Sports, Cardiovascular, and Wellness Nutrition
a dietetic practice group of the Academy of Nutrition and Dietetics

SCAN'S Pulse

Winter 2019, Vol. 38, No. 1

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Are Low Calorie Sweeteners Tools or Trouble for Regulation of Glycemia?

by Kelly Higgins PhD, MPH and Richard Mattes, PhD, MPH, RD

The food industry and consumers add low calorie sweeteners (LCSs) to foods or beverages to reduce the sugar and energy content while maintaining sweetness and palatability. Based on 2009-2010 and 2011-2012 cycles of the National Health and Nutrition Examination Survey (NHANES),[1] it is currently estimated that LCSs are consumed by approximately 41% of American adults and 25% of children. LCSs are especially popular among individuals attempting to lose or maintain body weight or control glycemia. Individuals attempting to lose weight are 64% more likely to consume LCSs.[2] Consumption of LCSs is associated with a higher healthy eating index score[3] and lower energy and sugar intake,[4] suggesting the LCSs are often incorporated into an overall healthy dietary pattern.

Despite certification as safe food additives or generally recognized as safe by the Food and Drug Administration and consistent evidence from randomized controlled trials (RCTs) that LCSs have no effect or no beneficial effects on body weight and glycemia,[5-7] the recommendation of LCS use for individuals with diabetes continues to be contested. It is important to determine the effect of LCS consumption on glycemia in order to provide recommendations for LCS use or avoidance among individuals with glucose intolerance. If LCSs have no effect on or help moderate glycemia, they can be used by individuals with diabetes to reduce their sugar intake while maintaining the palatability of the diet. If LCS consumption disrupts glucose homeostasis, their use should be discouraged or recommendations should be made for avoidance of specific LCS.

Theorized Mechanisms of Action

Historically, LCSs were believed to be inert outside the oral cavity. However, discovery of the expression of the sweet taste receptor throughout the body, including on tissues that regulate glycemia (e.g., enteroendocrine cells in the gastrointestinal tract, -cells in the pancreas), raises questions of how receptor binding past the oral cavity may affect glycemia. Some argue that if LCSs bind to receptors on enteroendocrine cells and lead to secretion of the incretins glucagon-like peptide 1 (GLP) and gastric inhibitory peptide (GIP), an increase in blood insulin in the absence of energy could disrupt glycemia and stimulate appetite.[8] While in vitro trials and select animal trials document

an increase in GLP-1 and insulin secretion with LCS consumption, trials in humans find no such effect.[9]

There is also speculation that LCS consumption leads to uncoupling of sweetness from energy provided by ingested carbohydrates, leading to energy dysregulation and overconsumption.[10,11] In addition, it has been posited that LCS consumption may lead to the development of a "sweet tooth," increasing appetite and intake of dietary sugars. However, there is little evidence of heightened energy intake with consumption of LCS or dietary sweetness in general.[6,12]

Finally, it has been hypothesized that if a LCS reaches the colon, gut microbiota composition and metabolite production may change, altering glucose tolerance. Chronic consumption of saccharin[13] and aspartame[14] lead to glucose intolerance in rodents by altering microbiotic population composition and metabolite production. A measurable change in the gut microbiota with aspartame consumption is surprising, because aspartame is digested and absorbed prior to reaching the colon.[15] While alterations to the gut microbiota with LCS consumption are linked to glucose regulation, the mechanism of action is unknown.[16]

Despite speculation of disrupted glycemia with LCS consumption, few trials have investigated the long-term effect of an individual LCS on glycemia in humans.

Differences in Chemical Structure

It is notable that the commercially available LCSs all have different chemical structures. These different structures determine their receptor binding domain, sensory properties, digestibility, and metabolic fate. For example, aspartame (a dipeptide) is hydrolyzed to aspartate, phenylalanine, and methanol in the proximal intestinal lumen, with limited opportunity to interact with receptors on enteroendocrine cells or the gut microbiota. Other LCSs (saccharin, ace-K, and a fraction of sucralose) are absorbed and excreted undigested in the urine. Sucralose and a fraction of saccharin are excreted undigested in the feces. Steviol glycosides and rebaudiosides (derived from the Stevia rebaudiana plant) are hydrolyzed to steviol and are either excreted in the feces or absorbed by intestinal cells, metabolized in the liver, and excreted in the urine. These different routes of digestion, absorption, and excretion (reviewed in Magnuson et al[15]) provide insight into the different metabolic processes they might affect. Because aspartame is digested in early stages of digestion, an effect on downstream glycemic responses is unlikely.

Evaluating LCS Effects on Disrupted Glycemia

To determine whether these hypothesized mechanisms translate to disrupted glycemia among humans, we conducted a 12-week RCT to determine the effects of two doses of aspartame equivalent to one can of soda (350 mg/day) or the 95th percentile of aspartame consumption (1,050 mg/day) on glycemia, body weight, and appetite among healthy, normal weight (body mass index [BMI]: 18-25 kg/m^2) adults aged 18 to 60 years.[17]

Method

The study involved 100 low consumers of LCS (consumption <1 time/week) who were assigned to consume either 0, 350, or 1,050 mg of aspartame daily for 12 weeks. Aspartame was delivered as a 500-mL fruit-flavored beverage for the 350 and 1,050 mg/day groups, with an additional 700 mg of encapsulated aspartame provided for the 1,050 mg/day group only; the 0 and 350 mg/day groups consumed placebo capsules.

Participants received no additional dietary guidance beyond instructions regarding the required consumption of the study beverages and capsules. A 240-minute oral glucose tolerance test (OGTT) was conducted at baseline and included measurement of appetite and selected hormones, including insulin, GIP, and GLP-1. Body composition was also measured at baseline. Throughout the study, par-

Knowledge Versus Wisdom

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

The humorist Miles Kington is often credited with saying: "Knowledge is knowing that a tomato is a fruit. Wisdom is not putting it in a fruit salad." While I'm guessing there are plenty of wise dietetics professionals who could pull off that feat, I totally get where he's coming from and I think it is an appropriate quote to introduce this issue's feature articles.

Our cover article by Kelly Higgins, PhD, MPH and Richard Mattes, PhD, MPH, RD provides excellent insights into the currently available literature regarding the potential impacts of low-calorie sweeteners on glucose metabolism. In another article, Daniel So, APD, Heidi Staudacher, APD, PhD and Katrina Campbell, AdvAPD, PhD share their wisdom on the latest and rapidly growing body of research about the effects of dietary fibers as prebiotics. Lastly, you'll find a fascinating article by Sarah La-Course that describes the hormonal dysregulation and psychological responses that occur with relative energy deficiency in sport and the female athlete triad.

You'll also find plenty of wisdom and opportunities to improve your knowledge throughout the pages of this issue. Be sure to check out all of the other information, including a book review, recent conference happenings, announcements in "Of Further Interest," and notable accomplishments of our members.

ticipants reported weekly for measurements of body weight and blood pressure and to collect their beverages for the following week. Baseline procedures were completed again after 12 weeks of the intervention.

Results

With the exception of one difference in serum glucose 60 minutes into the OGTT among participants in the 350 mg group at baseline, there was no difference in serum glucose or insulin among any of the groups at any time point during the 240-minute OGTT at baseline or after 12 weeks of consumption. In addition, there were no effects of either aspartame dose on GLP-1 and GIP concentrations. Body weight and fat mass did not change in response to aspartame or among the control group. Unpublished results using a similar study design assessing the differential effects of saccharin, aspartame, rebaudioside A, sucralose, and sucrose consumption for 12 weeks among healthy individuals with overweight or obesity (BMI ≥ 25 kg/m^2) yielded similar null effects of sweetener consumption on glycemic response during a 120-minute OGTT, yet it did exhibit differences in body weight change (unpublished).

Discussion

Overall, glycemic response, incretin release, and body weight were not affected by 12 weeks of aspartame consumption at two doses among healthy, normal weight individuals. These results are consistent with findings from trials examining acute and chronic glycemic responses to aspartame. Aspartame administered orally or intragastrically exhibited no effect on blood glucose,[18,19] insulin,[19,20] GLP-1,[19] and GIP [20] in acute feeding trials in humans. One trial reported reduced GLP-1 concentrations with aspartame oral exposure, but this was in comparison to an unsweetened, energetic control.[20] Chronic consumption of an aspartame-sweetened soda did not produce different results than water on glucose and insulin area under the curve concentrations during an OGTT when consumed by individuals with overweight or obesity for 6 months.[21] There is little evidence that any LCS affects glycemia or consistently initiate incretin release (see Tucker and Tan, 2017[7]). An increase in GLP-1 with consumption of a sucralose and ace-K sweetened cola was reported in one trial,[22] but this effect is likely due to the cola and not the sweeteners.[23]

Conclusion

Findings from these trials have implications for consumers, clinicians, policy makers, and the food industry. The United States Department of Agriculture 2015 Dietary Guidelines Advisory Committee gave no recommendation regarding LCS use for weight loss despite the available "moderate and consistent evidence,"[24] leaving it to the consumer to interpret if they should consume LCS. Our findings indicate that aspartame and other LCSs pose no risk to glucose dysregulation among healthy individuals who are of normal weight or overweight. Even doses of aspartame up to the 95th percentile of consumption had no adverse effect on glycemia or body weight. These trials were not conducted to test the efficacy of aspartame and other LCS for treatment of diabetes, but the results indicate that they do not pose a risk for glucose dysregulation.

