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FALL / WINTER 2010

From the Director

Commitment to Excellence

Dear Friends and Colleagues:

As I reflect upon the past year, and give thought to the contributions and dedication of my colleagues, staff, and benefactors, one word overwhelmingly comes to mind — commitment. During shifting economic times and a slow recovery from our nation’s recession, our collective efforts made 2010 a banner year for our Center. We raised the bar on Alzheimer’s disease research — receiving international accolades, awards and a significant increase in grant funding — all of which underscore and acknowledge our progress and dedication to the advancement of transformative science.

I would like to start by thanking Dean Robert Grossman and Vice-Dean Vivian Lee of NYU Langone Medical Center, for providing us visionary leadership and championing the mission of our Center; Bruce Silberstein and the Silberstein Foundation for their unwavering support and generosity which nurtures our commitment to funding novel research and providing the best patient-centered Alzheimer’s care; our many benefactors upon whom we rely on for their meaningful contributions; our dedicated scientists who relentlessly pursue innovation in finding new therapies, approaches and treatment options to help the many who suffer from Alzheimer’s. Parkinson’s and related neurodegenerative diseases; our devoted clinicians who provide expert medical expertise to patients and to their families; and my operational staff who manage all the details within the big picture. Last but most importantly, I want to extend my gratitude to our patients, who inspire us and put their trust and faith in us for answers, diagnoses, and treatments.

As we look ahead, our vision for 2011 is to broaden our focus on research and translational medicine, bringing what we learn in the laboratory to the patient’s bedside. Our research network within the Silberstein Alzheimer’s Institute will expand with a plan to form additional collaborations with other groups within the NYU Langone matrix, including the departments of Neurology, Pediatrics, Cardiology and Internal Medicine. For instance, our neuroscientists will be integrating with researchers in Pediatrics, investigating lysosomal storage disorders leading to mental dysfunction and exploring the overlap between developmental neurodegenerative diseases and diseases of aging. As one example of expanding collaborations with the Department of Medicine, our researchers are joining forces with scientists in Cardiology and Geriatric Medicine to study the intersectionality between cardiovascular abnormalities and risk for developing Alzheimer’s disease.

On the clinical front, we will continue to build on our patient-centered clinical capability, further developing newly established specialty clinics, such as our Lewy Body Disease Center — the first of its kind in the New York City area. Additionally, we are expanding our multidisciplinary clinical services. For example, with the addition of Geriatric Medicine we now offer cognitive remediation and a falls prevention program.

As you glance through the following pages, you will read about our committed researchers and clinicians, their collective achievements and groundbreaking progress in an effort to advance scientific discovery for the betterment of humankind.

Best Regards,

Ralph A. Nixon, M.D., Ph.D.
Director
Center of Excellence on Brain Aging

The Silberstein Alzheimer’s Institute Enters Fourth Decade of Research Advancement

As the Silberstein Alzheimer’s Institute celebrates 30 years of pioneering advancement in the field of Alzheimer’s research, we spoke with Bruce Silberstein, President of the Silberstein Foundation, to learn more about the Institute’s founding and obtain his insights and perspectives as well as his vision for the future.

EDITOR: Going back to inception in the early 1970s, could you share with us the tenets upon which the Silberstein Alzheimer’s Institute was founded?

MR. SILBERSTEIN: My mother was diagnosed with Alzheimer’s disease in the ’70s at NYU Medical Center. At that time, there really wasn’t a facility in the country that specialized in research, treatment and caregiver support. However, NYU had an Alzheimer’s Disease Center (ADC), which today is the oldest ADC in the country (supported by the National Institute on Aging) and is part of the Silberstein Alzheimer’s Institute. When my mother was receiving care at NYU, there were two volunteers who served as counselors at the ADC, Emma Shulman and Gertrude Steinberg. Alongside Dr. Steven Ferris, who managed my mother’s care, Emma and Gertrude helped my mother immensely, and worked with us, as a family, to cope with the disease and its eventual progression. Seeing the effect that the disease was taking on my mother was devastating. Yet, my father was so touched by Dr. Ferris’ dedication and the compassionate care she was receiving from Emma and Gertrude, that my father, out of gratitude and gratefulness, donated

Continued on Page 7
Novel Gene-Related Research at Silberstein Alzheimer's Institute Paves Way for New Directions in Alzheimer’s Disease Treatment

Unveiling a discovery offering a new lead for pharmaceutical companies, a collaborative team of researchers from NYU Langone’s Silberstein Alzheimer’s Institute and the Nathan S. Kline Institute for Psychiatric Research (NKI) have discovered how mutations in a gene linked to Alzheimer’s disease may cause the early-onset form of the disease by disrupting crucial cell function. The finding, reported online in the journal Aging, opens the door to developing novel treatments for this form of Alzheimer’s disease and for the more common, late-onset form that develops later in life and affects millions of people worldwide.

