An Upside to Risk Factors
Dr. Elaine Peskind: Lumbar Puncture Myth Buster
William Utermohlen: Self-Portraits of Alzheimer’s
Alzheimer’s disease causes neurons, to die or to become misshapen, and these misshapen brain cells make it difficult for healthy ones to perform their highly specialized responsibilities. Researchers have used animal models to learn more about how neurons become misshapen by Alzheimer’s, but this process has been slow and ineffective because the neurons look alike and are closely bunched together. As the researchers stared into their microscopes, the act of identifying particular neurons was in many ways like an airplane pilot attempting to make out distinct trees while flying over a uniformly green forest canopy. This problem was addressed by a group of Harvard scientists through the development of a new type of mouse, a “Brainbow” mouse. The randomly color-coded nature of a Brainbow mouse’s brain (as shown in the image above) makes identifying neuron changes much easier for scientists. Instead of seeing a homogeneous green canopy of neurons, it is as if researchers see the forest at the peak of autumn, a breathtaking view where individual trees—or neurons—are easily distinguishable from the rest of the forest.
The goal of this small study was to examine the effects of insulin nasal spray on the cognition, daily functioning, processing of sugar in the brain, and cerebrospinal fluid biomarkers in adults with amnestic mild cognitive impairment or Alzheimer’s disease. The study found that insulin treatment improved certain kinds of memory and preserved patients’ ability to function and think clearly. These findings support a longer study of intranasal insulin therapy for patients with amnestic mild cognitive impairment and patients with Alzheimer’s disease.

**Local Research Updates**

**“Intranasal Insulin Therapy for Alzheimer’s and Mild Cognitive Impairment.”**

**“Temporal Relationship between Depression and Dementia: Findings from a Large Community-Based Fifteen-Year Follow-Up Study.”** Archives of General Psychiatry. 2011; 68 (9): 970–7.

Local researchers include Drs. G. Li, L. Wang, S. R. Sholder, M. L. Thompson, E. B. Peskind, W. McCormick, J. H. Brown, J. K. Cross, and E. B. Larson. Depression after the age of fifty is associated with an increased risk of dementia, but the exact meaning of this relationship between depression and dementia is unclear—are people who are depressed more likely to get dementia? Or are people with dementia more likely to become depressed? This study confirmed that late-life depression is associated with an increased risk of dementia but found no evidence that early-life depression increases dementia risk. The findings suggest that late-life depression may be an early sign of dementia rather than a risk that increases one’s chances of developing dementia.

**“Intranasal Insulin Therapy for Alzheimer’s and Mild Cognitive Impairment.”**


Local researchers include Drs. J. B. Shofer, M. L. Thompson, E. B. Peskind, B. K. Walter, T. Postupna, J. L. Pirtle, and S. Craft. This study found that for normal older adults, increased high-intensity physical activity reduces the negative effects of a high-fat, high-sugar diet on amyloid-beta levels in cerebrospinal fluid. Moreover, for adults with mild cognitive impairment (MCI), high-intensity physical activity increases the positive effects of a low-fat, low-sugar diet on amyloid-beta levels in cerebrospinal fluid. These results suggest that adults with no memory problems who engage in high-intensity physical activity are less vulnerable to the brain changes caused by an unhealthy diet and that adults with MCI who pair high-intensity physical activity with a healthy diet are more likely to experience brain-related benefits.

**Alzheimer’s Program Support Fund**

The ADRC’s Program Support Fund helps junior faculty to use their expertise and innovation to pursue promising research studies within the UW ADRC. Your partnership in this Program Support Fund is essential to our continued efforts to find better treatments and a prevention for Alzheimer’s disease. For more information regarding the Support Fund, please contact Susan Martin at 206-346-5791 or sxm25@uw.edu. Checks can be made out to “UW ADRC” and mailed to: VAPSHCS, S-116 6-East, Att: Susan Martin, 1660 S Columbian Way, Seattle, WA 98108. To donate online, visit www.washington.edu/giving/make-a-gift and search for “Alzheimer’s Program Support Fund.”

**SPRING 2012**

**DIMENSIONS**

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Dimensions is produced and published by the UW ADRC.

