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A New Perspective on the Role of Sodium and Electrolytes in Exercise-Associated Muscle Cramps: A Review for Dietitians

by Andrew Dole, RDN, CEC

Exercise-associated muscle cramps (EAMCs) are painful, sudden, skeletal muscle spasms that occur during or after physical activity. EAMCs are most often specific to working muscles and are localized.[1,2] A definitive cause of EAMCs has yet to be uncovered, but there is evidence that widely accepted beliefs and nutrition-related protocols undertaken to prevent EAMCs are based largely on anecdotal or observational findings and possess limited evidence for justifying their efficacy and implementation. This necessitates an update on the current EAMC literature to help registered dietitian nutritionists (RDNs) reassess current recommendations and match protocols to the emerging science.

While the exact mechanisms contributing to the development of muscle cramping as well as evidence-based prevention protocols remain unknown,[1] research in the past 10 years further supports our understanding surrounding the exercise-associated muscle cramp. Even though the etiology of EAMCs needs to be elucidated, there is clear evidence contrasting old theories of hypohydration and electrolyte deficiency as primary contributors of EAMC.[1,3-7] New data suggest that fatigue and the central nervous system may, instead, play a significant role.[1,8-10] Presented here is a research update that aims to provide information that can be used to reevaluate current beliefs regarding the etiology of EAMCs and consider potential new evidenced-based nutrition protocols for EAMC treatment and prevention.

Observations and Factors Traditionally Correlated With EAMCs

Dehydration and EAMC

Historically, dehydration has been widely accepted as a root cause of EAMCs. However, as described here, recent studies have been unable to demonstrate a connection between dehydration and lowered body mass associated with sweat-related water loss.

In a 2009 review, Schwellnus makes acute observations about the origins of the dehydration theory behind EAMCs. He first identifies that the foundational literature regarding EAMCs, originating in 1904 by Edsall and in 1933 by Talbott, could be con-

sidered incomplete and was observational in nature.[1] For example, Edsall's paper provided no clinical data based on blood work, and instead relied upon less than seven observed cases of hospital admissions during hot weather.[2] Furthermore, Schwellnus makes the case that the samples of athletes that Edsall and Talbott observed had exclusively participated in extreme heat and humidity and exhibited high levels of sodium-concentrated fluid losses. However, it is now known that EAMCs also occur in cool climates, in cold water, and in temperature controlled environments.[1,10]

Despite the observational nature and limited clinical data presented in these early publications, dehydration is still often cited as a cause of EAMCs. More recent research that includes measurements of plasma volume, blood volume, and body weight differences between athletes reporting cramping and those without EAMCs do not support strong associations between dehydration or electrolyte loss and cramping.[5-7] A few studies stand out in this regard:

- In 2013, 10 men underwent an exercise intensity-controlled protocol inducing significant (3-5%) or serious (>5%) levels of dehydration. A resulting mean 4.7% body mass loss paired with a 4-g loss of sodium did not induce muscle cramping among the participants.[5]
- A study conducted in 2010 reported that among a sample of athletes participating in an Ironman triathlon, 43 reported EAMCs, whereas 166 were unaffected (i.e., the control group). Pre- and post-race body weight levels did not differ between the groups.[7]
- In 2005, Ironman triathletes (n=20) reporting EAMCs (n=11) who were compared with those not reporting muscle cramping (n=9) exhibited no differences in body mass/fluid loss during the race.[6]

Furthermore, in a controlled hydration and electrolyte study among adult men with a history of EAMCs, Jung found that 7 of 13 participants (54%) reported an EAMC in a trial inducing hypohydration.[3] This finding appears to support dehydration as a precursor to cramping. However, in a follow-up trial where participants were exposed to a calf-fatiguing exercise protocol in the heat and given a carbohydrate-electrolyte beverage, 9 of 13 participants (69%) still reported an EAMC, despite euhydration and electrolyte supplementation.[3] It should be noted that in Jung's study the subjects cramped much earlier in the hypohydration trial, which may highlight the role of proper hydration in prolonging onset of fatigue as a potential component of an intervention plan for preventing EAMCs.

Electrolyte/Sodium and EAMC

Low levels of serum sodium and other electrolytes have also been routinely associated with EAMCs by athletes and practitioners. As with dehydration, studies supporting sodium and electrolyte loss as a primary contributor to EAMCs largely stem from observational research.[11-13] More recent studies evaluating athletes from a variety of sports disciplines, including football, marathon, and triathlon, do not support a significant connection between electrolyte levels or electrolyte loss with the development of EAMCs:

- A research study conducted in 1986 found no significant differences in pre- or post-race serum electrolyte levels or plasma volume losses between those developing a cramp (n=15, 18%) and those without EAMCs among 82 male marathon runners.[14]
- In 2005, among the 20 Ironman athletes agreeing to evaluation immediately after the race, post-race serum electrolyte concentrations did not significantly differ between those reporting EAMCs (n=11) and those without cramping (n= 9).[6]
- Among the 210 Ironman athletes tested in 2011, pre- and post-race serum sodium and chloride levels did not differ between those reporting (n=43) or not reporting (n=166) EAMCs.[7]
- In 2005, the 69% of men develop-

From The Editor

Keep It Up

by Mark Kern, PhD, RD, Editor-in-Chief

Sometimes it's easy to become complacent about our careers or have a lull in our professional growth. I predict that if you read this edition of *PULSE,* that won't be the case for you. This issue is loaded with topics that are almost sure to expand your knowledge. I know it has mine, and my hope is it will help you to keep up with some of the latest in nutrition science and practice.

Our cover article by Andrew Dole, RDN is an excellent update on the current research regarding muscle cramps during exercise and the potential roles of electrolytes. Gregory Norris and Christopher Blesso, PhD have provided our free CPE article in this issue. In it they discuss the potential roles of sphingolipids in reducing risk for chronic diseases. You'll also find an interesting article by Becky Mehr, MS, RD that discusses a novel three-phase food exposure model for use in treating eating disorders. Finally, Zachary Grunewald, MS, RDN reviews recent research on the role of perivascular adipose tissue on the function and health of blood vessels.

There's plenty of other interesting topics to keep abreast of in these pages. Be sure to check out all of the great information provided by our editors and the news provided by our SCAN leaders.

ing an EAMC, upon performing calf-fatiguing exercise and consuming a carbohydrate- and sodium-enhanced hydration drink, cramped despite electrolyte supplementation.[3]

These findings are not to be confused with the known effect of serum electrolyte imbalances, specifically the depletion of calcium and magnesium, on the development of muscle spasms.[15] It is also known that the electrolytes, particularly sodium (Na), play an important role in health and sport performance. However, the above findings provide support to a more complex and multifaceted etiology of, and potential course of treatment for, EAMCs.

Emerging Literature

From as early as 1986, the central nervous system has been implicated in the development of EAMCs.[14] Most recently, several studies focusing on the role the central nervous system (CNS) and genetic predispositions to cramping contrast the traditional theories surrounding hypohydration and electrolyte imbalance as potential root causes of EAMCs.

Neuromuscular Theory

The neuromuscular fatigue theory (NMF) provides a counter to traditional understandings regarding the etiology of EAMCs by implicating the sensing of fatigue-related variables by the central nervous system and fatigue of working muscles due to increasing exercise intensity.[1,7,8,10,16-18]

Continuous and repetitive muscle contractions characterize most sports, although they can be emphasized in endurance running, swimming, and cycling, and can result in a "hyperexcitability"[15] of the nervous system. As a result of this chronic overload, muscles present with a higher potential risk of cramping. Electromyographic (EMG) data from triathletes reporting muscle cramping in the field demonstrate associations between increased motor neuron activity and EAMCs.[6]

> "Tired muscles affect the CNS at the level of the spine and contribute to altered neuron control."[1]

In addition to risk of "hyperexcitability" due to chronic overload, data support that neuromuscular fatigue may also increase EAMC risk. Numerous exogenous variables including temperature, humidity, energy stores, hydration, duration of exercise, and intensity of workload combined with repetitive motions contribute to muscle fatigue.[1] Tired muscles affect the CNS at the level of the spine and contribute to altered neuron control.[1] This fatigued state creates another "cramp-prone" scenario.

Moreover, the intensity of training or the race-day workload has been closely associated with the development of EAMCs.[7,8,16,17] Intensity of effort is linked with elevated relative perceived exertion (RPE) and muscular inefficiency, leading to a fatigued and a "cramp-prone" state.

In a case report by Wagner, the resolution of chronic hamstring cramping in one athlete was accomplished

through isolated muscle recruitment, strengthening of agonist muscle groups, and "neuromuscular reeducation" of the target muscle.[15] Clearly, the sample size of only one is limiting, although in the context of the topic and surrounding research, it is relevant to mention as support of the NMF theory. It is also encouraging that, in this case, muscle strengthening and CNS recruitment was associated with EAMC mitigation.

Prior History of Cramping and Genetic Factors

Prior research has shown that athletes with a previous history of muscle cramping may exhibit an increased risk of developing subsequent EAMCs. The reason is unknown, but the connection is clear:

- In 2011, all 20 of the 49 runners reporting EAMC in an ultramarathon race had experienced a prior EAMC.[16] In another study published in 2011 involving 433 Ironman athletes, 216 were classified with an EAMC during or immediately after the evaluated bout of exercise. Of the 216, 76% reported an EAMC within the past 12 months.[17]
- In 2014, 52 of 104 elite rugby players experienced at least one EAMC during the 2010 season. Of the 52 athletes, 22% were classified as "chronic crampers," with a history of EAMC.[8]

The aforementioned studies highlight a potential subset of athletes presenting with multiple prior EAMCs.This may support a potential genetic component to EAMCs. Further research supporting a genetic component includes:

- A study from 2011, in which 36% of the triathletes exhibiting EAMC (n=202) also reported a family history of EAMC.[17]
- In 2013, specific collagen genes associated with soft-muscle tissue injury were tested among a group of 268 Ironman or Ultra distance athletes. The athletes with a history of EAMC exhibited a lower prevalence of a specific variant of the *COL5A1* collagen gene compared with the non-EAMC group.[19]

While these studies do not identify a direct relationship between genetics and EAMC, they represent the depth of the emerging research and support future studies on genetic factors regarding muscle cramping in athletes.

Future Research

Current scientific literature is moving away from electrolyte, sodium, and hydration as primary determinants of EAMCs. However, a majority of the current dietetics texts, practice manuals, and professional protocols involve the manipulation of these variables to prevent the occurrence of EAMCs in active clients and competitive athletes.

