

Women's Health *REPORT*

A QUARTERLY PUBLICATION OF WOMEN'S HEALTH DIETETIC PRACTICE GROUP

ALZHEIMER'S DISEASE AND DEMENTIA: Role of Diet, Exercise and Overall Lifestyle

By Shoshana Werber-Flax, MS, RDN, CDN, CPT

This article is approved for 1 CPEU by the Commission on Dietetic Registration, the credentialing agency of the Academy of Nutrition and Dietetics. Upon reading this article, please take the quiz at <http://bit.ly/10uyqri>. To learn more about the author, see the Member Spotlight on page 8.



What are Alzheimer's Disease and Dementia?

Alzheimer's disease is the most common form of dementia (a group of symptoms including loss of memory, motor function and cognitive function due to damage/death of brain tissue) and accounts for 60-80% of cases. It is characterized by progressive loss of memory, cognition, and language, and loss of behavioral abilities related to activities of daily living.¹ There are many different types of dementia; each associated with specific brain abnormalities and symptom patterns.² Vascular dementia (VaD), the second most common type of dementia after AD, is caused by a reduced blood supply to the brain often as a result of stroke.³ The most significant risk factor for VaD is high blood pressure. "There is growing evidence that hypertension is the most important modifiable vascular risk factor for development and progression of both cognitive decline and dementia."⁴

Alzheimer's disease (AD) is a devastating irreversible neurodegenerative disease that attacks the brain's neurons, or nerve cells. The disease is named after Alois Alzheimer, the German doctor who first described the brain disease in 1906.⁵ Alzheimer's disease is a major public health threat as it currently affects 1 in 9 Americans over age 65 (11%) and 50% of Americans over age 85.⁶ Younger-onset Alzheimer's, also known as early onset, affects people who are younger than 65 and accounts

for 5% of all people with AD.¹ Women are more likely to develop AD than men, with women accounting for almost two-thirds of Americans with the disease.³

Alzheimer's is one of the top ten causes of death in the United States; however, it is the only disease among these that currently cannot be prevented, slowed or cured.³ In 2015, 5.3 million Americans were diagnosed with AD, a number projected to increase to 7.1 million by 2025 and 13.8 million by 2050.³ As of 2015, 46.8 million people around the world were living with dementia.⁷ The prevalence of AD and other types of dementia continues to rise and the trend will likely continue as average life expectancy increases.

In addition to being a major public health issue, the disease is a tremendous worldwide economic burden, costing \$818 billion in 2015.⁷ Currently, AD and other dementias are most common in Western Europe, followed by North America, and are least common in Sub-Saharan Africa.⁶ Concern or fear of developing AD seems to increase with prevalence. A 2011 MetLife® Foundation telephone survey revealed that American adults feared developing AD more than any other disease, second only to cancer,⁸ while a 2013 YouGov® survey found people 60 years of age and older feared developing AD or dementia more than any other chronic condition including cancer.²

Despite the increase in prevalence worldwide, current estimates suggest that the incidence of dementia may be on the decline in the Western world and in high-income countries due to better cerebrovascular care and enhanced brain health;⁹ a trend that has been observed by researchers involved with the Framingham Heart Study. Over the past 30 years, the incidence of dementia has decreased among study participants. The researchers write that "risk reduction was observed only among persons who had at least a high school diploma...The prevalence of most vascular risk factors (except obesity and diabetes) and the risk of dementia associated with stroke, atrial fibrillation, or heart failure have decreased over time, but none of these trends completely explain the decrease in the incidence of dementia."¹⁰

What Happens to the Aging Brain?

Aging causes changes to the brain's size and vasculature, as well as to perception and understanding. The brain's weight declines at a rate of approximately 5% per decade, beginning at age 40.

in this issue

From the Chair and Editor	2
Healthy Aging and Brain Health	7
WH DPG Member Spotlight	8
Research Brief	9
An Update on Zika	11
HOD Fact Sheet	13
WH DPG Member Benefits	15

Chair

Heather A. Goesch, MPH, RDN, LDN

Publications Editor

Kathleen Pellechia, RD

Assistant Publications Editor

Wendy Baier Cartier, RDN

Resource/Book Reviewer

Carrie Dennett, MPH, RDN, CD

Research Coordinator

Christine Garner, PhD, MS, RD, CLC

Member Spotlight

Lauren Manaker, MS, RD, LD

Design and Layout

Steve Bonnel

Editorial Board

Miri Rotkowitz, MA, RD

Denise Andersen, MS, RDN, LD, CLC

Kathleen Pellechia, RD

Lisa Hamlett Akers, MS, RD, IBCLC, RLC

Heather Goesch, MPH, RDN, LDN

Wendy Baier Cartier, RDN

Reviewers

Helen W. Lane, PhD, RD

Katie Smith, PhD, RD

Kendra Tolbert MS, RDN, CDN, CLC

Dolores M Wolongevicz, PhD, RD, LDN

Please send any questions or comments to publications@womenshealthdpg.org

The Women's Health Report (ISSN-3233) is an online quarterly publication of the Women's Health Dietetic Practice Group (WH DPG) of the Academy of Nutrition and Dietetics. The WH Report features articles, as well as information on programs, materials, positions, and products for use of its readers. News of members, book reviews, announcements of future meetings, requests for information, or other items of interest to women and reproductive nutrition dietetics practitioners should be sent to the Publications Editor at publications@womenshealthdpg.org.

The statements in this publication do not imply endorsement of the WH DPG or the Academy of Nutrition and Dietetics. © 2016.

We're on the web!

www.womenshealthdpg.org



FROM THE CHAIR Heather A. Goesch, MPH, RDN, LDN

Dear WH Members,

By the time you read this my term as DPG Chair will be done. It was a very exciting year! I believe we made great strides in strengthening our reach and our resources. Through every webinar, forum conversation, publication, tweet and post I learned something new – and hope you did, too.

This final Women's Health Report of 2015-16 tackles the topic of brain health. Our feature is a well-written piece on Alzheimer's and dementia from Shoshana Werber, MS, RDN, CDN, CPT. As a WH member who specializes in this area of nutrition, she is also the focus of our Member Spotlight. Elsewhere in the issue you will find complementary pieces, including a resource guide on brain health and healthy aging, information on the Zika virus, research updates, as well as DPG news.

If you haven't already, please join me in welcoming our new Chair, Catherine Sullivan, MPH, RD, LDN, IBCLC, RLC. Catherine brings a deep passion and wealth of knowledge of women's health and breastfeeding, and I'm thrilled to see what's in store for our DPG this year! Thank you to the returning and departing member volunteers who round out our multi-talented Leadership Team – your countless hours of devotion help make WH better than ever. And to the WH Membership: Your thoughtful participation and sharing of knowledge is invaluable to enhancing our DPG and our profession. Thank you!

It's been an honor and privilege to serve as Chair of the WH DPG. I look forward to continuing to work with and for you as Past Chair, and wish each of you a most enjoyable summer.

Cheers,
Heather



FROM THE EDITOR Kathleen Pellechia, RD

Dear WH members,

I am amazed that summer is upon us and we have reached our last issue for this year. We apologize for its late arrival, and beginning with our next issue we will fully transition to the new membership year with another four jam-packed issues to share with you.

This issue focuses on brain health and is something that we hear more and more about in the media and in the literature. Whether it be the brain-gut connection, the importance of diet and exercise, the role of keeping the mind active as we age, etc., there are endless opportunities for us to impact how our brain ages. I think it is always exciting when our feature author is also a member, so I thank Shoshana Werber, MS, RDN, CDN, CPT for taking time out of her busy schedule to write for us on the topic of Alzheimer's disease and dementia.

As we look ahead into the 2016-2017 membership year, we will be celebrating the 100th anniversary of the Academy of Nutrition and Dietetics with our own look at 100 years of women's health and nutrition. If you have stories and pictures to share, we would love to highlight you in an upcoming issue. Ideas can be sent to publications@womenshealthdpg.org.

I wish you a wonderful, peaceful summer and look forward to continuing to serve as your Publications Editor.

The actual rate of brain volume decline increases with age, especially over age 70 and shrinkage rates increase with diseases like AD.¹¹ Genetics, hormones, neurotransmitters, lifestyle and health conditions all influence brain aging.