Hello Readers,

We don’t have a cure. We can’t wind back the clock and give our loved ones their memories. We can’t prescribe a pill that will prevent future generations from developing Alzheimer’s disease. And so dementia may seem inevitable. It may seem that our bodies have spun a web so vast and impenetrable that nothing will stop the emergence and spread of the disease.

That may be true. But in this issue, we look to science, to art, and to love to show us how we might better struggle against the seeming inevitability of Alzheimer’s.

Take for example, “An Upside to Risk Factors,” and “Connectin the Dots: Dr. Gail Li & The Epidemiology of Aging.” In these pieces, we describe the use of risk factors as avenues for studying Alzheimer’s disease from a population perspective. This view of Alzheimer’s research places the emphasis on identifying individuals who may be particularly vulnerable to dementia and on discovering prevention strategies and lifestyle changes that sidestep the harm of risk factors before they can make a negative impact.

The issue addresses the less scientific and more personal aspects of Alzheimer’s disease through art and storytelling. William Utermohlen, a twentieth-century German artist, painted a series of self-portraits throughout his lifetime. His body of work, sampled in this issue, gives us a peek into the intimate changes a person with Alzheimer’s experiences and helps us see one way in which a person with dementia can step forward into the daunting fog of memory loss and maintain some sense of identity. And Gloria Roberts tells how Alzheimer’s affects marriage. Her story is a sad one, but it shows us something even more vast and impenetrable than dementia—it shows us how love can resist the effects of Alzheimer’s.

We hope you enjoy the issue. As always, let us know what you think and if there are other topics, stories, or opportunities you believe we should feature. Our team loves hearing from you.

Happy reading,

James Leverenz, MD

Education Core Director

Lindsey Beach

Dementia Managing Editor
Strokes and Hardening of the Arteries

THE BAD: Research shows that strokes are a major player in age-related cognitive decline and that many older people who suffer from Alzheimer’s disease also suffer from cerebrovascular disease, which is often related to the development of strokes. Moreover, some evidence suggests that the Alzheimer’s disease process may be jump-started by the buildup of cholesterol and other fatty materials within blood vessel walls which then causes hardening of the arteries. Johns Hopkins University researchers examined the relationship between this cholesterol-induced hardening of the artery walls, which is known as atherosclerosis, and two diagnostic measures of Alzheimer’s. Two hundred participants who were cognitively normal at the beginning of the study received medical evaluations and memory testing for an average of 8.7 years before their deaths. The researchers found no correlation between the presence of atherosclerosis or mini-strokes in the brain and the levels of abnormal deposits of plaques and tangles in the brain, which are the definitive signs of Alzheimer’s disease. However, the researchers observed a significant relationship between atherosclerosis and the clinical signs of dementia, that is, poor performance on cognitive testing. Atherosclerosis of brain blood vessels was calculated to be responsible for 34 percent of the dementia seen in this group.

THE GOOD: If the research on the hardening of the arteries is correct, improved control of atherosclerosis could be one way to reduce the risk of Alzheimer’s. Some practical ways of reducing atherosclerosis include eating a healthy, balanced diet; maintaining a healthy weight; and staying physically active. The Johns Hopkins study suggests that reducing one’s exposure to atherosclerosis risk factors, such as high cholesterol, diabetes, and smoking, could be one strategy for reducing the risk of dementia.

An Upside to Risk Factors

The National Institute on Aging (NIA) recently released its 2010 Alzheimer’s Disease Progress Report,* which outlines the latest discoveries and treatment research from centers across the United States. The report is full of seemingly bad news—it explains that high cholesterol, brain proteins, and some foods might contribute to the development of dementia. But with each of these findings, the NIA also offers a hint of good news, because unlike such risk factors as age and genetics, these are risks that can perhaps be decreased through lifestyle choices. NIA scientists are exploring prevention strategies to determine whether mental stimulation, exercise, and dietary supplements can delay or reduce the severity of age-related decline or the onset and progression of disease.

*Find the report at www.nia.nih.gov/alzheimers/publication.

Article adapted from the NIA 2010 Alzheimer’s Disease Progress Report.

Exercise

THE BAD: Brain-derived nerve growth factor (BDNF) is a protein produced by brain cells that is essential for both early brain development and healthy brain functioning in adulthood. BDNF is highly concentrated in a brain area known as the hippocampus, where it supports neuron-connection health and promotes the generation of new neurons. But scientists have noted that the levels of BDNF that are found in serum decline with age. A University of Illinois at Urbana-Champaign study suggests that this loss of circulating BDNF may actually contribute to age-related cognitive decline. In a group of 142 older adults without dementia, older age was associated with reduced levels of BDNF in serum, smaller hippocampal volumes, and poorer memory performance.

