



FISHER CENTER FOR
ALZHEIMER'S
RESEARCH FOUNDATION



2021 **ANNUAL** **REPORT**

TABLE OF CONTENTS

MESSAGE FROM THE EXECUTIVE DIRECTOR	1
ABOUT US	2
THE FISHER CENTER LAB AT THE ROCKEFELLER UNIVERSITY	3-7
ALZHEIMER'S EDUCATION AND RESEARCH PROGRAM AT NYU GROSSMAN SCHOOL OF MEDICINE	8-11
IMAGINE INSTITUTE, INSTITUTE OF BRAIN AND SPINE	12
MESSAGE FROM THE BOARD CHAIR	13
FINANCIALS	14-15
INFORMATION PROGRAM	16
GRANTS	17
IMPACT AND OUTREACH	18
BOARD AND STAFF	19
ADVISORY COMMITTEE / RESEARCH TEAM	20
HOW TO DONATE	21



FISHER CENTER FOR
ALZHEIMER'S
RESEARCH FOUNDATION

FDR Station, PO Box 220 | New York, NY 10150

T. 212.915.1328 | E. info@alzinfo.org

Message From THE EXECUTIVE DIRECTOR



Dear Friends,

This year's expanded Annual Report is a reflection of the dedication of our many scientists and staff. Despite the challenges of living through a second year of COVID-19, we made great strides in 2021. As you'll see in the following reports, our research scientists have been hard at work searching for a cure.

In addition to our scientific achievements, the Foundation's Information Program continues to expand its reach, providing helpful information to those with Alzheimer's, their families, friends, and caregivers.

As you read this report, I hope you feel as much pride in these accomplishments as I do, as it is your contributions that make our work possible. We are incredibly grateful for each and every dollar you entrust to us.

A handwritten signature in blue ink, which appears to read 'Lucretia', is positioned above the printed name.

Lucretia V. Holden, SHRM-CP

About Fisher Center for Alzheimer's Research Foundation

In 1995, philanthropists Zachary Fisher and David Rockefeller partnered to create and fund the Zachary and Elizabeth M. Fisher Center for Research on Alzheimer's Disease at The Rockefeller University (The Fisher Center lab) after Fisher's wife Elizabeth was diagnosed with Alzheimer's disease. A year later, Mr. Fisher founded the Zachary and Elizabeth M. Fisher Center for Alzheimer's Research Foundation to raise funds to support the lab.

Each year, the Fisher Center Foundation raises millions of dollars for novel Alzheimer's research at the Fisher Center lab in New York City, as well as supporting Alzheimer's research at the NYU Grossman School of Medicine (also in New York City), and the Imagine Institute, Institute of Brain & Spine in Paris, France.

Research conducted at The Fisher Center lab uses state-of-the-art methodologies, and the lab itself is highly-regarded as one of the largest and best-equipped scientific facilities for Alzheimer's research in the country. In 2021, under the direction of Dr. Marc Flajolet and his team of internationally-renowned scientists, progress was



Zachary and
Elizabeth M. Fisher

made to find the causes and cure for Alzheimer's disease.

We hold ourselves to the highest standards of fiscal accountability, with gold seal and four-star ratings from Candid and Charity Navigator. In addition to raising money for research, funds also support our Information Program which is comprised of award-winning resources that provide education and information to those who have been diagnosed with Alzheimer's, their family, friends and caregivers.

Our mission is to understand the causes of Alzheimer's disease, improve the care of people living with it, and find a cure.

Our vision is working towards a future when Alzheimer's is nothing but a memory.

"I know that an answer will not be found in time to help my beloved Elizabeth, but I want to do what I can to find a cure so that others will not have to suffer through the ravages of this disease as my wife and I have had to."

- Zachary Fisher

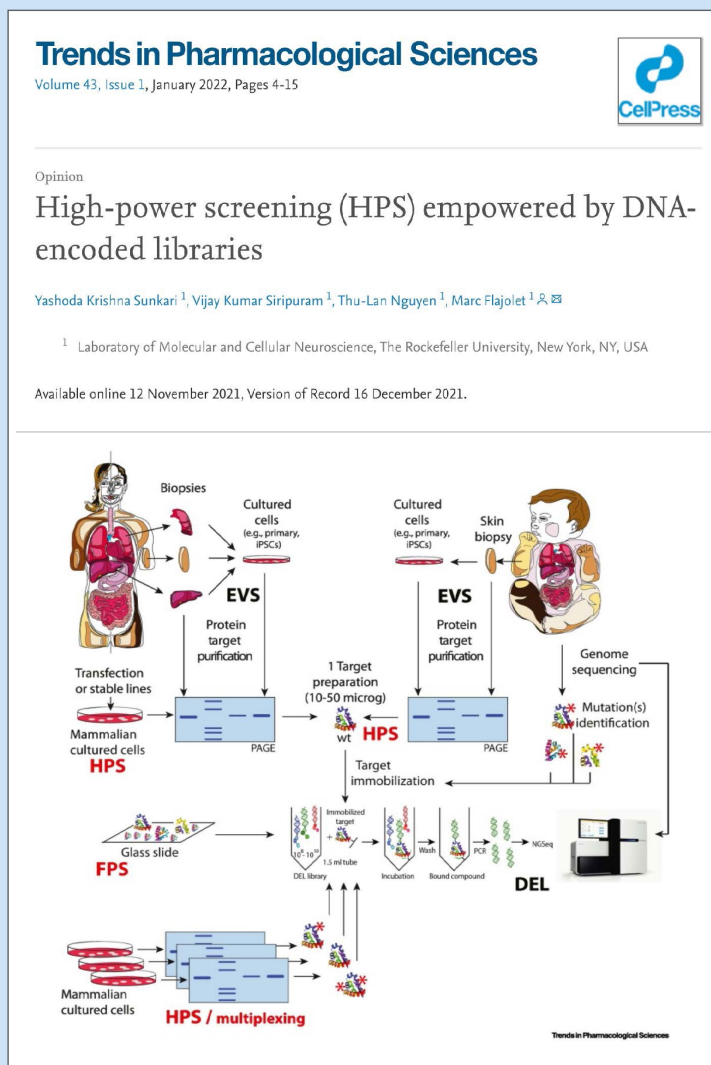
REPORT FROM:

The Fisher Center Lab at The Rockefeller University

Alzheimer's is an extremely complex disease. To address this complexity, Fisher scientists followed their long-standing approach of investigating different complementary aspects of the disease, while focusing more on targeted drug discovery using our own in-house platform.

This year, we made significant progress in five different areas:

- The main part of our project on the selective vulnerability of certain neuron types, which die very early on in the disease was published as we reported and we will continue this work in collaboration with Dr. Jean-Pierre Roussarie. A follow-up manuscript has been prepared and submitted for publication.
- We made great progress on the development of our drug discovery platform using the DNA-encoded library (DEL) technology. We have published a review/opinion piece in the journal *Trends* on this subject.
- We have also published a novel study on the role of microglia in AD pathology with the Memorial Sloan Kettering Institute. This project was initiated by us at the Fisher Center; a pre-clinical study is ongoing.
- We have made significant progress on our efforts to identify novel compounds binding to various types of tau.
- We completed our work on non-neuronal targets. We found that PS1, could affect microglia activation.



Analysis of selective cell vulnerability in Alzheimer's Disease

One of our goals was to identify genes/pathways that are implicated in the degeneration of neurons most vulnerable to Alzheimer's. Our strategy is to take advantage of a combination of state-of-the-art tools, and computational and experimental techniques developed by us over many years. We are developing a model system in order to test the role of each of the genes in the degeneration process.

Progress Report:

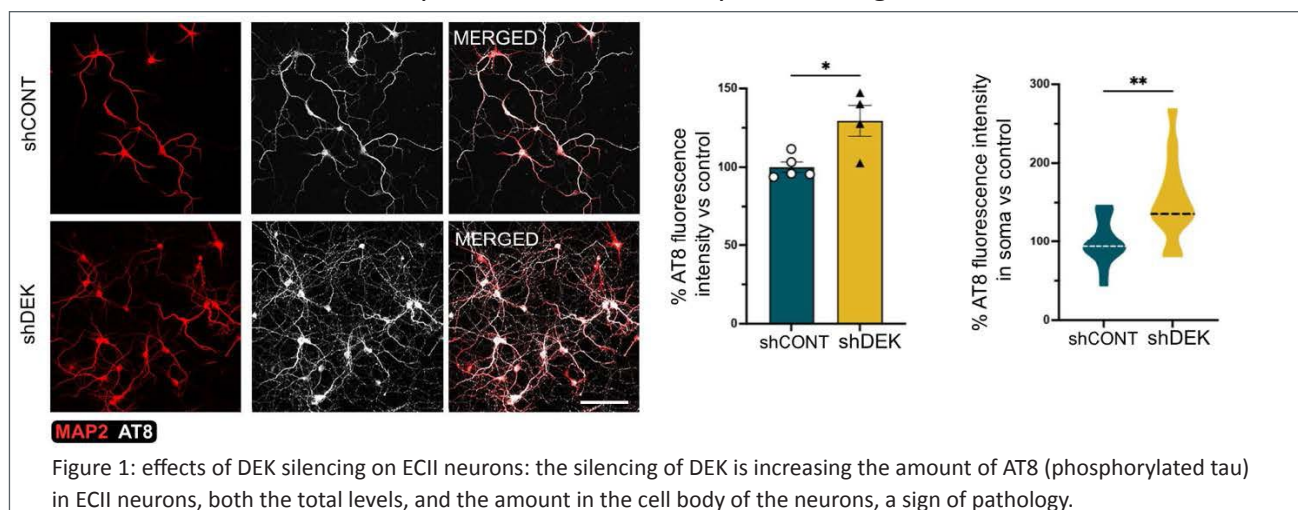
Over the last reporting period, we pursued our investigation of the molecular mechanisms that lead to the selective degeneration of vulnerable neurons in Alzheimer's disease, as well as our development of tools for the study of these mechanisms, and the identification of therapeutic targets.

Mechanisms of degeneration:

We are about to submit to the journal *"Neuron"* a manuscript about how the protein DEK drives tau pathology in the very vulnerable neurons of the brain. This publication summarizes three years of work funded, in part, by the Stringer Foundation. A very comprehensive study on the links between DEK and tau found that DEK, never before associated with neuronal function, was a crucial regulator of neuron excitability and of tau accumulation. We think that during the course of Alzheimer's, DEK becomes dysregulated and thus contributes to pathological tau accumulation and neurodegeneration.

