The Skinny on Fats: How Dietary Fats Influence Cardiovascular Disease Risk

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Introduction

Poor diets are a key risk factor for cardiovascular disease (CVD), the leading cause of death among adults in developed countries. While there is no doubt that poor diets contribute to CVD, precisely what constitutes a poor diet in terms of cardiovascular risk is still a matter of debate. Dietary fat has long been at the center of this controversy. This paper will explore the latest research regarding types and amounts of dietary fats in our diets, and how they influence our cardiovascular health.

The dietary fats at the center of the debate are saturated fatty acids, monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs). PUFAs are further broken down into two important groups—omega-6 and omega-3 fatty acids. Each of these types of fat, depending on how much is consumed in relation to the other types, has a different effect on blood lipids (such as LDL cholesterol) as well as on other risk factors for cardiovascular disease. Therefore, the amounts and types of fat in the diet influence the risk of developing and dying from cardiovascular disease.

Yet, exactly how dietary fats effect risk and what to do about it are astonishingly difficult questions to answer. After decades of research, there are still many inconsistencies in the data. Even nutrition and health experts have not come to complete consensus around fats in the diet. Arivale’s Translational Science team (www.arivale.com) has examined the research to identify how different types and amounts of dietary...
fats influence serum lipids and risk of CVD. This paper will first summarize the evidence that connects LDL particles with CVD. A brief review of the detrimental impact of saturated fat will follow, concluded by the evidence for replacing saturated fat with other macronutrients and the resulting consequences for CVD.

Evidence that cholesterol is linked to CVD risk

There is extensive evidence that LDL cholesterol and LDL particles are a fundamental determinant of CVD risk and a causal agent in atherosclerosis—the development of plaque within arteries.\(^1\)-\(^6\) Although LDL cholesterol levels do not tell the whole story and can sometimes be misleading, evidence strongly indicates that increased LDL cholesterol is associated with an increased risk of myocardial infarction and CVD-related death.\(^1\) Recently, genetic research has added to the already strong evidence that LDL cholesterol is a causal agent for the initial development as well as the progression of atherosclerotic plaques.\(^2\) For example, genetic studies in individuals with defective LDL-receptor genes reveal an increased CVD mortality risk. Elevated LDL levels for prolonged periods of time further increase this risk.\(^2\) Finally, lowering LDL cholesterol is strongly associated with reduced rates of CVD events.\(^4\)

While LDL cholesterol concentration is a useful tool in predicting CVD risk, conventional testing does not tell the whole story. For example, LDL cholesterol concentrations can often overestimate or underestimate risk.\(^5\) Aside from the cholesterol itself, the LDL particles, inflammation and oxidative stress are now known to be crucial factors in arterial plaque development. Today’s more advanced lipid testing can measure the number, size, and density of cholesterol-containing LDL particles. These advanced lipid tests are more accurate at predicting CVD risk.\(^6\) A comprehensive array of advanced lipid measurements and other biomarkers that relate to inflammation, oxidative stress and nutrient deficiencies allow for more personalized interventions.

Saturated Fat

Based on the media and popular culture’s fixation with the saturated fat “controversy,” one could easily assume that reducing saturated fat is no longer important for heart health. Indeed, the “butter/bacon is back” message is more ubiquitous than ever, and some believe that saturated fat, and the higher cholesterol levels that accompany it, are of no consequence in terms of CVD risk.

This recent movement fueled by both media and some health professionals is compelling at first glance. A few recently published studies conclude that there is no association between intake of saturated fat and CVD risk.\(^7,8\) One of these studies was a meta-analysis of observational studies reporting associations of saturated fat with all-cause mortality, CVD mortality, incidence of coronary heart disease (CHD), and stroke.\(^7\) The researchers concluded that saturated fats are not associated with any of these outcomes. Another popular study was The Minnesota Coronary Experiment (MCE), a randomized controlled trial conducted in 1968 with 9,423 men and women. Researchers recovered lost data from the MCE study, and findings were published in 2016 concluding that replacing saturated fat in the diet with PUFAs had no impact on CVD mortality, even though it did reduce cholesterol.\(^8\) Without taking the entire body of research into account, some popular health experts cite these and other similar studies to promote the idea that reducing saturated fat does not lower CVD risk.

While these studies and arguments may seem compelling, they are not representative of the vast majority of research linking saturated fat intake with increased CVD risk. They have also been criticized for containing major flaws.\(^9\) Critiques of the meta-analysis cited above show that the researchers based their conclusions on selected data rather than the entire set of data.\(^3\) Some of the ignored data showed a statistically significant relationship between saturated fat intake and all-cause mortality, as well as with all of the CVD-related outcomes. The ignored data also showed that replacing dietary saturated fat with high quality carbohydrates such as fruits, vegetables, and whole grains reduced the risk of CVD. When referring to the MCE trial, many experts have cited major flaws including a significant drop in subjects (75%) due to early discharge, and fake foods created for the trial that were artificially high in omega 6s, low in omega 3s, and likely containing trans fats.\(^10\)

While it is true that some saturated fat can certainly be a part of a healthy diet and that cholesterol is needed by the body for a number of critical biological functions, the fact remains that the vast majority of scientific evidence supports reducing saturated fat.\(^11-16\) One of the largest, most detailed, and powerful studies yet to examine the associations of specific dietary fats with total and cause-specific mortality, just published in JAMA Internal Medicine, found that when compared with the same number of calories from carbohydrate, every 5% increase in saturated fat intake was associated with an 8% higher risk of overall mortality.\(^17\) Wang et al also found that replacing 5% of energy from saturated fats with equivalent energy from PUFAs and MUFAs was associated with estimated reductions in total mortality of 27% and 13%, respectively. Lastly,
a meta-analysis that reviewed 15 randomized controlled trials (RCTs) of over 59,000 participants concluded that reducing dietary saturated fat reduced the risk of cardiovascular events by 17%. Clearly, the specific nutrients that replace saturated fat are important.

The next sections of this paper will explore the research around replacing dietary saturated fats with other nutrients and the associated cardiovascular outcomes.

Replacing saturated fat with carbohydrates

Replacing saturated fat with carbohydrates can be beneficial for reducing CVD risk, but this largely depends on the type and quality of carbohydrate. As mentioned above, Wang et al found that when compared with overall carbohydrates, every 5% increase of total calories from saturated fat was associated with an 8% higher risk of overall mortality. However, intake of saturated fat, when substituted for total carbohydrates, was not significantly associated with CVD mortality. The researchers expected this lack of association with CVD because the major sources of carbohydrates in a typical Western diet are highly processed foods with large amounts of refined starch and sugar, providing a high glycemic load that can increase CVD risk independent of saturated fat.

Overall, the body of research indicates that when saturated fats are replaced with refined carbohydrates, such as added sugars, CVD risk is unchanged, and some CVD risk factors, such as triglycerides and the number of small, dense LDL particles, actually increase. However, the consumption of fiber-rich, low glycemic index carbohydrates lowers LDL cholesterol, has no detrimental effects on triglycerides, and is associated with a decreased risk of heart disease.

Harvard researchers followed 84,628 women and 42,908 men for 24 to 30 years and found that higher intake of carbohydrates from whole grains was significantly associated with a 10% lower risk of coronary heart disease, whereas carbohydrates from refined carbohydrates/added sugars were significantly associated with a 10% increased risk. They showed that replacing 5% of energy (calorie) intake from saturated fats with equivalent energy intake from carbohydrates from whole grains was associated with a 9% lower risk of coronary heart disease.

Unfortunately, the researchers did not examine other healthy carbohydrate sources besides whole grains. Fruits, vegetables and legumes may be as beneficial as whole grains at reducing heart disease risk. Other studies have shown that carbohydrates with a low glycemic index and/or glycemic load are associated with reduced total and LDL cholesterol as well as lower risks of myocardial infarction and coronary heart disease. This suggests that in addition to replacing saturated fat with whole grain carbohydrates, other low glycemic carbohydrates, including many vegetables, fruits, and legumes, may also be beneficial replacements.