Therefore, the role of the RDN in preventing and treating EAMCs may require research updates to better reflect current evidence. This is not to say that hydration strategies, electrolyte repletion, and energy balance do not have a role in EAMC prevention. In fact, they may be fundamental to the prevention of EAMCs through complimentary interactions.

> "…supplementation of fluid, sodium, and electrolytes should not stand as our only solution for treating EAMC."

However, separating ourselves from antiquated literature allows us to actively seek new, more effective means of prevention to contribute to the EAMC solution, particularly surrounding alternative or complementary strategies geared toward preventing fatigue, including the following: 1) the role of glycogen status in delaying muscle fatigue; 2) the role of energy stores or exogenous energy consumption on CNS-related fatigue; and 3) optimal macronutrient distribution to delay muscular or CNS-related fatigue.

While the neuromuscular fatigue theory holds promise, more field studies are needed, because much of the electrical impulse-induced EAMCs are done within a laboratory setting. In addition, there are limited research or clinical data showing a relationship between nutritional interventions and CNS-related fatigue.

Guidelines for Dietetics Practice

Basing practice protocols on dated theories may limit the effectiveness of RDNs and mire the profession in old science. Therefore, supplementation of fluid, sodium, and electrolytes should not stand as our only solution for treating EAMC. Instead, practitioners should focus on the complete athlete and the broader spectrum of research, considering the athlete's individual history and genetic predisposition to EAMC in addition to hydration, electrolyte balance, diet periodization, exercise intensity, and energy balance. Concurrently, as a profession, we should continue to actively evaluate evidence that supports the impact of energy balance and hydration on delaying fatigue, a potential root cause of EAMC.

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CPE article

Dietary Sphingolipids for the Prevention of Obesity-Related Chronic Disease

by Gregory H. Norris and Christopher N. Blesso, PhD

This article is approved by the Academy of Nutrition and Dietetics, an accredited Provider with the Commission on Dietetic Registration (CDR), for 1 continuing professional education unit (CPEU), level 1. To apply for ***free*** *CPE credit, take the quiz on SCAN's Web site (www.scandpg.org/quiz/?id=91). Upon successful completion of the quiz, a Certificate of Completion will appear in your My Profile (under the heading, My History). The certificate may be downloaded or printed for your records.*

Learning Objectives

After you have read this article, you will be able to:

- Identify sources of dietary sphingolipids and discuss how its common form, sphingomyelin, is digested in the body.
- Describe the effects of dietary sphingolipids on blood lipids reported in animal and human studies and discuss their potential role in treating and/or preventing obesity-related dyslipidemia and fatty liver.
- Discuss the findings of animal and human studies regarding the effects of dietary sphingolipids on the inflammatory response.

Obesity often coincides with chronic low-grade inflammation and dyslipidemia, which accelerate the progression of cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), and non-alcoholic fatty liver disease (NAFLD).[1] Therefore, finding therapies to prevent dyslipidemia and inflammation may be beneficial for preventing obesity-related

complications. Sphingolipids, a class of phospholipids, are dietary bioactives that have been shown to reduce dietary fat and cholesterol absorption in pre-clinical studies. In addition, dietary sphingolipids have the potential to attenuate inflammation, a major underlying factor in cardiometabolic disease.

This article provides an overview of the potential for dietary sphingolipids to impact obesity-related chronic disease.

Sphingolipids as Part of the Human Diet

The health effects of dietary lipids, such as fatty acids and cholesterol, have been extensively studied. In addition, phospholipids have been examined for their ability to reduce liver fat and improve serum lipid profiles.[2,3] Dietary phospholipids are most commonly found in cellular membranes as well as in natural emulsions, such as those found in milk or eggs. Phospholipids can be divided into two distinct groups: glycerophospholipids and sphingolipids (Fig. 1).

Sphingolipids are a diverse class of polar lipids found most abundantly in animal products such as milk and eggs. Structurally, the sphingolipids contain a long base molecule, typically sphingosine, which makes them unique among phospholipids. This is in contrast to the glycerol backbone found in the glycerophospholipids, such as phosphatidylcholine.[4] The typical Western-type diet is estimated to contain approximately 200 to 400 mg/day of sphingolipids,[5] similar to the average intake of dietary cholesterol in the United States.[6] Dietary sphingolipids commonly exist in the form of sphingomyelin (SM), which is exclusive to animal products and found most abundantly in dairy products and egg yolk.

Dietary SM is not readily absorbed by the intestines; instead, it needs to be digested to ceramide and sphingosine prior to absorption.[7] Alkaline sphingomyelinase (alk-SMase), found on the intestinal brush border and in human bile, catalyzes the first step in SM digestion, converting SM to ceramide and phosphocholine.[8] Other dietary lipids are known to inhibit alk-SMase activity[9,10]; thus, SM digestion does not proceed until most other lipids have already been absorbed.[11] An additional intestinal enzyme will digest ceramide to yield sphingosine and a fatty acid, which are readily absorbed by intestinal cells.[8] Sphingomyelin digestion is typically incomplete, and roughly 20% of SM consumed will reach the large intestines intact or as ceramide.[12]

Potential to Improve Obesity-Related Dyslipidemia and Fatty Liver

Effects Observed in Animal Models

Interest in the effects of dietary sphingolipids on cardiometabolic diseases likely stemmed from the observation that SM has a high affinity for cholesterol in cell membranes.[13] The hypothesis was that the strong physical interactions between cholesterol and SM could alter cholesterol absorption.[14] Subsequent research showed that dietary SM and its digestion products, ceramide and sphingosine, could reduce cholesterol absorption.[14-17] Upon further investigation, it was found that SM could also inhibit the absorption of fatty acids and other lipids.[18,19]

After it was demonstrated that dietary SM could impair lipid absorption, SM and other sphingolipids were examined for their effects on blood lipids during long-term feeding studies. Several studies reported improvements in serum lipids,[20-23] whereas others showed no effect[24,25] or showed detrimental effects.[26] In mice, investigators found that feeding different sphingolipids as part of a high-fat diet for 6 weeks at roughly the equivalent of 1 g/day in a 70-kg human reduced serum cholesterol and triglyceride concentrations compared with a high-fat control diet.[20] Phytosphingosine, a sphingolipid found in both plants and animals, was further examined and shown to reduce lipid absorption and protect mice from developing fatty livers, known as hepatic steatosis. In 2013, Chung and colleagues[25] reported that supplementing a high-fat diet with egg-derived SM reduced cholesterol absorption and hepatic steatosis in mice. Reductions in liver fats

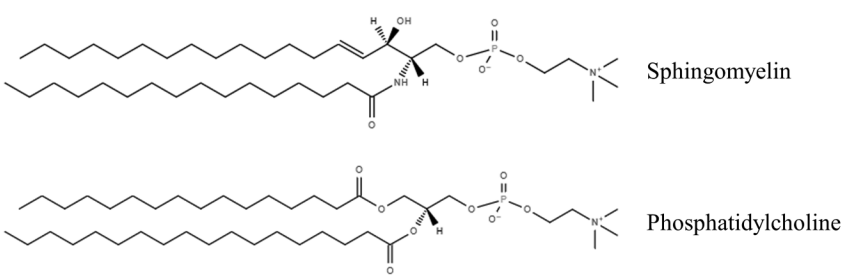

Figure 1. Structure of Common Phospholipids. Sphingosine is a common structural backbone for sphingolipids, such as sphingomyelin. Glycerol is the structural backbone for all glycerophospholipids, including phosphatidylcholine. Lipid structures were drawn using The LIPID MAPS Lipidomics Gateway, http://www.lipidmaps.org/

were found, even though serum cholesterol went unchanged in this study. Hepatic steatosis is common in obesity and is generally considered the liver's manifestation of insulin resistance; therefore, these studies highlight the potential of sphingolipids for the prevention of obesity-related chronic disease.

Our laboratory has compared the effects of SM sourced from both milk and eggs on obesity and inflammation in mice fed high-fat diets. We observed that supplementing diets with milk-derived SM attenuated the weight gain seen with feeding a high-fat diet over 4 weeks. This effect coincided with reductions in serum cholesterol, serum endotoxin, and liver fat content.[21] In a follow-up study of longer duration, we found that supplementing mice with egg-derived SM attenuated body weight and fat gain in response to a high-cholesterol, high-fat diet.[22] Egg-derived SM also reduced serum cholesterol and fasting glucose, suggesting an improved metabolic status compared with that observed in a high-fat control group. Inflammation is strongly related to insulin resistance and hepatic steatosis, which together increase the risk of obesity-related chronic disease. Feeding mice milk- or egg-derived SM caused reductions in serum free fatty acids, hepatic steatosis, and inflammation markers in serum as well as in adipose tissue.[22]

Effects Observed in Human Studies

Only a few clinical trials have been conducted examining the effects of dietary sphingolipids on blood lipids. Phytosphingosine (1 g/d) supplementation for 4 weeks was shown to reduce both serum cholesterol and low-density lipoprotein cholesterol (LDL-C) compared with placebo in men with metabolic syndrome.[27] In another crossover study, healthy adults who consumed 1 g/day of milk-derived SM added to prepared diets for 2 weeks had increases in serum high-density lipoprotein cholesterol (HDL-C) compared with those who consumed diets without SM.[28]

Mixtures of sphingolipids have also been tested using a more whole food approach. A sphingolipid-rich (975 mg/d) drink supplemented for 4 weeks in women reduced plasma LDL-C compared with a placebo.[29] Buttermilk powder, which is rich in both phospholipids and sphingolipids, reduced plasma cholesterol and triglycerides versus placebo in a 4-week crossover study in healthy adults.[30] Another study in overweight adults reported that consumption of 40 g of milkfat per day as whipping cream (19.8 mg of phospholipids) for 8 weeks reduced plasma cholesterol and LDL-C compared with phospholipid-depleted butter oil.[31] While the clinical evidence is still preliminary, the results are consistent with what has been shown in animal studies.

"Inflammation is strongly related to insulin resistance and hepatic steatosis, which together increase the risk of obesity-related chronic disease."

These recent trials suggest that dietary sphingolipids have the potential to treat and/or prevent dyslipidemia in humans. However, research in humans is still relatively scarce and larger studies of longer duration should be conducted.