The presenilin gene (PS1) is most commonly associated with the early-onset familial form of Alzheimer’s, which runs in families and can strike people in their 30s. The gene was discovered 15 years ago, but until now no one understood how mutations in the gene caused the disease.

The NYU Langone/NKI research team led by Ralph Nixon, MD, PhD, professor in the Departments of Psychiatry and Cell Biology at NYU Langone Medical Center and director of the Center for Dementia Research at the Nathan S. Kline Institute for Psychiatric Research, discovered that the presenilin 1 gene, when it is functioning optimally, performs a crucial house-cleaning service by helping brain cells digest unwanted, damaged and potentially toxic proteins — essential for brain cell survival. However, in its mutated form, the gene fails to help cells recycle these potential toxins, disrupting the recycling process, thereby killing nerve cells — suggesting an explanation for the brain damage evident in Alzheimer’s disease.

“We observed that in skin cells of patients with Alzheimer’s disease caused by presenilin mutations, the ability to break down and reuse normal proteins and to remove potentially toxic damaged proteins and organelles is severely impaired,” said Dr. Nixon, who is recognized in his field for his research efforts and excellence in teaching. He is a New Jersey native and graduate of New York University. Dr. Galvin joins NYU Langone from Washington University School of Medicine where he served as Director of the Memory Diagnostic Center within the Department of Neurology and held two recent positions within the Alzheimer’s Disease Research Center.

At the Barlow Center, Dr. Galvin’s practice focuses on providing state-of-the-art diagnostic evaluations, treatment and rehabilitation for all forms of dementia and cognitive impairment. His approach to treatment includes pharmacological intervention, psychosocial therapy, and the importance of proper nutrition, diet and exercise. Additionally, he leads numerous clinical trials — providing patients and families the opportunity to participate in research studies testing potential treatments; as well as longitudinal and neuroimaging studies for early detection; and psychosocial research for caregivers.

In addition to overseeing clinical operations at the COE and his clinical practice, Dr. Galvin’s laboratory program focuses on the discovery of therapies and treatment options for Alzheimer’s disease and related neurodegenerative disorders. Recognized for his research efforts and excellence in Geriatric Neurology and Psychiatry by the American Academy of Neurology, Dr. Galvin has published extensively in the area of neurodegenerative disorders, dementia and cognitive aging and is the editor of two textbooks on dementia. He also serves on the boards of various organizations.

Specialty Center Established for Diagnosis and Treatment of Lewy Body Disease — First Center of Its Kind in NYC Tri-State Area

The Center of Excellence (COE) on Brain Aging established a specialty center for the diagnosis and treatment of Lewy Body Disease, a multi-system disease involving disturbances of cognition, behavior, sleep and autonomic function. Lewy Body Disease, or LBD, affects an estimated 1.3 million individuals in the United States and is widely misdiagnosed because LBD symptoms closely resemble those of other more commonly known diseases such as Alzheimer’s and Parkinson’s disease.

Lewy Body Disease is named for the scientist Friederich H. Lewy who discovered abnormal protein deposits that disrupt the brain’s normal functioning. Though there is no cure for the disease, early detection can result in effective disease management through the use of non-pharmacological approaches to start, followed by maximizing use of antidepressant medications and SSRIs. Typically, Alzheimer’s patients are given antipsychotic drugs to control behaviors, but patients with Lewy Body Disease can have serious reactions to certain types of these medications including trouble initiating movements and severe rigidity, even muscle breakdown and renal failure.

The LBD Center, headed by Dr. James Galvin, offers a collaborative, patient-focused approach to diagnosis and treatment. Diagnostic evaluations involve both physical and neurological examinations, as well as patient and family interviews, and neuropsychological and mental status tests. The patient’s functional ability, attention, language, visuospatial skills, memory and executive functioning are assessed. Patient care teams include neurologists, neuropsychologists, psychiatrists, internists and geriatric specialists, as well as social workers to provide support and care for caregivers. Innovative programs in cognitive remediation and falls prevention can be tailored to individual patient needs.

The Center also offers a wide array of comprehensive and technologically advanced testing including brain imaging, sleep evaluations and other laboratory studies. Patients have the opportunity to participate in clinical research projects involving imaging, cerebrospinal fluid and other biological markers leading to development of new therapeutics that are not available elsewhere.

