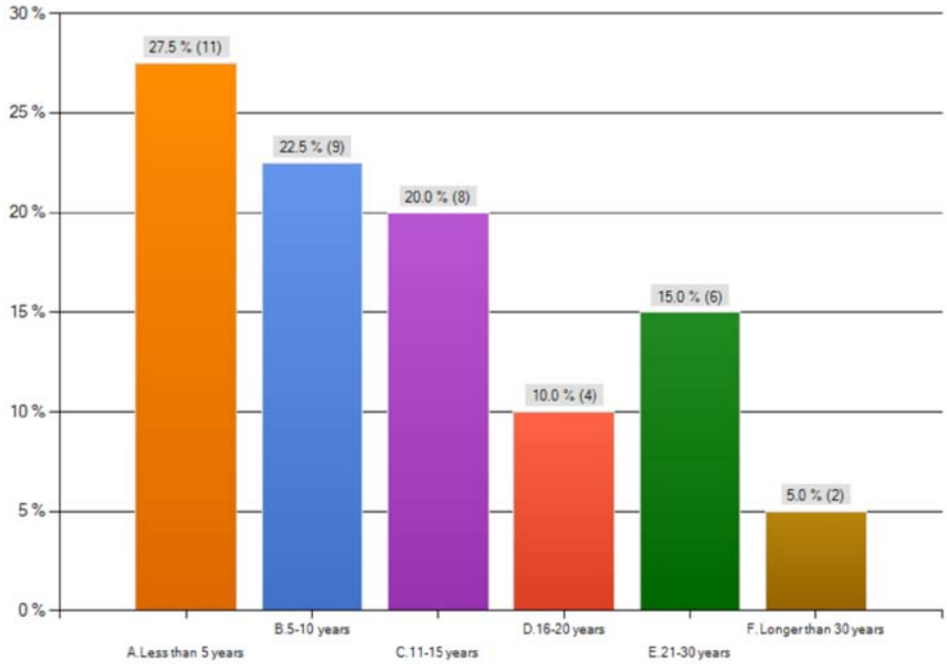
Participant Demographics

One hundred percent (n=40) of participants were female RDs 25 years of age or older living in the U.S. and working with individuals with eating disorders. The majority (62.2%) of RD participants practice in a private or community outpatient setting. Participants were asked separately about their years in practice specifically with individuals with EDs. Responses demonstrated a wide range of longevity in the field, shown in Figure 1, below.

The majority of participants reported continuing education in ED (97.3%), continuing education in counseling (79.5%), and continuing education

Figure 1

How many years have you been working with individuals with eating disorders?



in mental illness (56.4%). Over half (53.8%) of the RDs report holding an advanced degree in nutrition (MS or PhD) and 10.3% report holding a counseling license. Of the surveyed participants, 46.2% teach classes about EDs to students, and 35.9% teach continuing education about EDs.

Mental Health Referral

Of 40 participants, 37 (92.5%) responded to the primary objective question, regarding 33 symptoms that might trigger a MHC referral. An affirmative response to a symptom signified that it would trigger a MHC referral. Of those who responded, 100% responded affirmatively to "Patient reports being depressed" (100%). This was the only option that received a 100% affirmative response.

Other symptoms received between 37.8% and 97.3% affirmative response. Those symptoms receiving over 90% affirmative response were: "Patient reports cutting themselves or using self-mutilation" (97.3%), "Patient exhibits signs of other mental illnesses such as Obsessive Compulsive Disorder, Post Traumatic Stress Disorder and Panic Disorder" (94.6%), "Patient reports abusing drugs or alcohol" (91.9%) and "Patient reports extreme mood swings" (91.9%).

Those symptoms receiving less than a 45% responses were: "Patient looks frail at the first appointment" (40.5%), "Patient reports family history of eating disorders" (37.8%), "Patient reports not feeling physically attractive" (43.2%) and "Patient is secretive about their disease" (43.2%).

Affirmative responses to all 33 provided symptoms are summarized in Table 1, below (n=37).

Table 1

Responses to "How do you decide to make the recommendations that a patient meet with a mental health professional? (Check all that apply)"

Response	Affirmative Percent	Affirmative Count
Mental Illness		
Patient reports being depressed	100.0%	37
Patient exhibits signs of other mental illnesses such as Obsessive Compulsive Disorder, Post Traumatic Stress Disorder and Panic Disorder	94.6%	35
Patient reports extreme mood swings	91.9%	34
Self-harm		
Patient reports cutting themselves or using self-mutilation	97.3%	36
Patient reports abusing drugs or alcohol	91.9%	34
Patient reports history of suicide attempts	83.8%	31
Abuse from Others		
Patient reports history of sexual abuse	86.5%	32
Patient reports history of physical abuse	83.8%	31
Patient reports verbal abuse	83.8%	31
Poor Self-Esteem		
Patient reports having little or no self-confidence, and critiques oneself repeatedly	83.8%	31
Patient believes they will never get better	81.1%	30
Patient reports not feeling worthy of being loved	78.4%	29
Patient reports not feeling beautiful on the inside	56.8%	21
Patient reports they do not excel at anything	75.7%	28
Patient reports not being physically attractive	43.2%	16
Poor Progress during MNT		
Patient only focuses on body image	75.7%	28
Patient never talks about food, and digresses to other topics	78.4%	29
Patient continues to lose a significant amount of weight	78.4%	29
Patient is silent or extremely quiet at two or more appointments.	70.3%	26
Have counseled the patient five or more times, and have not have noticed any changes	62.2%	23
Patient is in denial about having an eating disorder	78.4%	29
Patient is secretive about their disease	43.2%	16
Patient looks frail at the first appointment	40.5%	15
Difficulty with Friends and Family		
Patient reports having arguments with co-workers/friends/family	67.6%	25
Patient reports being stressed with home life	62.2%	23
Patient reports being stressed with school/work	56.8%	21
Patient reports they want to feel more accepted by friends and family	54.1%	20
Patient reports feeling unappreciated	54.1%	20
Patient reports they want to feel more accepted by friends and family	45.9%	17
Patient reports feeling alone and scared	78.4%	29
Patient reports alienating themselves from friends and family	75.7%	28
Patient reports having marital issues	86.5%	32
Patient reports family history of eating disorders	37.8%	14

When sorted by length of time as an RD, responses were consistent throughout all groups. Additionally, 19 participants took the opportunity to report additional referral triggers in open-ended format in response to the prompt: "Please list any other signs or criteria that impact your decision to recommend a patient meet with a mental health professional." Write-in responses included (listed from those given by multiple respondents to those given by only one respondent):

- "All clients with an eating disorder" or similar (6 responses)
- "They are binging [sic], compulsively eating, purging, restricting" or similar (2 responses)
- "Conflict with family member/ family involvement" or similar (2 responses)
- "If there is lack of progress in our work" or similar (2 responses)
- "The level of care needed for their issues is beyond advanced nutrition counseling and requires a skilled mental health professional" or similar (2 responses)
- "Need for medication management/psychiatrist" (1 response)
- "If a patient is very "particular" about how they eat as in that they only eat organic/natural foods or are very picky to the point where there is apprehension around eating other foods." (1 response)
- "Requirement of the clinic" (1 response)
- "Recovered from ED, seeking healthy living support, History of dieting, no ED dx" (1 response)
- "Other risk factors --medical, prior or ongoing hx/saga of the ED, compulsive exercise" (1 response)

DISCUSSION

In the process of providing nutrition counseling, RDs often become privy to information outside the purview of MNT. Therefore RDs in all areas of practice will benefit from guidelines for making a MHC referral and improving their comfort level with making referrals. Due to the overlap of nutrition with psychiatric and psychological issues among ED patients, it is of even greater importance that RDs specializing in the ED field expand their knowledge of mental health issues as well as their network of mental health professional to whom they can refer.

As a pilot study, this project has several limitations. The intentional omission of common ED behaviors (binge eating, purging, compulsive exercise, etc.) as potential triggers may have unintentionally confused respondents, as evidenced by some open-ended responses. There is no way to measure the influence of "clinical impression" or "professional judgment" on responses, nor to validate that what respondents report is in fact what they practice. This study did not allow respondents to identify symptoms that would not trigger a MHC referral independently but that together with one or more other symptoms would combine to trigger a referral.

Interestingly, data compiled in this study indicated "Patient reports family history of eating disorder" is a low-level trigger for an RD to recommend MHC. Since a genetic correlation in the development of an ED has been documented,[1] this outcome raises the question of whether patients who report a family history of ED but do not exhibit other MHC-worthy qualifications should nevertheless be referred to MHC. Next steps could include surveying mental health professionals to see if they would identify the same referral triggers as the RDs and if they could contribute additional triggers for RDs to consider; identifying clusters of symptoms that trigger a referral only when reported in tandem; and designing a study that allows respondents to rate the urgency of a MHC referral for different symptoms.

CONCLUSION

Based on this survey of the professional practice of experienced ED RDs, a MHC referral is most often triggered by reports or signs of:

- Depression
- Self-harm
- Other mental illness
- Substance abuse
- Extreme mood swings

Many other symptoms may also warrant MHC, whether independently or in conjunction with other symptoms. The competent RD will continue to use a combination of clinical judgment and consultation with colleagues until standard guidelines for MHC referral are available.

Research DPG: CPE Article

Dietitians Referring to Mental Health Professionals

Question writer: Caroline Sullivan, MS, RD, CSSD, LD

1.) What co-morbid conditions may interfere with nutrition restoration in a patient with eating disorders?
- A. Addiction
- B. Depression
- C. Anxiety
- D. All of the above

2.) What type of guidelines would be helpful for new dietitians treating eating disorders?
- A. Medical nutrition therapy
- B. Mental health referral
- C. Standard refeeding process
- D. Weight gain protocol

3.) What was the main objective of the current study?
- A. Determine the experience level of eating disorder dietitians.
- B. Understand from where dietitians receive referrals.
- C. Observe the treatment practices of eating disorder dietitians.
- D. Investigate what triggers dietitians to refer to therapists.

