

— 23rd Annual —
ALZHEIMER DAY

ABSTRACT BOOK
THURSDAY, MAY 11, 2017



WELCOME



Dear Friends and Colleagues:

It is my great pleasure to welcome you to our 2017 annual Alzheimer Day.

This year's Mendelson Lecture by Dr. John Morris will focus on revolutionary advances in the early detection of Alzheimer's disease. Dr. Morris has pioneered many of these developments. They introduce exciting opportunities for stopping the disease at its earliest stages, perhaps even before the onset of any memory disorder. The goal of prevention, which appeared to be in the realm of fantasy a few years ago, is now being pursued through rigorous investigations such as the A4 trial that is being conducted in our center.

Following the keynote presentation, and partly overlapping with lunch, we will have a poster session where clinicians and scientists affiliated with the Northwestern Alzheimer's Disease Center will showcase their recent work in the areas of aging, dementia and Alzheimer's disease. The posters will cover topics ranging from basic science to patient care, from emerging medical treatments to behavioral interventions. In the afternoon, we will hold a program in honor of the Glen & Wendy Miller Alzheimer's Family Support Program to celebrate the 20th anniversary of the Northwestern Buddy Program.

It is becoming increasingly clear that Alzheimer's disease comes in different clinical and biological forms. The most common form impairs memory but there are forms that impair word finding, behavior or spatial orientation rather than memory. When it comes to caring for patients, we need to pay attention to these differences so we can personalize interventions according to the principles of precision medicine. We are also realizing that brain aging takes different forms and that incapacitating memory loss is not a necessary part of growing old. Identifying the factors that promote the preservation of memory in advanced age will help us understand the factors that increase resistance to Alzheimer's disease. These themes of heterogeneity in dementia and aging are being pursued through our Primary Progressive Aphasia and SuperAging research programs.

Our center reached a major milestone this year. A \$10 million campaign launched by the Feinberg School of Medicine to double the space of the Cognitive Neurology and Alzheimer's Disease Center and endow its research enterprises has crossed the threshold for starting the construction of the new 10,000 sq. ft. headquarters. We are scheduled to move to our new location by the fall of 2018. The expansion and endowment will allow us to greatly expand our activities and to reach new levels of

excellence. This great milestone would not have been reached without the generosity of our Community Advisory Board and especially the vision and dedication of the Davee Foundation and of Mr. Craig Grannon, who has served as chair of our Community Advisory Board.

In January 2011, President Barack Obama signed the National Alzheimer's Project Act (NAPA), which called for an aggressive and coordinated national plan to attack Alzheimer's disease and improve care and services. Goal 1 of NAPA is to 'prevent and effectively treat Alzheimer's Disease by 2025.' We are rapidly approaching the target date. Based on today's program, I hope you will agree with me that there is now light at the end of the tunnel.

Welcome and enjoy the day.

A handwritten signature in black ink, appearing to read "Marsel Mesulam MD". The signature is written in a cursive, flowing style.

Marsel Mesulam, MD

Ruth Dunbar Davee Professor of Neuroscience and Neurology
Director, Cognitive Neurology and Alzheimer's Disease Center

THANK YOU



The Cognitive Neurology and Alzheimer's Disease Center would like to thank the Mendelson Family for their generous support of this event.

In honor of Robert and Linda Mendelson's 50th wedding anniversary, David and Blythe Mendelson, Sharon and Scott Markman, and Debbie Mendelson Ponn established the Mendelson Lectureship, which brings a keynote speaker to the CNADC's annual Alzheimer Day.

GOLD SPONSORS

We greatly appreciate the support of our Gold Sponsors, Peck Ritchey, LLC and UsAgainst Alzheimer's.

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Alzheimer's Planning Attorneys

Estate Planning – Guardianship – Litigation

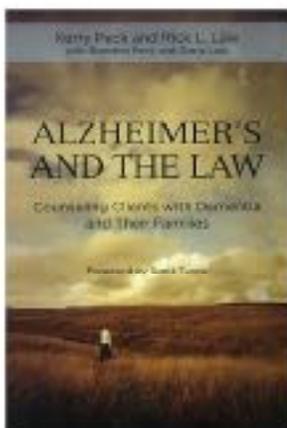


Do you have a family member recently diagnosed with Alzheimer's disease?

Peck Ritchey's Alzheimer's Planning Attorneys will assist you in meeting your family's needs to protect quality of life and financial security for the entire family.

Do you have a family member in the later stages of Alzheimer's disease without advanced planning in place?

After an individual is no longer deemed capable of making decisions for themselves, it can become complicated for them and family members to make medical and financial decisions legally. Having an attorney experienced in Alzheimer's disease planning will help your family gain control over this difficult situation as well as safeguard their wellbeing.



Kerry R. Peck, Managing Partner, is active in the Alzheimer's community as a member of the Alzheimer's Association Greater Illinois Chapter Board of Directors and a frequent presenter at Alzheimer's Association education seminars. His is also co-author of the American Bar Association published book, "Alzheimer's and the Law".

Peck Ritchey's attorneys possess unique expertise to provide guidance and implement legal solutions to protect your family.

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Us4gainstAlzheimer's

Invites you to a celebrity reading of the First Act of Trish Vradenburg's acclaimed play *Surviving Grace*, the touching and humorous story of one family's journey with Alzheimer's.

TUESDAY, JUNE 13, 2017

6:00 PM: Reception

7:00 PM: Celebrity Reading

8:30 PM: VIP Cast Dinner

The Mary Galvin Recital Hall
Northwestern University
Evanston, IL

S U R V I V I N G

Grace

STARRING



MARILU HENNER



DIANE REHM



LONI ANDERSON



MIKE DITKA

Additional Celebrities To Be Announced

PRESENTED BY

Us4gainstAlzheimer's

AND KAREN SEGAL, CHAIR

IN PARTNERSHIP WITH



Cognitive Neurology and
Alzheimer's Disease Center

TO PURCHASE TICKETS OR FOR MORE INFORMATION, VISIT: SurvivingGrace.org

SCHEDULE OF EVENTS

23RD ANNUAL ALZHEIMER DAY

Thursday, May 11, 2017

11:30 AM

Welcoming Remarks

M.-Marsel Mesulam, MD, Director, CNADC, and Ruth Dunbar Davee Professor of Neuroscience, Feinberg School of Medicine

Presentation of Marie and Carl Duncan Prize in Memory Research

John Disterhoft, PhD, Associate Director, Ernest J. and Hattie H. Magerstadt Memorial Research Professor of Physiology, Feinberg School of Medicine

12:00 PM

The Mendelson Lecture

“The Biomarker Revolution in Alzheimer Disease”

John C. Morris, MD, Friedman Distinguished Professor of Neurology and Director, Charles F. & Joanne Knight Alzheimer Disease Research Center at Washington University School of Medicine, St. Louis

1:00 PM

Lunch and Scientific Poster Viewing

2:30 PM

“Living with Dementia: Fostering Connection, Creativity, and Contribution”

Honoring the Glen & Wendy Miller Alzheimer’s Family Support Program and celebrating the 20th anniversary of The Buddy Program

Moderated by Darby Morhardt, PhD, LCSW

Panel Discussion with Northwestern Clinicians

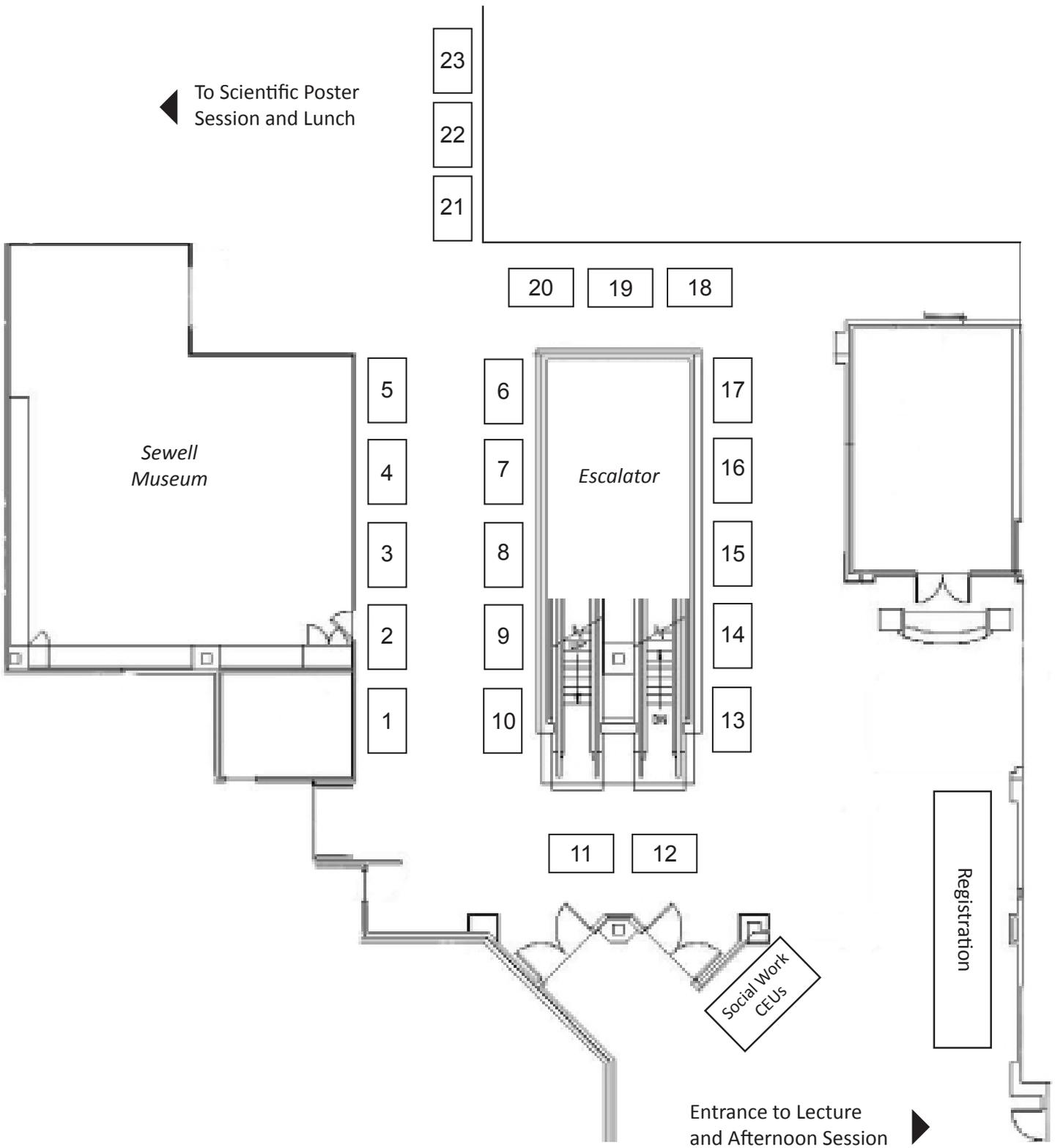
Moderated by Lauren Dowden, MSW, LCSW

4:00 PM

Adjourn

Disclaimer: The advertisements published are not an endorsement of services, nor do they represent the recommendations, opinions, or views of the Northwestern University Cognitive Neurology and Alzheimer’s Disease Center.

MAP OF VENDOR FAIR



LIST OF VENDOR TABLES BY NUMBER

The numbers for each vendor correspond to the Map of Vendor Fair on Page 8.

- 1.....Peck Ritchey, LLC
- 2.....Belmont Village
- 3.....Alzheimer's Association
- 4.....JourneyCare
- 5.....The Abington
- 6.....CJE SeniorLife
- 7.....Elderwerks
- 8.....All Trust Home Care
- 9.....SeniorBridge
- 10.....UsAgainst Alzheimer's
- 11.....Northwestern Cognitive Neurology and Alzheimer's Disease Center (CNADC)
- 12.....Northwestern CNADC Research
- 13.....Home Instead Senior Care
- 14.....Dutton, Casey, & Mesoloras
- 15.....Best Practices Home Care Alliance
- 16.....St. Paul's House
- 17.....The Clare
- 18.....Renewal Care
- 19.....Northwestern Geriatric Medicine
- 20.....Skyline Village and Encore Illinois, NFP
- 21.....Arts Camp for Brain Health
- 22.....Health Learning Center
- 23.....CATCH-ON

THANK YOU

We would like to thank our Silver Sponsor and Bronze Sponsors for their support of this event.