Brain changes related to AD begin years before any signs of the disease. This time period is referred to as preclinical AD.¹ Hallmark pathologies of AD include plaques, deposits of the protein fragment beta-amyloid and tangled, twisted strands of the protein tau;¹² these brain changes are eventually accompanied by damage and death of neurons. As the disease progresses over time, the brain noticeably shrinks and almost all of the brain's functions are affected. Short-term memory fails when AD first destroys nerve cells in the brain's hippocampus, an area of the brain that plays an important role in new memory formation. Neuron death in the cerebral cortex causes deterioration of judgment and language skills. In advanced AD, the brain's cortex shrinks, causing damage to areas of the brain involved in planning, thinking and remembering.¹²

What are Major Risk Factors for AD?

The following section will give examples of research studies for some major risk factors.

Age

After age 65, the likelihood of developing Alzheimer's doubles every 5 years. People who are 85 years of age and older have almost a 50% chance of developing the disease,³ however while older age is a risk factor for AD, the disease is not a normal part of getting older.¹

Family History and Genetics

People with one or more family member(s) (parents, siblings or children) who have the disease are at increased risk for AD. People with the epsilon 4 variant of the apolipoprotein E gene, which is the strongest genetic risk factor identified as of yet, are also at increased risk.²

Down Syndrome

People with Down Syndrome are 3 to 5 times more likely to develop AD and the disease generally develops at a younger age than in the general population.¹³

Mild Cognitive Impairment (MCI)

An estimated 10-20% of people over 65 have MCI and are at increased risk for AD and other types of dementia. While some memory loss is normal with aging, a diagnosis of MCI indicates more substantial memory loss and minor cognitive difficulties that generally do not interfere with independent function or activities of daily living.¹⁴

Education Level

Low education level may be a risk factor for AD and other dementias.¹⁵ Some studies suggest that greater educational and occupational achievement may decrease the risk of AD, "either by decreasing ease of clinical detection of AD or by imparting a reserve that delays the onset of clinical manifestations."¹⁶

Environmental/Lifestyle Factors

Environmental and lifestyle factors also play a major role in whether someone develops AD or other dementias. History of head trauma, especially traumatic brain injury (TBI), increases risk for AD.¹⁷ It is now well established that brain health is closely tied to overall heart and vascular health, as compromised cardiovascular function decreases blood supply and thus oxygen to the brain.

Type 2 diabetes is also linked with AD, conferring a roughly twofold increased risk.¹⁸ In 2005, Suzanne de la Monte and colleagues at Brown Medical School, coined the term "type 3 diabetes" to describe AD. De la Monte's team examined postmortem brain tissue from 45 patients with AD and found that the disease was associated with both a decrease in the production of insulin and a resistance to insulin receptors. These findings were further supported by the use of both an experimental animal model that mimics brain diabetes following intracerebral administration of a drug that is commonly used to produce Type 1 or Type 2 diabetes and a study on how PPAR agonists (used to treat Type 2 diabetes) have been shown to prevent many of the AD-associated neurodegenerative effects.¹⁹

In addition, insulin resistance,²⁰ heart disease,²¹ stroke,²² hypertension,²³ hypercholesterolemia,²⁴ hyperhomocysteinemia,²⁵ obesity,²⁶ smoking,²⁷ and inflammation²⁸ have all been linked to increased risk of AD, other dementias or cognitive decline.

Women and AD Risk

Women are more vulnerable to developing AD than men, with 60% of those diagnosed being female.²⁹ It is hypothesized that women account for a greater percentage of existing cases because on average they live longer than men and are therefore more likely to reach an age that puts them at risk.²

According to Laws, et al., there is "clear evidence from brain imaging, post-mortem analyses, hormone therapy and genetics suggesting that AD affects men and women differently."³⁰ Research also indicates that women are more likely to carry the APOE-ε4 allele gene than men.³¹ Researchers at the University of Southern California, refer to sex, age and the APOE gene as a "triad of risk of Alzheimer's disease."²⁹ In a paper published in March 2016, the authors wrote that "the bioenergetic aging perimenopause to menopause transition unique to the female, creates a risk profile for AD unique to the female."²⁹ Other researchers explain that the higher incidence of AD in women is due to "a reduction of estrogen in postmenopausal women, sex differences in AD pathology, and greater cognitive reserve in men."³¹ More studies are needed to examine the biological differences in the disease process between women and men. Researchers are hopeful that by identifying the role that gender differences play in the etiology of AD, more accurate treatments and prevention strategies will emerge.³⁰

What Lifestyle Factors are Neuro-protective and Help Prevent Cognitive Decline?

The following section will give examples of research studies for some lifestyle factors.

Continued on page 4

Diet

It is well established that a Western diet, one high in calories, saturated fat, trans fats and refined carbohydrates, increases risk for obesity, heart disease, diabetes, metabolic syndrome and cerebrovascular disease. Research indicates all of these conditions are strongly linked to cognitive impairment.³² Conversely, research has shown that cardio-protective diets seem to be neuro-protective. Therefore, it appears that what's good for the heart and the body is also good for the brain.

Oxidative stress, inflammation and vascular impairment contribute to age-related cognitive decline. "There is also evidence that brain tissue in patients with AD is exposed to oxidative stress (e.g., protein oxidation, lipid oxidation, DNA oxidation and glyco-oxidation) during the course of the disease."³³

The value of dietary changes involving dietary patterns appears to be more promising than the research on specific dietary components, including vitamin E,³⁴ omega-3 fatty acids,^{35,36,37} B vitamins,³⁷ vitamin D,³⁸ resveratrol,^{39,40} and curcumin.⁴¹

Mediterranean Diets

Mediterranean diets are culturally-based dietary patterns, emphasizing whole grains, fresh fruits, vegetables, legumes, nuts, fish, olive oil and moderate wine intake. Moderate wine intake is considered up to 1-5 ounce glass of wine per day for women and up to 2-5 ounce glasses for men.⁴² Many of the vegetables emphasized are green leafy vegetables, which are often cooked or eaten with olive oil, thereby intensifying their health benefits.⁴³ The olive oil, itself, especially extra virgin olive oil, is high in polyphenols like oleuropein, which has been shown to provide neuro-protection⁴⁴ and is the main source of dietary fat used for cooking, baking, and for dressing salads and vegetables. Meat, cheese and dairy products, processed foods and sweets are limited as part of the Mediterranean diet.⁴³

Mediterranean Diets and Cognitive Health Research

European researchers in Barcelona Spain compared a Mediterranean diet supplemented with either olive oil or nuts to a low-fat diet control group.⁴⁵ The Mediterranean diet pattern used was based on a 2009 systematic review conducted by Mente, et al that utilized the Bradford Hill guidelines to "derive a causation score for each dietary exposure in cohort studies and examined for consistency with the findings of randomized trials."⁴⁶ The randomized clinical trial included 447 cognitively healthy volunteers (average age 67) who were at high risk for cardiovascular disease and enrolled in the Prevencion con Dieta Mediterranea nutrition intervention from October 2003 until December 2009.⁴⁵

The study participants were assigned into three groups in batches of 50 balanced for sex and age using a computer-generated random number sequence. The first group (155 participants) was assigned to supplement a Mediterranean diet with 1 liter of extra virgin olive oil every week. The second group (147 participants) was assigned to a Mediterranean diet supplemented with 30 grams of mixed walnuts, hazelnuts and almonds each day (28 grams ≈ 1 ounce of nuts). The specific daily breakdown was 15 grams (g) of walnuts, 7.5 g of hazelnuts and 7.5 g of almonds. The

third group, the control group, (145 participants) was assigned to follow a low-fat diet. After the intervention, participants were evaluated for cognitive change, and results were ultimately available from 334 of the original 447 participants. Compared with the control group, cognitive improvement was seen for participants in both the Mediterranean diet plus nuts group and in the Mediterranean diet plus olive oil group, suggesting that these dietary patterns may help prevent age-related cognitive decline in an older population.⁴⁵

DASH Diet

The Dietary Approaches to Stop Hypertension (DASH) diet was designed to help treat or prevent high blood pressure.⁴⁷ Like the Mediterranean diet, the DASH diet is rich in fruits, vegetables, fat-free/low-fat dairy products, whole grains, fish, poultry, beans, seeds and nuts. Overall, the diet is high in fiber and other nutrients associated with lowering blood pressure: potassium, magnesium and calcium. The DASH diet is also low in sodium (sodium limited to either 2,300 or 1,500 milligrams a day depending on other risk factors), saturated fat, cholesterol, red meat, sweets, added sugars and sugary beverages.⁴⁸

DASH Diet and Cognitive Health Research

Researchers at Duke University Medical Center examined the combined effects of the DASH diet and aerobic exercise on neurocognitive function among overweight and obese adults with hypertension. As part of the larger Exercise and Nutrition Interventions for Cardiovascular Health (ENCORE) study, 124 overweight or obese sedentary participants with hypertension (none of whom were taking anti-hypertensive medication) were randomly assigned to one of three groups: DASH diet alone; DASH diet combined with a behavioral weight management program, including 30 minutes of supervised aerobic exercise three times per week and weekly group counseling sessions; or a "usual diet" control group.