4.) What was a strength of the polled population?
- A. Both genders were represented
- B. Length of experience in the field
- C. Diagnostic criteria knowledge
- D. Percentage with counseling licenses

5.) Dietitians appear to always refer to mental health professionals for which symptom?
- A. Obsessive calorie counting
- B. Negative body image
- C. Regular use of drugs
- D. Reports of depression

6.) Identify a weakness of the present study?
- A. The survey identified too many eating disorder symptoms.
- B. Study population did not exhibit sufficient experience.
- C. Data investigated impressions not objective symptoms.
- D. Typical eating disorder behavior was not investigated.

7.) What influence should further studies investigate?
- A. Impact of professional experience
- B. Multiple symptoms to trigger referral
- C. Frequency of referrals to therapists
- D. Mental health professionals' credentials

8.) What was the most frequent write-in response given?
- A. Long history of an eating disorder
- B. Medication recommended
- C. All patients given referrals
- D. Lack of resources to make referrals

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Research on Nuts Supports Current Global Dietary Recommendations, The Growing Evidence Base Demonstrates Multiple Health Benefits

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ABSTRACT

Dietary recommendations have been made for nuts on the basis of an impressive evidence base demonstrating many health benefits. This article reviews the current research that supports dietary recommendations for nuts. In addition, we discuss the PREDIMED Prevencion con Dieta Mediterranea Trial, a landmark primary prevention intervention study that evaluated the inclusion of nuts in a Mediterranean-style diet on cardiovascular disease. This trial showed a 30% reduction in risk of cardiovascular disease events, and a 49% reduction in risk of stroke in the Mediterranean diet supplemented with mixed nuts group compared to the low-fat group. Research on nuts is moving at a fast pace, and the dietetic community needs to keep abreast of the emerging findings to be best positioned to respond to patient, colleague, and media inquiries.

INTRODUCTION

The Global Burden of Disease Study (2010), largely funded by the Gates Foundation and conducted in concert with the World Health Organization (WHO), recently reported the world's top health problems.[1] Ischemic heart disease (IHD) was ranked as the number one health problem in the world, and stroke was number three. In 1990, IHD and stroke were ranked number 4 and 5, respectively, indicating that the burden of cardiovascular disease (CVD), a non-communicable, chronic disease, is growing globally.

Of interest to the nutrition community is that the top ranked (presented in parentheses below) nutrition-related risk factors causing the greatest "loss of health" include: low fruit consumption (# 5), high sodium intake (#11), low intake of nuts and seeds (#12), iron deficiency (#13), low intake of whole grains (#16), vegetables (#17), and omega-3 fatty acids (#18), high processed meat intake (# 22), low fiber intake (# 24), vitamin A deficiency (#29), and zinc deficiency (# 31). Other ranked risk factors related to poor nutrition practices include high blood pressure, alcohol use, high body mass index, high fasting plasma glucose, childhood underweight, suboptimal breast feeding, high total cholesterol, lead, and non-potable water. In a ranking of dietary risk factors that affect IHD, a diet low in nuts and seeds was ranked at the top, contributing 40% of disability-adjusted life years.[2]

In recognition of the importance of a heart healthy diet as one strategy to decrease CVD by 20% by the year 2020, the American Heart Association has recommended ≥ 4 servings/week of nuts, legumes, and seeds.[3] Likewise, because of their many health benefits, the 2010 Dietary Guidelines for Americans recommends choosing a variety of protein foods, including unsalted nuts and seeds, in the list of foods and nutrients to increase.[4]

In this article we review the research that supports the current dietary recommendations for nuts, specifically related to major chronic diseases. In addition, we summarize the very exciting research from the PREDIMED Trial, a large intervention trial evaluating the effects of two Mediterranean-style diets, one that recommends daily nut consumption or olive oil compared with a lower-fat prudent diet on CVD and other diseases and conditions.[5]

Nuts and CVD

Four epidemiologic studies conducted in the United States (Adventist Health Study, Iowa Women's Health Study, Nurses' Health Study, Physicians' Health Study) demonstrate a dose-response decrease in coronary heart disease (CHD) risk with increasing nut comsumption.[6] Participants who consumed nuts > 5 times/week had an approximate 40% reduction in CHD risk. Consuming nuts 1-4 times/week was associated with about a 30% decrease in CHD risk. In a comprehensive pooled analysis of 25 feeding trials with 1284 data points, Sabate and colleagues reported a dose-response decrease in total cholesterol, LDL-cholesterol (LDL-C), the LDL-C/HDL-C ratio, and triglycerides (TG) with increasing calories from nuts in the diet (10%, 12% and 20% of calories).[7] The consistent evidence showing benefits of nut consumption on lipids and lipoproteins is one widely accepted mechanism to explain the cardioprotective benefits of nuts. Interestingly, many studies have shown a greater cholesterol lowering effect of nuts than would be predicted from their fatty acid profile.[8] The conclusion that emerged from this is that there are other bioactive factors in nuts that contribute to their cholesterol-lowering effects beyond their

favorable fatty acid composition. This conclusion is supported by a study that demonstrated LDL binding to HepG2 cells increased by 50% in subjects who consumed a walnut-enriched diet (walnuts replaced approximately 35% of energy from unsaturated fat).[9] In this study, the uptake of alpha-linolenic acid (ALA)-enriched LDL particles was increased with increasing amounts of α-linolenic acid (ALA) in the LDL particle. Thus, it appears that walnut consumption may stimulate LDL receptor function to promote LDL-C clearance from the plasma, and, thereby, decrease LDL-C levels.

Other mechanisms to explain the cardiovascular benefits of nuts include improved endothelial function in hypercholesterolemic subjects,[10] decreases in cellular adhesion molecules,[11] and reduced blood pressure.[12,13] In addition, recent evidence has indicated that walnut oil increases cholesterol efflux (also referred to as reverse cholesterol transport) which suggests that HDL particle function is improved.[14] Moreover, a new study reported increased cholesterol efflux after postprandial walnut consumption.[15] Collectively, multiple mechanisms have been indentified that explain the cardioprotective benefits of including nuts in the diet.

Nuts and Body Weight

Epidemiologic studies consistently show that there is either a negative association or no association between nut consumption and body weight, despite a higher reported energy intake (reviewed in 16). In a unique analysis of four cohort studies of 120,877 U.S. men and women, Mozaffarian et al. evaluated changes in lifestyle factors and weight gain at 4-year intervals[17] Four year weight change was inversely associated with vegetable intake (- 0.22 lbs), whole grains (-0.37 lbs), fruits (-0.49 lbs), nuts (-0.57 lbs), and yogurt (-0.82 lbs). Thus, the epidemiologic studies demonstrate neither an increase in body weight in nut consumers nor an age-related increase in body weight typically observed in developed countries. A recent clinical study evaluated the effects of a weight loss diet with almonds compared with a hypocaloric nut-free diet and reported a similar weight loss (4 to 6 kg) after 18 months.[18] Thus, almonds and likely other energy-dense nuts can be included in a hypocaloric diet and still achieve weight loss. Research reported by Novotny et al.[19] evaluated the metabolizable energy content of almonds and found it was 32% less than that calculated from Atwater Factors. One explanation for this is an increased fecal fat excretion that has been reported in subjects consuming one serving of mixed nuts (almonds, walnuts, hazelnuts) versus none.[20] Interesting, fecal fat excretion is related to how thoroughly nuts (i.e., almonds) are masticated.[21] What is emerging is that nuts are not digested as well as other foods which could explain the results of nut studies with a higher calorie intake and no increase in body weight, as well as similar weight loss responses.

Nuts, Diabetes, and Metabolic Syndrome

In the Nurses' Health Study, nut consumption was inversely associated with risk of type 2 diabetes.[22] Consumption of nuts (≥ 5 times/week) was associated with a 27% reduction in risk of type 2 diabetes. Nut consumption of 1 to 4 times/week was associated with a 16% reduction in risk. In a clinical study conducted by Jenkins et al.[23] using 117 subjects with type 2 diabetes, nut consumption (475 calories per 2000 calorie diet; 75 g/day) versus muffins, or half portions of mixed nuts or muffins resulted in a reduced HbA(1c) of 0.21%. The authors reported that nut intake was inversely related to changes in HbA(1c) and LDL-C. Thus, nut consumption may decrease risk of diabetes by improving glycemic control. In a study that evaluated the effects of 56 g/day of walnuts added to subjects' usual diet for 8 weeks versus no walnuts, endothelial function (as measured by flow mediated dilation) was significantly improved on the walnut diet.[24] In this study, there was no effect of walnuts on HbA(1c) despite an increase in fasting blood glucose levels. Another study by Tapsell et al.[25] also found that 30 g/day of walnuts consumed by patients with type 2 diabetes did not affect HbA(1c); however, there was a beneficial effect on LDL-C (-10%) and increase in HDL-C. Given that vascular complications accompany diabetes, an improved endothelial function with walnuts may be of clinical benefit. However, further studies are needed to better understand the effects on diabetes control and the accompanying vascular complications.

The standard of care for metabolic syndrome includes weight loss because most patients have excess visceral adiposity. Two studies have been conducted evaluating the effects of nuts in a weight loss program on criteria for metabolic

syndrome. In a Spanish cohort (n = 50), Casas-Agustench, et al.[20] reported that 30 g/day of mixed nuts in a weight loss diet reduced fasting insulin and homeostatic model assessment (HOMA) as well as IL-6, a marker of inflammation. In a Chinese population, Wu, et al.[26] reported a similar reduction rate of metabolic syndrome in subjects who were counseled on the AHA guidelines, a heart healthy diet with flaxseed or walnuts (30 g/day for each). However, the reversion rate of central obesity was higher in the flaxseed and walnut treatment groups. Interestingly, the number of metabolic syndrome criteria was significantly reduced in the walnut group versus the control group. For both diabetes and metabolic syndrome, there are few nut studies. Nonetheless, the limited evidence is suggestive of health benefits.

