New Ways to Diagnose Alzheimer’s Disease

Over the past 25 years there have been few changes in the diagnosis of Alzheimer disease (AD) even though our knowledge of the disease and its progression has grown. For example, the current criteria have age cutoffs (between 40 and 90 years of age), but we know now that many people living into their 90’s are at risk for AD. Also, the current criteria do not include use of supporting information from genetic studies or biomarker results. Recent research has drawn attention to using images of the brain and of the hippocampus (the place in the brain where memory consolidation occurs) to measure changes that may mark the early stages of the disease.

New criteria have been proposed to integrate the biological and epidemiological information we now have. The diagnosis of “all cause dementia” is similar to the current diagnosis in that it focuses on problems in social or occupational function (i.e. every day activities), that are a clear decline from a previous level and are due to cognitive loss. However, unlike current criteria for dementia, memory impairment may be apparent, but is not required. The criteria for the clinical diagnosis of the specific condition of Alzheimer disease also includes gradual onset of problems. Probable AD would be defined by evidence of clinical decline which means worsening noticeable by families or doctors. However, if this type of information is not available, (such as when there is no way to be sure about the previous ability), biomarkers including genetic markers could provide support for the diagnosis. This approach suggests that we have growing confidence in biomarkers (such as brain scans) as an indicator of specific disease.

Diagnostic criteria were also proposed for symptomatic pre-dementia known as Mild Cognitive Impairment (MCI), consisting of 4 basic elements: 1) Concern about any cognitive change, not just memory; 2) poor performance in any cognitive area including but not exclusively memory; 3) relatively normal day to day function and 4) the absence of dementia. MCI cases could be

Ask the Expert: Judith Neugroschl, M.D.

Q: Do any of the prevention strategies for Alzheimer’s have any merit? I heard that there was a recent NIH panel that said that none of them were useful!

R: The NIH recently sponsored a panel made up of 15 specialists in a variety of fields of medicine and public health, along with 20 expert presenters, in order to assess the current research on Alzheimer’s disease prevention. They came up with a consensus statement with a couple of major points – the most general being that given the difficulty in diagnosing the “pre-clinical” stages of Alzheimer’s disease, firm conclusions cannot be drawn about the association of any modifiable risk factor with Alzheimer’s disease. They did, however, conclude that “the evidence is insufficient to support the use of pharmaceutical agents or dietary supplements to prevent cognitive
**Mumbai Corner**

**Meeting Across the Globe**

Kathleen Vandyk.

Meeting in person is important, and this certainly holds true for clinical research. There are several types of research meetings that require live communication, including diagnostic and autopsy conferences, as well as staff training sessions. These meetings provide an opportunity for the teams to work together to decide on a final diagnosis, and clinical information is then compared to neuropathological findings from the autopsy work completed in Mumbai.

The Mount Sinai ADRC and Mumbai teams can only visit in person a few times a year, so we have taken advantage of recent progress in web-based telecommunication in order to meet “face-to-face.” We are now able to hold conferences real-time between New York and Mumbai, using programs that permit audiovisual communication over the internet. These teleconferences often lead to lively educational discussions about how experiences with Alzheimer’s disease and dementia may differ between the two countries. While nothing can replace the immediacy of sitting in the same room, technology such as this opens the door for researchers from around the globe to collaborate on critical research issues.

**New York City Team**

**Mumbai Team**

*Pictured to the far left (from left to right):* Dr. Dushyant Purohit; Dr. Nirmala Batheja (standing); Dr. Mary Sano; Kathleen Van Dyk

*Pictured to the near left (from left to right):* Dr. Meghna Bhatnagar; Richa Patel; Dr. Urvashi Shah; Dr. Girish Nair; Vaishali Ganwir

**memory walk 2010**

*Pictured below: Wendy Samuel (front row, second from left) and “Nonnie’s Team”*

*Pictured above: The ADRC team (from left to right):* Dr. Mary Sano, Judy Creighton, Dr. Margaret Sewell, Angelica De La Fuente, Riana Moring-Parris, Aliza Romirovsky, Elisheva Bellin, and Priyanka Ghosh.