The Lewy Body Disease Center is located at the Pearl S. Barlow Center for Memory Evaluation and Treatment at the Silberstein Alzheimer’s Institute, 145 East 32nd Street in midtown Manhattan. Physicians, patients and families needing information may call 212.263.3210 or visit www.barlowcenter.org.
Elkhonon Goldberg receives Psychiatric Research and is a Professor at The Nathan Kline Institute for research at the Center for Dementia mutation which leads to increased levels of Alzheimer’s disease because they have a gene that mimic some of the characteristics of the Alzheimer’s disease. The reason is abnormal using experimental animals nerve cells develop after adulthood. It is thought to be very important because there is been studied before, and this small population of adult-born nerve cells is incorrect appear to be a small population that is important for learning and memory. The nerve cells that migrate to the brain from the hippocampus, an area of the brain that is important for learning and memory. The nerve cells that migrate incorrectly appear to be a small population that develops after maturity. They have been studied before, and this small population of adult-born nerve cells is thought to be very important because there are very few areas of the adult brain where nerve cells develop after adulthood.

Dr. Scharfman will study the reasons why the migration of the adult-born nerve cells is abnormal using experimental animals to mimic some of the characteristics of Alzheimer’s disease because they have a mutation which leads to increased levels of amyloid. Dr. Scharfman conducts her research at the Center for Dementia Research at The Nathan Kline Institute for Psychiatric Research and is a Professor of Child and Adolescent Psychiatry, Psychiatry, Physiology and Neuroscience at NYU Langone Medical Center.

Elkhonon Goldberg Receives Two Noteworthy Awards

Elkhonon Goldberg, Ph.D., has received the honor of being inducted into the foreign membership of Istituto Veneto di Scienze, Lettere ed Arti — an Italian Academy whose aim is “the advancement, dissemination and protection of the sciences, literature and arts.” The purpose of the academy is to bring together outstanding figures from the world of scholarship to promote cultural, social and economic life. The institute is composed of two classes: the class of mathematical, physical and natural sciences, and the class of ethical sciences, literature and arts. Dr. Goldberg’s election was formalized by a decree issued by the Italian Ministry for Heritage and Cultural Activities at the closing ceremony of the Institute’s academic year.

Additionally, during the annual meeting of the International Neuropsychological Society in Krakow, the Copernicus Prize 2010 was awarded to Dr. Goldberg by the Polish Neuropsychological Association. This award honors the contribution made by an individual to interdisciplinary dialogue between neuroscience and neuropsychology in the best tradition of Nicholas Copernicus.

An author, educator, scientist and clinician, Dr. Goldberg is renowned for his clinical work, research, writings and teaching in neuropsychology and cognitive neuroscience. He serves as Clinical Professor of Neurology at NYU School of Medicine.

Einar Sigurdsson Receives Award for Drug Discovery Research for Frontotemporal Dementia

The Alzheimer’s Drug Discovery Foundation (ADDF) and the Association for Frontotemporal Dementias (AFTD) announced recently the recipients of their third annual research award, Frontotemporal Dementia Drug Discovery Program. The ADDF/AFTD program awarded $300,000 to academic scientists on the cutting edge of research for FTD. The COE’s Einar M. Sigurdsson, Ph.D. was one of three researchers worldwide to receive this prestigious honor for his project, “Passive Immunotherapy for Frontotemporal Dementia.” FTD is a debilitating form of dementia characterized by extreme changes in behavior, personality, language and movement.

The grant to Dr. Sigurdsson will provide $100,000 over one year, and will support studies on the therapeutic potential of monoclonal tau antibodies in a mouse model of frontotemporal dementia.

The National Alliance for Caregiving and MetLife Foundation Honors NYU Caregiver Intervention Program

Representatives from leading aging organizations and agencies from around the nation convened in St. Louis, MO to honor the 2010 winners of the National Family Caregiving Awards, sponsored by the National Alliance for Caregiving with support from MetLife Foundation. The programs were recognized for their innovation, effectiveness and response to caregiver needs in their communities. This year’s winners included the NYU Caregiver Intervention program, an evidence-based intervention program at the COE headed by Mary S. Mittelman, Dr.P.H. The focus of the program is to improve the well-being of caregivers of those with Alzheimer’s disease by mobilizing the support of naturally existing family networks, improving caregiving skills and providing the opportunity for ongoing counseling and support. Dr. Mittelman is Director of the Psychosocial Research and Support Program at the COE, and Research Professor in the Department of Psychiatry at NYU Langone Medical Center. She also leads the Education and Psychosocial Cares of the NYU Langone Alzheimer’s Disease Center.
Researchers at the Silberstein Alzheimer’s Institute Accelerate Pace of Discovery for Early Detection and Treatment Options

Today, research in the field of Alzheimer’s is taking place against the backdrop of an urgent demographic reality. Recent data suggests that more than 5 million Americans have Alzheimer’s disease and that number is rapidly increasing as baby boomers reach their golden years.