(Figure 1)

This discovery of DEK, and of an entire new mechanism involved in Alzheimer's, represents a major proof of concept. We demonstrate that our approach that we have built over the past decade, meticulously investigating vulnerable neurons, and modeling them with computational tools, has the potential to reveal completely novel genes, never before associated with Alzheimer's. We are currently evaluating the possibility and the feasibility to target DEK therapeutically. We are now investigating a few other vulnerability genes that we have identified, all of which might also be potential therapeutic targets. We follow the same experimental framework as with DEK and hope to discover that similar cellular functions are affected by the other vulnerability candidate genes.



Description of the early stages of the disease:

In another line of research, we obtained brains from patients that had not been diagnosed for Alzheimer's before their death, and that have no (or very early) amyloid and neurofibrillary tangle pathology (control group). On these brains:

- We are comprehensively studying the modifications of tau present in vulnerable neurons at very early stages of the disease using mass spectrometry.
- We performed single-nucleus RNA-sequencing on these samples and found a few significant differences that are all starting to draw a picture of the early stages of the disease: we find that a specific category of cells is activated before microglia is activated.

We are starting to molecularly dissect the earliest stages of the disease, to identify the earliest events, postulate causation links between dysregulation of different cell types, and to uncover possible intervention points.

Construction, characterization and validation of our in-house drug-screening platform based on the DNA-encoded library (DEL) technology

The development and optimization of methodological alternatives to classical methods such as 'high throughput screening' (HTS) have the potential to significantly accelerate the identification of small molecular weight compounds for a given therapeutic target. This will be extremely valuable, especially for Alzheimer's disease, for which therapeutic interventions remain mostly underdeveloped. The few DNA-encoded libraries that exist today, or first generation DEL, are practically inaccessible and limited to a handful of large pharmaceutical companies.

Progress Report:

We introduced some exciting new features to our state-of-the-art drug screening platform, and are happy to report great progress on that front as well. We have now generated several DEL libraries and have made 125 million drug-like molecules. A patent application has been drafted that includes several of the new features.

Drug screening is often hampered by the absence of a robust biological read-out; however, the DEL technology will circumvent this problem altogether. This means that it is even suitable for targets that are usually extremely difficult, including some Alzheimer's targets, because they do not have an activity (or known activity) that can be measured. One of the novel tools (SELDA) that we have designed and validated is presented in **Figure 2 on the next page** and was used to perform DEL optimization.

During this reporting period, we have developed and characterized different reagents focused on tau and tau aggregates. We have designed and constructed molecular tools to express the full protein and

have expressed those constructs into human derived cells growing *in vitro* in the lab. A similar approach was used with truncated versions of tau because those are easier to work with and are known to aggregate faster and more easily.

Three series of DEL screens (for a total of 10 screens using 12.5 million compounds) have been performed, and have designed the very first molecules found in our screens that potentially bind to tau.

GSAP regulates lipid homeostasis and mitochondrial function associated with Alzheimer's disease

The existence of amyloid plaques, as the clearest sign of neuropathology in Alzheimer's, has directed recent

efforts to the clearance of them. One line of research has emphasized the possible inhibition of gammasecretase. Since the enzyme is responsible for processing APP, inhibition of it was hoped to either slow down or reduce beta-amyloid production. However, feasibility of such an approach has been refuted due to the importance of gamma-secretase in other cellular functions crucial for survival.

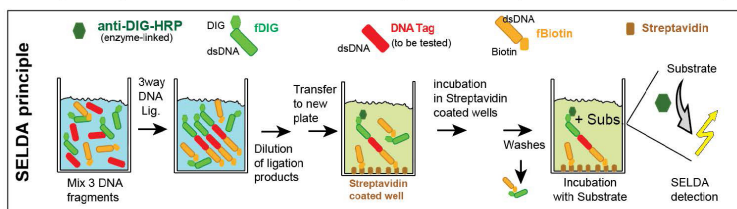
Former lab director Paul Greengard thought that finding a specific inhibitor that only targets gamma-secretase's activity would be ideal. Such precise inhibition would preserve the integrity of gamma-secretase in other critical cellular functions, while limiting its negative effects. A few years back, we discovered such a candidate: GSAP (Gamma Secretase Activating Protein). Prior to our discovery, nothing was known about the biological function of this protein and no association with Alzheimer's disease had been made. Our latest study – defining a novel biological function of GSAP – was published in the well-known *Journal of Experimental Medicine*.

Drug discovery approach to identify drug-like compounds binding to various types of tau aggregates

The protein tau, the second most known Alzheimer's culprit, has regained interest after a number

Visual abstract: SELDA principle

Three types of DNA fragments (fDIG, DNA Tag and fBiotin) are mixed in a tube at equimolar ratio and ligated using T4 DNA ligase. Ligation products are then diluted and incubated in wells of a 96-well streptavidin coated plate. Following washes, ligated products are retained in the wells and fragment ligated on both ends are quantified using horse a radish peroxidase (HRP) based assay.