Thousands came out to enjoy the sunny weather and to join the NYC Alzheimer’s Association’s annual memory walk on October 24th at Riverside Park. Mount Sinai walked as part of NY CARE, an organization that promotes awareness of AD research and represents the efforts of Mount Sinai, Columbia, and NYU, as well as the local Alzheimer’s Association. Many family, friends, and other supporters joined our efforts including Wendy Samuel and her “Nonnie’s Team.” Together, the NY CARE team raised over $10,000.00, our best year yet. Thank you to all who came out to support this important effort, and we hope to see you next year!
Alzheimer’s Disease Research Center News

• On September 29th, ADRC’s own Dr. Sam Gandy and Dr. Mary Sano presented at the 23rd Annual Chapter Meeting of the Alzheimer’s Association New York City Chapter, held before a packed house at the beautiful auditorium at Rockefeller University on the upper east side. Dr. Gandy spoke of the amyloid clumps called oligomers that are harbingers of Alzheimer’s disease, diminishing the role of neural plaques as a potential contributor, a theory that has long been held by the medical community. Dr. Sano presented on the functional and financial impact of Mild Cognitive Impairment (MCI), a condition that has been linked to later Alzheimer’s.

• On November 10th, ADRC Director Dr. Mary Sano was honored by Mount Sinai School of Medicine’s (MSSM) Office for Women’s Careers. The event, entitled Just Desserts, is an opportunity to spotlight an outstanding MSSM female professional, in an informal setting. Dr. Sano was the first to be recognized in a series featuring leading women physicians or scientists.

• On November 16th, we participated in the Alzheimer’s Foundation of America’s (AFA) National Memory Screening Day. National Memory Screening Day is one of the highlights of AFA’s efforts to promote early detection of memory problems, including Alzheimer’s disease, and to encourage appropriate intervention, including medical treatments, social services and other resources. Our ADRC offered free, confidential memory screenings, as well as follow-up resources and educational materials to those concerned about memory loss.

• The Mann Foundation recently held their 5th Annual Golf Outing at the Glen Head Country Club in Glen Head, Long Island. All proceeds went towards the Mount Sinai ADRC in the foundation’s continuing efforts to help find a cure for Alzheimer’s disease.

• A recent edition of the Huffington Post (11/15/2010) published an article discussing the relative increase in dementia prevalence within the workforce as the average age of retirement continues to rise. This article included input from numerous professionals involved in Alzheimer’s treatment and research, including Dr. Mary Sano:

“Raising the retirement age could increase the likely occurrence of the working population being at risk for the disease, but most problematic is the potential stigma that this risk may impose on [all] older workers.”

• Dr. Sam Gandy was featured recently in the AARP Bulletin and the New York Times noting the importance of amyloid as a significant contributor not only in developing Alzheimer’s disease, but also as a potential focus for future treatment and prevention. He has also appeared recently on Good Morning America and Fox News.

Have you considered brain donation?

Brain donation is a priceless contribution to knowledge that leads to the development of more effective treatment for Alzheimer’s disease. It is also critically important to be able to study brain tissue of individuals with no memory or other cognitive problems. This allows scientists to compare normal and abnormal brain tissue, which will lead to a greater understanding of factors that may protect the brain from disease in aging.

To update your brain donation registration information, or to learn about becoming a donor, contact Dr. Karen Dahlman at 212-241-2968, or email her at karen.dahlman@mssm.edu.
A drug used decades ago to treat high blood pressure has been shown to improve learning and memory in mouse models of Alzheimer’s disease, according to a new study by researchers at the National Institute on Aging (NIA), part of the National Institutes of Health. The study found that the drug, diazoxide, acted on nerve cells in the mouse brain in ways that slowed the development of the neurodegenerative disorder. The findings appear in the Nov. 15, 2010, print edition of the Journal of Alzheimer’s Disease.

Mark P. Mattson, Ph.D., chief of NIA’s Laboratory of Neurosciences in Baltimore, directed the research, in collaboration with colleagues at Konkuk University College of Veterinary Medicine, Seoul, South Korea, and the Indiana University School of Medicine, Indianapolis.

Mattson’s team found that diazoxide stabilized nerve cells in the brain and prevented a biological cascade in the mice that can result in the destruction of these cells. The drug also improved blood flow in the brain and prevented the harmful accumulation of two proteins, beta-amyloid and tau, which are hallmarks of Alzheimer’s. Widely used in the 1970s and ‘80s to treat patients with severe hypertension, diazoxide is currently used to treat hypoglycemia, or low blood sugar.

