

WORKING TO MAKE **ALZHEIMER'S DISEASE**

NOTHING BUT A **MEMORY**

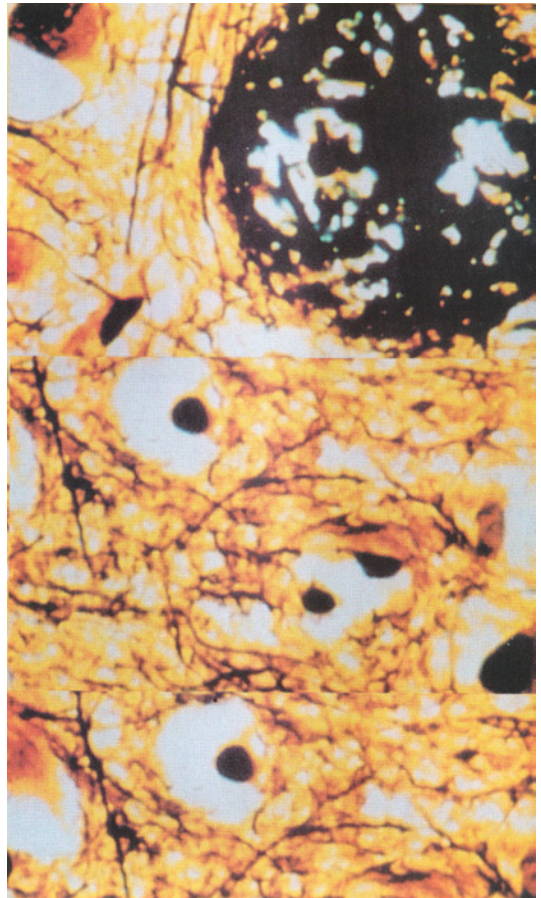


FISHER CENTER FOR
ALZHEIMER'S
RESEARCH FOUNDATION





Healthy Brain Cell



Brain Cells Afflicted by Alzheimer's Disease

The human brain is the most remarkable and complex structure known to man. Within its three-pound wrinkled mass are some 100 billion nerve cells (neurons), tiny bits of tissue that choreograph every aspect of our being. Crisscrossed among them is an astounding network of a trillion or more nerve fibers that act like electrical wires, forming synaptic connections where nerve impulses are transferred from one neuron to the next. Through these contact points, or synapses, flow the chemical and electrical signals that underlie every aspect of our being, not just our very basic, everyday abilities to walk and talk, but also our capacity to think, feel, reason and remember.

The cruelty of Alzheimer's disease is that it attacks the brain, profoundly altering who we are. Like a thief in the night, it robs us of our most basic functions and fundamental pleasures at the very time when we should be enjoying the fruits of lifelong labor. It steals from us the very qualities that make us human: our capacity for love, for planning and reasoning, for making and recalling memories. Even for making everyday decisions. As our society ages, and more people are afflicted, the need to hunt down and stop this killer disease in its tracks has never been greater.



Creating Hope Through Scientific Discovery

The opening of The Fisher Center for Alzheimer's Disease Research at The Rockefeller University marked the launch of one of the most intensive attacks ever on this dreaded illness. The brainchild of Zachary Fisher, whose wife Elizabeth suffered from Alzheimer's, the Center was founded with the singular mission of spearheading the crusade against Alzheimer's disease--to create hope for a cure or to delay the onset of the disease--through scientific discovery. A generous fund contributed by Mr. Fisher and David Rockefeller enabled the construction of a magnificent 10,000-square foot research laboratory equipped with the advanced facilities necessary to support an unprecedented interdisciplinary assault on this devastating illness.

Nobel Laureate Directs Research

A blue-ribbon search committee unanimously recommended world-renowned neuroscientist Paul Greengard, Ph.D., to be founding director of the Fisher Center. Dr. Greengard revolutionized our understanding of the molecular and cellular basis by which nerve cells communicate with one another, discoveries that earned him the 2000 Nobel Prize in Physiology or Medicine. He and his associates have also made fundamental contributions not only to our understanding of Alzheimer's disease, but also to the elucidation of depression, schizophrenia, Parkinson's disease, drug abuse and other brain disorders.

Dr. Greengard has assembled a team of outstanding scientists from all over the world to help us understand the causes of Alzheimer's disease and develop novel approaches to its prevention, early detection and treatment.

Today, the Fisher Center is one of the largest and most modern facilities in the world dedicated to solving the puzzle of Alzheimer's disease. Its strategy is to facilitate cross-fertilization of ideas by applying the discoveries of basic science to clinical studies with Alzheimer's patients. The strategy encourages a two-way "bench-to-bedside" exchange, ensuring that new developments in basic science can be rapidly applied to clinical situations and that clinical

weapons available to modern warriors in the battle against Alzheimer's. These include sophisticated "gene chips" and other essential tools of molecular biology that permit scientists to monitor hundreds of genes as they are turned on and off during nerve-cell function. A high-tech computer complex facilitates data analysis and expedites worldwide exchange of information.

The adjoining Fisher Conference Center

"This model center, where researchers and caregivers work side by side, is a crucial element in the course we follow to combat Alzheimer's disease."

