



**Alzheimer's  
Disease  
Research**

# 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, personality, and more, 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, the Alzheimer's Disease Research (ADR) program, which began in 1985, has awarded nearly \$130 million in research to understand and cure this disease. With our grants, 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 innovative. The generosity of our donors helps make this possible.

The current portfolio of active projects within the ADR program is expansive. As an organizational tool for this yearbook, our 105 projects have been arranged by broad themes that align with the international categorization of *Common Alzheimer's Disease Research Ontology* classification for the *International Alzheimer's Disease Research Portfolio* database for tracking and implementing the research goals of the *National Plan to Address Alzheimer's Disease*, and used by research funding agencies around the world.

- **Blood and the Brain in Dementia**
- **Genomics: DNA Blueprint for Alzheimer's Disease**
- **Battling Amyloid Beta**
- **Tangling with Tau**
- **Biology of Fats and Proteins (ApoE and Lipids)**
- **Cells and Circuits**
- **Immunity and Inflammation**
- **Biomarkers**
- **Finding New Drugs and Treatments for Alzheimer's Disease**
- **Translational Research and Clinical Interventions**
- **Research Tools and Resources**

*Note: The funding statistics and inclusion of research grants in this yearbook are based upon projects that were active as of July 1, 2019. Current as of July 15, 2019.*

## Blood and the Brain in Dementia

As a neurodegenerative disease, Alzheimer's is known for damaging neurons, which are the nerve cells of the brain. Neurons depend on oxygen and glucose carried through the brain's blood vessels, or vascular system. Their needs are great because the brain consumes more energy than any other human organ, up to 20 percent of the body's total supply. The brain relies heavily on an intricately laced system of arteries, veins and capillaries that, in adult brains, stretches an estimated 100 miles in length. For protection, the brain's circulatory system is sealed off from that of the rest of the body by a special blood-brain barrier that helps prevent bacteria, viruses, and other toxic substances from entering.

Together, the brain's circulatory system and protective barrier are important to Alzheimer's research because it is key to keeping neurons healthy.



**Peter Abadir, MD** (7/1/19 - 6/30/22)  
*Johns Hopkins University, Baltimore, MD*

Brain Changes in Alzheimer's Disease, Role of a Blood Pressure System

*Angiotensin receptors are found on brain cells and play an important function in brain vital functions. This study will examine changes in these receptors in brain cells in patients with Alzheimer's dementia. This project will also study the impact of a class of drugs that target these receptors and are commonly used to treat high blood pressure.*

[www.brightfocus.org/grant/A2019634S](http://www.brightfocus.org/grant/A2019634S)



**Alexandre Bonnin, PhD** (7/1/19 - 6/30/22)  
*University of Southern California, Los Angeles*  
Co-Principal Investigator **Axel Montagne, PhD**

Prenatal Inflammation Programs Alzheimer's Disease Risk Later in Life

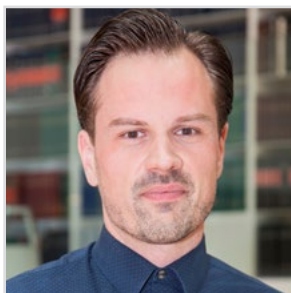
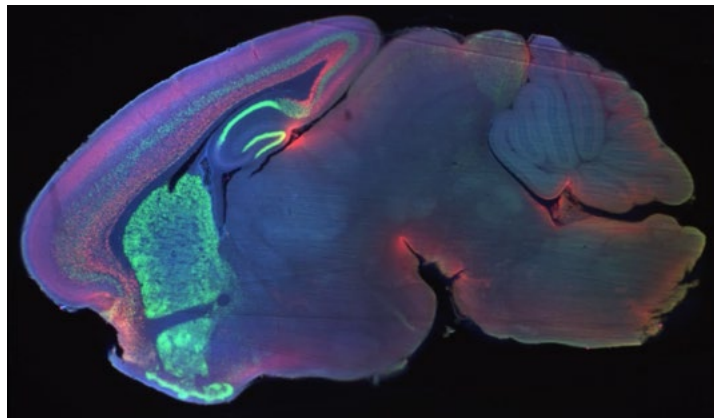
*Recent animal model studies suggest a causal link between inflammation during embryonic development and risk of AD-like neuropathology later in life. In light of recent research demonstrating that blood-brain barrier breakdown in the adult brain is a core cause of AD, the hypothesis is that inflammation-mediated disruption of blood-placenta and blood-brain barriers are key factors in the developmental origins of AD.*

[www.brightfocus.org/grant/A2019279S](http://www.brightfocus.org/grant/A2019279S)

**Courtesy of Dr. Alexandre Bonnin's Lab**

Different types of neurons have been labeled with different florescent colors in this picture of a mouse model brain.

One of the most important areas for memory, the hippocampus, can be identified by the bright green lines showing its characteristic layered structure.

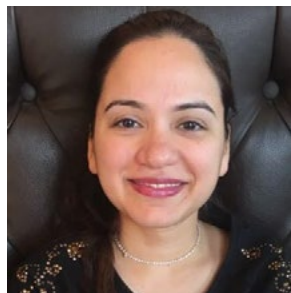


**Daniel Bos, MD, PhD** (7/1/17 - 12/30/19) **FELLOWSHIP**  
**Erasmus Medical Center, Rotterdam, Netherlands**

**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](http://www.brightfocus.org/grant/A2017424F)**



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

**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](http://www.brightfocus.org/grant/A2018165F)**



**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](http://www.brightfocus.org/grant/A2017290S)**



**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](http://www.brightfocus.org/grant/A2017094S)



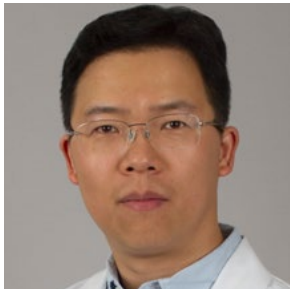
**Alex Smith, PhD** (7/1/18 - 6/30/21)

**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](http://www.brightfocus.org/grant/A2018351S)



**Zhen Zhao, PhD** (7/1/19 - 6/30/22)

**University of Southern California, Los Angeles**

Understanding the Vascular Link Between Traumatic Brain Injury and Alzheimer's Disease.

*Traumatic brain injury (TBI) is a leading cause of injury deaths and disabilities in the United States and the most robust environmental risk factor for AD. Vascular impairment is also a hallmark of the pathological events after TBI, including local edema, blood-flow reduction and breakdown of blood-brain barrier, which may significantly increase Alzheimer's risk. This project investigates the link between cerebrovascular impairment induced by TBI and its impact on the susceptibility to AD in animal models.*

[www.brightfocus.org/grant/A2019218S](http://www.brightfocus.org/grant/A2019218S)

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

## Genomics: DNA Blueprint for Alzheimer's

Genes are the “master blueprint” that instructs our cells to make unique proteins which in turn build, operate, and repair human tissue. Humans have an estimated 24,000 genes along our 23 matched pairs of chromosomes (46 in all), and “genomics” refers to the field that studies all of them at once. Even slight changes in a gene on one or both chromosomes can produce a protein that functions abnormally, possibly causing or increasing/decreasing the risk of a disease such as Alzheimer's. However, only one type of Alzheimer's disease (AD)—early-onset forms, representing less than 10 percent—can be traced consistently to changes, or mutations, in identified genes. The remaining 90 percent—late-onset AD—is associated with small genetic irregularities occurring throughout the genome. Using powerful and fast new technologies, researchers working in genomics look for variations, patterns, and interactions among all genes in hundreds of thousands of people. So far, several dozen “regions of interest” have been identified, only it gets complicated because gene signaling can be turned “on” or “off” by additional factors, such as environment and lifestyle. Thanks to genomics, all this is being sorted out, and ultimately will help provide answers to basic questions, such as: What causes AD to start? How do genes interact with environment to raise or lower Alzheimer's risk? Who is most at risk and apt to benefit from new treatments? What's most likely to work in any given individual (“personalized medicine”)?