“These intriguing findings open new avenues of basic research that may increase our understanding of how modulating the electrical activity of nerve cells may slow the damage wrought by Alzheimer’s disease pathology,” said NIA Director Richard J. Hodes, M.D. “More research will be needed before we can determine whether this may be a potential therapy for Alzheimer’s.”

NIA scientists studied two groups of Alzheimer’s mice, one given diazoxide in drinking water and one given a placebo. After eight months, the diazoxide group outperformed the placebo group on a standard test of learning and memory. The brain tissue of the treated group showed fewer deposits of amyloid and tau proteins, less damage due to oxidative stress, and better blood flow—all indications that diazoxide may have suppressed some of the harmful cellular changes associated with Alzheimer’s disease. “To better understand the complex biological mechanisms by which diazoxide may exert a positive effect on nerve cells, we then studied the effects of diazoxide on cultured nerve cells,” Mattson said.

The scientists found the drug activates and opens channels in the cell that enhance the movement of potassium, which then calms the electrical activity of nerve cells in parts of the brain involved in learning and memory. Diazoxide also lowered the excessive calcium often found in nerve cells in brains affected by Alzheimer’s. These beneficial effects were seen with a dose of diazoxide low enough to avoid a major decrease in blood pressure, Mattson noted.

Comment by Dr. Sam Gandy:

“Many groups are looking to re-purpose existing drugs with known safety profiles, evaluating them anew in mouse models of Alzheimer’s. Diazoxide has a number of clinical applications, but its benefit in the mouse model is believed to be associated with its ability to promote nerve cell communication. Work from many labs shows that more communication often translates to improved plasticity, which, in turn, helps nerve cells fight off the poisonous environment of amyloid clumps called oligomers. Moreover, as MSSM colleague Joe Buxbaum and I showed over 20 years ago, nerve cell communication reduces generation of amyloid, so these two actions of diazoxide might be predicted to synergize in the AD brain. Of course, the true test will come in a randomized clinical trial, and I look forward to seeing the Mount Sinai ADRC participate in a diazoxide trial when that time arrives.”

Need a Memory Evaluation?

The ADRC’s Memory & Aging Center (MAC) provides comprehensive evaluation for those who have memory complaints. **Experts**: Our team includes experts in geriatrics, geriatric psychiatry and neuropsychology, neurology, and radiology. **Quick**: The evaluation can be completed in one visit, including evaluation by a geriatric memory specialist, neuropsychological testing, and neuroimaging.

To make an appointment, please call us at (212) 241-8329
evaluated for the etiology (cause) of the problem and the likelihood of AD might be determined by the type of cognitive problem (such as memory loss), or by genetic (such as Apolipoprotein E4) and biomarker data that are strongly associated with AD diagnosis.

Perhaps most controversial is the attempt to define a “pre-clinical” Alzheimer disease. This asymptomatic (normal) condition would be defined by biomarkers associated with AD pathology in the absence of any clinical features. While this may be a step toward earlier detection of AD, more information is needed before we can confirm that biomarkers in AD are really predictive of specific conditions when there are no symptoms present. Currently, markers thought to indicate AD have been present in about a third of the elderly asymptomatic individuals and right now we have no evidence that these people get worse. This highlights the importance of following normal volunteer elders to determine if we can accurately predict the onset of AD.

New Ways to Diagnose AD (continued from page 1)

Ask the Expert (continued from page 1)

decline or AD.”

Many possible factors have been studied ranging from cognitive stimulation, and physical activity to blood pressure medications, vitamins, omega 3 fatty acids, red wine, and eating a Mediterranean diet, among others. There are studies that are ongoing, and hopefully will augment our understanding, but so far the jury is still out as to whether they can alter an individuals risk for developing a memory problem.

The bottom line is that researchers need to continue to look for early diagnostic markers and identify people at significant risk, so that further large randomized trials can help sort out these answers. One example of how researchers are approaching this is through the AD neuro-imaging initiative (ADNI) – a study looking at imaging and other markers in blood or spinal fluid and following people over time to better understand and define who will go on to develop a dementia.

In the meantime doing “healthy” things – getting exercise, staying mentally active, eating a healthy and balanced diet may help and certainly won’t hurt!
A List of Caregivers’ Resources

The following is a listing of Support Groups at Mount Sinai and in the New York City area:

- **Early-stage-dementia patient support group**, contact Elizabeth Fine, M.S.W. to register at (212) 659-9230.