SILVER SPONSOR



BRONZE SPONSORS

The Abington

All Trust Home Care

Alzheimer's Association

Best Practices Home Care Alliance

The Clare

CJE SeniorLife

Dutton, Casey, & Mesoloras

Elderwerks

Home Instead Senior Care

JourneyCare

Renewal Care

St. Paul's House

SeniorBridge

THE 23RD ANNUAL ALZHEIMER DAY PLANNING TEAM

LAUREN DOWDEN

JOSHUA KAPLAN LYMAN

JEANEANE QUINN

KRISTINE ZACHRICH

DARBY MORHARDT, DIRECTOR OF EDUCATION

Thank you to all CNADC staff and faculty who have made this day a success!

The CNADC appreciates your dedication and commitment
to making this day possible.

MORNING SESSION



Marsel Mesulam, MD

Marsel Mesulam is the Ruth Dunbar Davee Professor of Neuroscience and Director of the Cognitive Neurology and Alzheimer's Disease Center at Northwestern University. He served as president of the Organization for Human Brain Mapping, vice president of the American Neurological Association, and chair of the medical advisory board of the Association for Frontotemporal Degeneration. His research has addressed the connectivity of the cerebral cortex in the primate brain, anatomy of human cholinergic pathways, representation of cognitive functions by large-scale networks, and neurobiology of dementias. He

founded the Behavioral Neurology Unit at the Beth Israel Hospital of Harvard Medical School and the Cognitive Neurology and Alzheimer's Disease Center at Northwestern University. He has received the Potamkin Prize from the American Academy of Neurology, the Javits Award from the United States National Institutes of Health, the McKnight Foundation Director's Award, and the Bengt Winblad Life Achievement Award from the Alzheimer's Association. He held the Robert Wartenberg and Houston Merritt lectureships of the American Academy of Neurology. He served on the editorial boards of *Brain* and *Annals of Neurology*. His textbook, *Principles of Behavioral and Cognitive Neurology*, is used by multiple training programs. His current research focuses on the biology of neurocognitive networks and on the pathophysiology of focal dementias. His trainees in clinical, cognitive and basic neuroscience lead major research programs in the United States and abroad.

MORNING SESSION



John Disterhoft, PhD

John Disterhoft and his laboratory group are studying the neurobiology of associative learning in the young and aging mammalian brain with in vivo and in vitro techniques using eyeblink conditioning, spatial learning and fear conditioning as behavioral model systems.

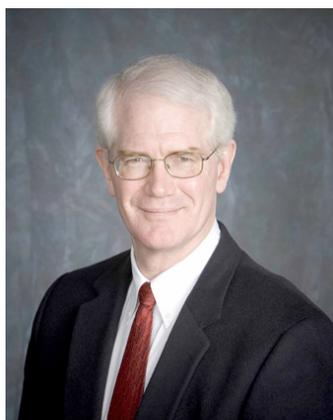
Many of their ongoing experiments focus on the hippocampus, a paleocortical region involved in transferring information during learning from short- to long-term memory storage. Single-neuron ensemble recording in the conscious animal is used to localize and functionally characterize the cell types involved in laying down the "memory trace" in the hippocampus and associated medial temporal lobe regions. In parallel experiments, biophysical measurements are made from brain slices taken from trained animals to define ionic mechanisms for the conditioning-specific alterations in postsynaptic intrinsic currents that have been observed. Synaptic alterations related to conditioning are also being explored in brain slices. Cellular and systems alterations in aging brain that may underlie learning deficits and agents which may be useful in enhancing learning rates in aging are being studied.

An overall goal of their studies is to understand both the mechanisms of learning and of memory storage. Hippocampus is especially involved in the initial acquisition of associative tasks. More permanent memory storage occurs in other brain regions after a process called memory consolidation. Some of their recent experiments are focusing on the manner in which prefrontal, sensory and temporal lobe neocortical regions, and the caudate nucleus of the basal ganglia change during both initial learning and after longer term storage of the eyeblink conditioned response. After regions are defined that store memories of the conditioned response after consolidation, more focused cellular and molecular studies can be done to characterize how this storage occurs at the subcellular level.

The portion of Dr. Disterhoft's research program investigating slow outward currents during learning in aging has received two consecutive MERIT award designations from the National Institute on Aging. He also has funding from the NIA to investigate the synaptic changes occurring in aging hippocampus using cutting edge molecular and 2P imaging approaches. The other portion of his research program involves studying the activity of many single neurons and doing brain imaging in conscious animals during learning and memory consolidation. Dr. Disterhoft directs the Northwestern University NIA funded predoctoral and postdoctoral training program on Mechanisms of Aging and Dementia and the NU IN-PREP postbaccalaureate training program, is Associate Director of the Northwestern University Alzheimer's Disease Center and is Executive Director of the Northwestern University Behavioral Phenotyping Core.

MENDELSON LECTURE

“THE BIOMARKER REVOLUTION IN ALZHEIMER DISEASE”



John Morris, MD

John C. Morris, MD is a leading researcher in the fight against Alzheimer’s disease. Dr. Morris is the Harvey A. and Dorismae Hacker Friedman Distinguished Professor of Neurology, Professor of Pathology and Immunology, Professor of Physical Therapy, and Professor of Occupational Therapy at Washington University. He also is the Director and Principal Investigator of the Charles F. and Joanne Knight Alzheimer’s Disease Research Center at Washington University School of Medicine.

Dr. Morris earned his medical degree from the University of Rochester School of Medicine and Dentistry in Rochester, New York. He completed residencies in internal medicine at Akron General Medical Center and in neurology at the Cleveland Metropolitan General Hospital, both in Ohio, and a postdoctoral fellowship in neuropharmacology at Washington University School of Medicine. The focus of Dr. Morris’ research and practice is Alzheimer’s disease and other neurological disorders associated with aging. He is a former member of the Alzheimer’s Association National Board of Directors and its Medical Scientific Advisory Council. He currently is a member of the Board of Directors for the American Academy of Neurology.

Dr. Morris is author or co-author of over 500 peer-reviewed journal articles, 50 chapters and reviews, and 4 books (current h-index 118). He has received many honors, including the Lifetime Achievement Award from the Alzheimer’s Association (2004), the 2004 MetLife Foundation Award for Medical Research, the 2005 Potamkin Prize for Research in Pick’s, Alzheimer’s, and Related Disease from the American Academy of Neurology, the 2006 Dr. Neville Grant Award for Clinical Excellence from the Barnes-Jewish Hospital Foundation (St. Louis, MO), and the 2008 Washington University Academic Women’s Network Mentor Award. In 2010, he received the Carl and Gerti Cori Faculty Achievement Award from Washington University and in 2013, the Peter H. Raven Lifetime Achievement Award from the Academy of Sciences St. Louis; the Washington University School of Medicine 2013 Second Century Award; and the 2013 Medical & Scientific Honoree from the Alzheimer’s Association. He is ranked in the top 1% of investigators in the field of Neuroscience and Behavior by Essential Science Indicators database.

AFTERNOON SESSION

“LIVING WITH DEMENTIA: FOSTERING CONNECTION, CREATIVITY, AND CONTRIBUTION”

Jakita Baldwin

Jakita Baldwin is currently a fourth-year medical student who is going into Neurology. She received a Bachelor's of Science Degree in Biology with a concentration in Cellular and Molecular Biology from Hampton University in 2011. During her undergraduate career, she conducted research at the National Institutes of Health's National Institute on Aging within the Laboratory of Molecular Gerontology. Her research in this lab examined intracellular localization of base excision repair proteins. Currently, her research interests in aging and cognitive disorders have turned from basic science to social aspects related to cognitive neurology. Under the mentorship of Darby Morhardt, LCSW at Northwestern's Cognitive Neurology and Disease Center, she is currently working to pursue the impact of burden on caregivers of patients with dementia, specifically within the African American community. Aside from research interests, she has participated in a variety of community service and extracurricular activities during medical school including the Good Neighbors Street Outreach Program, which aims to connect homeless populations surrounding Northwestern's medical campus with social services, and the Keep Your Heart Healthy program that strives to improve cardiovascular health in underserved communities around the city. Serving as program coordinator of the Health Professions Recruitment and Exposure Program, she organized a six-week curriculum for underserved and under-represented high school and college students in Chicago. She additionally served as co-president of Feinberg's chapter of the Student National Medical Association. She will continue her training in neurology at Northwestern Memorial Hospital after graduation.

Borna Bonakdarpour, MD, FAAN

Dr. Bonakdarpour received his medical degree from Tehran University of Medical Sciences. His doctoral research on aphasia therapy received international attention and brought him to Northwestern University for his research fellowship in aphasia rehabilitation and neuroimaging of language. Following that he finished his residency in neurology at The University of Arizona and Florane and Jerome Rosenstone cognitive neurology fellowship at the CNADC. He is board certified in neurology and behavioral neurology and was recently received a 5 year career development award by the NIH to study pathophysiology of primary progressive aphasia using functional and structural neuroimaging. He is also an active member of the CNADC clinical trials team and primary investigator of the Connect trial for Alzheimer disease.

He is the director of neurology residents' cognitive neurology training and aside from research/scientific lectures he has been giving invited lectures for patients and family members, medical and physician

AFTERNOON SESSION

“LIVING WITH DEMENTIA: FOSTERING CONNECTION, CREATIVITY, AND CONTRIBUTION”

assistant students, fellows, primary care physicians and medical specialists caring for patients with cognitive impairments and dementias. Dr. Bonakdarpour has authored more than 40 publications, including original scientific papers, reviews, and book chapters.

Irene Dove

Irene Dove was born and raised on Chicago’s south side. She was one of the first African Americans to attend her Catholic school in the 1950s and throughout the 1960s was active in the Civil Rights Movement. She traveled to Senegal as a young adult to learn more about her historical roots. She is a widow with one son and two granddaughters, 11 and 4 years old, whom she adores. Ms. Dove worked in the finance department at the University of Chicago for 35 years before retiring. She is a world history buff and mentored two Northwestern medical students in the buddy program during 2011-12 and 2012-13.

Lauren Dowden, MSW, LCSW

Lauren Dowden is a clinical social worker at the Cognitive Neurology and Alzheimer’s Disease Center, Northwestern University Feinberg School of Medicine, where she works closely with individuals and families, who are living with a dementia diagnosis or changes in cognition. She holds a Masters in Social Work from Loyola University Chicago specializing in mental health with a gerontology sub-specialization and a BA in Theater Arts from Pennsylvania State University. She is also an alumna of *The Second City* (Las Vegas) and is an adjunct faculty member at the Second City Training Center. She teaches medical improvisation for medical students at Northwestern University Feinberg School of Medicine and was an instructor at the 2014, 2015 & 2017 Woltman Inter-Professional Communications Scholars Program for medical interdisciplinary teams at Indiana University and is a co-facilitator for the CNADC and Lookingglass Theatre’s *Memory Ensemble* - an improvisational workshop for individuals with dementia. As an intern at the CNADC, she co-created the first Northwestern CNADC storytelling workshop, Don’t Look Away: Using Storytelling to Give Voice, Find Connections, and Change Perceptions, which she continues to facilitate, develop and research. The storytellers from Don’t Look Away have shared their story with over 1000 people, including health and social service professionals, researchers, students and community members and have been featured on WTTW’s *Chicago Tonight* and in *The New York Times*.