Dietary Sphingolipids and Inflammation

Chronic low-grade inflammation is a common characteristic of obesity and contributes to the development of cardiometabolic diseases. Dietary milk SM was shown to attenuate chemically-induced colitis in mice,[32] suggesting anti-inflammatory effects in the gut. Glucosylceramide and gangliosides, which are sphingolipids where the phosphorylcholine component is replaced by sugars, also reduce chemically-induced colitis in rodent models.[33,34] Both ceramide and sphingosine were reported to reduce the inflammatory activation of macrophages in vitro.[35]

Recently, a human trial examined serum inflammatory markers after the ingestion of a concentrate of butter serum, a milk byproduct rich in phospholipids.[36] They reported a decrease in post-prandial concentrations of inflammatory soluble intercellular adhesion molecule and an increase in anti-inflammatory interleukin-10 with the addition of butter serum to a high-fat meal.[36] Although research involving dietary sphingolipids shows some potential to alter inflammatory responses, further research is necessary to investigate how dietary sphingolipids impact chronic low-grade inflammation with obesity in humans.

Conclusion

Sphingolipids are not essential nutrients; however, like many plant bioactives, they could provide health benefits if consumed in appreciable amounts. Dietary sphingolipids show potential to improve obesity-related complications such as dyslipidemia, NAFLD, and inflammation. Additional research should expand on the preliminary findings from human studies and explore other obesity-related conditions. It will be important to determine if benefits observed in animal studies translate to humans before making recommendations to increase the intake of dietary sphingolipids.

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Addressing Food Rigidity Through Hierarchical Exposure Therapy

by Becky Mehr, MS, RD

It is common to hear about a client's food fears in a nutrition session through such statements as "Eating pizza will make me fat," "Eating two bananas in a day will cause my potassium to spike," or "White potatoes cause diabetes, so I avoid them." What is the best way to tackle these statements to support recovery?

Traditionally, methods utilized in this context have included nutrition education, meal plans, guidance with intake, and exploring the relationship of food and weight as it relates to the client's history and belief system. These methods all share components of cognitive behavior therapy (CBT), dialectic behavior therapy (DBT), acceptance and commitment therapy (ACT), and motivational interviewing (MI). Increasingly, mindfulness is also incorporated into nutritional therapy.[1] All of these tools are helpful in acknowledging the client's convictions that lead to food rules and food rigidity, while facilitating cognitive flexibility and subsequent behavioral changes.

It is widely recognized that individuals with eating disorders (EDs) are prone to rigid thinking about food and weight-related beliefs, and they seek to control their emotions via food behaviors such as restricting, bingeing, and purging. A connection between the presence of overanxious disorder in childhood and obsessive compulsive disorder (OCD) may increase the risk for anorexia nervosa (AN).[2] As such, EDs develop as a coping skill for emotional distress that has resulted in a pattern of rigid, inflexible thinking and eating patterns that perpetuate the cycle of the ED patterns.[3,4]

> "A connection between the presence of overanxious disorder in childhood and obsessive compulsive disorder (OCD) may increase the risk for anorexia nervosa (AN)." [2]

At the Renfrew Centers, a Unified Treatment Model for Eating Disorders™ is the foundation for therapeutic and nutritional interventions; it was developed as an adaptation of Barlow's Unified Protocol for the Trans-diagnostic Treatment of Emotional Disorders.[5] This model integrates a relational approach with an evidence-based, trans-diagnostic model, designed to address the needs of a complex medical and psychiatric patient population. The model assumes that EDs arise from and are perpetuated by internal disconnection from one's own emotional experiences, as well as a relational disconnection from others. The ED is the way the individual has learned to "cope" with or "deal" with internal uncomfortable emotional experiences. The concepts in this model help clients "lean in" to the emotions rather than use the ED behaviors or another means of avoiding and/or suppressing the emotion. Treatment of the ED involves breaking the cycle of emotional avoidance by addressing the ED behaviors and increasing the ability to tolerate "uncomfortable" emotional experiences. The "uncomfortable" emotional experience could be anxiety, anger, guilt, grief, loneliness, or even happiness.

The Unified Treatment Model: Three Phases

The Unified Treatment Model for Eating Disorders™ comprises three phases: Engaging, Acquiring, and

Transforming. These stages build on each other to support the client in "leaning in" to one's emotional experience, thus reducing the cycle of emotional avoidance through ED patterns.

In the first stage, Engaging, the client learns the function of his or her emotions and how to practice present-focused, nonjudgmental awareness of the emotional experience. Nutritionally, the client is also working on nourishing the body and increasing food variety. In addition, the client is being introduced to symptom interruption strategies to support more balanced, stable brain activity by eating regular, full meals and/or snacks. Throughout this process the client is developing insight into how food-related behaviors are connected to her emotional experiences by completing food emotion journals. The food emotion journals help to connect the physical sensations, thoughts, and behaviors/urges during an anxiety-provoking situation, i.e., eating.

The second stage, Acquiring, focuses on recognizing thinking patterns and typical emotional avoidance behaviors and cultivating an awareness and tolerance of physical sensations. At this stage, the client is starting to build self-efficacy with eating and food, learning to tolerate sensations of hunger and fullness and physical reactions to eating. The client is also identifying the urges to use food rituals or ED symptoms, and tolerates them rather than engages in them.

In the final stage, Transforming, the client creates a hierarchy of challenging, commonly avoided food and food-related behaviors. An understanding of food exposure principles is developed during this stage. It is essential that the client utilize the skills from the previous two stages to practice tolerance with the emotional experience to create a corrective learning experience while engaging in a food-related exposure. Exposures may be done in a group setting or as part of individually planned exposures. The insight, knowledge, and experience gained through all stages of the Unified Treatment Model for Eating Disorders™ help the client become an expert in understanding and managing emotional experiences and achieving sustained recovery. This last stage is a key concept for those in practice outside of Renfrew's model.

Exposure Therapy

The definition of exposure therapy is "psychotherapy that involves repeated real, visualized or simulated exposure to or confrontation with a feared situation or object or a traumatic event or memory in order to achieve habituation and that is used especially in the treatment of post-traumatic stress disorder, anxiety disorder or phobias"[6]

"Exposure therapy, in its proper form, is not used often in the field of nutrition."

Exposure therapy, in its proper form, is not used often in the field of nutrition. However, Steinglass et al[7] found that the overlap between AN and anxiety disorders suggests that exposure and response prevention could be a beneficial approach in the treatment of EDs and preventing relapse. They noted that up to 50% of adult hospitalized patients were rehospitalized within 1 year of discharge from an acute treatment.[7,8] They also noted in these studies that psychological symptoms improved, while eating behavior did not change during the acute treatment. Expanding upon previous research, Steinglass et al[9] completed a study evaluating exposure and response prevention for anorexia nervosa (AN-EXRP) as a strategy for improved eating patterns during weight restoration. They found that AN-EXRP improved caloric intake of a laboratory meal over time compared with the traditional approach of cognitive remediation therapy.

Exposure to naturally occurring unpleasant events occurs frequently in most people's lives. Over time, the more we are exposed to certain events and unable to avoid them, the more likely we are to develop tolerance for the events. It can take up to 10, 15, or 20 trials of a food for it to be accepted by a child.[10] With that knowledge of human development and learning, it is unreasonable to expect that one exposure to a highly feared food by someone with an ED would be sufficient to resolve the ongoing distress. Rather, with repeated and increasingly more difficult exposures—and with developing confidence to manage the emotional distress that comes from those exposures—clients can have their efforts supported by registered dietitian nutritionists (RDNs) to lean into their distress so they can build experience and confidence that lead to a healthy, normal relationship with food. Planned exposures help clients anticipate the factors that will lead to their distress and plan for ways to manage their emotional experience without using the previous avoidant ED behaviors.

Even without the benefit of a formal treatment model such as Renfrew's, RDNs often find that they are engaged in exposure-type activities from the very first session. Universal practices are often inherently distressing and avoided by clients with an ED. Developing a meal plan, challenging food rules and rituals, incorporating new foods, and planning for desserts are often experienced as emotionally distressing to clients. Dietitians need to be aware that setting and accomplishing these objectives will require education, building confidence, and supporting clients through trust and support of the RDN/client relationship. It requires empathy and an appreciation of how anxiety-provoking these tasks can be for someone with an ED. Clients are

coming to RDNs for support, and with this exposure model RDNs are asking clients to step out on a limb and go against their "habit" that keeps them "safe."

Applying Planned Exposures

RDNs provide science-based, food-neutral information to help increase awareness of the emotional experience of food-related behaviors, and to provide encouragement and support for the clients' attempts to change ED behaviors.

Scenario 1

A client comes to the session with the goal of eating two tacos at the Cinco de Mayo work party next week. The client recognizes why it is important to eat socially and has been incorporating fear foods at home. The goal is set to "just do it" without any additional thought or planning, as the client seems motivated and extremely excited to do this. However, the client has never shared a meal or had a snack with coworkers. The client arrives at the next session feeling defeated and sad, and has decreased food variety at home.

Scenario 2

The next client comes into session stating, "I'm invited to a cookout next week. Normally, I eat beforehand because while there I will be nervous that others are looking at me and judging what I eat. Then I only eat fruit to have at least something there. Eating in social settings increases my anxiety. One of my goals for treatment is to be able to do things like have a burger with my friends without freaking out." The RDN works with the client to create a short hierarchy of the foods that would be available and the pre-anticipated distress associated with those items. It is decided to approach the task of eating a burger in steps, i.e., having just a burger but no bun while at the cookout. The client and RDN discuss what it would be like to do something different and what seems like a manageable but slightly more challenging food item to eat socially in this scenario. It is taken a step further, and the client and RDN talk about and plan through the anticipated exposure to build confidence in tolerating accomplishing the task. The client arrives at the next session stating, "I did it" with glowing confidence.

In each of these scenarios, the client shows a glimmer of desire to change, and goals for exposure are set. So why was one client successful and the other client not?

Why the Hierarchy Helps

In the first scenario, the exposure was planned. However, a hierarchical component for the task was not explored prior to determining the sequence and timing of the exposure. In the second scenario, the anxiety component of the situation and ranking of food available were explored; this process fits more with the concept of a planned food exposure. Planned exposures help clients to gradually experience and tolerate the distress of food-related tasks and to build confidence in their ability to manage the emotional experience that arises when exposed to these tasks.

> "The planning part of an exposure requires that clients start with a low level (minimal distress and avoidance rating) hierarchy item."