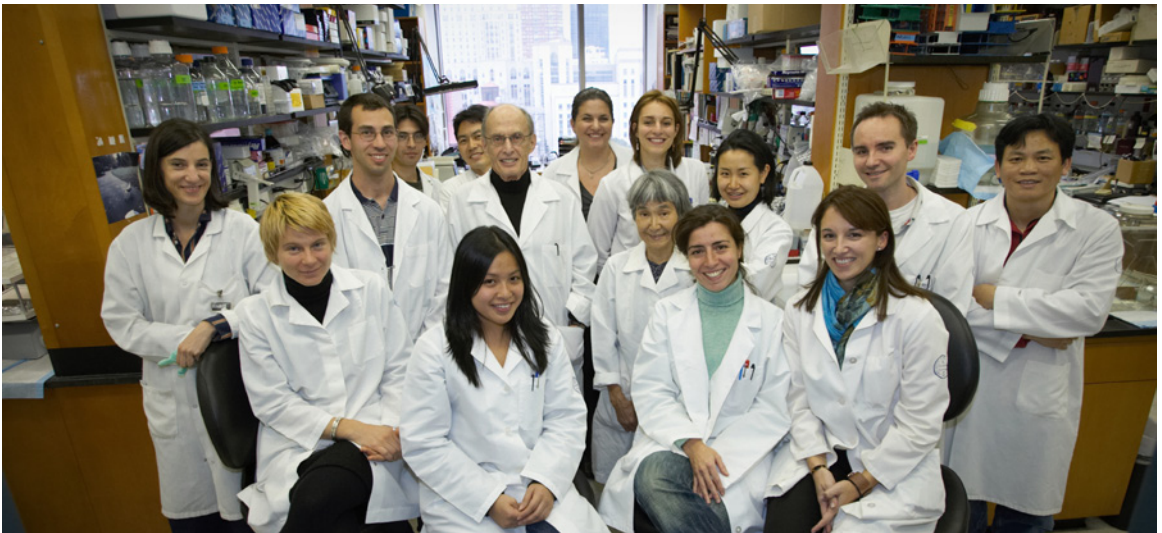
Dr. Richard J. Hodes, Director of the National Institute on Aging.

findings guide and stimulate strategies for scientific exploration. The Center is considered by many to be a prototype for Alzheimer's research centers.

Equipped for Battle

The Fisher Center houses an impressive armamentarium of the most advanced

functions as a kind of war room for plotting the next moves in the battle. Top scientists from around the world gather here to share scientific findings and collaborate in order to expedite research advances. Informal collaborations augment annual symposia at The Rockefeller University on related subjects.



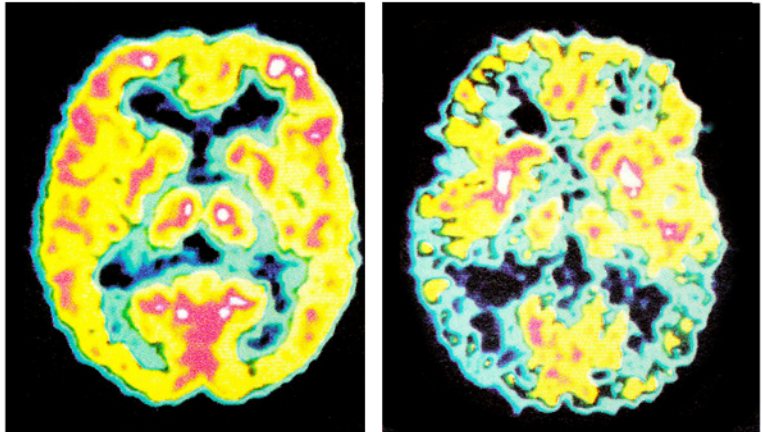
Dr. Paul Greengard (center) surrounded by some of the fifty scientists in the Rockefeller Research lab. The Fisher Center laboratory is one of the world's largest Alzheimer's research facilities.

Alzheimer's Disease

What is it?

Alzheimer's disease is a fatal brain disorder that today affects an ever-growing number of Americans. Alzheimer's is a disease of aging, and because the life span of our population is longer than ever before, the number of people affected is increasing rapidly. Scientific researchers have made huge strides in understanding Alzheimer's, raising strong hope that effective treatments, and possibly even a cure, are now within reach.

Alzheimer's is the most common form of dementia, a group of disorders that impairs mental functioning. (Dementia literally means "loss of the ability to think.") At the moment, Alzheimer's is progressive and irreversible. Abnormal changes in the brain worsen over time, eventually interfering with many aspects of brain function. Memory loss is one of the earliest symptoms, along with a gradual decline of other intellectual and thinking abilities, called cognitive functions, and changes in personality or behavior. Alzheimer's advances in stages, progressing from mild forgetfulness and cognitive impairment to widespread loss of mental abilities. In severe Alzheimer's, people become dependent on others for every aspect of their care. The time course of the disease from onset to death varies by individual, ranging from five to 20 years. The most common cause



Brain scans done with Position Emission Tomography (PET) demonstrate how Alzheimer's affects brain activity. The left image shows a normal brain, while the right is from a person with Alzheimer's. The blue and black areas in the right image indicate reduced brain activity resulting from the disease.

Images courtesy of National Institute on Aging

of death is a co-occurring infectious illness, such as pneumonia.

What Causes Alzheimer's Disease?