AFTERNOON SESSION

“LIVING WITH DEMENTIA: FOSTERING CONNECTION, CREATIVITY, AND CONTRIBUTION”

Tatiana Carrasquilla

Tatiana Carrasquilla is a third year medical student at Northwestern University Feinberg School of Medicine. Tatiana, born in the city of Bogota, Colombia, moved with her family to America when she was 7 and grew up in Slidell, Louisiana. During her childhood, she discovered her passions of playing soccer, painting, and working with people. She attended the University of Alabama and majored in Biology with a minor in Psychology. During that time, she partnered with her university to start a local food drive called "Swipe Away Hunger" which helped to feed 500 people Thanksgiving dinner through student donations and a school-prepared banquet. She also conducted research in both Alzheimer's disease, studying the relative rates of neurodegeneration in *C elegans* through multiple drug interventions, as well as qualitative research in psychology, interviewing adolescents regarding their perspectives on the parent-child relationship. During her senior year, she pursued a medical volunteer opportunity in Costa Rica before coming to the school of her dreams, Feinberg School of Medicine, where she hopes to create the foundation to later pursue a career in Neurology.

Christine M. Dunford, PhD

Christine Mary Dunford is Director of the School of Theatre and Music at the University of Illinois at Chicago. She has also been an ensemble member with the Tony Award winning Lookingglass Theatre Company since 1989, and has acted in, written/adapted and/or directed nearly three dozen Lookingglass productions. She directed her own adaptation of the New York Times best-selling novel, *Still Alice*, by Lisa Genova, for Lookingglass as part of the company's 25th anniversary season. Christine's production received a Joseph Jefferson nomination for Outstanding New Adaptation, and was selected by the *Chicago Sun-Times* as one of the top ten plays in Chicago in 2013. Christine is currently working on an adaptation project commissioned by Lookingglass' through the Glassworks new work development program; and she performed on the Lookingglass stage in 2016 as the Mother in *Blood Wedding*. Dunford co-founded the Lookingglass Education and Community program and has taught with the program since its inception. She also co-founded the Memory Ensemble, with Darby Morhardt and Mary O'Hara, with the CNADC at Northwestern University Feinberg School of Medicine. She has a PhD in performance studies (Northwestern University), a MA in anthropology (University of Illinois at Chicago), and a BA in theatre (Northwestern University).

Bill Finik

Bill's family moved to northwest Indiana from Pittsburgh PA in the early 50's. His dad was a supervisor in the steel industry and jobs were readily available. His mother was the 'stay at home' supervisor. After graduating from high school in Griffith, Bill began a rather checkered college experience ending with a

AFTERNOON SESSION

“LIVING WITH DEMENTIA: FOSTERING CONNECTION, CREATIVITY, AND CONTRIBUTION”

degree from Indiana State and a Master's degree from Michigan State. Bill started working in the steel industry and retired after 30+ years at the same mill. Bill's hospital experiences at NMH date back almost 50 years to Wesley Memorial Hospital, where Prentice now stands. The Storytelling class, the Memory Ensemble, and the Buddy program have all been extremely helpful in our current fight.

Jennie C. Finik

Jennie's family moved several times while she was growing up in Ohio due to her Dad's work as an engineer with AT & T. They finally settled in Barrington, Illinois where she graduated from Barrington High School. Jennie married Bill 25 years ago after being introduced at work. Since Jennie and Bill entered their marriage rather late in life, they have no children...just dogs. They moved from St. John, Indiana to Valparaiso, IN, which is a great place to live and it's the longest time Jennie has spent in one place without moving. Jennie was very happy when Bill introduced her to Northwestern for their medical needs. Everyone has been very friendly and they are both very satisfied. Thank you.

Natasha Ritsma

Natasha Ritsma is the Curator of the Loyola University Museum of Art (LUMA). She has been running the iLUMAnations program at LUMA since January 2016. iLUMAnations is a program designed specifically for people with memory loss and their care partners. The primary goal of the program is to spark creative dialogue and nurture positive interactions. With the guidance of specially trained docents, participants tour LUMA's rotating exhibitions and permanent collection to view and discuss works of art. Natasha has a dual Ph.D. in Communication and Culture and American Studies and a M.A. in modern and contemporary Art History.

Warren McGee

Warren McGee is an MD/PhD student in Northwestern's Medical Scientist Training Program (MSTP). His family moved around when he was growing up, but he considers Chicago to be his home. He graduated magna cum laude from Columbia University in the City of New York in 2011 with a BA in Biochemistry, Phi Beta Kappa. He then joined Northwestern's MSTP in the fall of 2011 and is currently a PhD candidate in the NU Interdepartmental Neuroscience (NUIN) PhD program.

His PhD research focuses on a protein called FUS that “misbehaves” in some patients with Amyotrophic Lateral Sclerosis (ALS, aka Lou Gehrig's Disease) and Frontotemporal Dementia (FTD). With his PhD mentor, Dr. Jane Wu from Northwestern's Department of Neurology, he is trying to understand the role FUS may have in regulating mitochondria in brain cells. He is doing this by combining the power of bioinformatics and “*Omics” data with molecular biology. He hopes this work will advance our

AFTERNOON SESSION

“LIVING WITH DEMENTIA: FOSTERING CONNECTION, CREATIVITY, AND CONTRIBUTION”

fundamental understanding of how proteins like FUS help maintain brain cells, and why their “misbehaving” may lead to neurodegenerative diseases.

In addition to participating in the Buddy Program, Warren has been active in several ways at Feinberg. He has been a member of the MSTP student council since starting at Feinberg, helping to spearhead an annual series of social and career formation events with the other MSTPs in Chicago, which are now in their sixth year. In 2013 he was a co-founder of the Good Neighbors Homeless Outreach Program, which built on previous student initiatives by partnering medical student volunteers with the Chicago Lights Elam Davies Social Services Center to meet people who are on the streets of Michigan Avenue, and provide emergency food and toiletries, as well as information about services available to them; this program is still active today. Finally, Warren has been involved in a leadership role with the Northwestern chapter of the Catholic Medical Association – Student Section, and is a joyful and active member of his Catholic parish, St. Clement in Lincoln Park.

Katrina Moncrief

Katrina Moncrief grew up in the mountains of North Carolina before attending college at the University of North Carolina at Chapel Hill. She met her husband Jamie at UNC and they moved to Wilmington, North Carolina immediately following graduation – they celebrated their 30th wedding anniversary just two weeks ago. “Kat” has worked as a massage therapist for the past 15 years, operating her own business in North Carolina, and now working in Chicago after she and her husband moved here in 2013. Kat is currently a mentor in the Buddy Program at the Cognitive Neurology and Alzheimer’s Disease Center at Northwestern University.

Darby Morhardt, PhD, LCSW

Darby Morhardt, PhD, LCSW is Research Associate Professor for the Cognitive Neurology and Alzheimer's Disease Center (CNADC) and Department of Preventive Medicine, Northwestern University Feinberg School of Medicine. She leads the CNADC’s Outreach and Recruitment, the Miller Alzheimer Family Support Program, in addition to social work services for the Northwestern Medicine Neurobehavior and Memory Clinic.

The focus of Dr. Morhardt’s work has been on the impact of cognitive impairment on the individual, family and their social networks. Areas of clinical research include the experience of families living with non-Alzheimer’s dementia such as frontotemporal dementia and primary progressive aphasia, the process of tailoring care to needs and symptoms; in addition to the development and evaluation of quality of life programs, support groups and other therapeutic interventions including the award

AFTERNOON SESSION

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winning Buddy Program pairing first year medical students with persons with dementia. The Buddy Program has been replicated in 11 schools nationally and internationally.

Dr. Morhardt participates on national, state and local advisory boards charged with developing dementia specific clinical curriculum. In her role as Outreach and Recruitment Core Leader, she is responsible for organizing the Northwestern Alzheimer’s Disease Center’s community education and outreach programs throughout Chicago and has worked to build community/academic research partnerships in the African American community and many limited English proficiency communities to identify education and service needs and expand and promote research opportunities for these underrepresented groups.

Annika Nilsen

Annika Nilsen is a Feinberg first year medical student from Columbus, Ohio. She graduated in May of 2016 from Vanderbilt University with a BA in Neuroscience, Phi Beta Kappa.

Throughout her Vanderbilt career Annika was a leader in residential life, including her role as head resident in her junior and senior years. She was formed by a variety of experiences—playing for the women’s club soccer team, researching cognitive deficits of schizophrenia in the Park Clinical Neuroscience lab, dancing in the South Asian Cultural Exchange’s Diwali Showcase, and starting a new campus group, Active Minds—an organization that worked to combat mental health stigma on college campuses. She also served as a senator for the College of Arts and Science and a cabinet member of Vanderbilt Student Government, was an active member of the Vanderbilt Programming Board, volunteered at elementary schools through Vanderbilt Student Volunteers for Science, and spent meaningful time at the Vanderbilt University Medical Center shadowing physicians and volunteering with patients.

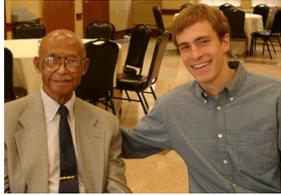
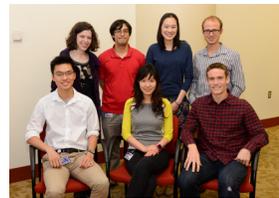
Annika chose Northwestern University Feinberg School of Medicine as the institution to pursue her medical doctorate because of its commitment to medical progress through research, holistic health and wellbeing for all, and collaboration and compassion over competition among medical students and faculty. Since being at Feinberg, Annika has served on the curriculum review committee, admissions student committee, and been an active member of the Buddy Program. She also currently serves as the president of Feinberg’s chapters of AMA and AMWA.

20TH ANNIVERSARY OF THE BUDDY PROGRAM

**DEVELOPED
IN 1997**



**220
BUDDY PAIRS
HAVE BEEN
MATCHED**



**REPLICATED AT 11
UNIVERSITIES NATIONALLY
& INTERNATIONALLY**



A heartfelt thank you to all of our mentors, family members and medical students for taking part in the Buddy Program! It has been an honor to witness these new friendships form over the course of each academic year.

Thank you to the Glen and Wendy Miller Family Foundation for their generous support throughout the years.

Congratulations and here's to the next 20 years!

MARIE AND CARL DUNCAN PRIZE IN MEMORY DISORDERS RESEARCH

Professor Carl Duncan is widely regarded as the first to demonstrate the existence of memory consolidation, showing the vulnerability of recently stored memories. His landmark work is cited more than half a century later. Upon his passing in 1999, his wife, Dr. Marie Duncan, who received her medical degree from Northwestern, set up the Duncan Fund to encourage research and discussion on issues related to memory.

In addition to an annual lecture on fundamental research on memory in the name of Professor Duncan, the Duncan Fund inaugurated in 2006 the Marie and Carl Duncan Prize in Memory Disorders Research to award accomplishments in clinically relevant arenas of inquiry.

MARIE AND CARL DUNCAN AWARD WINNERS

2016

Ashlee E. Rubino

Internalized Tau₄₅₋₂₃₀ Aggregates Can Spread Tau Pathology and Neuronal Degeneration in Alzheimer's Disease and Related Disorders

2015

Dina Simkin, PhD

Calbindin-D_{28k} Restores the Intrinsic Excitability Properties of Aged CA1 Pyramidal Neurons to Young-Like State

2014

Daniel M. Curlik II

Ameliorating Age-Related Cognitive Impairments by Reducing Expression of L-Type Calcium Channels in Area CA1 of the Hippocampus

2013

Diana Schwab Himmelstein

Characterization of the Oligomeric Form of Tau

2012

Tharinda Rajapaksha

The Alzheimer's β -Secretase Enzyme BACE1 is Required for Accurate Olfactory Sensory Neuron Axon Guidance and Normal Glomerulus Formation in the Olfactory Bulb

2011

Carmen Westerberg

Electrically Enhancing Memory Consolidation During Sleep: A Novel Method for Reducing Age-Related Memory Decline

2010

Nicolas Kanaan

Phosphorylation in the N-Terminal Region of Tau Can Regulate Tau-Mediated Inhibition of Anterograde Fast Axonal Transport in the Squid Axoplasm

2009

Katherine Sadleir

The Role of EIF2- α Phosphorylation in A β ₄₂ Induced BACE1 Elevation

2008

Carmen Westerberg

Relationships Between Poor Sleep and Poor Memory in Mild Cognitive Impairment

WHO WE ARE

COGNITIVE NEUROLOGY AND ALZHEIMER'S DISEASE CENTER NORTHWESTERN UNIVERSITY FEINBERG SCHOOL OF MEDICINE



MISSION

The Cognitive Neurology and Alzheimer's Disease Center (CNADC) is a multidisciplinary organization dedicated to the following pursuits:

- Conducting research to discover how the brain coordinates cognitive functions such as memory, language, attention, and emotion.
- Discovering causes and treatments for diseases that disrupt these functions, such as Alzheimer's disease and related dementias.
- Transferring the benefits of this research to patients and their families.
- Training researchers and clinicians who want to work in this field.