THE GOOD: Exercise could be an easy way to address age-related decreases in BDNF and the associated drop in hippocampal volume and memory. Indeed, exercise is known to increase BDNF levels in humans and has been linked to larger hippocampal volumes and better cognitive function in old age. Additionally, many observational studies associate physical exercise with successful cognitive aging. A University of California, San Francisco-led epidemiological study that had 9,344 women age 65 and older (with an average age of 71.6) report their physical activity as teenagers, thirty-year-olds, fifty-year-olds and found that women who were physically active displayed better cognitive performance and less risk of cognitive impairment in late life than those who were inactive. Women who exercised as teenagers had the best outcomes of all, but even women who were sedentary as teenagers but became more active later had better cognitive functioning in late life. These findings suggest that physical activity, particularly in early life, may help reduce the risk of age-related cognitive decline.
High-Fat, High-Carbohydrate Diets

THE BAD: Saturated fats and refined carbohydrates, such as white sugar, are the main ingredients contributing to weight gain in the modern Western diet. These dietary factors also have been associated with cognitive dysfunction and an increased risk of Alzheimer’s. To learn more about how dietary factors affect brain function, scientists at Purdue University fed rats a high-energy diet similar to the one found in Western cultures—high in fats and simple carbohydrates. Rats that were fed this high-energy diet for ninety days performed significantly worse on certain memory tests than rats that were fed a diet containing one-third the fat.

THE GOOD: Given that most of the evidence concerning high-fat, high-carbohydrate diets is based on animal studies, no definitive dietary recommendations are currently possible. However, recent epidemiological evidence suggests a possible association between consuming fish and the mono-unsaturated fatty acids and polyunsaturated fatty acids that are found in red meat, nuts, whole milk products, olives, and avocados and a reduced risk of cognitive decline and dementia. Moreover, in keeping with the dietary advice for lowering cardiovascular and metabolic risks, it is probably wise to consume high levels of fats from fish, vegetable oils, nonstarchy vegetables, and fruits with a low glycemic index; a moderate amount of wine; and few foods with added sugars.

Vitamin D

THE BAD: Studies show that a significant number of older people in the United States and Europe may have vitamin D deficiencies. To determine whether vitamin D plays a role in cognitive health, NIA investigators and an international team of researchers studied a group of 858 people age 65 or older living in Tuscany, Italy. During the six-year study, participants who were severely deficient in vitamin D experienced significantly faster rates of cognitive decline than participants with normal levels of vitamin D. This finding adds to emerging evidence that vitamin D may be important for brain health and functioning and that it may protect neurons from damage.

THE GOOD: Future clinical trials could tell us more directly about the importance of vitamin D. In the meantime, there are a few clear steps we can take to improve our vitamin D intake and to thereby safeguard against some causes of age-related cognitive decline. For instance, vitamin D can be obtained through a diet that is high in fatty fish or through sensible exposure to the sun.

Dr. Elaine Peskind:
Lumbar Puncture Myth Buster

Dr. Elaine Peskind, the director of the UW ADRC Clinical Core, reinvented the way research lumbar punctures, or spinal taps, are done, making them safer and less painful for research participants. But urban legends and myths of pain, meningitis, and paralysis are still associated with the procedure. Dr. Peskind, who has performed more than a thousand lumbar punctures during her work at the UW ADRC, takes a look at a handful of these fables and reveals the truth about research lumbar punctures.

MYTH: Lumbar punctures are really painful.

The discomfort associated with a lumbar puncture seems to vary from person to person. Most people report that the only painful or uncomfortable part of the procedure is a very brief sting they experience when the local anesthetic or numbing medicine is injected. This local anesthetic is similar to the one you would receive at the dentist, and it is used to prevent pain during the lumbar puncture. As the needle for the lumbar puncture is positioned to collect spinal fluid, most people describe the feeling as a pressure sensation. In a study we did at the UW ADRC, we found that, overall, anxiety and pain ratings were low among the research participants who had lumbar punctures. Most people are surprised at how comfortable the procedure is, and occasionally a person will sleep through the procedure.