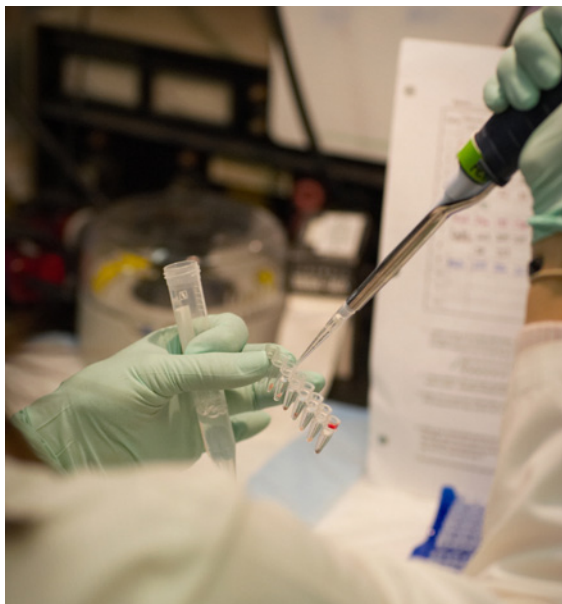
Scientists have made significant progress in understanding the possible causes of Alzheimer's disease, yet many questions remain. It is likely that multiple factors, both inherited and environmental, interact to cause the disease.

In addition to investigating what might trigger Alzheimer's in some people, scientists have identified a number of brain changes that are associated with the disease. These include the characteristic plaques and tangles around and inside nerve cells that were first associated with the clinical dementia syndrome described by the German physician Alois Alzheimer at a medical conference in 1906.

What Happens to the Brain with Alzheimer's?

The progressive loss of cognitive function in Alzheimer's disease is accompanied by pathologic (disease-associated) changes in the brain. One of these is the formation of plaques-sometimes described as tiny "Brillo pads" in the space between nerve cells. Unlike plaques in blood vessels, which are composed of the fatty substance cholesterol, the brain plaques in Alzheimer's are comprised of a stringy protein called beta amyloid. Another protein, called tau, which normally channels chemical messages inside nerve cells, deforms and collapses into neurofibrillary tangles, which appear like twisted bits of wire inside nerve cells.

The amyloid plaques consist principally of a single building-block protein called beta amyloid, which is derived from a much larger molecule known as the amyloid precursor protein (APP). The mechanisms regulating how rapidly APP is broken down to produce the amyloid protein were discovered at the Fisher Center and continue to be studied both at the Center and worldwide.



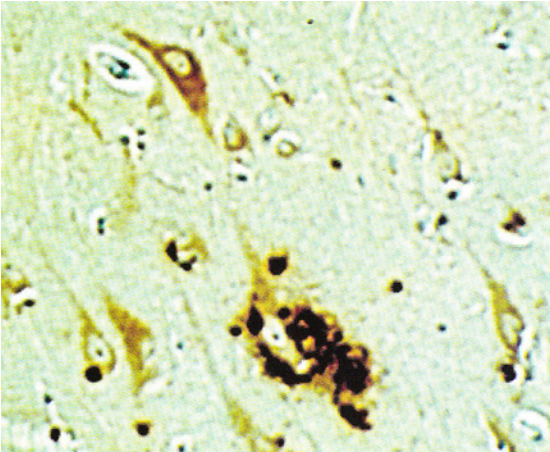
The Cost of Living Longer

The successes of medical science have led to an unprecedented increase in the life expectancy of Americans. With this "graying" of America, Alzheimer's disease looms as the most severe socio-medical problem our nation will face in the next few decades.

- 5.3 million Americans have Alzheimer's
- 54% of the U.S. population has been touched in some way by Alzheimer's
- There are 10.9 million unpaid caregivers
- Total payments for AD in 2010 are expected to be \$172 billion, including \$123 billion for Medicare and Medicaid
- The cost to U.S. businesses for employees who are caregivers of people with Alzheimer's and other dementias is \$36.5 billion
- U.S. businesses also pay an additional \$24.6 billion for health care, long-term care and hospice for people with Alzheimer's and other dementias
- Total healthcare costs are more than three times higher for people with Alzheimer's and other dementias than for other people age 65 and older
- Every 70 seconds someone in America develops Alzheimer's disease. By mid-century someone will develop Alzheimer's every 33 seconds. By the end of 2010, there will be nearly a half million new cases of Alzheimer's each year; and by 2050, there will be nearly a million new cases per year.

These financial estimates do not begin to account for the physical and emotional toll on the families and caregivers of those affected. For more information or to donate (only 8% of our funds are used on overhead and administrative purposes), please visit ALZinfo.org or call 1-800-ALZ-INFO.

Because the devastation of Alzheimer's doesn't stop with the person afflicted.



In Alzheimer's, plaques comprised of a protein called beta amyloid build up in the brain and interfere with nerve cell communication. Brown areas in image indicate amyloid accumulation; the lighter brown shows amyloid within nerve cells, while the darkest spots are where amyloid has formed plaques in the space between nerve cells.

Who Is Affected?

Today, as many as 5.3 million Americans are believed to have Alzheimer's disease. Alzheimer's affects about 10 percent of people aged 65 and up, and the prevalence doubles roughly every 10 years after age 65. Half of the population age 85 and up are thought to have Alzheimer's.