RESEARCH AREAS

- Treatment and Prevention of Alzheimer's Disease
- Causes and Treatments of Primary Progressive Aphasia, Frontotemporal Degeneration, and other Younger Onset Dementias
- Nature of Cognitive and Behavioral Changes in Alzheimer's Disease
- Human Cognitive Brain Mapping
- Experimental Treatments
- Chemistry of Memory
- Maintenance of Cognitive Functions in Aging
- Genetics
- Impact of Non-Pharmacological Interventions on Quality of Life

The CNADC has a number of research studies for which we are seeking volunteer participants. If you are interested in participating in memory research and/or you would like to be placed on our mailing list, please contact us at 312-926-1851 or memory-research@northwestern.edu.

320 East Superior Street
Searle 11th Floor
Chicago, IL 60611
Phone: 312-908-9339
Fax: 312-908-8789
CNADC-Admin@northwestern.edu
<http://www.brain.northwestern.edu>

NEUROBEHAVIOR AND MEMORY CLINIC

CARE FOR PATIENTS AND FAMILIES

The Neurobehavior and Memory Clinic is designed to meet the needs of persons experiencing memory loss or other symptoms of dementia, and their families.

SERVICES INCLUDE

- Evaluation and follow-up care by behavioral neurologists who specialize in the diagnosis and treatment of dementia syndromes
- Evaluation of memory and other thinking abilities with the use of specialized tests given by a clinical neuropsychologist
- Management of medication for memory disorders
- The opportunity to participate in clinical research and clinical drug trials
- Psychiatric evaluation and treatment for mood and behavior disorders associated with neurological disease
- Education and counseling for patients and families
- Symptom specific interventions and strategies
- Information and referral to other supportive services

A DEDICATED CLINICAL TEAM

BEHAVIORAL NEUROLOGISTS

M.-Marsel Mesulam, MD, Director
Borna Bonakdarpour, MD
Jay Gottfried, MD, PhD
Ian Grant, MD
Shoaib Memom, MD

NEUROPSYCHIATRISTS

Deborah Reed, MD
Fred Ovsiew, MD

NEUROPSYCHOLOGISTS

Nancy Kennedy, PhD, ABPP-CN
Jana Wingo, PhD
Sandra Weintraub, PhD, ABPP-CN

SOCIAL WORKERS

Darby Morhardt, PhD, LCSW
Lauren Dowden, MSW, LCSW
Joshua Kaplan-Lyman, AM, LCSW

CLINIC MANAGER

Megan Atchu, MA

PSYCHOMETRIST

Nicole Wright, BA, CSP

PATIENT ACCESS REPRESENTATIVES

Cheryl Brown
Anthony Nowaske

Call for an appointment: 312-695-9627

676 North St. Clair Street
Suite 945
Chicago, IL 60611
Phone: 312-695-9627
Fax: 312-695-6072

EXECUTIVE COMMITTEE MEMBERS

ADMINISTRATIVE CORE

M.-Marsel Mesulam, MD

*Director, Cognitive Neurology and Alzheimer's Disease Center
Ruth Dunbar Davee Professor in Neuroscience and Neurology*

John Disterhoft, PhD

Associate Director

Ernest J. and Hattie H. Magerstadt Memorial Research Professor of Physiology

Kevin Connolly, MS

Administrator

CLINICAL CORE

Sandra Weintraub, PhD

Professor

Departments of Psychiatry, Neurology, and Psychology

DATA MANAGEMENT & STATISTICS CORE

Alfred Rademaker, PhD

Professor

Department of Preventive Medicine

OUTREACH & RECRUITMENT CORE

Darby Morhardt, PhD, LCSW

Research Associate Professor

Cognitive Neurology and Alzheimer's Disease Center

NEUROIMAGING CORE

Emily Rogalski, PhD

Research Associate Professor

Cognitive Neurology and Alzheimer's Disease Center

NEUROPATHOLOGY CORE

Eileen Bigio, MD

Professor

Department of Pathology

ADVISORY BOARD

We would like to graciously thank our Advisory Board, founded and led from 1998 to 2008 by the late Jerome Rosenstone.

EXECUTIVE COMMITTEE OF THE ADVISORY BOARD

Terry Chapman, Co-Chair (2016-2018)
Craig C. Grannon, Co-Chair (2016-2018)
David Moscow, Past Chair (2014-2016)

CURRENT MEMBERS

Jason Boschan
Terry Chapman
Susan Chapman
Carole Feiger
Joyce Gallagher
Bill Goldberg
Ivan Himmel
Melissa Kahn
David Mendelson

LIFE MEMBERS

Robert Mendelson
Linda Mendelson
John Van Cleave
Kay Van Cleave
Donna Elrod
Gloria LaGrassa

The CNADC Advisory Board was formed to increase public awareness and knowledge of the Center, and to help garner ongoing philanthropic support for the CNADC's programs and facilities. The Board helps promote the Center both locally and nationally, and assists in securing the funding necessary to position the Center among the premier Alzheimer's research and patient care facilities in the United States.

If you are interested in learning more about the CNADC Advisory Board, please contact Kevin Connolly at 312-503-2832 or visit our website: <http://www.brain.northwestern.edu/about/giving.html>

WHY I PARTICIPATE IN RESEARCH

Responses from CNADC research participants and families.



"MY FAMILY HAS A HISTORY WITH ALZHEIMER'S DISEASE AND I AM WILLING TO DO WHATEVER I CAN TO FURTHER RESEARCH TO FIND A TREATMENT OR A CURE."

"Participation in these studies is our way to help research in important scientific projects."

"We will be judged by how we relate to other people."

"I enjoy it. Also, I am happy improving the health/research for others as well as myself."



"A FEW YEARS AGO MY WIFE LOST HER MOTHER TO ALZHEIMER'S, WE'VE BEEN INVOLVED EVER SINCE."

"I love to do it. It makes me feel good. It gives me some confidence and makes me feel that I am being monitored."

"I believe research is vital to the human condition."

"To be challenged, evaluate myself, and ask questions."



"BECAUSE RESEARCH IS ESSENTIAL TO UNDERSTANDING THE WORLD. ONE LEARNS THINGS FROM SYSTEMATIC ANALYSIS OF DATA THAT CANNOT BE LEARNED FROM COLLECTING ANECDOTES."

THE IMPORTANCE OF BRAIN DONATION

Please help us combat dementia.

To win the fight against Alzheimer's disease and other brain diseases that cause dementia we need more research. Brain donation at the time of death from individuals who have been well studied during life is one of the most important and generous gifts a patient who has lived with dementia and his/her family can make. Brain donations from older individuals who do not suffer from dementia are also critical for comparison and to learn why some people are able to withstand Alzheimer's and other dementias.



Brain donation is one of the most important contributions to research.

The study of brain tissue from individuals with and without disease who have been carefully studied during their lifetime allows scientists to understand the mechanisms of disease, and how those with and without disease differ in their genes and molecules. While major advances have already been made possible through the generosity of brain donation, there is still much more to be learned and a need for continued support.

Brain donation provides a valuable service to families.

A comprehensive autopsy is performed on the brain of donors. The family of the donor receives a full report detailing the neuropathologist's findings. At present, neurodegenerative diseases that cause dementia can only be diagnosed with 100% certainty through a brain autopsy, so families are provided with a definitive diagnosis. Such information is useful if other family members develop a dementia in the future or if there is a known strong family history. Making this generous donation provides the family with a way to potentially help others, which can create a sense of hope and power over the illness that affected their loved one.

Please consider that we are not able to accept every donation.

If someone interested in brain donation was never seen as part of research or for a clinical evaluation at Northwestern University's Alzheimer's Disease Center, we may not be able to accept the brain donation. We can determine on a case-by-case basis if the donation would be appropriate for our research.

Brain autopsy is a decision that individuals and their families can make only after thoughtful consideration. The decision has important emotional and practical implications.

Members of the professional staff at the Cognitive Neurology and Alzheimer's Disease Center at Northwestern are available to talk with you and answer your questions.

Phone: 312-926-1851

Email: memory-research@northwestern.edu

CORE CENTER RESEARCH STUDY

Goals

- Enroll individuals who have been diagnosed with Alzheimer's disease or a related disorder
- Enroll older healthy individuals without memory or other cognitive problems
- Identify a study partner who will be able to provide additional information about the participant
- Obtain information from participants that will support research studies of aging and memory in the larger NU community and the nation
- Understand needs of diagnosed individuals and their families
- Provide counseling, education, and referrals to community services as needed
- Encourage commitment to our brain donation program

Participants May Receive

- Participants receive annual evaluations of memory and other cognitive functions
- We will provide participants with information on the latest treatments and preventions of memory loss
- Participants will also receive our newsletter and other educational materials relevant to preserving memory health
- Social work advice is available to inform participants about community resources
- No cost for participation



Initial Research Visit Include

The enrollment visit takes approximately 2 hours. During this time:

- Demographic information and medical history is gathered from participants and their study partners
- Paper and pencil tests are given to evaluate memory and thinking skills
- A social worker meets with family members and/or care partners
- A blood sample is taken for research on genetic markers
- Participants are informed of our brain donation program

Annual Return Visits Include

The annual return visits take approximately 90 minutes. During this time:

- Information about the previous year is gathered from participants and their family members and/or care partners
- Paper and pencil tests are given to evaluate memory and thinking skills

Research Coordinators: Bitu Rad and Laura Martindale
(312) 926-1851

SUPERAGING STUDY

-OVER 80 AND GOING STRONG-

Does this sound like you or someone you know?

If so, join our research study!



Who?

Adults over the age of 80 who remain actively engaged in life

Why?

To help us better understand and identify factors that contribute to SuperAging, the maintenance of cognitive functioning in old age

Where?

**Northwestern University CNADC
320 E. Superior Street, Searle Building, Chicago, IL**

What is involved?

Participants in our study will visit our center in Chicago every 2 years for:

- **Cognitive testing**
- **An MRI brain scan**
- **Surveys and Questionnaires**

Compensation will be offered for your time

If interested, contact us for more information:

Phone: (312)-503-2716

Email: agingresearch@northwestern.edu

Website: www.brain.northwestern.edu

Study funded by: National Institute on Aging and The Davee Foundation

Grant #: 1R01AG045571-01, IRB #: STU00027225

Study Title: Super Aging study: Correlates of Active Engagement in Life in the Elderly

LANGUAGE IN PRIMARY PROGRESSIVE APHASIA RESEARCH STUDY

Funded by the National Institute of Deafness and Other Communication Disorder and the National Institute on Aging

INFORMATION ABOUT THE STUDY

The study lasts three days total, about seven hours each day including breaks and lunch. On days one and two, you will be in Chicago where you will participate in a battery of neuropsychological tests. Neuropsychological tests are paper and pencil tasks that evaluate your language, memory and other areas of cognition. Also during the first two days, you will have a magnetic resonance imaging (MRI) scan and participate in an event-related potentials (ERP) task. On the third day, you will travel to Northwestern's main campus in Evanston, IL (about 15 miles north) and participate in a variety of language and naming experiments, some that involve voice recording and another that includes ERP testing.

Over 100 people with PPA and 60 age-matched controls will participate in this study. Participants will be asked to return two years later to compare changes between the two visits.

Participants must be:

- Right-handed
- Not claustrophobic
- Safe for an MRI
- Free of any illness or condition other than PPA that would affect their ability to participate now or in the future.

Individuals not seen at the Northwestern Cognitive Neurology and Alzheimer's Disease Center will need to send records (neurology, neuropsychology and imaging reports) and have a phone interview before being approved by the study director to participate.