Replacing saturated fat with omega-6 polyunsaturated fatty acids

The predominant unsaturated fats in the western diet are the omega-6 polyunsaturated fatty acids (PUFA). Linoleic acid, obtained primarily from vegetable oils, nuts, and seeds, is the most abundant omega-6 PUFA. Current US dietary guidelines and the American Heart Association recommend higher intake of omega-6 PUFA to reduce the risk of coronary heart disease. These recommendations are based on the preponderance of evidence showing that the omega-6 PUFAs, particularly when they replace saturated fat, reduce the risk of cardiac events and deaths due to coronary heart disease and improve a number of CVD risk factors, such as blood pressure and LDL cholesterol. Wang et al found that among specific PUFAs, intake of linoleic acid was most strongly related to a lower risk of CVD mortality. Compared to those in the lowest quintile of linoleic acid intake, those in the highest quintile had a 22% reduced risk of CVD mortality. Similar results were found in the Cardiovascular Health Study, in which researchers followed 4707 participants for 18 years. Study participants with higher blood levels of linoleic acid had a decreased risk of death from coronary heart disease or any other cause.

Despite this evidence, the recommendation to replace saturated fat with omega-6 PUFA remains contentious. Not all observational studies and randomized trials have shown benefits, and concerns have been raised about higher omega-6 fatty acid consumption being harmful for heart health because of potential pro-inflammatory effects. Biochemically, linoleic acid can be elongated to arachidonic acid and subsequently synthesized to a variety of pro-inflammatory compounds, which could theoretically increase CVD risk. However, this speculation is not supported by randomized controlled studies, in which dietary intake of linoleic was not found to increase inflammatory markers including C-reactive protein or tumor necrosis factor-α. Another concern has to do with the fact that the omega-6s and omega-3s share metabolic pathways and can potentially compete with each other, causing the omega-6s to interfere with potential cardiovascular benefits of the omega-3s. However, this has not been shown to occur; rather, studies have found that the combination of both types of fatty acids...
acids is associated with the lowest levels of inflammation and the lowest risk of CVD.27,31

Overall, despite the concerns, the majority of the research clearly points to the benefits of omega-6 fatty acids for cardiovascular health.

Omega-3 Fatty Polyunsaturated Fatty Acids

The omega-3 PUFAs, α-linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), are heart-healthy and should be consumed regularly for optimal cardiovascular health. EPA and DHA are primarily found in fatty fish, such as salmon, mackerel, herring, and albacore tuna, whereas ALA is primarily found in plant-based foods such as olive, canola, walnut, and flaxseed oils, as well as in walnuts, flaxseeds, and chia seeds.

The omega-3 fatty acids play an important role in preventing and treating CVD. In terms of prevention, studies show that consumption of omega-3 fatty acids in the form of fish or fish oil is associated with a decreased risk for development of coronary heart disease and death due to CVD (cardiac mortality).32,33 Higher levels of these fats circulating in the bloodstream are linked to a lower risk of dying from CVD or any other cause.34 Although there are some inconsistencies, studies show that omega-3 fatty acids have beneficial effects on many CVD-related outcomes, including heart attacks and sudden cardiac death.34-38 Omega-3 fatty acids also improve important CVD-related measurements such as fasting triglyceride levels, blood pressure, heart rate, inflammation, and blood vessel (endothelial) function.39,40

Despite the many reported benefits, however, more recent trials have raised questions about the role of omega-3 fatty acids, and in particular of fish oil (EPA/DHA) supplementation, in the prevention and treatment of CVD, and there is renewed debate among experts around recommending omega-3s.40-42 The conflicting evidence is exemplified in two recent meta-analyses which reviewed many of the same studies but come to opposite conclusions regarding the effects of EPA and DHA on CVD.40,43 Some of the more recent data showing limited effects may be attributed to the fact that an increasing number of study participants are on more aggressive pharmaceutical treatment, or to the fact that a number of the studies are statistically underpowered, differ widely in EPA/DHA dosage, participants’ disease states, and time of follow-up.41,42

While this has undoubtedly contributed to confusion among medical professionals and the public, the current scientific literature provides strong concordant evidence that omega-3 fatty acids reduce risk of death from CVD. The strength of the data has compelled national and international guidelines to collectively recommend that healthy adults consume at least 250 mg per day of long-chain omega-3 fatty acids or at least 2 servings of oily fish per week to maintain cardiovascular health, with many organizations recommending higher amounts for those at greater risk of CVD.44-46

Finally, a fascinating and rapidly emerging area of research is now focused on the complex interaction between different nutrients, including omega-3 fatty acids, with relatively common mutations in genes involved in omega-3 fatty acid modulation. Results from research into these gene-nutrient interactions strongly suggests that the beneficial effects of omega-3 fatty acids are not dependent only on the intake of these essential fats, but on their interactions with genes.47

Replacing saturated fat with monounsaturated fat

Replacing saturated fats in the diet with MUFAs such as those found in olive oil, is associated with improvements in many cardiovascular disease-related risk factors, such as cholesterol and blood pressure, and a reduced risk of cardiovascular disease. Studies show that MUFAs are comparable to PUFAs (both linoleic and α-linolenic fatty acids) in terms of having a favorable effect on cholesterol as compared to saturated fat.48,49
Replacing saturated fat with MUFAs decreases total cholesterol, LDL-cholesterol, total-to-HDL cholesterol ratio, and LDL particle number.49 Individuals who fall into the top versus bottom third of MUFA and olive oil intake have a 12% reduction in death from cardiovascular disease, a 9% reduction in cardiovascular events, such as heart attack, a 17% reduction in risk of stroke, and an 11% overall risk reduction in death from all causes.39 Replacing just 5% of energy from saturated fat with equivalent energy from MUFAs is associated with a 13% estimated reduction in total mortality.17

Extra virgin olive oil, especially when consumed as part of a Mediterranean diet low in saturated fat, is associated with significantly reduced risks of CVD and resulting deaths in individuals at high cardiovascular risk.50 Part of what makes extra virgin olive oil such a particularly healthy source of MUFAs is the fact that it contains certain heart-healthy phytonutrients (“polyphenols”) in addition to the MUFAs themselves. These polyphenols help lower total and small LDL particle numbers, lower LDL oxidation, decrease oxidative stress, and enhance HDL function.51-53

Besides extra virgin olive oil, other healthy dietary sources of MUFAs include avocados and nuts such as pistachios. Replacing
saturated fats with these MUFA-rich foods is a scientifically validated way to improve heart health.54,55

Mediterranean Diet

The Mediterranean Diet, with its focus on fish, monounsaturated fats from olive oil, fruits, vegetables, whole grains, legumes, and nuts is arguably the best studied and most evidence-based diet to prevent CVD. This diet is known to reduce primary cardiovascular disease outcomes such as death and events such as stroke and myocardial infarction, in addition to improving many important biomarkers related to CVD, including lipids and markers of inflammation.56

In terms of preventing cardiovascular events, results from the Prevención con Dieta Mediterránea (PREDIMED) study, published in the New England Journal of Medicine in 2013, showed that adhering to an energy-unrestricted Mediterranean diet supplemented with extra-virgin olive oil or mixed nuts for 4.8 years reduces the incidence of myocardial infarction, stroke and cardiovascular death by 30% compared with a control diet.57 A follow-up study that classified participants to their level of adherence to the Mediterranean diet indicated that those who had the highest adherence to a Mediterranean-type diet, had a 48% lower risk of CVD compared to those who did not follow this type of dietary pattern.58

Numerous meta-analyses conducted over the last decade have shown that adherence to the Mediterranean diet has reduced overall mortality and reduced CVD incidence and mortality.59,60 Not only does the Mediterranean diet reduce the risk of CVD by lowering LDL cholesterol concentrations, it also lowers LDL particle number61 and oxidized LDL concentrations,62 and increases LDL size.63 In the PREDIMED study, participants who supplemented their diet with nuts showed significant reductions from baseline small LDL as well as decreased LDL particle number, both of which are known to reduce CVD risk.63

Trans fats

Industrially produced trans fats, produced through the hydrogenation of vegetable oils, are generally found in processed baked goods and snack foods such as muffins, pies, and cakes. These can show up as “partially or fully hydrogenated vegetable oils” on ingredient labels. A 2% increase in transfat consumption is associated with a 23% increase in the incidence of CVD, and a 16% increase of premature death.17,64 Consuming industrial trans fats has been found to increase the risk of coronary heart disease events, such as heart attacks, by 30%, and to increase the risk of death from coronary heart disease by 18%.7 One way trans fats increase cardiovascular disease risk is by adversely affecting serum lipids, including not only traditional lipid measurements such as LDL and total cholesterol, but also more advanced lipid measurements such as LDL particle number.65 LDL particles are directly involved in the formation of atherosclerotic plaques and, as discussed previously, LDL particle number is superior to traditional lipid markers in terms of predicting adverse events related to coronary heart disease. The current dietary guidelines recommend that trans fats should be limited to less than 1% of energy or as low as possible.