Because the population of the U.S. is aging, the number of people with Alzheimer's will continue to rise unless something can be done to stem it. At current rates, by the year 2050, more than 16 million people will have the disease. By that date, more than 60% of people with Alzheimer's disease will be age 85 or older.

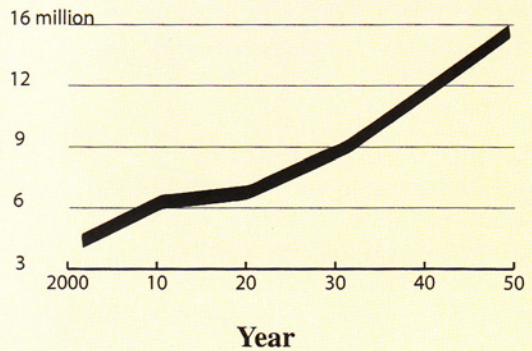
What Are the Risk Factors For Alzheimer's?

Two proven risk factors for typical Alzheimer's disease are age and family history. The disease usually strikes after age 65, and risk increases with advancing age. Having a family member with Alzheimer's increases one's risk, particularly if the relative has the early-onset form of the disease (beginning before

age 65). However, half of people with the far more common late-onset form have no family history.

Many non-genetic so-called "environmental" factors may also increase one's risk, and a great deal of research is aimed at understanding these factors. Examples would be a past that includes a serious head injury, lower levels of formal education, and lower socioeconomic status, although scientists are not sure why or how these things interact to produce the disease in some people but not others. There is recent evidence that one's environment and experiences early in life may also play a role in the eventual development of Alzheimer's. Continuing research is urgently needed to try to solve these mysteries.

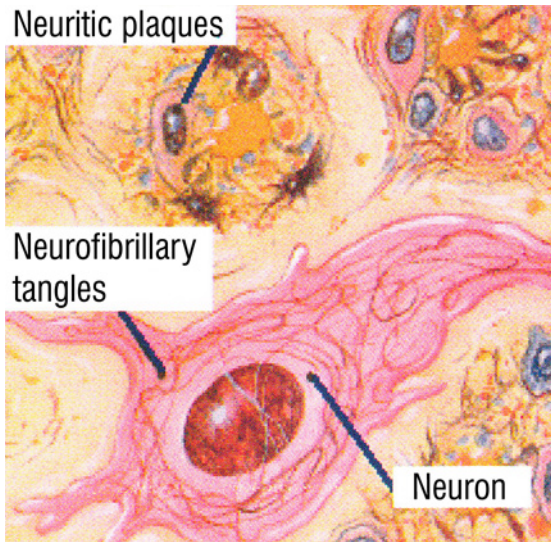
Projected Alzheimer's cases in the U.S.



Source: Evans et al, International Conference on Alzheimer's Disease, Stockholm, July 2002

How is Alzheimer's Disease Treated?

Until scientists better understand the causes of Alzheimer's disease, a cure will continue to elude us. While great progress has been made, there is still no effective treatment for the illness, and none that addresses the underlying disease processes.



Hallmarks of Alzheimer's disease include neuritic plaques (outside neurons), and neurofibrillary tangles



Supportive-care approaches and anti-agitation therapists improve the quality of Life for AD patients

Drugs currently approved by the U.S. Food and Drug Administration (FDA) for treating Alzheimer's are only minimally effective in relieving some of the symptoms in a small proportion of patients. These medicines are most effective when begun early in the course of the disease, yet early diagnosis is difficult. No currently approved medication strikes at the cause of the disease.

At the Fisher Center laboratory, pioneering research aimed at understanding the biological mechanisms underlying Alzheimer's has helped scientists understand the cause better than ever before. However, as with cancer and other complex diseases, the true roots of Alzheimer's continues to escape us. So researchers have started with the proximate cause - beta amyloid proteins - and worked backward in an attempt to identify approaches to delaying the onset or altogether preventing the disease. These leads are being aggressively pursued by scientists in academic laboratories and pharmaceutical companies. In fact, dozens of potential Alzheimer's therapies are now undergoing

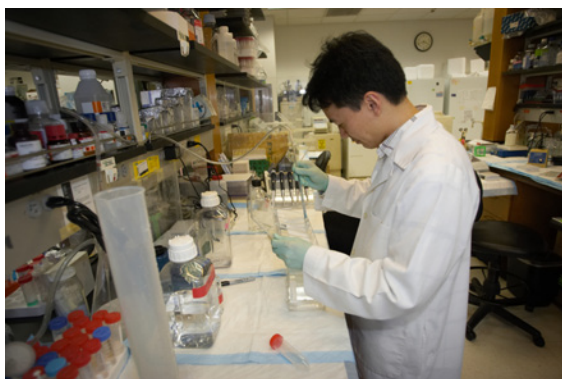
human clinical trials and many more are being investigated in pre-clinical trials in laboratory animals. These studies offer great hope that truly effective treatments for Alzheimer's will soon be available.

To find clinical trials near you, visit: www.clinicaltrials.gov

At the moment, in the absence of truly beneficial treatment, various supportive-care approaches may be employed to ease the suffering of the person with Alzheimer's and the burden on their families. Considerable research is ongoing to develop more effective strategies for managing the troublesome behavioral symptoms of Alzheimer's, such as agitation, wandering and delusions. As the disease progresses, changes in thought, reason, emotion, and memory may be so pervasive and the person's emotional state so disrupted that management with strong anti-agitation drugs may become necessary. These drugs have serious limitations, and improving them is an area of intense research at the Fisher Center.

