

Current Alzheimer's Disease Research Projects

There is scarcely a person alive today who has not seen or felt the impact of Alzheimer's disease (AD). AD ranks sixth among the leading causes of death in the United States. By disrupting memories, cognition and personality, AD is extremely stressful for family members, friends and caregivers. Alzheimer's disease will claim a greater toll as our population ages—unless something more is done.

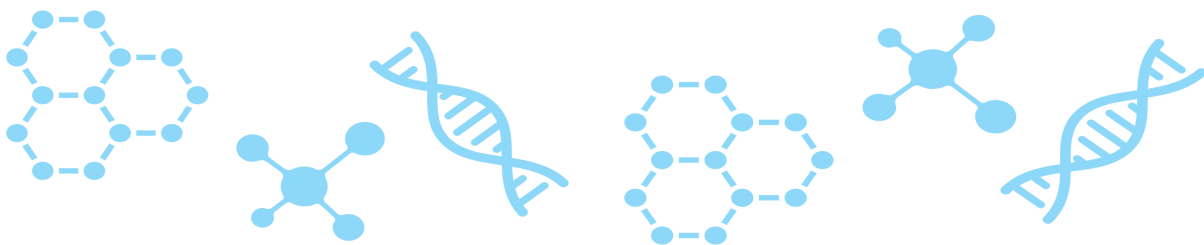
With generous donor support, BrightFocus Foundation's Alzheimer's Disease Research (ADR) program, which began in 1985, has funded nearly \$120 million in research to understand and cure this disease. With our support, scientists around the world have developed and tested thousands of hypotheses about how the disease destroys brain function over time and investigated hundreds of ideas to treat and cure dementia.

This yearbook provides an overview of BrightFocus' current ADR grant projects. BrightFocus Foundation does not direct funding to specific techniques or categories of research. Applications from scientists for funding can be on any aspect of understanding and curing dementia. Each year, the proposals are evaluated by a Scientific Review Committee (SRC) comprised of expert scientists from around the world. Our SRC recommend projects that are the most cutting-edge and the most likely to be successful. The generosity of our donors helps make this possible.

***Note:** The funding statistics and inclusion of research grants in this yearbook are based upon award offers and grants that were active as of July 1, 2018. Excerpts are from research profiles available at BrightFocus.org and may have been edited for clarity and space constraints. This information is accurate as of 2/6/2019*

The current portfolio of active projects within the ADR program is expansive. As an organizational tool for this yearbook, more than 90 projects have been arranged by broad themes:

- **Stopping Alzheimer's by Understanding Disease Progression**
- **Beta Amyloid and Tau: The Debate Continues**
- **Role of Immune Factors and Clearance Mechanisms**
- **Blood Vessels and the Brain**
- **Drug Discovery and New Treatment Approaches**
- **Intervening with Genetic Risk Factors**
- **Lifestyle Changes to Reduce Alzheimer's Risk & Impact**
- **Pioneering New Ways to Image and Assess the Brain**
- **New Methods and Resources to Fight Alzheimer's**



Stopping Alzheimer's by Understanding Disease Progression

On November 4, 1906, Dr. Alois Alzheimer described the symptoms of one of his patients, Mrs. Auguste Deter. After she died, Dr. Alzheimer examined her brain (with new techniques developed by his friend and colleague Dr. Frank Nissl) to describe the plaques and tangles that define the disease today. With the support of our donors, scientists around the world are working back from Dr. Alzheimer's reports to understand how the disease starts. One goal of this research is to identify who is likely to develop dementia in the future by measuring something in the body that doctors can use to define an individual's risk.

The following projects test theories about specific biomarkers for Alzheimer's disease that predict the formation of plaques, tangles and dementia.



Special Opportunity Grant

Jill M. Goldstein, PhD (3/30/18 - 3/29/20)

Massachusetts General Hospital, Harvard University, Boston

Clinical Algorithm to Identify Alzheimer's Disease Risk in Early Midlife

This project will support the launching of a comprehensive effort (integrating clinical, physiological and brain biology traits) to identify in early midlife biomarkers for AD risk informed by sex differences in brain aging and memory decline.

www.brightfocus.org/grant/CA2018607



Inma Cobos, MD, PhD (7/1/17 - 6/30/20)

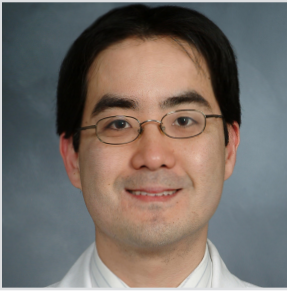
University of California, Los Angeles

Alzheimer's in the Human Brain: Focusing on One Neuron at a Time

What makes some neurons more vulnerable or resistant to disease? This project uses a new technique called "single cell RNA sequencing" to isolate thousands of single neurons from human brain tissue, study all the genes that are expressed in each individual cell, and make cell-to-cell comparisons between normal, early stage and late stage AD.

www.brightfocus.org/grant/A2017346S

This grant is made possible in part by support from Alzheimer's Los Angeles.



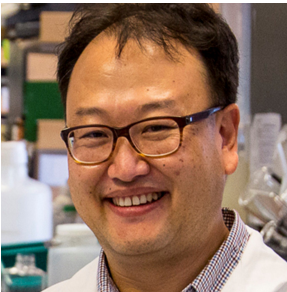
Makoto Ishii, MD, PhD (7/1/15 - 12/30/18)

Weill Cornell Medicine, New York, NY

Identifying How Fat Hormones That Regulate Body Weight Are Affected in Alzheimer's Disease

Before memory and thinking problems occur, people who develop AD start to lose body weight. This project studies whether there are problems with hormones that control body weight early on in AD.

www.brightfocus.org/grant/A2015485S



Doo Yeon Kim, PhD (7/1/16 - 6/30/19)

Massachusetts General Hospital & Boston Children's Hospital

Co-Principal Investigator **Clifford J. Woolf, PhD**

A Human Cellular Alzheimer's Disease Model Based on 3D Culture Technology

This project further develops and characterizes a human 'Alzheimer's in a dish' model based on a unique three-dimensional culture technique that uses human cells growing in a dish. This will provide a novel and valid platform for basic mechanistic studies and drug screening in an environment similar to the human brain.

www.brightfocus.org/grant/A2016362S



Tae Ho Lee, PhD (7/1/17 - 6/30/20)

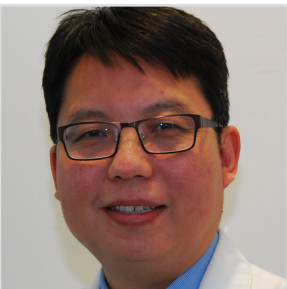
Beth Israel Deaconess Medical Center,

Harvard Medical School, Boston, MA

New Mechanism to Regulate Neuron Death in Alzheimer's Disease

The goal of this project is to study the role of death-associated protein kinase 1 (DAPK1) in AD using mouse models of AD.

www.brightfocus.org/grant/A2017180S



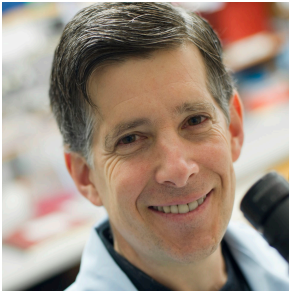
Tao Ma, MD, PhD (7/1/17 - 6/30/20)

Wake Forest School of Medicine, Winston-Salem, NC

A Potential New Therapeutic Target for Alzheimer's Disease

This project evaluates the mechanism of AD that leads to cells making fewer new proteins through the activity of an mRNA translational factor, eEF2, and its associated kinase as the basis for future work to identify novel diagnostic markers and therapeutic targets.

www.brightfocus.org/grant/A2017457S



Benjamin Wolozin, MD, PhD (7/1/15 - 6/30/19)
Boston University, MA

Harnessing Reversible Protein Aggregation to Treat Alzheimer's Disease

Biological stresses, including beta amyloid, induce formation of a particular type of protein complex termed stress granules. The researchers hypothesize that the process of aggregation associated with stress granules might stimulate pathology in AD. Reducing stress granule formation could be a novel therapeutic strategy.

www.brightfocus.org/grant/A2015256S



Jessica Young, PhD (7/1/18 - 6/30/21)

University of Washington School of Medicine, Seattle, WA

A New Method to Assess Cellular Dysfunction in Alzheimer's Using Human Neurons

The overall goal is to use "induced pluripotent stem cell technology", derived from adult human cells, to better understand pathogenic events that may occur early on in neurons that could represent novel therapeutic targets for AD. The focus is on the endosomal network (how proteins are moved within a cell), which may become dysfunctional in AD before amyloid and tau deposits are reported.

www.brightfocus.org/grant/A2018656S



Beta Amyloid and Tau: The Debate Continues

Alzheimer's well-known plaques and tangles are made of beta amyloid and tau proteins that have clumped together outside (plaques) and inside (tangles) of brain cells. These clumps of proteins are difficult to remove. Exactly how they are involved in cell death is uncertain, it is clear that both proteins interact and play a role. Much attention has focused on toxic tau, which increases later in AD and usually signals that the disease is worsening. As plaques and tangles increase, they are associated with damage to the brain's network and subsequent cognition loss.