MYTH: There is a chance that a person could get meningitis by participating in a lumbar puncture.

People cannot develop meningitis from a lumbar puncture. Meningitis by participating in have a lumbar puncture. The spinal cord ends about five inches above the spot where the spinal punctures, or spinal taps, are done, making them safer and less painful for research participants.

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MYTH: If a person gets a lumbar puncture, that person will have a bad headache afterwards.

When doctors perform a lumbar puncture, they puncture a fluid-filled sac that surrounds the brain and spinal cord. Spinal headaches occur when the spinal fluid continues to leak (under the skin) from this puncture. A true spinal headache worsens when a person is sitting or standing and improves when that person lies down. After a lumbar puncture that is conducted for medically necessary reasons, 10 to 30 percent of people develop a spinal headache. However, for research lumbar punctures at the UW ADRC, we use techniques that make the headache rate much lower—less than 1 percent of our subjects report having spinal headaches. This difference is mainly caused by the gauge (thickness) of the lumbar puncture needle and the shape of the needle tip. In the UW ADRC research lumbar punctures, the needle inserted into the sac has a smaller gauge and duller tip than the needles that are commonly used during medically necessary lumbar punctures. This difference means that in a research lumbar puncture at the UW ADRC, the tip of the research needle slides between the fibers of the sac that contains the spinal fluid rather than cutting through it. Because those fibers are not cut and because the hole left by the needle is smaller, the puncture site seals quickly and prevents the spinal fluid from leaking out.

MYTH: If the doctor sneezes while someone is undergoing the procedure, that person will become paralyzed.

The spinal cord ends about five inches above the spot where the lumbar puncture needle is inserted. Because the needle is inserted well below where the spinal cord ends, there is almost no chance of nerve damage or paralysis.

Vitamins and Minerals

Vitamin D: The BAD: Studies show that a significant number of older people in the United States and Europe may have vitamin D deficiencies. To determine whether vitamin D plays a role in cognitive health, NIA investigators and an international team of researchers studied a group of 858 people age 65 or older living in Tuscany, Italy. During the six-year study, participants who were severely deficient in vitamin D experienced significantly faster rates of cognitive decline than participants with normal levels of vitamin D. This finding adds to emerging evidence that vitamin D may be important for brain health and functioning and that it may protect neurons from damage. The GOOD: Future clinical trials could tell us more directly about the importance of vitamin D. In the meantime, there are a few clear steps we can take to improve our vitamin D intake and to thereby safeguard against some causes of age-related cognitive decline. For instance, vitamin D can be obtained through a diet that is high in fatty fish or through sensible exposure to the sun.
In the world of science, epidemiologists are detectives of the big picture. They study health from a population perspective, looking for patterns in large groups of people. By connecting the dots between people, they hope to reveal factors that cause disease or that increase or decrease the risk of disease. Epidemiologists are on the scene when the flu breaks out in Los Angeles; they study the frequency of heart disease in Vietnam; and at the UW ADRC, our own Dr. Gail Li uses the tools of epidemiology to seek out causes, risk factors, and preventions for Alzheimer’s disease.

Epidemiologists like Dr. Li perform their research on a grand scale, searching for health-related patterns in large swaths of people. Thus, in some sense, Dr. Li’s own research trajectory is a fitting one for an epidemiologist because hers is a global career, a pursuit for knowledge that spans continents. Dr. Li’s story begins in Beijing, China, where she was born and raised, completed medical school, and earned a PhD in psychiatric epidemiology. And Beijing was also where she first began sleuthing for epidemiological clues about the development of Alzheimer’s disease.

In the mid-1980s, when Dr. Li was completing her graduate training and taking her first steps into dementia research, most doctors in mainland China were about thirty to forty years behind US doctors in their understanding and diagnosing of Alzheimer’s disease. At that time, the Mini-Mental State Examination, an important yet simple test of memory impairment, was unavailable in Chinese. Dr. Li worked to translate the test and to establish that the translated version worked in Chinese residents. As part of research supported by the World Health Organization and Beijing Medical University, Dr. Li also went door to door in Beijing, administering a survey with residents who experienced significant memory loss. Dr. Li and her colleagues canvassed the city because they were interested in discovering the prevalence and incidence of dementia in mainland China—in the language of epidemiology, prevalence refers
to the number of disease cases in one thousand individuals per year whereas incidence refers to the number of new disease cases in one thousand individuals per year. This door-to-door study resulted in the first major report of dementia in mainland China.