**Holly Cukier, PhD** (7/1/18 - 6/30/21)

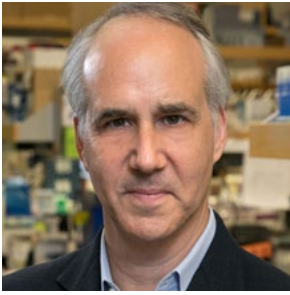
**University of Miami, FL**

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](http://www.brightfocus.org/grant/A2018197S)**



**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](http://www.brightfocus.org/grant/A2018700S)



**Catherine Kaczorowski, PhD** (7/1/16 - 6/30/20)  
*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](http://www.brightfocus.org/grant/A2016397S)



**Farid Rajabli, PhD** (7/1/18 - 6/30/20)  
*University of Miami, FL*

*FELLOWSHIP*

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](http://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**

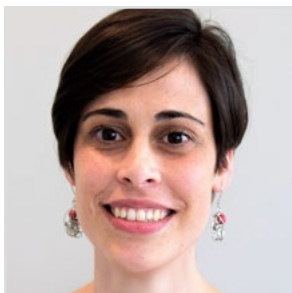
Using Population Sequence Differences to Identify a DNA Change that Reduces the Risk of ApoE for Developing Alzheimer's Disease

*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](http://www.brightfocus.org/grant/A2018425S)

## Battling Amyloid Beta

There are many versions of amyloid protein in the human body, and most serve a useful role. Amyloid beta (A $\beta$ ) is a type of amyloid that is prone to molecular changes that create fragments that accumulate in the brain. A healthy brain is able to break down amyloid beta and eliminate it, but in Alzheimer's disease (AD), amyloid beta forms hard, insoluble plaques that are toxic to neurons and are sometimes (not always) associated with AD-related memory loss and other changes. Once only seen after autopsy, new technologies have made it possible to measure amyloid beta plaques and learn which parts of the brain are most affected. Anti-amyloid drugs are being tested in clinical trials, with the hope of preventing formation of amyloid beta plaques in the future.



**Ana Batista, PhD** (7/1/19 – 6/30/21)

*University of Massachusetts, Worcester*

*FELLOWSHIP*

The Effect of the TTR Protein on Alzheimer's Disease

*Our goal is to answer conclusively whether transthyretin (over) expression in a post-developmental setting is an effective approach to either prevent or change the course of disease progression in AD and assess the potential of AAV gene therapy approach for translation into human clinical trials.*

[www.brightfocus.org/grant/A2019468F](http://www.brightfocus.org/grant/A2019468F)





**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 amyloid beta metabolism and prevents neuronal inflammation in the Alzheimer's brain.*

**[www.brightfocus.org/grant/A2018100S](http://www.brightfocus.org/grant/A2018100S)**



**Masato Maesako, PhD** (7/1/19 – 6/30/21)

**FELLOWSHIP**

**Massachusetts General Hospital, Boston**

A New Method to Visualize Amyloid Beta Generation

*This project will identify which cells, and where within cells, amyloid beta is produced using a new kind of biosensor that will allow for visualize of the protein in living cells.*

**[www.brightfocus.org/grant/A2019056F](http://www.brightfocus.org/grant/A2019056F)**



**Henrietta Nielsen, PhD** (7/1/19 - 6/30/22)

**Stockholm University, Sweden**

Assessment of Associations Between a Liver-Generated Profile in the Blood, Behavior and Alzheimer's Disease Related Changes Inside the Brain

*Alzheimer's is a disease of the brain and for which the risk is determined by a heritable factor, APOE4. We will investigate potential effects of a specific APOE4-linked liver-generated blood profile on disease-related changes inside the brain. A successful discovery of a factor that can be targeted in the periphery rather than the in brain, for the cure or prevention of Alzheimer's disease, would facilitate the development of medication to prevent the disease.*

**[www.brightfocus.org/grant/A2019446S](http://www.brightfocus.org/grant/A2019446S)**



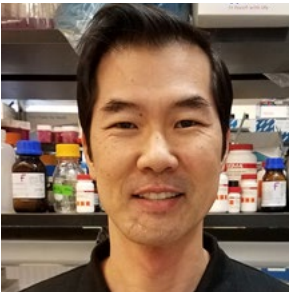
**Bryndon Oleson, PhD** (7/1/19 – 6/30/21)  
**University of Michigan, Ann Arbor**

**FELLOWSHIP**

Understanding the Function of the Biomolecule Polyphosphate During Aging and Alzheimer's Disease

*The highly-conserved polymer polyphosphate was recently found to protect cells and organisms from the toxic effects of amyloidogenic proteins such as amyloid- $\beta$ . The goal of this project is to characterize how polyphosphate changes with age, modifies amyloid- $\beta$  toxicity, and influences susceptibility to Alzheimer's disease.*

[www.brightfocus.org/grant/A2019250F](http://www.brightfocus.org/grant/A2019250F)



**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](http://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 Amyloid Beta Burden in Alzheimer's Disease

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

[www.brightfocus.org/grant/A2017443S](http://www.brightfocus.org/grant/A2017443S)



**Goonho Park, PhD** (7/1/18 - 6/30/20)  
**University of California, San Diego**

**FELLOWSHIP**

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](http://www.brightfocus.org/grant/A2018212F)



**Jiri Safar, MD** (7/1/16 - 12/30/19)  
**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](http://www.brightfocus.org/grant/A2016085S)



**Matthias Truttmann, PhD** (7/1/19 - 6/30/20)  
**University of Michigan, Ann Arbor**

The Alzheimer's Mystery: Why Proteins Clump Up and Kill Our Memories

*As we get older, proteins become less and less stable and will occasionally engage in the formation of protein clumps within cells. Some of these protein clumps are very toxic to neurons and will damage our brains, thus triggering neurodegenerative diseases such as AD. This project aims to better understand the processes that prevent the formation of protein clumps and seek to learn why these processes become less efficient in the older population.*

[www.brightfocus.org/grant/A2019157S](http://www.brightfocus.org/grant/A2019157S)



**Eitan Wong, PhD** (7/1/19 - 6/30/21)  
**Memorial Sloan-Kettering Cancer Center, New York, NY**

The circadian regulation of  $\gamma$ -secretase activity in Alzheimer's disease

*Alzheimer's disease (AD) is a progressive and fatal neurodegenerative disease which becomes increasingly prevalent worldwide with no effective treatments available causing a healthcare problem of epidemic proportion. Although the cause of AD is poorly understood, the disease progression is associated with beta-amyloid peptide senile (A) plaques and sleeping disorder, suggesting malfunction in internal biological clock and alteration of circadian rhythm. Interestingly, our initial data discovered that gamma-secretase activity, the enzyme responsible for amyloid beta plaques generation, also exhibits a daily circadian oscillation. In this proposal we aim to reveal the molecular interaction between circadian function and gamma-secretase activity and the connection to AD.*

[www.brightfocus.org/grant/A2019356F](http://www.brightfocus.org/grant/A2019356F)