Conclusion

Dietary fat—what types and how much—is an important consideration for cardiovascular health. While controversies surrounding dietary fat will continue as the research advances, it is currently possible to come to some evidence-based conclusions. Lowering the amount of saturated fat consumed in the diet is beneficial for cardiovascular health. However, what replaces the saturated fat is crucial. Replacing saturated fat with PUFAs (both omega-6 and omega-3) and MUFA s reduce the risk of cardiovascular disease. Omega-3 fatty acids are typically insufficient in western diets, and should be emphasized to optimize cardiovascular health. Replacing saturated fat with whole, unrefined carbohydrates, including whole grains, is also beneficial for reducing heart disease risk, whereas replacing saturated fat with added sugars and refined carbohydrates is deleterious and these carbohydrates should be avoided.

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**Additional Resource**

Physical Therapy: A Multidisciplinary Approach to Pelvic Floor Dysfunction

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Physical therapists graduate from master’s or doctoral programs with extensive knowledge of the neuromusculoskeletal systems. This training can be furthered with continuing education courses to become certified or competent in treating areas of specialty practice within the scope of each state’s physical therapist practice act. Pelvic floor physical therapists (pelvic floor PTs) are physical therapists by background, and specialize in the conservative treatment of bowel/bladder dysfunction, pelvic pain, abdominal pain, constipation, pregnancy-related changes to the musculoskeletal system, and sexual dysfunction.

Pelvic floor PTs assess and provide interventions for the musculoskeletal limitations of the spine, pelvis, hips, and pelvic floor musculature that may be contributing to any of the above dysfunctions. The musculature of the spine, pelvis, and hips provide support to the pelvic floor muscles (PFM). The PFMs are those that line the bottom of the bony ring that forms the pelvis. These muscles have four functions: support the contents of the abdomen (organs, soft tissue); act as sphincters around the urethra and anus, making them responsible for continence; provide stabilization for posture; and, as muscles of sexual function, obtain/maintain erection and orgasm. Patients with dysfunction of these muscles may present with constipation, urinary/fecal incontinence, urinary/fecal urgency, urinary/fecal frequency, pelvic pain, abdominal pain, or pain with intercourse.1-3

Urinary incontinence can occur because of PFM weakness. Age, complications related to child birth, surgery related to cancer treatment, or other pelvic trauma can contribute to PFM weakness. As tissue loses elasticity with age or has been stretched during child birth, women can experience incontinence. Some experience this as stress incontinence, meaning that when the PFM are stressed, they are unable to keep their contraction to avoid loss of urine. This can happen with coughing, jumping, running, sneezing, or any sudden movement that does not allow the PFMs to sustain a contraction. Urge incontinence can occur with or without stress incontinence and can happen when there is a strong urge to urinate, however,
the PFM are unable to sustain a contraction long enough for the individual to make it to the toilet on time. PFM are responsible for urinary continence as well as fecal continence. Because the muscle can be weak in both of these cases, fecal incontinence can also be a result. It is very important to note that pain often disguises itself as weakness. Pain can cause the PFM to be overused. Pelvic floor PTs refer to this as a high tone pelvic floor, or hypertonicity. In this case, the muscles are not weak, they are fatigued from overuse, and subject to pain. This can cause pain during gynecological exams, painful intercourse, or referred pain affecting the abdomen or lower back. High tone PFM can also contribute to difficulty having bowel movements. Pelvic pain can also lead to a decline in the ability to coordinate contraction and relaxation of the pelvic floor muscles.1,5

As the lower back and hips are closely related to the PFM, the strength of the hips, abdomen, and back muscles are important for success in treatment. Tight musculature in these areas can also provide a barrier to improvement and is addressed during treatment. If the pelvic floor muscles are weak and have not responded to strength training, abdominal and hip muscles can be strengthened and incorporated into techniques to maintain continence. Having weakness in these groups of muscles can present as difficulty in maintaining posture or stabilizing the spine/pelvis during activity. In the presence of weakness of the postural muscles listed above, the PFM must work harder to assist with posture. This can cause fatigue and tightness of the PFM which can lead to some of the other dysfunctions discussed.1,3,5

A multidisciplinary approach to pelvic floor dysfunction is essential for this population. Having a medical team to perform bowel/bladder studies can provide important insight into the ability to decrease symptoms and make realistic goals for treatment.4,5 Mental health is also important for this population, especially those with pelvic pain. Being able to refer to mental health professionals that are familiar with pelvic pain can assist with helping patients through the process. Therapists often refer to RDNs in order to further establish a diet that works best for individual diagnoses. This is the case with many people receiving treatment for gynecological, genitourinary, and colorectal cancers. These diseases carry with them the likelihood of radiation to the pelvis, and/or abdomen. Along with causing bowel/bladder changes and colitis, radiation causes fibrosis of the soft tissue and fascia within the radiation field.6 Pelvic floor PTs are in the unique position of being able to address these types of musculoskeletal limitations that can cause stenosis, decreased transit time, inability to empty, or a list of other impairments related to treatment for cancer.7,8,9 Pelvic floor PTs provide basic nutrition information and assist with diet changes. Recommendations are made for fluid intake, fiber intake, avoidance of foods and beverages that cause bladder irritation, and how to manage intolerance to certain ingredients. However, many patients would benefit from a more in-depth diet analysis and evaluation from an RDN that reaches beyond the scope and knowledge of the pelvic floor PT. The marriage between RDNs and pelvic floor PTs is one that can help each patient receive a comprehensive course of care to not only address functional limitations of the body’s soft tissue, but uncover nutritional deficits that could potentially improve patient outcomes.

It becomes readily apparent when working with this population that the decreases in quality of life can be severe and detrimental. Many women are under the impression that incontinence is part of aging or natural following childbirth, but this is not the case and should not be considered normal.1,3 Patients with incontinence can become fearful of social situations and therefore avoid them. The use of incontinence products are not only a financial burden, but cause people to become self-conscious of clothing choices. Patients with pelvic pain may stop going for annual gynecological exams because they cannot tolerate speculum insertion, they stop participating in sexual activity because it is too painful and no longer enjoyable, and pain can become worse without treatment and perpetuate the pain cycle.2 For many people with pelvic floor dysfunction, symptoms have been interrupting daily life for years. While symptoms may not completely resolve, pelvic floor PTs are able to utilize treatment techniques in conjunction with patient education to assist patients in self-managing their symptoms.7-10 When people are able to understand the causes/triggers of pelvic floor dysfunction, they can better manage it independently. The goal for physical therapy is to ensure safe, well-planned self-management of what is sometimes a chronic condition. For those with dysfunction resulting from cancer treatment, the pelvic floor PT provides a transition from the treatment phase into survivorship.11 Patients often report that as follow up appointments become few and far between, they feel a sense of “now what?” As cancer patients move into the role of cancer survivors, it is helpful to have a transition into the ‘new normal,’ and to be able to understand what has changed with their bodies following cancer treatment.