Ottavio Arancio, MD, PhD (7/1/18 - 6/30/21)

Columbia University, New York, NY

Co-Principal Investigator **Russell Nicholls, PhD**

Identifying How Tau Impairs Nerve Cell Communication in Alzheimer's Disease

Toxic forms of tau play a central role in AD and other neurodegenerative conditions, in part by interfering with how neurons connect to each other at synapses (the tiny gap where electrical signals are transmitted). The goal of this project is to better understand how tau interferes with synaptic function so that we can develop effective strategies to block the impairments it causes."

www.brightfocus.org/grant/A2018816S



Kathryn Bowles, PhD (7/1/17 - 6/30/19)

FELLOWSHIP

Icahn School of Medicine at Mount Sinai, New York, NY

Discovering New Factors That Control the Production of Different Versions of Tau Protein

There are six different versions of tau protein in the human brain. This project aims to discover which genes are responsible for regulating the different versions of tau so that we may better understand how and why an imbalance occurs, and what we could do to fix it.

www.brightfocus.org/grant/A2017144F



Resham Chhabra, PhD (7/1/17 - 6/30/19)

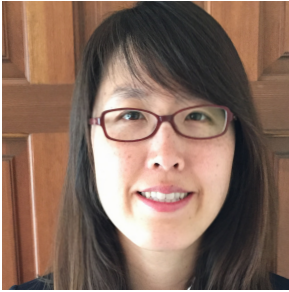
FELLOWSHIP

Johns Hopkins University, Baltimore, MD

TDP-43 Depletion as a Risk Factor for Tau Pathology in Alzheimer's

In 30%-70% of AD cases, a protein called TDP-43 is lost from the nucleus of cells (a round 'central-command' structure that contains the DNA). The aim of this study find out if TDP-43 loss plays a role in the initiation or acceleration of tauopathy.

www.brightfocus.org/grant/A2017102F



Karen Chiang, PhD (7/1/15 - 6/30/19)
University of California, San Diego

FELLOWSHIP

The Impact of Beta Amyloid on the Spread of Tau Toxicity in the Brain

This research will study the role of beta amyloid in tau toxicity by using mouse models that express both tau and amyloid beta in a specific cell population within the hippocampus to determine whether tau spread is altered in neurons which form synapses with these amyloid-expressing cells.

www.brightfocus.org/grant/A2015595F



Cara Croft, PhD (7/1/18 - 6/30/20)
University of Florida, Gainesville

FELLOWSHIP

Using Brain Slices to Understand and Target Tau in Alzheimer's Disease

This project uses mouse models and an engineered, non-infectious virus delivery system to make the animal brains develop similar buildup of tau that is seen in AD patients. These are compared to mouse tissue without tau to see why or if these cells die. Then, novel treatments to prevent or reverse the buildup of tau will be tested.

www.brightfocus.org/grant/A2018149F



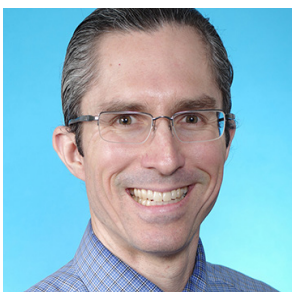
Karen Duff, PhD (7/1/17 - 6/30/20)
Columbia University, New York, NY
Co-Principal Investigator **Natura Myeku, PhD**

Slowing Alzheimer's Disease by Enhancing Cellular Garbage Disposal

This project tests the effectiveness in animal models of a drug that stimulates the brain's own "garbage disposal units" (the proteasome) to remove the toxic proteins that form clumps in the brain and cause the memory loss.

www.brightfocus.org/grant/A2017393S

This grant is made possible in part by support from Lois and Duane Luallin in memory of Denver E. Perkins and Edwin H. Luallin.

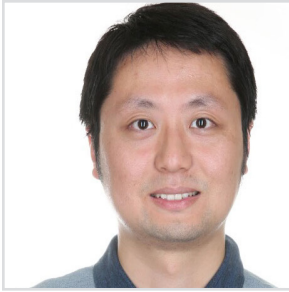


Chadwick Hales, MD, PhD (7/1/17 - 6/30/20)
Emory University, Atlanta, GA

How Proteins Contribute to the Formation and Spread of Pathology in the Alzheimer's Disease Brain

This project studies how three specific proteins that support RNA processing in healthy brains will seed tangle-like aggregates in AD using cell cultures and animal models of the disease.

www.brightfocus.org/grant/A2017281S



Zhuohao He, PhD (7/1/18 - 6/30/21)
University of Pennsylvania, Philadelphia
Co-Principal Investigator **Virginia Man-Yee Lee, PhD**

Studying a Type of Tau Protein that Aggregates in Alzheimer's Disease Brains

This project investigates a distinct form of pathological tau in AD and will further create antibodies capable of recognizing such AD-specific pathological protein.

www.brightfocus.org/grant/A2018802S



David Irwin, MD (7/1/16 - 6/30/19)
University of Pennsylvania, Philadelphia

Non-Amnestic Alzheimer's Disease Biology

This study will integrate genetic, neuroimaging and digital histopathology data to determine the genetic influence and progression of tau pathology in non-amnestic AD (people with underlying AD pathology and cognitive difficulties, but no memory problems). This approach will improve the diagnosis of AD patients who may benefit from emerging therapies that aim to halt or slow the progression of plaques and tangles in the brain and also identify new genetic targets for drug development in AD.

www.brightfocus.org/grant/A2016244S



Shahrnaz Kemal, PhD (7/1/17 - 6/30/19)

FELLOWSHIP

Northwestern University, Chicago, IL

Unexplored Toxic Pathways in Alzheimer's Disease: Potential New Drug Targets

This project is designed to find unique pathways by which beta amyloid, a toxic peptide associated with AD, adversely affects neurons in the absence of tau, another toxic peptide involved in AD. In doing so, novel drug targets may be identified. Additionally, new drugs will be tested for their ability to reverse some of the detrimental effects of beta amyloid.

www.brightfocus.org/grant/A2017033F





Daniel Lee, PhD (7/1/15 - 6/30/19)
University of South Florida, Tampa, FL
Co-Principal Investigator **Maj-Linda Selenica, PhD**

Gene Therapy with Arginine Decarboxylase and the Regulation of Tau

This proposal will identify how an enzyme called 'arginine decarboxylase' regulates tau pathology. The results will identify if increasing arginine metabolism and polyamines in mouse models impacts tau neuropathology.

www.brightfocus.org/grant/A2015504S



Goonho Park, PhD (7/1/18 - 6/30/20)

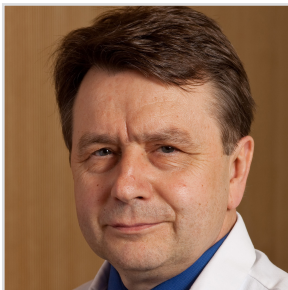
FELLOWSHIP

University of California, San Diego

A Novel Mechanism of Neuronal Disconnection in Early Stage Alzheimer's

Beta amyloid peptide, which is generated from amyloid precursor protein (APP), is hypothesized to be one of the major reasons for synaptic damage in AD. APP also generates another fragment called C31. This project will test whether the blocking of the generation of C31 from APP can protect synapses from injury and damage.

www.brightfocus.org/grant/A2018212F

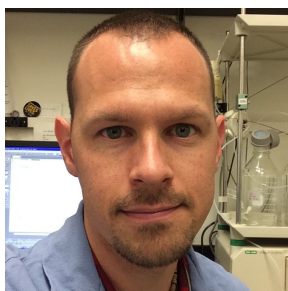


Jiri Safar, MD (7/1/16 - 6/30/19)
Case Western Reserve University, Cleveland, OH
Co-Principal Investigator **Qingzhong Kong, PhD**

Profiling Prion-Like Strains of Beta Amyloid that Control Alzheimer's Progression

This project tests the hypothesis that rapid progression of AD is caused by specific molecular structural features of beta amyloid using mouse models of the disease.

www.brightfocus.org/grant/A2016085S



Paul Seidler, PhD (7/1/16 - 6/30/19)

FELLOWSHIP

University of California, Los Angeles

Blocking Assembly of Tau Protein into Toxic Structures Associated with Alzheimer's Disease

This project will expand our understanding of Alzheimer's by discovering the many different structural assemblies (toxic or non-toxic) that are available to tau.

www.brightfocus.org/grant/A2016588F

This grant is made possible in part by support from Alzheimer's Los Angeles.