After her groundbreaking work in China, Dr. Li relocated to New York to spend several years as a visiting scholar at Mount Sinai Medical Center. Together with her fellow researchers, Dr. Li sought to learn how factors like a family history of disease or race might increase one’s chances of developing Alzheimer’s disease. In one of these studies, Dr. Li and her colleagues found that Italian and Jewish families had a higher rate of dementia than Puerto Rican or Chinese families.

Next, after time in China and New York City, Dr. Li arrived in Seattle for her psychiatric residency training and geriatric psychiatry fellowship. She then joined forces with the ADRC and Adult Changes in Thought (ACT), a large longitudinal study of dementia and risk factors that is led by Dr. Eric Larson. Since 1994, the ACT study has randomly selected Seattle Group Health Cooperative patients who lack memory difficulties and are sixty-five and older to volunteer for cognitive assessments every two years. Dr. Li became interested in using this epidemiological treasure trove of data to learn more about whether cholesterol was a risk factor for Alzheimer’s disease and whether cholesterol-lowering medications known as statins may have some protective properties. It turned out that there was no explicit link between cholesterol and Alzheimer’s disease, but Dr. Li found that statins appear to reduce the risk of Alzheimer’s, particularly for individuals who took them early in life. She also found that people who had taken statins in their sixties and early seventies had reduced levels of Alzheimer’s brain changes and tangles when their brains were examined during research autopsies.

Dr. Li began studying this connection between statins and Alzheimer’s changes more intensively. She joined Drs. Robert Riekse and Elaine Piskind in conducting a pilot study that compared the effects of two different statins, pravastatin and simvastatin, on the levels of biological markers, or biomarkers, of Alzheimer’s. They found that pravastatin, which is unable to pass from the blood into the brain, did not change the level of Alzheimer’s biomarkers in the spinal fluid. In contrast, participants who took three months of simvastatin, which can pass from the blood into the brain, had reduced levels of the Alzheimer’s biomarker phosphorylated tau. Based on these results, Dr. Li speculated that simvastatin (“Zocor”) might lower some people’s chances of developing Alzheimer’s.

We now know that early Alzheimer’s changes often begin decades before any symptoms appear. If we could find a drug to prevent the disease or slow its progression during this time before symptoms emerge, we could tremendously reduce the suffering of patients and the burden on family and society. The National Institute on Aging was impressed by the evidence provided by Dr. Li and her colleagues that simvastatin could potentially lower the risk of developing Alzheimer’s disease if it is taken earlier in life. They therefore awarded Dr. Li a three-year, $1.2 million grant to perform a larger, more definitive study of the effects of simvastatin on Alzheimer’s disease biomarkers in healthy, middle-aged persons. In this study, which is called the SimBio study, volunteers randomly receive twelve months of simvastatin or placebo (a placebo is an inactive substance identical to the experimental medication but has no pharmacological effect). Unlike conventional clinical trials, the SimBio study uses spinal fluid biomarkers to measure study outcomes rather than examining how the drug affects clinical symptoms. At the end of the study, Dr. Li will compare the people who received simvastatin to the people who received the placebo to see whether there are distinct differences in the levels of such Alzheimer’s biomarkers as tau protein, phosphorylated tau, amyloid beta protein, and brain-derived neurotrophic factor. This study is currently under way and the results will be analyzed in the next few years.

In addition to the SimBio study, Dr. Li is also sticking to her epidemiological examinations of the ACT study data by investigating other risk factors such as the ways in which high blood pressure and high glucose levels may be associated with an increased risk of Alzheimer’s in later life. She is also re-searching whether depression in early life may increase the risk of dementia in later life or whether the depressive symptoms that are commonly seen in later life may be an early symptom of dementia.

When Dr. Li is not researching preventions for Alzheimer’s disease, she is a very busy ballet/soccer mom who enjoys exploring new types of cooking, playing table tennis, and working as a psychiatrist one day a week at the Asian Counseling and Referral Service. The organization is a nationally recognized nonprofit that offers a broad array of services, including mental health treatment to Asian Pacific immigrants, refugees, and American-born residents who live in the King County area of Washington. In addition to providing outpatient psychiatric care to a variety of patients, Dr. Li uses her time at the Asian Counseling and Referral Service to teach UW psychiatry residents about specific mental health issues related to Eastern culture, immigration, acculturation, and the integration of Eastern and Western philosophy in the treatment of mental illness.