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References

1. Sylvetsky AC, Jin Y, Clark EJ, et al. Consumption of low-calorie sweeteners among children and adults in the United States. *J Acad Nutr Diet.* 2017;117:441-448.

2. Drewnowski A, Rehm CD. The use of low-calorie sweeteners is associated with self-reported prior intent to lose weight in a representative sample of US adults. *Nutri Diabetes.* 2016;6:e202.

3. Drewnowski A, Rehm C. Consumption of low-calorie sweeteners among U.S. adults is associated with higher healthy eating index (hei 2005) scores and more physical activity. *Nutrients.* 2014;6:4389.

4. Gibson S, Horgan G, Francis L, et al. Low calorie beverage consumption is associated with energy and nutrient intakes and diet quality in British adults. *Nutrients.* 2016;8:9.

5. Miller PE, Perez V. Low-calorie sweeteners and body weight and composition: a meta-analysis of randomized controlled trials and prospective cohort studies. *Am J Clin Nutr.* 2014;100:765-777.

6. Rogers PJ, Hogenkamp PS, de Graaf C, et al. Does low-energy sweetener consumption affect energy intake and body weight? A systematic review, including meta-analyses, of the evidence from human and animal studies. *Int J Obes* (Lond). 2016;40:381-394.

7. Tucker RM, Tan S-Y. Do non-nutritive sweeteners influence acute glucose homeostasis in humans? A systematic review. *PhysiolBehav.* 2017;182:17-26.

8. Egan JM, Margolskee RF. Taste cells of the gut and gastrointestinal chemosensation. *Mol Interv.* 2008;8:78-81.

9. Bryant C, McLaughlin J. Low calorie sweeteners: evidence remains lacking for effects on *human gut function*. *Physiol Behav.* 2016;164:482-485.

10. Swithers SE, Davidson TL. A role for sweet taste: calorie predictive relations in energy regulation by rats. *Behav Neurosc.* 2008;122:161.

11. Rogers PJ, Carlyle JA, Hill AJ, et al. Uncoupling sweet taste and calories: comparison of the effects of glucose and three intense sweeteners on hunger and food intake. *Physiol Behav.* 1988;43:547-552.

12. Appleton KM, Tuorila H, Bertenshaw EJ, et al. Sweet taste exposure and the subsequent acceptance and preference for sweet taste in the diet: systematic review of the published literature. *Am J Clin Nutr.* 2018;107: 405-419.

13. Suez J, Korem T, Zeevi D, et al. Artificial sweeteners induce glucose intolerance by altering the gut microbiota. *Nature.* 2014;514:181.

14. Palmnäs MSA, Cowan TE, Bomhof MR, et al. Low-dose aspartame consumption differentially affects gut microbiota-host metabolic interactions in the diet-induced obese rat. PLoS ONE. 2014;9(10):e109841.

15. Magnuson BA, Carakostas MC, et al. Biological fate of low-calorie sweeteners. *Nutr Rev.* 2016;74:670-689.

16. Nettleton JE, Reimer RA, Shearer J. Reshaping the gut microbiota: impact of low calorie sweeteners and the link to insulin resistance? *Physiol Behav.* 2016;164:488-493.

17. Higgins KA, Considine RV, Mattes RD. Aspartame consumption for 12 weeks does not affect glycemia, appetite, or body weight of healthy, lean adults in a randomized controlled trial. *J Nutr.* 2018;148:650-657.

18. Bryant CE, Wasse LK, Astbury N, et al. Non-nutritive sweeteners: no class effect on the glycaemic or appetite responses to ingested glucose. *Eur J Clin Nutr.* 2014;68:629-631.

19. Steinert RE, Frey F, Töpfer A, et al. Effects of carbohydrate sugars and artificial sweeteners on appetite and the secretion of gastrointestinal satiety peptides. *Br J Nutr.* 2011;105:1320-1328.

20. Hall WL, Millward DJ, Rogers PJ, et al. Physiological mechanisms mediating aspartame-induced satiety. *Physiol Behav.* 2003;78(4–5):557-562.

21. Engel S, Tholstrup T, Bruun JM, et al. Effect of high milk and sugar-sweetened and non-caloric soft drink intake on insulin sensitivity after 6 months in overweight and obese adults: a randomized controlled trial. *Eur J Clin Nutr.* 2018;72:358-366.

22. Brown RJ, Walter M, Rother KI. Ingestion of diet soda before a glucose load augments glucagon-like peptide-1 secretion. *Diabetes Care.* 2009;32;2184-2186.

23. Sylvetsky AC, Brown RJ, Blau JE, et al. Hormonal responses to non-nutritive sweeteners in water and diet soda. *Nutr Metab* (Lond). 2016;13:71.

24. Dietary Guidelines for Americans Committee. Scientific Report of the 2015 Dietary Guidelines Advisory Committee. In: USDA, ed2015.

Fiber as a Modulator of the Gut Microbiota

by Daniel So, APD, Heidi M. Staudacher, APD, PhD and Katrina L. Campbell, AdvAPD, PhD

The gut microbiota describes the trillions of microbes living along our gastrointestinal (GI) tract.[1] Most of these microbes reside in our colon, where the anaerobic conditions, large surface area, and availability of undigested nutrients provide an environment that supports their growth.[2,3] The relationship between the helpful gut microbiota and human host is mutually beneficial. In exchange for the favorable colonic environment, the microbiota provides several health benefits, including producing beneficial metabolites, supporting immune function, and contributing to nutrition status.[3-5]

Bacteria make up nearly all (>99%) of the microbial population of the gut microbiota.[6] Broadly speaking, these bacteria are considered potentially beneficial or harmful based on their metabolic activities or capabilities. Specific carbohydrate-utilizing bacteria, such as *Bifidobacterium* and *Lactobacillus* species, as well as a group of bacteria known as "keystone" species, are recognized as potentially beneficial to health.[7,8] One key mechanism for this health benefit is in the generation of butyrate, a short-chain fatty acid (SCFA) metabolite with potent anti-inflammatory properties.[5] Other major SCFAs, acetate and propionate, also confer a number of putative health benefits both systemically and locally in the colon.[5] The abundance of these bacterial species, as well as the diversity of the microbial population and the types of metabolites produced, are typically used as indicators of the health of the gut microbiota.[9]

Multiple studies have recently shown that disturbances to the composition of the gut microbiota are associated with a range of different inflammatory and metabolic conditions[10] such as type 2 diabetes mellitus[11] and inflammatory bowel disease.[12] Such links present the gut microbiota as a potentially modifiable risk factor for the development of these conditions.

Modulators of the Gut Microbiota

The composition of the gut microbiota evolves throughout the lifecycle, affected by modifiable and unmodifiable factors,[3,9] with antibiotic use and diet considered the most influential modifiable drivers of gut microbiota composition.[13]

Antibiotics dramatically alter the gut microbiota, decreasing the abundance of many bacterial species and reducing the diversity of microbial population.[14,15] Although the microbiota may be capable of returning to its original composition following antibiotic use, a complete return to the initial state is rare, with composition alterations persisting for up to 2 years.[14,16] Furthermore, not all bacterial species will return following antibiotic use, with some microbes lost from the community indefinitely.[16]

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Diet has been shown to modulate gut microbiota composition in both the short and long term by altering the availability of nutrients for bacterial fermentation. Undigested nutrients, such as fiber and protein, pass through to the colon and come into contact with the gut microbiota. Here they undergo fermentation, providing energy to support bacterial growth and enabling production of a range of metabolic byproducts with various implications on host health.[17] Broadly speaking, the fermentation of fiber leads to generation of the SCFAs, while protein fermentation produces less favorable byproducts such as ammonia and phenols.[18]

Short-term dramatic dietary change leads to rapid and marked changes in gut microbiota composition. This has been shown in a landmark study involving 11 healthy adults consuming either entirely animal or entirely plant-based diets for 5 days.[19] The animal-based diet impacted the composition of the gut microbiota in a profound manner, leading to a distinctly different microbiota composition from increases in the abundance of bile-tolerant bacteria as well as decreases in the abundances of several important, carbohydrate-utilizing bacteria species.[19] These shifts in bile-tolerant bacterial abundances may lead to greater production of secondary bile acids and alter fecal bile profiles, potentially promoting enteric diseases such as liver cancer.[20] Although changes to the gut microbiota composition were less pronounced on the plant-based diet, it was able to maintain diversity of the microbial population as well as produce significantly greater amounts of SCFAs.[19] This demonstrates the rapidity in which extreme dietary shifts can lead to compositional and metabolic changes in the gut microbiota.