Role of Immune Factors and Clearance Mechanisms

The brain has 'immune privilege,' which means the entire central nervous system is separated from the body's immune system. Instead, scavenger 'microglia cells' are spaced throughout the brain to maintain a healthy environment. These cells can clear damaged cells and other unwanted particles, and dispose of them into the bloodstream. Microglia and star-shaped astrocytes, another type of brain cell, have a complex role in the development of dementia. Grantees are looking at what causes the immune response to become unbalanced and whether there are ways to help the brain's immune cells to counter cell death and the aggregation of proteins.



Mickael Audrain, PhD (7/1/18 - 6/30/20) *FELLOWSHIP*
Icahn School of Medicine at Mount Sinai, New York, NY

Role of the Microglial Protein Tyrobp in the Pathogenesis of Tauopathy

Using AD mouse models and primary cultures, the goal of this project is to investigate the involvement of a macroglial protein, Tyrobp, in tauopathy progression and its associated inflammatory response.

www.brightfocus.org/grant/A2018253F



Wei Cao, PhD (7/1/18 - 6/30/21)
Baylor College of Medicine, Houston, TX

New Immune Molecule in Inflamed Alzheimer's Brain

This project will study a new family of cytokines, recently detected in Alzheimer's brains, that participate in the inflammatory process.

www.brightfocus.org/grant/A2018377S



Charles Glabe, PhD (7/1/18 - 6/30/21)
University of California, Irvine

Mechanism of Neuronal Death in Alzheimer's Disease

Cell death pathways that are involved in the progression of the inflammatory response, one of the hallmarks of AD, are of highest interest. Detailed knowledge about this specific type of inflammatory cell death pathway in AD brains might allow us to identify potential therapeutic strategies to prevent neurodegeneration.

www.brightfocus.org/grant/A2018718S



Congcong He, PhD (7/1/18 - 6/30/21)
Northwestern University, Chicago, IL

How Autophagy Recognizes & Degrades Alzheimer's Disease-Causing Amyloids in the Brain

The goal is to understand how autophagy, a protein degradation pathway, regulates beta amyloid metabolism and prevents neuronal inflammation in the Alzheimer's brain.

www.brightfocus.org/grant/A2018100S



Benjamin Hogan, PhD (7/1/18 - 6/30/21)
The University of Queensland, Brisbane, Australia
Co-Principal Investigator **Neil Bower, PhD**

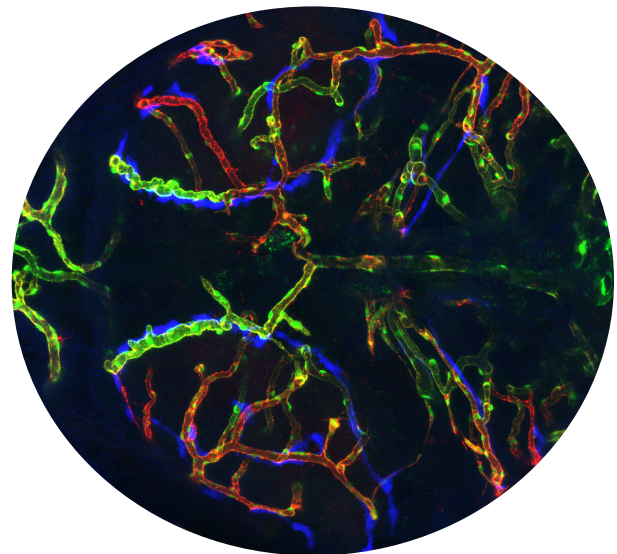
Characterization of Waste Clearance Pathways in the Vertebrate Brain

This project aims to develop a new understanding of how a specific cell type in the brain, called 'mural lymphatic endothelial cells,' clear waste from aging brains.

www.brightfocus.org/grant/A2018807S

Courtesy of Dr. Benjamin Hogan's Lab

In this picture of the blood vessels of a zebrafish brain, the arteries are green, veins are red and a new type of cell involved with clearing out waste from the brain are blue.



Bruce Lamb, PhD (7/1/15 - 6/30/19)
Indiana University, Indianapolis, IN

The Role of TREM2, a Key Immune Regulating Protein, in Alzheimer's Disease

Understanding the biology of TREM2, the newly identified immune molecule in Alzheimer's disease risk, will enable identification of new drug targets. These researchers will study its role in Alzheimer's tau pathology and discover how it interacts with different brain cells.

www.brightfocus.org/grant/A2015296S



Wenjie Luo, PhD (7/1/16 - 6/30/19)
Weill Cornell Medicine, New York, NY

Cellular Mechanisms Underlying Microglia-Mediated Amyloid Degradation

Accumulation of abnormal amyloid and tau proteins in the brain is detrimental for brain functions. This project will evaluate how a specific small molecule helps scavenger microglia cells clean up these proteins.

www.brightfocus.org/grant/A2016399S



Zixu Mao, PhD (7/1/16 - 6/30/19)
Emory University, Atlanta, GA

Understanding Brain Inflammation in Alzheimer's Disease

The goal is to understand the critical signaling pathways that underlie microglial inflammatory response in the context of Alzheimer's pathogenic stress as the disease progresses over time.

www.brightfocus.org/grant/A2016501S



Edoardo Marcora, PhD (7/1/17 - 6/30/20)
Icahn School of Medicine at Mount Sinai, New York, NY
Co-Principal Investigator **Anne Schaefer, MD, PhD**

Understanding the Role of Apolipoprotein E in Microglia

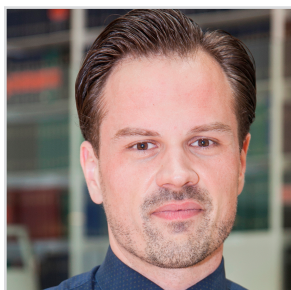
In normal conditions, microglia cells do not make APOE; however, in disease conditions, they sense the brain damage and respond by churning out APOE. The researchers are investigating what happens in mouse models if the APOE gene is removed from microglia.

www.brightfocus.org/grant/A2017458S



Blood Vessels and the Brain

Most AD is 'mixed', meaning that in addition to amyloid plaques and tau tangles, there can be changes in the brain's blood vessels that interfere with normal circulation. In some cases, beta amyloid can deposit in vessels, and Alzheimer's-related inflammation causes vessels to grow 'sticky', both of which may stall blood flow and compromise the supply of oxygen and nutrition to the brain. In addition, the extremely tight junctions of the blood-brain barrier (BBB), which close the brain's circulation from the rest of the body, also may be affected in AD. While certain BBB changes appear harmful, scientists are exploring how to safely manipulate it to allow better clearance of toxins and easily get drugs into the brain, which is notoriously difficult to accomplish.