**Justyna Dobrowolska Zakaria, PhD** (7/1/19 - 6/30/22)  
**Northwestern University, Chicago, IL**  
Co-Principal Investigator **Robert J Vassar, PhD**  
**Bateman Washington University**

**A New Method to Separate Sub-groups of Alzheimer's Disease by Measuring sAPP $\beta$  in Human Cerebrospinal Fluid**

*The goal of this project is to measure how quickly an AD patient's brain makes a protein known as sAPP $\beta$ , and compare this to a healthy patient's brain, to determine if in AD there is more sAPP $\beta$  being made than normal. Also, there is increasing evidence that not every patient's AD has the same cause. So additionally, we want to use sAPP $\beta$ , and other proteins such as sAPP $\beta$  and amyloid beta, to determine if there are subgroups within AD patients that might respond in different ways to drugs that target AD.*

**[www.brightfocus.org/grant/A2019520S](http://www.brightfocus.org/grant/A2019520S)**

## **Tangling with Tau**

Another protein associated with Alzheimer's disease (AD), tau is abundant inside neurons, where its fibrous shape lends stability to tubes that transport nutrition and waste in and out. However, in AD, tau goes through molecular changes that cause it to misshape and collect in messy tangles. Unlike amyloid plaques, which can form years and even decades before AD symptoms occur, tau tangles typically are a sign that AD is rapidly getting worse. Current theories hold that amyloid beta and tau interact in ways to make that happen, and scientists are investigating how tau may be involved in spreading AD throughout the brain.



**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](http://www.brightfocus.org/grant/A2018816S)**



**Nick Cochran, PhD** (7/1/19 – 6/30/21) **FELLOWSHIP**  
**HudsonAlpha Institute for Biotechnology, Huntsville, AL**

How an Important Gene for Alzheimer's called MAPT is Turned On

*This project will find out how the MAPT gene, which encodes tau, is turned on in neurons. Tau causes problems in Alzheimer's disease, and scientists think that reducing tau might be helpful as a treatment. Figuring out how MAPT is turned on in neurons might help determine how to turn it off and reduce tau in AD.*

[www.brightfocus.org/grant/A2019129F](http://www.brightfocus.org/grant/A2019129F)



**Luca Colnaghi, PhD** (7/1/19 – 6/30/21) **FELLOWSHIP**  
**Instituto di Ricerche Farmacologiche Mario Negri, Milan, Italy**

Molecular Mechanisms in Alzheimer's Disease

*Protein aggregation and deposition in the brain is a striking feature of several neurodegenerative disorders, including Alzheimer's disease. The aim of this proposal is to understand the role of understudied post-translational modifications, such as Ubiquitin and Ubiquitin-like modifications, towards tau aggregation and deposition using mouse models of AD.*

[www.brightfocus.org/grant/A2019296F](http://www.brightfocus.org/grant/A2019296F)



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

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](http://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](http://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](http://www.brightfocus.org/grant/A2017281S)

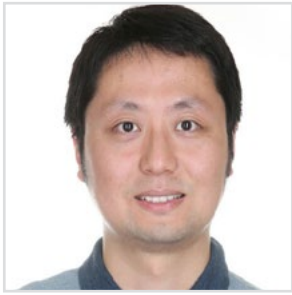


**Michelle Farrell, PhD** (7/1/19 – 6/30/21) **FELLOWSHIP**  
**Massachusetts General Hospital, Boston**

Improving Detection of the Earliest Signs of Alzheimer's Disease  
to Help Prevent Memory Loss

*The project aims to use brain imaging (PET scans) in healthy older adults to visualize the earliest signs of amyloid plaques, and determine how the buildup of these amyloid plaques contributes to the appearance of tau tangles inside brain cells and subtle changes in memory and thinking. This research will provide urgently needed information about the early stages of development of AD, and help the next generation of prevention trials target individuals who are at an optimal point in the development of AD for successful intervention.*

[www.brightfocus.org/grant/A2019029F](http://www.brightfocus.org/grant/A2019029F)



**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](http://www.brightfocus.org/grant/A2018802S)



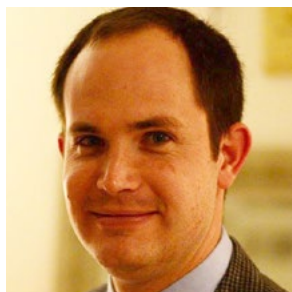
**Lukasz Joachimiak, PhD** (7/1/19 - 6/30/22)

*University of Texas Southwestern Medical Center, Dallas, TX*

Detecting the Shape Changing Protein Tau in Alzheimer's Disease

*The tau protein normally adopts a "good" shape and with age converts into a "bad" shape. This project aims to understand how tau changes into the "bad" shape to help understand how to detect this in patients and develop therapies to prevent it.*

[www.brightfocus.org/grant/A2019060S](http://www.brightfocus.org/grant/A2019060S)



**Jeremy Strain, PhD** (7/1/18 - 6/30/20)

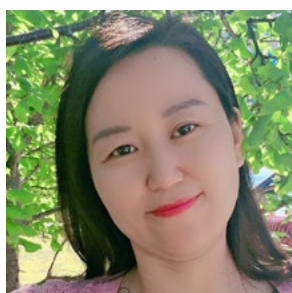
*Washington University, St. Louis, MO*

**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](http://www.brightfocus.org/grant/A2018817F)



**Wenyan Sun, PhD** (7/1/19 - 6/30/21)

*University of Texas Health Science Center at San Antonio*

**FELLOWSHIP**

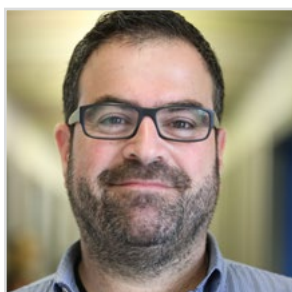
Determine Whether PIWIL and piRNAs are Dysregulated in Tau Transgenic Mice and Human Neurodegenerative Tauopathies

*Recently, research with fruit flies identified a new mechanism to address tau toxicity and jumping genes through PIWI protein and PIWI-interacting RNAs (piRNAs). To investigate if this mechanism is conserved in vertebrate tauopathy, this project will be determined if this same mechanism is intact in mouse models of AD so that they can be used to determine if piRNAs identified in AD are directly bound to PIWI-like proteins and determine if they are relevant to brain cell death in AD.*

[www.brightfocus.org/grant/A2019223F](http://www.brightfocus.org/grant/A2019223F)

## Biology of Fats and Proteins (ApoE and Lipids)

Initially recognized for its role in cardiovascular disease, the *APOE* gene also plays a role in Alzheimer's disease (AD). Its primary function is to regulate a class of proteins involved in the metabolism of fats (lipids) in the body. However, *APOE* has several common variants (or "alleles") whose effect vary. The e4 allele, in particular, is the most prevalent genetic factor associated with late-onset Alzheimer's disease, and may cause an increased risk and/or earlier onset. Its impact varies depending on whether the mutation appears on one or both chromosomes, and on a person's race and ethnicity (i.e., risk is not increased uniformly across all ethnic groups). Scientists are still trying to find out the reasons why. Some clues may lie with ApoE4 interactions with the immune system, where it influences inflammation and a type of cellular damage known as oxidation. Also, whereas ApoE helps break down amyloid beta protein located in and around neurons, the ApoE4 version is less effective at doing so.