In addition to the potential for short-term dietary change to shift gut microbiota composition, there is evidence that long-term dietary habits dictate the microbial profile of the host. Cross-sectional studies of in-

dividuals from Africa and Europe have demonstrated that those who consume a plant-based diet high in dietary fiber have greater microbial diversity in the gut compared with matched populations who follow low-fiber Western style eating patterns.[21,22] Collectively, these dietary studies demonstrate that the intake of fiber, frequently fermentable in nature, appears to support a more favorable bacterial composition. In fact, recent data suggest that intake of more than 30 types of plants is linked with not only greater diversity of the gut microbiota, but also a higher abundance of beneficial *Faecalibacterium prausnitzii* and a reduced number of antibiotic-resistant genes.[23] Increasing fiber intake may also impact the gut microbiota indirectly by altering the colonic environment through increasing transit time and lowering colonic pH.[24,25]

Dietary Fiber

Dietary fiber describes a group of carbohydrates that escape digestion in the small intestine.[26] There are a large range of types of fibers that vary in chain length (number of monomeric units), particle size, viscosity, and fermentability.[27,28] Based on these properties, different fibers provide distinct physiological effects along the GI tract. For example, fibers with high viscosity delay gastric emptying and attenuate nutrient absorption, while other less viscous fibers can contribute to stool bulk and speed up colonic transit.[27,29,30] Furthermore, fermentable fibers provide fuel for carbohydrate-utilizing bacteria to support their growth, producing byproducts such as SCFAs as a result.[27]

A large and consistent body of evidence has linked the physiological effects of fiber, such as blood glucose control, interference with cholesterol production, and laxation, to a wide variety of health benefits when it is consumed in adequate quantities. These benefits include a reduced risk of cardiovascular disease and colorectal cancers, and promotion of GI health.[28,31]

Prebiotic Fibers

In addition to the general physiological effects of dietary fiber, some types of fiber have specific properties related to their classification as prebiotics. A prebiotic is defined as "a substrate that is selectively utilized by host microorganisms conferring a health benefit."[8] While most types of prebiotics are dietary fibers, only a subset of fibers meet the criteria for prebiotic classification. The microorganisms typically investigated as targets of this selective stimulation are *Bifidobacterium* and *Lactobacillus* species, while increasing attention is being turned to the keystone bacterial species such as *F. prausnitzii* and *Ruminococcus bromii,* some of which are involved in the production of butyrate.[8,32]

It should be noted that all fibers have the potential to impact gut microbiota composition, and the prebiotic distinction lies within the selectivity of its impact.

The prebiotic definition has been a moving target over the past decade, with previous iterations the distinguishing prebiotics as either "accepted" or "candidate" based on the level of evidence available.[33] As such, inulin-type fructans and galacto-oligosaccharides (GOS) that are rapidly and readily fermentable fibers are classified as accepted prebiotic fibers, while candidate prebiotic fibers have included a broader, more varied range of fermentable fibers. Candidate prebiotics include resistant oligosaccharides (polydextrose and resistant starch) that have demonstrated evidence for shifting microbiota composition, but their effects in selectively stimulating the gut microbiota are not as well understood.[33] It should be noted that all fibers have the potential to impact gut microbiota composition, and the prebiotic distinction lies within the selectivity of its impact.

Gaps in Our Understanding

Most of the understanding of the impact of dietary fiber on the gut microbiota until recently has been centered on whole-diet interventions, cross-sectional associations, and interventions involving specific prebiotic fibers. The totality of the evidence makes it clear that a higher fiber intake is linked with altered microbiota profile compared with lower fiber intakes. However, evidence from whole diet alterations and cross-sectional studies are unable to tease out the precise impact of fiber due to the presence of multiple confounders (e.g., other lifestyle factors in cross-sectional studies and change in other dietary components in whole diet studies).[13,19,21,22] Although prebiotic fiber has been tested in robust supplementation trials,[33] these represent only a subset of total dietary fiber. Furthermore, large disparities exist across the prebiotic trials, including differences in study design, the type and amount of prebiotic tested, and patient population tested. Therefore, our understanding of the precise effect of dietary fiber and the gut microbiota is still limited, particularly with regard to its effect on diversity, abundances of specific important species, and byproducts known to be important to health.

Our Research on Fibers and Gut Microbiota

We attempted to better understand how fibers impact the gut microbiota

by conducting a systematic review and meta-analysis. Following is a summary of our research:

Methods

We performed a systematic review and meta-analysis to assess the impact of dietary fiber intervention on the gut microbiota in healthy individuals.[34] We included randomized controlled trials (RCTs) of healthy adults that compared the effect of fiber intervention on gut microbiota composition of fecal samples with the effect of either a placebo or low fiber comparator.

The difference in the diversity of gut microbiota between the two groups was the primary outcome of this study. Between-group differences in specific bacterial abundances (including *Bifidobacterium* and *Lactobacillus* spp) were secondary outcomes and fecal SCFAs were exploratory outcomes.

Additional analyses included a subgroup analysis to evaluate whether there were differences in effect based on fiber type. For this, fibers were classified as either accepted prebiotic fibers, candidate prebiotic fibers, or general fiber if it did not fit under the two previous classifications.

Findings

The primary finding of this systematic review and meta-analysis review was that dietary fiber intervention did not lead to changes in the diversity of the gut microbiota but did lead to a greater abundance of *Bifidobacterium* ($P<.001$) and *Lactobacillus* ($P=.02$) species as well as fecal butyrate ($P=.05$) compared with comparators. Fiber intervention did not affect abundances of other bacterial species assessed (e.g., *F. prausnitzii*) or other SCFAs.

The subgroup analysis showed that not all types of fibers affected the gut microbiota in the same manner. For example, only accepted prebiotic fibers and candidate prebiotic fibers led to greater abundances of *Bifidobacterium* ($P<.001$) but general fibers had no effect compared with placebo and low fiber comparators ($P=.24$). The effect was even more specific for *Lactobacillus* species, with only the accepted prebiotic fiber intervention leading to greater abundance compared with placebo and low fiber comparators ($P=.002$).

Translating Results into Practice and Research

Although increases in short-term dietary fiber intake may not impact diversity of the gut microbiota, consumption of certain fibers, namely the prebiotic fibers fructans and GOS, stimulate growth of beneficial bacteria *Bifidobacterium* and *Lactobacillus* species. Not all types of fiber, according to our findings, directly impact the composition of the gut microbiota. However, this does not discount the other beneficial physiological effects of fibers throughout the GI tract that are known to have favorable impactors on health. Together with the capacity of some fibers to modulate microbiota composition and functions, this confirms the place of fiber at the center of a nutritious and varied diet.

Together with the capacity of some fibers to modulate microbiota composition and functions, this confirms the place of fiber at the center of a nutritious and varied diet.

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References

1. Sender R, Fuchs S, Milo R. Revised estimates for the number of human and bacteria cells in the body. *PLoS Biol.* 2016;14(8):e1002533.

2. Morgan XC, Huttenhower C. Chapter 12: Human microbiome analysis. *PLoS Comput Biol.* 2012;8(12): e1002808.

3. Sekirov I, Russell SL, Antunes LC, et al. Gut microbiota in health and disease. *Physiol Rev.* 2010;90:859-904.

4. Backhed F, Ley RE, Sonnenburg JL, et al. Host-bacterial mutualism in the human intestine. *Science (New York, NY).* 2005;307:1915-1920.

5. Gill PA, Zelm MC, Muir JG, et al. Review article: short chain fatty acids as potential therapeutic agents in human gastrointestinal and inflammatory disorders. *Aliment Pharmacol Therapeutics.* 2018;48:15-34.

6. Qin J, Li R, Raes J, et al. A human gut microbial gene catalogue established by metagenomic sequencing. *Nature.* 2010;464:59-65.

7. Banerjee S, Schlaeppi K, van der Heijden MGA. Keystone taxa as drivers of microbiome structure and functioning. *Nature Rev Microbiol.* 2018;16;567-576.

8. Gibson GR, Hutkins R, Sanders ME, et al. Expert consensus document: The International Scientific Association for Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of prebiotics. *Nat Rev Gastroenterol Hepatol.* 2017;14:491-502.

9. Marchesi JR, Adams DH, Fava F, et al. The gut microbiota and host health: a new clinical frontier. *Gut.* 2016;65: 330-339.

10. de Vos WM, de Vos EA. Role of the intestinal microbiome in health and disease: from correlation to causation. *Nutr Rev.* 2012;70 (Suppl) 1:S45-56.

11. Karlsson FH, Tremaroli V, Nookaew I, et al. Gut metagenome in European women with normal, impaired and diabetic glucose control. *Nature.* 2013;498:99-103.

12. Ni J, Wu GD, Albenberg L, et al. Gut microbiota and IBD: causation or correlation? *Nat Rev Gastroenterol Hepatol.* 2017;14:573-584.

13. Zhernakova A, Kurilshikov A, Bonder MJ, et al. Population-based metagenomics analysis reveals markers for gut microbiome composition and diversity. *Science (New York, NY).* 2016;352:565-569.

14. Francino MP. Antibiotics and the human gut microbiome: dysbioses and accumulation of resistances. *Frontiers Microbiol.* 2015;6:1543.

15. Modi SR, Collins JJ, Relman DA. Antibiotics and the gut microbiota. *J Clin Invest.* 2014;124:4212-4218.

16. Jernberg C, Lofmark S, Edlund C, Jansson JK. Long-term ecological impacts of antibiotic administration on the human intestinal microbiota. *ISME Journal.* 2007;1(1):56-66.

17. Vieira-Silva S, Falony G, Darzi Y, et al. Species–function relationships shape ecological properties of the human gut microbiome. *Nat Microbiol.* 2016;1:16088.

18. Yao CK, Muir JG, Gibson PR. Review article: insights into colonic protein fermentation, its modulation and potential health implications. *Aliment Pharmacol Ther.* 2016;43:181-196.

19. David LA, Maurice CF, Carmody RN, et al. Diet rapidly and reproducibly alters the human gut microbiome. *Nature.* 2014;505:559-563.

20. Yoshimoto S, Loo TM, Atarashi K, et al. Obesity-induced gut microbial metabolite promotes liver cancer through senescence secretome. *Nature.* 2013;499:97-101.