Daniel Bos, MD, PhD (7/1/17 - 6/30/19)

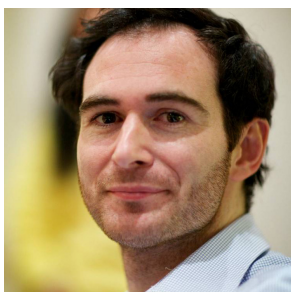
FELLOWSHIP

Erasmus Medical Center, Rotterdam, Netherlands

How Atherosclerosis Affects Brain Structure, Cognitive Function, and Dementia

Atherosclerosis - or hardening of the arteries - is increasingly being recognized as an important risk factor for dementia. Yet, it remains unclear whether the progression of atherosclerosis at different locations in the arterial system also contributes to changes in the structure or function of the brain and, ultimately, dementia.

www.brightfocus.org/grant/A2017424F



Matthew Campbell, PhD (7/1/15 - 6/30/19)

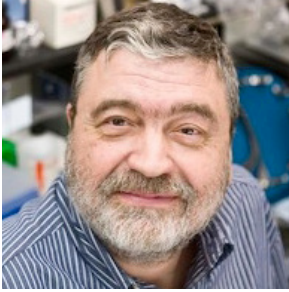
Co-Principal Investigator **Peter Humphries, PhD**

Trinity College, Dublin, Ireland

A New Method to Remove Toxic Material from the Brains of Alzheimer's Disease

These researchers are examining the role of novel 'tight junction' proteins in AD. Tight junctions are barriers between cells that limit movements of proteins and fluid between two cells. These researchers will assess whether tight junction protein modulation with anti-amyloid antibodies could act as a novel therapy.

www.brightfocus.org/grant/A2015548S

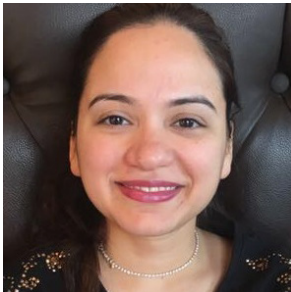


Jorge Ghiso, PhD (7/1/15 - 6/30/19)
NYU Langone Health, New York, NY

Effect of Aging and Dysfunction of Cerebral Microvasculature in Alzheimer's Disease

The project aims to provide a better understanding of the brain removal and catabolism of beta-amyloid in health and disease through a comprehensive approach encompassing the use of biochemical and proteomic methodologies in combination with genetically engineered mouse models with severe compromise of blood vessels in the brain

www.brightfocus.org/grant/A2015275S



Saima Hilal, PhD (7/1/18 - 6/30/20)
Erasmus Medical Center, Rotterdam, Netherlands

FELLOWSHIP

The Impact of 'Silent' Small Strokes on Brain Function and Alzheimer's Development

These researchers aim to find the cause for Alzheimer's disease by detecting small strokes using structural and functional brain scans of thousands of people.

www.brightfocus.org/grant/A2018165F

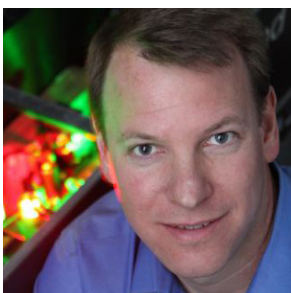


Ethan Lippmann, PhD (7/1/17 - 6/30/20)
Vanderbilt University, Nashville, TN
Co-Principal Investigator **Laura Dugan, MD**

Identification of Genes/Proteins Involved in Leakage of Blood Vessels in the Brain

In AD patients, the blood vessels of the brain become leaky, which worsens symptoms like memory loss. This project aims to identify why they become leaky.

www.brightfocus.org/grant/A2017094S



Chris Schaffer, PhD (7/1/17 - 6/30/20)
Cornell University, Ithaca, NY

Improving Brain Blood Flow in Alzheimer's Disease to Improve Cognitive Function

Screen drugs that interfere with white blood cell adhesion-and have already been proven safe for humans-to find candidates that reduce tiny capillary vessel stalling, and improve blood flow to the brain in AD.

www.brightfocus.org/grant/A2017488S

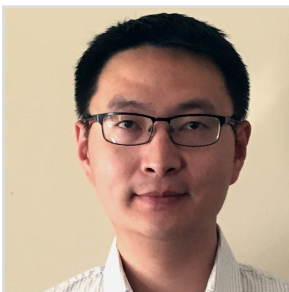


Alex Smith, PhD (7/1/18 - 6/30/21)
University of California, San Francisco

Why Is Brain Glucose Uptake Reduced in Alzheimer's Disease?

Blood vessels in the brain are surrounded by cells that contain a very large amount of a protein called aquaporin-4. In Alzheimer's disease, the amount of aquaporin-4 around vessels is reduced. This project will test if this is causing the cells to swell around the vessels and block sugar from getting into the brain.

www.brightfocus.org/grant/A2018351S



Yi Su, PhD (7/1/17 - 6/30/20)
Washington University School of Medicine, St. Louis, MO

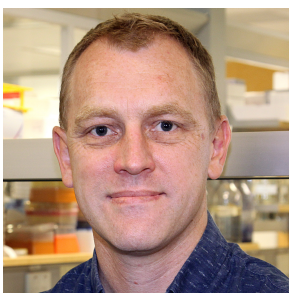
Neurovascular System Function and its Relationship with Aging and Alzheimer's Disease

The goal of this project is to test a new way of analyzing the data from positron-emission tomography (PET) brain scan images to examine changes in brain metabolism and the loss of blood-brain barrier (BBB) integrity as a consequence of aging and AD.

www.brightfocus.org/grant/A2017272S

Drug Discovery and New Treatment Approaches

The painstaking work to uncover Alzheimer's pathology has paid off with a bounty of 'druggable targets', but a lack of effective drugs. With a disease as complex as this one, it's very helpful to find multiple points where it may be possible to slow or halt Alzheimer's progress. ADR grantees are discovering and contributing to early development of molecules and compounds aimed at reducing toxic protein buildup and improving clearance of toxic particles and delivering treatments to brain areas. Support of these exploratory projects at early stages is essential to get the next generation of drugs and therapies to patients.



Brett Collins, PhD (7/1/18 - 6/30/21)
The University of Queensland, Brisbane, Australia

Stabilizing the Retromer Protein Complex with Molecular Chaperones for Alzheimer's and Parkinson's Diseases

Cellular processes regulating protein turnover could be manipulated to prevent the build-up of the toxic beta amyloid peptides that cause neurological failure. This work will develop novel small molecules and peptides with the goal to enhance this protein turnover in neurons, and provide a starting point for designing new AD drugs.

www.brightfocus.org/grant/A2018627S



Shermali Gunawardena, PhD (7/1/18 - 6/30/21)
SUNY Buffalo, NY

A Novel Therapeutic Device to Clear Axonal Blocks to Prevent Alzheimer's Disease

The research team is using a highly innovative approach to develop synthetic biomolecules that will deliver therapeutics to specific sites within the brain to modify defects that activate AD pathways.

www.brightfocus.org/grant/A2018509S

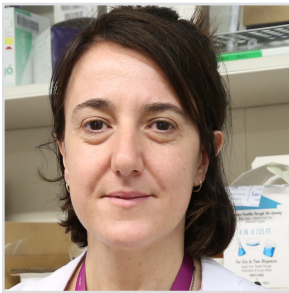


Mark Henkemeyer, PhD (7/1/16 - 6/30/19)
University of Texas Southwestern Medical Center, Dallas

Identification of Novel Compounds to Promote Synapse Health and Prevent Alzheimer's Disease

The researcher will use novel high-throughput screens of small drug-like chemical libraries for compounds that disrupt the ability of EphB, an important protein for synapse function, to bind with beta amyloid. This project is looking for a new class of medicines that will halt the destruction of synapses and avert memory loss.

www.brightfocus.org/grant/A2016345S



Mar Hernandez Guillamon, PhD (7/1/17 - 6/30/20)
Vall d'Hebron Research Institute, Barcelona, Spain

Modulating Brain Cholesterol to Treat Alzheimer's Disease

The aim of this proposal is to determine the effect of a new treatment based on the administration of a natural modified protein, the ApoA-I-Milano variant, able to mobilize lipids in a transgenic mouse model of AD.

www.brightfocus.org/grant/A2017243S



Tsuneya Ikezu, MD, PhD (7/1/16 - 6/30/19)
Boston University, MA

Validation of Drug Candidates for Enhancing the Clearance of Amyloid Accumulation and Dead Cells in the Alzheimer's Disease Brain

Focuses on identifying a drug to enhance the clearance of unwanted build-up of proteins and dead cells in AD by targeting the microglia protein TREM2.

www.brightfocus.org/grant/A2016551S



Patrick Kehoe, PhD (7/1/16 - 6/30/19)

University of Bristol, England, United Kingdom

Co-Principal Investigators **Scott Miners, PhD & Mark Good, PhD**

Helping the Brain to Fight Back Against Alzheimer's—Using Old Drugs for New Purposes

Investigate whether a drug already developed, but unlicensed for use in people for blood pressure, and previously not considered in AD, can protect against both cognitive decline and tissue damage in an established mouse model of AD.

www.brightfocus.org/grant/A2016582S

This grant is made possible in part by a bequest from the Trust of Edward & Irene Schlosser.