Visual abstract: SELDA parameters and flexibility

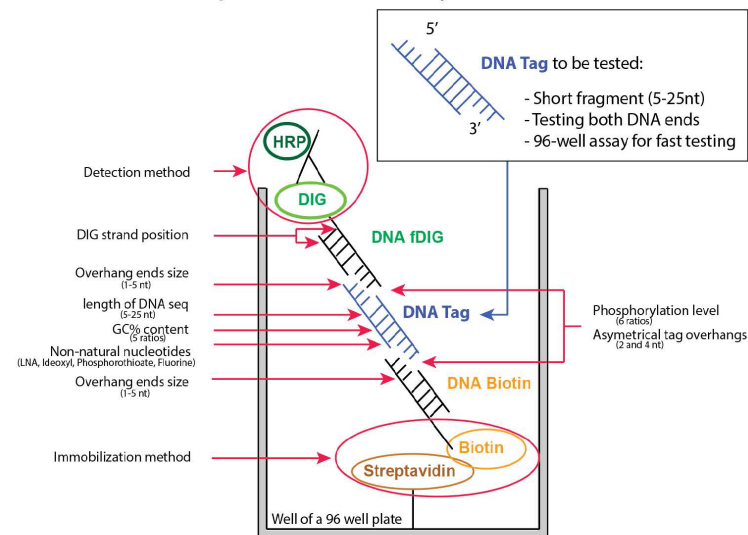


Figure 2 : Schematic representation of a novel tool that we have designed, constructed and validated to improve the quality of our DEL platform.

of important clinical trials targeting Abeta plaques have failed. Tau remains at the center of various important biological functions including disease progression, relevance for memory related functions, expression in brain regions involved in learning and memory, and use as a diagnostic tool. For these reasons, a large effort has been put over the years to target tau aggregates, to either attempt removing them or to reduce their production.

Progress Report:

We initiated work to produce tau in cells at the milligram scale, to purify it, to generate aggregates *in-vitro* and to separate them based on their size. The larger aggregates might represent the best target for identifying compounds binding to them, but they might be less important for the disease state. However, compounds binding to larger aggregates might be best suited for diagnostic purposes. The smaller aggregates, also referred to as oligomers, are believed to be the most toxic ones, possibly also largely involved in disease spreading and progression. We made good progress last year, generating tools, testing antibodies, forming and purifying the aggregates, and we have recently initiated several DEL screening campaigns using tau monomers.

We are hoping that DEL molecules that we might identify could be useful for various aspects of tau biology. The most useful application would be to block or reduce tau aggregation as mentioned above. We have two *in-vitro* validation techniques for that goal but we also would like to optimize one method in cells, and perhaps even *in-vivo* in a tau mouse model of Alzheimer's in collaboration with Dr. Roussarie. Our strategy to block tau aggregation should allow us to address the disease at a very early stage and could possibly stop the spreading of tau seeds and the generation of neurofibrillary tangles, blocking the disease progression. Finally, a compound with strong affinity to tau pathological aggregates, if not associated with a relevant biological activity, could still be useful for diagnostic purposes.

Conclusion

In summary, we have made significant progress for each of our projects. **We currently have six manuscripts in preparation.** Three of those manuscripts were submitted and two are under revision and will be resubmitted soon. **The work on vulnerability is progressing extremely well** and we will pursue our effort as outlined previously. A number of improvements for our DEL platform were successful, and new ones are in the works. **We have worked on a large patent application that has now been filed as a provisional patent** application. We will focus now on target preparation and drug screening campaigns with the DEL compounds. We made significant progress on **generating tools and reagents to identify drug-like compounds that could bind to tau.** More importantly, **we are excited to initiate the validation work of the first batch of tau binding compounds identified.**

REPORT FROM:

The Zachary and Elizabeth M. Fisher Alzheimer's Disease Education and Research Program at the NYU Grossman School of Medicine

With the support of the Fisher Center Foundation, we published our discovery of a new stage of eventual Alzheimer's disease that precedes the stage of Subjective Cognitive Decline, called "Psychometric Cognitive Decline." This was the first discovery and identification of normal persons with no subjective memory complaints, who eventually progress to Subjective Cognitive Decline, Mild Cognitive Impairment and the eventual dementia of Alzheimer's disease.

We recruited 60 subjects with "no cognitive decline (NCD); 47 of these subjects were followed. We collected information longitudinally, approximately every two years on these subjects, which included a psychometric test battery. This battery incorporates tests of Initial and Delayed Paragraph Recall, Initial and Delayed Paired Associates Recall, Memory for designs, Digit Span Recall Forwards, Digit Span Recall Reverse, the Digit Symbol Substitution Test, and the WAIS-R Vocabulary subtest.

A Combinatorial Psychometric Deterioration Score (PDS) was computed based upon an equal weighting of these nine psychometric tests. We analyzed the subject's cognitive decline based on their Global Deterioration Scale stage. This allowed us to determine those subjects who declined, i.e. those who progressed to Global Deterioration Scale stage 2 (Subjective Cognitive Decline [SCD]), Mild Cognitive Impairment (MCI), or dementia, and those who did not decline.