Responding to this impending health crisis, The National Institutes of Health (NIH) has established an extensive program of AD research led by the National Institute on Aging (NIA). This program applies the expertise of many scientific disciplines in an attempt to answer difficult questions about what causes AD, how it can be diagnosed early and accurately, how it can be treated, and how it might ultimately be prevented. The scientific accomplishments highlighted in the next few pages by researchers within the COE’s Silberstein Alzheimer’s Institute have been made possible through significant funding received by the NIH/NIA, as well as by the Alzheimer’s Association, and other noteworthy associations and private benefactors.

Olfaction Research Provides New Clues

Olfactory dysfunction as it relates to Alzheimer’s disease and other neurodegenerative disorders has been clearly documented over the years. There is an established relevance between detection and distinction of odors and AD patients since smell activates a large portion of the brain. Problems with olfaction often start before other classical signs of AD are noticed in patients. However, research linking olfactory sensory loss to specific AD pathologies is lacking. Ongoing research being conducted at the COE on Brain Aging, with researchers at The Nathan S. Kline Institute for Psychiatric Research (NKI) and across the NYU Langone network, reveals the significance of olfactory disturbance and its link with early diagnosis and treatment management.

A study published by COE investigators, in the Journal of Neuroscience, links a loss of smell function in Alzheimer’s disease model animals with amyloid-ß protein accumulation in the brain, a distinguishing hallmark of Alzheimer’s disease. The formation of amyloid plaques and neurofibrillary tangles are believed to contribute to the degradation of the neurons and circuits in the brain and the subsequent symptoms of AD. In this study, COE scientists used genetically engineered mice, which developed high levels of amyloid-ß burden in their brains, reflecting a progressive Alzheimer’s disease pathology similar to humans. They found that Alzheimer’s disease amyloid pathology occurs first in a region of the mouse brain responsible for smelling — which is directly above their noses. This pathology also coincided with the animals having abnormal abilities to smell. The mice with a high concentration of amyloid in their brains had to sniff odors longer to “learn” them than mice with less amyloid. They also had problems differentiating between odors.

“Olfaction research presently taking place at the COE is novel in its approach because for the first time in a dementia-related study, in this specific sensory field, there is collaboration between olfactory investigators, psychiatrists, and AD researchers bringing to bear multi-faceted intelligence, insight and expertise,” noted co-author of the study, Daniel W. Wesson, PhD, NYU School of Medicine and Nathan S. Kline Institute.

Healthy Older Adults with Subjective Memory Loss May Be at Increased Risk for Mild Cognitive Impairment and Dementia

Subjective Cognitive Impairment (SCI), the earliest sign of cognitive decline, is marked by situations such as when a person recognizes they can’t remember a name like they used to or where they recently placed important objects the way they used to. Studies have shown that SCI is experienced by one-quarter and one-half of the population over the age of 65. A new study, published in the journal, Alzheimer’s & Dementia, finds that healthy older adults reporting SCI are 4.5 times more likely to progress to the more advanced memory-loss stages of mild cognitive impairment (MCI) or dementia than those free of SCI.

The long-term study completed by researchers at the COE on Brain Aging tracked 213 adults with and without SCI over an average of seven years, with data collection taking nearly two decades. Further cognitive decline to MCI or dementia was observed in 54 percent of SCI persons, while only in 15 percent of persons free of SCI.

“This is the first study to use mild cognitive impairment as well as dementia as an outcome criterion to demonstrate the outcome of SCI as a possible forerunner of eventual Alzheimer’s disease,” said Barry Reisberg, MD, professor of psychiatry, NYU School of Medicine. According to the study authors, scientists and physicians can now target the prevention of eventual Alzheimer’s disease in the SCI stage, beginning more than 20 years before dementia becomes evident.
Brain Plaques May Explain Higher Risk of Alzheimer’s Based on Mother’s History

Recent developments in neuroimaging have greatly widened our knowledge on how AD progresses. Brain imaging techniques have aided us in understanding that damage in the brain begins early on, before any clinical symptoms are evident and this process develops gradually over the years. Unfortunately, by the time an individual is diagnosed, the disease is well-established. New imaging methods being developed at the COE are being used as powerful research tools and may soon prove to be invaluable diagnostically among pre-symptomatic individuals.