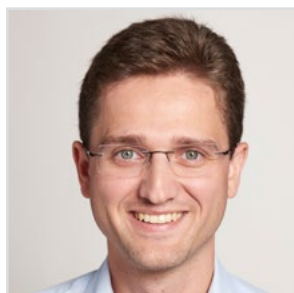
**Edoardo Marcora, PhD** (7/1/17 - 6/30/20)

*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](http://www.brightfocus.org/grant/A2017458S)



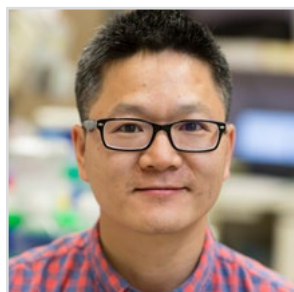
**Joseph Castellano, PhD** (7/1/18 - 6/30/21)

*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](http://www.brightfocus.org/grant/A2018213S)



**Weiwei Fan, PhD** (7/1/18 - 6/30/20)

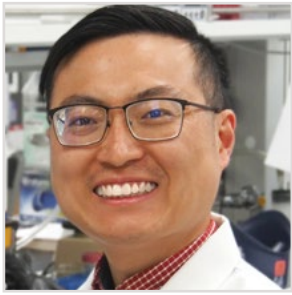
*Salk Institute for Biological Studies, La Jolla, CA*

Developing a New Drug for Alzheimer's Disease by Improving Lipid Metabolism in the Brain

*PPARδ is a protein that is present in the brain. It is suggested to have important functions in brain. This proposal will help us understand its exact functions in brain. I will also test whether we can target PPARδ to treat AD.*

[www.brightfocus.org/grant/A2018325S](http://www.brightfocus.org/grant/A2018325S)





**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](http://www.brightfocus.org/grant/A2018128F)



**Na Zhao, MD, PhD** (7/1/18 - 6/30/20)

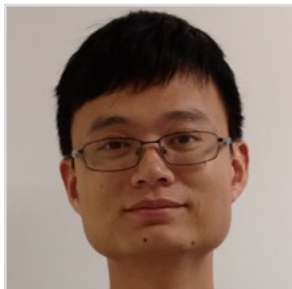
*FELLOWSHIP*

*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](http://www.brightfocus.org/grant/A2018777F)



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

*FELLOWSHIP*

*Xiamen University, China*

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 apoptosin, can alter the brain to disrupt communication between neurons to cause problems with memory.*

[www.brightfocus.org/grant/A2018214F](http://www.brightfocus.org/grant/A2018214F)

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



## Cells and Circuits

The human brain has an estimated 100 billion neurons. Extending from each of them is a long fiber, known as an “axon,” which can run several feet. Each axon forms a connection, known as a “synapse” with another neuron, creating a circuit over which brain signals travel. In Alzheimer’s disease (AD), individual neurons die and do not regenerate; yet some brains are resilient and will remodel themselves to meet new communications demands. If a circuit is too damaged to connect by the most direct route, signaling will sometimes take detours, known as indirect neural pathways. It’s not until the communications network completely breaks down that the worst AD symptoms—things like forgetting loved ones, or becoming lost in familiar places—begin to occur. Scientists are studying the brain’s many cells and circuits, looking for ways to preserve communications for as long as possible after the onset of AD.



**Marc Aurel Busche, MD, PhD** (7/1/19 - 6/30/22)  
*University College London, England, UK*

**Mechanisms of Neuronal Dysfunction in Early Alzheimer's Disease**

*This project will explore in particular the effects that tau and amyloid proteins seen in the brains of patients with AD have on the activity of interacting nerve cells in the hippocampus, a brain region which is known to be important for learning and memory.*

[www.brightfocus.org/grant/A2019112S](http://www.brightfocus.org/grant/A2019112S)

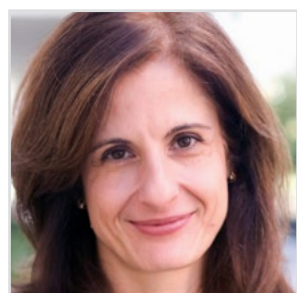


**Maria Calvo-Rodriguez, PhD** (7/1/19 – 6/30/21) *FELLOWSHIP*  
*Massachusetts General Hospital, Boston*

**Dysfunction of Mitochondria in Astrocytes in Alzheimer's Disease**

*This project will clarify if mitochondria mobility, distribution and calcium dynamics are altered in astrocytes in the pathology of AD, and eventually determine the contribution of mitochondria and astrocytes to this disease. The final goal is to reverse mitochondrial dysfunction with appropriate drugs, suggesting novel molecular targets for therapeutic development that can be used in people.*

[www.brightfocus.org/grant/A2019488F](http://www.brightfocus.org/grant/A2019488F)

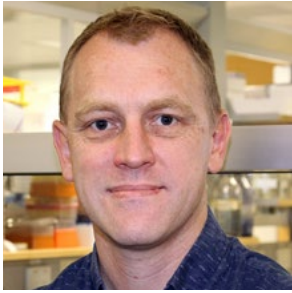


**Inma Cobos, MD, PhD** (7/1/17 - 6/30/20)  
*Stanford University, CA*

**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](http://www.brightfocus.org/grant/A2017346S)



**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](http://www.brightfocus.org/grant/A2018627S)



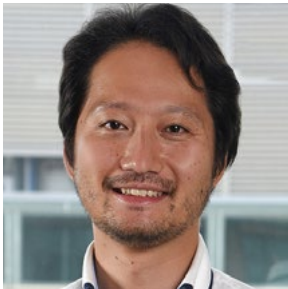
**Sara Gallant, PhD** (7/1/18 - 6/30/20)  
*University of Southern California, Los Angeles*

**FELLOWSHIP**

**Arousal-Induced Memory Selectivity in Aging & 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](http://www.brightfocus.org/grant/A2018449F)



**Kei Igarashi, PhD** (7/1/19 - 6/30/22)  
*University of California, Irvine*

**Rescuing Impaired Memory in Alzheimer's Disease Using Reactivation of Brain Network Activity**

*Drs. O'Keefe, Moser and Moser, three Nobel prize researchers, previously found that brain cells called "place cells" and "grid cells" are important to keep our memory. Are these cells broken in AD patients? If so, does fixing of these cells heal memory lost in AD patients? This project will answer to these questions using animal models of AD.*

[www.brightfocus.org/grant/A2019380S](http://www.brightfocus.org/grant/A2019380S)



**Thomas Kukar, PhD** (7/1/19 - 6/30/22)  
*Emory University, Atlanta, GA*

**Understanding Lysosome Dysfunction in Alzheimer's Disease**

*The health and survival of neurons in the brain is dependent on a recycling pathway carried out by lysosomes, cellular organelles that help degrade and recycle proteins. Defects in the function of lysosomes are increasingly thought to be involved in the development of Alzheimer's disease (AD). We are trying to understand why decreases in a protein called progranulin impair lysosome function and increase the risk of developing Alzheimer's disease.*

[www.brightfocus.org/grant/A2019380S](http://www.brightfocus.org/grant/A2019380S)



**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](http://www.brightfocus.org/grant/A2017180S)



**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](http://www.brightfocus.org/grant/A2017286S)



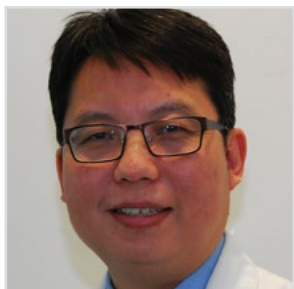
**Arjun Masurkar, MD, PhD** (7/1/19 - 6/30/22)  
*New York University, NY*