21. Schnorr SL, Candela M, Rampelli S, et al. Gut microbiome of the Hadza hunter-gatherers. *Nature Commun.* 2014;5:3654.

22. De Filippo C, Cavalieri D, Di Paola M, et al. Impact of diet in shaping gut microbiota revealed by a comparative study in children from Europe and rural Africa. *Proc Nat Acad Sci USA.* 2010;107:14691-14696.

23. McDonald D, Hyde E, Debelius JW, et al. American gut: an open platform for citizen science microbiome research. *mSystems.* 2018;3(3).

24. Falony G, Vieira-Silva S, Raes J. Richness and ecosystem development across faecal snapshots of the gut microbiota. *Nat Microbiol.* 2018;3:526-528.

25. Gibson GR, Scott KP, Rastall RA, et al. Dietary prebiotics: current status and new definition. *Food Sci Technol Bull: Functional Foods.* 2010;7:1-19.

26. Food and Agriculture Organization of the United Nations (FAO)/World Health Organization (WHO). CODEX Alimentarius (CODEX). Guidelines on Nutrition Labeling CAC/GL 2–1985.

27. Eswaran S, Muir J, Chey WD. Fiber and functional gastrointestinal disorders. *Am J Gastroenterol.* 2013;108: 718-727.

28. Stephen AM, Champ MM, Cloran SJ, et al. Dietary fibre in Europe: current state of knowledge on definitions, sources, recommendations, intakes and relationships to health. *Nutr Res Rev.* 2017:1-42.

29. Clemens R, Kranz S, Mobley AR, et al. Filling America's fiber intake gap: summary of a roundtable to probe realistic solutions with a focus on grain-based foods. *J Nutr.* 2012;142: 1390s-1401s.

30. Lattimer JM, Haub MD. Effects of dietary fiber and its components on metabolic health. *Nutrients.* 2010;2:1266-1289.

31. Veronese N, Solmi M, Caruso MG, et al. Dietary fiber and health outcomes: an umbrella review of systematic reviews and meta-analyses. *Am J Clin Nutr.* 2018;107:436-444.

32. Flint HJ, Duncan SH, Louis P. The impact of nutrition on intestinal bacterial communities. *Curr Opin Microbiol.* 2017;38:59-65.

33. Roberfroid M, Gibson GR, Hoyles L, et al. Prebiotic effects: metabolic and health benefits. *Brit J Nutrition.* 2010;104 (Suppl 2):S1-63

34. So D, Whelan K, Rossi M, et al. Dietary fiber intervention on gut microbiota composition in healthy adults: a systematic review and meta-analysis. *Am J Clin Nutr.* 2018;107:965-983.

Role of Psychoneuroendocrine Factors in Menstrual Dysfunction Among Athletes

by Sarah LaCourse, NASM-CPT

The concurrence of menstrual dysregulation with other physiological and psychological conditions has been a topic of discussion related to female health. Relative energy deficiency in sport (RED-S) and the female athlete triad ("triad") are two conditions addressing the interrelationship between an athlete's energy status, physiological health, and subsequent injury risk.[1-3] Whereas the triad involves a syndrome of menstrual dysfunction, low energy availability (EA), and low bone mineral density (BMD),[1] RED-S addresses additional complex factors resulting from inadequate EA, including psychological involvement, cardiovascular effects, and immune system function, and it also includes males.[2]

While the athletic population is susceptible to these conditions, non-athletic females are also at risk for developing menstrual dysregulation and associated physiological concerns potentially due in part to stress-related underpinnings of hormone cycle fluctuations.[4-6] Because of the multitude of factors involved in menstruation, determining the root of dysregulation is critical for prevention of downstream effects from irregular cycles that could include low BMD resulting in osteopenia or osteoporosis, reproduction concerns, and changes in cardiovascular health.[1,2] Ideally practitioners, coaches, and athletes should focus on understanding the risk and potential for developing RED-S and associated concerns, and aim for prevention by supporting the physiological and psychological needs of individual athletes during and beyond athletic endeavors.

Neuroendocrine Pathways

The factors affecting menstrual function and those contributing to menstrual irregularities are multifaceted. Abnormal menses can be described by two primary categories: oligomenorrhea (cycles >35 days in duration) and amenorrhea (lack of three consecutive menstrual cycles, or absence of a cycle for >90 days).[1] A type of amenorrhea common for female athletes is functional hypothalamic amenorrhea (FHA), which occurs when disruption to the hormones involved in neuroendocrine and reproductive functions occurs as a result of low EA due to either reduced dietary intake, increased energy expenditure, or a combination of both.[1-3]

Changes in the neuroendocrine axes, which include the hypothalamic-pituitary-adrenal (HPA), the hypothalamic-pituitary-gonadal (HPG), and the hypothalamic-pituitary-thyroid (HPT) axes, may contribute to the clinical or subclinical menstrual disturbances previously discussed.[5,7,8] Moreover, the hormones involved in communication between axes, including estrogen, estradiol, progesterone, gonadotropin releasing hormone (GnRH), luteinizing hormone (LH), follicle stimulating hormone (FSH), corticotropin releasing hormone (CRH), adrenocorticotropic hormone (ACTH), cortisol, leptin, and growth hormone (GH), also affect menstruation.[5,7-10] In addition to the individual functions of each chemical, multiple feedback mechanisms are involved in the regulation of hormone levels and overall functioning within the neuroendocrine axes.[9] Specifically, ovulation is controlled in part by feedback throughout the HPG system by way of GnRH secretion from the hypothalamus, and LH and FSH pulses from the pituitary that signal the ovaries, the site of estrogen and progesterone production.[9]

An adequate level of energy intake (EI) is essential for both optimal physiological functioning and overall health.[1,2,8-11] Deviations in energy status, specifically low EA, have been shown to alter the cascade of regulatory neuroendocrine pathways. Specifically, one alteration with profound impact to menstrual function is LH pulsatility.[9,10] This begins with a change to the pattern of GnRH release from the hypothalamus, the hormone that stimulates the gonadotropins LH and FSH. A change in LH pulsatility creates disruption in ovary function, thereby affecting estrogen and progesterone release.[8-12] Beyond ovarian function, differences in LH pulsatility can also increase chemicals released within the HPA axis and decrease the chemicals released from the HPT axis, which may amplify ongoing hormonal dysfunction.[7,9,13] Women with FHA show variance in GnRH, LH and FSH, estrogen, progesterone, thyroid hormones, and cortisol.[7-13]

A threshold may exist regarding overall EA, with research in young adult women indicating that EA <30 kcal/kg fat free mass (FFM) per day contributes to a decrease in frequency and an increase in the amplitude of LH pulses.[12] Of note, that study was conducted during an acute period and may not represent females in a state of chronic low EA, chronic stress, or male athletes.[12]

Psychological Involvement and Stress

The triad and RED-S both include underlying EA concerns, with or without the presence of disordered eating or a clinical eating disorder (ED).[1,2] While it is important to evaluate for disordered eating or a clinical ED, ongoing stress may also be present with menstrual dysfunction.[7,14,15] Psychological stressors are common among the athletic population and can lead to

various effects on hormones depending on how the individual athlete responds to stressors.[7] Individual response to stress can be classified as "allostatic load," which purportedly considers the body's ability to integrate and adapt to stress as a means to support homeostasis.[7]

Various stressors can be at play, including home, school, and work problems, daily living, and the stress of physically pushing one's body and having increased energy demands.[7,14,15] A higher resilience to external and internal stressors is supportive of hormonal function because fewer adaptations to sustain homeostasis are required.[7] Response and tolerance to stress will vary from person to person, based on multiple factors. Indeed, personality traits including perfectionism and desire for social approval have been found in females with FHA,[15] and these traits are likely involved in how stressors are tolerated.[7] To add to personality traits that potentially work against an individual's level of resilience to stress, Marcus et al[15] found increased disordered eating patterns even with an exclusion criterion of diagnosable EDs. Additional research identified an increase in cognitive restraint and body dissatisfaction among oligo-amenorrhoeic athletes.[16]

A potential explanation behind the physiological effects of stress is HPA axis activation leading to increases in cortisol secretion from the adrenal glands.[7,13-15,17] Females with FHA present with an increased level of serum cortisol compared with those with other patterns of menstrual irregularity and healthy controls.[15] Consistent with this theory, a sample of adolescents diagnosed with FHA reported that a stressful event such as death of a loved one, chronic disease, or change of school occurred prior to the onset of menstrual irregularity.[17] With previous stressful events, incoming stimuli may prematurely activate the HPA axis, which may reduce the threshold required to activate this negative-feedback loop in the future.[17]

Interventions for Supporting Female Athletes

Optimizing EA remains the focus when addressing FHA in female athletes, with an emphasis on providing nutritional recommendations specific to individual needs. While a level of EA <30 kcal/kg FFM/day is described as "low," the International Olympic Committee, the Female Athlete Triad Coalition, and the American College of Sports Medicine (ACSM) recommend aiming for a target of 45 kcal/kg FFM/day.[1-3] When conducting an intervention to support females dealing with low EA and menstrual dysfunction, the specific guidelines will vary depending on the individual's calculated energy needs, BMI, nutrient intake, and other nutritional risks. Research shows that appropriate EA is the key factor in overcoming neuroendocrine changes leading to disrupted or ceased menses.[12,18,19]