Clients are more likely to have a successful experience and to "complete" exposures if they are able to plan for how they will manage the experience prior to the task. The planning part of an exposure requires that clients start with a low level (minimal distress and avoidance rating) hierarchy item. Completing this task, while navigating one's emotional experience, leads to confidence to try increasingly more "difficult" tasks. Would a child be successful in learning to ride a bike if the training wheels weren't on at first? Perhaps he would, but, the likelihood of falling, becoming injured, and developing a fear of trying again also increases. With training wheels a child is able to increasingly learn balance, speed, and coordinating turns, while gaining confidence to manage the task. Ultimately, this leads to increased self-confidence and reassurance in being able to handle the bike. The process of food exposures works in much the same way. By starting with a low distress hierarchy item and gradually moving to more difficult tasks, the client becomes well-versed in implementing newly developed skills and gains the confidence to manage food-related challenges.

Summary

As noted in the two scenarios and in the AN-EXRP study, approaching fear foods by addressing the relationship of the intake and the anticipated anxiety helps to sustain and maintain momentum in the recovery process. Mark Twain summarized the principle behind exposure therapy quite nicely by stating, "Habit is habit and not to be flung out of the window by any man, but coaxed downstairs a step at a time." Thus, by using the technique of a food exposure hierarchy, clients are learning to rely on their own ability to do the task rather than getting stuck because they jumped right into the deep end of the feared foods.

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Influence of Perivascular Adipose Tissue on Vascular Health and Function

by Zachary Ian Grunewald, MS, RDN

An emerging topic in the field of obesity and cardiovascular research is in the area of perivascular adipose tissue (PVAT) and its effects on vascular health and function. PVAT is defined as the adipose tissue that surrounds a vessel (typically an artery) that has been found to increase in size with caloric excess and obesity.[1] Understanding the influence of PVAT on the vasculature is particularly important today given that almost three in four (74%) men and women over the age of 20 are either overweight or obese[2] and at greater risk of cardiovascular disease.[3] Notably, obesity is characterized by adipose tissue expansion and is associated with chronic inflammation and cardiovascular disease.[3] Initially thought to be an inert storage for triglycerides, adipose tissue has recently been identified as a major endocrine organ, secreting factors such as leptin, adiponectin, and cytokines, which can influence local and systemic vascular health.[4,5] Through the actions of these secreted molecules, PVAT has been found to exert both protective[6] and detrimental[7] actions on the vasculature. However, in the setting of obesity, recent evidence has implicated inflamed PVAT in the pathogenesis of endothelial dysfunction and aortic stiffness,[3,8,9] important risk factors for the development of cardiovascular disease. This brief review of the literature explores the influence of PVAT on the cardiovascular system, primarily in the setting of obesity.

PVAT Phenotype

Interestingly, PVAT phenotype has been determined to vary depending on anatomical location. For instance, PVAT surrounding the thoracic region of the aorta (periaortic adipose tissue) has been found to be morphologically more similar to brown adipose tissue (BAT) than subcutaneous white or visceral white adipose tissue (WAT)[10] containing higher amounts of uncoupling protein-1 (UCP-1), a hallmark component of brown adipose tissue. The brown phenotype of the thoracic periaortic adipose tissue is suggested to be more resistant to obesity-induced inflammation and vascular impairments.[11] As such, under healthy physiological conditions thoracic periaortic adipose tissue has been determined to confer an anti-contractile action on the aorta,[6,12] whereas in the abdominal region, this property is lost.[12] Particularly in the setting of obesity, PVAT surrounding the abdominal aorta has been reported to be more susceptible to inflammation and to negatively impact local vascular function.[10,12] Notably, it is believed that the phenotype of the PVAT, healthy or inflamed, that determines whether PVAT exerts protection on the vessel or causes harm. Although periaortic adipose tissue has been at the forefront of this area of research, PVAT surrounding coronary vessels has also been determined to influence the health of the coronary arteries.[4,7]

Furthermore, the phenotype of PVAT surrounding both the aorta and coronary vessels is altered by obesity.[7] Obesity is associated with chronic low-level inflammation, particularly at the level of the adipocyte.[3] Inflamed adipose tissue is associated

with arterial dysfunction and vascular insulin resistance in humans.[3] Additionally, obesity induced by a high-fat, high-sucrose diet has been demonstrated to reduce UCP-1 expression and cause BAT to become more like WAT,[13] which is believed to negatively impact adipose tissue phenotype. Indeed, unhealthy obese and inflamed PVAT has been reported to confer detrimental effects on the vasculature, whereas healthy PVAT exerts a protective action.[6,7,14] The ability of PVAT to act on the vessel is suggested to be through linking metabolic signals to the local vessel that it surrounds,[4,9,14] as described below.

PVAT-Vascular Cross-Talk

PVAT has been identified as a highly capable "vasocrine" organ, secreting adipokines and inflammatory markers that have been shown to exert direct action on the cardiovascular system.[4,9] Current evidence indicates that adipose tissue can secrete many types of signaling molecules termed adipokines (adipose tissue-derived cytokines),[4,8-10,15-17] classic inflammatory cytokines,[5] vasoactive peptides,[18] and catecholamines.[19] Interestingly, PVAT surrounding the aortic arch has been shown to secrete larger amounts of pro-inflammatory cytokines than other peripheral or subcutaneous fat depots.[9] In particular, PVAT secrets elevated amounts of interleukin (IL)-6, IL-8, monocyte chemoattractant protein-1 (MCP-1) compared with other fat depots, while secreting lower amounts of adiponectin, an anti-inflammatory adipokine.[9] PVAT-derived adiponectin has been implicated in the protective actions of PVAT on the vasculature, which is often reduced with obesity.[6,14] Additional research indicates that MCP-1, IL-6, and tumor necrosis factor alpha expression are reduced in the PVAT of rats who voluntarily exercise versus sedentary rats,[20] suggesting that exercise can foster a healthier PVAT phenotype. Collectively, a more brown-like, lean adipose depot is thought to have a healthier secretome, whereas obese or inflamed PVAT secretes a greater abundance of pro-inflammatory factors.[7-9] Moreover, the phenotype of PVAT influences its secretome and, thereby, its action on the vessel.

Leptin has been implicated as a major signaling adipokine between PVAT and the artery.[4,21] Leptin, secreted by adipocytes, is well known to modulate food intake but has also been shown to alter vascular homeostasis.[22-25] With obesity or following consumption of a high-fat diet (HFD), basal leptin has been found to be elevated.[26] Chronic elevation of leptin has been reported to confer negative effects on the endothelium, altering nitric oxide (NO) bioavailability and contributing to endothelial dysfunction.[23-25] Leptin secreted by epicardial adipose tissue has been reported to contribute to coronary endothelial dysfunction in a swine model of metabolic syndrome,[4] providing evidence that PVAT secretions can directly influence local vasculature.

Effects of PVAT on Vascular Function

Vascular function is typically defined by an artery's ability to dilate and contract.[12,14] A primary modulator of vascular function is the endothelial cell that lines the lumen of all vessels. When endothelial cells become dysfunctional, as is often observed with obesity, the dilatory function of the vessel often becomes impaired. Endothelial dysfunction is characterized by an imbalance of vasoactive substances such as NO and endothelin-1, potent signaling molecules.[27] PVAT has been shown to alter the balance between NO and endothelin-1, implicating PVAT as a modulator of endothelial function.[3,27] Ma and colleagues found that when male Wistar rats consumed a HFD for 6 to 8 weeks, periaortic fat mass was significantly increased and aortic endothelial cells showed marked reductions in endothelial nitric oxide synthase (eNOS)-dependent vasodilation,[1] a major regulator of vasodilation. Additionally, epicardial PVAT-derived leptin impairs endothelium-dependent dilation in a model of metabolic syndrome.[4] These findings and the findings of others[28] demonstrate that obese and inflamed PVAT exerts a negative action on the vessel, altering the function of endothelial cells and impairing vascular function.

In addition to endothelial-mediated impairments in vascular function, PVAT also influences vascular compliance or aortic stiffness. Importantly, aortic stiffness is considered an independent risk factor for the development of cardiovascular disease.[5,29] PVAT-derived IL-6 has been shown to contribute to aortic stiffness in LDL receptor deficient mice as measured by intrinsic mechanical stiffness.[5] Additionally, age-related increases in superoxide production in PVAT have been directly linked to aortic stiffness as assessed by aortic pulse wave velocity, the gold-standard measurement for aortic stiffness.[29] Thus, the metabolic and phenotypic changes that occur at the level of the perivascular adipocyte affect the mechanical properties of the local vessel, increasing the risk for the development of cardiovascular disease.

Conclusions

In conclusion, PVAT is implicated in the pathophysiology of cardiovascular disease. Current evidence demonstrates that PVAT phenotype and its secretome, whether healthy or obese, can exert protection on local vessels or can cause harm. Research suggests that improving PVAT phenotype may improve vascular function and reduce the risk for cardiovascular disease. As more literature accumulates about this unique fat depot, evidence suggests that obese and inflamed PVAT can cause considerable harm to the function of local vessels. Although PVAT is not readily accessible in humans to examine, it is important to consider that obesity may increase cardiovascular disease risk through the actions of PVAT. Moreover, regular exercise may improve the negative impact of obese PVAT on the vasculature. Future research should continue to elucidate the role of the PVAT secretome on vascular health, or vice versa, and identify strategies to improve the phenotype of obese PVAT.

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Forging Ahead with SCAN's New Initiatives

by Cheryl Toner, MS, RDN

While the heat and humidity will hang on here in Northern Virginia for a while longer, September brings the promise of cooler, crisper air along with the routine of the school year for those who have or work with children or who work in academia. I am longing for that routine as I write this letter. My teenagers are home for the summer, and as a working parent I am torn between wanting to play and surrender to the slower rhythm, and needing to continue an aggressive pace of work for my business and for SCAN.

Well-Fueled to Move Forward

There are three things that fuel me to be able to push ahead on SCAN's initiatives, even during the "lazy days of summer." First, the people. The SCAN Executive Committee (EC), our executive director, publication editors, project leaders, and task force members dedicate such passion to our work together—it is truly contagious. At SCAN's EC Retreat in June, we kicked off the year with team building in the mountains of Keystone, Colorado, hiking, doing a little yoga, cooking together, and enjoying s'mores around the fire pit. I hope to see you all back in the mountains of Keystone for our 2018 SCAN Symposium in the spring!

Second, the strategic plan gives me a clear sense of direction. At the EC Retreat, we clarified our initiatives for the year. While I cannot list everything SCAN is doing in this letter, it's humbling to see our progress, and exciting to plan for significant impact through programs that originated in our strategic planning session held in the summer of 2015.