Towards New Stimulation Methods to Correct Memory in Alzheimer's Disease

*Memory fails early in Alzheimer's disease because the entorhinal cortex, a brain area first affected by the disease, cannot properly communicate with the hippocampus, the second brain area affected by the disease. However, there is another brain region, the thalamus, that communicates with the hippocampus but is not affected at early stages. This project deciphers the structure and function of the "wiring diagram" between thalamus and hippocampus, and then examines how this functional connectivity changes in AD.*

[www.brightfocus.org/grant/A2019602S](http://www.brightfocus.org/grant/A2019602S)

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



**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](http://www.brightfocus.org/grant/A2017457S)

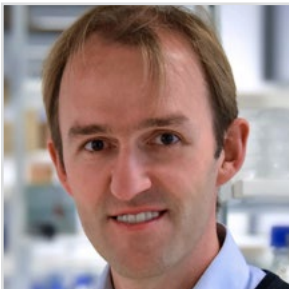


**Jerome Mertens, PhD** (7/1/19 - 6/30/22)  
*University of Innsbruck, Innsbruck, Tyrol, Austria*

**Reprogramming of Skin Cells from Alzheimer Patients into Brain Neurons to Understand and Fight Cellular Memory Loss on the Molecular Level**

*Everybody ages, and unfortunately, this banal fact represents a huge health threat for us, because old age is the major risk factor for many human diseases with AD leading the way. Our laboratory has recently found a way to reprogram cultured skin cells from Alzheimer patients directly into brain neurons, which are unique for each patient and also biologically "remember" how old the patient was. In this project we aim to better understand this connection and try to find ways to give Alzheimer neurons their own memory back.*

[www.brightfocus.org/grant/A2019562S](http://www.brightfocus.org/grant/A2019562S)

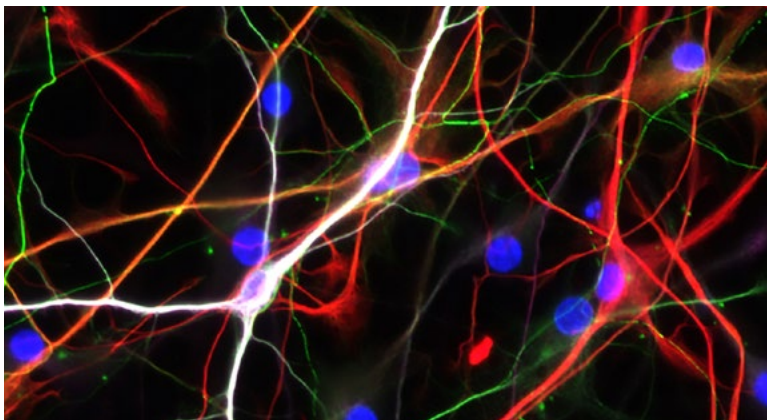


**Dominik Paquet, PhD** (7/1/19 - 6/30/22)  
*Ludwig-Maximilians University, Munich, Germany*

**A Human Brain-In-A-Dish Model to Investigate Central Factors Required for the Formation of Alzheimer's Disease Pathology**

*We currently do not understand well why the brains of Alzheimer patients contain aggregates of proteins and how these aggregates relate to the death of millions of nerve cells over time. To better understand the formation of plaques and tangles, we would like to investigate the building blocks that are required for the formation of AD. For this purpose, we will turn human stem cells into nerve cells and other cell types found in the human brain, grow them together in a dish to assemble artificial human brain tissue, and introduce alterations in genes and cellular physiology that are typical for patients with inherited forms of AD. We will investigate if these models display Alzheimer pathology in a dish, and then modify the composition of cell types or the function of cells and their genes to learn which factors cause protein aggregation or nerve cell death in AD.*

[www.brightfocus.org/grant/A2019604S](http://www.brightfocus.org/grant/A2019604S)



**Courtesy of Dr. Dominik Paquet's Lab**  
Photo of what a long-term co-culture of iPSC-derived human cortical neurons (green and white) and astrocytes (red) grown together in a lab look like under a microscope.



**Bede Portz, PhD** (7/1/19 – 6/30/21)  
**University of Pennsylvania, Philadelphia**

**FELLOWSHIP**

**New Protein Modifiers and Therapeutic Targets to Combat Toxic RNA Foci in Frontotemporal Dementia**

*Frontotemporal dementia is caused by expanded repeats in the C9orf72 gene, which encode toxic repeat RNAs that aggregate, forming RNA foci. This project will elucidate the machinery overwhelmed by C9orf72 repeat expansion by testing the hypothesis that DDX3X is an RNA disaggregase capable of dissolving these foci, and by screening for new protein modifiers of C9orf72 RNA foci in live cells.*

**[www.brightfocus.org/grant/A2019612F](http://www.brightfocus.org/grant/A2019612F)**



**Min-Kyoo Shin** (7/1/19 – 6/30/21)  
**Case Western Reserve University, Cleveland, OH**

**FELLOWSHIP**

**Determination of Whether a Novel Biological System in the Brain Regulates Nerve Cell Death and Behavioral Abnormalities in Alzheimer's Disease**

*Alzheimer's disease (AD) is one of the most highly prevalent and devastating conditions in society, and there are currently no treatments that prevent or slow disease progression. We have discovered a new biological system governing neurodegeneration in traumatic brain injury: enzymatic activity of 15-prostaglandin dehydrogenase in the brain that controls levels of prostaglandin E2, an endogenous agent that protects neurons. We also have preliminary evidence that levels of 15-PGDH are pathologically increased in animal models of AD, as well as human AD brain.*

**[www.brightfocus.org/grant/A2019551F](http://www.brightfocus.org/grant/A2019551F)**



**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](http://www.brightfocus.org/grant/A2018656S)**

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

## Immunity and Inflammation

One theory about Alzheimer's disease (AD) is that it may be triggered, in part, by a breakdown in the brain's immune system. Normally our brain has ways of clearing damaged cells and unwanted particles in its midst and disposing them into the bloodstream—which can be thought of as “taking out the garbage.” However, a chronic rise in unwanted debris, including toxic amyloid beta and tau proteins, can short-circuit that process and lead to chronic inflammation and cell damage. Grantees are looking at what causes the immune response to become unbalanced and whether there are ways to help the brain's cells and immune system do a better job of fighting Alzheimer's.



**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](http://www.brightfocus.org/grant/A2018253F)



**Darrick T. Balu, PhD** (7/1/19 - 6/30/22)  
*McLean Hospital, Harvard Medical School, Belmont, MA*

Understanding How Inflammation Kills Brain Cells During Alzheimer's Disease Progression

*As AD progresses, inflammation changes the characteristics of particular cells in the brain called astrocytes. Inflammatory astrocytes release chemical compounds that are toxic to neurons. This project aims to understand how one of the molecules released by reactive astrocytes kills neurons, in hopes of finding new drugs to treat patients*

[www.brightfocus.org/grant/A2019034S](http://www.brightfocus.org/grant/A2019034S)



**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](http://www.brightfocus.org/grant/A2018377S)



**Hemraj Dodiya, PhD** (7/1/19 – 6/30/21)  
*University of Chicago, IL*

*FELLOWSHIP*

Microbiome Influences Microglia Phenotypes and Beta-Amyloid Amyloidosis in a Sex-Specific Manner

*These experiments will assess the role of gender-specific gut microbes in regulating inflammation and beta-amyloid deposition using mouse models of AD. This will advance understanding of link between different gut microbes and AD susceptibility in men and women.*