In order to be seen by a pelvic floor PT, a prescription from MD/PA/CRNP requesting physical therapy evaluation and treatment for pelvic floor dysfunction is required. Specific disorders/dysfunctions can be
listed on prescriptions, but are not necessary. Precautions following surgery or any modalities to be avoided should be listed on the prescription. Pelvic floor PT is considered a specialty area of practice, and patients are typically able to attend a clinic that is out of network if the capitated site for PT does not offer this service. The American Physical Therapy Association (APTA) has a section on Women’s Health that is helpful in providing information on the role of the pelvic floor therapist. There is a directory on the section’s website for locating a pelvic floor PT at: www.womenshealthapta.org.

References

Genetics of Celiac Disease

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Like so many diseases encountered by RDNs, celiac disease (CD) does not have a single cause, but many contributing factors that lead to disease progression. Genetic predisposition is a significant factor. Nearly all individuals with CD have the human leukocyte antigen DQ2 or DQ8. However, not all individuals with these polymorphisms go on to develop CD. Over 90% of people with CD have the DQ2 or DQ8 antigens, but 40% of people without the disease also have these antigens.

Studies have shown that these HLA genes are highly significant in the development of CD, but they are not the whole story. HLA-DQ2 and HLA-DQ8 do not have a high degree of affinity for native gluten, but for modified gluten peptides that are created by an enzyme released following tissue damage. However, due to the often lengthy and invasive procedure of diagnosing CD, as well as the large number of people who may go undiagnosed (usually due to a lack of the classic gastrointestinal symptoms), this genetic test may be highly useful in the clinical setting.

Diagnosis

CD is an autoimmune disorder where the immune system has an aberrant sensitivity to gluten, a protein found in wheat, barley, and rye. As a result, the body attacks its own tissues, leading to a number of problems, but most significantly results in villous atrophy and nutrient malabsorption. The overactive immune response related to CD can increase the risk of a wide variety of diseases, including many types of cancer, autoimmune diseases, diseases of inflammatory origin, as well as kidney failure, liver damage, and osteoporosis. Strict lifelong gluten avoidance can reduce these risk factors down to near normal levels. Unfortunately, many individuals with CD do not exhibit any of the classic gastrointestinal symptoms and do not know they have the disease.

Because diagnosing CD can be quite complicated and invasive, there is great interest in genetic testing.

Genetic Markers

Polymorphisms in human leukocyte antigen genes are currently the only clinically relevant genetic markers of CD. Following that, it is HLA-DQ2 and HLA-DQ8 that are considered the markers of genetic predisposition for CD. Several other genetic markers have been associated with CD including chemokines, cytokines and their receptors, cell adhesion molecules, as well as T cell and B cell activators. However, these genes are not currently considered in calculating CD risk. Only HLA polymorphisms are considered clinically relevant. In particular, the DQA1*0501 and DQB1*0201 allele combination, encoding the DQ21*0501-DQB1*02 (DQ2) heterodimer, and to a lesser extent the DQA1*0301 and DQB1*0302 combination that encodes the DQ21*0301-DQB1*0302 (DQ8) heterodimer are strongly associated with the development of CD.

The pathway of gluten-induced immune response is not yet fully elucidated. However, understanding has greatly increased due to genome-wide association studies. The genes identified with CD development shed light on the pathogenesis of the disease, as well as the immunologic pathways involved.

Immunologic Model of Celiac Disease

Genetic and immunologic data suggest that CD is primarily a T-cell mediated immune disorder where CD4+ T cells recognize gluten peptides, in which MHC (HLA) class II molecules play a significant role. Human leukocyte antigen class I and class II genes code for cell surface markers involved in antigen presentation and self-recognition by the immune system. HLA molecules bind to antigens and present them to T cells. Class I includes HLA-A, HLA-B, and HLA-C. Class II includes HLA-DP, HLA-DQ, and HLA-DR. HLA class I molecules are recognized by CD8+ T cells, which activate the cytotoxic response, while HLA class II molecules are recognized by CD4+ T cells that trigger the humoral response.

The gene dosage of HLA-DQ2 and DQ8 haplotypes are related to the severity of CD. Individuals who are homozygous for HLA-DQ2.5 are at a fivefold increased risk for CD development compared to individuals who are
heterozygous for HLA-DQ2.5.1 Patients with refractory CD, those who do not respond to a gluten-free diet, are more likely to be homozygous for the DR3-DQ2 haplotype, which produces the HLA-DQ2.5 heterodimer. Additionally, individuals with refractory CD, which involves elevated levels of intraepithelial lymphocytes, are at high risk for developing lymphoma.2 HLA-DQ2 or DQ8 molecules have a stronger binding affinity for deaminated gluten over native gluten, which is not negatively charged. Both HLA-DQ2 and HLA-DQ8 molecules have positively charged regions that preferentially bind to negatively charged peptides. Transglutaminase 2 (TG2) facilitates the gluten deamination reaction.3

TG2 has been shown to modify gluten molecules such that they will bind preferentially to the HLA-DQ2 or DQ8 molecules, thus stimulating an immune response. However, TG2 is primarily located intracellularly in its inactive form, and activated after release due to tissue damage. Therefore, there must be a triggering event involving tissue damage to release TG2, which deaminates gluten. The deaminated, positively charged gluten binds to the negatively charged pocket in the HLA-DQ2 or DQ8 molecules, leading to an inflammatory response and thus to CD.4

**Take Home Message**

CD does not follow simple inheritance patterns. It is the result of multiple gene and environmental factors. However, because a large number of individuals are thought to have undiagnosed CD, which untreated can have significant negative consequences, the genetic test for CD-related genes can be clinically useful. There is a very strong association between CD and the HLA-DQ2, and to a lesser extent HLA-DQ8 genes. Over 90% of individuals with CD are DQ2+ or DQ8+.2 These two molecules have a strong affinity to modified gluten peptides, which are produced by an enzyme released by tissue damage.

While the HLA genes and the immunologic pathway leading to CD progression are of great interest to researchers and clinicians, the road to disease is likely even more winding than it appears. Increasing use of genetic testing, genome-wide studies, and bioinformatics are likely to yield better understanding, and perhaps improved clinical outcomes for this disease as well as many others in the coming years.

**References**


Gluten-Free Certification: A Food Manufacturer’s Perspective

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Abstract

This article will examine the gluten-free certification process from a food manufacturer’s perspective. Differentiation will be made between the process of gluten-free certification for a food product versus a facility. Additionally, practical recommendations will be made for RDNs counseling patients who present with either celiac disease (CD) or non-celiac gluten sensitivity (NCGS).

Introduction

According to the Celiac Disease Foundation, CD is estimated to affect 1 in 100 people worldwide and more than 3 million in the United States alone. Of these, 2.5 million Americans are undiagnosed and at risk for long-term health complications. Non-celiac gluten sensitivity (NCGS) is now said to affect as many as 18 million Americans, according to the National Foundation for Celiac Awareness. There are more than 200 symptoms associated with celiac disease and NCGS which makes them difficult to diagnose, however testing is suggested. With such staggering figures, RDNs are poised to play a critical role in counseling those with celiac or NCGS.

Food manufacturers have taken note of the rising incidence and, in combination with consumer interest, have supported the rise of “gluten-free” as one of the fastest-growing grocery trends over the past decade. At the time the first products were entering the market, there was no federal regulation or even a single definition for gluten-free. In 2013, the US Food and Drug Administration (FDA) came out with official gluten-free guidelines for food manufacturers.

This article provides a look at the gluten-free certification process from a food manufacturer’s perspective, addressing gluten-free certification, the certification process, and options for manufacturer compliance.