Stephen Martin, PhD (7/1/16 - 8/30/18)

The University of Texas at Austin

A New Approach to Treating Alzheimer's Disease

This project will explore a novel strategy to treat AD by targeting a biological pathway different from those of all existing drugs and known clinical candidates. If successful, a compound will be identified that holds promise for those suffering from AD, thus representing a new way to treat this devastating disease.

www.brightfocus.org/grant/A2016443S



Randy McIntosh, PhD (7/1/17 - 6/30/20)

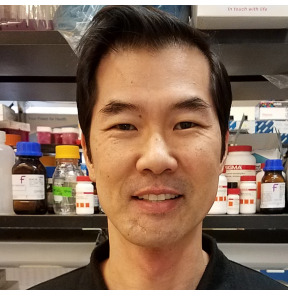
Baycrest Centre for Geriatric Care, Toronto, Canada

Co-Principal Investigator **Kelly Shen, PhD**

Building a Personalized Virtual Brain with Alzheimer's Disease to Guide Clinical Decisions

This research provides software to "reconstruct" the brain, building models of different dementias to characterize the unique features of each disease and cognitive impairment. As this work progresses, it will be used to evaluate the potential of therapeutic interventions.

www.brightfocus.org/grant/A2017286S



Daniel Pak, PhD (7/1/17 - 6/30/20)

Georgetown University, Washington, DC

Testing a Novel Amyloid-Promoting Factor as an Alzheimer's Disease Therapy

This project studies how a novel protein, PLK2, stimulates the production of pathogenic amyloid fragments and tests the effectiveness of new drugs in mouse models of AD.

www.brightfocus.org/grant/A2017508S



Angèle Parent, PhD (7/1/17 - 6/30/20)
University of Chicago, Illinois

Targeting APP Intracellular Fragment to Improve Memory and Reduce Beta Amyloid Burden in Alzheimer's Disease

This research tests the hypothesis, using mouse models of AD, that production of an intracellular fragment originating from APP [amyloid precursor protein] could initiate signaling events that benefit memory process and reduce beta amyloid generation when delivered through modified viral vectors.

www.brightfocus.org/grant/A2017443S



Ana Pereira, MD (7/1/16 - 6/30/19)
Icahn School of Medicine at Mount Sinai, New York, NY

Enhancing Glutamate Levels as a Way to Treat Alzheimer's Disease

Glutamate needs to be at the correct place and time to allow efficient neuronal communication, and to avoid toxicity. Glutamate levels are regulated by GLT-1. The researchers investigate the mechanisms through which GLT-1 becomes dysregulated.

www.brightfocus.org/grant/A2016478S

This grant is made possible by the support from the Ping Y. Tai Foundation.

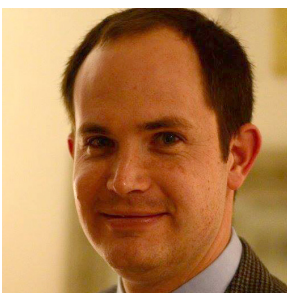


Dianne Perez, PhD (7/1/16 - 6/30/19)
Cleveland Clinic, Ohio

Novel Drugs Against a New Receptor Target to Treat Alzheimer's Disease

The researchers of this project will validate a novel target for treatment of AD, make new drugs that are selective for this target, and then test these drugs in an animal model of the disease.

www.brightfocus.org/grant/A2016272S



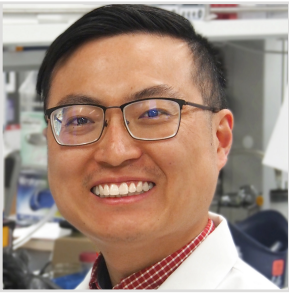
Jeremy Strain, PhD (7/1/18 - 6/30/20)
Washington University, St. Louis, MO

FELLOWSHIP

How Connections in the Brain Break Down in Alzheimer's Disease

This project is directed at understanding how and where structural connections in the brain are damaged in two variants of AD. Two known causes of 'white matter' (brain nerve fibers) damage commonly seen in this population will be detected by combining different neuroimaging and analytical techniques.

www.brightfocus.org/grant/A2018817F



Chao Wang, PhD (7/1/18 - 6/30/20)

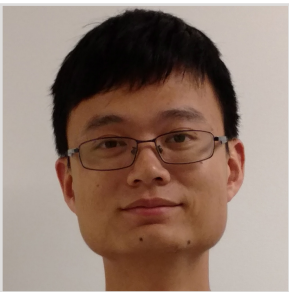
FELLOWSHIP

Washington University School of Medicine, St. Louis, MO

A New Approach to Treating Tauopathy by Lowering Apolipoprotein E Level

The researchers will determine if decreasing apolipoprotein E (apoE) levels in the brain can alter tau aggregation and tau-induced neurodegeneration, and we will also try to determine how apoE exerts its effects on tau.

www.brightfocus.org/grant/A2018128F



Yingjun Zhao, PhD (7/1/18 - 6/30/20)

FELLOWSHIP

Sanford Burnham Prebys Medical Discovery Institute, La Jolla, CA

A Novel Approach for Memory Improvement in Alzheimer's Disease

The goal of this project is to understand how proteins that are overproduced in AD, such as apoptosis, can alter the brain to disrupt communication between neurons to cause problems with memory.

www.brightfocus.org/grant/A2018214F

This grant is made possible in part by the support from the J.T. Tai Foundation.



Intervening with Genetic Risk Factors

Late-onset Alzheimer's results from a complex interaction of genes, environment, and individual biology (i.e., a person's age, gender, and underlying health). So far, approximately 30 genes have been found to influence Alzheimer's disease (AD) risk in various ways, such as dysregulating immune function; introducing damaging proteins into the brain; disrupting metabolism; lowering clearance of toxic wastes; and more. Possession of a variant of the apolipoprotein E gene (APOE4) confers a strong risk for earlier onset for AD. This is a rare, but difficult, conditional for certain families. While the promise of individualized drug therapy and gene editing remain in the future, our scientists are performing the necessary work to intervene with AD risk factors to support healthy aging.



Joseph Castellano, PhD (7/1/18 - 6/30/21)
Icahn School of Medicine at Mount Sinai, New York, NY

ApoE4's Effects on Blood Proteins and Brain Function in Alzheimer's Disease

The researchers will directly investigate how manipulating proteins in the blood influences the ability of the risky APOE4 gene to influence development of AD and the extent to which it can be rescued with more neutral forms of the gene.

www.brightfocus.org/grant/A2018213S



Holly Cukier, PhD (7/1/18 - 6/30/21)
University of Miami, Miller School of Medicine, FL
Co-Principal Investigator **Derek Dykxhoorn, PhD**

Elucidating the Roles of ABCA7 in Neurons and Glia Created from Alzheimer's Patients' Adult Stem Cells

This project seeks to investigate the role of a gene shown to be a risk factor for AD, ABCA7, and the consequence of a mutation that was first identified in African Americans. Using two cell types from induced pluripotent stem cells generated from African Americans, the researchers will investigate how this deletion may affect the normal way neurons and microglia develop, and how that may lead to AD.

www.brightfocus.org/grant/A2018197S



Pierre De Rossi, PhD (7/1/17 - 6/30/19)
University of Chicago, Illinois

FELLOWSHIP

BIN1 as a Genetic Risk Factor in Alzheimer's Disease Pathology

Investigates how the BIN1 gene, one of the most significant late-onsite AD risk factors, functions in the development and progression of AD using a specially created mouse model of the disease.

www.brightfocus.org/grant/A2017366F



Joachim Herz, MD (7/1/16 - 6/30/19)
University of Texas Southwestern Medical Center, Dallas

Targeting the Molecular Cause of the ApoE4 Risk in Alzheimer's Disease

Based on prior discovery of a novel drug target, NHE6, and on a new mechanism by which ApoE4 weakens synaptic strength in neurons, this proposal aims to establish the necessary mechanistic and infrastructural baseline to screen for NHE6 specific inhibitors and for discovering novel potential methods to target NHE6 function.

www.brightfocus.org/grant/A2016396S



Catherine Kaczorowski, PhD (7/1/16 - 6/30/19)
The Jackson Laboratory, Bar Harbor, ME