[www.brightfocus.org/grant/A2019032F](http://www.brightfocus.org/grant/A2019032F)



**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](http://www.brightfocus.org/grant/A2018718S)

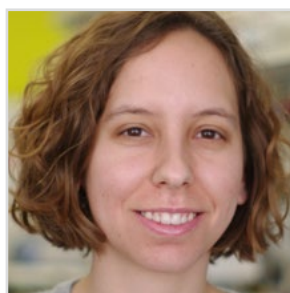


**Benjamin Hogan, PhD** (7/1/18 - 6/30/21)  
*The University of Queensland, Brisbane, Australia*  
Co-Principal Investigator **Neil Bower, PhD**

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](http://www.brightfocus.org/grant/A2018807S)



**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](http://www.brightfocus.org/grant/A2018349S)





**Zixu Mao, MD, PhD** (7/1/16 - 6/30/20)  
**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 pathogenesis as the disease progresses over time.*

[www.brightfocus.org/grant/A2016501S](http://www.brightfocus.org/grant/A2016501S)



**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 amyloid beta 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](http://www.brightfocus.org/grant/A2018169S)



**Anna Pimenova, PhD** (7/1/19 – 6/30/21) **FELLOWSHIP**  
**Icahn School of Medicine at Mount Sinai, New York, NY**

**Uncovering the Features of PU.1-Protective Microglia in Alzheimer's Disease**

*Analyses of genetic information in patients with AD have identified many factors for the predisposition to develop dementia. These factors are very common in human populations, and together with rare familial cases of Alzheimer's disease, suggest that brain inflammation contributes to disease progression. This project will focus on the protective features of immune cell types in the brain and define molecules that regulate brain response to disease.*

[www.brightfocus.org/grant/A2019299F](http://www.brightfocus.org/grant/A2019299F)



**Yuxiang Sun, PhD** (7/1/19 – 6/30/21)  
**Texas A&M University, College Station**

**A New Intervention to Control Inflammation in Alzheimer's Disease**

*Low-grade chronic inflammation is a hallmark of aging, and inflammation in the brain causes and worsens Alzheimer's Disease (AD). We have evidence that suppression of a gene called GHS-R in immune cells produces an anti-inflammatory effect in the brain and improves spatial memory.*

[www.brightfocus.org/grant/A2019630S](http://www.brightfocus.org/grant/A2019630S)



**Nicholas Varvel, PhD** (7/1/19 - 6/30/22)  
**Emory University, Atlanta, GA**

**Brain-Invading Monocytes at the Intersection of Alzheimer's Disease and Seizures**

*In addition to memory loss, a certain subset of those with AD also suffer from seizures. We have recently identified an immune cell type, called a monocyte, that enters the brain after seizures. The studies are designed to determine if seizure-induced monocyte entry into the brain enhances the progression of AD.*

**[www.brightfocus.org/grant/A2019077S](http://www.brightfocus.org/grant/A2019077S)**

## **Biomarkers**

Biomarkers are early markers of biological changes associated with AD. Alzheimer's disease (AD) may begin causing gradual changes in the brain some 10-20 years before the onset of symptoms. The best hope of stopping AD is during this phase, and the need for earlier treatment is what makes the search for biomarkers so important. Some of the earliest examples of biomarkers, still in development, include tests to measure elevated amyloid beta (A $\beta$ ) levels in blood and/or cerebrospinal fluid; use of advanced imaging to detect tiny changes in brain structure; and techniques identifying beta amyloid protein deposits in the retina of the eye that mirror those in the brain. Biomarkers like these can help identify who is most likely to develop AD in the future, and what type, and also provide reliable measures of disease progress. This will help guide treatment decisions in the future, when drugs become available, determining such things as who needs treatment, when to start, and which drugs and treatment strategies are most likely to be successful.

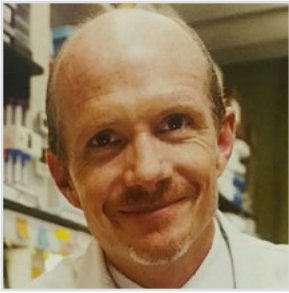


**Iman Aganj, PhD** (7/1/16 - 6/30/20)  
**Massachusetts General Hospital, Boston**

**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](http://www.brightfocus.org/grant/A2016172S)**

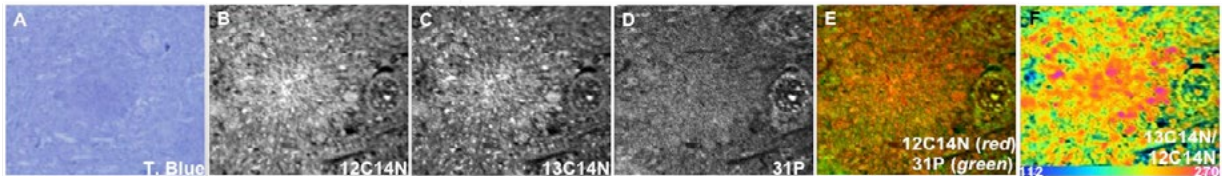


**Randall Bateman, MD** (7/1/17 - 9/30/20)  
**Washington University School of Medicine, St. Louis, MO**  
 Co-Principal Investigators **Norelle C. Wildburger, PhD**  
 & **Robert Schmidt, MD, PhD**

**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 amyloid beta 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](http://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).



**David Berron, PhD** (7/1/19 – 6/30/21)  
**Lund University, Sweden**

**FELLOWSHIP**

**Learning About the Early Consequences of Alzheimer's Disease on our Brain and Cognitive Functions**

*With the powerful and novel combination of state-of-the-art positron emission tomography (PET) imaging for amyloid/tau and structural as well as functional ultrahigh-field magnetic resonance imaging (MRI) at 7 Tesla, this project will unravel the early effects of AD pathology on brain functional connectivity and memory task-related functional activity, grey matter loss and specific memory functions.*

[www.brightfocus.org/grant/A2019401F](http://www.brightfocus.org/grant/A2019401F)



**Becky Carlyle, PhD** (7/1/19 - 6/30/22)  
**Massachusetts General Hospital, Boston**

**Investigating Neuropeptides as Biomarkers and Novel Therapeutics for Alzheimer's Disease**

*AD is currently defined by the abundance of two insoluble proteins, A $\beta$  and tau, but the amount of these proteins does not accurately predict cognitive problems in people with AD. Recent studies have found that neuropeptides are widely dysregulated in AD, and might play roles in the AD disease process. In this proposal, we investigate whether neuropeptides may be used to more accurately assess AD patients, and whether supplementation with these peptides might eventually prove a new potential therapy for AD.*

[www.brightfocus.org/grant/A2019182S](http://www.brightfocus.org/grant/A2019182S)



**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](http://www.brightfocus.org/grant/A2018093S)



**Ryan Darby, MD** (7/1/17 - 8/30/19)

*FELLOWSHIP*

*Vanderbilt University, Nashville, TN*

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](http://www.brightfocus.org/grant/A2017226F)



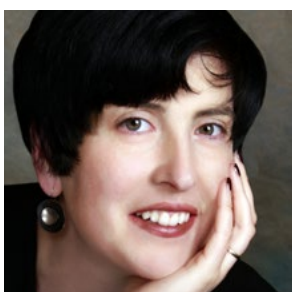
**Jill M. Goldstein, PhD** (3/30/18 - 3/29/20)

*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/A2018607](http://www.brightfocus.org/grant/A2018607)