Gluten Considerations from a Food Manufacturer’s Lens

Labeling

The core of the FDA’s 2013 gluten-free regulation asserts that any foods labeled as “gluten-free” must contain less than 20 ppm. The agency cites that some celiac disease research suggests that most individuals with celiac disease can tolerate variable trace amounts and concentrations of gluten in foods, at levels less than 20 ppm gluten, without causing adverse health effects. Any food product bearing a gluten-free claim labeled on or after August 5, 2014, must meet the requirements of the agency’s labeling rule, which defines a gluten-free food as either inherently gluten-free or does not contain an ingredient that is:

1) A gluten-containing grain (ex. wheat, spelt, barley, rye);
2) Derived from a gluten-containing grain that has not been processed to remove gluten (ex. wheat flour); or
3) Derived from a gluten-containing grain that has been processed to remove gluten (e.g., wheat starch), if the use of that ingredient results in the presence of 20 ppm or more gluten in the food.

An important element of the regulation is that the FDA has defined the term “gluten-free” for voluntary use in food labeling. Meaning, it is up to the food manufacturer to ensure their products contain less than 20 ppm gluten and the FDA will only take action if a food has been shown to have more than the threshold after being manufactured.

Testing and Certification

How do manufacturers ensure their foods comply with the FDA regulation? Essentially, it is up to the manufacturer. Below are the five methods most commonly employed:

1) Conduct in-house gluten testing
2) Employ a third-party laboratory to conduct gluten testing
3) Request certificates of gluten analysis from ingredient suppliers
4) Follow Good Manufacturing Practices (GMPs)
5) Seek third-party gluten-free certification

In-House Testing

Whether a manufacturer is third-party certified or elects to market their products as gluten-free, in-house testing is imperative to verify that products are, in fact, gluten-free. Table 1 below illustrates the many available tests for in-house detection of gluten.
ELISA$^3$ testing is reliable and is used to ensure consistent gluten-free status in house. ELISA has a robust, portable swab kit that tests down to 10 ppm. It can be implemented and is most useful at the beginning, middle and end of each batch. Its portability, reliability and ease of use make it an excellent option for high-quality gluten-free protocols. Other types of tests include polymerase chain reaction (PCR), which detects DNA rather than a protein; and adenosine triphosphate (ATP) swab tests for assessing cleanliness of equipment surfaces.

**Employment of an Outside Laboratory**

Sending samples to an external lab is a way to verify the absence of gluten, but is not practical or affordable for ongoing compliance. As one can imagine, it creates an added step in the production process and added costs for third-party lab testing. Therefore, this method is instead employed after-the-fact to test products that are suspected of contamination.

**Supplier Certification**

A third method for ensuring a product is gluten-free is to request gluten-free certification analysis from ingredient suppliers. Obtaining supplier documentation as proof of a product’s gluten-free status saves a company the expense of testing on its own. Testing from suppliers that may have a cross-contamination issue, such as with gluten-free oats, should
be required. However, from suppliers that provide ingredients where the risk is minimal, such as chocolate, fruit and nuts, supplier certification may not be necessary.

**Follow Good Manufacturing Practices**

Every single bar cannot be tested, therefore, having a quality assurance program in place is just as important as periodic testing. GMPs are a set of quality control and production procedures that minimize risk of allergen or foreign object contamination and include protocols from ingredient sourcing to employee training, cleaning, cross-contact controls, management and testing. Manufacturers can refer to the Safe Quality Food (SQF) Code, Edition 7.2 that outlines extensive safety, cleaning and good manufacturing protocols for food manufacturers.

**Third-Party Certification**

To ensure products are dependably gluten-free, obtaining third-party program certification is the best option. All certifying agencies require in-house product testing and audit plants to make sure they employ comprehensive Quality Assurance protocols for sequestering allergenic ingredients and cleaning machinery. In other words, methods 1-3 above are effective, but susceptible to human error and it is only through a third-party certification program that all the critical quality assurance methods are brought together to effect the lowest possible risk.

Food manufacturers may include the logo of a gluten-free certification program on their labels, provided that its use is truthful. The FDA notes that it does not endorse, accredit or recommend any particular third-party gluten-free certification program over another; the choice is up to the manufacturer. Major players in third-party certification include The Gluten-Free Certification Organization (GFCO), the Gluten-Free Certification Program (GFCP) and NSF International (NSF). All three organizations employ a comprehensive gluten-free compliance audit but differ slightly in their requirements. Importantly, they require that certified products contain 10 ppm or less gluten (and similar proteins from rye, barley and hybrids of these grains), and audit a plant to ensure strict Good Manufacturing Practices (GMPs) are followed. Certification by one of the approved third-party certification organizations is the gold standard for ensuring that products meet FDA standards of less than 20 ppm gluten.

**Considerations for the Practitioner**

Not everyone will enter the gluten-free food manufacturing industry. However, it is important for RDNS and consumers to be educated about the product label for the protection of their clients, themselves, or their family members.

**Label Nuances**

Certain food products may indicate their gluten-free certification via use of a symbol alone. Others may claim they are “made with no gluten-containing ingredients” or “not made with gluten-containing ingredients.” Unless the label of such a food includes a “gluten-free” claim, consumers should not assume that the food meets the FDA “gluten-free” requirement.

**Understanding Oats**

While oats in their natural state are gluten-free, they are often processed in facilities that process wheat, thus leading to cross-contamination. Any oats labeled gluten-free must adhere to the FDA's regulations and thus contain less than 20 ppm gluten. In addition to suggesting naturally gluten-free foods and products, RDNs can empower clients with knowledge of certification and labeling rules. Further, understanding where to turn for gluten-free starches and flours also takes the mystery and anxiety out of eating gluten-free. Common gluten-free starches include:

- Root vegetables such as potatoes, yams, sweet potatoes, turnips
- Squash: acorn, butternut, delicata, spaghetti
- Rice: preferably brown or wild for the added fiber, minerals and B vitamins
- Corn, corn tortillas and polenta
- Gluten-free grains such as quinoa, millet, amaranth, buckwheat

Lastly, as an RDN, having a thorough understanding of the gluten-free certification process can help provide guidance to clients and patients. And if for those with the entrepreneurial spirit who desire to bring a gluten-free food to market, knowing labeling, certification, testing and compliance options are essential.

**References**


NEW! Integrative and Functional Nutrition Online Certificate of Training Program

Develop opportunities in this evolving area and earn CPEUs online with this brand-new program. The Academy’s Center for Lifelong Learning, planned with the Dietitians in Integrative and Functional Medicine DPG, are offering a new program to prepare RDNs to apply the integrative and functional medicine constructs in practice. Topics such as digestive health, detoxification, inflammation and more will be covered. Don’t miss out on your chance to become an expert in this fast-growing field!

The Level 2 program consists of five separate modules that build on each other:

• Module 1: Introduction to Integrative and Functional Nutrition
• Module 2: Digestive Health
• Module 3: Detoxification
• Module 4: Inflammation
• Module 5: Dietary Supplements

To receive the certificate associated with this training, all five modules and related tests must first be completed and a final post-test in Module 5 must be passed with a minimum of an 80 percent score. The Certificate of Training can then be downloaded and printed from the site.

Academy members enjoy a reduced rate of $24 for each module or may complete all five modules and earn 8.0 hours of CPEUs for $96.

Learn more and earn CPEUs at: http://www.eatrightstore.org/products/cpe-opportunities/certificates-of-training.
suggested consuming a low-

Two highly active threads included topics on tinnitus and low oxalate diets. Several users suggested consuming a low-

In our commitment to being fair and unbiased, discussions regarding individuals or organizations, as well as certificate programs, testing, and nutrient analysis programs can be found on the electronic mailing list under: https://groups.yahoo.com/neo/groups/DIFM_Listserv/info.