In the future, Dr. Li hopes to continue her epidemiological research of Alzheimer’s disease. She hopes that her work with biomarkers in spinal fluid and the ACT study will provide researchers with a way of predicting which people will develop Alzheimer’s based on their profile and risk factors. And Dr. Li hopes that this research will lead to early detection and interventions that can prevent Alzheimer’s years before people lose their ability to think clearly.
Throughout his life, the painter William Utermohlen created work that was noteworthy for its intense psychological scrutiny, particularly in his self-portraits. In 1995, at the age of sixty-two, Utermohlen was diagnosed with Alzheimer’s disease, but he kept on painting. The last of his self-portraits, painted between 1995 and 2001, are therefore unique artistic, medical, and psychological documents. They portray a man who felt doomed by a disease that was encroaching on his mind but who fought to preserve his identity and his place in the world. With perseverance, courage, and honesty, Utermohlen adapted his style and technique to the growing limitations of his perception and motor skills, still attempting to communicate with clarity from within his predicament. Utermohlen died in London on March 21, 2007, but his lifelong dedication to translating psychological self-observation into art allows us the opportunity to see how dementia affected his life, perhaps before the disease was even diagnosed.

Adapted from www.williamutermohlen.org
Making a Greater Investment in Alzheimer’s

By Megan Brooks

In February of this year, the Obama administration announced a commitment of $156 million over the course of the next two years to address Alzheimer’s disease, including making $50 million immediately available to the National Institutes of Health (NIH) for research to identify effective treatments, delay disease progression, and ultimately, prevent Alzheimer’s altogether.

“Alzheimer’s disease is “quickly becoming one of our nation’s most critical health challenges,” Sebelius said. As many as 5.1 million people currently suffer from the disease, and that number “could double or more by 2050,” she said, with a “steep” economic price.

In addition, the fiscal year 2013 budget will include $80 million dollars in new research funding,” US Department of Health and Human Services Secretary Kathleen Sebelius said during a media briefing at the National Press Club. “Altogether, the fiscal years 2012 and 2013 investments total $130 million in new Alzheimer’s research funding over two years, more than a 25 percent increase over the current annual Alzheimer’s research investment,” she said. The initiative also includes an additional $26 million in caregiver support, provider education, public awareness, and improvements in data infrastructure.

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"We can’t wait to confront the growing threat that Alzheimer’s disease poses to families and our nation as a whole. The time for bold action . . . is right now," she said. Sebelius said the investments build on the “historic” National Alzheimer’s Project Act, passed by Congress in January 2011 and signed by President Obama, which calls for an aggressive and coordinated national plan to fight Alzheimer’s disease.

NIH Director Dr. Francis Collins said, “We at the NIH wholeheartedly share the vision expressed [by the administration] and the sense of urgency when it comes to fighting Alzheimer’s disease. With new insights and new technologies, we are getting an increasingly clear picture of our brains. We are getting a far better handle on the molecular basis of Alzheimer’s disease, providing real hope for developing new and targeted approaches to treatment and prevention.”

“Thanks to the new infusion of funds announced today, I think Alzheimer’s research is poised for some great discoveries,” Dr. Collins added. “The research supported by these funds will make a difference in many key areas.” These areas, he said, include, first, application of comprehensive DNA sequencing to identify additional gene variants that play a role in the various forms of the disease and in protecting the disease; second, the development of new cell-based models of Alzheimer’s disease that will enable us to screen hundreds of thousands of molecules for their potential as therapeutic agents; and third, the testing of new therapies in people at high risk for Alzheimer’s disease.”

Adapted from Medscape Medical News.
CURRENTLY ENROLLING RESEARCH STUDIES

Volunteers needed for Alzheimer’s prevention study

The University of Washington Alzheimer’s Disease Research Center is currently investigating the use of a commonly used cholesterol medication, simvastatin, for its potential to prevent Alzheimer’s. Participation consists of nine research visits over one year. Healthy persons between the ages of 45 and 64 without Alzheimer’s disease or memory problems are eligible. Each study participant will have a 50:50 chance of being on the medication, simvastatin, or on placebo (a sugar pill). Participants will be compensated for their time.