Third, to support our strategic plan, SCAN's EC has prioritized a deep focus on the things that enable us to produce value for our members. Our website, the public hub of our organization, is getting an overhaul this year. External relationships are a significant priority as we seek to enrich our members' ability to engage with allies in our fields of work. We are improving the SCAN volunteer experience, from the process of signing up, to finding that first project or role, to advancing towards greater responsibility and influence. We know that getting involved with SCAN helps our members to build stronger networks, advance their careers, and strengthen our profession. In this vein, we are building a culture of leadership to engage SCAN members in becoming stronger leaders both within SCAN and in our day-to-day work.

"We know that getting involved with SCAN helps our members to build stronger networks, advance their careers, and strengthen our profession."

Click to Stay Connected and Informed

SCAN has a lot going on. So I always encourage members to pay attention when a SCAN email comes to your inbox—*open it, read it,* and *click it*. Click on at least one thing to learn more about what SCAN is doing for you or how you can get involved. Of course, you did it already—you are leading the way by reading this issue of *PULSE,* our first digital fall issue! We are greener, more financially efficient, and over the course of the year will be increasingly more useful to you in this format.

Be on the lookout for information regarding the forthcoming Food & Nutrition Conference & Expo™ (FNCE®) in the upcoming emails from SCAN so you can join us at our events at FNCE®. With all of the amazing programming and networking at this year's event as we celebrate the Academy's Centennial, the SCAN family will be there to help you learn and network to fuel your professional endeavors. See you in Chi-Town!

Conference Highlights

Multi-service Eating Disorders Association (MEDA) Annual Conference

March 10-11, 2017
Boston, MA

MEDA is the foremost eating disorders nonprofit organization in New England. MEDA provides education about eating disorders (EDs) and their underlying causes, and offers a range of services to support individuals and families through the various stages of recovery. MEDA's annual conference for health professionals is an important part of its educational program. Here are a few highlights from the 2017 lineup of exceptional speakers:

Prevention of EDs Through Societal Changes

Presented by Bryn Austin, ScD, of Harvard T.H. Chang School of Public Health, Boston, MA; and Rachel Rodgers, PhD, of Bouve College of Health Sciences, Northeastern University, Boston

Remember when the media glamorized cigarettes back in the 1950s, and smoking was the cool thing to do? Fast forward to today's culture, where smoking is banned in restaurants and public places, smokers feel ashamed of indulging in this health-harmful habit, and teens cannot legally buy cigarettes. Times have changed!

Other examples of large-scale public health prevention efforts that led to massive improvements in health include the use of seat belts and air bags. These are the kind of changes we want to see with eating disorders, so we can prevent them from ruining people's lives in the first place—but how do we make that happen? Austin and Rodgers explained that we need "strategic science." According to this concept, when researchers set out to design a new study, they should also think about how their study findings could be used by policymakers and communities to make change happen to benefit real people, including those at risk of developing eating disorders.

A review of the current eating disorders literature identifies at least 100 interventions that help prevent EDs. These studies focus on improving self-esteem, improving body acceptance, and teaching media literacy. The problem is, these programs all focus on the individual person. While this is good, we now need to focus on a bigger view and expand those findings to focus on health care policy and laws, with the goal of changing beauty ideals, cultural values, and media's messages.

"…we now need to focus on a bigger view and expand those findings to focus on health care policy and laws, with the goal of changing beauty ideals, cultural values, and media's messages."

To do this, we need help from a team of strategists who can create economic models to identify health care costs associated with ED treatment and compare those to the cost of interventions that could prevent EDs. Once we have a cost-analysis, in addition to the support of clinicians and voters, we can take that information to policymakers. Could we create legislation on the state and local level, if not the national level, to ban the sale of diet pills and laxatives to teens? Stop ads for fraudulent weigh loss products? Stop coercing models to get down to size zero to get a job? Discourage photoshopping of models' bodies?

To train the next generation of health care professionals, the Harvard T.H. Chang School of Public Health and Boston Children's Hospital have created the Strategic Training Initiative for the Prevention of Eating Disorders (STRIPED; www.hsph.harvard.edu/striped). The goal of STRIPED is to bring together scientists, policymakers, and community advocates to work together to prevent eating disorders on a large scale—on the scale that public health prevention efforts usually focus. Preventing eating disorders can prevent undue suffering, health care costs, and other costs to individuals, families, and society—to say nothing of saving lives.

Integrating Obsessive-Compulsive Disorder (OCD) Knowledge Into Anorexia Work

Presented by Marcia Black, PhD, psychologist in private practice, Amherst MA

A common belief among health care providers is that the obsessive-compulsive behavior seen in anorexia will resolve once the client is better nourished and weight-restored. This has been proven untrue. In fact, once weight is restored, helping the client abate the struggle with obsessive thoughts and compulsive behaviors becomes an important part of recovery. The fact is 65% of patients with anorexia have at least one anxiety disorder, and 70% of those patients say that the anxiety preceded the

eating disorder. Childhood anxiety commonly ends up as an eating disorder during puberty. Obsessive-compulsive patterns are "safety behaviors" or "compensatory behaviors" that play a key role in anxiety, OCD, and anorexia.

When a client is anxious and in a state of fear when eating, the goal is to calm down the obsessive thoughts and reduce the fear via exposure and response prevention work (E/RP). For example, exposure might start with just sitting in the same room with a jar of peanut butter, then on another day tasting a tiny bit of peanut butter, and so on. Each time clients sit through an exposure and learn to tolerate their anxiety/fear, they must also be taught to not engage in any of their "safety behaviors" (such as running 10 miles after having eaten peanut butter).

Fully debriefing the client after E/RP work is also important. You could ask the client who fears that the peanut butter would lead to weight gain the following: *"Why do you think you didn't balloon up after you ate the peanut butter? "* … *"Did you notice the anxiety was able to return to baseline without using safety behaviors?"* With time, clients learn they can master their own anxiety and fears.

Black encourages asking clients about their compulsive behaviors, for example: *"How often do you do a body check? Compare yourself to others? Count bites? Rearrange your clothing on your body? Carefully arrange your food on the plate so that the peas do not touch the mashed potato?"* Black recommends sharing this insight with the client: *"My guess is you are feeling very ashamed about your behaviors. I want to let you know that all humans—actually, all animals—engage in these kinds of behaviors when they are facing a threatening situation."* Most clients are very relieved to learn that they are not the only one with hidden secrets.

By making a list with the client of his/her behaviors, the clinician can then address the fears by asking, for example: *"What would happen to you if you stopped looking at yourself in every mirror? Or stopped using the Internet to look at models' bodies?"* The next step is to have the client identify a behavior he or she could stop doing for an hour, a day, or a week. Not doing a behavior will make the client very anxious, but if the client understands that anxiety goes up and it also comes down, the client can be encouraged to not start another behavior to deal with the anxiety. The goal is to learn to tolerate several different feelings. Black emphasized the benefits of doing E/RP with clients with anorexia.

When Elimination Diets Don't Work: Solutions Supporting ED Recovery and Gastrointestinal Health

Presented by Marci Evans, MS, CEDRD-S, private practice specializing in eating disorders, Cambridge, MA; and Lauren Adler Dear, MS, RDN, private practice specializing in digestive health, Brookline MA

Eating disorders can trigger gut issues, and gut issues can trigger eating disorders. With ED recovery, sometimes gut problems resolve, but at other times they remain problematic. Among people with eating disorders, 98% report having a functional gut issue such as belching, heartburn, constipation, and/or diarrhea. The degree of the issue relates to the stress level of the client: the higher the stress and anxiety, the worse the symptoms.

Functional gut disorders have no biological markers. The physician often tells the client that "everything looks normal." Hence, the symptoms seem to be linked to the nervous system. Evans explained that the vagus nerve links the gut's "brain" to the head's brain. The gut-brain works independently of the head brain.

The microbiota in the gut affects the signals to the brain and regulates appetite, energy extraction, and mood. Preliminary research suggests that people with anorexia have a significantly reduced number of health-promoting bacteria in their gut as well as a significantly reduced variety of microbes. This correlates with an increase in eating disorder pathology, inclucing negative body image. As clients restore weight and become better nourished, their microbiome improves but does not match the levels of non-ED control groups.

When a client with an eating disorder complains about gastrointestinal issues, the RD should think twice before recommending an elimination diet; it can be too triggering. Because the mind is often at the center of the ability to overcome gut issues, the dietitian could instead recommend alternative therapies such as mindfullness, meditation, biofeedback, and even hypnosis. Presenter Laure, Dear, an RDN and a trained hypnotherapist, reported that hypnosis has helped her clients resolve their gut issues.

The therapeutic value of hypnosis in patients with irritable bowel syndrome (IBS) was documented in an Australian study (Peters SL, et al, *Aliment Pharmacol Ther*; 2015:41: 1104-1115). Patients with IBS received either hypnosis or a low FODMAP Diet; the results showed that both groups had similiar improvements in intestinal issues at 6 weeks and at 6 months. The group that had hypnosis acquired an additional benefit: less anxiety.

Summarized by "Conference Highlights" editor Nancy Clark, MS, RD, SCCS, who has a private practice in the Boston area and is author of Nancy Clark's Sports Nutrition Guidebook, *available at www.NancyClarkRD.com.*

Reviews

Fueling the Teen Machine, 2nd edition

Ellen Shanley, MBA, RD, CD-N and Colleen Thompson, MS, RD
Bull Publishing Co., Box 1377, Boulder, CO 80306
800/676-2855; www.bullpub.com
2011, softcover, 279 pp, $16.95
ISBN 978-1-933503-37-0

It is well known that adolescence is a crucial period for growth and development, with proper nutrition being a key component. As teenagers notoriously gravitate toward less-than-healthy options, it can be difficult to convince them to consume nutritious foods. In response to this conundrum, Ellen Shanley and Colleen Thompson have provided a relatable guide that not only can help teenagers better understand nutrition and how it impacts their bodies, but also can effectively encourage them to take responsibility for their health and make better choices.

This guide begins by reviewing basics, such as defining macro- and micronutrients and introducing the principles of the *2005 Dietary Guidelines for Americans* and the Food Guide Pyramid. (There is no updated edition to reflect MyPlate or the 2015 Dietary Guidelines.) A variety of topics tailored to the teenage reader is explored, including weight management, sports nutrition, meal planning, functional foods, and supplements.