Corporate Sponsorship and Nutrition Research

There is a fair reason to be skeptical about nutrition research studies when they are funded by large food industries. Several University of California San Francisco (UCSF) investigators, Cristin E. Kearns, DDS, MBA, Laura A. Schmidt, PhD, MSW, MPH, and Stanton Glantz, PhD, conducted a retrospective examination of the relationships between the sugar industry and certain nutrition scientists, going back as early as the 1960s. 1 Hundreds of documents were analyzed while investigating the influence of the Sugar Research Foundation (SRF) on research conducted at Harvard’s School of Public Health on sugars, fats, and coronary heart disease (CHD) in 1967. UCSF investigators found that the three nutrition professors publishing the review that shifted the blame of CHD from sugar consumption to fat and cholesterol, were paid over $48,000 in today’s dollars.

Another timely article, published by the same authors, Kerns, Schmidt, and Glantz 2 reviewed the sugar industry’s role in downplaying the effect of sugar on coronary heart disease, while blaming dietary fat as the culprit in heart disease. The authors concluded that policymakers should give less weight to research funded and conducted by the food industry and include more animal and mechanistic studies evaluating the effect of sugar on coronary heart disease biomarkers and disease development. Not all studies sponsored by food industries have an ulterior motive; however, it is important to pay attention to who is funding such studies and who is really benefiting from the results.

Big business has funded the following research studies:


**Dietary Influence on Neurogenesis**

A research review discusses the role dietary intervention can play in neurogenesis and stem cell function with age. As we age, there is a decrease in cell regeneration within the body and brain which impairs stem and progenitor cell function, and decreases these stem cells' ability to regenerate and differentiate into specialized cells. Throughout their life, stem cells can change their activities dependent upon organismal demands and nutritional changes, which can rapidly influence the extrinsic signals delivered to these cells. Nutrient availability to molecular pathways and calorie restrictive diets were reviewed in regards to their influence on stem cell function. Different experimental studies involving parabiosis (joining of circulatory systems allowing for the exchange of blood) of both youthful and ageing mice were reviewed to determine the effect of circulating factors in the bloodstream on tissue function. It is suggested that circulating factors changed by diet can positively influence stem cell regeneration and function. Research on animals exposed to a youthful milieu has shown that consumption of a calorie-restricted diet (in the absence of malnutrition) has a beneficial effect on stem cell number and function in various tissues, reversing the declines in stem cell activity with age. Calorie restricted diets were also shown to influence metabolic growth in maintenance and repair functions, and promote oxidative phosphorylation in glycolysis. One of the authors of this review, Sandrine Thuret, PhD, concluded these findings in a Ted Talks presentation, in addition to further research. She discussed how active mice were shown to have increased neurogenesis as well as diets higher in flavonoids, folic acid, omega-3 fatty acids, and zinc. Conversely, diets high in sugar, fats, ethanol or deficient in vitamins A, B, and E were shown to decrease neurogenesis.


**Metabolic and Inflammatory Benefits of Pairing Walnuts with Whole Foods**

Certain foods have been shown to provide different benefits when eaten alone versus in combination with other foods. An animal feeding study examined the effect of metabolic disease conditions by combining whole foods with English walnuts, which are rich in α-linolenic acid (ALA), an ω-3 polyunsaturated fatty acid (PUFA). Twelve groups of 96 (n=8 each) mice were provided high-fat (HF) and low-fat (LF) control diets, a HF diet paired with walnuts, or a combination of walnuts with different whole foods for nine weeks. Each of the groups pairing walnuts and whole foods consisted of whole foods of either: blueberries, apples, cranberries, tart cherries, broccoli sprouts, olive oil, soy protein, or green tea. By week nine, the body weights of the low-fat group were significantly lower at 5.5 ± 0.5 g, compared to the high-fat group at 17.3 ± 1.0 g (P < 0.05). There was no variance in weights between the different high-fat groups except for a lower body weight in the green tea and walnut group and contrariwise the soy-protein group had an increased in body weight. A glucose tolerance test performed at week eight showed the area under the curve was significantly higher in the mice fed the high-fat control group in comparison to the mice fed the walnut and raspberry, apple, or green tea group. A liver histology showed the groups fed with green tea and broccoli sprouts had a decreased hepatic fat concentration as compared to the mice fed the HF-control diet. Metabolites including 7-α cholesterol, several ω-3 PUFAs, phospholipids, diacylglycerols, and glycerol 3-phosphate were higher in the HF diet than the LF diet groups. There was a higher reduction in glutathione, cysteinylglycine, γ-glutamylcysteine, bile acid metabolites, and free coenzyme A, in the HF diet compared to mice who consumed the LF diet. In comparison to the mice in the LF- and HF-diet control groups, all mice fed walnuts in the diet had moderately higher levels of ALA, eicosapentaenoic acid, and docosahexaenoic acid, all ω-3’s. All mice with the walnut-fed diet had lesser concentrations of arachidonic acid (a polyunsaturated omega-6 fatty acid) than the LF and HF control groups. Changes in gene expression and hepatic biochemical concentrations were shown from pairing walnuts with whole foods, improving carbohydrate and lipid metabolism and inflammation. This study shows that regular consumption of foods high in ω-3 PUFAs and polyphenols could lead to metabolic improvement.


**Documentary Series Review: Cooked**

The Netflix series, *Cooked*, is a documentary by Michael Pollan based on his book published in...
2013, Cooked: A Natural History of Transformation. Each of the four episodes focuses on a food that is representative of one of the four natural elements: fire, water, air and earth. Pollan investigates each food by traveling to the countries of origin, discussing its history, demonstrating their cooking techniques and teaching us how integral the food is to the country’s daily life and culture. Even in a short amount of time (just under an hour per episode), Pollan clearly connects each food to its respective element. The series gives us a grounding experience as Pollan shows us how the quest for food—hunting, gathering and preparing—shapes the day for many people of the world. Further, foods are not just nourishment, but represent health, income, and in some instances, faith. Each episode reminds the viewer to slow down, be mindful and show gratitude for the food in front of us. More information on Michael Pollan’s Cooked can be found here: http://michaelpollan.com/videos/netflix-documentary-series-cooked/. Michael Pollan. Cooked [TV Mini-Series]. USA: Jigsaw Productions, Netflix; 2016.

Co-reviewed by Jena Savadsky Griffith, BA, BS, Associate Newsletter Editor.


Nutrigenomics in the modern era. Proc Nutr Soc. 2016;7:1-11. [Epub ahead of print] (PubMed ID: 27819203) Genetic variation alone does not adequately explain diet-related phenotypes like obesity or common complex diseases. However, nutritional genomics continues to develop and can provide useful tools for further research. When combined with behavioral and lifestyle changes, personalized nutrition has the potential of making a major contribution to better health worldwide.

Nutrigenomics at the interface of aging, lifespan, and cancer prevention. J Nutr. 2016;146(10):1931-1939. Epub 2016 Aug 24. (PubMed ID: 27558581) This review focuses on telomerase activity, energy metabolism (bioenergetics), DNA repair and oxidative stress in relation to aging and age-related health, and to cancer prevention. The author concludes by suggesting that nutrigenomics has the potential to “revolutionize” our understanding of more individualized nutritional needs. Because questions still remain, continued research is encouraged.

The importance of gene-environment interactions in human obesity. Clin Sci (Lond). 2016;130(18):1571-97. doi:10.1042/CS201600221. (PubMed ID: 27503943) An introduction to obesity-related gene-environment interactions is provided, along with a review of the evidence, the mechanisms, and the challenges. It concludes by suggesting that our growing understanding may have “tremendous applications” for individualized and/or population-wide health and lifestyle recommendations.

Moving towards specific nutrigenetic recommendation algorithms: Caffeine, genetic variation and cardiovascular risk. J Nutrigenet Nutrigenomics. 2016;9(2-4):106-115. Epub 2016 Jul 29. (PubMed ID: 27467525) Individual response to caffeine ingestion includes a genetic element that can affect cardiovascular risk. This paper discusses nutrigenetic considerations as an example of an approach that can be taken toward practical applications of nutritional genomics. Table 3 provides a listing of genes and gene variants that are suspected to be relevant to the cardiovascular effects of caffeine.