Nuts and Cancer

Limited epidemiologic data exist which evaluate the relationship between nut consumption and cancer risk. In a review of the literature, Gonzalez and Salas-Salvado[27] noted that a problem in evaluating the data is that nuts, seeds and legumes are often aggregated. Consequently, only suggestive evidence exists of a protective effect on colon, rectal and prostate cancer. In contrast, animal studies are more compelling for a protective effect of walnuts on mammary gland tumorigenesis and colorectal cancer.[28,29] In a transgenic mouse model predisposed to mammary gland tumors, a walnut-containing diet fed from the time of weaning significantly reduced tumor incidence, multiplicity, and size.[28] Compared with corn oil fed mice, mice injected with HT-29 human colon cancer cells, tumor growth was significantly slowed in walnut fed and flaxseed fed mice by 27% and 43%, respectively.[29] Tumor weight also was reduced by 33% and 44%, respectively. While the animal data demonstrate benefits of walnuts on certain cancers and epidemiologic studies suggests nut consumption reduces the risk of certain cancers, further research is needed to get a clear understanding of how nut consumption affects cancer risk.

Nuts and Cognition

There is some exciting new evidence that suggests that walnut consumption may confer beneficial effects on cognitive function in animals[30,31] and humans.[32] Walnut extracts have been shown to protect against age-related cellular dysfunction in aged rats.[30] In a cell culture model, walnut extracts reduced amyloid beta-protein mediated cell death.[31] Amyloid beta-protein is a major component of senile plaques in individuals with Alzheimer's disease. In the PREDIMED Trial (described below), participants assigned to the Mediterranean diet with nuts had an improvement in plasma brain-derived neurotrophic factor (BDNF).[32] BDNF is a member of the neurotrophin family of growth factors that function to support the survival of existing neurons, as well as promote the growth and differentiation of new neurons and synapses. There are important ramifications of the current research; however, further studies are needed to advance our understanding about the effects of nuts on cognitive function.

Experiences from the Prevencion con Dieta Mediterranea (PREDIMED) Trial and Other Clinical Studies

The lower incidence of cardiovascular disease amongst those who live in the Mediterranean area suggests that the Mediterranean diet provides a healthier eating plan and may be the key to lowering disease risk. Identifying what role the diet, or parts of the diet play in disease prevention has been the focus of numerous of studies with somewhat varying outcomes, which may be a result of disparities within the Mediterranean diet.

Mediterranean Diet. The Mediterranean diet is defined in different ways ranging from the definition in Segen's Medical Dictionary – "A diet that differs somewhat by country, but which is generally characterized by increased consumption of olive oil, complex carbohydrates, vegetables, fish, and decreased red meat and pork consumption" to that of Oldways Preservation Trust; "The Mediterranean Diet is a way of eating based on the traditional foods (and drinks) of the countries surrounding the Mediterranean sea."[33,34] The core of the Mediterranean diet is reliance on plants, specifically vegetables, grains, fruits, nuts, beans, legumes and seeds. For many countries fatty fish, such as tuna, mackerel or sardines, is also a part of the menu and the use of other animal proteins is limited. The fats of predominance are olives, olive oil, nuts and seeds. Plant foods provide a wide variety of nutrients, phytonutrients and fiber all of which contribute to the prevention of cardiovascular disease. The inclusion

of fatty fish provides a healthy intake of omega-3 fatty acids which have been shown to aid in the reduction of blood triglycerides, prevention of blood clotting and reduction of inflammation. Since fatty fish is not consumed in all countries of the Mediterranean, the question of the possible role of walnuts, the nut with the highest level of omega-3 fatty acids in the form of alpha-linolenic acid (ALA), has been a focal point of several research studies. Walnuts contain 2.57 grams of ALA per ounce.

Research Study Outcomes. In the "A Walnut Diet Improves Endothelial Function in Hypercholesterolemic Subjects: A Randomized Crossover Trial" Ros, et. al. looked at how a traditional Mediterranean diet compared to a Mediterranean diet where walnuts provided 18% of total energy.[10] The small scale, randomized, controlled, crossover trial evaluated 21 hypercholesterolemic men and women who followed each diet for four weeks; 18 subjects completed the trial. Endothelial function improved in 12 subjects who followed the walnut diet versus just the Mediterranean diet.

As a follow-up to this smaller scale study the "Prevention with the Mediterranean Diet" PREDIMED study, a Spanish, multi-center, parallel, randomized, six-year clinical trial was designed. PREDIMED followed 7500 subjects who were at risk for cardiovascular disease but did not exhibit any disease. The trial consisted of men between the ages of 55-80 years and women ages 60-80 years. All subjects either had type 2 diabetes or had three or more risk factors for cardiovascular disease where the risk factors included, smoking, hypertension, elevated LDL-C, low HDL–C, overweight or obese or a family history of cardiovascular disease.[35]

A one year report was published in Archives of Internal Medicine in 2008. The study, "Effect of a Mediterranean Diet Supplemented with Nuts on Metabolic Syndrome Status: One Year Results of PREDIMED Trial" found that after one year, prevalence of metabolic syndrome dropped by 13.7% in the Mediterranean diet plus nuts group (which consisted of 50% walnuts and 25% each of almonds and hazelnuts), double that of the olive oil group and six times the control group.[35] The PREDIMED study was published in the New England Journal of Medicine in February 2013, showing a 30% reduction in risk of cardiovascular disease and a 49% reduction in risk of stroke in the Mediterranean diet supplemented with mixed nuts group compared to the low-fat group.[36]

The PREDIMED study design consisted of three groups of subjects; a control group and two Mediterranean diet groups. The control group followed a diet that was similar to the American Heart Association or the National Cholesterol Education Program low-fat diet and received dietary counseling at baseline and were provided with low fat diet leaflets yearly for three years. One Mediterranean diet group consumed a liter of olive oil each week and the other Mediterranean diet group consumed 15 grams of walnuts, 7.5 grams of almonds and 7.5 grams of hazelnuts each day for a total of 30 grams or approximately one ounce of mixed nuts. The two Mediterranean diet groups also received nutritional education to help them adhere to the diets. Subjects in the trial were randomized to assure matched characteristics of weight, age, smoking, disease incidence and medication use. Diet recalls were completed to provide nutritional information on key food group intake differences in the two Mediterranean diet groups versus the control. The analysis shows the variation in fatty acid distribution based on consumption of nuts versus the olive oil. After one year, the incidence of metabolic syndrome (MetS) declined in all three study groups. The group that consumed the mixed nuts showed the largest drop in incidence of MetS. Assessment after three months showed significant declines in fasting blood glucose, fasting insulin levels and a smaller reduction in the homeostatic model assessment (HOMA) (score estimates a person's insulin sensitivity or resistance), in both of the Mediterranean diet groups. Those on the low-fat diet actually showed an increase in all three.

A study by Vinson and Cai[37] investigated the antioxidant capacity of nuts. The researchers analyzed the catechin content of eight different nuts as well as creamy and crunchy peanut butter. The nuts were assessed in both their raw and roasted forms. The results suggest that in the US, nuts provide 162 mg of polyphenols each day or 19% of the 954 mg per day intake of polyphenols from all food sources. Polyphenols bind lipoproteins helping to reduce oxidation, improve the lipid profile and improve endothelial function, thus reducing inflammation. Inclusion of nuts,

especially raw walnuts, which contain 575 mg of polyphenols in a 1 ounce portion, may be the component of nuts that helps change endothelial flow in both of the previous studies.[37]

Practical Implications. The PREDIMED trial included nutrition consultations, motivational interviewing and group sessions, for the two Mediterranean Diet groups.[38] Assessment of diet adherence found that subjects who received the nutritional consults had better diet compliance. One of the key factors in assessing the value of dietary change is the ability to adhere to the diet for longer than just the length of a study. In assessing adherence to the PREDIMED diet, the two Mediterranean diet groups had diet changes at 12 months that mimicked those they had achieved at three months, indicating a better ability to maintain the dietary changes. Along with assessing dietary adherence, markers of cardiovascular disease improved in both of the Mediterranean diet groups. This study supports the practical aspects of dietary compliance for the two Mediterranean diets.

While research studies continue to look at the right balance of fats, making small changes in meal plans utilizing healthy fats can help promote cardiovascular health. A few simple tips include the following:

- Use olive oil instead of butter for bread and preparation (sautéing)
- Add walnuts to dishes or use them to replace other less nutrient dense foods. For example, top your salad with seasoned walnuts instead of croutons or instead of using breadcrumbs as a coating, use finely chopped walnuts.
- Consume fatty fish such as salmon in place of other animal proteins.
- Boost frequency of vegetarian dishes
- For a simple dessert, top Greek yogurt with walnuts and drizzle with honey

Implications for the Nutrition and Dietetic Community

There is impressive evidence that nuts have cardioprotective effects and other beneficial health effects. Whereas we have a robust evidence base for some diseases, for others our current understanding is in the early phases. Despite this, there are dietary recommendations for nut consumption. Dietitians need to be aware of the current research and dietary recommendations so that they are best positioned to counsel patients about how to include nuts in a healthy diet. It also is imperative that dietitians keep abreast of emerging research on nuts so that they are well informed and respond accurately to patient, colleague, and media inquiries about nuts and health.