The Research Program Leading the Battle

Scientists at the Fisher Center are attacking the scourge of Alzheimer's disease with a three-pronged assault that seeks to understand the cause, improve care, and find a cure. Elucidating the processes that lead to the formation of amyloid plaques is central to understanding the cause and preventing the disease by somehow interrupting these processes. Developing better anti-agitation medications will improve the care of people who have Alzheimer's by controlling behavioral symptoms and enhancing the quality of life for victims and their families. The vanguard scientists at the Fisher Center have also been at the forefront of research to induce nerve cells to grow new synaptic connections that may be able to compensate for dying nerve cells—a strategy that could delay the onset of the disease long enough to effectively cure it. This multi-level approach offers the best hope for people suffering from Alzheimer's and holds great promise for achieving genuine scientific breakthroughs in the short term.



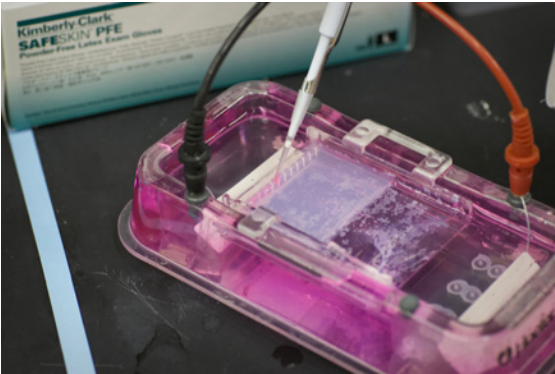
Researcher checking molecules



A scientist working with molecules in test tubes.

Important Advances Close At Hand

In the November 14, 2008 issue of the journal *Cell*, researchers at the Fisher Center for Alzheimer's Disease Research at The Rockefeller University report a breakthrough in cellular analysis that solves a problem that has perplexed neurological research since the neuron was first discovered over 100 years ago. Lead author, Myriam Heiman, and her colleagues have developed a method to reveal the kinds and amounts of proteins different cells produce, what biologists call its translational profile. The technique involves isolating the genetic messages that govern protein production in these different cell types. The new method, "translating ribosome affinity

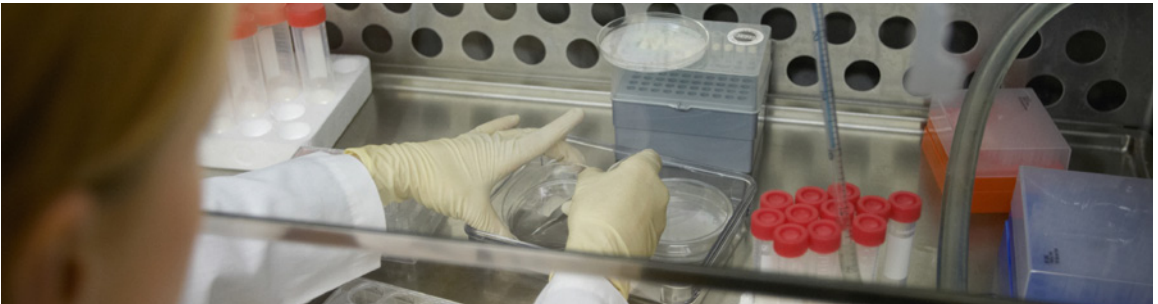


The Center is equipped with state-of-the-art technology

purification”, TRAP, can identify all the genetic messages that give that cell type its unique identity, including, perhaps, its susceptibility to disease.

type in any tissue in the body. Its usefulness is not just limited to brain cells, meaning it has far reaching research applications—cancer, heart disease, diabetes, as well as many others. Dr. Paul Greengard, the director of the Fisher Center for Alzheimer’s Disease Research where Myriam Heiman is a Research Associate, says about half of the research in his lab now employs the new technique to study the biochemical bases of Alzheimer’s, Parkinson’s and other diseases. It is also being applied to the still-mysterious ways in which psychoactive drugs fight schizophrenia and depression.

The TRAP tool advances the speed at which researchers can yield results and should fundamentally change biochemical studies of



Our scientists are among the country’s leaders in applying the sci-fi technology of molecular genetics to unravel the many mysteries of Alzheimer’s and Parkinson’s diseases, among others.

Like skilled assassins, many diseases seem to know exactly what types of cells to attack. While destroying one type of cell, a disease will inexplicably spare a seemingly identical group of neighbors. What makes cells vulnerable or not depends largely on these translational profiles. For this reason, scientists have struggled to analyze the subtle molecular differences among the hundreds of specialized cell types that are tangled together in tissues like the brain. The new TRAP procedure solves a problem that has been a fundamental barrier to a deeper understanding of the brain and how neurological diseases attack it. The true breakthrough lies in its ability to distinguish the profile of any cell

the brain. “We can look at a thousand genes instead of one at a time, so things should clear a thousand times faster,” says Dr. Greengard, who won the Nobel Prize in Physiology or Medicine in 2000 for research into how neurons communicate.