Association between the vitamin D receptor gene polymorphism and osteoporosis. Biomed Rep. 2016;5(2):233-236. Epub 2016 May 31. (PubMed ID: 27446548) The Apal variant (rs17879735) of the vitamin D receptor (VDR) gene was found to be associated with lower bone mineral density among the Chinese subjects tested and is described as "important" with regard to osteoporotic risk.

Genes, environment and gene expression in colon tissue: a pathway approach to determining functionality. Int J Mol Epidemiol Genet. 2016;7(1):45-57. eCollection 2016. (PubMed ID: 27186328) The combination of genetic variations with environmental and lifestyle factors can affect gene expression and the risk of cancer. Table 2 provides a listing of genes and gene variants that were evaluated. Table 3 lists associations between lifestyle factors and gene expression, with Tables 4-5 providing more details on specific gene variants. The authors conclude by suggesting that more attention should be given to gene expression within particular pathways and suggest that their data illustrates the importance of environment and lifestyle.


Consumer perceptions of interactions with primary care...
.providers after direct-to-consumer personal genomic testing. *Ann Intern Med.* 2016;164(8):513-22. doi:10.7326/M15-0995. Epub 2016 Mar 1. (PubMed ID: 26928821) A total of 1249 customers of 23andMe, and 589 customers of Pathway Genomics participated in this study. Sixty-three percent of these participants planned to share their results with their primary care providers, some of which reported the results of such interactions. Sharing of results is expected to increase in the future. This PDF can be found at http://www.genomes2people.org/wp-content/uploads/2016/02/Van-Der-Wouden-et-al-2016.pdf.

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A most interesting session presented at FNCE® titled “Emerging Nutrition Interventions in Urology” presented recommendations and justification for including physical therapy referrals for urological patients along with nutrition intervention.

Barbara Shorter, EdD, RDN, CDN, and Barbara Gordon, RDN, LD gave a very thorough presentation on the differing urological conditions that affect many women, leaving them uncomfortable and often in pain. Two of these conditions, stress urinary incontinence (SUI) and interstitial cystitis/bladder pain syndrome (IC/BPS) have become increasingly well-known and better understood.

SUI affects 20-40% of American women and is characterized by leakage of urine when laughing, coughing, sneezing and/or during intense physical activity. Women of any age who are overweight are at risk of SUI as excess abdominal fat can place pressure on the bladder, causing leakage. Excess weight can also put pressure on the pelvic floor muscles making them weak and susceptible to leakage. Nutrition assessment and diagnosis begins with asking patients about SUI, evaluating excess energy intake and inadequate fluid intake, and reviewing sleep patterns, food sensitivities, activity and bowel patterns. Nutrition intervention includes education with goal setting, counseling on healthy weight management and constipation strategies, referral to a physical therapist for constipation treatment and bladder control training, and coordination with the patient’s PCP. Women with SUI are often afraid to exercise because they are afraid of leakage. However, they should be encouraged to exercise as moderate levels of activity can help improve symptoms and control leakage.

IC/BPS is a chronic condition characterized by pelvic pain, pressure in the bladder and pelvic region, which is associated with urinary frequency and urgency. Research has shown that dietary modification can help patients manage IC by identifying the foods that cause pain and symptoms, meal patterns and size, fluid intake, either excess or inadequate, and certain medications. Assessing for allergies or sensitivities, bloating, irritable bowel syndrome, bowel related comorbidities, medications and supplements, and anthropometric data can provide insight into the nutrition related aspects of the condition and help establish a nutrition diagnosis. Once diagnosis has been established, education and intervention can begin with emphasis on coordinating care between the RDN, PCP and other healthcare providers, including physical therapists. Pelvic floor muscle strengthening activities, including Kegel exercises can help relieve symptoms; a physical therapist can help the patient know if they are being done correctly. Nutrition monitoring and evaluation is key to assuring that the patient’s goals are met and symptoms relieved, not only with IC/BPS, but with SUI and stones as well.

The handouts for this session were extensive, including descriptions for Kegel exercises, food sensitivity questionnaires, and a suggested elimination diet to help identify problematic foods. A list of bladder flare triggers other than foods reported by IC/BPS patients provides suggestions for things other than foods that may cause symptoms. The handouts and slides, available for download for FNCE® attendees, would be helpful resources for RDNs who see patients with urinary symptoms.
**Recipes: Mediterranean Red Lentil Soup With Herbs and Creamy Red Chai With Turmeric**

**Kory DeAngelo, MS, RDN, CD**

**Creamy Red Chai with Turmeric**

- 2 cups water
- 4 whole cloves
- 1 star anise
- ½ vanilla bean, seeded and chopped
- 2 cinnamon sticks
- 6 green cardamom pods, crushed
- 6 whole black peppercorns
- ½ inch fresh ginger, grated
- ¼ teaspoon ground turmeric
- 1 heaping tablespoon red rooibos tea
- ½ cup non-dairy milk
- Raw honey (optional)

Combine water, cloves, star anise, vanilla, cinnamon, cardamom, peppercorns, ginger, and turmeric in a pot and bring to a boil. Turn down heat, cover, and simmer for 30 minutes. Add red rooibos tea and milk and steep for 5 minutes. Strain and serve. Add honey if desired. Serves 2.

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**The Avocado Gets a Serving Size Makeover**

In healthy-fat news, the FDA recently increased the official serving size of a medium avocado from 1/5 (30 grams) to 1/3 or 50 grams. These serving sizes are not mandated by the FDA, but are intended to reflect the amount consumers are eating. The FDA last updated the avocado serving size in 1993. Over the last 15 years, per capita annual avocado consumption has increased from two pounds per person to seven pounds per person and is continually growing. This is not surprising to many RDNs as the avocado is praised for being a good source of fiber, mono and polyunsaturated fat, folate, potassium, vitamin E, vitamin C and is cholesterol free. Additionally, according to NHANES, avocado consumption is associated with better diet quality, higher nutrient intake and lower metabolic syndrome risk. With all of the attention now given to this valuable food, many may consume ½ or even an entire avocado, which is certainly easier to measure!

**Sources**


Information compiled by Jena Savadsky Griffith, BA, BS, Associate Newsletter Editor
The Academy Takes Action to Address Food and Nutrition Security

Helene Kent, MPH, RDN

Some Facts from the Feeding America's Hunger in America 2014 Report

- Fourteen percent of American households (17.4 million households) were food insecure with 6% of households (6.9 million households) experiencing very low food security.
- Households with children reported food insecurity at a significantly higher rate than those without children—19% compared to 12%.
- Households that had higher rates of food insecurity than the national average included households with children (19%), especially households with children headed by single women (35%) or single men (22%), Black non-Hispanic households (26%) and Hispanic households (22%).
- Food insecurity exists in every county in the US with rates of food insecurity among rural households generally higher than urban households.
by Feeding America and the Academy. Keep informed by subscribing to the Healthy Food Bank Hub Digest.

- **Conduct food insecurity screening** within your practice setting and work with local partners to make referrals for food access.
- **Volunteer or otherwise support activities** to address food and nutrition security.
- Respond to the Academy’s Action alerts on food and nutrition security.
- Speak with legislators, community leaders and other healthcare professionals **about the causes and solutions to food and nutrition insecurity**.
- Share your expertise by submitting your preferred tools and best practices to the Healthy Food Bank Hub.
- If you are in a position to educate and engage in research, we also ask you to teach students and interns about food insecurity and the role of Academy members in these efforts. An excellent resource is the Food Insecurity/Food Banking Supervised Practice Concentration. In 2017, the Nutrition and Dietetic Educators and Preceptors (NDEP) will make available a classroom resource for educators.
- Engage in research activities to determine how members can best relieve hunger and food insecurity. Learn more about the Dietetics Practice-Based Research Network (DPBRN).
- Lead community-based efforts to map and intervene in food and nutrition insecurity.
POM Wonderful®
100% Pomegranate Juice

Pure 100% Pomegranate Juice
- Every 16 oz. bottle has the juice of 4 pomegranates.
- No added sugars*, preservatives, colorants, or artificial sweeteners.