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Member Spotlight

Suzanne ("Suzi") Domel Baxter, Ph.D., R.D., L.D., F.A.D.A

Erin Gaffney-Stomberg, PhD, RD

Suzanne ("Suzi") Domel Baxter, Ph.D., R.D., L.D., F.A.D.A

For this edition of The Digest, we are spotlighting RDPG member Suzanne ("Suzi") Domel Baxter, PhD, RD, LD, FADA. Dr. Baxter is a Research Professor in the Institute for Families in Society in the College of Social Work at the University of South Carolina, Columbia, SC. She conducts nutrition research, usually as Principal Investigator or Co-Investigator. As such, she writes research grants to obtain funding; obtains approval from the Institutional Review Board to conduct research; recruits school districts, schools and children to participate in research; hires, trains, and supervises research staff; prepares protocols for data collection, entry, and coding; conducts team meetings; collaborates with statisticians, co-investigators, and consultants on analyses and interpretation of results; writes abstracts and manuscripts of research results; gives poster and oral presentations of research results; reviews manuscripts for peer-reviewed journals; serves on the Board of Editors for the Journal of the Academy of Nutrition and Dietetics; reviews grant applications for NIH on an ad hoc basis; writes book chapters; and participates in professional activities at the local, state, and national levels. Read our interview with Dr. Baxter below to learn about her interesting career as an academician.

Dr. Baxter, please tell us about your background. How did you get to where you are now?

After receiving an Easy Bake Oven as a Christmas gift when I was 6 years old and baking every mix within a couple of days, I knew my career would involve food! I started college at Valparaiso University, IN, as a double major in home economics and music. Because I was born and raised in the Dallas, TX area, I lasted one semester in the cold north before begging my mother to let me come home, attend community college, and regroup. That fall, I started attending Texas Christian University in Fort Worth and three years later (1981), graduated with a BS having completed a coordinated undergraduate program, and became a registered dietitian. However, I knew clinical dietetics was not for me so I pursued non-clinical opportunities. My past experiences include working as an out-patient RD at the following: at a residential school for cerebral palsy children; in school foodservice program; and with the Women, Infants, and Children Program. At that point in my career, I decided to go to graduate school. For several years during graduate school, I worked as a graduate assistant which involved teaching and counseling in the nutrition center. My thesis and dissertation involved developing and pilot-testing weight loss programs for low-income black and Hispanic women in Dallas, TX. After receiving my masters' followed by a PhD in 1990 from Texas Woman's University in Denton, TX, I accepted a post-doctoral fellowship at the Medical College of Georgia under Dr. Tom Baranowski and Dr. Bill Thompson for 2.5 years. After completing my fellowship, I remained at the Medical College of Georgia for the next 10 years as an Assistant and then Associate Professor, with my responsibilities centered primarily around research. In 2003, I became a Research Professor at the University of South Carolina in Columbia where I currently work.

What is your current research interest?

The primary area of my work is methodological research concerning the accuracy of children's dietary recalls. This work is funded through the NIH Research Project Grant Program (an R01 grant). This R01 grant is a four-year project with fourth-graders with three aims to identify the combination of retention interval and prompts that maximizes dietary recall accuracy. Aim 1 compares dietary recall accuracy using a 2 x 4 factorial design: prior-24-hour recall obtained in the afternoon or previous-day recall obtained in the morning combined with four prompts (forward; reverse; open; meal). Each of 480 children is observed eating school breakfast and school lunch, and then interviewed to obtain a 24-hour dietary recall using one of the eight conditions, with 60 children (30 per sex) per condition. Aim 2 assesses relationships of social desirability, food security, body mass index, socioeconomic status, and achievement test scores with dietary recall accuracy of the 480 children in Aim 1. Aim 3 investigates one-month test-retest reliability for a 14-item social desirability survey and a 5-item food security survey via classroom administration to approximately 100 children.

The secondary area of my work concerns childhood obesity. A current project is funded through the NIH Exploratory/Developmental Research Grant Award (an R21 grant). This grant is a two-year secondary-analyses project that uses data from a previous R01 grant for 1,542 fourth-grade children from schools in

Augusta, GA. This grant encompasses 2 Aims. Aim 1 investigates a possible relationship between childhood obesity and participation in school meal programs. Aim 2 examines behaviors and selections in a subset of 344 children to understand differences in actual (observed) school-meal intake.

Although NIH funding has ended, we are writing manuscripts for a third grant with two aims. Aim 1 was to develop, pilot-test, and validate an interview protocol to be used with children. This protocol utilized an integrated recall of dietary intake and physical activity that occurs at school. Aim 2 determined the influence of grade (third through fifth) and retention interval (same school day with an afternoon interview; or the previous school day with a morning interview) to validate our interview protocols for diet and/or physical activity.

How did you become involved / interested in your current line of research?

I thank Dr. Tom Baranowski for exposing me to the research bug and giving me the idea for methodological research! After receiving my PhD, I interviewed with Tom and, when he offered me a position, I told him I had another offer to teach. He asked me whether I wanted to keep reinventing the wheel, or whether I wanted to investigate what makes the wheel roll faster and further. I embarked on a post-doctoral position with Tom performing research. My work experience with Tom instilled my interests conducting methodological research concerning the accuracy of children's dietary recalls.

What advice would you give to a young researcher for developing a successful line of research?

Find one or more mentors or experienced collaborators. Learn from the critiques provided by reviewers. This is especially true with NIH grant applications. Some get funded on first submission while others take several revisions (e.g new title and one or two new aims) before being funded. If you truly believe you have a good idea, keep applying until funding is obtained. Offer to serve as a grant reviewer for NIH to help learn the process. Be willing to think outside the box. Some of the best collaborators in my career have been cognitive psychologists. Their expertise in memory crosses paths with my primary research focus.

What are your career goals?

I would like to reach and exceed the century mark for research publications in refereed journals; be more involved professionally at the national level; and be a mentor to one or more post-doctoral fellows. I enjoy serving others and my profession. One of my favorite sayings is, "We make a living by what we get, but we make a life by what we give."

How has your affiliation with the Academy impacted your career progression?

Because of my affiliation with the Academy, I was awarded the Kraft-General Foods Foundation, Inc. Fellowship (through what was then ADA Foundation) for two consecutive years during my dissertation research. Being awarded this generous fellowship impacted my career. It allowed me to focus my time and effort to complete my PhD. The Fellowship has impacted my ability to increase my donations and efforts to the AND Foundation for Research. In honor of a dear friend and research colleague, Amy Joye, MS, RD, I led the effort with Amy's family and friends, the South Carolina Dietetic Association, and the Georgia Dietetic Association to create a Named Fund through what was the ADA Foundation (now the Academy Foundation) to raise a minimum of $50,000. The $50,000 minimum endowment level needed to establish the Amy Joye Research Fund was reached in March, 2009, just a few months prior to Amy's passing. The principal will remain intact in perpetuity and the interest will be used to award nutrition research grants on an annual basis. To date, three Amy Joye Memorial Research grants have been awarded. Although Amy is no longer with us, her memory and potential continue to bless our profession and nutrition research.

If someone were to ask you to explain why research is important to the field of dietetics, what would you say?

Dietetics is science; every science progresses to the degree to which its research has been developed and refined. We become what we behold. We shape our research, and then our research shapes us. Research is our future.

Hot Topics in Food Science and Health: Impact of Food Matrix on Satiety, Intake, and Weight

Robin A. Ralston, MS, RD
Department of Food Science and Technology, The Ohio State University

Food matrix is defined by the USDA as "the nutrient and non-nutrient components of foods and their molecular relationships, i.e. chemical bonds, to each other".[1] Because the matrix of a food can impact the rate of digestion and absorption, it has potential to impact satiety, energy intake, and in the long-term, weight change. The impact of the matrix of fruits and vegetables plays a role in the development of healthy eating guidelines. In fact, the 2010 Dietary Guidelines for Americans suggests that fresh, canned, frozen and dried fruits should be chosen over 100% fruit juice, especially in overweight or obese children and adolescents.[2] This is a recommendation that is not too difficult to follow for most Americans, as recent data shows that fresh fruit is the most common and fastest growing snack food consumed in the U.S.[3]

Several studies support choosing solid fruits over fruit juices. In one study, a preload of one of four forms of apple – peeled raw apple, applesauce, apple juice, or apple juice plus soluble fiber – was consumed by 58 adults on four separate occasions followed by a standard test meal a*d libitum*.[4] All preload foods were matched for weight and energy, and all except regular apple juice were matched for dietary fiber. The amount of the test meal consumed was compared following the four apple preloads or no preload (control). While participants consumed less of the test meal when any preload was given, both the fresh apple and applesauce preloads resulted in lower energy intakes of the entire meal (test meal + preload) compared to control and both apple juice preloads, indicating greater than 100% dietary compensation. Energy intake after the fresh apple was lower than after the applesauce. Fiber has previously been suggested as a reason fresh fruit elicits greater satiety than juice.[5] This study suggests additional factors are involved because even with equal fiber levels, fresh apple resulted in lower energy intake than apple juice with fiber. Alternatively, because the intact cell walls of fresh apple create a larger volume than the applesauce or juice, the fresh apple could fill more space in the stomach, reducing food intake.[6] The act of chewing could additionally impact total energy intake and satiety,[7] also favoring the fresh apple.

In a similar study, participants consumed a preload of solid fruits (raw apple and grapes, dried apples, raisins, and water), a preload of fruit juices (apple and grape juices containing soluble fiber), or no preload (control), followed by an *ad libitum* test meal.[8] Preloads were matched for energy, volume, and dietary fiber. In overweight/obese participants, hunger was significantly higher following the meal with the beverage preload compared to the solid preload. This result was not seen in lean participants. Again, participants consumed less of the test meal with all preloads, but total energy intake of the complete meal (test meal + preload) was higher with the beverage preload compared to both the solid preload and no preload and was not impacted by weight status of the study participant. Again, chewing may have played a role, but because both treatments had equal volumes, the variable of volume was eliminated. This short-term study was continued with an 8-week intervention supplementing usual dietary intake with either liquid or solid fruits and vegetables throughout the day.[9] Interventions were matched for energy and fiber but not volume. Due to differences in energy intakes, lean participants gained weight during the beverage intervention only, while the overweight/obese group gained weight during both the beverage and solid interventions. While the inclusion of dried fruit in the solid intervention can confound the results, fruits and vegetables in liquid form may satiate less, result in increased energy intake at a meal, and lead to weight gain compared to solid fruits and vegetables, especially in adults who are overweight or obese.