A family history of Alzheimer’s disease is one of the biggest risk factors for developing the memory-robbing disease. Now, an international collaboration led by COE researchers has found the likely basis for this heightened familial risk — especially from the maternal side. Aided by a new version of a brain-scanning technique, the researchers discovered a far greater number of protein clumps linked to the disease among healthy adult children of parents with Alzheimer’s disease compared to counterparts with no family history of dementia. The average increase in these clumps, amyloid-beta plaques, was particularly striking among study volunteers whose mothers had been diagnosed with the disease. The plaques appeared throughout most regions of the brain.

The findings, published in the Proceedings of the National Academy of Sciences, may help explain why a family history is such a big risk factor for the brain disease — individuals with an affected parent have a four- to ten-fold greater risk than those with no family history.

The new study combines positron emission tomography (PET) with a fluorescent dye called Pittsburgh Compound B (PiB) that highlights brain amyloid plaques, enabling researchers to actually see the deposits. The dye attaches to plaques and acts like a temporary beacon to highlight their presence during a PET scan.

New Key Factor Identified in the Development of Alzheimer’s Disease

Inheritance of an extra copy of the gene β-amyloid precursor protein, APP, in individuals with Down syndrome leads to the inevitable development of early onset Alzheimer’s disease, known to be linked to the deposition of Amyloid β peptide or Aβ in the brain. However, a new study published online by Proceedings of the National Academy of Sciences identifies βCTF, a small protein found in APP, as a novel factor for the development of Alzheimer’s disease-related endosome abnormalities, which have also been tied previously to the loss of brain cells in Alzheimer’s disease.

“Identifying people at risk for Alzheimer’s disease is the necessary first step in developing preventive therapies,” said co-author Mony de Leon, EdD, professor, Department of Psychiatry at NYU Langone and lead investigator of this study. “There is a great effort underway to find early markers of disease, before symptoms appear, so that therapeutic approaches will one day delay or ultimately prevent this disease.”

Damaged Protein Identified as Early Diagnostic Biomarker for Alzheimer’s Disease in Healthy Adults

COE researchers have found that elevated cerebrospinal fluid levels of phosphorylated tau231 (P-tau 231), a damaged tau protein found in patients with Alzheimer’s disease, may be an early diagnostic biomarker for Alzheimer’s disease in healthy adults.

The study, published online by Neurobiology of Aging, shows that high levels of P-tau 231 predict future memory decline and loss of brain gray matter in the medial temporal lobe — a key memory center. Prior studies found the medial temporal lobe to be the most vulnerable brain region in the early stages of Alzheimer’s disease, accumulating damaged tau proteins in the form of neurofibrillary tangles. Tangles are one of the signature indicators of Alzheimer’s disease, in addition to beta amyloid plaques.

“Our results show for the first time that elevated levels of P-tau 231 in normal individuals can predict memory decline and accompanying brain atrophy, suggesting that P-tau 231 has the potential to be an important diagnostic tool in the pre-symptomatic stages of Alzheimer’s disease,” said lead author of the study Lidia Glodzik, MD, PhD, assistant research professor, Department of Psychiatry at the Center for Brain Health at the COE.

New imaging methods being developed at the COE are being used as powerful research tools and may soon prove to be invaluable diagnostically among pre-symptomatic individuals.

Endosomes are membrane compartments in cells that support cell survival by absorbing outside nutrients and are crucial in neuronal functions. In Alzheimer’s disease, endosome abnormalities are the earliest neuropathologic features to develop, appearing even earlier in cases where one of several major genetic risk factors for the disease is inherited. Endosomes are also suspected sites of Aβ production in the cells.

“Given that brain pathology begins to accumulate years ahead of memory problems in Alzheimer’s disease, our findings are intriguing,” says Lisa Mosconi, PhD, research assistant professor of psychiatry at NYU Langone and lead investigator of this study. “There is a great effort underway to find early markers of disease, before symptoms appear, so that therapeutic approaches will one day delay or ultimately prevent this disease.”

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In the study, using the cells from individuals with Down syndrome that are genetically predisposed to developing Alzheimer’s disease, we showed that elevated levels of βCTF, independent of Aβ, cause a specific pattern of endosome defects with similar pathology of brain cells in Alzheimer’s disease,” said Ying Jiang, PhD, lead author and clinical instructor in the Department of Psychiatry at NYU Langone Medical Center. “Our research was successfully able to pinpoint that βCTF causes Alzheimer’s disease-related endosome defects and that we could successfully reverse these endosome defects by lowering βCTF levels in the cells.”