Participants in both of the treatment groups had lower blood pressure compared to those in the control group after four months (the end of the study period).⁴⁹ Perhaps, unsurprisingly, the DASH diet plus weight management group exhibited the greatest blood pressure reduction, the greatest weight loss and the best aerobic capacity. The results of this study demonstrate that the DASH diet, especially when combined with aerobic exercise, can improve neurocognitive performance among people with hypertension. Furthermore, the most apparent improvements in neurocognitive performance were seen among participants with the poorest vascular health.⁴⁹

MIND Diet

The Mediterranean-DASH Intervention for Neurodegenerative Delay (MIND) diet is a hybrid between the Mediterranean and DASH diets and includes many of the same dietary components. The MIND diet was specifically developed as an eating plan for better brain health and highlights natural, plant-based foods, with a particular emphasis on berries and green leafy vegetables.⁵⁰ The diet is comprised of 15 dietary components (ten "brain-healthy" & five "unhealthy" food groups).

The “brain-healthy” components include green leafy and other vegetables, nuts, berries, beans, lentils and soybeans, whole grains, seafood, poultry, olive oil and wine. The “unhealthy” food components include red meat, butter and stick margarine, cheese, pastries and sweets and fried or fast foods. The MIND diet foods contain nutrients demonstrated to reduce oxidative stress, decrease inflammation, slow cognitive decline and lower risk for AD.⁵⁰

MIND Diet and Cognitive Health Research

A prospective study led by Martha Clare Morris, a professor at Rush University, examined 923 participants, ages 58 to 98 years old. All participants were dementia-free (average age 81) at the start of the trial and were followed for an average of 4.5 years.⁵⁰ Diet was assessed yearly by a semi-quantitative food frequency questionnaire and cognitive brain tests were conducted yearly. Dietary components from the food frequency questionnaire were then scored using guidelines for the MIND diet, Mediterranean-type diet, and DASH diet. Overall guidelines for the MIND diet included daily consumption of at least three servings of whole grains, a salad plus one other vegetable and a glass of wine. Weekly recommendations included nuts as a snack most days of the week, bean consumption every other day, berries and poultry at least twice a week and fish at least once per week. Study participants were encouraged to limit butter or stick margarine to less than one tablespoon per day, cheese and fast or fried foods to less than one serving each per week and in general to limit red meat, pastries, and sweets.⁵⁰

High adherence to all three dietary patterns was associated with a decreased risk of AD. However, the MIND diet was most effective overall and was shown to reduce the risk of AD by as much as 53% in study participants who strictly followed the diet, and by 35% in participants who adhered to the diet moderately well. Study participants who adhered most strictly to the diet had brains roughly 7.5 years younger than study participants who did not at the end of the trial. Interestingly, the authors found that strict compliance to the MIND diet was not necessary in order to reap at least some neuro-protective benefits.⁵⁰

Exercise

An increasing number of studies have explored the role of physical activity and cardiorespiratory fitness as a way to decrease risk for cognitive decline and AD. For example, in a prospective study conducted with women 65 and older, researchers found that of the 5,925 women enrolled in the study, those who exhibited greater physical activity at baseline were less likely to develop cognitive decline during the 6-8 year follow-up.⁵¹

A community-based, pilot randomized controlled trial found that “an individual’s cardiorespiratory fitness response was a better predictor of cognitive gains than exercise dose (i.e., duration) and thus maximizing an individual’s cardiorespiratory fitness may be an important therapeutic target for achieving cognitive benefits.”⁵² Further findings from a recent study published in the *Journal of Alzheimer’s Disease*, suggests that regular physical

activity including dancing and gardening, may decrease risk of AD by as much as 50%.⁵³

Sleep

Sleep is restorative for the brain and helps keep the mind and memory sharp. It is recommended that adults get between 7 and 8 hours of sleep per night.⁵⁴ People with sleep difficulties due to insomnia or disruptions from sleep apnea should seek medical help as sleep disturbance can increase AD risk.⁵⁴ “Because late-life sleep disturbance can be treated, interventions to improve sleep or maintain healthy sleep among older adults may help prevent or slow AD to the extent that poor sleep promotes AD onset and progression.”⁵⁵

Smoking Cessation and Limiting Alcohol

It is well established that smoking increases cardiovascular disease risk. Cardiovascular risk factors and smoking are both linked to an increased likelihood of developing AD and other forms of dementia.²⁷ “The risk of dementia, AD, and VaD was dose-dependent such that the risk increased with increasing the amount of smoked cigarettes. Indeed, very heavy smokers (those who reported smoking >2 packs per day in middle age) were at the greatest risk of dementia even decades later in life.”²⁷

Furthermore, drinking heavily causes neurodegenerative changes in the brain, accelerating shrinkage or atrophy of the brain.⁵⁷ Since heavy drinking is associated with greater risk for cognitive decline, alcohol should be consumed in moderation.

Mental Health

Depression can affect brain health. Late-life depression is a prevalent psychiatric disorder and research demonstrates that it is associated with an increased risk for dementia.⁵⁸ Depression is not a normal part of aging and can be treated with counseling and/or medications.

Head Safety

Brain injuries increase risk for cognitive decline and dementia. Suffering from TBI early in life or in midlife is linked to an increased risk of dementia later in life.¹⁷ TBI is also linked to an earlier age at onset in people with AD or MCI.⁶⁰ To help avoid head injuries, always wear a seat belt and a helmet when appropriate.

Learning

Challenging the mind benefits the brain; examples include doing something creative, playing a new board game, learning a new card game or completing a puzzle. Studies show that increased participation in these types of activities in middle and late life is associated with decreased rates of cognitive decline.⁶¹

Social Activity and Engagement

Older adults with greater social ties tend to live longer.⁶² Volunteering, social hobbies, community involvement and simply staying connected to friends, family and neighbors all help the brain. Social engagement also appears to be extremely important for overall quality of life.⁶²

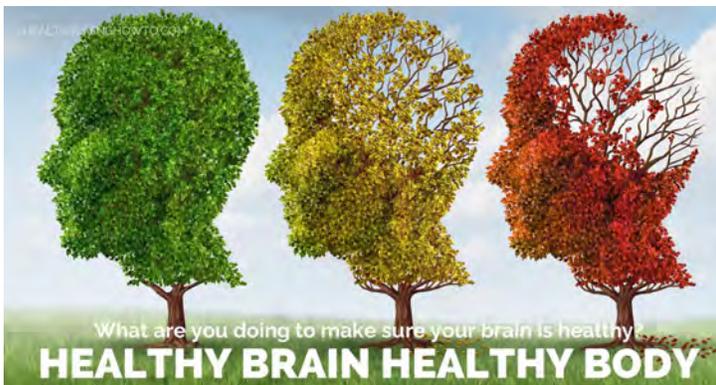
Continued on page 6

Summary

Dietary patterns that include anti-inflammatory and antioxidant-rich foods appear to be potentially promising nutritional tools to combat AD and slow the rate of cognitive decline. Despite some encouraging research on certain dietary supplements, further studies are needed to investigate their role in neuro-protection. Nutrition interventions that focus on protecting the brain while combating diseases that increase risk of AD such as cardiovascular disease and Type 2 diabetes also appear to be beneficial. Combining a brain-healthy diet with positive physical, mental and social activities as well as proper sleep and no smoking, may be the most supportive strategy to possibly prevent age-related cognitive decline, and dementia/AD.