- **Spanish-Speaking family caregivers group**, meets on the 1st and 3rd Monday of the month from 11 to 12:30pm at Settlement Health located at 212 E. 106th Street. Call Dante Tipiani, M.S.W. at (212) 659-8872 to register.

- **The Memory Tree at DOROT** meet Tuesdays from 1pm-5pm. For more information, visit their website www.thememorytree.org, or call Elizabeth Fine, M.S.W. at (212) 659-9230.

- **Riverstone Senior Life Services** located at 99 Fort Washington Avenue Washington Heights offers Spanish-speaking groups every other Wednesday from 11am-12pm. English groups meet every Thursday from 11am-12pm. For more information, call Riverstone Senior Life Services at 212-927-5600.

- **The Elmhurst Senior Center** in Queens offers a Spanish-speaking family caregiver group that meets on the 2nd and 4th Wednesday of each month from 10am-11:30am. For more information or to register, call Mercedes Pichardo at (718) 478-7171, extension 27.

- **Monthly Caregivers’ Support Groups at Mt. Sinai**, Wednesdays from 12 noon - 1:30 at 1440 Madison Ave, Martha Stewart Center for Living. Groups will be held on January 12th, March 9th, & April 6th, 2011. Please call Elizabeth Fine, M.S.W. at 212-659-9230 to register.

- **The Caregivers Program at Mount Sinai** is a 4 week telephone class for people caring for someone with a memory problem. The following is a schedule of topics discussed at each session:
  - Week 1: Understanding the nature, cause, symptoms, treatment and research options related to memory disorders.
  - Week 2: Managing the communication and behavior changes associated with memory problems.
  - Week 3: Coping with the caregiving role: Time and stress management techniques.
  - Week 4: Resources available for people with memory problems and their caregivers.

  If you are interested in registering, please contact Elizabeth Fine, M.S.W. at (212) 659-9230.

Gene Associated with Type 2 Diabetes Is Low in People with Alzheimer’s Disease

Mount Sinai School of Medicine researchers have found that a gene associated with the onset of Type 2 diabetes also is found at lower-than-normal levels in people with Alzheimer’s disease. The research, led by Giulio Maria Pasinetti, MD, PhD, The Sauder Family Professor in Neurology, and Professor of Psychiatry and Geriatrics and Adult Development at Mount Sinai School of Medicine, was published this month in Aging Cell.

The new study provides insight into a potential mechanism that might explain the relationship between Type 2 diabetes and the onset and progression of Alzheimer’s disease. Recent evidence indicates that healthy elderly subjects affected by Type 2 diabetes are twice as likely to develop Alzheimer’s disease, but researchers have been unable to explain how.

“The relationship between Type 2 diabetes and Alzheimer’s disease has been elusive,” said Dr. Pasinetti. “This new evidence is of extreme interest, especially since approximately 60 percent of Alzheimer’s disease cases have at least one serious medical condition primarily associated with Type 2 diabetes.”

Using mice that were genetically engineered to have Alzheimer’s disease comparable to that seen in humans, Dr. Pasinetti and colleagues found that a gene known as proliferator-activated receptor coactivator 1 (PGC-1), a key regulator of glucose currently investigated as a potential therapeutic target for Type 2 diabetes, is decreased in Alzheimer’s disease. The team reports that this decrease might be causally linked to promotion of Alzheimer’s disease. They found that PGC-1 promotes degradation of a specific enzyme known as beta-secretase (BACE). BACE is directly involved in the processing and eventual generation of β-amyloid, an abnormal protein highly linked to Alzheimer’s disease and brain degeneration.

Dr. Pasinetti and his colleagues are optimistic that if they find that PGC-1 can be manipulated pharmacologically to prevent BACE accumulation in the brain, these studies will provide important insights for the formulation of novel treatments and possible preventative strategies in Alzheimer’s disease.
ADRC Studies Currently Enrolling

**Alzheimer’s Disease Neuroimaging Initiative – Grand Opportunity (ADNI-GO)**

In this study, we hope to determine whether imaging of the brain through MRI, PET and amyloid imaging scans can help predict and monitor the onset and progression of Alzheimer’s disease. In addition to neuroimaging, the study will collect and test blood and cerebral spinal fluid to determine if biomarkers can predict and monitor the disease. This study is sponsored by the National Institutes of Health and will take place at about 50 major universities across the US and Canada. No study drug is used in this research. Participants cannot be involved in other clinical trials while in this study. This is a longitudinal study which will span several years. We are looking for volunteers who can participate for the full duration. The study needs volunteers who: are between 55 and 90 years of age, in good health but have memory problems and concerns, are fluent in English or Spanish, are willing and able to undergo the test procedures, and have a study partner – a friend or relative who can accompany the volunteer to all clinic visits. Participant’s health will be closely monitored by a team of doctors and nurses. Participants will receive compensation for their time and costs incurred for travel, parking and meals. For more information, please contact Aliza Romirowsky at (212) 241-1514.