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Chair's Report

Dear Research DPG Members

Chris Taylor, PhD, RD, LD

Being deeply rooted in the Midwest, I can attest that extreme climatic events (tornados, blizzards, flood, etc.) can be difficult to predict. These often hazardous events bring two major thoughts to mind in the area of dietetics. First, like the weather, it is difficult to predict the exact impact of nutrition in the promotion of health and chronic disease prevention, so we should not get hung up on minute details. Secondly, we should carefully consider what metrics we use to measure the success or failure of nutrition interventions.

Much like the weather, the dietetics research environment faces the same ebb and flow of opportunity and scrutiny. In the emerging markets of health care reform and financial saliency, dietetics professionals will need to be creative, have ingenuity, patience and persistence to forge ahead. Further, as nutrition experts, we must also rely on sound science, an understanding of all of the variables involved and our best estimates in order to predict how our efforts will improve health. Though these challenges seem daunting, we are ideally positioned to make our stamp on the future of health in the US and beyond.

Sincerely,
Chris Taylor, Research DPG Chair, 2012-13

Treasurer 's Report

Spring Greetings, Research DPG Members!

Karin Pennington, M.S., R.D., L.D.
RDPG Treasurer

Our annual budget allows for $24,035 of income and $24,076 in expenses. The fiscal year is from June 2012 to May 2013. We are doing well financially! Our current reserves are at 105%, with additional sponsorship revenue from the National Cattlemen's Beef Association and Solae not yet officially reflected in our totals. The goal is to keep reserves near 100%. Thank you to our sponsors for their generous support!

Also, we have recently submitted the Program of Work and Budget for the next fiscal year which will start in June. As with this year, the income will meet expenses as we strive to maximize RDPG member benefits. Thank you to our current members, and we look forward to serving you and others into the future!

Karin Pennington, M.S., R.D., L.D.
RDPG Treasurer

Research DPG 2012-13 Budget

		Annual Budget ($)	As of February ($)
Revenue	Membership	17,535	14,844
	Grants/Contracts	6,500	18,009
	Interest Income	—	2,430
		24,035	35,283
Expenses	Lodging/ Subsistence	993	2,892
	Transportation	5,700	1,461
	Professional/ Consulting	2,000	2,190
	Postage	450	24
	Teleconferences	230	65
	Member Dues/ Fees	1,203	261
	Outside Services	3,200	0
	Awards	4,600	4,400
	Audio Visual	0	2,916
	Food Service	5,000	11,773
	Printing/Copying	650	281
	Other	50	0
		24,076	26,263
	NET	**-1,653**	**9,020**
Reserve	**February 2013 Reserve**	**24,076**	**19,247**
	Reserve Percentage	**—**	**105%**

History of Food Allergies

Jody L. Vogelzang Ph.D., R.D., L.D., F.A.D.A., C.H.E.S.

Adverse reactions to foods, especially large dietary proteins in foods resulting in an allergenic response are referred to as a food allergy. Although food allergies are not a new development and occur in only a small percent of the population, diagnosis and treatment remain a topic of interest with practioners.[1]

In the United States it is estimated that approximately 6-8% of children and 3-4 % of adults display true food allergies.[2] In using data gleaned only from hospital discharge records, Chafen, et al. (2010)[3] identified that the number of diagnosed food allergies has increased over time. According to their research in 1997, 3.3% of US children had a diagnosed food allergy and 10 years later this number increased to 3.9%, which was considered a statistically significant difference.[3] The cause of this increase was unclear; it could be attributed to an actual increase in an immune triggered reaction to food or better reporting mechanisms. Nonetheless, food allergies remain a public health problem, accounting for over 30,000 severe allergic food reactions each year, including hundreds of deaths.[2]

Causation and Symptoms

Food allergies result from abnormal immunologic reactions to the protein in foods. Traditionally, these reactions have been categorized by those that are immunologically mediated (IFA) by immunoglobulin (Ig)E, and all others which are nonimmunologically non-IgE mediated (NFA).

In IgE- mediated reactions, symptoms are rapid, and onset can begin in seconds or as long as two hours after the ingestion of the food substance. In this type of hypersensitivity, the onset is rapid and the duration of the event is short but not without the possibility of severe consequences.

IgE mediated reactions are usually accompanied by symptoms relating to histamine production including flushing, swelling of the lips, face or throat, urticaria and pruritus. In some cases gastrointestinal (GI) involvement occurs and is demonstrated by cramping, nausea and diarrhea.

Non-IgE mediated food allergies were first considered to be a benign condition mediated by cellular immune responses, primarily affecting the GI mucosa. More recent research has focused on "subtle changes in interactions between environmental factors (microbiota, dietary components, etc.) and the gut immune responses… which can result in undesired adverse reactions to food proteins (FPs)."[4]

The onset of symptoms in NFA occurs more slowly than in IFA making the food offender difficult to assess. The lack of easily accessible diagnostic measures also contributes to the problem.[5] Clients suffering from NFA may report symptoms as widely ranging as joint pain, chronic headaches and irritable bowel disease. Non–IgE-mediated gastrointestinal food allergies include food protein–induced enterocolitis syndrome (FPIES), food protein–induced proctocolitis and food protein–induced enteropathy.[6] While not life threatening, these symptoms affect quality of life and may cause morbidity in rapidly growing infants and toddlers when the NFA is caused by infant formula proteins.

The cause of the NFA appears to be related to the ability of the gut to recognize and tolerate large dietary proteins (DPs). Immune tolerance to (DPs) is partially maintained by active suppressive mechanisms involving antigen (Ag)-specific regulatory T cells.[5]

Healthy subjects without a food allergy frequently have low concentrations of food-specific IgG, IgM, and IgA antibodies in their serum. Food protein–specific IgG antibodies tend to rise in the first months after the introduction of a food and then begin a gradual decline, even though the DP continues to be ingested.[6] If homeostasis is not reached and antibodies remain high, an inflammation of the gastric mucosa will occur.

To further complicate the diagnosis and treatment of NFA and IFA, both IgE and non-IgE mediated reactions can occur in some eosinophilic GI disorders which involve the gastric mucosa.[4]

The role of food proteins

Allergic reactions to egg, milk, peanut, tree nuts, fish, shellfish, wheat and soy account for most significant food allergies in the United States, although any food can trigger an allergic response (see Table 1).[7]

Jenkins et al. compared animal food allergens and their human homologs by evaluating protein families, sequence analysis and evolutionary relationships. They noted that "sequence identities to

Table 1

Estimated food allergy rates in North America		
Prevalence	**Infant/child**	**Adult**
Milk	2.50%	0.30%
Egg	1.50%	0.20%
Peanut	1.00%	0.60%
Tree nuts	0.50%	0.60%
Fish	0.10%	0.40%
Shellfish	0.10%	2.00%
Wheat/soy	0.40%	0.30%
Sesame	0.10%	0.10%
Overall	5%	3-4%

human homologs of greater than 62% typically excluded the protein from being allergenic in human subjects."[8] This evolutionary look at protein relationships may offer more diagnostic clues when evaluating adverse food reactions. Interestingly, food preparation appears to affect allergenicity. It is thought that preparation methods may explain the higher rates of peanut allergy in the US and other westernized countries, where peanuts are consumed after roasting. China has lower prevalence rates of peanut allergies which may be attributed to the consumption of peanuts after they have been boiled or fried. The high temperature of roasting (180 °C) peanuts leads to a Maillard reaction that appears to increase protein stability and allergenicity.[9] [10]

Diagnosis

Food allergies are frequently misdiagnosed by consumers, resulting in a higher number of self-reported cases than are found when clinical testing occurs. In one study, self-reported cow's milk allergy was claimed to occur in 3.5% of the research population while testing of the same population found that the rate of cow's milk allergy was actually 0.6%-0.9%.[3]

Expert interviewing skills are needed to assess symptomology, to determine time of onset and to obtain a list of foods consumed around the time of the adverse reaction. Many times the food is unusual in the diet and may be identified with careful questioning. Questions regarding new ingredients added to previously well-tolerated dishes may prove to be the allergen. In other instances the allergen may be introduced through possible cross-contamination. This may occur in food buffets, salad bars, food manufacturing and processing, and the reuse of oil in a deep fat fryer. In addition to questions regarding food consumption, it has been documented that exercise, close to the time of the adverse reaction, may have served as a trigger instead of an actual food. Medications and environmental exposures may also serve as triggers in a small number of people.[11]

Clinical laboratory testing such as Mediator Release Testing for NFA and for IFA antibody titers, elimination diets, skin prick tests and oral challenge tests can help narrow the field of client specific food allergens.

Conclusion

An adverse reaction to a food substance can be classified as IgE mediated or non IgE mediated. These classifications differentiate the onset, symptomology and severity of the reaction. While IgE mediated allergies have been well researched, non IgE allergies have been topics of more recent discovery and involve intricate interactions between large dietary proteins and the gut mucosa. The NFA is a topic of much discussion in the Academy of Nutrition and Dietetics Integrative and Functional Medicine listserve as members share expertise in uncovering dietary triggers in the elimination of adverse food reactions in clients.