This new technique will help accelerate scientific research into discovering the subtle molecular differences amongst the hundreds of specialized cell types. A deeper understanding of body cell mechanisms will help researchers investigate the causes of Alzheimer’s and Parkinson’s diseases.

History and Overview

The Fisher Center for Alzheimer's Research Foundation

The Fisher Center for Alzheimer's Research Foundation was organized as a not-for-profit corporation on October 24, 1995. At an earlier date, a Senate committee representing the federal Labor, Health and Human Services and Education departments directed the National Institutes of Health to gather for them information on the cause, care, and cure of Alzheimer's. The first meeting dealt with cause; the next two dealt with care and potential cure. From these findings, the scientists drew up a blueprint that outlined the most promising research areas. This first coordinated effort has set a benchmark standard for the U.S. Congress and for the scientific world to follow.

The Fisher Foundation was established for purposes that are exclusively charitable, scientific and educational. Supporters of its charitable programs include individuals, corporations, foundations, quasi and governmental organizations. This support is primarily directed toward research to be conducted at the Fisher Center lab at The Rockefeller University in New York City, as well as related research facilities in the U.S. and abroad.

For example, the Fisher Alzheimer's Disease Education and Resources Program at the New York University School of Medicine focuses on the care of people suffering from Alzheimer's.

Led by internationally recognized Alzheimer's care expert Barry Reisberg, M.D., investigators

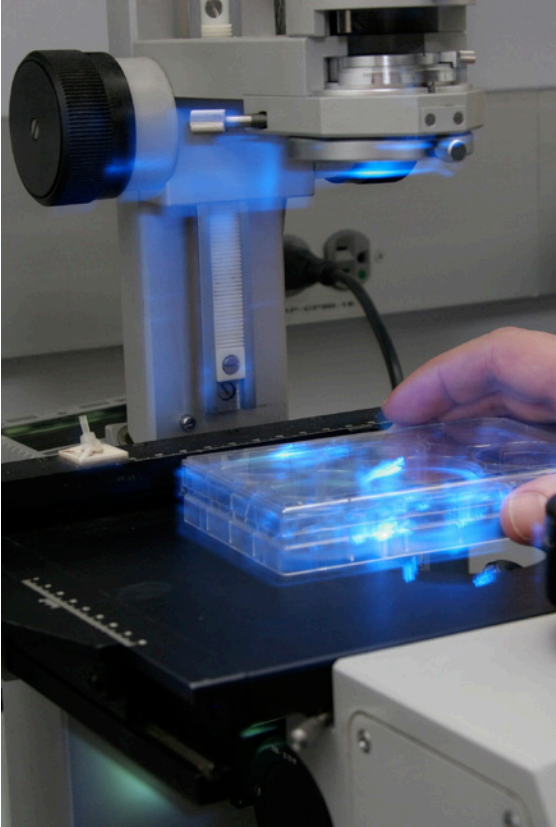
have developed a new approach to Alzheimer's care that addresses the basic mechanisms of the disease, resulting in new techniques for managing the disease. At The University of Genoa, Italy, researchers under the direction of neuroscientist Fabio Benfenati, Ph.D., are investigating the mechanisms by which nerve cells in the brain communicate, with the goal of enabling healthy cells to compensate for cells that have died in Alzheimer's and related diseases.

The Foundation hosts numerous events throughout the year, including forums for exchange of scientific information about Alzheimer's disease, educational conferences and fundraising events.

A singular genuine belief drives the mission of the Fisher Center Foundation and its scientists: that Alzheimer's disease can and will one day be eliminated.



Rockefeller neuroscience laboratories span the areas of molecular and cellular neurobiology, sensory neuroscience and the neurobiology of behavior.



Scientists examine cells via our state-of-the-art microscope.

About Our Research

- Beating Back Beta Amyloid
- Improving The Quality Of Life For Alzheimer's Patients
- Reversing Nerve Cell Damage
- Recruiting Stress Proteins to clean up tangles in the brain
- Curing Early-Onset Alzheimer's
- The Science Of Caring

Beating Back Beta Amyloid

The Fisher Center Foundation funds scientists who continue to be at the forefront of research into the understanding of beta amyloid, a protein that is at the root of Alzheimer's

disease. Beta amyloid forms dense protein deposits called plaques. Sometimes compared to tiny scouring pads, these beta amyloid plaques accumulate in the spaces between brain cells, choking them off and causing them to die. Fisher Center scientists were among the first to define the specific steps by which beta amyloid is produced, and their discovery that pharmacological substances can interrupt this process set off a worldwide race to develop drugs to inhibit beta amyloid buildup. The Foundation's beta amyloid research is now focused on a protein called amyloid precursor protein (APP). When broken down in a certain way, APP results in the formation of beta amyloid plaque. Fisher scientists are unraveling the process by which APP processing is regulated. Their immediate goal is to develop ways to slow the accumulation of plaque and other forms of beta amyloid in the brain and reduce their toxic effects on nerve cells, moving much closer to a cure for Alzheimer's disease. In fact, Fisher Center Foundation scientists are making significant progress in developing therapies aimed at reducing the production of toxic beta-amyloid, an achievement that will ultimately prevent, slow or even cure the disease. Fisher scientists recently identified several proteins that regulate the amount of beta-amyloid produced in the brain. What's new is that these "regulator" proteins exert control over one or both of the enzymes that are known to produce beta-amyloid directly. Discovery of this "molecular fine-tuning" mean that Fisher scientists can now search for, or design, new drugs for treating Alzheimer's.