**Nerve Growth Factor (NGF) Study**

CERE-110 is a research drug being used in gene therapy research for Alzheimer’s disease. In this study, genes are transferred to brain cells via neurosurgery so that the body can make NGF, a naturally occurring protein that might increase the survival of neurons that die in AD. The purpose of this phase 2 clinical trial is to find out if this type of gene therapy technique, neurosurgically injecting CERE-110, is safe, well-tolerated, and of benefit when given to people with AD. For more information, please contact Priyanka Ghosh at 212-659-8885 or priyanka.ghosh@mssm.edu. GCO#09-0367; Principal Investigator: Judy Neugroschl, M.D., MSSM IRB approved through 4/13/11.

**The Gammaglobulin Alzheimer’s Partnership Study**

The Gammaglobulin Alzheimer’s Partnership (GAP) Study is designed to evaluate the safety, efficacy, and tolerability of the investigational drug Immune Globulin Intravenous (IGIV) for the treatment of mild-to-moderate Alzheimer’s disease (AD). IGIV is a biologic agent with anti-inflammatory and immunomodulating properties; this study is being conducted to determine if IGIV can help slow the progression of AD and its symptoms. The Gammaglobulin Alzheimer’s Partnership (GAP) Study is seeking volunteers who: 1. Are aged 50-89, and who have probable mild-to-moderate AD. 2. Have a Mini-Mental Status Exam (MMSE) score of 16-26. 3. Have not suffered from serious or unstable diseases within the past 3 months. 4. Have a study partner who can actively participate in the study with the volunteer. For more information, please contact Priyanka Ghosh at (212) 659-8885, or via email at priyanka.ghosh@mssm.edu. MSSM #08-1326; Principal Investigator: Hillel Grossman, M.D. MSSM approved through 3/16/11.

**Functional Deficits of ACC in MCI**

This study examines the effects of aging on memory and attention. Volunteers will be trained for a simple computer task and will perform this task in an MRI scanner. All participants will be compensated for time and travel. Participants are eligible to participate if they meet the following criteria: are between 55 - 90 years of age; are either free of memory problems or are experiencing some memory problems; have a Mini-Mental Status Exam (MMSE) score higher than 24 (if not known, this can be determined through evaluation); have no metal in their body; do not have any current psychiatric disorders; are not claustrophobic. For more information, please contact Joanna Micek at 212-241-1613; joanna.micek@mssm.edu. #08-00443 MSSM approved through 6/19/11.

**Trial of NIC5-15, a natural product in subjects with Alzheimer’s disease.**

We are seeking Veterans with Alzheimer’s disease to participate in a research study of a natural product, NIC5-15 at the Bronx James J. Peters Veterans Medical Center. NIC5-15 is a natural product found in legumes and soybeans. In laboratory studies it interferes with the accumulation of a protein in the brain that is involved in the development of Alzheimer’s disease. Researchers hypothesize that this may slow down the progression of the disease. Some study participants will receive NIC5-15 and some will receive a placebo (sugar pill). Participation in the study includes physical exams, neurological exams, blood tests and tests of memory and thinking skills. Participation of 8 months is necessary. For more information please contact Riana Moring-Parris at (718) 584-9000 ext 5179, or email at Riana.moring-parris@mssm.edu. Principal investigator: Hillel Grossman, MD. VA GRO#10-029 MSSM approved 12/6/2010-6/30/2011.