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Upcoming Conferences

May 22-25, 2013
Annual Meeting of the International Society of Behavioral Nutrition & Physical Activity
(Ghent, Belgium)
Call for abstracts: Closed
Website: *http://www.isbnpa2013.org*

August 9-12, 2013
Society for Nutrition Education and Behavior Annual Conference
(Portland, OR)
Call for abstracts: Closed
Website: *www.sneb.org*

September 15-20, 2013
IUNS 20th International Congress of Nutrition
(Granda, Spain)
Call for abstracts: Closed
Website: *www.icn.2013.com*

October 19-22, 2013
Food and Nutrition Conference Expo
(Houston, TX)
Call for abstracts: Closed
Website: *http://www.eatright.org/fnce/sessionproposals*

June 21-25, 2013
American Diabetes Association 73rd Scientific Sessions
(Chicago, IL)
Call for abstracts: Closed
Website: *http://professional.diabetes.org/Congress_Display.aspx?TYP=9&CID=91271&loc=dorg-homepage*

November 12-16, 2013
Obesity Society Annual Scientific Meeting
(Atlanta, GA)
Website: *http://www.obesityweek.com*

Student Research

Association of Urinary Levels of Estrogens and Estrogen Metabolites with Mammographic Density in Postmenopausal Women

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INTRODUCTION

Breast cancer is the most common and second deadliest cancer of women living in the United States. It is estimated that 232,340 new breast cancer cases in women will be diagnosed, and 39,620 women will lose their lives as a result of breast cancer in 2013.[1]

Currently, there is convincing evidence from epidemiological, animal, and in vitro studies that endogenous sex hormones, particularly estrogens, play a critical role in the etiology of breast cancer.[2] Sex steroids, including estrogens, promote cellular growth and proliferation,[3] and their circulating and urinary concentrations are elevated in hormone-dependent breast cancers.[4-6]

The metabolism of parent estrogens, estradiol and estrone, first starts with irreversible hydroxylation through the action of cytochrome P450 (CYP450) enzymes. This leads to the conversion of the parent estrogens to catechol estrogens including 2- and 4-hydroxyestradiols (2- and 4-OHE2) and 2- and 4-hydroxyestrones (2- and 4-OHE1), and 16-α-hydroxyestrone (16-α-OHE1). Catechol estrogens are further metabolized (methylated) to methoxyestrogens (e.g., 2- and 4- MeoE2, and 2- and 4- MeoE1) by the catechol-O-methyltransferase (COMT) enzyme (Figure 1). The COMT gene is polymorphic; a single G to A transition at codon 158 of COMT (SNP rs4680) results in a 3- to 4-fold decrease in enzymatic activity (GG vs. AA genotype). Also, individuals with heterozygous genotype (A/G) show intermediate levels of COMT activity.[7-8] Given the role of the COMT enzyme in the conversion of catechol estrogens to methoxyestrogens, any genetic variation in this enzyme might influence the risk of breast cancer as a result of significant changes in the estrogen metabolites levels.[9] Therefore, it is intriguing to speculate that individual genetic variability in the COMT enzyme may affect breast density and consequently influence breast cancer risk.

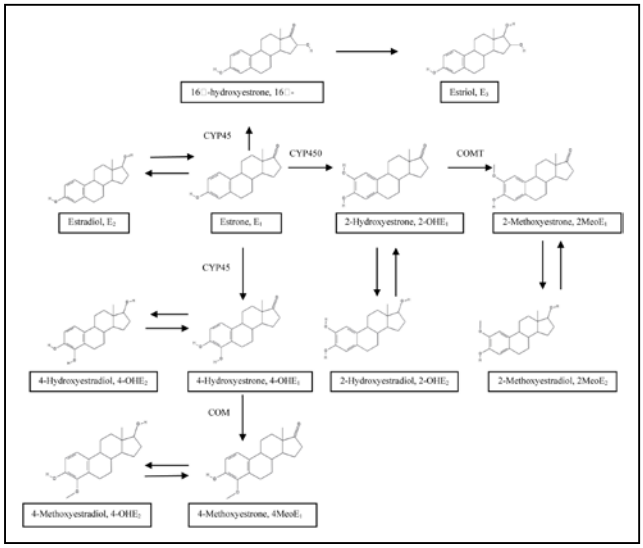
Mammographic density is a measure of the amount of fibroglandular tissue that appears on a mammogram. This measure is compared to the fat content in the breast tissue and is usually expressed as percent mammographic density (PMD). Mammographic density is a strong established risk factor for breast cancer.[10] It has been shown that high breast density (more than 75%) is linked with 4-6 times greater risk of breast cancer compared with no densities;[10-14] however, the involved mechanisms have not yet been fully elucidated. One of the proposed mechanisms through which mammographic density may modify breast cancer risk is by means of sex steroids.[15-17]

To date, the published data on the association between estrogens and mammographic density is scarce with mixed results. Current data is mostly limited to the circulating levels of estrogens and not the urinary hormones and their metabolites.[18-23] The purpose of this study is to test the hypothesis that estrogens and their metabolites are directly related to breast density. We also tested a secondary hypothesis that this effect is modified by the genetic variation in the COMT genotype within a cross-sectional analysis of postmenopausal women.

MATERIALS AND METHODS

Participants' eligibility and recruitment. The study sample used for this paper is a sub sample of a larger parent clinical trial "Green Tea and Reduction of Breast Cancer Risk." The parent trial is a randomized, double-blind, placebo-controlled trial. It aims to investigate the effects of green tea catechin intake on well-established biomarkers of breast cancer risk in 1000 healthy

Figure 1
Endogenous estrogen metabolism.



postmenopausal women at high risk of breast cancer due to dense breast tissue. Biomarkers that are studied include mammographic density, circulating and urinary reproductive hormones and their metabolites, insulin-like growth factor axis proteins, as well as oxidative stress. In addition, a possible differing effect of COMT genotype will be examined in all of the above biomarkers. Eligible participants for this sub sample analysis (n=208) were healthy postmenopausal women at increased risk of breast cancer due to high breast density, aged 50-70 years, non-smokers who do not regularly consume green tea (more than one cup of green tea per week) and have not taken hormone replacement therapy within six months of being screened for this study. Additional inclusion criteria include: alcohol consumption equal or less than 7 servings/week (one alcoholic serving size was defined as 12-oz of beer, 4-oz of wine, or 1.5-oz of liquor), BMI 19-35 kg/m^2, stable weight (less than 10 pounds change) for the past year, no previous diagnosis of breast cancer or proliferative breast disease, and no elevated levels of liver enzymes (above 1.5 times the upper limit of normal). Recruitment was conducted at the University of Minnesota Medical Center (UMMC), Fairview Southdale, and Fairview Maple Grove Breast Clinics in Minnesota.

The study radiologist supervised the reviews of the mammogram reports and the identification of those women with "heterogeneously dense" or "extremely dense" breasts. "Heterogeneously dense" breasts are approximately 51-75% glandular, and "extremely dense" breasts are >75% glandular. Once identified, an IRB approved recruitment letter was sent to prospective subjects explaining the intent of the study (IRB Approval Code Number: 0806M36121). If interested, the prospective subjects would call a screening phone number and subsequently attend an orientation session where the study was explained in more detail. Written informed consent was obtained at the end of each orientation session. Consented subjects then came for a screening in which blood was drawn for assessing the liver function status and COMT genotyping, and height and weight were measured to calculate BMI. Based on the results of the screening, final eligibility status of each participant was determined. If the participant was found eligible, she was scheduled for her baseline clinic visit, and she was randomized into either the green tea extract or placebo group. At baseline clinic visit, blood was drawn for measurement of all of the parent study biomarkers, and all subjects received their study supplements/ placebo. Data used for this paper is cross-sectional in nature, and only participant's baseline information, prior to beginning supplementation or placebo, was used in this analysis.

Urine collection and measurement and analysis of hormone levels. Subjects were instructed to collect urine for 24 hours prior to their morning baseline clinic visit times. They were asked to refrigerate the urine sample and avoid alcohol consumption during the 24 hour urine collection. Collected urine volumes were measured, and urine samples were aliquoted and stored at -80º within 2 hours upon receipt. Twelve urinary estrogens, including the primary estrogens and their metabolites, were analyzed by the liquid chromatography-tandem mass spectrometry (LC-MS/MS) assay developed by Xu *et al*[24] as modified by our laboratory. All samples were measured in duplicate to ensure the accuracy of measurements.

COMT genotype. DNA was extracted from buffy coats of peripheral blood samples using a DNeasy Blood & Tissue Kit (QIAGEN Sample and Assay Technologies). TaqMan assays were developed for determining the COMT polymorphism gene variant using a TaqMan Drug Metabolism Genotyping Assay (Applied Biosystems).

Mammographic density measurement. The left cranio-caudal view of the digital screening mammograms of each participant was assessed to determine the PMD. Reading of the participants' mammograms was done by an experienced reader using the computer-assisted Madena method developed at the University of Southern California in which total breast area and dense area are quantified.[25] Percent density was calculated by dividing the dense area by the total area multiplied by 100. In order to measure the reliability of the percent density readings for quality control, approximately 10% of the total mammograms were read twice where the intra-class correlation coefficients of the duplicate readings were found to be more than 95%.

Statistical analyses. Urinary levels of estrogens and their metabolites were not normally distributed, so they were analyzed on the log-scale and are reported as medians and ranges. Association between the urinary estrogens and mammographic densities was determined using Spearman correlation coefficients adjusted for age, BMI, COMT

genotype and reproductive history factors. Data were analyzed using SAS software, version 9.2 (SAS Institute Inc.), and the value of P < 0.05 was considered statistically significant.

RESULTS

Selective characteristics of the study participants are summarized in Table 1. The mean +/- the standard deviation (SD) age of participant was 60.0 (5.1) years. Approximately 44% of the participants were either overweight or obese (BMI ≥ 25 kg/m^2). Most of the participants were white (96.1%) and non-Hispanic (98.1%), parous (73.3%), never smoked (67%); 83% had a previous history of using birth control, and 46% had a history of using HRT. The mean (SD) percent breast density was 32.3% (16.8). As expected in the Caucasian populations, about half of the participants possessed the COMT AG genotype, 18% had the GG and 27% had AA genotypes.

Table 2 displays the distribution of urinary estrogens and their metabolite concentrations and age and BMI. Parent estrogens comprised 28.2% of the total estrogens and metabolites, and were statistically significantly correlated with the hydroxylated estrogens including 2, 4, and 16-hydroxylated metabolites (P<0.0001 for all metabolite pathways). In contrast, estrogens and hydroxylated metabolites were negatively associated with age (P<0.05 for all of them) but non-significant positively related with HRT; however, their correlation direction with BMI was mixed and mostly non-significant (data not shown).