Indeed, Lagowska et al[18] implemented a 3-month individualized dietary intervention and found that LH concentrations moved in the direction for supporting a menstrual cycle. In this study, female athletes with oligomenorrhea or amenorrhea were prescribed a dietary plan designed around individual needs. The results showed that despite not having a return of menses at the 3-month mark, the individualized diet was associated with a positive change in LH, indicating that EA plays a large role not only in cessation of menses but also in supporting hormone cycles.[18]

Additional findings from Guebels and colleagues[19] provide insight into the connections among EA, EI, and energy balance (EB). This study examined the role of a 6-month nutritional intervention on overall EB and resumption of menses in exercise-related menstrual dysfunction. The basis of the nutritional intervention was to increase EI. Findings showed all intervention participants had return of menses, and this occurred around EA of ~40 kcal/kg FFM/day.[19]

While nutrition interventions are the preferred approach to ameliorating hormonal disruptions associated with menstrual dysfunction in athletes,[1-3,18,19] coupling nutritional counseling with mental health support may yield greater positive outcomes.[20-22] Early intervention is also key to prevention of longer-term effects such as BMD loss.[3] Exploring the connection between one's mental health and menstrual dysfunction may aid in the process of identifying appropriate and individualized interventions. In an intervention that focused on the use of cognitive behavioral therapy (CBT) as a treatment option to restore menses, it was observed that those with FHA who underwent a 20-week CBT program were more likely to resume menses than those who did not undergo CBT.[20] A follow-up study indicated that those who underwent CBT exhibited positive changes in blood concentrations of cortisol, TSH, and leptin, further indicating the connections between mental health, endocrine function, and FHA.[21]

A recent review by Kyriakidis et al[22] examined the roles of various treatment approaches when working to overcome FHA. Treatments reviewed included leptin therapy, counseling, and dietary intervention, and each style yielded positive results on hormonal status over time. Nutritional approaches to treating FHA may contribute an additional benefit of the individual adopting dietary habits that are supportive of their needs. In addition, because various forms of stress may serve as an etiological factor of oligomenorrhea and amenorrhea, it is possible that increased ability to manage stress through behavioral interventions could benefit menstrual health status.[22]

Physiological Outcomes and Long-Term Health

In addition to the neuroendocrine and metabolic adaptations occurring with FHA and associated menstrual disturbances, other possible short- and long-term implications may emerge. Two review articles focused on the role menstrual cycles play in women's health and outlined how

stress may promote changes in ovarian, neuroendocrine, and brain function.[23,24] Longer-term, chronic effects linked to FHA include adverse impacts on reproduction and fertility, bone health including osteopenia and osteoporosis, and cardiovascular health.[23,24]

Menstrual dysfunction is highly complex and involves EA, neuroendocrine axes, metabolic pathways, and stress.[1-3] Due to interactions among multiple systems, acute and chronic symptoms and outcomes, as well as various stages of menstrual irregularity, evaluation and treatment must be individualized. An individualized approach to overcoming menstrual dysfunction, including FHA, is most appropriate as it will address the specific needs of the individual while optimizing the potential for a positive outcome.[1-3] Prevention protocols include education on RED-S, FHA, and EA, in addition to supporting a healthy relationship with nutrition, exercise, and performance.[2] These practices can be employed by athletes themselves and anyone directly involved in their lives including coaches, trainers, loved ones, and practitioners. If an underlying FHA pathology is occurring, taking steps to mitigate the systemic effects is crucial in the prevention of downstream complications. Moreover, a multifaceted approach to treatment that encompasses a dietitian, mental health practitioner, and physician will ultimately provide the athlete with a well-rounded support network and framework to heal the complexity of menstrual dysfunction and associated neuroendocrine networks.[1-3]

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References

1. Nattiv A, Louks AB, Manore MM, et al. American College of Sports Medicine position stand: the female athlete triad. *MSSE*. 2007;39:1867-1882.

2. Mountjoy M, Sundgot-Borgen J, Burke L, et al. The IOC consensus statement: beyond the female athlete triad – relative energy deficiency in sport (RED-S). *BJSM*. 2014;48:491-497.

3. De Souza MJ, Nattiv A, Joy E, et al. Female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad: 1st international conference held in San Francisco, California, May 2012 and 2nd international conference held in Indianapolis, Indiana, May 2013. *BJSM*. 2014;48:289

4. Torstveit MK, Sundgot-Borgen J. The female athlete triad exists in both elite athletes and controls. *MSSE*. 2005;37(9):1449-1459

5. Fourman LT, Fazeli PK. Neuroendocrine causes of amenorrhea – an update. *JCEM*. 2015;100:812-824.

6. Laughlin GA, Dominguez CE, Yen SSC. Nutritional and endocrine-metabolic-aberrations in women with functional hypothalamic amenorrhea. *JCEM*. 1998;83:25-32.

7. Pauli SA, Berga SL. Athletic amenorrhea: energy deficit or psychogenic challenge? *Ann NY Acad Sci*. 2010;1025:33-38.

8. Misra M. Neuroendocrine mechanisms in athletes. *Hand Clin Neurol*. 2014;124:373-386.

9. Berga S, Naftolin F. Neuroendocrine control of ovulation. *Gynecol Endocrinol*. 2012;28:9-13.

10. Nappi RE, Facchinetti F. Psychoneuroendocrine correlates of secondary amenorrhea. *Arch Womens Ment Health*. 2003;6:83-89.

11. Warren MP, Chua AT. Exercise-induced amenorrhea and bone health in the adolescent athlete. *Ann NY Acad Sci*. 2008;1135:244-252.

12. Loucks AB, Thuma JR. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *JCEM*. 2003;88:297–311.

13. Berga SL, Mortola JF, Girton L, et al. Neuroendocrine aberrations in women with functional hypothalamic amenorrhea. *JCEM*. 1989;68:301–308.

14. Genazzani AD, Chierchia E, Santagn S, et al. Hypothalamic amenorrhea: from diagnosis to therapeutical approach. *Ann Endocrinol*. 2010;71:163-169.

15. Marcus MD, Loucks TL, Berga SL. Psychological correlates of functional hypothalamic amenorrhea. *Fertil Steril*. 2001;76:310-316.

16. Sokoloff NC, Eguiguren ML, Wargo K, et al. Bone parameters in relation to attitudes and feelings associated with disordered eating in oligo-amenorrheic athletes, eumenorrheic athletes, and nonathletes. *Int J Eat Disord*. 2015;48:522-526.

17. Bomba M, Gambera A, Bonini L, et al. Endocrine profiles and neuropsychologic correlates of functional hypothalamic amenorrhea in athletes. *Fertil Steril*. 2007;87:876-885.

18. Lagowska K, Kapczuk K, Friebe Z, et al. Effects of dietary intervention in young female athletes with menstrual disorders. *JISSN*. 2014;11(21):1-8.

19. Guebels CP, Kam LC, Maddalzzo GF, et al. Active women before/after an intervention designed to restore menstrual function: resting metabolic rate and comparison of four methods to quantify energy expenditure and energy availability. *IJSNEM*. 2014;24:37-46.

20. Berga SL, Marcus MD, Loucks TL, et al. Recovery of ovarian activity in women with functional hypothalamic amenorrhea who were treated with cognitive behavior therapy. *Fertil Steril*. 2003;80:976–981.

21. Michopoulos V, Mancini F, Loucks TL, et al. Neuroendocrine recovery initiated by cognitive behavioral therapy in women with functional hypothalamic amenorrhea: a randomized, controlled trail. *Fertil Steril*. 2013;99:2084-2091.

22. Kyriakidis M, Caetano L, Anastasiadou N, et al. Functional hypothalamic amenorrhea: leptin treatment, dietary intervention and counselling as alternatives to traditional practice – systematic review. *EJOG*. 2015;198:131-137.

23. Prokai D, Berga SL. Neuroprotection via reduction in stress: altered menstrual patterns as a marker for stress and implications for long-term neurologic health in women. *IJMS*. 2017;17(2147):1-8.

24. Meczekalski B, Katulski K, Czyzyk A, et al. Functional hypothalamic amenorrhea and its influence on women's health. *J Endocrinol Invest*. 2014;37:1049-1056.

SCAN's New Frontier: Optimizing Human Performance

by Lindzi S. Torres, MPH, MS, RDN, CSSD

"The only thing worse than being blind is having sight but no vision." — Helen Keller

As you read this we will have wrapped up another great gathering at the Food & Nutrition Conference & Expo™ (FNCE®). One highlight of FNCE® was the exciting reveal of SCAN's new vision and mission. Helen Keller's words ring true in our goal to move the vision of our practice group into a new frontier. We hope that you, as a member of SCAN and the dietetics community at large, will embrace our new vision and mission that seek to be more inclusive of all realms of physical performance, across the full spectrum of an individual's human potential.

Our New, More Inclusive Vision and Mission

SCAN's new vision and mission, developed and approved by your Executive Committee, are as follows:

Vision: A world where all people perform to their potential, powered by nutrition and physical activity

Mission: Optimize health and human performance through the integration of nutrition with sports, physical activity, cardiovascular health, and well-being

What Is Human Performance?

If you google "human performance," you'll get more than 1.2 billion results. There are many definitions of human performance, ranging from organizational change to optimization at the microcellular level of the human body. For SCAN, we view human performance as the potential within all individuals to optimize their health through nutrition and physical activity in the arenas where they choose to engage.

Why Human Performance?