An interesting segment was featured on ABC’s Nightline, following a patient who participated in a neurosurgical research study evaluating the effectiveness of nerve growth factor (NGF) as a treatment for Alzheimer’s disease. The patient discusses his thoughts and experiences participating in this type of research. This video can be viewed online at: [http://www.hulu.com/watch/187633/abc-nightline-alzheimer’s-breakthrough](http://www.hulu.com/watch/187633/abc-nightline-alzheimer’s-breakthrough)
Researchers are one step closer to discovering the genetic precursors to late-onset Alzheimer’s disease, the most common type of the disease. Investigators from four institutions, including Dr. Joseph Buxbaum of Mount Sinai Medical Center, collaborated to discover the role of variations in a gene named MTHFD1L. The presence of this altered gene appears to result in a nearly two-fold increase in an individual’s risk of developing Alzheimer’s.

The research was conducted by a team of researchers led by Margaret Pericak-Vance, Ph.D., director of the John P. Hussman Institute for Human Genomics (HIHG) at the University of Miami Miller School of Medicine (details appear September 23 in the open-access journal *PLoS Genetics*). According to Dr. Pericak-Vance, identification of this gene “is important because the gene is known to be involved in influencing the body’s levels of homocysteine, and high levels of homocysteine are a strong risk factor for late-onset Alzheimer’s disease.”

In a previous study conducted at Queen’s University Belfast and published in *Stroke*, it was reported that even moderate elevations of homocysteine were associated with a nearly threefold increased risk for Alzheimer’s disease. Homocysteine is an amino acid believed to be toxic to blood vessels. “We are hopeful our identification of MTHFD1L as a risk gene for Alzheimer’s disease will help us to better understand how this disease develops and potentially serve as a marker for people who may be at increased risk,” said co-author Dr. Adam Naj.

“This finding gives us unique insight into possible interactions between genetic and environmental risk factors that contribute to AD,” said Dr. Buxbaum. “We know of environmental and lifestyle factors that can impact homocysteine levels and it will be important to understand whether variations of the MTHFD1L gene can modulate these effects.”

This latest discovery of a gene variation that appears to increase the risk of this devastating disease “will lead to a better understanding of what’s happening in Alzheimer’s disease, and how we can improve treatment,” added Dr. Jonathan L. Haines of Vanderbilt Center for Human Genetics Research. This discovery comes at a time when new insights into the prevention and treatment of Alzheimer’s disease are desperately needed. The latest statistics released by the Alzheimer’s Association show that about 5.3 million Americans have this form of dementia, with a nearly threefold increase anticipated by 2050.

Gene Linked to Increased Risk of Alzheimer’s Disease

Researchers are one step closer to discovering the genetic precursors to late-onset Alzheimer’s disease, the most common type of the disease. Investigators from four institutions, including Dr. Joseph Buxbaum of Mount Sinai Medical Center, collaborated to discover the role of variations in a gene named MTHFD1L. The presence of this altered gene appears to result in a nearly two-fold increase in an individual’s risk of developing Alzheimer’s.

The research was conducted by a team of researchers led by Margaret Pericak-Vance, Ph.D., director of the John P. Hussman Institute for Human Genomics (HIHG) at the University of Miami Miller School of Medicine (details appear September 23 in the open-access journal *PLoS Genetics*). According to Dr. Pericak-Vance, identification of this gene “is important because the gene is known to be involved in influencing the body’s levels of homocysteine, and high levels of homocysteine are a strong risk factor for late-onset Alzheimer’s disease.”

In a previous study conducted at Queen’s University Belfast and published in *Stroke*, it was reported that even moderate elevations of homocysteine were associated with a nearly threefold increased risk for Alzheimer’s disease. Homocysteine is an amino acid believed to be toxic to blood vessels. “We are hopeful our identification of MTHFD1L as a risk gene for Alzheimer’s disease will help us to better understand how this disease develops and potentially serve as a marker for people who may be at increased risk,” said co-author Dr. Adam Naj.

“This finding gives us unique insight into possible interactions between genetic and environmental risk factors that contribute to AD,” said Dr. Buxbaum. “We know of environmental and lifestyle factors that can impact homocysteine levels and it will be important to understand whether variations of the MTHFD1L gene can modulate these effects.”

This latest discovery of a gene variation that appears to increase the risk of this devastating disease “will lead to a better understanding of what’s happening in Alzheimer’s disease, and how we can improve treatment,” added Dr. Jonathan L. Haines of Vanderbilt Center for Human Genetics Research. This discovery comes at a time when new insights into the prevention and treatment of Alzheimer’s disease are desperately needed. The latest statistics released by the Alzheimer’s Association show that about 5.3 million Americans have this form of dementia, with a nearly threefold increase anticipated by 2050.