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A Story of Hope

Medical misdiagnosis is a persistent problem in the United States. Many research studies have demonstrated the frequency of medical misdiagnoses, detecting frequent clinical errors and misdiagnoses, with some rates as high as 47 percent. For George Kaplan*, continual misdiagnoses over the course of nine years and several clinicians resulted in deterioration of his medical condition, poor quality of care, anxiety and caregiver stress.

In 2001, Mr. Kaplan began having violent nightmares — he became aggressive and abusive in his sleep — screaming, throwing, using foul language. His wife realized that this behavior was completely out of the ordinary for someone who was typically a calm, gentle individual. Immediately, she took her husband to see a neurologist, who ran a few tests on Mr. Kaplan and then shrugged and told him that there was really nothing wrong. Over time, the nightmares continued and other symptoms emerged, such as forgetfulness and decreased organizational skills. Mrs. Kaplan then decided to seek a second, third, and as it would end up being, several medical opinions for her husband — but unfortunately, none of the clinicians they consulted with considered Mr. Kaplan’s condition to be serious. They would play down his illness, stating that this may be the onset of Alzheimer’s disease, and dismiss his violent dreams. However, clonazepam, an anti-anxiety drug, was prescribed for his sleep disorder and treatment of dreams. Years passed, and in 2008, Mr. Kaplan began having movement issues — his gait was shuffled, he began slopping, there was slight slurring of speech and shaking of hands. At this point, again, the Kaplans went to see a specialist who told Mr. Kaplan that the movement issues were related to his dementia. Mrs. Kaplan thought otherwise.

It wasn’t until Mr. Kaplan came to see Dr. James Galvin at the Pearl Barlow Center for Memory Evaluation and Treatment in May, that George Kaplan was accurately diagnosed with Lewy Body Disease (LBD), which affects an estimated 1.3 million individuals and their families in the United States. Presently, the disease is misdiagnosed 80 percent of the time because LBD symptoms closely resemble those of other more commonly known diseases such as Alzheimer’s and Parkinson’s disease. The effects include a degradation of cognitive functioning, similar to Alzheimer’s disease, or a degradation of motor control, similar to Parkinson’s disease. Lewy Body Dementia is the second most common form of dementia after Alzheimer’s disease. In degenerative dementias, nerve cells die over time, causing a progressive decline in the ability to think, reason and remember. In Lewy Body Dementia, microscopic protein deposits (Lewy bodies) are found in the dying nerve cells disrupting the brain’s normal function causing gradual deterioration.

Though Mrs. Kaplan had taken an active role in her husband’s condition since the onset, it was when she first met Dr. Galvin at a Manhattan support group, that she realized her husband had been chronically misdiagnosed for all these years. “As Dr. Galvin was explaining Lewy Body Dementia to the audience, I sat there in amazement. I thought to myself, he is talking about my husband. This is what he has.” She immediately made an appointment to see Dr. Galvin and was astonished at how much she and her husband learned from their initial consultation. “He took the time to discuss my husband’s case with me privately and then spoke with both of us together. He explained my husband’s condition to us in a compassionate, kind and comforting manner. More so, he was expertly knowledgeable and spot-on,” says Mrs. Kaplan. After nine long years, their life took a different turn. Now, knowing the disease they are fighting, understanding how it affects them, and having a guided treatment plan, they are able to face the future armed with facts instead of fragile with fear.

“When I am alone, it becomes a bit depressing,” says Mrs. Kaplan softly and a bit tearful. Married happily for 54 years, Mrs. Kaplan describes the future as “a journey for us to endure together. I know how this story will end for us, but I keep having hope that Dr. Galvin and NYU will find a treatment for my husband that will help him with this disease.” Advice that Mrs. Kaplan would share with other caregivers in a similar situation: “The day you give up hope, is the day you stop living. Choose to live today. Enjoy your time together. This is a journey, but remember to plan for the long term, the eventual destination.”

Whether reading a novel, watching a game of baseball, playing cards or listening to Gershwin, Mr. Kaplan lives each day appreciating the gifts of family and friends who have provided extraordinary support, love and care.

* For patient confidentiality purposes, names have been changed.

Winter Crisp

Filling
• 1/2 cup of sugar
• 3 tablespoons all-purpose flour
• 1 teaspoon lemon peel, grated
• 3/4 teaspoon lemon juice
• 5 cups of apples, unpeeled, sliced
• 1 cup of cranberries

Topping
• 2/3 cup of rolled oats
• 1/3 cup of brown sugar, packed
• 1/4 cup of whole wheat flour
• 2 teaspoons ground cinnamon
• 1/2 teaspoon soft margarine, melted

1. Prepare filling by combining sugar, flour, and lemon peel in medium bowl. Mix well. Add lemon juice, apples, and cranberries. Stir to mix. Spoon into 6-cup baking dish.
2. Prepare topping by combining oats, brown sugar, flour, and cinnamon in small bowl. Add melted margarine. Stir to mix.
3. Sprinkle topping over filling. Bake in 375°F oven for approximately 40–50 minutes or until filling is bubbly and top is brown. Serve warm or at room temperature.