Models of nutrition in the realm of "performance" have focused mostly on more traditional physical activity and sports. The goal of our new mission is to be more inclusive and consider all human activities in the quest to optimize performance. This mindset reminds me of Nike's definition of an athlete: "If you have a body, you are an athlete." We aim to empower individuals in multiple aspects of their life through nutrition and physical activity.

These individuals may be professional athletes, construction workers, clerical aids, first responders, artists, dancers, or musicians. They may be operating at an elite or beginner's level. They may be able-bodied, or have physical or cognitive disabilities. SCAN's new vision and mission embrace the power of nutrition to tap the full potential of human performance for individuals across a range of physical, cognitive, occupational, and life stage realities. SCAN is strongly committed to our practice areas, and this new effort seeks to amplify our programing in current areas and expand the aperture of our skill sets to the next level of human performance.

I eagerly look forward to taking this journey with you. We'll have an opportunity to discuss in person at the 35th Annual SCAN Symposium, *Navigating the Path of Wellness*, in Phoenix, AZ, on April 26-28, 2019. For more details, see "Upcoming Events" in this issue (page 20), and be sure to mark your calendars now!

What Else Happened at FNCE®?

In addition to introducing SCAN's new mission and vision, FNCE® 2018 provided attendees with a host of educational and networking opportunities, such as:

- **SCAN Sports Workshop.** This insightful workshop—*Enhancing Your Sports Nutrition Practice*—was sponsored by Gatorade Sports Science Institute and featured Janet Rankin, Scott Sehnert, and Brett Singer as presenters.

- **SCAN Yoga Session.** Many members took a break from educational endeavors and took advantage of the rejuvenating yoga session planned by SCAN and sponsored by RXBAR.

- **SCAN Morning and Afternoon Sessions.** Sunday's CE session, sponsored by Eating Recovery Center, delved into *Unmasking & Understanding BED: Nutrition Interventions for the Most Common (and Most Overlooked?) Eating Disorder.* Monday's CE session, sponsored by RXBAR, explored *The Science Behind Protein, Nuts and Dates.*

- **SCAN Reception.** Held at the International Spy Museum in Washington, DC, the SCAN Reception offered lively networking and was the backdrop for unveiling SCAN's new vision and mission at a special "TopSecret" event.

- **SCAN FNCE® Spotlight Session.** Themed *Heart of an Athlete: Managing Hypertension in Athletic Populations,* this in-depth presentation featured speakers Jackie Buell and Alan Hinderliter.

Whether you were in attendance at the recent FNCE® or were unable to make it this year, you can nevertheless view pictures from these events and see how your colleagues took advantage of connecting with others and staying on top of their professional know-how. Be sure to check out these photos at #FNCE. And plan to attend next year!

American College of Sports Medicine 65th Annual Meeting

Minneapolis, MN • May 29 – June 2, 2018

If you haven't yet attended an annual meeting of the American College of Sports Medicine (ACSM), you might want to plan to do so. The conference is excellent for sports dietitians who want to be on the cutting edge of sports nutrition research. While the meeting officially starts on a Wednesday, free pre-conferences that focus on nutrition are usually scheduled on Tuesday. Below is a summary of information presented at the Gatorade Sport Science Institute (GSSI) Sports Nutrition Pre-Conference: *Revisiting the Basics.*

Protein Intake and Muscle-Building

If an athlete wants to build muscle, when is the best time to eat protein: before, during, or after lifting weights?

Trent Stellingwerff, PhD of the Canadian Sport Institute suggested that timing might not actually matter. While resistance exercise stimulates a muscle-building effect that is most robust within the first 4 hours after a workout, the effect lasts for about 2 days, at least in untrained to moderately trained individuals. Hence, athletes want to pace their protein intake evenly throughout the day, but they need not carry a protein shake around the gym!

Resistance exercise is far more potent than a high-protein diet for increasing strength and muscle gains. That said, most athletes could expect to see a gain of only about 2 lb (1 kg) of muscle in 13 weeks. That's not much in comparison to what they really want to see.

Maximal anabolic (muscle-building) effects are seen with about 25 g to 30 g of protein per meal. More precisely, that is 0.75 g of protein per pound (1.6 g/kg) body weight per day, or 0.1 to 0.2 g of protein per pound (0.25-0.4 g/kg) *per meal* in young men. More than that has little or no further benefit.

These recommendations, however, change with age. Older athletes need more of the amino acid leucine to trigger the anabolic response seen in young men. Hence, individuals older than age 50 should target about 0.16 g of protein per pound (0.35 g pro/kg) for their post-exercise recovery meal, and should choose high-quality dietary protein (milk, egg, fish, soy, etc.). For a 150-lb (68-kg) athlete, that's about an additional 10 g of protein per meal. That's not much—only a glass or milk or 1.5 oz of meat, fish, or chicken.

Nicholas Burd, PhD of the University of Illinois reminded the audience that, despite rumors to the contrary, protein neither damages the kidneys nor causes a decline in kidney function. Even people with chronic kidney disease should consume the recommended dietary allowance for protein (0.8 g/kg). A high-protein diet also does not cause bone loss. Bone is 40% to 50% protein (collagen).

Burd went on to say that over-consuming protein is not only a waste of money but it also stresses the environment. Athletes need to take a holistic, whole foods approach to their diets. Natural protein-rich foods, as opposed to processed supplements, are best (if compatible with the training schedule) because they offer a complex and complete matrix that is more effective than processed proteins. One example of the benefits of whole foods can be seen with eggs. A whole egg promotes 40% greater muscle protein synthesis in the 5 hours post-exercise compared with eating just the egg white (van Vliet S, et al. *Am J Clin Nutr.* 2017;106:1401-1412). Nutrient interactions seem to facilitate a more robust response compared with eating isolated protein.

High-Carbohydrate vs High-Fat Sports Diet

Which will better enhance athletic performance: a high-carbohydrate or a high-fat sports diet?

Despite growing interest in a high-fat sports diet, Gareth Wallis, PhD of the University of Birmingham, UK reported that research does not support this kind of diet for athletes who exercise at high intensity. Rather, research supports consuming 3 g to 4.5 g of carbohydrate per pound (7-10 g carb/kg) body weight per day to be well-fueled for hard training and competitive events.

Grains, fruits, and vegetables are crucial for athletes who want to exercise vigorously. Some athletes eat a high-fat diet for training and then switch to carb-loading before a competitive event, but that's a bad idea. The enzymes involved in metabolizing carbohydrate become less active, so the muscles are less able to access carbohydrate for fuel when it is needed for winning sprints and surges.

Sports Supplements

Is there one single sport supplement that works for all athletes?

To better understand why a supplement is unlikely to be effective for all athletes, Eric Rawson, PhD, RD of Messiah College in Pennsylvania suggested that we need a more specific

scientific approach to studying supplements based on age, sex, body size, training status, and genetics. That would help us give better advice to target groups of athletes, rather than simply make population-wide recommendations. Many athletes take multiple supplements, so research with "stacked" supplements would also be helpful. Here are some findings regarding three popular sport supplements:

- *Creatine* enables an athlete to lift harder in the training room and build more muscle. However, not everyone is a responder. For example, 3 of 11 subjects in a research study had a strong positive response, 5 had a slight response, and 3 did not respond at all (Syrotuik DG, Bell GJ. J *Strength Cond Res.* 2004;18:610-617). Why not? Their daily diets might have impacted their baseline creatine levels. Creatine is found in meat and other animal proteins. When meat-eating athletes consumed a lacto-ovo-vegetarian diet for 26 days, their creatine levels dropped (Lukaszuk JM, et al. *J Strength Cond Res.* 2005;19:735-740). Hence, to normalize creatine levels for a research study, vegetarian athletes might need to take creatine monohydrate supplements (the most effective form of creatine).

- *Caffeine* is a known energy-enhancing sport supplement. An athlete's response to caffeine will depend on his or her genetics. Caffeine works best when an athlete is starting to fatigue. Athletes can consume caffeine in coffee, tea, soda, gels, gum, and pills, preferably in combination with carbohydrate to boost effectiveness.

- *Sodium bicarbonate* is used by some athletes to buffer the lactic acid that builds up during intense bursts of exercise. Research suggests that response times to peak blood levels can vary widely, from 40 to 165 minutes. (Jones RL, et al. *Int J Sport Nutr Exerc Metab.* 2016;26:445-453). This variability makes it hard for exercise scientists to offer firm recommendations. Hence, outcomes vary from athlete to athlete. Sub-elite athletes seem to respond better than elite athletes. Because sodium bicarbonate easily causes nausea and vomiting, a solution is to take it in gastro-resistant capsules.

Sodium Lost in Sweat

How concerned do athletes need to be regarding sodium lost via sweating?

The amount of sodium lost in sweat varies from a lot to a little, related to both sweat rate and how well an athlete is acclimated to exercising in the heat, among other factors. Lindsay Baker, PhD of the Gatorade Sports Science Institute reminded the audience that when athletes sweat, they lose proportionately more water than sodium. Hence, sodium levels in the blood increase with dehydration. A high concentration of sodium in the blood stimulates thirst.

Athletes have three ways they commonly manage fluid replacement: (1) "hit-or-miss" ad-libitum drinking as desired; (2) drinking to quench thirst; and (3) drinking on a set schedule. The effectiveness of these strategies depends on the individual athletes, availability of fluids, the weather, and exercise intensity and duration. Athletes with several tattoos should take note: tattooed skin may sweat less and excrete saltier sweat.