GOALS OF THE STUDY

The materials collected from your participation in the research study will be used to investigate a variety of topics. The information that we obtain in three days from you and other participants could lead to exciting developments in the knowledge and treatment of Primary Progressive Aphasia (PPA)

Specifically, the goals of this study are:

- To characterize individuals with PPA using neuropsychological testing and brain imaging.
- To investigate naming and word processing problems in PPA and see how they relate to brain changes.
- To increase awareness of PPA, educate others about this unique disorder and encourage more research to eventually develop a treatment.

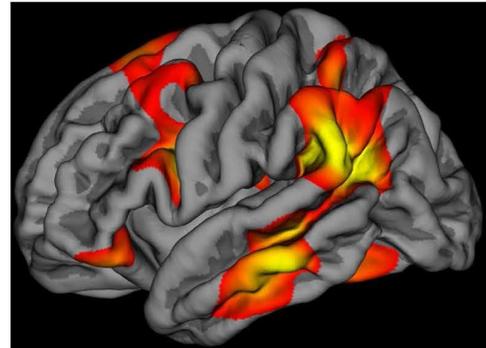


LANGUAGE IN PRIMARY PROGRESSIVE APHASIA RESEARCH STUDY, CONT.

ABOUT MRI

MRI, or magnetic resonance imaging, is a special technique that researchers and clinicians use to see the tissues inside the body. For this study, we will be looking at the brain.

The MRI portion of the study takes about forty-five minutes. You will change into a hospital gown and remove all metallic objects (Jewelry, hearing aids, etc.). You will be asked to lie still on your back on a table with a specifically designed headrest. This will help keep you from moving your head. After you are positioned, the table will slide into the enclosed portion of the scanner.

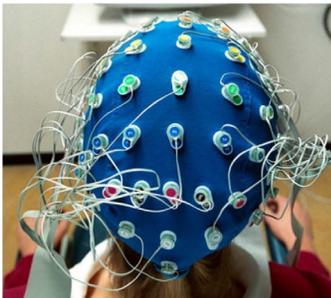


The MRI scanner is loud and you may feel a small vibration, but this is normal. To communicate with the staff through an intercom system and to protect you from the noise, you will be wearing headphones specifically designed for MRI scanners. You will have constant contact with the researchers while you are in the scanner.

The images obtained will be used to compare with other participants, learn more about the brains of people with PPA, and examine the relationship between brain changes and test performance.

ABOUT ERP

ERP stands for event related potentials. This technique is used to analyze electroencephalogram (EEG) results and to learn about specific functions of the brain. In this type of study, the electric activity of the brain is recorded through electrodes placed on the scalp.



To prepare for this experiment, you will sit down while research assistants place an elastic EEG cap with small holes over your scalp. After the cap has been put on your head, a small amount of water-based gel is put into each hole. Sensors are then placed on top of each gel spot. You will be asked to perform a variety of cognitive tasks while the sensors record the signals that your brain emits. This will last about an hour.

After the experiment is finished, the researchers will take off the cap. You may have some gel remaining in your hair. We will have shampoo, towels and a hair dryer available for you if you prefer. Please bring a comb or brush if you require one.

COMPENSATION

TRAVEL | Out of town participants and study partners will have air travel and accommodations paid for and booked in advance by the research study. Local participants will have travel expenses reimbursed.

MEALS | Participants and study partners who are from out of town will be reimbursed for three meals a day; local participants will be reimbursed for lunches.

OTHER COMPENSATION | In addition to travel and meal costs, participants will be paid \$100 per day up to \$300.

PAYMENT | Participation compensation and meal and travel reimbursement will be paid in the form of a check. The check should arrive to the subject's home 30-60 days after the receipts are received.

FOR MORE INFORMATION, PLEASE CONTACT:

Benjamin Rader, benjamin.rader@northwestern.edu, (312) 908-9681
Northwestern University CNADC
320 E Superior Street, Searle 11-579
Chicago, IL 60611

CLINICAL TRIALS

A The A4 Study NOW IS THE TIME

If you are a healthy older adult with normal thinking and memory abilities, **now is the time** to join the fight to prevent memory loss associated with Alzheimer's disease.



NOW IS THE TIME

What is the A4 Study?

The A4 Study (also known as the Anti-Amyloid Treatment in Asymptomatic Alzheimer's study) is a landmark clinical trial to prevent the memory loss associated with Alzheimer's disease (AD). The A4 study is for individuals ages 65 to 85 who may be at risk for AD-related memory loss but who have no outward signs of the disease.

The A4 study is investigating a new treatment that may reduce the impact of a protein known as "amyloid" or "beta amyloid" which forms plaques in the brain. Scientists believe that the accumulation of amyloid in the brain may play a key role in the eventual development of AD-related memory loss.

The investigational treatment used in A4 targets the excess amyloid in the brain with the aim of slowing possible AD-related damage in the brain and delaying symptoms of memory loss.

Learn more about the A4 study:

Cognitive Neurology and Alzheimer's Disease Center (CNADC) at Northwestern University
PI Sandra Weintraub, PhD | STU00087736
1-855-NU-STUDY (312-503-6227)
Email: nustudy@northwestern.edu
<http://tinyurl.com/CNADC-trials>



Phone: 844-A4STUDY (247-8839)
Email: brainlink@ucsd.org

A4study.org

Join the fight to prevent memory loss associated with Alzheimer's disease.

What is involved in the A4 study?

The study lasts approximately three years and participants will be required to visit the clinical research site once a month. Participants will have their health monitored throughout the study using assessments such as:

- Memory and thinking tests
- ECGs
- PET scan
- MRI scans
- Blood and urine tests

You may be eligible to participate in the A4 study if you:

- Are 65 to 85 years old
- Have normal thinking and memory abilities
- Have an A4 study partner who has at least weekly contact with you and can answer questions once a year
- Are willing and able to receive intravenous infusions (IV) of the investigational treatment or placebo (monthly infusions).

A4 participants must be willing and able to participate in all required procedures for the duration of the A4 study.

CLINICAL TRIALS



Worrying About Your Memory?

Join the MIND Study

A Treatment Study for
Mild Cognitive Impairment (MCI)



MIND

Memory Improvement Through Nicotine Dosing

Northwestern University CNADC
PI: Ian Grant, MD | STU00204222
Kristine Lipowski, Research Manager
Phone: 312-503-2486
Email: k-lipowski@northwestern.edu

The Memory Improvement Through Nicotine Dosing (MIND) study will determine whether daily transdermal nicotine will have a positive effect on early memory loss in people diagnosed with MCI.

We need your help.

If you are a healthy, non-smoking adult age 55+ and are interested in learning more about this study, please visit MINDstudy.org or call **866-MIND-150**.

There is no cost to participate.

This study is being conducted by Vanderbilt University and the University of Southern California Alzheimer's Therapeutic Research Institute and funded by the National Institute on Aging (NIA).

CLINICAL TRIALS

Did You Know You Can Help Make Alzheimer's History?

Join the ADNI Study

An Observational Study of Brain Aging



ADNI is a historic study of brain aging which could help change the future of Alzheimer's disease.



We need your help.

ADNI is seeking people over age 55, who are healthy, as well as those with mild memory problems and those who have been diagnosed with mild dementia due to Alzheimer's. There is no experimental medication involved.

To learn more, please visit ADNI3.org or call:

1-888-2-ADNI-95
(1-888-223-6495)

Your local site is:

Northwestern University CNADC
PI: Emily Rogalski, PhD | STU00203359
Jordan Robson, Research Coordinator
Phone: 312-503-5212
Email: Jordan.robson@northwestern.edu

Funded by the National Institutes of Health (NIH) and the Foundation for the National Institutes of Health (FNIH).

GLEN & WENDY MILLER ALZHEIMER'S FAMILY SUPPORT PROGRAM

QUALITY OF LIFE & RESEARCH OPPORTUNITIES

The Northwestern University Cognitive Neurology and Alzheimer's Disease Center (CNADC) provides a number of programs to help support the quality of life of persons living with memory loss, mild cognitive impairment, or other forms of dementia like Alzheimer's disease.

If you would like to learn more about one of the following programs, please contact us at 312-908-9023 or visit www.brain.northwestern.edu



Support Groups

The CNADC offers two support groups for patients and families:

- Frontotemporal Degeneration (FTD) & Primary Progressive Aphasia (PPA) Caregiver Support Group
- Younger Onset Support & Education Group (for persons living with Alzheimer's disease under the age of 65 and their families)

The Buddy Program™

This unique program matches first year students from Northwestern's Feinberg School of Medicine with persons in the early stages of cognitive decline. The Buddy Program provides an opportunity for persons with Alzheimer's disease and related dementias to mentor a medical student and gives medical students the unique advantage of spending time with diagnosed individuals at an early stage of illness and outside of the clinical setting.

The Memory Ensemble™

A collaboration between the CNADC and the Lookingglass Theatre Company, the Memory Ensemble is an improvisational theatre experience for persons in the early stages of memory loss. Program participants learn to use their instincts, creativity, and spontaneity as they explore and create together. During this 8-week program, benefits of this non-pharmacological intervention are investigated.

ilLUMAnations

Designed as a program for people with memory loss and their care partners, the primary goal of ilLUMAnations is to spark creative dialogue and foster meaningful exchange around art in a supportive environment. With the guidance of specially trained docents, participants tour exhibits at the Loyola University Museum of Art.

Storytelling Workshop

This workshop offers individuals in the early stages of cognitive decline and their partners an opportunity to develop and write a shared story from their lives through reminiscence and exploration of the impact dementia has had on their lives. The program seeks to preserve couplehood and decrease social isolation.

SEED: SUPPORT & EDUCATION FOR EARLY DEMENTIA PROGRAM

Join us for an 8-week group for individuals with Alzheimer's disease or related disorders and their families. Group sessions will provide:

- Education and resources from professionals
 - Coping strategies
 - Discussion
 - Emotional support
-

Fall and Spring Sessions Offered

WEEK #1

Welcome and Introductions

WEEK #2

The Basics of Dementia

WEEK #3

Coping with Changes: Practical and Functional Interventions

WEEK #4

Coping with Changes: Maintaining Your Relationships and Disclosing the Diagnosis with Others

WEEK #5

Coping with Changes: Supportive Community Resources & Interventions

WEEK #6

Research Update and Opportunities

WEEK #7

Legal & Financial Considerations

WEEK #8

Life After SEED: Creative & Supportive Interventions and Q &A with Families Living with a Diagnosis

[Schedule is subject to change.]

INTERVIEW REQUIRED TO PARTICIPATE

There is a \$150 charge per person for the program (Scholarships are available)

Discount parking will be provided.

Please contact facilitators with any questions:

Lauren Dowden, MSW, LCSW
lauren.dowden1@northwestern.edu
312.503.0604

Darby Morhardt, PhD, LCSW
d-morhardt@northwestern.edu
312.908.9432

Joshua Kaplan-Lyman, AM, LCSW
jkl@northwestern.edu
312.503.5209

COUNSELING, EDUCATION AND SUPPORT

Clinical Social Work Consultation: A Customized Approach to Care



The Northwestern Neurobehavior and Memory Clinic offers a multidisciplinary team approach. Your care team includes neurologists, psychiatrists, neuropsychologists and social workers. Clinical social workers are available to discuss your questions and work with you to develop a personal and customized approach to care. Following are some questions you may have:

- **“Do I understand the diagnosis?”**

Your social worker will:

- Review the diagnosis and provide the opportunity to ask questions and get up-to-date disease information.
- Discuss changing behaviors and other diagnosis-related symptoms, and offer helpful communication strategies.

- **“How do I cope with this now and as it progresses?”**

Your social worker can:

- Provide counseling regarding changing roles as the disease progresses.
- Help you to assure your own self-care and to strengthen your support network.
- Provide referrals for individual, couples, and/or family counseling.

- **“How can I plan for future care?”**

Your social worker can:

- Connect you to trusted elder law attorneys for estate planning and to establish powers of attorney for health care and finances.
- Provide counseling regarding advance directives.
- Help you to explore long-term care options and funding sources.