**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](http://www.brightfocus.org/grant/A2017560S)



**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](http://www.brightfocus.org/grant/A2018202S)**



**Xiong Jiang, PhD** (7/1/16 - 6/30/20)  
**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](http://www.brightfocus.org/grant/A2016251S)**

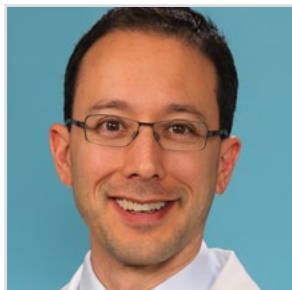


**Hosung Kim, PhD** (7/1/19 - 6/30/22)  
**University of Southern California, Los Angeles**  
Co-Principal Investigator **Arthur Toga, PhD USC**

**Machine-Learning Applied to Neuroimaging Data Can Predict Brain Biological Age and Acceleration of Aging in Early Alzheimer's Disease**

*This proposed research seeks to predict physiological brain age for individuals in healthy condition by leveraging deep learning-based modeling with brain image datasets. This project expands the model to predict how abnormality expands incrementally to different brain areas as mild cognitive impairment and AD develop to estimate the "survival" probability explaining the remaining days in healthy status prior to the onset of MCI or AD, ultimately allowing disease-specific risk scoring as a clinical tool to be used in routine patient care.*

**[www.brightfocus.org/grant/A2019052S](http://www.brightfocus.org/grant/A2019052S)**

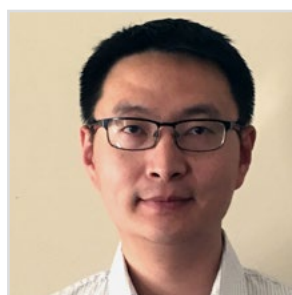


**Terrance Kummer, MD, PhD** (7/1/17 - 6/30/20)  
*Washington University School of Medicine, St. Louis, MO*

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](http://www.brightfocus.org/grant/A2017084S)

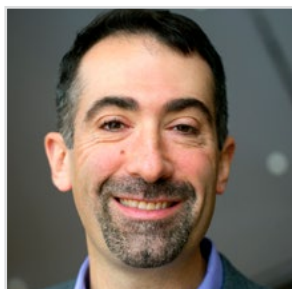


**Yi Su, PhD** (7/1/17 - 6/30/20)  
*Banner Health, Phoenix, AZ*

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](http://www.brightfocus.org/grant/A2017272S)

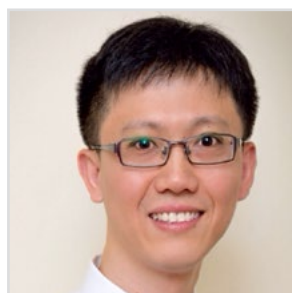


**Peter Tessier, PhD** (7/1/17 - 8/31/20)  
*University of Michigan, Ann Arbor*  
Co-Principal Investigators **Brian Ross, PhD & Henry Paulson, MD, PhD**

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](http://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

*Our new PET-MRI [positron emission tomography–magnetic resonance imaging] method takes pictures of the brain in elderly people. Whereas these PET-MRI images have commonly been used to identify tumors or strokes in patients, we have found a new way to use PET-MRI to measure the brain's injury and immune response.*

[www.brightfocus.org/grant/A2017330S](http://www.brightfocus.org/grant/A2017330S)

## Finding New Drugs and Treatments for Alzheimer's Disease

Years of innovative and dedicated research have paid off with the discovery of numerous factors contributing to Alzheimer's disease (AD) pathology. These discoveries have produced a bounty of "druggable targets", and 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 its progress. Unfortunately, only a handful of potential "disease-modifying treatments" ever make it to the point of being tested in clinical trials, where a few are being tested today. Support is needed for the type of high-risk, high-reward early research where discoveries in basic science may lead to the development of molecules and compounds aimed at reducing toxic protein buildup in AD, improving clearance of toxic particles, and delivering treatments to brain areas. The contributions gained from these exploratory projects at early stages are essential to get the next generation of drugs and therapies to patients.



**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](http://www.brightfocus.org/grant/A2017243S)



**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](http://www.brightfocus.org/grant/A2018509S)



**Mark Henkemeyer, PhD** (7/1/16 - 12/31/19)  
*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](http://www.brightfocus.org/grant/A2016345S)



**Tsuneya Ikezu, MD, PhD** (7/1/16 - 6/30/20)  
*Boston University, MA*

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

*Focused 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](http://www.brightfocus.org/grant/A2016551S)



**Patrick Kehoe, PhD** (7/1/16 - 6/30/20)  
*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](http://www.brightfocus.org/grant/A2016582S)







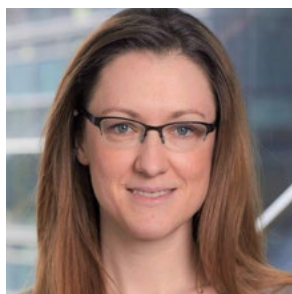
**WonHee Kim, PhD** (7/1/19 – 6/30/21)  
**Tufts University, Boston, MA**

**FELLOWSHIP**

### Understanding Alzheimer's Disease to Avoid Side Effects of Drugs

*Pharmaceutical companies have developed a drug, called BACE inhibitor, that has the potential to prevent and cure AD. However, it is still uncertain whether this drug is safe for AD patients. Prior research suggests that this medicine could cause worsen side effects in people born with Down syndrome or carrying a specific genetic mutation causing AD. The goal of this project is to better understand AD caused by genetic risk factors, and ultimately find a safe drug treatment for AD patients.*

[www.brightfocus.org/grant/A2019021F](http://www.brightfocus.org/grant/A2019021F)

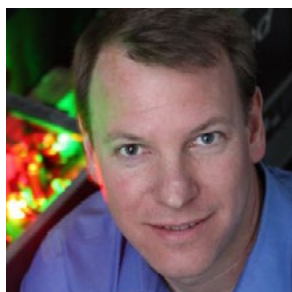


**Anna Orr, PhD** (7/1/19 - 6/30/22)  
**Weill Cornell Medicine, New York, NY**  
Co-Principal Investigators **Adam Orr, PhD**  
**Weill Cornell Medicine, New York, NY**

### Alleviating Alzheimer's Disease with Novel Therapeutic Agents that Can Precisely Block the Production of Reactive Oxygen

*Aging and neurodegenerative disease are associated with the accumulation of free radicals (also called oxidative stress) in the brain and other organs. Oxidative stress can damage cells and organs, promote disease, and impair brain function. We recently discovered small molecules that can block specific causes of oxidative stress without affecting normal cell functions. This research project will test whether these small molecules have therapeutic benefits in experimental models of dementia.*

[www.brightfocus.org/grant/A2019363S](http://www.brightfocus.org/grant/A2019363S)



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

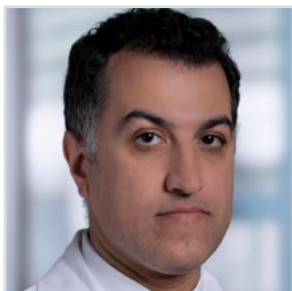
### Improving Brain Blood Flow in Alzheimer's Disease to Improve Cognitive Function

*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](http://www.brightfocus.org/grant/A2017488S)

## Translational Research and Clinical Interventions

“Translational” research refers to the effort to take basic science knowledge from the laboratory or research setting into the “real world” in the form of potential treatments or cures, in other words, to literally “translate” science into useful ways of diagnosing, treating, and managing Alzheimer’s disease (AD). It can take many different forms, from using smartphone-based testing to monitor cognitive status in AD, to finding ways for individuals with AD to get better sleep and exercise, since scientists have associated these lifestyle activities with brain health and possible protective benefits. Another very important undertaking is that of testing new drugs and interventions in humans once they are deemed safe, and this is done through clinical trials and another studies which rely on volunteers who are willing to participate. These activities will help speed drugs, treatments, and critical knowledge from “bench to bedside” and put them in the hands of people living with AD today or facing the risk of it in the future.