References

1. What is Alzheimer's? Alzheimer's Association Website. http://www.alz.org/alzheimers_disease_what_is_alzheimers.asp#basics. Accessed March 14, 2016.
2. Alzheimer's Association, 2014 Alzheimer's Disease Facts and Figures, Alzheimer's & Dementia, Volume 10, Issue 2.15.
3. 2015 Alzheimer's Diseases Facts and Figures. Alzheimer's Association Website. <http://www.alz.org/facts/overview.asp>. Accessed March 14, 2016.
4. Gąsecki D, Kwarciany M, Nyka W, Narkiewicz K. Hypertension, Brain Damage and Cognitive Decline. *Current Hypertension Reports*. 2013;15(6): 547-558.
5. Major Milestones in Alzheimer's and Brain Research. Alzheimer's Association Website. http://www.alz.org/research/science/major_milestones_in_alzheimers.asp. Accessed March 14, 2016.
6. 2015 Alzheimer's Statistics. Alzheimers.net Website. <http://www.alzheimers.net/resources/alzheimers-statistics/>. Accessed March 14, 2016.
7. Dementia Statistics. Alzheimer's Disease International. <http://www.alz.co.uk/research/statistics>. Accessed March 14, 2016.
8. MetLife Foundation. What America Thinks MetLife Foundation Alzheimer's Survey. <http://www.madrc.org/sites/madrc/files/alzheimers-2011.pdf>. Published February 2011. Accessed March 24, 2016.
9. Scheltens P, Blennow K, Breteler MM, et al. Alzheimer's disease. *Lancet*. 2016; (15)01124-1.
10. Satizabal CL, Beiser AS, Chouraki V, Chêne G, Dufouil C, Seshadri S. Incidence of dementia over three decades in the Framingham heart study. *New England Journal of Medicine*. 2016;374(6):523-32.
11. Peters, R. Ageing and the brain. *Postgraduate Medical Journal*. 2006; 82(964): 84-88.
12. Brain Tour. Alzheimer's Association Website. http://www.alz.org/braintour/plaques_tangles.asp. Accessed March 14, 2016.
13. Down Syndrome and Alzheimer's Disease. Alzheimer's Association Website. <http://www.alz.org/dementia/down-syndrome-alzheimers-symptoms.asp>. Accessed March 15, 2016.
14. Mild Cognitive Impairment. Alzheimer's Association Website. <http://www.alz.org/dementia/mild-cognitive-impairment-mci.asp>. Accessed March 14, 2016.
15. Caamaño-Isorna F, Corral M, Montes-Martínez A, Takkouche B. Education and dementia: a meta-analytic study. *Neuroepidemiology*. 2006;26(4):226-32.
16. Stern Y, Gurland B, Tatemichi TK, Tang MX, Wilder D, Mayeux R. Influence of education and occupation on the incidence of Alzheimer's disease. *Journal of the American Association*. 1994;271(13):1004-10.
17. Shively S, Scher AI, Perl, DP. Dementia resulting from traumatic brain injury. *Archives of Neurology*. 2012;69(10): 1245-1251.
18. Mayeux R, Stern Y. Epidemiology of Alzheimer Disease. *Cold Spring Harbor Perspectives in Medicine*. 2012;2(8): a006239.
19. de la Monte SM, Wands, JR. Alzheimer's Disease Is Type 3 Diabetes-Evidence Reviewed. *Journal of Diabetes Science and Technology*. 2008; 2(6):1101-1113.
20. Luciano R, Barraco GM, Muraca M, et al. Biomarkers of Alzheimer disease, insulin resistance, and obesity in childhood. *Pediatrics*. 2015;135(6):1074-81.
21. B Ng J, Turek M, Hakim AM. Heart disease as a risk factor for dementia. *Clinical Epidemiology*. 2013;5:135-145.
22. Erkinjuntti T. Vascular cognitive deterioration and stroke. *Cerebrovascular Diseases*. 2007;24(1):189-94.
23. Perrotta M, Lembo G, Carnevale D. Hypertension and dementia: epidemiological and experimental evidence revealing a detrimental relationship. *International Journal of Molecular Sciences*. 2016;17(3): 347.
24. Solomon A, Sippola R, Soininen H, et al. Lipid-lowering treatment is related to decreased risk of dementia: a population-based study (FINRISK). *Neurodegenerative Diseases*. 2010;7(1-3):180-2.
25. Kamat PK, Vacek JC, Kalani A, Tyagi N. Homocysteine Induced Cerebrovascular Dysfunction: A Link to Alzheimer's Disease Etiology. *Open Neurology Journal*. 2015;9:9-14.
26. Hildreth KL, Van Pelt RE, Schwartz RS. Obesity, Insulin Resistance, and Alzheimer's Disease. *Obesity*. 2012;20(8):1549-1557.
27. Peters R, Poulter R, Warner J. Smoking, dementia and cognitive decline in the elderly, a systematic review. *BMC Geriatrics*. 2008;8:36.
28. Miklossy J, McGeer PL. Common mechanisms involved in Alzheimer's disease and type 2 diabetes: a key role of chronic bacterial infection and inflammation. *Aging*. 2016;8(3).
29. Riedel BC, Thompson PM, Brinton RD. Age, APOE and sex: Triad of risk of Alzheimer's disease. *The Journal of Steroid Biochemistry and Molecular Biology*. 2016; S0960-0760(16)30058-9.
30. Laws KR, Irvine K, Gale TM. Sex differences in cognitive impairment in Alzheimer's disease. *World Journal of Psychiatry*. 2016;6(1):54-65.
31. Irvine K, Laws KR, Gale TM, Kondel TK. Greater cognitive deterioration in women than men with Alzheimer's disease: a meta analysis. *Journal of Clinical and Experimental Neuropsychology*. 2012;34(9):989-98.
32. Kanoski SE, Davidson TL. Western diet consumption and cognitive impairment: Links to hippocampal dysfunction and obesity. *Physiology & Behavior*. 2011;103(1):59-68.
33. Gella A, Durany N. Oxidative stress in Alzheimer disease. *Cell Adhesion and Migration*. 2009;3(1):88-93.
34. Dysken MW, et al. Effect of vitamin E and memantine on functional decline in Alzheimer disease: the TEAM-AD VA cooperative randomized trial. *Journal of the American Medical Association*. 2014;311(1):33-44.
35. Denis I, et al. Omega-3 fatty acids and brain resistance to ageing and stress: body of evidence and possible mechanisms. *Ageing Research Reviews*. 2013;12(2):579-94.
36. Dyal SC, Michael-Titus AT. Neurological benefits of omega-3 fatty acids. *Neuromolecular Medicine*. 2008;10(4):219-35.
37. Oulhaj A, et al. Omega-3 fatty acid status enhances the prevention of cognitive decline by B vitamins in mild cognitive impairment. *Journal of Alzheimers Disease*. 2016;50(2):547-57.
38. Miller JW, et al. Vitamin D status and rates of cognitive decline in a multiethnic cohort of older adults. *Journal of the American Medical Association Neurology*. 2015;72(11):1295-1303.
39. Anekonda TS. Resveratrol-A boon for treating Alzheimer's disease? *Brain Research Reviews*. 2006; 52(2):316-326.
40. Evans HM, Howe PR, Wong RH. Clinical evaluation of effects of chronic Resveratrol supplementation on cerebrovascular function, cognition, mood, physical function and general well-being in postmenopausal women-rationale and study design. *Nutrients*. 2016;8(3):E150.
41. Menon VP and Sudheer AR. Antioxidant and anti-inflammatory properties of curcumin. *Advances in Experimental Medicine and Biology*. 2007;595:105-25.
42. Alcohol and Public Health. Centers for Disease Control and Prevention Website. <http://www.cdc.gov/alcohol/faqs.htm>. Accessed March 14, 2016.
43. Characteristics of the Mediterranean Diet. Oldways Website. <http://oldwayspt.org/resources/heritage-pyramids/mediterranean-diet-pyramid/traditional-med-diet>. Accessed March 14, 2016.
44. Omar SH. Cardioprotective and neuroprotective roles of oleuropein in olive. *Saudi Pharm J*. 2010;18(3):111-121.
45. Valls-Pedret C, et al. Mediterranean diet and age-related cognitive decline. *Journal of the American Medical Association Internal Medicine*. 2015;175(7):1094-1103.
46. Mente, A et al. A Systematic Review of the Evidence Supporting a Causal Link Between Dietary Factors and Coronary Heart Disease. *Archives of Internal Medicine*. 2009; 169(7): 659-669.
47. Appel, LJ et al. A Clinical Trial of the Effects of Dietary Patterns on Blood Pressure. *New England Journal of Medicine*. 1997; 336:1117-112.
48. DASH Diet to Lower High Blood Pressure. Medline Plus Website. <https://www.nlm.nih.gov/medlineplus/ency/patientinstructions/000770.htm>. Accessed March 15, 2016.
49. Smith PJ, Blumenthal JA, Babyak MA, Craighead L, Welsh-Bohmer KA, Browndyke JN et al. Effects of the Dietary Approaches to Stop Hypertension Diet, Exercise, and Caloric Restriction on Neurocognition in Overweight Adults With High Blood Pressure. *Hypertension*. 2010;55 (6):1331-8.
50. Morris MC, Tangney CC, Wang Y, Sacks FM, Bennett DA, Aggarwal NT. MIND diet associated with reduced incidence of Alzheimer's disease. *Alzheimer's & Dementia*. 2015;11(9):1007-14.
51. Yaffe K, Barnes D, Nevitt M, Lui LY, Covinsky K. A prospective study of physical activity and cognitive decline in elderly women: women who walk. *Archives of Internal Medicine*. 2001;161(14):1703-8.
52. Vidoni ED, Johnson DK, Morris JK, Van Sciver A, Greer CS, Billinger SA, et al. (2015) Dose-Response of Aerobic Exercise on Cognition: A Community-Based, Pilot Randomized Controlled Trial. *PLoS ONE* 10(7): e0131647.
53. Even Gardening or Dancing Might Cut Alzheimer's Risk. Health Day Website. <http://health.usnews.com/health-news/articles/2016-03-11/even-gardening-or-dancing-might-cut-alzheimers-risk>. Accessed March 15, 2016.
54. Are you Getting Enough Sleep? Centers for Disease Control and Prevention Website. <http://www.cdc.gov/features/sleep/>. Accessed March 14, 2016.
55. Burke SL, Maramaldi P, Cadet T, Kukull W. International Psychogeriatrics. Associations between depression, sleep disturbance, and apolipoprotein E in the development of Alzheimer's disease: dementia.. 2016:1-16.
56. Spira AP, et al. Self-reported Sleep and β -Amyloid Deposition in Community-Dwelling Older Adults. *Journal of the American Medical Association Neurology*. 2013;70(12):1537-1543.
57. Rusanen M, et al. Heavy Smoking in Midlife and Long-term Risk of Alzheimer Disease and Vascular Dementia. *Archives of Internal Medicine*. 2011;171(4):333-339.
58. Meyer JS, Terayama Y, Konno S, Akiyama H, Margisvili GM, Mortel KF. Risk Factors for Cerebral Degenerative Changes and Dementia. *European Neurology*. 1998;39(1):7-16.
59. Dotson VM, Beydoun MA, Zonderman, AB. Recurrent depressive symptoms and the incidence of dementia and mild cognitive impairment. *Neurology*. 2010; 75(1):27-34.
60. Li W, Risacher SL, McAllister TW, Saykin AJ. Traumatic brain injury and age at onset of cognitive impairment in older adults. *Journal of Neurology*. 2016.
61. Stern C, Munn Z. Cognitive leisure activities and their role in preventing dementia: a systematic review. *International Journal of Evidence-Based Healthcare*. 2010; 8(1): 2-17.
62. Bowling A, Grundy E. The association between social networks and mortality in later life. *Reviews in Clinical Gerontology*. 1998;8:353-361.