A New Method to Identify Genes Critically Involved in Alzheimer's Disease

This proposal seeks to identify genes that modify the onset and severity of AD in a well-characterized, genetically diverse mouse population. Subsequent gene therapy will be used to validate identified genes and rescue/treat memory failure in a mouse model of AD.

www.brightfocus.org/grant/A2016397S



Celeste Karch, PhD (7/1/18 - 6/30/21)
Washington University School of Medicine, St. Louis, MO

Defining the Role of CXCR4 in Alzheimer's Disease

Common variants in a chemokine receptor found in microglia, CXCR4, contribute to tauopathies. The objective of this study is to begin to determine how CXCR4 drives AD and whether an existing drug improves tauopathy outcomes in a mouse model of AD.

www.brightfocus.org/grant/A2018349S



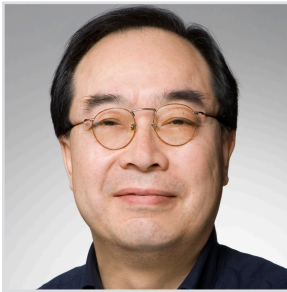
Chaeyoung Kim, PhD (7/1/17 - 6/30/19)
Gladstone Institutes, San Francisco, CA

FELLOWSHIP

The Effect of ApoE4 on Mitochondrial Function in Alzheimer's Disease

This project aims to determine how ApoE4, the major genetic risk factor for late-onset Alzheimer's, and its neurotoxic fragments, disrupt mitochondrial activity to drive neurodegeneration. Mitochondria are the energy powerhouses of cells, where disrupting the power supply could lead to disease.

www.brightfocus.org/grant/A2017214F



Joseph H. Lee, DrPH (7/1/15 - 12/31/18)
Columbia University, New York, NY

Co-Principal Investigator **Richard Mayeux, MD**

Genome Search for Genetic Modifiers of Alzheimer's Disease Age of Onset

The main goal of this project is to identify novel genes that modify the effects of the G206A mutation in the PSEN1 gene, which causes early-onset AD. For these G206A mutation carriers who were identified in Puerto Rico, their age at onset can range from the 40s to 70s. By identifying genes that contribute to delayed age at onset, we hope to better understand ways to protect against AD.

www.brightfocus.org/grant/A2015633S

This grant is made possible in part by a bequest from the Estate of Frederick J. Pelda



Timothy Miller, MD, PhD (7/1/18 - 6/30/21)

Washington University School of Medicine, St. Louis, MO

Decreasing a Novel Genetic Risk Factor for Alzheimer's and its Effect on Pathology and Cognition in Mouse Models

A gene involved in inflammatory responses (TREM2) increases risk for developing AD and mediates the accumulation of beta amyloid in the brains of experimental mouse models. These researchers will use TREM2-lowering antisense oligonucleotides to explore new avenues for future treatments for AD.

www.brightfocus.org/grant/A2018169S



Farid Rajabli, PhD (7/1/18 - 6/30/20)

FELLOWSHIP

University of Miami, Miller School of Medicine, FL

Evaluating the Role of Ethnicity, Race, and Genetic Ancestry in Alzheimer's Disease

The goal of this project is to evaluate the role of race/ethnicity and examine ancestry-specific genetic variants in a multi-ethnic dataset. The researchers aim to identify novel areas of the genome that correlate risk of AD with genetic ancestry.

www.brightfocus.org/grant/A2018556F



Jeffery Vance, MD, PhD (7/1/18 - 6/30/21)

University of Miami, FL

Co-Principal Investigators **Margaret Pericak-Vance, PhD,**
Gary Beecham, PhD & Anthony Griswold, PhD

Using Population Sequence Differences to Identify a DNA Change that Reduces the Risk of ApoE for Developing Alzheimer's Disease

Carriers of ApoE4 with African ancestry have a lower risk for AD than carriers of European ancestry. These researchers have isolated the region that protects African ApoE4 carriers and will use this study to identify DNA changes among the populations that can be tested in biological models.

www.brightfocus.org/grant/A2018425S



Huda Zoghbi, MD (7/1/16 - 6/30/19)

Baylor College of Medicine, Houston, TX

Co-Principal Investigator **Juan Botas, PhD**

A Genetic Screen to Identify New Drug Targets for Alzheimer's Disease

This project will identify new therapeutic targets using an unbiased, high-throughput screen using two different assay systems in parallel (human neuronal cell lines and fruit flies expressing human APP) to identify those proteins whose reduction results in lower levels of APP, and rescues neuronal degeneration in flies.

www.brightfocus.org/grant/A2016151S

Lifestyle Changes to Reduce Alzheimer's Risk & Impact

The conditions for Alzheimer's disease (AD) may develop over a lifetime, and the risks are not necessarily confined to one's later years. The factors contributing to sporadic (i.e., not genetically inherited) late-onset forms of AD begin to develop throughout middle age, when conditions such as diabetes, cardiovascular disease, sedentary lifestyle, and declining mental activity (reading, playing music, or learning new things) may begin to take their toll. Heart disease, lipid disorders, sleep problems, depression, and prior brain trauma are being investigated as risk factors. To offset increased risk, beneficial lifestyle modifications, including exercise and diet aimed at lowering lipids and cholesterol, improved cardiovascular health, and even support for gut bacteria, are being explored as possible ways to delay disease onset and severity.



Jason Brandt, PhD (7/1/16 - 6/30/19)
Johns Hopkins University, Baltimore, MD

A High Fat, Low Carbohydrate Diet for MCI and Early Alzheimer's Disease

This study will test whether a diet very low in starches and sugars, and very high in fat (which has been used to treat other brain disorders) may be useful to treat AD.

www.brightfocus.org/grant/A2016073S

This grant is made possible in part by support from the Jerome Jacobson Foundation.



Sarah Fritschi, PhD (7/1/17 - 9/30/18) *FELLOWSHIP*
Washington University School of Medicine, St. Louis, MO

Understanding the Interplay between Sleep and Alzheimer's Disease

By switching sleep on and off in a specially created mouse model, this project will assess whether sleep disturbances and poor sleep quality are an early factor that contributes to the risk of developing AD.

www.brightfocus.org/grant/A2017114F



Jennifer Gatchel, MD, PhD (7/1/16 - 6/30/19) *FELLOWSHIP*
McLean Hospital & Massachusetts General Hospital, Boston, MA

Depressive Symptoms, Proteins Beta Amyloid and Tau, and Neuronal Network Activity in Prodromal and Early Alzheimer's Disease

This study will involve novel investigation of whether psychiatric and behavioral symptoms relate to build-up of beta amyloid, tau, and changes in brain circuit connectivity patterns in living, cognitively normal older adults and those in the pre-AD and early AD stages. Findings from this work could enhance prevention and treatment approaches of late life psychiatric symptoms and AD.

www.brightfocus.org/grant/A2016434F

This grant is made possible by a bequest from the Howlett Revocable Trust.



Majken Jensen, PhD (7/1/17 - 6/30/20)
Harvard University, Boston, MA

Using Blood Samples to Assess the Role of Nutritional Factors in Alzheimer's Risk

In this project, key healthy dietary patterns will be identified that can form the foundation of dietary recommendations to lower a risk of Alzheimer's Disease.

www.brightfocus.org/grant/A2017290S



Brendan Lucey, MD (7/1/16 - 6/30/19)
Washington University School of Medicine, St. Louis, MO

Sleep Quality and Decreasing Beta Amyloid Levels in the Human Brain

This study proposes to answer several of these questions: 1) Does poor sleep quality increase beta amyloid and 2) Does improving sleep quality in poor sleepers decrease beta amyloid?

www.brightfocus.org/grant/A2016180S



Robert Newton, Jr., PhD (7/1/17 - 6/30/20)
Pennington Biomedical Research Center, Baton Rouge, LA

Program for African American Cognition and Exercise (PAACE)

The goal of this project is to increase the knowledge of the effects of a physical activity program on dementia prevention in African-American adults.

www.brightfocus.org/grant/A2017547S



Stephanie Rainey-Smith, PhD (7/1/18 - 6/30/20) *FELLOWSHIP*
Edith Cowan University, Perth, Western Australia, Australia

Can Good Sleep Prevent Alzheimer's Disease?