Conference Highlights Editor Nancy Clark, MS, RD counsels both casual and competitive athletes at her private practice in the Boston area. She is author of the best selling Nancy Clark's Sports Nutrition Guidebook *and co-leader of an online sports nutrition workshop. For more information, visit www.NancyClarkRD.com.*

Reviews

Nutrition for Sport, Exercise, and Health

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www.HumanKinetics.com
2018, softcover, 476 pp, $129.00
ISBN 978-1-4504-1487-6

Nutrition and physical activity have become common themes in today's society. It is now so common that we're starting to be constantly exposed to new research, conflicting perspectives, and misinformation. In *Nutrition for Sport, Exercise, and Health*, Marie A. Spano, Laura J. Kruskall and D. Travis Thomas break down the concepts of basic nutrition and reconstruct these basics to compliment exercise science based on science and its implementation.

Paraphrasing the most recent research, this full-color text begins by presenting the basics of nutrition, introducing the essential energy processes necessary to understand the human body, food, and physical activity. It goes on to explore the foundations of nutrition, and then takes a dive into the functions and recommendations of carbohydrate, fat, and protein along with their micronutrient counterparts. The text continues on to explain the importance of hydration and electrolyte status; the role of dietary supplementation; body weight and composition; and nutrition considerations for various degrees of training and sports alike. As a final note, *Nutrition for Sport, Exercise, and Health* touches on specific nutrition concerns that may arise when considering special populations. It also includes more than 100 illustrations, a glossary, chapter objectives, reflection questions, and perspective sidebars to help prompt the reader to consider certain things in practice.

Overall the book discusses nutrition and its relationship with physical activity in a seemingly logical sequence. This allows the reader to build upon previous information and advance at a progressive pace. The authors provide readers with tools to help readers distinguish fact from fiction in future research, and perhaps in their own personal lives.

Marie Spano, Laura Kruskall and D. Travis Thomas are coauthors specializing in the area of sports nutrition and dietetics through university and research practices.

Reviewed by Arda Buyuktimkin, MS, RD, renal dietitian for Fresenius Kidney Care and consultant dietitian for Nutrition Solutions in Beaumont, TX

Research Digest

Blueberry Metabolites Exhibit Vascular Benefits

Bharat D, Cavalcanti RRM, Petersen C, et al. Blueberry metabolites attenuate lipotoxicity-induced endothelial dysfunction. *Mol Nutr Food Res.* 2018;62:1700601-170068.

Blueberries are an excellent source of anthocyanins, which have been found to improve cardiovascular health in clinical and laboratory trials; however, the mechanism is unknown. When blueberries are digested, anthocyanins are quickly metabolized, suggesting that vasculoprotection may be caused by circulating metabolites. This study examined the association between anthocyanin metabolites and their attenuating effect on vascular endothelial inflammation and dysfunction. Several of the metabolite compounds were synthesized for this study, because they are not commercially available. Human aortic endothelial cells (HAECs) were treated in vitro with parent anthocyanins or blueberry metabolites mixture for 6 hours, and then provoked with lipotoxic stress via palmitate. A physiologically relevant dosage was applied in concentrations known to circulate in humans following blueberry consumption. The HAECs were tested for cell viability, monocyte adhesion, and indices of endothelial inflammation. To analyze the effect of metabolites on lipotoxicity-induced vascular dysfunction using isometric tension techniques, the aortae of C57B1/6J mice were incubated with palmitate and blueberry metabolites. Compared with a control, palmitate significantly induced lipotoxic stress ($P < .0001$). Palmitate-induced monocyte binding to HAECs was prevented when treated with blueberry metabolites ($P < .0001$), but not as significantly as with the parent anthocyanins ($P = .099$). Blueberry metabolites were also suggested to decrease inflammatory markers and improve vessel function ($P = .0162$). This study indicates blueberry anthocyanins can be included in a healthful diet for patients with metabolic syndrome, diabetes, and cardiovascular complications as they might im-

prove lipotoxicity-induced endothelial dysfunction. This work was supported by research grants from the University of Utah.

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

Association Between Gestational Diabetes and Cardiovascular Disease

Tobias DK, Stuart JJ, Li S, et al. Association of history of gestational diabetes with long-term cardiovascular disease risk in a large prospective cohort of US women. *JAMA Intern Med.* 2017;177:1735-1742.

Approximately 6% of pregnancies are complicated by gestational diabetes (GD), and there is evidence that the prevalence is increasing. GD has been identified as a risk factor for cardiovascular disease (CVD), but the association has not been evaluated to control for other common risk and lifestyle factors. This study included 89,729 women from the Nurses' Health Study II who reported at least one pregnancy and who were free from CVD and cancer at baseline. Self-reported fatal and nonfatal myocardial infarctions (MIs) and strokes were validated using medical records. Physical activity (calculated in metabolic equivalents), diet quality (as calculated by healthy dietary pattern adherence score), smoking incidence, type 2 diabetes mellitus (T2DM) progression, and body mass index (BMI) were also evaluated. Individuals with GD during pregnancy had a higher incidence (60%, *P*<.001) of total CVD, although correction with a multivariate model (including years since first birth age, menopausal status, current hormone therapy, race/ethnicity, family history of MI or stroke, history of pregnancy hypertension disorders, pre-pregnancy BMI, and parity) and current lifestyle factors (physical activity, diet quality, smoking status, aspirin use, change in weight from pre-pregnancy weight, and alcohol intake) lowered the increased risk to 20% (*P*=.02). GD is associated with increased incidence of cardiovascular disease later in life, but this risk can be significantly decreased by adhering to a healthy lifestyle. This study was funded by the National Institute of Health and Intramural Research Program of Eunice Kennedy Shriver National Institute of Child Health and Human Development.

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

Protein and Antioxidant Effect on Muscle Function and Soreness Following Exercise

Ives SJ, Bloom S, Matias A, et al. Effects of a combined protein and antioxidant supplement on recovery of muscle function and soreness following eccentric exercise. *J Int Soc Sports Nutr.* 2017;14:21.

Short bouts of eccentric muscle contractions (ECC) induce mechanical and metabolic disturbances in muscle, impairing muscle function (MF) and performance and increasing muscle soreness (MS). Both protein (PRO) and antioxidant (AO) supplementation have been shown to improve recovery from eccentric exercise, but their combined effects have not been examined. Therefore, the purpose of this study was to determine the effects of PRO and AO supplementation on muscle fatigue and soreness following eccentric contractions. Sixty college-aged, sedentary males participated in this randomized, single-blind, placebo-controlled, parallel design study examining the effects of the consumption of one of three isocaloric (124 kcal) and isovolumetric supplements—PRO (31 g whey protein-isolate in water), PRO+AO (31 g whey protein-isolate and antioxidants + water), or a carbohydrate (CHO) control (31g sugar + water)—on assessments of peak isokinetic torque (PIKT) and peak isometric torque (PIMT) of the knee extensors of the right leg; perceived muscle soreness (MS), measured via Visual Analog Scale (VAS); and thigh circumference (TC). Measures were obtained prior to 100 maximal ECC extensors of the right thigh and at 0, 1, 2, 6, and 24 hours post-ECC. Supplements were consumed at 0, 6, and 22 hours post-ECC. Both PIKT and PIMT decreased significantly over time (-25%), with no group differences in PIMT. However, PIKT was higher with PRO (+11%) and PRO + AO (+17%) when compared with CHO (P<.001). Muscle soreness increased significantly over time (*P*< .001). However, PRO+AO supplementation resulted in the lowest MS (*P*=.0333). The resulting lower perceived muscle soreness over time in the PRO + AO group compared with the PRO and CHO groups may prove beneficial when repeated exercise bouts are paired with inadequate recovery. Sports dietitians may consider emphasizing protein in combination with antioxidant-rich foods for recovery from eccentric exercise.
This study was funded by a grant from Scott Connelly Foundation to coauthors Paul J. Arciero and Stephen J. Ives.

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

Nitrate Supplementation Improves High-Intensity Intermittent Exercise Performance

Nyakayiru J, Jonvik K, Trommelen J, et al. Beetroot juice supplementation improves high-intensity intermittent type exercise performance in trained soccer players. *Nutrients* 2017;9:314.

Previous studies have shown an ergogenic effect of nitrate supplementation on endurance performance.

However, little is known about the effects of nitrate supplementation on other types of exercise. The purpose of the study was to investigate the effects of 6 days of moderate nitrate supplementation on high-intensity intermittent exercise performance. In this randomized, double-blind crossover study, 32 trained male soccer players ingested either a bolus of 140 ml nitrate-rich (~800 mg nitrate/day) beetroot supplement (BR) or a nitrate-depleted beetroot juice (PLA) at the same time of day for 6 days with the last bolus ingested 3 hours prior to completing repeated 2 x 20 sprints at progressively increasing speed (Yo-Yo IR1 test). Crossover treatment was separated by 8 days. Blood and saliva samples to measure nitrate levels were collected 2.5 hours following the last bolus. Heart rate (HR) was measured during exercise testing. Consumption of beetroot for 6 days increased plasma and salivary nitrate and nitrite concentrations compared with placebo concentrations ($P<.001$). Mean HR was lower in the BR (172 ± 2 bpm) vs. PLA (175 ± 2 bpm; P=.014) during exercise trials. Exercise performance improved 3.4 ± 1.3% compared with PLA (1623 ± 48 vs. 1574 ± 47 m, respectively; $P=.027$). The results of this study indicate that beetroot juice supplementation increases plasma and salivary nitrate and nitrite concentration, which may improve high-intensity intermittent exercise. Dietitians may consider using a beetroot supplement or increasing nitrate-rich food consumption for athletes who participate in high-intensity intermittent sports. This study was financially supported by a grant from the Dutch Technology Foundation STW.