Thirty-six subjects declined to a GDS of ≥ 2 , indicating that they had Subjective Cognitive Decline or more advanced decline. Eleven subjects remained in GDS stage 1, indicating that they had not declined. The subjects' PDS scores were computed and it was found that the PDS was significantly lower at baseline in the future decliners than in the future non-decliners. We published this discovery of a new stage, which we termed, "Psychometric Cognitive Decline" (PCD), as the invited feature article in the *30th Anniversary Research Articles issue* of the journal of *Dementia and Geriatric Cognitive Disorders*.

Additionally, we began our study of risk factors for this newly discovered stage of Psychometric Cognitive Decline. There were no significant between-group differences at the time of entry into the study in terms of gender or education. We also found that, for the future decliner cohort, the mean time to decline was 3.81 ± 1.89 years.

At the present time, age is the only clearly identifiable demographic risk factor for Psychometric Cognitive Decline. Interestingly, although we found that certain Behavioral and Psychological Symptoms of Dementia (BPSD) are a harbinger for decline of SCD persons to MCI, there was no significant difference in these symptoms in our PCD or NCI cohorts.

An investigation into the role of behavioral disturbances in persons with Subjective Cognitive Decline (SCD) has been completed. Seventy-three subjects with SCD, who were participants in our Alzheimer's Disease Research Center longitudinal study, with behavioral disturbance symptomatic assessments, who were 40 years of age or greater and otherwise healthy, were followed for 2.13 ± 0.30 years. Subjects were ≥ 40 years of age at baseline. The majority of subjects, specifically, 48 subjects, were females and 25 of the subjects were males. Subjects were divided into two categories at follow-up: (A) subjects who declined to MCI (Global Deterioration Scale [GDS] stage 3), or dementia (GDS stage ≥ 4), and (B) subjects who remained unimpaired (GDS stage ≤ 2).

We investigated the differences in individual symptoms and categorical symptomatic classifications of BPSD at baseline for subjects who declined at follow-up, and for subjects who had not declined at follow-up. We found there was a significant difference between the two groups.

We assessed whether disease progression was associated with the presence of individual BPSD using the Behavioral Pathology in Alzheimer's Disease Assessment Scale. Additionally we controlled for age, gender, and educational background, and determined the risk for a subject having any BPSD of manifesting cognitive decline at follow-up. We also investigated whether age, education and gender were significant independent risk factors. A manuscript describing these findings has been prepared and is in the process of being submitted for peer-reviewed publication.

We also studied the care and treatment of persons with moderately severe Alzheimer's disease through the effects of a Comprehensive, Individualized, Person-Centered Management (CI-PCM) Program that we developed for persons living with Alzheimer's disease. We examined the impact of the CI-PCM program on psychotropic medication usage and on the BPSD in a 28-week randomized controlled trial. First, we examined the magnitude of usage of psychotropic medications used to treat the BPSD in subjects who had been randomized to receive Usual Community Care (UCC) or the CI-PCM

Program. Psychotropic medications used to treat conditions apart from BPSD were excluded. Subjects were evaluated at baseline and at weeks 4, 12 and 28. We examined the ratio of daily psychotropic medication usage in the two groups.

Psychotropic medications were compared by examining the percent of the maximum daily dosage of each medication taken cumulatively throughout each study observation period from week 4 to week 28. At baseline, the group randomized to UCC consumed a significantly different amount of psychotropic medication than subjects randomized to the CI-PCM program. Because there was a significant difference between the two groups at baseline, we analyzed psychotropic medication usage in comparison with baseline. We then determined the amount of psychotropic medication taken on a per day average relative to baseline from week 4 to week 28 for the UCC subject group and the CI-PCM subject group. The total amount of psychotropic medication taken, in comparison with baseline, was significantly lower in the CI-PCM program subjects. We assessed the effect of the CI-PCM program on the BPSD using the Behavioral Pathology in Alzheimer's Disease Frequency-Weighted Severity Scale (BEHAVE_AD_FW) at baseline and weeks 4, 12, and 28.

In conclusion, we found that the subjects in the CI-PCM program took significantly less psychotropic medication and their BPSD symptomatology significantly improved. Psychotropic medications notably include anxiolytic medications, antidepressant medications, mood stabilizers, soporific medications, and antipsychotic medications. They are prescribed to treat BPSD, including anxiety, depression, agitation, diurnal rhythm disturbances, and psychosis. Many of these medications carry "black box warnings" indicating the need for caution in prescribing because of the possibilities of causing deleterious effects, including death. BPSD are a major cause of burden to care partners of persons living with dementia and are a frequent cause of institutionalization of AD persons. They have also been reported to cause substantial distress and decreased quality of life for both persons with dementia and their care partners (Desai AK, et al., Recognition and Management of Behavioral Disturbances in Dementia.



Prim Care Companion J Clin Psychiatry. 2001; 3(3):93-109). We conclude that the CI-PCM program is a safe, highly efficacious, non-pharmacologic intervention that significantly reduces both BPSD symptomatology and psychotropic medication usage in advanced AD persons. A manuscript describing these findings is being prepared for submission to a peer-reviewed journal for publication.

Dr. Kenowsky served as chair of an oral research session entitled, “Dementia Care Research: Person-centered care and characteristics of care,” where she presented these findings to the scientific and medical community on July 28, 2021 at the premier international scientific meeting for Alzheimer’s Disease and Related Disorders.