Yield: 6 servings
Serving Size: 1, 3/4-inch by 2-inch piece

Each Serving Provides:
Calories: 252 / Total Fat: 2 g / Saturated Fat: less than 1 g / Cholesterol: 0 mg / Sodium: 29 mg / Total Fiber: 5 g / Protein: 3 g / Carbohydrates: 58 g / Potassium: 225 mg

Source: www.nhlbi.nih.gov
The Importance of Safety-Proofing Your Home

Is your home a safe environment to support the needs of a person with Alzheimer’s disease? In order to provide a secure home area when caring for an Alzheimer’s patient, it is necessary to pay attention to certain risk factors and safety hazards that could be detrimental to the well-being of your loved one. Following are a few measures that may be beneficial to caregivers as you evaluate your home environment and implement safety-proofing. Remember, it is important to frequently re-examine home safety measures to protect the one you love from a dangerous mishap or tragic accident.

Improving Safety Within Your Home

- Be sure to always work with fire extinguishers, smoke detectors and carbon monoxide detectors in the house. Test them regularly.
- Lock or disguise hazardous areas. Cover doors and locks with a painted mural or cloth. Use swinging or folding doors to hide entrances to the kitchen, stairwell or garage.
- Install locks out of sight. Place deadbolts either high or low on exterior doors to make it difficult for the person to wander out of the house.
- Remove locks in bathrooms or bedrooms so the person cannot get locked inside.
- Use child-proof locks and door knob covers to limit access to places where knives, appliances and poisonous cleaning fluids are stored.
- Enroll the person in MedAlert + Alzheimer’s Association Safe Return®, a 24-hour nationwide emergency response service for individuals with Alzheimer’s or related dementia that wander or who have a medical emergency.
- Beware of Dangerous Objects and Substances
  - Use appliances that have an auto shut-off feature.
  - Install a hidden gas valve or circuit breaker on the stove so a person with dementia cannot turn it on. Or, consider removing the knobs from the burner.
  - Store grills, lawn mowers, power tools, knives and firearms and cleaning products in a secure place.
  - Discard toxic plants and decorative fruits that may be mistaken for real food.
  - Remove vitamins, prescription drugs, sugar substitutes and seasonings from the kitchen tables and counters. Medications should be kept in a locked area at all times.
- Supervise the use of tobacco and alcohol. Both may have harmful side effects and may interact dangerously with some medications. A person with dementia who smokes should not be left alone; a forgotten cigarette left burning could start a fire.

Avoid Injury During Daily Activities

- Watch the temperature of water and food — it may be difficult to tell the difference between hot and cold.
- Install walk-in showers and grab bars in the shower or tub and at the edge of the vanity to allow for independent, safe movement.
- Add textured stickers to slippery surfaces. Apply adhesives to keep throw rugs and carpeting in place — or remove rugs completely.

Adapt to Vision Limitations

- Use contrasting colored rugs in front of doors or steps to help the individual anticipate staircases and room entrances. Avoid using a dark-colored rug because it may appear to be a “hole.”
- Remove throw rugs altogether to reduce the likelihood of tripping.
- Use night lights in hallways, bedrooms and bathrooms.

Source: Alzheimer’s Association. For more information visit www.alz.org.

CAREGIVER TIPS

The Silberstein Alzheimer’s Institute Enters Fourth Decade

Continued from Page 1

a significant sum to NYU so that countless others in our position may also be helped.

A few years later, research on Alzheimer’s began to take flight at NYU and a working group of accomplished researchers was assembled. Through funding provided by our family, the William and Silvia Silberstein Institute for Aging and Dementia was formed — a much smaller operation than what the Institute for Aging and Dementia was formed. Through the Silberstein family’s involvement at NYU Langone in 2004, I desperately wanted to continue reaching accomplishments of the Institute, alongside a nationally recognized caregiver intervention program is a key initiative and we continue to play a pioneering role in to eradicate the disease. If we are successful, then the Institute will have accomplished its mission. In the shorter term, I want us to continue playing a pioneering role in finding fine treatments and slowing disease progression. Additionally, the caregiver intervention program is a key initiative and we must continue to help the many caregivers who suffer alongside their loved ones.