- **“What services are available at Northwestern or in my own neighborhood?”**

Your social worker can guide you to:

- Specialized support and education groups for newly diagnosed individuals and families.
- Quality-of-life programs designed to offer meaningful and purposeful activity.
- Other community programs in which you can find enriching opportunities.

Please call the Northwestern Neurobehavior and Memory Clinic, 312-695-9627 or ask your doctor for a referral for a clinical social work consultation.

ARTS CAMP FOR BRAIN HEALTH

Leading Chicago non-profit organizations join together to create a day of arts programming for those with early-stage memory loss, diminished cognitive and neuro-motor function, and those who care for them.

Arts Camp for Brain Health

When

Thursday, June 15, 2017

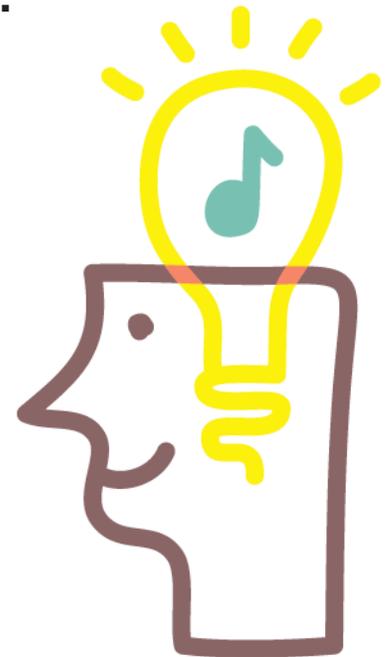
9:00 am – 12:30 pm, \$10 per person

Where

Old Town School of Folk Music
4545 N Lincoln Ave, Chicago

ADA Accessible

registration opens April 27, 2017
at oldtownschool.org/braincamp



- 9:00-9:30am Registration and Refreshments in the Lobby
- 9:30-9:45am Welcome and gathering of participants with performance by Old Town School's Memory Singers
- 10:00-11:00am Workshop Sessions 1, Art making, Dance, Theatre Improv, Video interviews, and Music
- 11:15-12:15pm Workshop Sessions 2, Art making, Dance, Theatre Improv, Video interviews, and Music
- 12:30pm Farewell gathering in the Lobby

Participating organizations include:

Art Institute of Chicago, Boomers Plus, Hubbard Street Dance Chicago, Lookingglass Theatre Company, Loyola University Museum of Art, Northwestern Medicine-Cognitive Neurology and Alzheimer's Disease Center, Old Town School of Folk Music, and Video Family Biographies

Additional support provided by:

All Trust Home Care, Health Research & Educational Trust, and Mel Winer Graphic Design

"...one of the things that art does, is it heals" Leonard Cohen



Voices of Belmont Village

"The staff was able to see through Dad's dementia to recognize and appreciate his real personality."

When the symptoms of dementia affect a loved one, it can be confusing and heartbreaking. Created in partnership with leading universities, Belmont Village memory programs help residents and family members focus on what is there — not what is lost. Through uniquely personalized care and research-based exercises and activities, our specially trained staff provides the structure and support you both need. He'll always be your dad.

BELMONT *Village*
SENIOR LIVING
OAK PARK

Distinctive Residential Settings | Chef-Prepared Dining and Bistro
Premier Health and Wellness Programs | Award-Winning Memory Care
Professionally Supervised Therapy and Rehabilitation Services

**Belmont Village proudly salutes the extraordinary work of
Northwestern University's Cognitive Neurology and Alzheimer's Disease Center.**

The Community Built for Life.®

708-848-7200 • belmontvillage.com

Winner of the Argentum 2016 Best of the Best and George Mason University Healthcare
Award for the Circle of Friends® memory program for Mild Cognitive Impairment.



MAP OF POSTER SESSION

Lunch Buffet

To Vendor Tables
and Lecture

49	25	24	1
48	26	23	2
47	27	22	3
46	28	21	4
45	29	20	5
44	30	19	6
43	31	18	7
42	32	17	8
41	33	16	9
40	34	15	10
39	35	14	11
38	36	13	12
	37		

Lunch Buffet

POSTER SESSION

CELL & MOLECULAR BIOLOGY

- 1 Improving the Tool Kit for Characterization of Pathogenic A β Oligomers in Alzheimer's Disease**
Erika Cline, Arighno Das, Josette Kamel, Anthea Weng, Jake Paschall, Nadia DiNunno, Adriano Sebollela, Kirsten Viola, William Klein
- 2 Cognitive Improvement in an Alzheimer's Disease Mouse Model Mediated by Autophagy Hyperactivation**
Altea Rocchi
- 3 A β and Tau in Dystrophic Axon Formation in Alzheimer's Disease**
Shahrnaz Kemal, Katherine R. Sadleir, and Robert Vassar
- 4 Alsin^{KO}-UeGFP Mice, the CSMN Reporter Line for Alsin, Display CSMN-Specific Cellular Defects Without Major Cell Loss**
Megan Schultz, Mukesh Gautam, Gabriella Sekerkova, Javier H. Jara, Marina V. Yasvoina, Han-Xiang Deng, Marco Martina, P. Hande Özdinler

CLINICOPATHOLOGIC STUDIES

- 5 Diagnostic Challenges of Fahr's Disease: A Case Report**
Ellen Fitzmorris, Qinwen Mao, Esther Bit-Ivan, Eileen Bigio, Borna Bonakdarpour
- 6 APOE ϵ 4 Is a Lesser Risk Factor for Frontotemporal Than Amnesic Dementia with Alzheimer's Neuropathology**
Letizia G. Borges, Alfred W. Rademaker, Eileen H. Bigio, M-Marsel Mesulam, Sandra Weintraub
- 7 Neuropsychiatric Symptoms Related to Neuropathologic vs. Clinical Diagnosis of Dementia**
Letizia G. Borges, Alfred W. Rademaker, Eileen H. Bigio, M-Marsel Mesulam, Sandra Weintraub
- 8 Cognitive Deficits in the First 24 Hours After the Onset of First Unprovoked Generalized Tonic Clonic Seizure**
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POSTER 1

IMPROVING THE TOOL KIT FOR CHARACTERIZATION OF PATHOGENIC A β OLIGOMERS IN ALZHEIMER'S DISEASE

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Purpose. Amyloid beta oligomers (A β O) accumulate early in Alzheimer's disease (AD) and experimentally cause memory dysfunction and the major cellular pathologies associated with AD (e.g., tau abnormalities, synapse loss, oxidative damage, etc.). However, the structures of the A β O species most germane to AD pathogenesis are ill-defined. This uncertainty regarding the pathophysiologically relevant A β O structures has impeded therapeutic advances (e.g., high-profile failures of A β immunotherapies in clinical trials) and, consequently, diminished the perceived therapeutic value of A β . My long-term research goal is to elucidate the structural characteristics of A β O that contribute to their role in the pathogenesis of AD. As a first step towards this goal, we have developed improved methods to stabilize, isolate, and characterize AD-relevant A β O.

Novel Methodology Introduced. We have developed (1) a cross-linking protocol capable of locking A β O conformation; (2) an array of antibodies targeting distinct sub-populations of AD-relevant A β O; and (3) an ultrasensitive assay for quantification of A β O, which are present in very low quantities in AD brain and CSF.

Findings. Using these tools, we have identified an A β O species in the brain of AD patients and mouse models, which is between 100 and 300 kDa, and can be identified specifically by our antibody NUsc1. This A β O species is capable of binding synapses in cultured neurons and inducing AD-associated pathologies, including tau phosphorylation and oxidative stress. Current efforts aim to achieve a more detailed structural characterization of this A β O species as well as to track its accumulation and spread during AD progression.

Practical Implications. We have identified an A β O species relevant to the development of Alzheimer's disease in the human brain and have developed an antibody targeting this A β O species. We predict this antibody, NUsc1, to have value as a disease-modifying therapeutic and/or early diagnostic for Alzheimer's disease.

POSTER 2

COGNITIVE IMPROVEMENT IN AN ALZHEIMER'S DISEASE MOUSE MODEL MEDIATED BY AUTOPHAGY HYPERACTIVATION

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Autophagy is a lysosomal degradation pathway evolutionarily conserved, and it is essential for physiological functions, cellular homeostasis and nutrient recycling. Impairment of the autophagy pathway has been associated with the pathogenesis of Alzheimer's disease (AD), a neurodegenerative disorder characterized by abnormal deposition of extracellular and intracellular amyloid β ($A\beta$) peptides, leading to progressive neuronal loss and memory deficits. To study whether autophagy plays a beneficial role in $A\beta$ clearance and cognitive improvement in AD, we developed a novel mouse model to hyperactivate autophagy *in vivo*. We found that knock-in of a point mutation F121A in the essential autophagy gene Beclin 1/Becn1 in mice, significantly reduces the interaction of Becn1 with its inhibitor Bcl-2, and thus leads to constitutively active autophagy in multiple tissues, including brain. Becn1F121A-mediated autophagy hyperactivation markedly decreases amyloid accumulation and prevents cognitive decline in AD mouse models. In addition to genetic activation of autophagy by the Becn1 mutation, we also found that ML246, a small-molecule autophagy inducer, as well as voluntary exercise, a physiological autophagy inducer, exert similar Becn1-dependent protective effects on $A\beta$ removal and memory improvement in AD mice. Taken together, these results established useful new approaches to activate autophagy *in vivo*, and revealed the important function of autophagy hyperactivation in brain for the prevention of Alzheimer's disease.

POSTER 3

A β AND TAU IN DYSTROPHIC AXON FORMATION IN ALZHEIMER'S DISEASE

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Alzheimer's disease (AD) is characterized by amyloid plaques composed of the β -amyloid (A β) peptide surrounded by swollen presynaptic dystrophic neurites consisting of dysfunctional axons and terminals that accumulate the β -site amyloid precursor protein (APP) cleaving enzyme (BACE1) required for A β generation. In addition, we have shown that they accumulate BACE1 cleavage products of APP, indicating they are a source of A β generation and could contribute to progressive pathology. The cellular and molecular mechanisms that govern presynaptic dystrophic neurite formation are unclear, and elucidating these processes are necessary to develop novel AD therapies. Previous studies suggest A β disrupts microtubules, which we hypothesize have a critical role in the development of presynaptic dystrophies. To investigate this further, here we have assessed the effects of A β , particularly neurotoxic A β 42, on microtubules during the formation of presynaptic dystrophic neurites *in vitro* and *in vivo*. Live-cell imaging of primary neurons revealed that exposure to A β 42 oligomers caused varicose and beaded neurites with extensive microtubule disruption, and inhibited anterograde and retrograde trafficking.

In addition to amyloid plaques, another hallmark pathology of AD is intraneuronal deposits of tau, a microtubule binding protein that plays a role in microtubule stability and in vesicle trafficking. Data indicate that tau hyperphosphorylation and tangle formation in AD are downstream of amyloid accumulation, and are the cause of neuronal death. For this reason we are investigating whether A β induced microtubule disruption and toxicity are tau dependent or independent. We treated primary hippocampal neurons from tau knockout mice with A β 42 oligomers, and using live cell imaging, observed microtubule beading equivalent to that seen in cultures from tau wild type mice. We also examined the formation of dystrophic neurites and plaque formation in a tau knockout Alzheimer's disease mouse model (5XFAD). In preliminary analysis, we find that the absence of tau does not ameliorate plaque or dystrophy formation *in vivo*. These results indicate that some effects of A β , such as microtubule breakdown and dystrophic neurite formation, are not mediated by tau.

From a therapeutic standpoint, these experiments are informative because they highlight the importance of blocking amyloid induced microtubule dysfunction either by targeting amyloid directly, or by restoring microtubule networks and vesicle trafficking through microtubule stabilization. We propose that brain-penetrant microtubule stabilizers could be of use in treating or preventing the cognitive decline in AD by preventing dystrophic neurite formation around plaques that lead to increased A β generation.