Each chapter contains easy-to-navigate subtopics, balancing nutrition facts with "hot topics" in nutrition. Many tables used throughout the book compare various foods and their nutritional values, illustrating the importance of nutrient density. Numerous healthy meal and snack ideas are provided to help the reader replace the empty calories of commonly consumed foods. Not only does this book provide sound nutritional information, it also equips the reader with strategies for shaping a healthy diet as well as identifying false food claims and misleading marketing.

"A variety of topics tailored to the teenage reader is explored, including weight management, sports nutrition, meal planning, functional foods, and supplements."

In all, the second edition of *Fueling the Teen Machine* provides in-depth information on a variety of nutrition topics with a relatable tone for the teenage reader. While some of the information may be a bit outdated, many of the principles presented still ring true today and can help the reader make more informed choices for life-long health.

Ellen Shanley, RD, CD-N, MBA is a registered dietitian and a faculty member in the Department of Allied Health Sciences at the University of Connecticut, where she directs the dietetics program and teaches courses in food system management. Colleen Thompson, MS, RD is also a registered dietitian and director of Hawley Armory, the fitness center for employee health at the University of Connecticut.

Reviewed by Caitlin Flanary, RD, clinical dietitian in Bakersfield, CA.

Research Digest

Hydration and American Football Players

Judge LW, Kumley RF, Bellar D, et al. Hydration and fluid replacement knowledge, attitudes, barriers, and behaviors of NCAA Division 1 American football players. *J Strength Cond Res.* 2016;30:2972-2978.

Because proper fluid balance is critical for sports performance, evidence-based fluid recommendations have been established for athletes. However, research suggests that some athletes, particularly collegiate football players, have difficulty meeting these recommendations. The purpose of this study was to examine hydration behaviors, attitudes, overall knowledge, and practices in NCAA Division 1 (DI) American football athletes. In this cross-sectional study, 100 male student football athletes (ages 18-24 y) from two different NCAA D1 universities completed a validated hydration survey administered immediately after voluntary summer conditioning sessions. Average hydration knowledge score (HKS), hydration attitude score, and hydration behavior scores were calculated. Out of a possible score of 17, the mean HKS score was 11.8±1.9 (69.4% correct). The four key hydration misunderstandings identified were as follows: 57% indicated that athletes should drink water rather than sports drinks, 52% disagreed that sports drinks are better than water for glycogen restoration, 49% indicated that athletes should use salt tablets during training/competition, and 55% indicated that thirst is the best indicator of hypohydration. Only 24% of athletes surveyed indicated appropriate intakes of fluids before, during, immediately after, and 2 hours after practice. Nutrition education (P=.045), position played (P=.025), and HKS (P=.045) were significant predictors of outcome variables. Furthermore, knowledge of appropriate hydration practices were obtained primarily from athletic trainers or coaches, with registered dietitians ranking sixth out of 11 knowledge sources. The results indicate that collegiate football players exhibit inadequate hydration knowledge and practices. Dietitians should focus on educating football athletes on appropriate hydration strategies to meet fluid recommendations.

Summarized by Amanda Bishop, ASCM-CEP, graduate assistant instructor, Department of Health, Kinesiology, and Recreation, University of Utah, Salt Lake City, UT.

"Dietitians should focus on educating football athletes on appropriate hydration strategies to meet fluid recommendations."

Pre-Workout Caffeine Supplement and Power/ Strength Performance

Martinez N, Campbell B, Franek M, et al. The effect of acute pre-workout supplementation on power and strength performance. *J Int Soc Sports Nutr.* 2016;13:29-35.

Athletes often consume pre-workout supplements containing caffeine, but research is lacking on the effect of such supplements on power and strength performance. The purpose of this study was to investigate the effects of a specific pre-workout supplement on performance measures of anaerobic and explosive power and upper body strength. In this randomized, double-blinded, crossover study, 13 recreationally active male participants completed a familiarization exercise trial followed by baseline exercise testing, then took either a supplement (Assault™) or placebo prior to an exercise trial (crossover treatment started on week 4), for a total of four exercise trials, each separated by 1week. Exercise trials consisted of a 5-minute dynamic warm-up followed by a medicine ball put, vertical jump, 1 repetition (1-RM) maximum bench press, and Wingate anaerobic test, each separated by 3 minutes. The supplement (Assault™; 14.5 g powder, 10 kcal, 3 g carbohydrate, 1,750 mg proprietary caffeine-containing blend) or placebo powder (flavored maltodextrin) were mixed with 10 ml of water and ingested 20 minutes prior to the third and fourth exercise trial. Peak power and mean power were significantly greater with the supplement than with placebo (782±191 W vs 722 ± 208 W (P=.003) and 569±133 W vs 535 ± 149 W (P=.006), respectively. There were no significant differences in medicine ball put, vertical jump, or 1-RM maximum bench press. The results of this study indicate that this particular pre-workout supplement may improve anaerobic peak and mean power, but not upper and lower body explosive power. Athletes considering supplementation to augment performance should consult a registered dietitian regarding safety and drug testing concerns.

This study was funded by an International Society of Sports Nutrition Educational Research Grant.

Summarized by Ashleigh Libs, graduate student, Department of Nutrition and Integrative Physiology, Coordinated Master's Program, Sports Nutrition Concentration, University of Utah, Salt Lake City, UT.

Disordered Eating, Food Addiction, and Nutrition Major College Students

Yu Z, Tan M. Disordered eating behaviors and food addiction among nutrition major college students. *Nutrients*. 2016;8:673-688.

College students are at risk for poor eating behaviors including disordered eating, eating disorders, and food addiction. The prevalence of eating disorders among college students ranges from 8% to 20.5%. The prevalence of food addiction ranges from 8% to 25% among young adults, but may be as high as 58% among those exhibiting disordered eating. Whether nutrition students are at higher or lower risk of disordered eating behaviors and food addiction compared with non-nutrition students is unclear. Therefore, the purpose of this study was to assess the prevalence of disordered eating behaviors and food addiction among nutrition and non-nutrition major college students at a public university. A total of 962 (73% female, 75% white) college students aged 18 to 25 years were recruited from Florida State University via campus email; nutrition students were incentivized with bonus points to participate. Participants were categorized into three groups: nutrition majors (n=147), non-nutrition health majors (n=136), and other majors (n=678). Participants provided demographic information (age, sex, race/ethnicity, height, weight) and completed the Eating Attitude Test (EAT-26), the Three Factor Eating Questionnaire (TFEQ-R18), and the Yale Food Addiction Scale (YFAS). According to the EAT data, approximately 10% of respondents showed a high level of concern for eating, body weight, and body shape, but a low level of concern for dieting behaviors, bulimia and food preoccupation, and oral control. There were no differences between groups. TFEQ-R18 data indicated moderate restrained, controlled, and emotional eating with no group differences. In addition, 10.3% of respondents met criteria for food addiction. Food withdrawal behaviors were significantly greater among non-nutrition health majors than nutrition or other majors (P=.013). This study indicates that both nutrition and non-nutrition majors are at risk for disordered eating behaviors and food addiction. Early eating disorder screening of college students may help increase awareness of eating disorders.

Summarized by Austin Henderson, graduate student, Department of Nutrition and Integrative Physiology, Coordinated Master's Program, Nutrition, Education and Research Concentration, University of Utah, Salt Lake City, UT

"This study indicates that both nutrition and non-nutrition majors are at risk for disordered eating behaviors and food addiction."

EPA and DHA and Cardiovascular Disease Risk Factors

Asztalos IB, Gleason JA, Sever S, et al. Effect of eicosapentaenoic acid and docosahexaenoic acid on cardiovascular disease risk factors: a randomized clinical trial. *Metabolism*. 2016;65:1636-1645.

Studies examining the effects of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) in fish oil have shown a decrease in coronary heart disease (CHD) events and CHD-related deaths. Most studies have combined EPA and DHA to examine their effects. This study explored the effects of individual doses of EPA and DHA on lipid and lipoprotein levels and on CHD-related inflammatory biomarkers in healthy normolipidemic individuals. In this randomized, double-blinded, placebo-controlled study, 121 participants were split into four groups: olive oil 6 g/day (n=30), EPA 600 g/day (n=30), EPA 1,800 g/day (n=31), and DHA 600g/day (n=30). Participants took two capsules three times daily for 6 weeks. Results showed increased lipid levels of EPA in the EPA 600 group (138%; P<.0001) and the EPA 1,800 group (417%; P<.0001) compared with a 13% increase in the placebo and DHA groups. Lipid DHA increased in the DHA group (87%; P<.0001) compared with a <1% increase in the other groups. The DHA group showed increases in low-density lipoprotein cholesterol (+18.4%, P <.01 fasting; +20.0%, P<.0005 fed state) compared with the EPA and placebo groups. However, the DHA group displayed a decrease in postprandial triglyceride (TG) levels (-20.0%; P<.04). Inflammatory marker Lp-PLA_2 was significantly decreased in the EPA 1,800 group (P=.003), whereas the EPA 600 and DHA groups showed no difference. This study indicates that individual doses of EPA and DHA may be responsible for separate, meaningful aspects of heart health protection.

This study was funded by a DuPont Applied Biosciences, Department of Agriculture Research service contract (53-3K-06), and an NIH grant (P50 HL083813-01).

Summarized by Vanessa Browning, graduate student, Department of Nutrition and Integrative Physiology, Coordinated Master's Program, Nutrition, Education, and Research Concentration, University of Utah, Salt Lake City, UT.

SCAN Notables

by Traci Roberts

■ **Tanya M Halliday, PhD, RD** was the recipient of the 2016 Michael Houston Memorial Scholarship, presented by Virginia Polytechnic Institute and State University (Virginia Tech). This award is given annually in memory of Michael Houston, professor emeritus and former Virginia Tech Human Nutrition, Foods and Exercise (HNFE) department head. It supports the research pursuits of a graduate teaching assistant who demonstrates a passion for teaching and an excitement for learning. Shortly after receiving this award, Tanya was named the HFNE Outstanding Doctoral Student in recognition of her academic achievements, leadership, and experiences beyond traditional course work. Tanya is now a postdoctoral fellow at the University of Colorado School of Medicine, Anschutz Medical Campus, in Aurora, CO.

■ **D Enette Larson-Meyer, PhD, RD, CSSD, FACSM** was honored to be selected as a member of the International Olympic Committee Expert Conference on Dietary Supplements and the Elite Athlete. The conference took place on May 3-5, 2017 in Lausanne, Switzerland. Enette is an associate professor of human nutrition and director of the Nutrition & Exercise Laboratory at the University of Wyoming. Information on the consensus statement can be accessed at www.olympic.org/news/ioc-tackles-the-topic-of-nutritional-supplements-their-use-and-their-effects.