Dr. Kenowsky presented a keynote address entitled, “Health Effects of a Comprehensive, Individualized, Person-Centered Management Program in Persons Living with Advanced Alzheimer’s Disease,” at the 13th Annual National Psychiatry Conference in Romania. Additionally, she presented a lecture entitled, “Comprehensive, Individualized, Person-Centered Management Program for Persons with Advanced Alzheimer’s Disease,” as Chair of the Symposium entitled, “New Ways to Improve Health Outcomes with Personalized Alzheimer’s Disease Management” at this conference.

Dr. Kenowsky also presented a lecture entitled, “Comprehensive, Individualized, Person-Centered Management (CI-PCM) Improves Health Outcomes in Alzheimer’s Persons,” as part of the *Troiani-Sweeney Endowed Fund Lecture Series on Caregiving for Caregivers*, at East Stroudsburg University, College of Nursing, in East Stroudsburg, Pennsylvania.

In summary, over the course of the past year, we have continued to advance the understanding and treatment of Alzheimer’s disease. We have also determined that the BPSD are significantly associated with subsequent cognitive decline. We are grateful to the Fisher Center Foundation for their continued support of our work and look forward to sharing future discoveries with you.

REPORT FROM:

Imagine Institute, the Institute of Brain & Spine

Alzheimer's disease (AD) is the most common neurodegenerative disease, but the underlying molecular and cellular events that lead to it are not completely understood. It is known that inflammation plays an important role, so our goal is to study the role of mast cells, a type of immune cells involved in inflammation. Mast cells can play both detrimental and protective roles in various pathological settings. Importantly, these cells have recently been suggested to play a role in neurodegenerative diseases and neuroinflammation, but their contribution to these processes is poorly understood. For example, mast cells are found in higher numbers around amyloid plaques in AD patients and possibly at an early stage, possibly even before other important cells and could therefore be considered as early responders.

Masitinib, a drug inhibiting mast cell functions, has been proven efficient (as an adjunct therapy) in AD in a phase II clinical trial. In an AD mouse model, masitinib had beneficial effects on learning. This result and others suggest that mast cells can, in specific conditions, be toxic for the neurons. Our lab has identified three gene families in mast cells with reported roles in neuropathological settings:

- a. Insulin-regulated aminopeptidase (IRAP): We suggest that IRAP is a new regulator of the inflammatory response of mast cells. A chemical targeting of IRAP by a small molecule inhibitor reduced pro-inflammatory cytokine secretion upon mast cell activation in vitro and in vivo.
- b. Epdr1 is a secreted protein that was found to be secreted/expressed in an IRAP-dependent manner. GWAS data analysis yielded that genetic variants of Epdr1 are associated with the risk of developing Alzheimer's disease. This data provides a strong rationale for the study of Epdr1 in mast cell-dependent inflammation and neurodegeneration.
- c. Phospho-histidine kinases and phosphatases: Increased extracellular Nme1 has been detected both in vitro in a model of brain injury as well as in post-mortem cerebrospinal fluid (a model for neurodegeneration) suggesting that it could also be a marker or functional player in neurodegenerative diseases. Nme1 increased neuronal survival in an injury model suggesting a neuroprotective activity.

These findings, and our continued research, provide hope that a treatment for Alzheimer's disease is within reach.

Message From **THE BOARD CHAIR/TREASURER**



Dear Colleagues and Friends,

We entered the year 2021 hopeful that the COVID-19 pandemic was nearing its end. Little did we know then that we'd be facing another 12 months of face masks, lock-downs and heightened concern over the safety and well-being of our family, friends, and colleagues.

Despite this challenge, our teams of scientists at The Fisher Center lab at The Rockefeller University, NYU Grossman School of Medicine, and Imagine Institute/Institute of Brain & Spine in France continued their ground-breaking work, the results of which have been reported in this document. I hope you feel, as I do, a sense of hope that one day we will find a cure for this debilitating disease, as each new experiment edges us closer to finding the key to unlocking the mystery of Alzheimer's.

In addition, this year we received our tenth consecutive Four Star rating by Charity Navigator, and a Guidestar Gold Seal of Transparency, indicators of our impeccable stewardship of your financial gifts.

We are so very grateful to our donors for their continued belief in our mission to find a cure; thank you for your generosity and support.

A handwritten signature in blue ink, appearing to read "Barry R. Sloane".

Barry R. Sloane

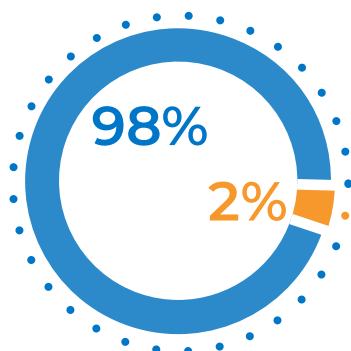
Audited Financial Position

Statement of Financial Position
December 31, 2021
(with comparative amounts at December 31, 2020)