Understanding how the brain changes as we age is something that all of us should strive to learn more about. As nutrition professionals, we can be aware of how diet, activity, medication use, stress, sleep, and aging itself impact brain health. Here are a few resources to help you get started:

1. [Alzheimer's Prevention](#) – Alzheimer's Foundation of America. Lifestyle choices can impact how the brain and the rest of the body ages. These include nutrition, mental exercise, physical activities and stress management. Find resources and tips to help promote optimum brain health, as well as the [Brain Health: 7 Tips for Successful Aging Infographic](#).

2. [Brain Health As You Age: You Can Make a Difference](#) – Administration for Community Living, U.S. Department of Health and Human Services. Evidence-based resources that can help professionals, older adults, and people with disabilities promote brain health. Includes PowerPoint presentations, educator's guides, handouts and brochures on the following topics: Brain Health Basics, Medicine, Age, Your Brain, Brain Injury, and Dementia.

3. [Global Council on Brain Health](#) (GCBH) – AARP and Age UK. "An independent collaborative of scientists, doctors, scholars and policy experts convened by AARP to provide the best thinking on what people and professionals can do to maintain and improve brain health. GCBH members will debate the latest scientific advancements in brain health research. Their recommendations on what works and what doesn't will provide practical, trustworthy information on brain health." The website includes a fact sheet on the collaboration, information on AARP's brain health survey, and a list of AARP milestones related to brain health.

4. [Healthy Aging](#) – University of California, San Francisco Memory and Aging Center. This website offers a range of resources related to healthy aging and the brain. Topics include: Normal Aging vs. Dementia; Maintaining Your Brain; Memory, and Art; and Creativity. Also includes links to a monthly newsletter, Mind Matters, and information on clinical trials.

5. [Healthy Aging Data Portal](#) – Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services (DHHS). The Healthy Aging Data Portal provides access to CDC data on "a range of key indicators of health and well-being, screenings and vaccinations, and mental health among older adults at

the national and state levels." Data can be viewed by location or indicator, and custom reports and visualizations can be created.

6. [Healthy Brain Initiative](#) – CDC, DHHS. The Healthy Brain Initiative recognizes that "public health's role in maintaining cognitive health, a vital part of healthy aging and quality of life, is emerging." [The Healthy Brain Initiative: A National Public Health Road Map to Maintaining Cognitive Health](#) provides guidance on how "state and local public health agencies and their partners can promote cognitive functioning, address cognitive impairment for individuals living in the community, and help meet the needs of care partners." Other resources include a logic model, research and reports, partnership resources, and surveillance data on cognitive decline from the Behavioral Risk Factor Surveillance System (BRFSS).

7. [Women's Brain Health Initiative](#) – "A charitable non-profit with a mandate to provide education and fund research to combat brain aging diseases that affect women." Find here the Mind over Matter campaign videos and magazines, as well as the Young Person's Cabinet, which has the goal of encouraging millennials to start promoting their brain health now.

CALL FOR VOLUNTEERS WH DPG LEADERSHIP TEAM

The Women's Health DPG is looking for enthusiastic members to serve in volunteer positions for the 2016-2017 membership year! We are seeking individuals for the following positions:

[Resource/Book Reviewer](#)

[Assistant Publications Editor](#)
(small stipend offered)

If you are interested and would like to learn more, please contact info@womenshealthdpg.org.

our vision

*"Optimizing the future of
women's health at all ages."*

WH DPG MEMBER SPOTLIGHT



Shoshana Werber-Flax, MS, RDN, CDN, CPT is a Registered and Certified Dietitian Nutritionist practicing in New York City. She is a certified personal trainer through the National Association of Sports Medicine (NASM), and holds a certificate in Culinary Nutrition from the Natural Gourmet Institute. Shoshana founded **Neuro Nutrition**, a private nutrition counseling practice offering a holistic "food as medicine, non-diet" approach to helping people achieve and maintain their health and wellness goals. She specializes in brain health, pre/post-natal nutrition, and disease prevention.

Why did you choose a focus of brain health for your practice? What drew you to this area of nutrition?

As a migraine sufferer since the age of 10, I have always been interested in the brain. I became more interested in brain health a number of years ago after one of my close family members suffered from a stroke and another relative was diagnosed with dementia. Since then, I have focused on the role nutrition and exercise can play in protecting the brain.

Please share with us a brief career history so we can learn how you arrived to this point.