PMD was significantly and inversely associated with age and BMI (the Spearman correlation coefficient were -0.15 and -0.3, P= 0.03 and <0.0001, respectively). As shown in Table 2, the majority of primary estrogens and their metabolites, except 2-methoxy estradiol, were inversely associated with the PMD. However, this relationship was weak and never reached the statistical significance level. These results remained unchanged after further adjustment for age, BMI, reproductive and dietary factor covariates mentioned in Table 1. Further adjustment for the effect of COMT genotype on breast density and estrogen levels was performed, and no interaction was found for either the PMD or estrogens and their metabolites. Since age and BMI adjusted Spearman coefficients were not statistically significant for any estrogen metabolites, the associations between mammographic density and different hormones were no longer investigated based on their concentration categories.

Table 1
Baseline characteristics and reproductive histories of participants

Variable	n	Mean ± SD or %
Age, y	208	60.0 ± 5.1
BMI, kg/m^2	208	25.1 ± 4.0
≤24.9 (normal-underweight)	116	55.8%
25.0-29.9 (overweight)	70	33.6%
≥30.0 (obese)	22	10.6%
Race		
White	200	96.1%
Other	8	3.8%
Ethnicity		
Hispanic	4	1.9%
Non-Hispanic	204	98.1%
COMT genotype distribution		
GG	37	17.8%
AG	115	55.3%
AA	56	26.9%
Mammographic density, %	200	32.3 ± 16.8
Age at menarche, y	204	13.0 ± 1.6
Age at first live birth, y	148	32.2 ± 4.9
Number of live child birth	205	1.60 ± 1.2
Parity		
Nulliparous	55	26.7%
Parous	151	73.3%
Past use of birth control pills		
No	35	17.1%
Yes	170	82.9%
Family history of breast cancer		
No	122	58.7%
Yes	86	41.3%
HRT history		
No	110	53.7%
Yes	95	46.3%
Past smoker		
No	138	67.0%
Yes	68	33.0%
Current Alcohol intake		
No	47	22.7%
Yes	160	77.3%
Current black/green tea intake		
No	83	40.3%
Yes	123	59.7%
Current soy consumption		
No	136	68.0%
Yes	64	32.0%

NOTE: Values are presented as mean ± standard deviation (SD) for continuous variables, and n and % for categorical variables. BMI, body mass index; COMT, Catechol-O-methyl transferase; HRT, hormone replacement therapy.

Table 2
Urinary concentrations of estrogens and estrogen metabolites (µg/day), age, BMI, and corresponding Spearman correlations with PMD

Hormones	Median (range)[a]	Spearman correlation coefficient [b]	*P* value
Estrone	1.7 (0.2-21.3)	-0.13	0.07
Estradiol	0.4 (0.05- 7.9)	-0.11	0.14
Estriol	2.37 (0.004-22.9)	-0.05	0.51
2-Hydroxyestrone	1.79 (0.01- 25.5)	-0.03	0.64
4-Hydroxyestrone	0.23 (0.006- 4.7)	-0.07	0.29
16-Hydroxyestrone	0.18 (0.005- 4.1)	-0.01	0.89
2-Methoxyestrone	0.12 (0.003- 3.5)	-0.04	0.57
4-Methoxyestrone	0.02 (0.003- 0.5)	-0.04	0.55
2-Hydroxyestradiol	0.09 (0.002- 1.0)	-0.02	0.81
4-Hydroxyestradiol	0.01 (0.002- 1.1)	-0.13	0.07
2-Methoxy estradiol	0.04 (0.002- 0.8)	0.12	0.08
4-Methoxy estradiol	0.01 (0.003- 0.5)	-0.05	0.45
Total estrone and estradiol	2.15 (0.3- 29.2)	-0.12	0.09
Total estrogens and metabolites	6.98 (1.3- 85.9)	-0.08	0.28
Age (y)	59.8 (50.1- 71.2)	-0.15	0.03
BMI (kg/m^2)	24.2 (18.2- 38.6)	-0.3	<0.0001

NOTE: Values are presented as median (range) for baseline hormone levels, age and BMI. *P* values for baseline hormone levels are unadjusted for age and BMI.
[a] n=208
[b] n=200

DISCUSSION

The current literature is inconsistent regarding the associations between reproductive hormones in blood and/or urine and mammographic density. Most of the existing evidence comes from studies assessing the foregoing association in the blood, and their results vary from a null relationship to either a direct or inverse association.[18-22] In this study of postmenopausal women at increased breast cancer risk, higher urinary concentrations of primary estrogens and their metabolites were not associated with mammographic density. These null results remained unchanged with or without adjustment for age, BMI, hormone therapy history and COMT genotype.

To date, a total of eleven studies have evaluated the relationship between estrogens and mammographic density in postmenopausal women.[18-22, 26-30] Similar to the association direction found in this research, six previous studies[18-20, 28-30] have shown inverse associations between blood estrone or estradiol and PMD; however, three of these studies[19-20, 30] lost their statistically significant association after controlling for BMI. In contrast, three studies[21-22, 27] have found positive associations between circulating estrogens and PMD after adjustment of BMI. To the best of our knowledge, only one study[23] has investigated the association of urinary estrogens and their metabolites with PMD thus far. Fuhrman *et al* have reported positive correlations between estrone and estradiol with PMD; however, these associations did not reach statistical significance level. The only significant result found in their study was an inverse association between 2-methoxyestrone and 4-methoxyestrone and PMD. These conflicting findings in the studies can be due to several potential factors including different sample size, diverse study populations, difference in sex hormones analysis methods (e.g., immunoassays vs. LC-MS/MS), as well as restrictions in the present breast density quantifying procedures and techniques.

In the interpretation of our study results, some limitations should be considered. This study has a relatively small sample size compared with earlier studies, which had sample sizes up to 1400 participants. Another limitation is that this study was a cross-sectional study; therefore, inferring causal relationship is limited. In addition, urinary sex hormone concentrations were only measured at one time point which may not reflect the long-term levels and may not be an appropriate surrogate for breast tissue levels. Finally, a two month gap existed between the time of the mammogram and the baseline hormone measurements. We are unsure if this time gap may have influenced the hormonal measurements as compared to the breast density observed 2 months earlier. This study also has several strengths that should be noted. It is part of a large prevention clinical trial so we can update the current results with a much larger sample size of approximately 1000 postmenopausal women in the near future. We have also used digital screening mammograms, which have less limitation than using traditional film mammograms. Furthermore, the reader of the mammograms was blinded to the characteristics of the subjects. Mammograms were read twice, and both readings produced similar results. Lastly, analyzing the urinary estrogens and their metabolites was done using advanced LC-MS/MS methodology, which is very accurate and reproducible.

In conclusion, we found no evidence that urinary estrogens and their metabolites are associated with the mammographic density in healthy postmenopausal women at increased risk of breast cancer. Also, our findings do not suggest any significant interaction between COMT genotype and estrogens and mammographic density. These results should be evaluated and confirmed in larger studies.

ACKNOWLEDGEMENT

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Spring in to Beef & Heart Health: Research Sheds New Light on Role of Lean Beef in Improving Heart Health

By Shalene McNeill, PhD, RD, Executive Director, National Cattlemen's Beef Association, a Contractor to the Beef Checkoff Program. Every beef farmer and rancher and every beef importer contributes to a fund called the beef checkoff, which is used to support the Human Nutrition Research Program.

Many misperceptions exist about the science-based evidence on the role of beef in a heart-healthy diet. For example, limiting consumption of red meat, including beef, is often one of many strategies suggested for reducing the risk of heart disease; however, significant research published in 2012 demonstrates that lean beef can be enjoyed as part of a diet recommended for improved heart health, including lower cholesterol levels.

BOLD Research Indicates Eating Lean Beef, as Part of a Heart-Healthy Diet, Can Help Improve Cholesterol Levels

The 2010 Dietary Guidelines for Americans and many health professionals recognize the Dietary Approaches to Stop Hypertension (DASH) eating plan as an evidence-based dietary pattern for a heart-healthy lifestyle. The DASH eating plan emphasizes vegetables, fruits, and low-fat milk and milk products; includes whole grains, poultry, seafood, and nuts; and is lower in sodium, red and processed meats, sweets, and sugar-containing beverages than typical intakes in the United States. Published in the January 2012 edition of the *American Journal of Clinical Nutrition*, the **"Beef in an Optimal Lean Diet" (BOLD) study offers evidence for increasing the flexibility of the DASH eating plan to include more frequent servings of lean beef while improving cholesterol levels.**[1]

The BOLD study utilized a randomized, crossover, controlled feeding design to measure the impact of healthy dietary patterns including varying amounts of lean beef on total and LDL cholesterol levels in a low saturated fat diet (<7%) in adults with moderately elevated cholesterol levels. Conducted by The Pennsylvania State University researchers, the study included 36 participants (ages 30-65y) who were assigned to a treatment order and consumed a total of four diets for five weeks each. The crossover design allowed each participant to serve as his or her own control, reducing any errors associated with biological variation.

The four diets tested in the study were: Healthy American Diet (HAD) as control, DASH, BOLD and BOLD-PLUS. The BOLD and DASH diets were matched for macronutrient composition, both rich in fruits, vegetables, whole grains and low-fat dairy products, however, they differed in their primary protein source. The BOLD and BOLD-PLUS diets' primary protein source was lean beef, such as 95% lean ground beef, top round, and chuck shoulder pot roast, while DASH and HAD included white meat and plant protein as their primary protein sources. The BOLD diet included an average of 4.0 oz. of lean beef every day and the BOLD-PLUS diet included 5.4 oz. of lean beef every day. The HAD and DASH diets included 0.7 and 1.0 oz. of lean beef every day on average, respectively.