Antioxidant Superpower®
- Every 8 oz. serving of POM® has 700 mg of polyphenol antioxidants. Antioxidants may help fight free radicals that damage our cells.
- An in vitro study at UCLA found that pomegranate juice has, on average, more antioxidant capacity than red wine, grape juice or green tea[1].

Food for Thought
Scientists are beginning to examine the potential impact of pomegranate polyphenols on memory and cognition in older adults.

A preliminary 2013 UCLA study indicated that a small group of older adults with age-related memory complaints who drank 8 oz. of pomegranate juice daily showed increased verbal memory performance and functional brain activity in fMRI testing after just four weeks[2].

These early scientific results on the impact of PJ on the brain have not yet been adequately studied. Further clinical research on larger populations is needed to help establish causation and to determine the long term effects of pomegranate on memory and cognition.

[1] Seeram et al., 2008
[2] Bookheimer et al., 2013
*Not a low calorie food, see nutrition information for sugar and calorie content
Drink it Daily. Feel it Forever.

Morning Boost

POM Green Warrior

Ingredients:
- ½ cup green grapes
- ½ cup pineapple pieces
- 1 cup packed spinach
- 1 inch fresh ginger, peeled
- ¼ cup POM 100% Juice
- 4 ice cubes

Method: Combine all ingredients in blender; puree until mixed. Pour into your favorite glass and enjoy!

Afternoon Delight

POM Wonderful is deliciously refreshing and is known for its sweet-tart taste.

If you want to try something new, try:
- Mixing it with sparkling water
- Adding it to lemonade for POM lemonade
- Freezing POM Ice cubes and adding them to your favorite drink

Smart Sip

Beet Em Bust Em™ Smoothie

- 4 tbsp chia seeds
- 1 ¼ cup POM Wonderful 100% pomegranate juice
- 1 ripe banana, frozen
- 1 tbsp coconut chips
- 1 tbsp cacao nibs or carob chips
- 2 small slices raw beet

Method: Combine all ingredients in blender; puree until mixed. Pour into your favorite glass and enjoy!

Workout Companion

POM fits perfectly in your workout bag for a before & after refreshment.

- POM is a good source of potassium
- 16 oz. of POM Wonderful 100% Pomegranate Juice has as much potassium as 2 medium bananas
- Potassium is an electrolyte key to muscle function and fluid balance
Editor’s Notes

The New Year is upon us and it seems like just yesterday I had the opportunity to speak with many of you at the 2016 FNCE®! Thank you for your kind words and support of the newsletter, one of the great member benefits of DIFM membership.

The newsletter is a team effort and I must give credit where it is due. Without the keen eye of our Copy Editor, Emily Davis Moore, MS, RD, LD/N, and the editorial expertise of our ‘new’ Associate Editor, Jena Savadsky Griffith, BS, BA the information would not meet editorial standards of the Academy. I must also acknowledge the hard work of our CPE Editor, Shari B. Pollack, MPH, RD, LDN who tirelessly searches for, edits, and coordinates reviews and educational credits with the Academy for our exceptional CPE articles.

You may have noticed that we are working towards a newer format for the newsletter, one in which we will have columns specific to DIFM and member interests. Stay tuned for the changes and updates. Each of these columns have editors that are talented and hardworking as well. Dina Ranade, RD, LD is the editor for the Botanicals, Supplements and Functional Foods column; you may remember her as the Resource Review and Networking Editor who contributes great ideas and book reviews for the newsletter. Raquel Praino, Dietetic Intern, joined us last year, helping with EML reviews and has now taken over the helm of the Resource Reviews as well as News You Can Use and Member Spotlight column. We have had two new, well-versed and enthusiastic RDs join us—Doris Piccinin, MS, RD, CDE, LDN, the Mind/Body column editor who comes to us with extensive experience in cancer care and as a former educator at Bastyr University, not to mention her enthusiasm and energy for all things DIFM; and Danica Cowan, MS, RD, the Biochemistry/Nutritional Genomics column editor who has already shared her expertise with members in her article in the Fall 2016 newsletter entitled “Sarcopenia and the mTOR Pathway” and the article in this issue, “The Genetics of Celiac Disease.” Stay tuned for more from her in the near future. Our most recent addition to the team, Sarah Greenfield, RD, CSSD, will be assisting Shari Pollack with CPE. And, thanks to our Communications Chair, Malorie Blake, MS, RDN, LDN, CSNC, and Chair-Elect, Mary Purdy, MS, RDN for their continued support and guidance. And last, but not least, our article editors/reviewers, Linda Lockett Brown, ABD, M.Ag., RDN, LDN, CLC, Christian Calaguas, RD

Upcoming Issues

• Summer 2017, Editor’s Deadline April 1
• Fall 2017, Editor’s Deadline July 1
• CPE Deadline, June 15
• Winter 2018, Editor’s Deadline November 1, 2017
• Spring 2018, Editor’s Deadline February 1, CPE Deadline, January 15

The views expressed in this newsletter are those of the authors and do not necessarily reflect the policies and/or official positions of the Academy of Nutrition & Dietetics.

We invite you to submit articles, news and comments. Contact us for author guidelines. Send change-of-address notification to the Academy of Nutrition & Dietetics, 120 South Riverside Plaza, Ste. 2000, Chicago, IL 60606-6995.

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Sarah
During the annual DIFM Executive Committee meeting at FNCE® in Boston, we reviewed our strategic plan and made some new goals for the second half of the year. What was at the top of the list? Connect with our members more. We have now grown to be the third largest Dietetic Practice Group (DPG) in the Academy and our expertise is more in demand than ever. We want to enlist your support to help us continue to grow and to tap into the experts among us to help write, speak, consult and spread the word about integrative and functional nutrition.

Here are the top four ways we encourage you to connect with us:

1. **Join our electronic mailing list (EML) if you haven’t already.** It’s easy and accessible and the best way to connect with your fellow DIFM members. It’s an active group and a great way to learn, share and stay up to date on what’s going on in our DPG. Go to the [www.integrativerd.org](http://www.integrativerd.org) members only tab for details on how to sign up. If you are an **educator**, consider joining our connecting educators electronic mailing list by emailing your request to info@integrativerd.com.

2. **Engage in our social media platforms.** Facebook, Twitter, LinkedIn and/or DIFM blog. You know the drill—click, like, post and share…It’s the fastest and most fun way to keep in touch! Students, please join our student Facebook page (DIFM Student members). Search for Dietitians in Integrative and Functional Medicine on all platforms and you will find us!

3. **Submit an application to our Expert Database and Speakers Bureau.** We get requests almost daily for input from our members on integrative and functional nutrition—to speak at conferences, author articles and position papers, and comment on policy initiatives. We need you to help us meet this demand so that we can continue to participate and stay engaged with the Academy and affiliates, the US government and local and international organizations. Interest in our practice area is increasing and we need you. Please submit your application here [http://integrativerd.org/strategic-plan/speakers-bureau](http://integrativerd.org/strategic-plan/speakers-bureau). Don’t underestimate yourself and the contribution you could make!

In addition to connecting our members, we also want to connect with other dietetic practice groups (DPGs) in the Academy. Integrative and functional nutrition can be applied across every practice area in nutrition. Our professional advancement team is working on cross promoting webinars and we are currently seeking other opportunities. If you are a member of another DPG and would like to propose a collaborative project please email Kory DeAngelo, MS, RDN, CD at kdeangelo@bastyr.edu.

In 2017 we hope to get to know you better. Please reach out and connect with us—join one of our electronic mailing lists, follow us on social media, apply to the expert database and speakers bureau, become a state ambassador or help us collaborate with other Academy practice groups. We need your help to keep growing DIFM and spreading the word about integrative and functional nutrition.

Wishing you a Happy and Healthy New Year,

Kelly

Dietitians in Integrative and Functional Medicine
a dietetic practice group of the Academy of Nutrition and Dietetics

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