I started out in corporate wellness, community nutrition and private practice. After working at the Migraine Research Foundation, a health nonprofit where I was able to combine my knowledge and skills in nutrition, marketing and events to raise money for a cause that is close to heart, my focus began to change. At the Foundation,

I communicated with migraine sufferers from around the world and became more interested in the influencing dietary and lifestyle factors. Weight loss and weight management were originally major focuses of my private practice, but my experiences here led me to reevaluate my true passions and interests. Ultimately, both personal and professional experiences led me to the place I'm at right now.

What is the most rewarding part of your job?

I find connecting with people either one-on-one or in a group setting rewarding. I have always loved nutrition education, and knowing that I am helping people improve their overall health and wellbeing is gratifying. As a migraine sufferer myself, I find it extremely fulfilling to help others suffering from a condition that I know all too well find relief.

Why do you think that brain health is an important area for women to focus on?

Women suffer from migraines three times as often as men. Women are also more vulnerable to developing Alzheimer's Disease (AD) than men, with 60% of those diagnosed being women. In addition, women are more often impacted by both migraine and AD even if they are not suffering personally, as they tend to be the caretakers of those who are suffering.

You also work in media and magazines. What is one piece of advice you would offer dietetics professionals interested in working with these outlets?

Be approachable, be prompt in your response if someone reaches out to you, and be willing to say "yes" to opportunities that come your way.

MEMBERSHIP UPDATE – Complimentary Webinars By Maya Feller, MS, RD, CDN, CLC

We completed our annual webinar series! During the 2015-2016 membership year the WH DPG hosted a trio of webinars for members, and offered the opportunity for non-members to participate for a small fee.

Our series started in March, with an outstanding presentation by Melainie Rogers, MS, RD, CDN, CEDRD, with a discussion on Eating Disorders in Pregnancy. Ginger Hultin, MS, RD, CSO gave a thought-provoking and engaging talk on Breast Cancer and Weight Considerations in April. And in May, Judy Simon, MS, RDN, CD, CHES spoke about Nutrition Challenges for Women with Diabetes.

This year we collaborated with the Oncology DPG to offer each other's members complimentary continuing education credits. Oncology members were invited to attend our breast cancer presentation in April, and in return, WH members were welcomed to their May webinar that focused on dietary supplements common in oncology treatment.

Webinars are 1 hour in length and offer 1 CPEU, pending approval from the Academy of Nutrition and Dietetics. Recordings are archived on the WH DPG website, and also available for 1 CPEU.

We look forward to planning the 2016-2017 webinar series, and are always on the lookout for innovative speakers who present up-to-date, evidence-based nutrition information.

If you have ideas for topics, or are interested in being a presenter, please send an email to membership@womenshealthdpg.org. Thank you!

RESEARCH BRIEF: Maternal Choline Supplementation as a Potential Therapy to Reduce Neurological Symptoms and Progression of Down Syndrome and Alzheimer's Disease

By Christine D. Garner, PhD, MS, RD

Strupp BJ, Powers BE, Velazquez R, Ash JA, Kelley CM, Alldred MJS, Myla, Caudill MA, Mufson EJ, Ginsberg SD. Maternal choline supplementation: a potential prenatal treatment for Down Syndrome and Alzheimer's Disease. *Current Alzheimer Research*. 2016;13(1):97-106. doi: <http://dx.doi.org/10.2174/1567205012666150921100311>.

Choline is an essential nutrient for which needs increase during pregnancy. It is essential during fetal development in several ways. Choline phospholipids (phosphatidylcholine and sphingomyelin) are required in large amounts for membranes of new cells and myelination of nerve axons. Additionally, acetylcholine, the choline-derived neurotransmitter, is essential for organization and function of the developing brain. Finally, betaine, an oxidized metabolite of choline, is a methyl group donor for S-adenosyl-methionine, which plays a role in DNA methylation and thus affects the fetal epigenome. To complement the feature article on Alzheimer's Disease (AD) and brain health, this issue's Research Brief will summarize a recently published review by Strupp et al.¹ This article examines maternal choline supplementation as a potential treatment for intellectual disability and brain disorder that accompany Down Syndrome and AD.

Down syndrome (DS), caused by a triplication of chromosome 21 (trisomy 21), is estimated to affect about 6,000 infants born in the U.S. each year.² This translates to 1 in every 691 babies born with DS, an increase from the previous statistic reported of 1 in every 733. People with DS typically exhibit impaired central nervous system function with significant dysfunction in learning and memory, attention, and language and communication skills. Interestingly, these individuals also develop AD pathology much earlier in life than is typical among other adults. Often by their thirties, individuals with DS may have neurodegenerative changes consistent with AD, such as amyloid-beta plaques and degeneration of cholinergic basal forebrain neurons. Mouse models have been created to study DS and AD pathology. In the trisomic mouse model, Ts65Dn, cognitive functioning declines during adulthood with loss of basal forebrain cholinergic neurons in a similar fashion to that observed in DS and AD.

There is high demand for choline during pregnancy. The adequate intake (AI) set for adult women is 425 mg/d, and increases to 450 mg/d for pregnant women.³ The 25 mg/d increase in pregnancy was based on fetal and placental choline accumulation observed in rat models. Importantly, more recent studies indicate that the current AI may not be set high enough. Yan et al.⁴ found that increasing maternal choline intake to 930 mg/d did not increase excretion of choline, thereby indicating that such levels of intake did not exceed metabolic requirements. Furthermore, animal studies have provided functional evidence that current recommendations may be insufficient for optimal cognitive functioning in offspring. Studies in normal rodents indicate that supplementing the maternal diet with choline levels four times higher than that in normal diet show improved memory, spatial cognition, and attention in normal offspring.¹

Spatial Cognition

To examine choline as a possible early intervention on cognitive function in DS, studies have been conducted to assess the effect of maternal choline supplementation using the Ts65Dn trisomic mouse model. In one study, normal disomic and Ts65Dn mice were trained to use cues in a radial arm water maze to locate a hidden escape at the end of one arm. These mice were born to mothers either on a choline-supplemented diet or a control diet. Ts65Dn mice born to choline-supplemented mothers exhibited lifelong functional improvements in the spatial learning involved in this task, and impressively, performed nearly as well as the normal disomic mice. Biologic evidence of these functional improvements was observed in the brain, in that basal forebrain cholinergic neurons, which typically atrophy by around 6 months of age in the trisomic mice, were protected in Ts65Dn offspring of supplemented mothers such that their number and density were higher than those of offspring of mothers on the control diet. The evidence of neurogenesis and neuronal density correlated with the indices of spatial cognition, supporting the concept that maternal choline supplementation may normalize brain neuron structure, thus, impacting function. Further studies have confirmed these proposed effects of maternal choline supplementation on protection of basal forebrain cholinergic neurons as well as the protection of functions that are dependent on these neurons.

Attention and Emotional Reactivity

Children with DS exhibit deficits in sustained attention, which typically worsen with age, particularly in adolescence and adulthood. The Ts65Dn mice exhibit attentional dysfunction in studies consistent with DS such that they miss a higher proportion of brief visual cues and are more often off-task between presentation of cues. To test the effect of additional choline in the maternal diet on attention, mothers of Ts65Dn offspring were put on a control or a choline-supplemented diet. Attention of offspring was tested using a 5-choice serial reaction time task for which one of five ports was briefly illuminated at varying intervals, and the mouse was rewarded for poking its nose into the illuminated port. The performance of the trisomic offspring of choline-supplemented mothers was superior to those born to control-diet mothers; the largest improvement was seen in accuracy of responses with brief cues, which demand more focused attention. The offspring of choline-supplemented mothers also exhibited less emotional reactivity following an incorrect response, indicating potentially improved regulation of emotion or negative affect in trisomic offspring of choline-supplemented mothers. Although little is known about the neural basis for attentional dysfunction or emotion regulation, it was postulated that the reduced density of choline acetyltransferase-immunoreactive basal forebrain neurons observed in these mice might play a role.