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

SCAN Notables

- **Zachary Grunewald, MS, RDN**, the recipient of a SCAN Graduate Student Research Grant for 2017, presented two posters on preliminary data regarding the role of endothelin-1 on the development of cardiovascular disease-associated obesity at the Experimental Biology Conference held in San Diego in April 2018. Zachary is a doctoral candidate in Exercise Science at the University of Missouri in the Department of Nutrition and Exercise Physiology, and he is nearing completion of his research funded with the assistance of SCAN.

- **Nicole Litwin, MS, RDN** was awarded the SCAN Graduate Student Research Grant for 2018. Nicole is a doctoral candidate in Food Science and Human Nutrition at Colorado State University, where she studies the efficacy of functional foods and bioactive compounds for improving vascular function.

- **Karen Collins, MS, RDN, CDN, FAND** was a speaker at two conferences on cardiovascular health held in September 2018. She presented "Nutrition News in Review: Clearing Through Headline Hype to Focus on Key Messages for Healthy Eating Strategies" at the annual meeting of the American Association of Cardiovascular ad Pulmonary Rehabilitation (AACVPR), and she spoke on "Talking to Your Patients About Trending Diets" at a session on dyslipidemia management held during the 2018 National Lipid Association Clinical Lipid Update.

- A group of SCAN members were invited by the National Athletic Trainers' Association (NATA) to collaborate for the first-ever *NATA News* "Nutrition Issue" published this past July. The project was organized by the SCAN-NATA Alliance Committee, which is led by national liaisons **Jennifer Doane, MS, RD, CSSD, ATC** and **Dana Angelo White, MS, RD, ATC**. Sports dietitians from around the country contributed articles and infographics highlighting evidence-based practice information and recommendations for athletic trainers. Topics included nutrition for injury recovery, hydration, metabolic syndrome, low- energy availability, and nutrient timing.

If you have an accomplishment that you would like to be considered for an upcoming issue of PULSE, please contact Mark Kern, PhD, RD at kern@sdsu.edu.

Of Further Interest

■ Cast Your Vote for SCAN Leaders

Your vote counts! Take an active role in how SCAN is governed by participating in the upcoming election for SCAN leaders. Once again, SCAN will use an electronic ballot. For details on the 2019 Election taking place this winter, check out SCAN's website (www.scandpg.org/nominations/) and watch for eblasts

■ View SCAN's Latest Annual Report Online

Members can find SCAN's Annual Report for fiscal year 2017-2018 posted on the SCAN website (www.scandpg.org/about-us/annual-reports/). The report provides an inside look at SCAN's programs, services, initiatives, and more—giving you important highlights on what SCAN has to offer, and how it is continually working for you.

■ Looking for Past *PULSE* Articles?

If you're doing research or simply want to locate content that appeared in an archived issue of *SCAN'S PULSE*, check out the annual "Index of Topics" posted for each year on SCAN's website. You'll find the issue and page number for each feature article (conveniently listed by practice area), and each item in the "Conference Highlights," "Reviews," and "Research Digest" departments. You can then instantly access the archived issue online. As a member benefit, all PULSE issues and annual indexes are available you for free at www.scandpg.org/nutrition-info/pulse-newsletters.

■ News from Wellness/CV RDNs Subunit

Here is an update from the Wellness/CV subunit:

- **New Webinar on Health Coaching.** Health and wellness coaching is becoming more and more popular and dietitians are uniquely suited to this role. Well-developed coaching skills can positively affect patient outcomes by helping clients work toward positive behavior changes. Our new webinar, *Health & Wellness Coaching for the RDN*, is almost ready and is a great introduction to health and wellness coaching and how it can help you and your clients. Watch for its release this fall at SCAN's e-library at www.scandpg.org/e-library.

- **CV Reimbursement Trends/Efforts.** RoseAnna Holliday, PhD, RDN and Geeta Sikand, MA, RDN, CDE, CLS, FAND, FNLA are SCAN's new reimbursement co-representatives. If you're interested in becoming involved in our efforts to increase awareness of reimbursement issues/topics, please contact Wellness/CV co-director Carol Kirkpatrick at fellcaro@isu.edu.

- **2019 SCAN Symposium Preview.** We hope you'll plan to join us in Phoenix, AZ, April 26-28 for the 2019 SCAN Symposium, which has a wellness-focused theme and emphasizes integrating wellness with sports and cardiovacular nutrition.

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

Below are some highlights from the SD-USA subunit:

- **Athletes and the Arts Partnership.** SCAN now has an official partnership with Athletes and the Arts (an initiative of the American College of Sports Medicine). This ties in with our Expanding the Arena Initiative by promoting opportunities for sports dietitians to work with performing artists. We are looking for volunteers who are interested in developing this partnership. Please visit the SCAN volunteer page at www.scandpg.org/volunteer-opportunities/.

- **New! Changes to the CSSD Exam Window**. Beginning February 11, 2019, the CSSD exam will be administered year-round! Visit the Commission on Dietetic Registration (CDR) website at https://www.cdrnet.org/certifications/board-certification-as-a-specialist-in-sports-dietetics.

- **SCAN Speaking Opportunity.** The SCAN-NATA (National Athletic Trainers' Association) Committee has developed a PowerPoint presentation that highlights the collaborative working relationship of sports RDNs and certified athletic trainers (ATCs). Any SCAN member can apply to offer this presentation at any NATA-approved provider program. SCAN benefits from increased exposure, NATA members benefit by connecting with a local nutrition expert, and you can benefit from potential referrals and an honorarium. Visit https://www.scandpg.org/sports-nutrition/working-with-a-sports-nutritionist/ for more information.

- **I'm a Sports RDN**. As part of the Expanding the Arena Initiative, SCAN wants to profile our greatest asset—our members! Do you work in a unique role or with a specialized niche of athletes as a sports RDN? Please contact Christina Figueroa at christinafig.rd@gmail.com if you would like to share your story.

- **Spring Is Conference Time**. Mark your calendars for the upcoming conferences you won't want to miss—the SCAN Symposium in April; the American College of Sports Medicine (ACSM) Conference, with the "PINES 10 Experts, 10 Questions" session, in May; and the Female Athlete Conference in June. See "Upcoming Events" in this issue for details.

■ 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 all 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 "Research Digest" by volunteering to abstract a recently published study on any of the above practice areas. For details on this opportunity, contact Kary Woodruff, MS, RD, CSSD, co-editor of "Research Digest," at kary.woodruff@health.utah.edu. Become a contributor to PULSE!

■ Manuscripts for *PULSE* Welcome

SCAN'S PULSE welcomes the submission of manuscript 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 relative 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/.

Mark Your Calendars

SCAN Symposium 2019

Navigating the Path to Wellness

April 26-28, 2019
Pointe Hilton Tapatio Cliffs Resort
Phoenix, Arizona

Come to the 35th Annual SCAN Symposium and put yourself on the path to adopting best practices—and integrating them into your wellness, sports nutrition, and cardiovascular health endeavors.

We'll gather at the Pointe Hilton Tapatio Cliffs Resort in Phoenix at the perfect time of the year to enjoy the splendors of the desert.

Attendees will:

- Explore evidence-based approaches and techniques
- Learn practical applications, skills, and knowledge to enhance your work
- Gain new tools to support clients, patients, and athletes
- Obtain superb advice and insights from experts
- Comingle with colleagues and make new friends

For more information and updates, watch for eblasts and visit www.scandpg.org/symposium-2019/.

Upcoming Events

March 21-24, 2019
ACSM's International Health & Fitness Summit & Exposition, Chicago, IL. For information: acsmsummit.org

April 6-9, 2019
Experimental Biology 2019, Orlando, FL. For information: http://experimentalbiology.org/2019/Home.aspx

April 26-28, 2019
Join your colleagues at the 35th Annual SCAN Symposium, Navigating the Path to Wellness, Phoenix, AZ. For more information: www.scandpg.org/symposium-2019/

May 28-June 1, 2019
ACSM Annual Meeting, World Congress on Exercise is Medicine®, and World Congress on the Basic Sciences of Exercise and the Brain, Orlando, FL. For information: www.scandpg.org/symposium-2019/

June 6, 2019
Female Annual Conference, Boston. For information: For information: https://bostonchildrens.cloud-cme.com/Aph.aspx?P=1&EID=910

SCAN'S PULSE

Publication of the Sports,Cardiovascular, and Wellness Nutrition (SCAN) dietetic practice group of the Academy of Nutrition and Dietetics. ISSN: 1528-5707.

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Appropriate announcements are welcome. Deadline for the Spring 2019 issue: Jan. 1, 2019. Deadline for the Summer 2019 issue: April 1, 2019. Manuscripts (original research, review articles, etc.) willl be considered for publication. Guidelines for authors are available at www.scandpg.org/nutrition-info/pulse/ Email manuscript to the Editor-in-Chief; allow up to 6 weeks for a response.

Subscriptions: For individuals not eligible for Academy of Nutrition and Dietetic membership: $50. For institutions: $100. To subscribe: SCAN Office, 800/249-2875

To contact an editor listed above, visit www.scandpg.org. (Click Nutrition Info tab, then "SCAN's PULSE")