■ **Arielle "Dani" Lebovitz, MS, RDN, CSSD, CDE** published her first book in a series of children's nutrition books funded through Kickstarter. Titled *Where Do Bananas Come From? A Book of Fruits,* the book is designed for the whole family to enjoy together. The content lets children discover "good for my body" nutrients; learn how fruit is grown; understand when a particular fruit is in season; and obtain guidance on how to pick, store, and eat fruit. The book engages the child's five senses and uses words from the book to describe the flavor and texture of each fruit. The book is available on Dani's website at www.experiencedeliciousnow.com as well as at www.amazon.com.

■ **Heather Rae Mangieri, RDN, CSSD** has authored a newly released book, *Fueling Young Athletes* (Human Kinetics, 2017). The book addresses the issues that families and athletes most often face, such as late night practices, inconvenient school lunch times, demanding tournament schedules, travel, and lack of sleep. It stresses the importance of balanced daily nutrition as part of an overall eating pattern for health, growth, development, and sports performance, and walks readers through ways to develop and implement that plan. In addition to the book, *Fueling Young Athletes* is available as a continuing education course with credits through Human Kinetics. It is designed to help RDNs and other fitness professionals understand adolescent growth and development and how to help young athletes fuel themselves to win.

■ **Sherri Stastny, PhD, RD, CSSD**, associate professor in the Department of Health, Nutrition, and Exercise Sciences at North Dakota State University (NDSU), was honored with the 2017 James Lebedeff Endowed Professorship Development Award, bestowed annually by NDSU's College of Human Development and Education. The award recognizes her collaborative research team leadership as well as her supervision of PhD students, who are studying the impact of nutrition, physical activity, and other lifestyle choices on muscle health. In addition to receiving this award, Sherri is a 2017 winner of the Outstanding Dietetics Educator Award, presented annually to deserving educators by the Nutrition and Dietetics Educators and Preceptors dietetic practice group.

If you have an accomplishment that you would like to be considered for an upcoming issue of PULSE, please contact Traci Roberts at: fivespotjones@gmail.com

Of Further Interest

■ News from Wellness and Cardiovascular Nutrition (Wellness/CV) Subunit

Here's an update on developments from the Wellness/CV subunit:

• **Wellness Task Force.** At the 2017 SCAN Symposium, about 40 members participated in the Wellness Task Force "campfire"/"story-telling" session. Ideas were shared regarding how SCAN can support its members who work in wellness. Watch for new initiatives that develop from this session!

• **Wellness/CV at FNCE®.** Heading to Chicago in October for the Academy's Centennial and Food & Nutrition Conference & Expo™ (FNCE®)? For more information on this event, read "See You at FNCE®!" on page 23. In particular, the SCAN Networking Reception will provide a great opportunity to meet and connect with the Wellness/CV leadership team and

other SCAN colleagues. We would love to hear about your suggestions for strengthening our subunit, and to get you involved as a volunteer!

• **Resources.** Looking for trusted resources to use in your wellness and/or cardiovascular-focused practice? Visit SCAN's website to download the latest fact sheets and find links to evidence-based position stands and partner organization sites at www.scandpg.org/cardiovascular. You can also find new webinars in SCAN's e-library at www.scandpg.org/e-library. Remember, webinars are free to members for the first month!

• **Joining Forces.** *SCAN Connection* is the new member newsletter that includes updates and content from all three of SCAN's subunits. We hope that in addition to reading the Wellness/CV articles, you will take this opportunity to enhance your professional development in the areas of sports and disordered eating/eating disorder nutrition practice.

■ News from DEED Subunit

Following are announcements from the Disordered Eating & Eating Disorders (DEED) subunit:

• **New Fact Sheets.** Four new fact sheets were recently released: *Men and Eating Disorders; Family Based Therapy; Identifying Eating Disorders;* and *Mindfulness.* You can access them at www.scandgp.org/fact-sheets/disordered-eating.

• **Latest Webinar.** Our latest webinar titled *Integration of Exposure Response Prevention with Dietary Application in the Treatment of Eating Disorders* is now available at the SCAN online store at www.scandpg.org/store/default.aspx?search=Webinars.

• **Let's Hear from You!** We are always looking for new ideas for educational materials to produce. To share your thoughts and ideas, contact the DEED director, Sarah Gleason, RDN, CEDRD at sarah@sarahthedietitian.com.

■ News from Sports Dietetics—USA (SD-USA) Subunit

Below are some highlights from the SD-USA subunit:

• **Expanding the Arena Initiative: Optimizing Performance on Every Stage.** Through this new initiative, SCAN will be working to promote untapped opportunities to our members and other professionals. In the sports arena, we know that the traditional view of the sports RDN working strictly with athletes in organized sports does not reflect our diverse work settings and client populations—in reality, sports RDNs may work with first responders, military personnel, adventure racers, musicians, and more! Be on the lookout for new resources from this initiative as well as for professional development opportunities and inspirational member profiles. Also, consider volunteering to be involved in this initiative or on other SCAN projects. Sign up at www.scandpg.org/volunteer-opportunities/ and indicate your area of interest.

• ***Sports Nutrition: A Practice Manual for Professionals*** **(6th edition).** The latest revised edition of this sports nutrition manual, available this fall, was written and reviewed by esteemed sports RDNs and other exercise experts. Highlights include a new chapter discussing emerging opportunities in sport nutrition, a completely revised overview of exercise physiology, strategies for sports nutrition assessment, updated population- and sport-specific recommendations, and more. The price is $65 for Academy members, and free faculty preview copies are available for educators and program directors. Visit the Academy store at www.eatrightSTORE.org.

• **New Partnership.** SCAN now has an official partnership with Professionals in Nutrition for Exercise and Sport (PINES)! This relationship and other official sports-related partnerships are managed by SD-USA. Learn more about PINES and its commitment to sport nutrition around the globe at www.pines.org/14a/pages/index.cfm?pageID=3295.

• **External Relations.** Did you know that SCAN also has official partnerships with the National Athletic Trainers' Association (NATA) and the National Strength and Conditioning Association (NSCA)? If you are interested in growing these relationships, please contact the SCAN Office at info@scandpg.org.

• **New Fact Sheets.** Check out our latest fact sheets at www.scandpg.org/sports-nutrition/sports-nutrition-fact-sheets/sn-fact-sheets/: *Celiac Disease and Gluten Sensitivity in Athletes; Irritable Bowel Syndrome in Athletes;* and *Creatine Supplementation and Athletic Performance.* As always, they are free with your SCAN membership.

■ Nominations for 2018 Academy Election

The call for nominations for the 2018 Academy Election began a while ago, and nominations for president-elect, speaker-elect, and treasurer-elect closed on September 1. However, it's not too late to submit other names. All other nominations are due October 25, 2017. The Nominating Committee is looking for leaders with the skills and vision to further the profession. Visit www.eatrightPRO.org/elections to view the positions available on the 2018 ballot and download the nominations form.

■ Qualified Health Claim Petition for Macadamia Nuts

The Food and Drug Administration has reviewed data for a qualified health claim that you may now begin seeing on food labels. The claim reads: "Supportive but not conclusive research shows that eating 1.5 ounces per day of macadamia nuts, as part of a diet low in saturated fat and cholesterol and not resulting in increased intake of saturated fat or calories, may reduce the risk of coronary heart disease. See nutrition information for fat [and calorie] content."

■ Quality Management Resources from the Academy

The Academy's Quality Management Committee has collaborated with the Commission on Dietetic Registration and other Academy entities to develop practice resources for the dietetics profession. Quality Management efforts of the Academy promote and support registered dietitian nutritionists (RDNs) and nutrition and dietetics technicians, registered (NDTRs) from all areas of practice in providing competence self-evaluation, and measuring and reporting quality of nutrition care and services. This is paramount to our nation's health care, to the marketplace and, to the business of dietetics.

The Academy's foundational documents inform and support the practice of the RDN and the NDTR. Quality Management practice documents address and include scope of practice, standards of practice and professional performance, standards of excellence, practice decision tools, definition of terms, practice tips, case studies, and quality improvement (QI), performance improvement (PI) provider accountability, systematic methods for process improvement, and performance measurement (PM) systems initiatives at the practitioner level.

An overview of all Quality Management resources is available in the learning module "Quality Management: Resources for Excellence in Nutrition and Dietetics," which can be accessed at www.eatrightpro.org/resource/practice/quality-management/quality-care-basics/resources-for-excellence-in-nutrition-and-dietetics-module.

■ Pediatric Weight Management Guide Released

Integrating the latest practice guidelines, the second edition of the *Pocket Guide to Pediatric Weight Management* helps practitioners create individualized nutrition care blended with practical advice. This handy guide contains appendixes with formulas, assessment methodologies, and professional resources. Available at a discounted Academy price ($26.99) at www.eatrightSTORE.org.

■ Call for Abstractors for "Research Digest"

The "Research Digest," which appears in each issue of *SCAN'S PULSE,* provides summaries of published papers relating to any of SCAN's practice areas: nutrition for sports and physical activity, cardiovascular health, wellness, and disordered eating and eating disorders.

You can contribute to the pages of *PULSE* by volunteering to abstract a recently published study of sports nutrition. For details on this opportunity, contact Stacie Wing-Gaia, PhD, RD, CSSD, co-editor of "Research Digest," at stacie.wing@health.utah.edu.

■ Manuscripts for *PULSE* Welcome

SCAN's PULSE welcomes the submission of manuscripts to be considered for publication. In particular, *PULSE* is interested in receiving original research reports and review articles. Manuscripts presenting practical guidelines, case studies, and other information relevant to SCAN will also be considered.

Manuscripts must be prepared and submitted in accordance with *PULSE's* Guidelines for Authors; only manuscripts that follow these guidelines will be considered. The Guidelines for Authors can be accessed at www.scandpg.org/nutrition-info/pulse/.

See You at FNCE®!