POSTER 4

ALSIN^{KO}-UeGFP MICE, THE CSMN REPORTER LINE FOR ALSIN, DISPLAY CSMN-SPECIFIC CELLULAR DEFECTS WITHOUT MAJOR CELL LOSS

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Corticospinal motor neurons (CSMN) are unique in their ability to collect, integrate, translate and transmit cerebral cortex's input towards spinal cord targets. Their degeneration is the key in numerous neurodegenerative diseases, such as amyotrophic lateral sclerosis (ALS). Mutations in the Alsin 2 (ALS2) gene are reported to be responsible for juvenile primary lateral sclerosis, infantile onset ascending hereditary spastic paraplegia, and are the most common cause for autosomal recessive juvenile ALS. In addition, upper motor neuron signs and bulbar symptoms are often prevalent in patients with juvenile ALS. However, cellular and molecular aspects of CSMN degeneration has not been studied in detail due to lack of selective markers to visualize these neuron populations *in vivo*. By crossing UCHL1-eGFP with Alsin^{KO}, we generated Alsin^{KO}-UeGFP mice, a CSMN reporter line to investigate upper motor neuron defects in the absence of Alsin. This novel reporter line helped us visualize and study CSMN at different stages of disease progression. Different from the hSOD1^{G93A} mice or the TDP-43 mouse models, the numbers of CSMN do not show dramatic reduction in the absence of Alsin. However, detailed cellular analysis using immunocytochemistry coupled with electron microscopy (EM) revealed very precise aspects of cellular defects that are restricted to CSMN. We find that even though CSMN do not undergo massive cell loss, the neurons are not healthy. The apical dendrites of CSMN become vacuolated, and this cellular defect is observed only in CSMN in the motor cortex. In addition, there are defects in the mitochondria, and there are signs of defective autophagy, with enlarged lysosomes that contain defective mitochondria, in addition to other proteins. The integrity of the cell membrane is impaired and becomes leaky especially towards end-stage. These findings suggest that Alsin is an important protein for proper CSMN function, and in its absence CSMN display precise neuronal defects, but such defects do not initiate their clearance. Therefore, even though the neurons are still present at layer V of the motor cortex, they are unhealthy and potentially nonfunctional.

POSTER 5

DIAGNOSTIC CHALLENGES OF FAHR'S DISEASE: A CASE REPORT

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BACKGROUND: Fahr's disease (or 'striopallidodentate calcinosis') is a rare, genetic, neurodegenerative condition. The most common clinical manifestations are movement disorders similar to those seen in Parkinson's disease, and the second most common are cognitive and/or psychiatric disturbances similar to those seen in dementia with Lewy bodies, schizophrenia, and autoimmune encephalitis. The imaging hallmark of Fahr's disease is abnormal, bilateral calcium deposits in the basal ganglia and other regions on brain computed tomography (CT) and magnetic resonance imaging (MRI) scans. However, this imaging finding is often interpreted as benign, because it can also occur in 0.7% of asymptomatic older individuals (Manyan, 2005). The low specificity of signs and symptoms pose a challenge to diagnosis of Fahr's disease. Here, we report a case and offer suggestions for improving detection.

METHODS: Case study

RESULTS: The patient was a 76-year-old woman with a 1-year history of non-amnestic cognitive impairment with delusions, hallucinations, and obsessive-compulsive behavior. Family history included schizophrenia in the mother. Neuropsychological testing revealed mildly impaired attention, executive function, and working memory, and examination confirmed the patient's history of severely impaired comportment (i.e. behavior). MRI and CT scans showed mild generalized atrophy and calcification in the basal ganglia and cerebellum that was interpreted as benign. The patient was initially diagnosed with dementia with Lewy bodies and treated with antipsychotic medications, but her delusions persisted. Subsequent blood tests showed elevated antithyroglobulin and thyroid peroxidase antibody, suggesting the patient had Hashimoto's encephalitis, yet she did not respond to steroid treatment. The patient soon died from complications of acute pancreatitis. Post-mortem neuropathologic evaluation of the patient's brain tissue found no signs of Hashimoto's encephalopathy. Instead, it revealed severe and extensive cerebrovascular mineralization, thereby confirming the diagnosis of Fahr's disease.

DISCUSSION: This case underlines two clues that should raise the possibility of Fahr's disease as the underlying cause in an individual with cognitive impairment, psychosis, and brain calcification. First, more attention should be paid to a finding of widespread calcification on head CT and/or MRI scans. Whereas incidental and mild calcification in asymptomatic elderly individuals is typically restricted to the globus pallidus, calcification in patients with Fahr's disease calcification is more extensive and severe affecting the cerebellum (Manyam, 2005). Second, our patient's mother most likely also had the disease but it was misdiagnosed as schizophrenia. Associated family history should raise the possibility of Fahr's disease as the underlying cause of cognitive impairment, psychosis, and brain calcification.

POSTER 6

APOE ϵ 4 IS A LESSER RISK FACTOR FOR FRONTOTEMPORAL THAN AMNESTIC DEMENTIA WITH ALZHEIMER'S NEUROPATHOLOGY

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OBJECTIVE: The present analysis was carried out in a large dataset to determine if the Apolipoprotein E ϵ 4 allele (APOE ϵ 4) frequency in behavioral variant frontotemporal dementia with Alzheimer's Disease neuropathology (FTD/AD) is the same as in dementia of the Alzheimer type with AD neuropathology (DAT/AD).

BACKGROUND: AD neuropathology produces different clinical phenotypes, most typically a syndrome of progressive amnesia (DAT) and more rarely, progressive aphasia (PPA) or progressive behavioral disturbance (FTD). FTD is commonly associated with frontotemporal lobar degeneration (FTLD) but it can be related to AD in 20% of cases. The most common genetic risk factor for DAT/AD is APOE ϵ 4 but it is not known if AD with clinical FTD has the same genetic risk profile.

METHODS: Retrospective analysis of post-mortem data obtained from 1304 cases in the National Alzheimer Coordinating Center (NACC) database compared APOE ϵ 4 frequency in cases with AD or FTLD and a clinical diagnosis of DAT or FTD. A secondary analysis compared APOE ϵ 4 frequency in those with FTD/AD as a function of the presence of motor symptoms. Chi-square analyses were used.

RESULTS: In clinical FTD cases, APOE ϵ 4 was more common with AD (46%) than with FTLD neuropathology (26%; $p=0.011$). However, there was a higher frequency of APOE ϵ 4 in those with DAT/AD (61%) than with FTD/AD (46%) ($p=0.026$). Motor symptoms were not associated with APOE ϵ 4 in FTD/AD.

CONCLUSION: AD can have multiple clinical presentations, each with a different anatomical pattern of peak neurodegeneration. In its most common amnesic form, APOE ϵ 4 is a major risk factor. However, it has been reported that APOE ϵ 4 is not a risk factor for the AD pathology that presents as PPA. The presentation of AD as the FTD syndrome appears to have an intermediate relationship to APOE ϵ 4. Our results strengthen the conceptualization of AD as a heterogeneous entity with distinct clinical, anatomic and molecular features.

Scientific relevance of this abstract

Apolipoprotein E ϵ 4 allele (APOE ϵ 4) is a known genetic risk factor for Alzheimer's disease (AD) neuropathology that causes amnesia (memory loss). The results of the study showed that APOE ϵ 4 is less frequent in individuals with a clinical diagnosis of frontotemporal dementia and AD neuropathology than in those with a clinical diagnosis of amnesic dementia and AD neuropathology. Thus, APOE ϵ 4 risk differs based on the clinical symptoms, a reflection of selective regional brain vulnerability, even though the underlying disease pathology is the same.

POSTER 7

NEUROPSYCHIATRIC SYMPTOMS RELATED TO NEUROPATHOLOGIC VS. CLINICAL DIAGNOSIS OF DEMENTIA

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OBJECTIVE: Amnestic dementias of the Alzheimer-type (DAT) and behavioral dementias of the frontal-type (FTD) can be associated with the neuropathology of either Alzheimer's Disease (AD) or frontotemporal lobar degeneration (FTLD). The primary aim of the present study was to compare the frequency of apathy and disinhibition in DAT and FTD caused by either AD or FTLD.

BACKGROUND: Neuropsychiatric symptoms such as apathy and disinhibition are associated with the clinical syndromes of both DAT and FTD. Clinical studies suggest apathy alone or in combination with disinhibition is the most common initial symptom in FTD and more prevalent than in DAT. The differential frequencies of these features alone or in combination in AD vs FTLD have not been determined.

METHODS: Retrospective analysis of data from 1304 cases with autopsy confirmed disease in the National Alzheimer Coordinating Center (NACC) database. The frequencies of apathy, disinhibition, or the combination of both were compared in 4 groups DAT/AD (n=1055), DAT/FTLD (n=51), FTD/AD (n=57), FTD/FTLD (n=141). Chi-square analyses were used.

RESULTS: In AD neuropathology, the most common symptom was apathy without disinhibition (33%; $p<0.001$). FTLD neuropathology was more frequently associated with the combination of apathy and disinhibition (44%; $p<0.001$).

CONCLUSION: Neuropsychiatric symptoms such as apathy, disinhibition or the combination of the two have differential associations with AD and FTLD neuropathology. In this sample of 1304 patients, AD pathology was more frequently associated with apathy alone, whether the clinical syndrome was DAT or FTD. In contrast, FTLD pathology was more frequently associated with the combination of both neuropsychiatric symptoms in its clinical manifestations of either DAT or FTD.

Scientific relevance of this abstract

This study is important because symptoms of apathy and disinhibition have different associations with Alzheimer's neuropathology and frontotemporal lobar degeneration (FTLD). There are biomarkers for Alzheimer's disease but not for FTLD. Therefore, in clinical practice, the nature of the behavioral symptoms can perhaps increase the level of diagnostic accuracy.

POSTER 8

COGNITIVE DEFICITS IN THE FIRST 24 HOURS AFTER THE ONSET OF FIRST UNPROVOKED GENERALIZED TONIC CLONIC SEIZURE

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Many people with epilepsy suffer from cognitive deficits as a consequence of their seizure episodes. Many studies have been conducted to investigate the long term effects of seizure on cognitive function; however, relatively few researches have been undertaken to assess the short term impacts of seizure on cognitive function. This study aimed to evaluate changes in cognitive performance in adults with a new onset tonic-clonic seizure in the first 24 hours after the onset of their seizure.

After having given inform consent, 40 patients with first seizure were investigated with a neuropsychological test battery of Wechsler Memory Scale III.

Their performance was compared with 40 healthy individuals from the patients' companions. Newly diagnosed patients had significantly worse performance in paired association recall which is a parameter of verbal memory.

Our data suggested that even with a one-time seizure episode, patients are susceptible to transient memory impairment.

POSTER 9

VULNERABILITY AND INTEGRITY OF VON ECONOMO NEURONS IN HUMAN ANTERIOR CINGULATE CORTEX ACROSS THE COGNITIVE LIFESPAN

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Age is the strongest risk factor for Alzheimer's Disease (AD), likely due to typical "wear-and-tear" that occurs in permanently post-mitotic neurons across the human lifespan. The Alzheimer dementia stage is preceded by a prodromal amnesic mild cognitive impairment stage (aMCI) where both AD pathology and cognitive impairment are less pronounced. What remain unknown are the intrinsic vulnerabilities of specific neuronal subpopulations that potentially contribute to neurodegeneration and cognitive decline across the lifespan.

von Economo neurons (VENs) are highly specialized spindle-shaped cells unique to the anterior cingulate cortex (ACC) and frontoinsular cortex in primate hominids, and are implicated in higher-order cognition. VENs were found to be reduced in certain neurodegenerative diseases such as behavioral variant Frontotemporal Dementia, whereas cognitive "SuperAgers" (age 80+ individuals with memory scores equal-to-or-above individuals ~25 years younger) have an abundance of VENs in ACC. The current study sought to determine whether VENs are preferentially reduced in individuals with amnesic dementia due to AD versus other healthy cognitive aging outcomes.

Postmortem brain specimens stained with Nissl were analyzed using modified stereological methods (Stereoinvestigator, MBF) from the following cohorts (N=5, per group): SuperAgers, cognitively-normal elderly (age 65+), cognitively-normal young (age 20-60), elderly individuals diagnosed with aMCI (age 65+) and elderly individuals diagnosed with amnesic dementia (age 65+) and with severe postmortem AD pathology. VEN density and total neuronal counts were obtained in ACC based on cellular morphology.