Maternal Supplementation and Offspring Choline Metabolism

To better understand the mechanisms by which maternal choline supplementation exerts effects on structural and functional

[Continued on page 10](#)

brain changes in offspring, choline metabolism in the offspring was examined. Choline labeled with an isotope was administered to both Ts65Dn offspring and normal disomic offspring of mouse mothers that had consumed either a control or a choline-supplemented diet. Metabolites were measured in the liver, plasma, and different brain regions. In both the Ts65Dn and disomic offspring of choline-supplemented mothers, greater choline metabolite levels were observed compared to those in offspring of control-diet mothers. Specifically, more metabolites were found in the basal forebrain, hippocampus, cerebellum and liver in both genotypes, but the difference was greater among Ts65Dn offspring. This evidence indicates that maternal choline supplementation had lasting effects on offspring choline metabolism, including increased metabolism in the liver and increased access of choline to the brain. This suggests that increasing choline intake by pregnant mice with a trisomic fetus may help normalize their aberrant choline metabolism, which may also contribute to the improvements in cognitive functioning.

Conclusions

Findings from mouse models indicate that maternal choline supplementation may be a promising avenue for prenatal treatment and prevention of cognitive dysfunction in DS. One caveat, however, is that the Ts65Dn mouse is an imperfect model for comparison in humans because some of its genes that correspond to human genes in DS are not triplicated, and other genes that are

not triplicated in humans with DS are triplicated in the Ts65Dn mouse. Strupp et al.¹ suggest that additional trisomic mouse models should be used next to examine the effects of maternal choline supplementation on offspring. Although each trisomic mouse type will have limitations, together they can provide greater insight on the potential of maternal choline supplementation as a therapy for DS.

Overall, the available evidence indicates that choline supplementation during pregnancy attenuates cognitive and affective alterations in trisomic Ts65Dn mice. Clinical trials will be needed to determine whether similar effects are observed in humans with DS. The authors conclude that increased maternal choline intake by all mothers may be prudent: "In light of growing evidence that all pregnancies would benefit from increased maternal choline intake, this type of recommendation could be given to all pregnant women, thereby providing a very early intervention for DS fetuses."¹

References

1. Strupp BJ, Powers BE, Velazquez R, et al. Maternal choline supplementation: a potential prenatal treatment for down syndrome and alzheimer's disease. *Current Alzheimer Research*. 2016;13(1):97-106.
2. Parker SE, Mai CT, Canfield MA, et al. Updated national birth prevalence estimates for selected birth defects in the United States, 2004–2006. *Birth Defects Research Part A: Clinical and Molecular Teratology*. 2010;88(12):1008-1016.
3. Institute of Medicine. *Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline*. Washington (DC): National Academies Press (US); 1998. Available at <http://www.ncbi.nlm.nih.gov/books/NBK114308/>.
4. Yan J, Jiang X, West AA, et al. Maternal choline intake modulates maternal and fetal biomarkers of choline metabolism in humans. *Am J Clin Nutr*. 2012;95(5):1060-1071.

The advertisement features the logo for 'eat right. Academy of Nutrition and Dietetics' at the top right. The main title 'MNT Provider' is in a large, blue, sans-serif font. Below it, a subtitle reads 'Your source for practice management news'. The text describes the newsletter as free to Academy members and an essential tool for Registered Dietitians (RDs), published monthly with up-to-date information on reimbursement, billing, coding, practice and business management, and healthcare reform issues. A central image shows several overlapping copies of the newsletter, with one clearly visible showing a table of 'The 2013 Medicare Physician Fee Schedule'. At the bottom, it directs readers to visit www.eatright.org/mnt for the latest issue.

NEW ONLINE MEMBER MARKETPLACE PROVIDES OPPORTUNITY FOR WH DPG MEMBERS TO SHINE

The Women's Health (WH) DPG is excited to announce its new Member Marketplace where members can feature new books, webinars, training series, etc. that they have developed.

The WH DPG Member Marketplace exists to promote the sale of WH DPG member products and services to other Academy of Nutrition and Dietetics members (Academy), as well as to the general public.

ELIGIBILITY

Only current WH DPG members are eligible to participate.

FEE

The fee per listing is \$20 per Academy membership year (June 1 - May 31).

Review the complete guidelines or submit a product using our online form. Questions can be directed to publications@womenshealthdpg.org.

Recent news has included extensive coverage on the Zika virus and its associated outbreaks. The Zika virus was relatively unknown in the United States until its recent South American outbreak and subsequent, ongoing spread. As members of the health care community, patients and clients may turn to dietetics professionals with questions about Zika and its consequences. This article will provide you with background information and resources.

Transmission of Zika

The majority of Zika cases are transmitted by bites from infected *Aedes* species mosquitos. This type of mosquito in particular is an aggressive daytime biter, and feeds on humans both indoors and outdoors near dwellings.

Recent findings confirm that it is possible for the disease to be sexually transmitted from a man to his sexual partners, which can occur before, after, or during the symptomatic stages. At this time there are no documented cases of transmission from a woman to her sexual partners.

The most concerning transmission is from mother to child during pregnancy. A pregnant woman can pass the Zika virus to the fetus in utero or around the time of birth. The Centers for Disease Control and Prevention (CDC) recently concluded that Zika virus infection during pregnancy is a cause of microcephaly and other severe fetal brain defects.

Microcephaly is a condition that causes a baby's head to develop much smaller than expected. This condition can be isolated, or can occur in combination with other major birth defects including seizures, developmental delay, intellectual disability, problems with movement and balance, feeding problems, such as difficulty swallowing, hearing loss, and/or vision problems. These problems can range from mild to severe and are often life-long.

To date there have been no confirmed cases of transmission connected with breastfeeding, nor with transmission via blood transfusion in the United States. There have been suspected cases of Zika transmission through blood transfusion in Brazil.

Animals do not appear to be involved in the transmission of Zika at this time.

Epidemic Areas as of May 26, 2016 (Image sourced from the CDC)



Cases in the United States as of June 8, 2016

There have been 691 confirmed cases in the United States, all of which are considered travel-associated cases. There have been 0 reports of locally acquired vector-borne cases.

Symptoms

The incubation period (time from exposure to symptoms) for Zika is not known, but is likely a few days to one week. The most common symptoms are fever, rash, joint pain, and conjunctivitis (red eyes). These symptoms are usually mild, typically lasting for several days to one week. Many people are asymptomatic, and do not realize they are infected.

Protection

The first way to protect yourself is to avoid mosquito bites. Some strategies include:

- Wear long-sleeved shirts and long pants.
- Stay in places with air conditioning and secure screens on doors and windows.
- Sleep under a mosquito bed net.
- Use EPA approved bug repellent with one of the following active ingredients: DEET, picaridin, IR3535, oil of lemon eucalyptus, or para-menthane-diol.
 - These are proven safe and effective, even for pregnant and breastfeeding women. However, insect repellent should not be used on children under 2 months of age.
- Treat clothing and gear with permethrin or purchase permethrin-treated items.

If you feel you have been exposed to Zika, you should continue to avoid mosquito bites, as a bite can infect the mosquito, putting others at risk.

To protect against sexual transmission, it is advised to abstain from sex or use condoms with a male partner for 6 months after symptoms begin. If someone has traveled to an area with Zika but has not developed symptoms, it is recommended to abstain or use condoms for 8 weeks after their return.

If a couple is trying to get pregnant, it is recommended to wait 8 weeks after a woman is exposed to Zika (with or without symptoms) to start having unprotected sex.

Travel

The CDC offers extensive information for travelers including pregnant women on areas with endemic Zika, travel notices, and information on travel to the 2016 Summer Olympics in Brazil. This information can be found on the [CDC website](#), as well as on [Facebook](#) and [Twitter](#). [Email updates](#) are also available.

Testing and Treatment

Preliminary diagnosis is based on clinical symptoms. Locations/dates of travel are also taken into consideration. Serum testing is available to confirm a diagnosis.

[Continued on page 12](#)

Testing may be offered to pregnant women who have traveled to a Zika-infected area 2 to 12 weeks after their return. Women living in a Zika-infected area should be offered screening at initiation of prenatal care and in the middle of the second trimester.

To help monitor the disease, health care providers are encouraged to report suspected cases to state or local health departments. These state or local health departments are then encouraged to report laboratory-confirmed cases to the CDC through ArboNET, the national surveillance system for arboviral disease.