	2021	2020
ASSETS		
Cash and cash equivalents	\$ 6,076,692	\$ 6,476,525
Pledges receivable from federated campaigns	277,890	229,416
Contributions receivable	1,229,862	443,271
Investments	33,173,796	27,626,607
Other assets	32,033	21,853
Furniture and equipment, net of accumulated depreciation of \$12,263 and \$10,728 in 2021 and 2020	<u>12,158</u>	<u>2,078</u>
	<u>\$ 40,802,431</u>	<u>\$ 34,799,750</u>
LIABILITIES AND NET ASSETS		
Liabilities		
Accounts payable and accrued liabilities	\$ 143,669	\$ 240,532
Grants payable, net of discount	<u>10,192,274</u>	<u>2,204,998</u>
Total Liabilities	<u>10,335,943</u>	<u>2,445,530</u>
Net Assets		
Without donor restrictions	30,267,901	32,129,483
With donor restrictions	<u>198,587</u>	<u>224,737</u>
Total Net Assets	<u>30,466,488</u>	<u>32,354,220</u>
	<u>\$ 40,802,431</u>	<u>\$ 34,799,750</u>

2021 Financial Report

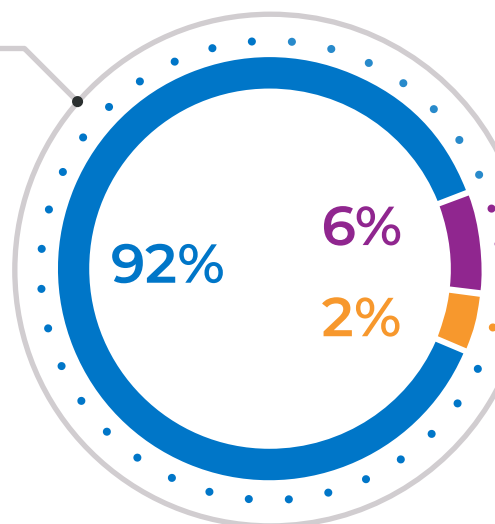
Contributions Breakdown \$8,524,977



- **Contributions, Gifts & Grants**
\$8,326,390
(98%)
- **Federated Campaigns**
\$198,587
(2%)

Expenses Breakdown \$15,211,602

- **Programs**
\$13,957,764
(92%)



- **Administrative**
\$963,405
(6%)
- **Fundraising**
\$290,433
(2%)

Programs

The Foundation's primary goal is to fund the Fisher Center lab, underwriting research to find a cure and understand the causes of Alzheimer's disease. Programs include:

- Our website, ALZinfo.org, which hosts a wealth of information about Alzheimer's disease.
- *Preserving Your Memory*®, a tri-annual magazine filled with caregiving tips and the latest news on Alzheimer's research and treatments.
- *Alzheimer's Research News You Can Use*, an e-newsletter that provides information about how to better live with, and care for, someone with Alzheimer's disease.

Fundraising

Fundraising allows us to promote our mission to end Alzheimer's by raising awareness and providing funding for novel Alzheimer's research.

Administrative

Our administrative expenses support:

- A small, talented staff,
- Legal and accounting services,
- Office equipment and supplies.



Our Information Program

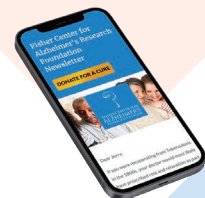
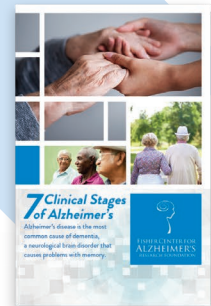
The Fisher Center Foundation curates the findings of scientific research and provides public education and disease awareness through our Information Program, which consists of the following:

Our website, [ALZinfo.org](https://alzinfo.org), provides a library of research findings and includes a resource locator that allows visitors to find doctors, nurses, disease centers, elder attorneys, Medicare information, and home health agencies in their area. The website also features “Ask the Experts,” which allows users to ask questions that are answered by our Fisher Center scientists.

Our free e-newsletter “Alzheimer’s Research News You Can Use,” is a bi-weekly resource that includes articles on the latest news and studies on Alzheimer’s prevention, healthy living and support for caregivers.

Our award-winning magazine, *Preserving Your Memory®*, features research, recipes, exercises, and therapies to alleviate or prevent symptoms, and tips for how best to relate to your loved one with Alzheimer’s. All editorial content is reviewed by our team of scientists for accuracy.

Our social media platforms – Facebook, Instagram, LinkedIn and Twitter – keep followers informed of recent news, articles and events related to Alzheimer’s and the Foundation.



Moving Toward Our Goals

In addition to funding for the Fisher Center lab at The Rockefeller University, the Alzheimer's Disease and Education Program at NYU, and the Imagine Institute in France, the Fisher Center for Alzheimer's Research Foundation has granted additional funds to The Rockefeller University to establish professorships and fellowships to support Alzheimer's research and carry forward the mission of our founders: Find a cure for Alzheimer's disease.

The following grants are ongoing:



Paul Greengard Professorship

This grant honors Nobel laureate Dr. Paul Greengard and the nearly 25 years he spent as Director for the Zachary and Elizabeth M. Fisher Center on Alzheimer's Research lab at The Rockefeller University before his death in 2019. The named chair will be someone who honors Greengard's legacy in conducting research to find a cure for Alzheimer's disease.



The Zachary and Elizabeth M. Fisher Professorship in Alzheimer's and Neurodegenerative Disease

This grant to The Rockefeller University establishes a professorship named for our Founders that will help to advance our understanding of, and treatment for, Alzheimer's and other debilitating diseases. This new professorship is currently held by Dr. Sidney Strickland who heads the University's Patricia and John Rosenwald Laboratory of Neurobiology and Genetics.