October 21-24, Chicago

Come to the 2017 Food & Nutrition Conference & Expo™—and celebrate the Academy's Centennial! Here are some SCAN highlights won't want to miss:

SUNDAY
3:30 pm - 5:00 pm
Putting Heart Into Performance Nutrition for Collegiate Athletes
Presenters: Jennifer Ketterly, MS, RD, CSSD and Caroline Mandel, MS, RD, CSSD

MONDAY
9:00 am - Noon
SCAN Booth
DPG/MIG Showcase

6:30 pm - 8:30 pm
SCAN Networking Reception
Apogee Rooftop Lounge at the Dana Hotel

TUESDAY
8:00 am - 9:30 pm
SCAN Spotlight Session:
Fueling Teen Athletes: Unique Challenges and Winning Strategies
Presenters: Christine Rosenbloom, PhD, RDN, CSSD, FAND and Anastasia Fischer, MD, FAAFP, FACSM

For more information and updates, be on the look-out for eblasts, and visit www.scandpg.org/cpe/fnce-2017

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Upcoming Events

October 7-10, 2017
AACVPR Annual Meeting, Charleston, SC. For information: American Association of Cardiovascular and Pulmonary Rehabilitation, www.aacvpr.org

October 21-24, 2017
2017 Food & Nutrition Conference & Exhibition™ (FNCE®)—Centennial Anniversary, Chicago, IL. For information: eatrightfnce.org.
SCAN events at FNCE are detailed on page 23 of this issue of *PULSE*. For more information: www.scandpg.org/cpe/fnce-2017

October 30-November 2, 2017
Obesity Week, Washington, DC. For information: American Society for Metabolic & Bariatric Surgery and The Obesity Society, www.obesity.org/meetings/obesity-week

November 10-12, 2017
Annual Renfrew Center Foundation Conference, Philadelphia, PA. For information: www.renfrew.org

April 21-25, 2018
Experimental Biology (EB) 2018, San Diego, CA. For information: experimentalbiology.org/2018/Home.aspx

May 4-6, 2018
Join your colleagues at the 34th Annual SCAN Symposium, *No Limits Nutrition: Extreme & Unique Practices,* Keystone, CO. More information to come.

SCAN'S PULSE

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Appropriate announcements are welcome. Deadline for the Spring 2018 issue: **Jan. 1, 2018.** Deadline for the Summer 2018 issue: **April 1, 2018.** Manuscripts (original research, review articles, etc.) willl be considered for publication. Guidelines for authors are available at www.scandpg.org. E-mail manuscript to the Editor-in-Chief; allow up to 6 weeks for a response.

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SOYBEAN OIL FOR HEALTH

By **Mark Messina, Ph.D.**

SoyConnection
By the United Soybean Board

Composition

In contrast to other beans, soybeans are relatively high in fat, with approximately 40 percent of their calories coming from fat.

Soybean oil is one of the few non-fish sources of omega-3 polyunsaturated fatty acids.

The oil from crushed soybeans is commonly labeled "vegetable" oil. Soybean oil is primarily comprised of two types of polyunsaturated fatty acids (PUFA): omega-6s and omega-3s. Soybean oil is made up of 12 to 15 percent saturated fat, 22 to 30 percent monounsaturated fat (oleic acid) and 55 to 58 percent PUFA. In turn, those PUFA contain 5 to 7 percent alpha-linolenic acid (ALA), an essential omega-3 fatty acid. Soybean oil's composition varies slightly between soybean varieties and growing conditions.[1]

Fatty Acid Composition of Soybean Oil*

Fatty Acid	g/100 g[1]
Total Polyunsaturated	**57.740**
Linoleic (n-6, 18:2)	50.418
Linolenic (n-3,18:3)	6.789
Total Monounsaturated	**22.783**
Oleic acid (C18:1)	22.550
Gondoic acid (20:1)	0.233
Total Saturated	**15.650**
Palmitic acid (C16)	10.455
Margaric acid (C17)	0.340
Stearic acid (C20)	4.435
Arachidic acid (C20)	0.361
Behenic acid (C22)	0.366

* USDA, National Nutrient Database for Standard Reference Release 28: Full Report (All Nutrients): 04669, USDA Commodity Food, oil, vegetable, soybean, refined

Soybean oil accounts for over 40 percent of the intake of linoleic acids (LA) and ALA in the United States because of its widespread use.[2] It contains a ratio of about 8:1 or 9:1 LA to ALA. The change in availability of vegetable oils in the U.S. food supply has changed the LA:ALA ratio from about 6.4:1 to 10.0:1. At the same time, the total omega-6 to omega-3 ratio was 5.4:1 in 1909 and 9.6:1 in 1999, as estimated by Blasburg et al.[2] The health implications of this ratio are discussed in the next section.

The ALA found in soybean oil is the principal source of omega-3s in the U.S. diet.

Dietary Fat, Coronary Heart Disease (CHD) and LA to ALA Ratio

Reduced saturated fat intake has been recommended for decades to protect against CHD.[3,4] The basis for this recommendation is the well-established hypercholesterolemic effect of saturated fat and to a lesser extent, the hypocholesterolemic effect of PUFA.[5] Not surprisingly given its composition, soybean oil has been shown to lower low-density lipoprotein (LDL)-cholesterol.[6,7] Nevertheless, there is some controversy about the impact of dietary fat, and in particular, saturated fat, on CHD.[8,9] Research suggests that the confusion over saturated fat's impact on CHD risk stems in part from failing to consider that the effect depends on whether saturated fat is replaced with carbohydrates or unsaturated fats.

Replacing 5 percent of energy from saturated fat with equivalent energy from PUFA was associated with a 25 percent reduction in CHD risk.

A combined analysis of the Nurses' Health Study (1980 to 2010; n = 84,628) and the Health Professionals Follow-up Study (1986 to 2010; n = 42,908 men) made this point clear. The investigators found that replacing 5 percent of energy intake from saturated fat with equivalent energy intake from PUFA, monounsaturated fat, or carbohydrates from whole grains was associated with statistically significant reductions in CHD risk: 25 percent, 15 percent and 9 percent, respectively. Whereas, replacing saturated fat with carbohydrates from refined starches/added sugars was not protective.[10]

Analysis of these two cohorts by Wang et al.[11] showed that in addition to affecting incidence of CHD, saturated fat intake increases total mortality, whereas PUFA lowers it. Finally, with respect to individual fatty acids, replacing just 1 percent of energy from palmitic acid (the most abundant saturated fat in the diet) with 1 percent of energy from PUFA lowered CHD risk by a statistically significant 12 percent.[12]

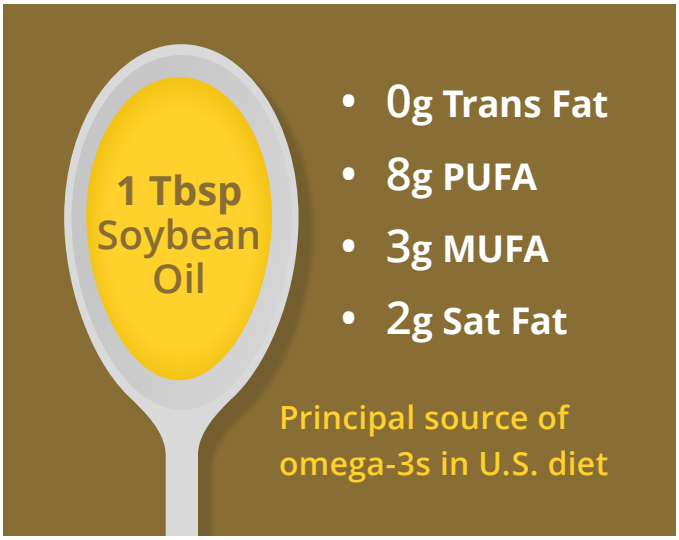
Despite the protective effects of LA intake observed in prospective epidemiologic studies, some argue that diets high in LA lead to inflammation, a likely underlying cause of cardiovascular disease (CVD).[13] However, the clinical evidence does not support this reasoning.[14-17] In fact, a systematic review of 15 trials concluded that "virtually no evidence is available from randomized, controlled intervention studies among healthy, non-infant human beings to show that addition of LA to the diet increases the concentration of inflammatory markers."[15]

NEW: Soybean Oil Achieves FDA's Heart-Health Claim

Supportive but not conclusive scientific evidence suggests that eating about 1½ tablespoons (20.5 grams) daily of soybean oil, which contains unsaturated fat, may reduce the risk of coronary heart disease. To achieve this possible benefit, soybean oil is to replace saturated fat and not increase the total number of calories you eat in a day. One serving of this product contains [x] grams* of soybean oil."

*U.S. Food and Drug Administration. "Soybean Oil and Reduced Risk of Coronary Heart Disease." July 31, 2017. https://www.fda.gov/downloads/Food/IngredientsPackagingLabeling/LabelingNutrition/UCM568508.pdf

The evidence indicates that diets high in omega-6 PUFA are not pro-inflammatory.



The Food and Agricultural Organization (FAO) of the United Nations recently concluded that that there is no rationale for a specific recommendation regarding the ratio of omega-6 to omega-3 fatty acids as long as the omega-6 fatty acid intake is between 2.5 percent and 9 percent of energy and omega-3 fatty acid intake is between 0.5 percent and 2 percent of energy.[18] The National Academy of Medicine does not recommend a specific ratio of omega-6 to omega-3 PUFA either. According to U.S. food disappearance data, LA and ALA intakes fall within the United Nations' specifications: these fatty acids represent 7.21 percent and 0.72 percent of energy intake, respectively.[2] Increasingly, evidence suggests that both LA and ALA have a role in reducing the risk of CHD and that the absolute amount consumed of these essential fatty acids should be emphasized rather than the ratio.[19]

There is no rationale to recommend a specific dietary ratio of omega-3 and omega-6 fatty acids.

One key issue remains: the inhibitory effect of LA intake on the conversion of ALA to the longer chain omega-3 PUFA (such as eicosapentaenoic acid).[20] However, according to the American Heart Association (AHA), because this conversion is already low,[21] it isn't clear that additional small changes would have net effects on CHD risk after the other benefits of LA consumption are taken into account. In a review of this issue, internationally recognized CHD expert William S. Harris concluded that the focus should not be on dietary ratios but rather on intake levels of each type of essential fat.[22] Harris et al.[14] also noted that decreasing LA intake as a means of increasing the dietary ratio of LA to ALA, as some have called for,[23-25] could very well have the opposite effect of that intended.

Finally, a more recent argument asserts that diets high in LA are harmful because LA is easily oxidized in vivo and the resulting metabolites increase CHD risk.[9] However, evidence in support of this hypothesis is unimpressive as none of studies cited in its defense showed that LA intake actually increases endogenous levels of oxidized metabolites of LA.[26-31]

View the full resource and more soy health facts at SoyConnection.com. Visit Soy Connection booth #1711 at FNCE® 2017 in Chicago.

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