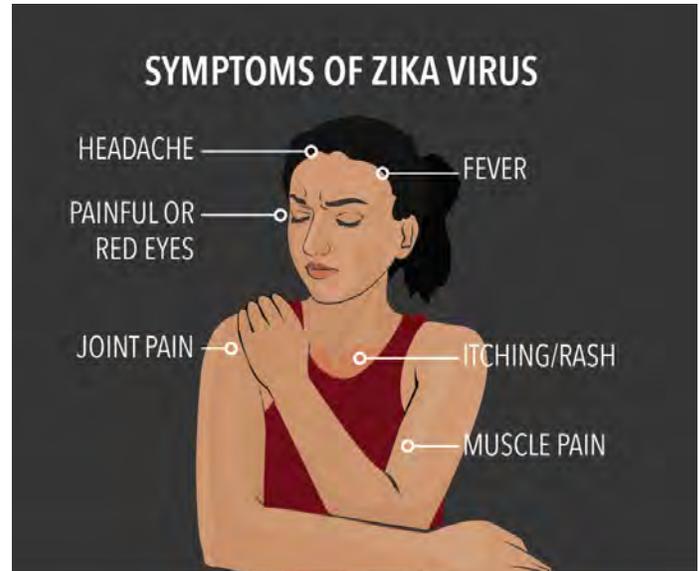
There is currently no specific antiviral treatment for Zika. Treatment recommendations typically include rest, lots of fluids, and medications to reduce pain and fever. Aspirin and other NSAIDs should be avoided until dengue can be ruled out, to reduce the risk of hemorrhage.

References and Resources

Centers for Disease Control and Prevention

- About Zika Virus <https://www.cdc.gov/zika/index.html>
- Fact Sheets and Posters <http://www.cdc.gov/zika/fs-posters/index.html>
- Information for Healthcare Providers <http://www.cdc.gov/zika/hc-providers/index.html>

- Information for Men and Women Thinking about Pregnancy <http://www.cdc.gov/zika/pregnancy/thinking-about-pregnancy.html>
- Information for Pregnant Women <http://www.cdc.gov/zika/pregnancy/index.html>
- Information for Travelers <http://wwwnc.cdc.gov/travel/page/zika-information>



Premiere Education in One Location!

This year's Food & Nutrition Conference & Expo™ will offer a number of professional development opportunities:

- Over 130 educational sessions feature expert speakers and outcomes-based learning.
- Dedicated tracks focus on advances in diabetes, functional nutrition, long-term care and sports nutrition.
- Quality and research symposia, workshops, excursions, Research Posters and Innovations Sessions spotlight research and new trends.

eat right. Academy of Nutrition and Dietetics
FNCE 2016
Food & Nutrition Conference & Expo™
Boston Convention and Exhibition Center | Boston, MA | October 15-18

Cambridge Boston

Learn more at www.eatrightFNCE.org.

HOD Fact Sheet: Outcomes from the Spring 2016 HOD Meeting

House of Delegates

Spring 2016

The House of Delegates (HOD) conducted a virtual dialogue on the Academy’s Second Century and Technological Innovations that Impact Food and Nutrition on April 30 and May 1, 2016. One hundred twenty-five (125) delegates and invited Academy members convened electronically to discuss the two topics over two days.

Saturday, April 30, 2016- Envisioning Our Second Century

Delegates and meeting participants:

Discussed critical events that have impacted the profession

Envisioned the next 100 years

Considered actions to engage members in the Second Century

Feedback from the meeting has been shared with the Academy’s Second Century Team.

Updates to the HOD

The HOD received electronic updates from the following key leaders:

- Evelyn Crayton, EdD, RDN, LDN, FAND, Academy President
- Kay Wolf, PhD, RDN, LD, FAND, Academy Treasurer
- Jean Ragalie-Carr, RDN, LDN, Academy Foundation Chair

Sunday, May 1, 2016- Technological Innovations that Impact Food and Nutrition

Delegates and meeting participants discussed how we can transform all areas of dietetics practice and move the profession forward in a world where rapid advances in technology continually change the way we learn, work and live. Delegates and meeting participants:

**Used the Council on Future Practice's
Technology Change Driver**

**Proposed strategies to help members shift to
higher skills and services**

**Generated ideas of technological innovations
that RDNs and NDTRs can spearhead**

**Discussed ways to empower members to
transform practice through technology**

Outcomes from the Spring HOD Meeting and Dialogue:

As a result of the discussion on technological innovations, one motion was developed and approved by the HOD following the dialogue.

HOD Motion #1: Therefore, be it resolved that the House of Delegates requests that:

- A. The Nutrition Informatics Committee review the input from the Spring 2016 HOD Meeting dialogue, create an action plan and recommendations to address the dialogue objectives, and communicate the plan to the HOD by the Fall 2016 HOD Meeting.
- B. All Academy organizational units identify and promote best practices related to technology and integrate technological innovations that impact food and nutrition into their program of work.
- C. The Academy create a hub on the Academy website where technology resources related to food and nutrition are shared.
- D. The Academy consider highlighting technology in an annual awareness campaign.
- E. The Academy's Second Century Team review the input from the House of Delegates 2016 dialogue and support incorporation of technological advancements into the opportunity areas for the September 2016 Summit and forthcoming innovations projects.

All materials pertaining to the Spring 2016 HOD Meeting can be found on the Academy website at www.eatrightpro.org/resources/leadership/house-of-delegates/about-hod-meetings >Spring 2016 Meeting Materials.

For more information, feel free to contact your delegate: Denise Andersen at dandersster@gmail.com.

WH MEMBER BENEFITS

The **Women's Health Dietetic Practice Group (DPG)** addresses women's health and nutrition issues relative to the life stages unique to women, including preconception, prenatal, postpartum, lactation and menopause.

Members provide education to healthy women of reproductive age and Medical Nutrition Therapy (MNT) to women with reproductive or medical issues, ultimately helping improve the health of women and their children, and reduce pregnancy complications and lifelong disease risk.

The Women's Health DPG offers the following Member benefits:

- **Women's Health Report quarterly digital newsletter** offering cutting-edge professional articles that keep Members up-to-date with the latest research and events in all areas of women's health. Available for CPEUs!
- **Professional WH education and networking sessions** available to members at FNCE®.
- **FREE webinars** on various topics related to women's health. Available for CPEUs!
- **Active conversation with other members** on the **WH DPG website**, electronic mailing list (EML), member forum, **Facebook page**, and **Twitter account** (@WomensHealthDPG).

- **Mentoring Program** pairing students and those new to the profession with seasoned professionals who share a similar focus to encourage skill enhancement, and help map out potential career pathways and future goals.

- **Member Marketplace** to share Member products and services with other WH Members and the public.

- **Award opportunities** offered to Members each year: Excellence in Practice in Women's Health, Emerging Professional in Women's Health, and Outstanding Student in Women's Health.



2015-2016 WOMEN'S HEALTH DIETETIC PRACTICE GROUP LEADERS

Executive Committee

Chair

Heather Goesch, MPH, RDN, LDN

Chair-Elect

Catherine Sullivan, MPH, RDN, LDN, IBCLC, RLC

Past Chair

Lisa Hamlett Akers, MS, RD, IBCLC, RLC

Treasurer

Dawn Balloosingh, MPA, RD, LMNT

Chairs/Coordinators

Social Media Coordinator

Miri Rotkowitz, MA, RD

Membership Chair

Maya Feller, MS, RD, CDN, CLC

Manager, DPG Relations

Susan DuPraw, MPH, RD

Nominating Committee Chair

Maria Bournas, MS, RD

Nominating Chair-Elect

Sarah Borowicz, MS, RDN, LDN

Committee Coordinators

Website/Electronic Mailing List (EML) Coordinator

Leila Shinn

Publications Editor

Kathleen Pellechia, RD

Assistant Publications Editor

Wendy Baier Cartier, RDN

Policy and Advocacy Leader

Lisa Eaton Wright, MS, RDN, LDN

Academy House of Delegates Representative

Denise Andersen, MS, RDN, LD, CLC

Membership Retention Coordinator

Katie Leahy, MS, RDN, LD

Reimbursement Representative

Rita Kashi Batheja, MS, RDN, CDN, FAND

Awards Coordinator

Ginger Carney, MPH, RD, LDN, IBCLC, RLC, FILCA

Research Coordinator

Christine Garner, PhD, MS, RD, CLC

Mentoring Coordinator

Judy Simon, MS, RD, CD, CHES

Please send any questions or comments to
info@womenshealthdpg.org.