Fisher scientists have also recently discovered how beta-amyloid damages communication between brain cells and how fibers connecting brain cells can be grown or made to shrink. With this understanding, it may be possible to

devise therapies that protect the brain even when beta-amyloid production goes awry.



A scientist works with a cell culture

Improving The Quality Of Life For Alzheimer's Patients

Most Alzheimer's disease patients show some signs of agitation and as Alzheimer's disease progresses through its later stages, as many as 75% of patients begin to exhibit aggressive or agitative behaviors, which are often treated with potent antipsychotic drugs. While useful, these drugs may cause incapacitating side effects. Building on two decades of research, Fisher Center Foundation scientists are exploring the mechanisms by which these antipsychotic drugs work in an effort to develop new and safer "anti-agitation" therapies that will improve the quality of life for patients while easing the burden on caregivers.

Reversing Nerve Cell Damage

The devastating loss of memory that is the hallmark of Alzheimer's disease is caused by the death of nerve cells "strangled" by beta amyloid. In what would have seemed like science fiction a decade ago, the Foundation's scientists are making progress in reversing this damage by actually inducing nerve cells to grow new connections with other cells, thus

improving communication between remaining healthy cells. This work builds on Fisher Foundation scientists' recent discovery that a protein called "WAVE1" regulates the growth of structures called spines that ultimately connect nerve cells; the Foundation's scientists are exploring ways to compensate for the death of nerve cells in Alzheimer's. These techniques might someday not only reverse symptoms such as memory loss in Alzheimer's patients, but might also treat other nervous system disorders such as Parkinson's and Huntington's disease, strokes, head trauma and spinal cord injuries.

Recruiting Stress Proteins to Clean Up Tangles in the Brain

The brains of people with Alzheimer's contain large numbers of plaques and tangles. Plaques are formed from beta-amyloid. Tangles are made of a protein called tau. Like beta-amyloid, tangles can also damage the brain and thus contribute to the devastating loss of mental function in Alzheimer's disease. Scientists have known for a while that tangles are caused when the tau protein does not fold properly. All proteins need to fold to have normal function. When a protein does not fold properly, it not only loses its own function, but also may damage many other proteins in cells, especially in the brain. Fisher scientists recently discovered that they could prevent the formation of tangles in a model of Alzheimer's disease by supplying a drug that blocks a type of protein known as a "chaperone" or "stress protein." This could lead to treatments that prevent much of the devastating damage to brain cells that occurs in Alzheimer's. Such treatment might be applied alongside an anti-amyloid treatment and this combination may turn out to be especially beneficial.

Curing Early-Onset Alzheimer's

While being diagnosed with Alzheimer's can be overwhelming at any age, it is particularly devastating when the disease strikes early in one's life. Early-onset Alzheimer's can present itself in people as young as 30 and it is strongly inherited, affecting generation after generation. It is known that certain genetic mutations alter a set of proteins in the brain called "presenilins" and these alterations in turn lead to increased production of a toxic form of beta amyloid. Research funded by the Foundation recently shed light on the particular roles of three presenilin-associated proteins, nicastrin, PEN2 and APH1, which are involved in critical steps in the onset of Alzheimer's. Foundation scientists are also continuing to research the two presenilin proteins, PS1 and PS2, which are cause of most cases of early-onset Alzheimer's. Understanding the exact roles of PS1, PS2, PEN2, APH1 and nicastrin proteins in the development of Alzheimer's is a crucial step in developing therapies and drugs to slow or reverse the progression of the disease.



Scientist at our Rockefeller University Lab

The Science Of Caring

While searching for the cause and developing a cure for Alzheimer's disease, the Foundation is also funding projects to support the many family members and friends who are on the front lines of caring for the more than 5 million Alzheimer's patients in the US and beyond. Despite the massive burden of coping with a long list of patients' behavioral problems, such as aggressiveness and anxieties, little information is available for caregivers about effective treatments and interventions.

The Foundation funds research at the Fisher Alzheimer's Disease Education and Resources Program at New York University School of Medicine under the direction of Dr. Barry Reisberg. This research has led to the

development of a new science of management for Alzheimer's and other dementias. Our next challenge is to raise funds to implement a caregiver training program based on this research and management principles. This program allows Alzheimer's patients to regain basic skills of daily living and reduces the patient's dependence on a caregiver, thus improving the quality of life for all involved.

In addition to numerous research projects, the Foundation also funds the Alzheimer's Information Program, www.ALZinfo.org. ALZinfo.org is a tool provided by the Fisher Center for Alzheimer's Research Foundation to educate people about this devastating disease. The mission of the website is to

educate, engage and create an online community with 24 hours a day, 7 days a week access to information and support via online chats, message boards and the most comprehensive resource databases available.

Whether you are afflicted with Alzheimer's, or a caregiver, or a loved one of an Alzheimer's patient, you are sure to find answers to your questions. Our Resource Locator will help you find local support on a variety of topics.

Another great feature of the website is www.ALZTalk.org. ALZTalk.org, is a free and easy way to make new friends and stay connected with those in the Alzheimer's community. Join today to post messages and share pictures and favorite links. ALZTalk.org gives users a voice and allows them to share tips and stories about coping with loved ones with Alzheimer's. It also offers the ability to ask our experts questions no matter how large or small.