This study will explore the relationship between sleep, memory, thinking, and markers of brain health by investigating whether improved sleep causes better memory and thinking, slows protein build up in the brain, and slows the shrinking of the brain.

www.brightfocus.org/grant/A2018402F



Donald Redelmeier, MD (7/1/15 - 6/30/19)
Sunnybrook Research Institute, Toronto, Ontario, Canada

A New Way to Recover from a Concussion and Avoid Alzheimer's Disease

The researchers will perform a computerized study with tens of thousands of healthcare databases to test whether statin treatment decreases the risk of subsequent dementia in seniors following a concussion.

www.brightfocus.org/grant/A2015284S



Na Zhao, MD, PhD (7/1/18 - 6/30/20) *FELLOWSHIP*
Mayo Clinic, Jacksonville, FL

The Regulation of ApoE on Insulin Signaling and Energy Metabolism in the Brain of Alzheimer's Disease

The project uses mouse models to test whether insulin can change how brain cells utilize blood sugar in individuals with the APOE4 genotype. The findings will be very useful in understanding how ApoE4 impairs brain health and how we can use insulin as an effective treatment for AD.

www.brightfocus.org/grant/A2018777F

Pioneering New Ways to Image and Assess the Brain

Solving Alzheimer's disease (AD) requires knowing as much as possible about how the brain works throughout our lifespan, including normal aging, and how its different regions and circuitry are ravaged by the disease over time. Thanks to pioneering new methods and techniques, diagnosing Alzheimer's disease and other forms of dementia no longer requires postmortem examination. The hallmarks of the disorder, plaques and tangles, can be imaged by researchers and discussed with patients and caregivers. Alzheimer's Disease Research grantees are working to develop and validate high-definition imaging that will differentiate between early disease and normal aging, and support earlier diagnoses, potentially before symptoms occur. Imaging will help doctors assess the impact of beta amyloid, tau, and other aspects of AD on brain circuitry. Ultimately these techniques will help us monitor brain health and the impact of treatment.



Iman Aganj, PhD (7/1/16 - 6/30/20)

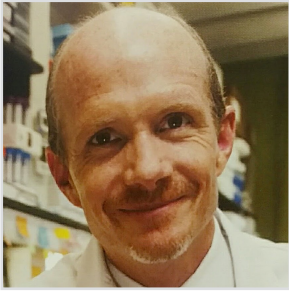
Massachusetts General Hospital, Boston

Imaging Biomarkers for Alzheimer's: New Methods to Account for Indirect Brain Connections

The goal of the proposed project is to develop and validate novel computational methods - such as those accounting for indirect neural pathways - and subsequently derive more accurate AD imaging biomarkers based on brain connectivity with different versions of MRI.

www.brightfocus.org/grant/A2016172S



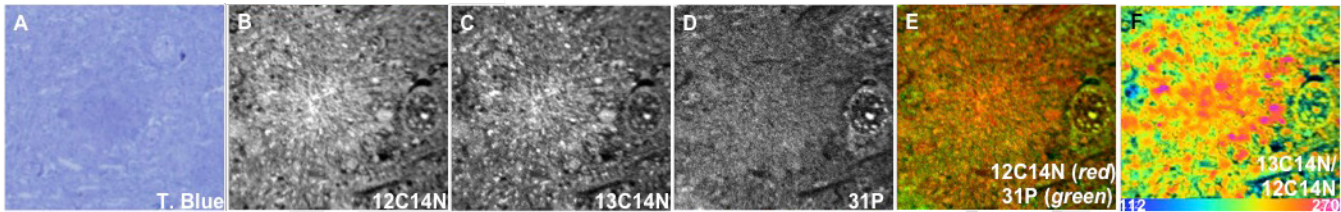


Randall Bateman, MD (7/1/17 - 9/30/20)
Washington University School of Medicine, St. Louis, MO
 Co-Principal Investigators **Norelle C. Wildburger, PhD**
 & **Robert Schmidt, MD, PhD**

A New Way to Image Amyloid Plaque Growth in Human Alzheimer's Disease

Critical to the development of therapeutics that may treat and even cure AD is an understanding of beta amyloid dynamics in the human brain. This project uses the most advanced imaging technology to study the rate of plaque pathology in patients.

www.brightfocus.org/grant/A2017081S



Courtesy of Dr. Randall Bateman's Lab

A plaque build-up in a mouse model of AD has been photographed four different ways (A-D) to see cell membranes (E, green) and learn how the plaque is formed (F).



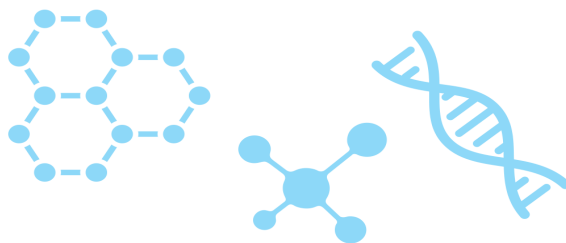
Ryan Darby, MD (7/1/17 - 6/30/19)
Vanderbilt University, Nashville, TN

FELLOWSHIP

Neuroimaging and Behavioral Correlates of Delusions and Hallucinations in Alzheimer's Disease

Delusions and hallucinations commonly occur in Alzheimer's disease, causing considerable distress for patients and families. The goal of this research is to determine why these symptoms arise, using different types of brain scans and behavioral tests.

www.brightfocus.org/grant/A2017226F





Sara Gallant, PhD (7/1/18 - 6/30/20)
University of Southern California, Los Angeles

FELLOWSHIP

Arousal-Induced Memory Selectivity in Aging and Alzheimer's Disease

The locus coeruleus is a small brainstem region that becomes active during highly emotional or arousing events and is one of first sites to develop AD-related tau pathology. Using brain imaging, this research will examine functioning of the locus coeruleus under emotional "fight-or-flight" arousal as well as its relation to selective memory processes in aging and AD.

www.brightfocus.org/grant/A2018449F

This grant was made possible in part by support from Alzheimer's Los Angeles.



Lea Grinberg, MD, PhD (7/1/17 - 6/30/20)

University of California, San Francisco

Co-Principal Investigator **Duygu Tosun, PhD**

A Neuroimaging Biomarker for Asymptomatic Alzheimer's Disease

This project will develop a biologically-validated clinical MRI template for detecting shrinkage in the locus coeruleus (the part of the brain that responds to stress and panic). This brainstem nucleus is especially vulnerable and earliest-damaged in AD.

www.brightfocus.org/grant/A2017560S

This grant was made possible in part by support from The Carl and Judy Moore Charitable Trust.



Xiong Jiang, PhD (7/1/16 - 6/30/19)

Georgetown University, Washington, DC

A Novel Non-Invasive MRI-Based Biomarker of Early Stages of Alzheimer's Disease

Develop and validate multimodality MRI techniques, a collection of individual techniques which, when they are added together, can help to detect and quantify Alzheimer's progression, even when it occurs without behavioral symptoms.

www.brightfocus.org/grant/A2016251S



Terrance Kummer, MD, PhD (7/1/17 - 6/30/20)
Washington University School of Medicine, St. Louis, MO

An MRI Fingerprint of Brain Circuit Breakdown in Alzheimer's Disease

In AD, neural circuits begin to break down throughout the brain over many years, yet remain invisible to clinical imaging techniques like MRI until the disease is so advanced that it is likely irreversible. The goal of this project is to develop new MRI approaches that can reveal these microscopic circuit injuries in model systems and in patients suffering from AD.

www.brightfocus.org/grant/A2017084S

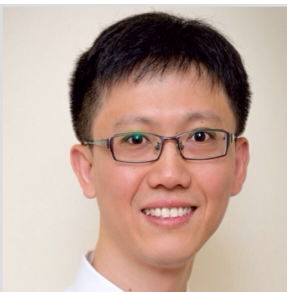


Peter Tessier, PhD (7/1/17 - 8/31/20)
University of Michigan, Ann Arbor
Co-Principal Investigators **Jennifer Cochran, PhD & Mehrdad Shamloo, PhD**

New Tau Imaging Agents for Early Diagnosis of Alzheimer's Disease

These scientists will use an innovative design and evolution method for generating imaging probes specific for tau. These novel probes will be used to image toxic protein particles in mouse models of AD.

www.brightfocus.org/grant/A2017395S



Yong Wang, PhD (7/1/17 - 6/30/20)
Washington University School of Medicine, St. Louis, MO

A New Imaging Method to Measure White Matter Damage and Inflammation in Alzheimer's Disease

A PET-MRI method has been commonly used to identify tumors or strokes in patients with other diseases. These researchers adapt this method to measure the brain's injury and immune response in patients who have AD, and to get a sense of the problems developing in their brain before memory problems occur.

www.brightfocus.org/grant/A2017330S



Laura Wisse, PhD (7/1/16 - 1/30/19)
University of Pennsylvania, Philadelphia

FELLOWSHIP

Separating Early Alzheimer's Disease and Aging Effects in Search of Markers to Track Alzheimer's Treatment Effects

To separate the effects of preclinical Alzheimer's disease from normal aging, these researchers will investigate the medial temporal lobe with a precise, high resolution MRI scanner, which will allow investigation of smaller regions within this brain region which, in contrast to the cruder measurements, are expected to show differential effects of aging and preclinical Alzheimer's disease.

www.brightfocus.org/grant/A2016432F

This grant is made possible in part by a bequest from the Trust of Elenore Lundeen.