Coming in 2022...

The Fisher Fellows Program at The Rockefeller University.

Impact and Outreach

Our mission includes raising funds for Alzheimer's research, and increasing public awareness of the disease through our Information Program. This year, we were successful in both areas, growing our audience reach through radio, and finding new ways to increase revenue.

From the latest in Alzheimer's research, to preventative measures you can take to keep your brain healthy, our Information Program includes our website, ALZinfo.org; a free, bi-weekly e-newsletter; social media platforms; informational booklets and brochures; and our award-winning magazine, *Preserving Your Memory*®.

Alzheimer's Research News You Can Use

Our free bi-weekly e-newsletter provides information on the latest studies related to Alzheimer's disease – information that is helpful to those affected by the disease, their families and caregivers.

116.47% Increase in revenue from our email newsletters

\$31,716.00 (2021) vs \$14,651.75 (2020)

Radio Publicity

A total of nearly 500 Fisher Center Foundation radio spots ran throughout the month of November (Alzheimer's Awareness Month) in the tri-state area, increasing traffic to our website by 2,700 visits.

VIA THE FOLLOWING STATIONS



91%

DIRECT TRAFFIC

Visitors that came to our website directly increased by 91% in comparison to the prior year.

32%

REFERRAL TRAFFIC

Visitors that came to our website from a link on another website increased by 32% in comparison to the prior year.

14%

NEWSLETTER OPEN RATE INCREASE

Our newsletter open rate increased by 14% to be at the industry average of 20%.

Board of Trustees

Barry R. Sloane
Chairman & Treasurer

Dr. Manny Alvarez

James L. Nederlander

Martin Edelman
Vice Chairman

Gerry Byrne

Richard J. Salem

Howard W. Lutnick
Vice Chairman

Hadley M. Fisher

Dr. Moshe Shike

David H.W. Turner
Secretary

The Honorable Betsy Gotbaum

Fisher Center Foundation Staff

Lucretia Holden
Executive Director

Jerry Louis
Web Developer & Graphic Designer

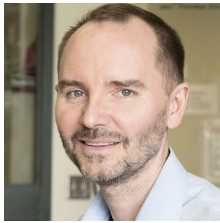
Christina Hall
Development & Communications Director

Josephine Rubin
Electronic Funds Transfer (EFT) Data Specialist

Rebecca Libed
Database Specialist & Analyst



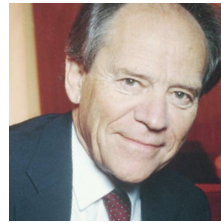
Neuroscience Advisory Committee



Dr. Marc Flajolet



Dr. Michael W. Young



Dr. Hermann Steller



Dr. Torsten N. Wiesel



Dr. Cornelia Bargmann



Dr. Nathaniel Heintz



Dr. Sidney Strickland

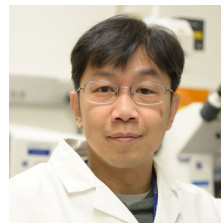
Research Team



Dr. Marc Flajolet



Dr. Yota Sagi



Dr. Jerry Chang



Dr. Revathy Chottekallapanda



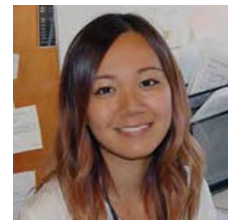
Dr. Lucian Medrihan



Dr. Yashoda Sunkari



Dr. Vijay Siripuram



Dr. Thu-Lan



Dr. Fei Ma



Dr. Benoit Delatour



Dr. Sunnie Kenowsky



Dr. Barry Reisberg

Researchers not photographed:

Dr. Marc Dhenain, Dr. Patrice Dubreuil, Dr. Olivier Hermine

Our Donors are Bringing Us Closer To A Cure

ONLINE Donate online by going to alzinfo.org/donate	BY MAIL FDR Station, PO Box 220 New York, NY 10150	BY PHONE/E-MAIL 1-800-259-4636 1-212-915-1328 info@alzinfo.org
MATCHING GIFTS alzinfo.org/matching-gift Tax ID # 13-3859563	WORKPLACE DONATIONS Inquire with your employer about workplace giving.	BEQUESTS/ PLANNED GIVING For more information please call: 212-915-1322
STOCK TRANSFERS Receiving Bank: Wells Fargo Acct Name: Fisher Center for Alzheimer's Research Foundation DTC #: 0141 Acct #: 3733-3729	MEMORY WALL alzinfo.org/memory-wall	FUNDRAISING alzinfo.org/fundraising

The Fisher Center for Alzheimer's Research Foundation Board of Trustees, Advisory Committee, and Staff humbly thank our donors who enable us to invest in scientific research and information programs for Alzheimer's patients and caregivers. We remain committed to fiscal prudence to ensure sustainability and meaningful resources for the Alzheimer's community.



FDR Station, PO Box 220
New York, NY 10150

212.915.1328

ALZinfo.org

REMAINING A LEADER IN OUR FIELD



WINNING TOP AWARDS AND RATINGS
FOR OUR ACCOUNTABILITY, TRANSPARENCY,
AND HIGH QUALITY
DIGITAL HEALTH RESOURCES