Details on each of the dietary interventions are as follows:

	HAD	DASH	BOLD	BOLD-PLUS
Calories	2,097 kcals	2,106 kcals	2,100 kcals	2,104 kcals
Protein (% of total calories)	17%	18%	19%	27%
Carbohydrate (% of total calories)	50%	55%	54%	45%
Fat (% of total calories)	33%	27%	28%	28%
Saturated Fat (% of total calories)	12%	6%	6%	6%
Lean Beef (oz./day) (weight before cooking)	0.7	1.0	4.0	5.4

After following treatment diets for five weeks, participants' total and LDL cholesterol levels were significantly reduced with the BOLD, BOLD-PLUS and DASH diets compared to the HAD diet. In addition, participants following the BOLD, BOLD-PLUS and DASH diets experienced a 10 percent decrease in LDL cholesterol from the start of the study. The improvements in heart health risk factors seen from the BOLD diets were as effective as those from the DASH and other heart-healthy dietary patterns such as OmniHeart which emphasized plant proteins.[2,3]

Lean beef also uniquely and favorably affected apolipoprotein concentrations; the BOLD and BOLD-PLUS diets significantly reduced apolipoproteins A1 and C-III concentrations compared to the HAD. And, the BOLD-PLUS diet was the only diet that resulted in the favorable response of significantly decreasing apolipoprotein B compared to the HAD.

Meta-Analysis Illustrates Lean Beef, Poultry, and Fish Have Similar Positive Effects on Heart Health

In addition, a meta-analysis of eight randomized control trials (RCTs) published in the July 2012 issue *Journal of Clinical Lipidology* evaluated the effects of beef protein compared to poultry and/or fish on lipoprotein lipids. The review authors hypothesized that beef consumption would not contribute to an increase in atherogenic blood lipid concentrations compared to poultry and/or fish, since 50% of beef's fatty acids are monounsaturated, and approximately 30% of its saturated fatty acid content is stearic acid. Therefore, this review and analysis identified peer-reviewed RCTs that investigated the effects of beef, independent of other red or processed meats, on the fasting lipoprotein lipid profile in healthy individuals or those with chronic disease risk factors. **The review of the eight RCTs indicated that beef, fish, and/or poultry have similar effects on lipoprotein lipid profiles, including small reductions in total cholesterol and LDL-C.**[4]

There's an Opportunity to Further Evaluate – and Better Translate – Evidence on the Role of Lean Beef in Heart Health

When considered in the context of the total body of research, the results from the BOLD trial, as well as the meta-analysis, provide **convincing evidence that nutrient-rich lean beef can be consumed as part of a diet recommended for improved heart health.**

At a time when most people have trouble meeting the recommendations in the Dietary Guidelines for Americans, it's increasingly important for nutrition research and education to evaluate and translate science into guidance that considers customs and habits that can improve compliance. For example, research indicates people are more likely to follow heart-healthy diets if they feature a variety of enjoyable, familiar foods. With 94% of American adults reporting that they eat beef at least monthly,[5] nutrition research that further evaluates the role of lean beef as part of an overall healthful diet – as well as dietary guidance that increases the flexibility of lean protein choices in heart-healthy recommendations – may be more likely to help promote positive, sustainable dietary change for improved heart health.

A free full text version of the BOLD study, which was funded in part by the Beef Checkoff Program, is available at: http://www.ajcn.org/content/early/2011/12/13/ajcn.111.016261.

The abstract of the meta-analysis, which was funded in part by the Beef Checkoff Program, is available online at http://www.lipidjournal.com/article/S1933-2874(12)00015-3/abstract.

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4. Maki KC, Van Elswyk ME, Alexander DD, et al. A meta-analysis of randomized controlled trials comparing lipid effects of beef with poultry and/or fish consumption. *J Clin Lipidol*. 2012;6(4):352-361.
5. *Consumer Beef Index*, July 2011. As of: April 9, 2012.

Funded by The Beef Checkoff

2012-13 Awards

UNDERGRADUATE AND GRADUATE STUDENT RESEARCH AWARDS

One undergraduate student award of $400

One graduate student award of $400

Applicants must have an abstract accepted for presentation at FNCE in Houston October, 2013.

All applications should be one pdf file sent by email. Applications provided in another format will not be accepted

Please include:

1. Your name and your academy member number (All award winners must be RDPG members as of 5/15/13)
2. Your mailing address, email address, your phone number
3. Your mentor's name
4. The name of the project
5. A copy of your acceptance letter from and (per and: notification of abstract acceptance or non-acceptance will be e-mailed by and.)
6. A copy of the abstract including title and authors
7. Applications should be submitted electronically to: Jeanene Fogli jeanenefogli@gmail.com
8. Applicant will be notified by email of receipt of application. It is the responsibility of the applicant to follow-up if an email notification of receipt is not received.
9. Applications due June 1, 2013

Award winners will be notified via email and phone by July 15, 2013.

Award will be given at the RDPG Member Breakfast during FNCE.

RDPG PILOT GRANT AWARD

A $3000 seed grant will be given to one RPDG Pilot Project.

All applications should be one pdf file sent by email. Applications provided in another format will not be accepted

Please include in your application package:

1. Your name, contact information, and number. (All applicants must be RDPG members as of 5/15/13)
2. A 250 word abstract to include: research question, methods, statistical methods, and anticipated results
3. Budget justification (no salary support will be paid).
4. Research justification: how will this project advance our understanding of nutrition?
5. Applications are due June 30, 2013.
6. Applications should be submitted electronically to: Jeanene Fogli jeanenefogli@gmail.com
7. Applicant will be notified by email of receipt of application. It is the responsibiility of the applicant to follow-up if an email notification of receipt is not received.

Winner will be notified by phone and email by August 1, 2013.

Winner will be introduced at the RDPG breakfast at the FNCE meeting in Houston. Winner will be expected to give a brief description of the project at this meeting. This can be given in writing if necessary.

***Winner will be expected to provide a brief report of results in* The Digest.**

Questions? Please contact Jeanene Fogli jeanenefogli@gmail.com or 617.875.3274

PUBLISHED PAPER JUNIOR FACULTY AWARD

One RDPG member will be selected to receive this award.

Please include:

1. Your name and member number (All award winners must be RDPG members as of 5/15/13)
2. Your mailing address, email address, and phone number
3. Electronic copy of paper; paper must have been published in an issue of a refereed journal dated between May 1, 2012 and April 30, 2013. Papers submitted or accepted but not published will not be considered this year but could be submitted for a future award.

Applicant must be within the first 5 years of post-doctoral career.
Candidate must currently hold one of the following ranks:

- Post-doctoral researcher within 5 years of completion of doctoral degree
- Clinician or research assistant professor within 5 years of completion of doctoral degree
- Tenure-track assistant professor.

4. A 250 word explanation of why the paper should receive this award from the RDPG
5. Applications should be submitted electronically to Jeanene Fogli jeanenefogli@gmail.com
6. Applicant will be notified by email of receipt of application. It is the responsibility of the applicant to follow-up if an email notification of receipt is not received.
7. Applications due July 1, 2013

Award winners will be notified via email and phone by September 1, 2013.

Awardee will be introduced at the RDPG member breakfast at FNCE. The award certificate will be presented at this meeting or mailed to the recipient.

2013 Election Results

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The Nominating Committee is pleased to report the outcome of the 2013 national elections. The following individuals have been elected from among many excellent candidates. The Committee thanks all who participated in the nomination and election processes.

2013 ELECTION RESULTS

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Nominating Committee

- Nancy Becker, MS, RD, LD (OR)
- Deanne Brandstetter, MBA, RD, CDN (NY)
- Martha Peppones, MS, RD, CD (WA)

Commission on Dietetic Registration

Registered Dietitian:

- Molly Gee, MEd, RD, LD (TX)
- Karen Lacey, MS, RD, CD (WI)
- Laura Matarese, PhD, RD, FADA, LDN (NC)

For more information regarding nominations and elections, visit www.eatright.org/elections.

Continuing Professional Education

Certificate of Attendance – Attendee Copy

2013 RDPG – Beginning to Identify Trends Among Dietitians Treating Eating Disorders: When Do You Make Referrals to Mental Health Care? (Newsletter)

(Activity #108727)

CPE Accredited Provider

Commission on Dietetic Registration

the credentialing agency for the Academy of Nutrition and Dietetics

_____

Participant Name

_____

RD/DTR ID Number

_____

Date Completed

_(2.0)_ (1) _____

CPEUs Awarded CPE Level Learning Need Code*

CDR Accredited Provider # **AM003**

Diane Moon Enos, MPH, RD

CDR Accredited Provider Signature

RETAIN ORIGINAL COPY FOR YOUR RECORDS.

**Refer to your Professional Development Portfolio Learning Needs Assessment Form (Step 2)*

COPY II: STATE LICENSURE VERIFICATION

Please complete a separate Certificate of Attendance Form for each session attended. Present a completed form to your Licensure Board upon request.

Continuing Professional Education

Certificate of Attendance – Licensure Copy

2013 RDPG – Beginning to Identify Trends Among Dietitians Treating Eating Disorders: When Do You Make Referrals to Mental Health Care? (Newsletter)

(Activity #108727)

CPE Accredited Provider

Commission on Dietetic Registration

the credentialing agency for the Academy of Nutrition and Dietetics

_____

Participant Name

_____

RD/DTR ID Number

_____

Date Completed

_(2.0)_ (1) _____

CPEUs Awarded CPE Level Learning Need Code*

CDR Accredited Provider # **AM003**

Diane Moon Enos, MPH, RD

CDR Accredited Provider Signature

RETAIN ORIGINAL COPY FOR YOUR RECORDS.

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Research DPG: CPE Article
Dietitians Referring to Mental Health Professionals

Answers

1.) D	5.) D
2.) B	6.) D
3.) D	7.) B
4.) B	8.) C