To contact the Fisher Center for Alzheimer's Research Foundation staff, please:

Email:
info@alzinfo.org or
visit: www.ALZinfo.org

Call:
1 (800) ALZ-INFO or 1 (800) 259-4636

Write:
Fisher Center for
Alzheimer's Research Foundation
1 Intrepid Square
West 46th Street and 12th Avenue
New York, NY 10036



Our Scientists



Dr. Paul Greengard

Director of the Fisher Center for Alzheimer's Disease Research at The Rockefeller University Dr. Paul Greengard was awarded the year 2000 Nobel Prize in Physiology or Medicine for his pioneering work in delineating how neurons communicate with one another in the brain. During a half-century of research, he has been lauded for his singular contribution to our understanding of the complex signaling process that occur within each of the 100 billion or more nerve cells in the human brain. He is the Vincent Astor Professor at The Rockefeller University and Director of the Fisher Center laboratory. Dr. Greengard is also a member of the National Academy of Sciences and has received more than 50 awards and honors throughout his career. He has authored nearly 1000 scientific publications.



Dr. Barry Reisberg

Professor of Psychiatry at New York University School of Medicine Clinical Director of the Aging and Dementia Research Center of the NYU School of Medicine Dr. Reisberg has directed research over the past quarter century which has significantly advanced the current understanding and treatment of Alzheimer's disease. He was the first to describe many of the most important symptoms of Alzheimer's and the characteristic clinical course of the disease with the Global Deterioration Scale in 1982 and the Functional Assessment Staging measure in 1984.

Dr. William J. Netzer

Research Associate at the Fisher Center for Alzheimer’s Research Foundation, and Scientific Liaison to the Fisher Foundation

Dr. Netzer’s research led to the recent discovery that Gleevec, a successful anti-cancer drug, lowers beta-amyloid production, suggesting that Gleevec or compounds similar to it might be useful in treating Alzheimer’s disease.

Dr. Marc Flajolet

Sr. Research Associate at the Fisher Center for Alzheimer’s Research Foundation

Dr. Flajolet’s research led to the recent discovery that a protein called “casein kinase one” regulates the production of beta-amyloid in the brain, suggesting that inhibitors of this protein might be useful in treating Alzheimer’s disease.

Dr. Wenjie Luo

Research Associate at the Fisher Center for Alzheimer’s Research Foundation

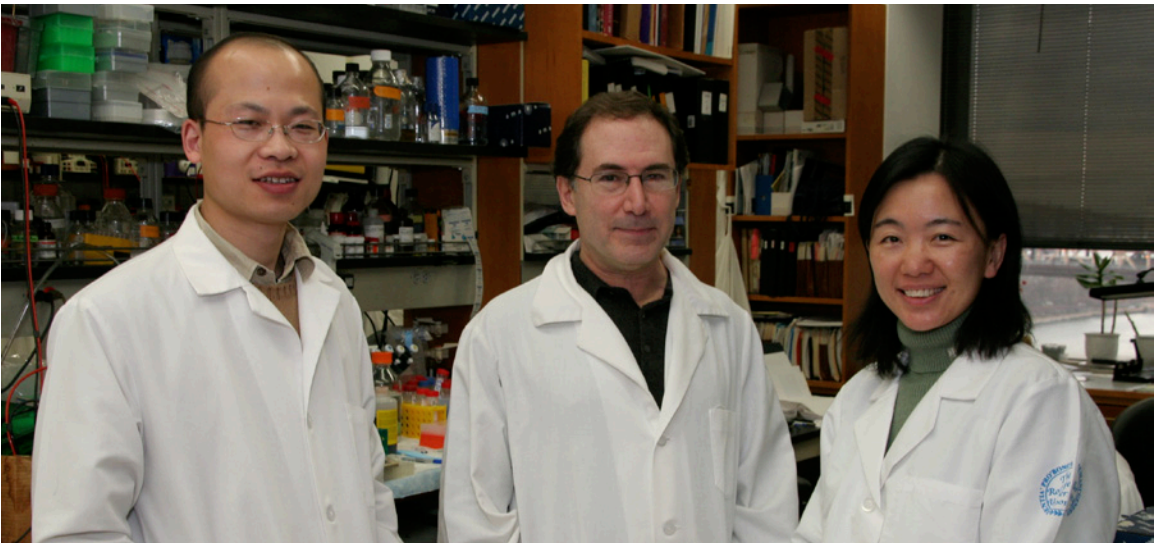
Dr. Luo’s research led to the recent discovery that the formation of tau tangles could be prevented by using a drug that reduces the activity of a protein called a “chaperone,” suggesting that this drug or compounds related to it could be used to treat the degeneration of brain cells in Alzheimer’s disease.

Dr. Ann Schaefer

Post doctoral fellow at the Fisher Center for Alzheimer’s Research Foundation

Dr. Schaefer’s research led to the discovery that molecules called “micro RNAs” (produced in the brain) are necessary to maintain normal brain development and function, suggesting that these molecules might be harnessed to treat Alzheimer’s disease.

For more information on our latest scientific findings, visit: <http://www.alzinfo.org/our-publications>



Dr. William J. Netzer



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