New Methods and Resources to Fight Alzheimer's

The world is alive with emerging technologies and resources that can be brought together in the quest to end Alzheimer's. These range from using artificial intelligence to detect Alzheimer's in eye tissue, to encouraging citizen-scientists worldwide to play the first-ever video game assisting with Alzheimer's research. We are privileged to be a driving force behind new collaborations and projects that will move research forward into exciting new territory. Ultimately these techniques will help us monitor brain health and the impact of treatment.



Special Opportunity Grant
Ann-Charlotte Granholm-Bentley, PhD, DDS (7/1/18 - 6/30/21)
University of Denver, CO

International Brain Bank for Down Syndrome-Related Alzheimer's Disease

The focus of this special project is to develop a strong collaborative network between six different research groups, with the long-term goal to determine the neurobiological mechanisms underlying the onset of AD-type dementia in Down syndrome.

www.brightfocus.org/grant/CA2018010



Special Opportunity Grant
Pietro Michelucci, PhD (7/1/17 - 6/30/19)
Human Computation Institute, Ithaca, NY

EyesOnALZ: Crowdsourced Research to Accelerate Future Alzheimer's Treatments

This project accelerates existing Alzheimer's treatment research that would otherwise take decades to complete by transforming a time-consuming laboratory task into an online game called "Stall Catchers" that anyone can play. This project also provides a hands-on way for people affected by Alzheimer's disease to make an impact in their own future or in that of a loved one, while also educating the general public about the disease. This is the first citizen science project supporting Alzheimer's research.

www.brightfocus.org/grant/CA2017606

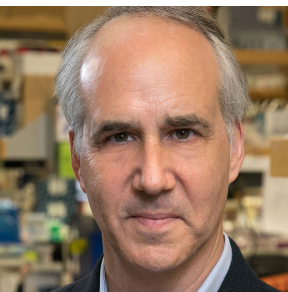


Carol Yim Lui Cheung, PhD (7/1/18 - 6/30/21)
The Chinese University of Hong Kong, China

Recognizing "Retinal Fingerprint" for Alzheimer's Disease Using Artificial Intelligence

In this study, an artificial intelligence will "learn" structural patterns in the eyes of AD patients using deep learning methods to create a "retinal fingerprint" of the disease. This technique only requires a routine eye-check, and represents an inexpensive, non-invasive, efficient and accessible method to screen for AD.

www.brightfocus.org/grant/A2018093S



Daniel Geschwind, MD, PhD (7/1/18 - 6/30/21)
University of California, Los Angeles
Co-Principal Investigator **Jessica Rexach, MD, PhD**

Identifying Disease Mechanisms in Neurodegeneration Using Genomics and Bioinformatics

Using cutting edge technology, these researchers will profile the different cells of the dementia brain at unprecedented resolution to understand the complexity of the brain's different cell types, which presents a unique challenge to scientific inquiry.

www.brightfocus.org/grant/A2018700S



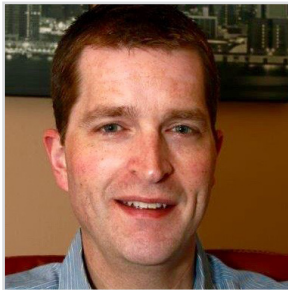
Joshua Grill, PhD (7/1/18 - 6/30/21)

University of California, Irvine

Improving Recruitment to Prodromal Alzheimer's Disease Clinical Trials

Most Alzheimer's clinical trials now enroll patients with mild cognitive impairment, which in many cases may be an early form of AD. This project will identify the challenges to enrolling these patients in clinical trials and develop methods to improve recruitment to these critical studies.

www.brightfocus.org/grant/A2018405S



Jason Hassenstab, PhD (7/1/18 - 6/30/21)

Washington University School of Medicine, St. Louis, MO

Rapid Assessment of Cognition using Smartphones to Track Early Changes in Alzheimer's Disease

One of the biggest challenges we face in Alzheimer's disease research and clinical trials is tracking the subtle cognitive changes that appear years before a clinical diagnosis. Standard cognitive tests were not designed for this purpose and thus have poor sensitivity and poor reliability. This study will adapt cognitive measures for rapid and repeatable administration on participant's personal smartphones with the goal of producing highly reliable assessments.

www.brightfocus.org/grant/A2018202S



Sanjeev Kumar, MD (7/1/18 - 6/30/21)

Centre for Addiction and Mental Health, Toronto, Ontario, Canada

Co-Principal Investigators **Tarek Rajji, MD, Daniel Blumberger, MD, Zafiris J. Daskalakis, MD, Corinne E. Fischer, MD, Nathan Herrmann, MD, Benoit H. Mulsant, MD, Bruce G. Pollock, MD & Reza Zomorodi, PhD**

Identifying Disease Mechanisms in Neurodegeneration Using Electrophysiology

Agitation and aggression affect the majority of patients with AD. Medications used to treat these symptoms are associated with many side effects. This project will use magnetic brain stimulation and electroencephalography to understand the mechanisms of agitation and use a non-invasive brain stimulation technique called Transcranial Direct Current Stimulation (tDCS) to treat it.

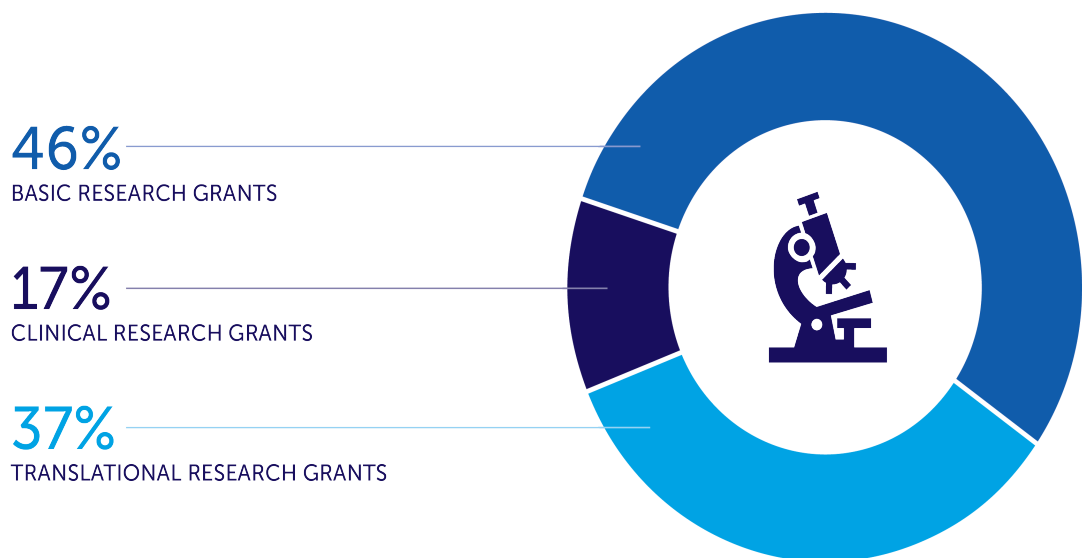
www.brightfocus.org/grant/A2018667S

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➤ BrightFocus Grants at a Glance



Basic - Research that aims to better understand how a disease happens, and to obtain new ideas of how to stop the disease.

Clinical - Research involving volunteer participants to test the safety and effectiveness of drugs, devices, or other treatment candidates.

Translational - Research to move findings from the lab bench to the "bedside" by testing potential treatments.



Alzheimer's Disease Research, a BrightFocus Foundation program