For more information call: 1-800-317-5382 or 206-764-2069

University of Washington Alzheimer’s Disease Research Center

Alzheimer’s Association Walk to End Alzheimer’s Saturday, September 16, 2012

Join the Alzheimer’s Association Walk to End Alzheimer’s and unite in a movement to reclaim the future for millions. This walk is the nation’s largest event to raise awareness and funds for Alzheimer’s care, support, and research. Start a team, join a team, or join the UW ADRC Research Biomarkers team today.

Research Biomarkers contact: 206.277.1337

www.alz.org/walk

Bingo Karaoke at the Greenwood Senior Center Last Friday of the month, 7–10 p.m.

It’s the hottest game in town. If you are twenty-one or over, join the Greenwood Senior Center on the last Friday of the month, January through October, for a fun-filled night. Reserve your seats or table now! All tickets must be reserved in advance, either online or by calling the Greenwood Senior Center: 206.297.0875

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If you are interested in learning more about this exciting study please call my office, the study coordinator: 206-616-5550 or 486-330-4664 (full-time) or memoware@uw.edu

University of Washington Alzheimer’s Disease Research Center

University of Washington Alzheimer’s Disease Research Center

Insulin for Memory Loss?

Memory Research Study

You may be eligible to participate in a study examining the effects of insulin (inside or outside the body) on memory. If you answer YES to the following:

• 50 years or older
• MCI or memory concerns
• No other health problems
• Not taking diabetes medications

Participants will receive:

• Memory-related educational materials
• Medical monitoring
• Free breakfast
• Free transportation to clinic

University of Washington Veterans Administration Memory Wellness Program

memorywellness.org • 866-638-8813

Currently enrolling research studies

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University of Washington Veterans Administration Memory Wellness Program

memorywellness.org • 866-638-8813

Gloria’s Story

Gloria Roberts is the community chair of the UW ADRC African American Advisory Board and an active advocate for Alzheimer’s disease research and caregivers. This is her story.

I met Nathaniel at work. He was coming down the hall, and we exchanged hellos. I quickly found that Nathaniel was very down-to-earth and easy to talk to. We started growing closer and closer, and then he asked me to be his lady. I had been nine years since my first husband passed away, and my husband had also passed away, but I told him no: “We’re best friends. I don’t need to cross that bridge.” He asked me again a couple months later, and finally I told him I would give it a shot. It was easy being with Nathaniel, and so we were married in 1997. We had a huge wedding, and we had all of our children participate.

In 2007, Nathaniel was diagnosed with Alzheimer’s disease. There was a part of me that said, “He’s going to be just fine,” because that’s how I could handle it emotionally. But I could see changes in his behavior. He could never find keys. He would go into the kitchen, and when he returned, he would leave the water running. Our conversations changed—he kept repeating himself, asking me the same questions over and over again. His personality had been changing all the time, but it’s all I can do. But sometimes I’m just sitting there, and I’ll burst into tears. I miss my husband. I grieve for the man he used to be. There are times when I think he’s fully present—he may have a day like that, maybe two days, and then he goes back into that other place. I find myself wanting to argue, but it doesn’t do any good.

When my first husband passed I was numb, and I really couldn’t cry, but now I cry all the time. It’s a different kind of grief, and to me it’s deeper, because I can see him, but he’s not there. I truly can’t imagine my future without Nathaniel being in it, but I know that the Alzheimer’s will continue to progress. So I’ve made the decision to stand and to love my husband as he is. That changes all the time, but it’s all I can do.

Gloria’s Story was recorded by StoryCorps in Seattle, Washington on October 14, 2008. This story is adapted from All These Love Stories from StoryCorps by Dougal, Penguin Press 2012. The book is available for purchase at your local bookstore or at www.storycorps.com.
Each participant will have a 50:50 chance of being on prazosin or placebo (an inactive substance) for the first twelve weeks of the study.

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Agitated and disruptive behaviors in people with Alzheimer’s can make the good times fade and the future seem bleak. The UW ARDC is investigating a drug* that could reduce these behaviors and brighten the lives of people with Alzheimer’s disease and family members.

If you are interested in participating please call 206.764.2069 or 800.317.5382.
Additional information is available at uwadrc.org.

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