EDITOR: Today, the Institute plays a leadership role in the global sphere when it comes to research related to Alzheimer’s disease. How does that make you feel?

MR. SILBERSTEIN: A great sense of satisfaction combined with remorse. We have made great strides in understanding the disease and providing patients ways in which to manage their affliction and slow disease progression. But we have not found a cure. I have a brother who suffers from Alzheimer’s, Parkinson’s disease and Lewy Body Dementia, and I can’t help him. I find that criminal. But, we have to forge ahead. Today, with science moving at such a fast pace due to technology and the sharing of information, I believe we are closing the gap.

EDITOR: Where do you see the Institute headed in the long-term? Your vision?

MR. SILBERSTEIN: In the long term, quite frankly, I hope the Institute is no longer needed. Let me expand on that — our goal is to eradicate the disease. If we are successful, then the Institute will have accomplished its mission. In the shorter term, I want us to continue playing a pioneering role in finding fine treatments and slowing disease progression. Additionally, the caregiver intervention program is a key initiative and we must continue to help the many caregivers who suffer alongside their loved ones.

EDITOR: How has the mission of the Institute evolved over time?

MR. SILBERSTEIN: Since the 1970s, NYU Langone has played a pioneering role in understanding the origins of Alzheimer’s disease and developing novel treatments and therapies. Initially, our mission was to support research programs. Today, we are a world-renowned research institute and we offer a comprehensive clinical care center for memory evaluation and treatment alongside a nationally recognized caregiver support program.

EDITOR: What is your involvement with the Institute?

MR. SILBERSTEIN: Upon my father’s death in 2004, I desperately wanted to continue the Silberstein family’s involvement at NYU Langone Medical Center. I am committed to working closely with Dr. Nixon in providing funding to increase the public’s awareness of the disease and further expanding our research and clinical capabilities as well as caregiver support programs. In some small way, I hope I have helped to shape the Institute into what it is today.

EDITOR: What would you say are the Institute’s major accomplishments?

MR. SILBERSTEIN: The Silberstein Alzheimer’s Institute has always been at the forefront of research advances — from advancing the scientific community’s understanding of the genetic and molecular structure of the disease to developing state-of-the-art brain imaging and clinical assessment techniques. Our investigators continue to be top-ranked in the areas of clinical treatment, early diagnosis, genetic and molecular causes and drug discovery.

In 2007, in order to bring our research directly to the patient, we inaugurated the Pearl Barlow Center for Memory Evaluation and Treatment providing an integrated approach to the care of memory impairment and age-related brain disorders. Later, in 2008, NYU Langone recognized the far-reaching accomplishments of the Institute, under the leadership of Dr. Nixon, and we evolved into being part of what is now the Center of Excellence on Brain Aging.

The Silberstein Alzheimer’s Institute Enters Fourth Decade

A publication of NYU Langone Medical Center | Center of Excellence on Brain Aging

Source: Alzheimer’s Association. For more information visit www.alz.org.
I have an aging parent who seems to be experiencing memory loss and lack of recall. How does one differentiate normal aging-related memory issues versus Alzheimer’s disease?

I’m very glad you asked this question, because quite often, this issue arises and individuals are uncertain of how to proceed. It is normal to be concerned because the first symptoms of Alzheimer’s disease mimic the normal aging process. First, let me say that if you have any apprehension regarding your parent’s health, you should visit his/her physician as soon as possible. Early diagnosis provides the best opportunity for treatment. If cognitive decline can be slowed through treatment, a patient’s quality of life can be vastly improved and independence prolonged. In order to help distinguish the early warning signs of Alzheimer’s from age-related memory loss, the Alzheimer’s Association has issued the following guidelines, which we, as physicians, strongly endorse:

- Memory changes that disrupt daily life (e.g., forgetting recently learned information or important dates)
- Challenges in planning or solving problems
- Difficulty completing everyday tasks
- Confusion with time and place
- Trouble understanding visual images and spatial relationships
- Poor or decreased judgment
- Problems with words in speaking or writing
- Misplacing things and having trouble retracing steps
- Withdrawal from work or social activities
- Changes in mood/personality

For more information on these warning signs, I suggest you visit www.alz.org/10signs, or please call the Barlow Center at 212.263.3210 to schedule a memory evaluation with one of our expert neurologists.

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Clinical Trials
Presently, at the COE, a very active clinical trials program exists with numerous trials in progress. To participate or inquire about clinical trials, please visit our website (http://aging.med.nyu.edu/) or call our Clinical Trials Coordinator at 212.263.5708.