**Alireza Faridar, MD** (7/1/19 – 6/30/21)  
*Houston Methodist Research Institute, TX*

*FELLOWSHIP*

Does Immune System Play a Role as a Potential Therapeutic Target in Alzheimer’s Disease?

*Regulatory T cells (Tregs) are the major immunomodulatory cell in the blood that might lose functionality in AD. For the first time in AD research, dysfunctional Tregs will be expanded in dishes to restore their suppressive function and the impact of these expanded/normalized Tregs on Alzheimer pathology will be evaluated.*

[www.brightfocus.org/grant/A2019083F](http://www.brightfocus.org/grant/A2019083F)





**Jason Brandt, PhD** (7/1/16 - 6/30/20)  
**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](http://www.brightfocus.org/grant/A2016073S)



**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](http://www.brightfocus.org/grant/A2018405S)



**Giacomo Koch, MD, PhD** (7/1/19 - 6/30/22)  
**IRCCS Santa Lucia Foundation, Rome, Italy**  
Co-Principal Investigator **Martorana Alessandro, MD, PhD**  
**Rome University Tor Vergata**

**Magnetic Stimulation to Treat Alzheimer's Disease**

*The primary aim of this project is to investigate a non-invasive brain stimulation, repetitive transcranial magnetic stimulation (rTMS), on memory skills in patients with mild AD. rTMS is considered a safe, well tolerated and relatively cheap treatment. The appealing idea of our intervention is to improve memory by directly modulating the activity of precuneus, key area linked to memory impairment. This project will provide a valid treatment to slow the worsening of symptoms and improve quality of life for those with AD and their caregivers.*

[www.brightfocus.org/grant/A2019523S](http://www.brightfocus.org/grant/A2019523S)





**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](http://www.brightfocus.org/grant/A2018667S)



**Brendan Lucey, MD** (7/1/16 - 6/30/20)  
*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](http://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](http://www.brightfocus.org/grant/A2017547S)





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

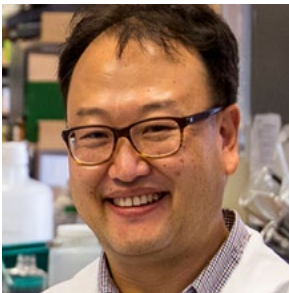
Can Good Sleep Prevent Alzheimer's Disease?

*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](http://www.brightfocus.org/grant/A2018402F)

## Research Tools and Resources

The world is alive with emerging technologies and resources that can be brought together in the quest to end Alzheimer's disease (AD). These range from high through-put science and artificial intelligence to citizen science. We are privileged to be a driving force behind new collaborations that will move research forward into exciting new territory. Ultimately these efforts will lead to better understanding of risks and treatment approaches for all types of AD.



**Doo Yeon Kim, PhD** (7/1/16 - 9/30/19)

*Massachusetts General Hospital and 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](http://www.brightfocus.org/grant/A2016362S)



**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.*

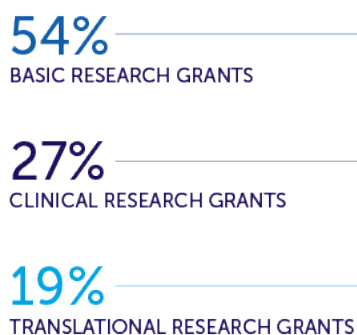
[www.brightfocus.org/grant/CA2018010](http://www.brightfocus.org/grant/CA2018010)

## Index:

Abadir, Peter .....	3	Grinberg, Lea .....	28
Aganj, Iman .....	26	Gunawardena, Shermali .....	31
Arancio, Ottavio .....	12	Hales, Chadwick .....	13
Audrain, Mickael .....	23	Hassenstab, Jason .....	29
Balu, Darrick .....	23	He, Congcong .....	9
Bateman, Randall .....	27	He, Zhuohao .....	14
Batista, Ana .....	8	Henkemeyer, Mark .....	32
Berron, David .....	27	Hernandez Guillamon, Mar .....	31
Bonnin, Alexandre .....	3	Hilal, Saima .....	4
Bos, Daniel .....	4	Hogan, Benjamin .....	24
Brandt, Jason .....	35	Igarashi, Kei .....	19
Busche, Marc Aurel .....	18	Ikezu, Tsuneya .....	32
Calvo-Rodriguez, Maria .....	18	Jensen, Majken .....	4
Cao, Wei .....	23	Jiang, Xiong .....	29
Carlyle, Becky .....	27	Joachimiak, Lukasz .....	15
Castellano, Joseph .....	16	Kaczorowski, Catherine .....	7
Cheung, Carol Yim Lui .....	28	Karch, Celeste .....	25
Cobos, Inma .....	18	Kehoe, Patrick .....	32
Cochran, Nick .....	13	Kim, Doo Yeon .....	37
Collins, Brett .....	19	Kim, Hosung .....	29
Colnaghi, Luca .....	13	Kim, WonHee .....	33
Croft, Cara .....	13	Koch, Giacomo .....	35
Cukier, Holly .....	6	Kukar, Thomas .....	19
Darby, Ryan .....	28	Kumar, Sanjeev .....	36
Dodiya, Hemraj .....	24	Kummer, Terrance .....	30
Duff, Karen .....	14	Lee, Tae Ho .....	20
Fan, Weiwei .....	16	Lippmann, Ethan .....	5
Faridar, Alireza .....	34	Lucey, Brendan .....	36
Farrell, Michelle .....	14	Ma, Tao .....	20
Gallant, Sara .....	19	Maesako, Masato .....	9
Geschwind, Daniel .....	7	Mao, Zixu .....	25
Glabe, Charles .....	24	Marcora, Edoardo .....	16
Goldstein, Jill M .....	28	Masurkar, Arjun .....	20
Granholt-Bentley, Ann-Charlotte ..	37	McIntosh, Randy .....	20
Grill, Joshua .....	35		

Mertens, Jerome.....	21	Schaffer, Chris .....	33
Miller, Timothy .....	25	Shin, Min-Kyoo.....	22
Newton, Robert Jr .....	36	Smith, Alex .....	5
Nielsen, Henrietta.....	9	Strain, Jeremy.....	15
Oleson, Bryndon.....	10	Su, Yi.....	30
Orr, Anna .....	33	Sun, Wenyan.....	15
Pak, Daniel .....	10	Sun, Yuxiang .....	26
Paquet, Dominik .....	21	Tessier, Peter.....	30
Parent, Angèle .....	10	Truttman, Matthias .....	11
Park, Goonho .....	10	Vance, Jeffery.....	8
Pimenova, Anna .....	25	Varvel, Nicholas.....	26
Portz, Bede.....	22	Wang, Chao .....	17
Rainey-Smith, Stephanie .....	37	Wang, Yong.....	30
Rajabli, Farid.....	7	Wong, Eitan.....	11
Safar, Jiri .....	11	Young, Jessica.....	22
		Zakaria, Justyna Dobrowolska .....	11
		Zhao, Na .....	17
		Zhao, Yingjun.....	17
		Zhao, Zhen.....	5

## ➤ 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