Stereological results revealed lowest VEN counts in the AD group, followed by higher counts in aMCI, older, and younger groups, respectively. VEN counts and total neuronal counts in the AD group were significantly lower compared to old and young groups ($p < 0.01$), but not the aMCI group. Consistent with prior findings, SuperAgers continued to show the highest VEN counts compared to all other groups ($p < 0.05$). Total neuronal counts in ACC were highest in young controls.

Similar to other neurons in human ACC, VENs are indeed vulnerable to loss in AD, but appear to be preserved in individuals with well-preserved cognition.

POSTER 10

ACTIVATED MICROGLIA IN CORTICAL WHITE MATTER OF COGNITIVELY INTACT ELDERLY

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Objectives: Microglial activation is a major indicator of neuroinflammation in a number of age-related neurodegenerative disorders, such as Alzheimer disease. We recently found that activated microglia are extremely abundant throughout white matter—at levels that surpass grey matter densities—in brains of individuals who show post-mortem Alzheimer's disease or TDP-43 pathology.

Methods: In this study, we evaluated microglial densities in brains of cognitively normal elderly (over age 65, N=4) and cognitive "SuperAgers", individuals over age 80 who show outstanding memory capacity (N=3). One cognitively normal young case (45 years) was included for preliminary age-related comparisons. Specimens were cut into whole-hemisphere sections and immunohistochemically stained to visualize HLA-DR-positive activated microglia. Five cortical regions (inferior frontal, anterior cingulate, inferior parietal, superior temporal, and entorhinal) were examined for microglial activation in both grey and white matter. Densities were ranked based on a 0-5 scale, with 0 denoting absence of activated microglia.

Results: Preliminary rankings revealed higher microglial activation in white matter compared to adjacent grey matter in all cases. Greatest white matter microglial activation was observed in entorhinal cortex compared to other regions. Examination of the one young case showed substantially less white matter microglia than elderly post-mortem cases.

Conclusions: Patterns of white matter microglia were prominent in brains of cognitively intact elderly, at levels that exceeded grey matter densities. Future directions will investigate age-related white matter microglial differences in larger groups of both young and old specimens, and will examine the contribution of white matter microglia to cognitive performance.

POSTER 11

CONCORDANCE BETWEEN CORTICAL ATROPHY AND DISTRIBUTION OF MICROGLIA IN PRIMARY PROGRESSIVE APHASIA WITH TDP-43 INCLUSIONS

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Primary progressive aphasia (PPA) is a neurodegenerative clinical dementia syndrome characterized by a gradual dissolution of language. The asymmetric nature of the language network and focal atrophy render PPA an excellent model for investigation of the relationships between the regional distribution of pathologic markers, cortical atrophy, and clinical phenotype. At autopsy, approximately 30% of PPA patients have TAR-DNA binding protein-43 (TDP-43) pathology (PPA-TDP). We previously reported concordance between microglia activation and cortical atrophy in PPA-TDP participants with progranulin mutations. The current study investigated whether a similar concordance exists in PPA-TDP participants without progranulin mutations. Whole hemisphere sections from five PPA-TDP cases were immunohistochemically stained for HLA-DR, a marker of activated microglia. Using unbiased stereology, densities of activated microglia were quantified in the following regions: inferior frontal gyrus (IFG), middle frontal gyrus (MFG), inferior parietal lobule (IPL), superior temporal gyrus (STG), middle or inferior temporal gyrus (MTG or ITG), entorhinal cortex (ERC), and hippocampus (HIP). Cortical atrophy was assessed using one of three methods: 1) structural MRI scans collected close to death analyzed using the FreeSurfer software, 2) clinical MRI scans, or 3) raw images of the postmortem brain. Cases 1, 2 and 3, all right-handed males, displayed substantial asymmetry of microglia distribution, greater in the left hemisphere in all cortical areas. This showed concordance with atrophy patterns visualized by quantitative MRI. Case 4, a left-handed male, had reversed asymmetry of atrophy and right hemisphere language dominance; clinical scans exhibited severe atrophy of the right temporal lobe. Microglia density displayed substantial asymmetry in right hemisphere cortical areas, with greatest asymmetry in STG and ITG, concordant with patterns of atrophy on MRI. Microglial counts in Case 5, a right-handed male participant, were fairly symmetric across all cortical regions, aligning with the absence of visible left-sided asymmetric atrophy in most cortical areas. These findings demonstrate significant microglial activation in PPA-TDP that match patterns of atrophy. It remains to be determined whether microglial activation contributes to cortical damage in PPA-TDP.

POSTER 12

TDP-43 PRE-INCLUSIONS IN PRIMARY PROGRESSIVE APHASIA: MORPHOLOGY AND DISTRIBUTION

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A hallmark of neurodegenerative disorders is aggregation of misfolded proteins in inclusions, such as accumulation of abnormally phosphorylated TAR-DNA binding protein-43 (TDP-43) in frontotemporal lobar degeneration (FTLD). In recent years, TDP-43-positive pre-inclusions (diffuse cytoplasmic staining) were described, suggesting that TDP-43 inclusions develop in stages. Here we investigated morphologic subtypes and distribution of TDP-43 pre-inclusions in primary progressive aphasia (PPA), a clinical language dementia syndrome in which a subpopulation is characterized by TDP-43 inclusions (PPA-TDP). Frozen and paraffin embedded sections were immunohistochemically stained for phosphorylated TDP-43, and subtypes and densities of pre-inclusions were examined in brains from one cognitively normal and five PPA-TDP participants. Seven distinct morphologic subtypes of pre-inclusions were identified in the PPA brains: 1) smooth, non-granular, non-fibrillar immunoreactivity in cytoplasm and proximal dendrites; 2) diffuse staining throughout the cell; 3) smooth cytoplasmic staining + granules; 4) granular staining in cytoplasm; 5) granular staining throughout the cell; 6) small fibrils in cytoplasm; and 7) fibrils throughout the cell. The normal brain was devoid of inclusions and pre-inclusions. Cortical areas with a high density of mature TDP inclusions contained lower densities of pre-inclusions than areas with low densities of inclusions. Consistent with the clinical PPA phenotype, the language dominant hemisphere showed higher mature inclusion densities. Pre-inclusions were considerably more numerous in fixed frozen sections than in paraffin embedded sections from the same regions. These observations suggest that TDP-43 inclusions originate in neurons with pre-inclusions, which may develop through progressive stages. These findings also suggest that fixed frozen sections are advantageous to paraffin embedded sections in studying pre-inclusions. It remains to be seen whether subtypes and distribution of TDP-43 pre-inclusions are common to all TDP-43 proteinopathies.

POSTER 13

ROD-SHAPED MICROGLIA SHOW DISEASE AND REGIONAL HIPPOCAMPAL SPECIFICITY

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Microglial dysfunction is strongly tied to the pathogenesis of neurodegenerative diseases. Remarkable heterogeneity in microglial morphology has been described. The morphologic subtypes include ramified, hypertrophic, dystrophic and rod-shaped microglia. Rod-shaped microglia were first described by Nissl more than 100 years ago; however, little is known about the specific functions of the rod-shaped microglia in neurodegeneration. In this study, we determined if rod-shaped microglia are involved in the process of neurodegeneration, by evaluating the distribution of rod-shaped microglia in the hippocampi of brains with the neuropathology of frontotemporal lobar degeneration with TDP-43 proteinopathy (FTLD-TDP), Alzheimer disease (AD), or no abnormality. We conducted immunohistochemistry with total microglia marker, IBA-1 antibody, followed by semi-quantitation of the density of rod-shaped microglia in the hippocampal sub-regions. This was based on the idea that if rod-shaped microglia contribute to the pathogenesis of neurodegeneration, they would likely exhibit different distribution patterns in the different regions of vulnerability of the hippocampus associated with these different pathologic states. We found that rod-shaped microglia were mainly located in the hippocampal CA1 region, and that the density of rod-shaped microglia in CA1 was higher than the densities in the CA2-4 regions and dentate gyrus, in all groups. Interestingly, there was an increased number of rod-shaped microglia in CA1 in FTLD-TDP as compared to normal brains and those with AD. The densities of rod-shaped microglia were not related to the severity of hippocampal sclerosis in either AD or FTLD-TDP. In addition, the densities of rod-shaped microglia were not related to mutations in GRN or C9ORF72. These data suggest that the rod-shaped microglia have a disease specificity in FTLD and a location specificity in the CA1 sector of the hippocampus.

POSTER 14

GRN MUTATIONS DECREASE THE PROPORTION OF PGRN-POSITIVE CA1 MICROGLIA

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Heterozygous loss-of-function mutations in the progranulin gene (GRN) cause FTLD-TDP, a common form of autosomal dominant dementia. Progranulin (PGRN) is a highly conserved, secreted glycoprotein, and may function in the central nervous system as a growth factor and key modulator of microglial function. Studies have suggested that altered microglial function caused by PGRN deficiency is tied to the pathogenesis of FTLD-TDP. In this study, we semiquantitatively evaluated the distribution of PGRN-immunopositive microglial cells in the CA1 region of the hippocampus in FTLD-TDP with GRN mutations, normal controls, Alzheimer disease without hippocampal sclerosis (HS), Alzheimer's disease with HS (where the hippocampus contains abnormal TDP deposits), and FTLD-TDP without GRN mutations. In normal controls and AD without HS, the PGRN-positive microglia comprised approximately 50% of the total microglial population. In two settings with abnormal hippocampal TDP, AD with HS and FTLD without GRN mutation, this proportion was increased to 80%, at the same time that the total number of microglia was increased, indicating an overall microglial activation in CA1. In FTLD-TDP with GRN mutation, the CA1 region also showed increased total microglial number but the number of PGRN-positive microglia was only 30% of the total population. We conclude that translocated and truncated TDP deposition in the hippocampus is associated with microglial proliferation and that the haploinsufficiency of the GRN mutations also extends to PGRN expression in microglia.

POSTER 15

TAU PET, AMYLOID PET, AND STRUCTURAL IMAGING IN PRIMARY PROGRESSIVE APHASIA

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Primary progressive aphasia (PPA) is a clinical dementia syndrome caused by neurodegenerative disease and characterized by asymmetric atrophy of the language-dominant (usually left) hemisphere. The most common neuropathologies reported for PPA are Alzheimer's disease (AD; ~40%), frontotemporal lobar degeneration with tauopathy (~30%), or with TDP-43 proteinopathy (~30%). Recent innovations in PET technology have allowed quantitation and spatial localization of amyloid and tau. These technologies are particularly promising in diseases such as PPA where there is no one-to-one relationship between clinical symptoms and underlying pathology. We used the tau ligand [¹⁸F] AV-1451 to scan three PPA participants whom also underwent [¹⁸F] AV-45 amyloid PET and structural MRI (sMRI). Two participants were diagnosed with the agrammatic subtype of PPA (PPA-G) and the other was diagnosed with the logopenic subtype (PPA-L). The sMRI was processed with FreeSurfer to derive measures of cortical atrophy. Amyloid was quantified using a previously described FreeSurfer method (Landau et al. J Nucl Med 2013). Two participants (1 PPA-L and 1 PPA-G) showed elevated amyloid binding ($A\beta+$; using the 1.11 whole cerebellar SUVR threshold), consistent with AD pathology. Both of these $A\beta+$ patients had elevated tau PET uptake patterns that mirrored the asymmetric left hemisphere atrophy, presumably reflecting the distribution of the neurofibrillary degeneration underlying the cortical atrophy. The amyloid-negative PPA-G patient had asymmetric left cortical atrophy and low tau PET binding. Longitudinal studies of neuropsychological performance, MR imaging, amyloid PET, and tau PET are needed in PPA to determine the temporal relationship among these measures and the usefulness of tau PET as biomarker for tracking the progression of disease.

POSTER 16

HIGH DENSITIES OF ACTIVATED MICROGLIA ARE PRESENT IN CORTICAL WHITE MATTER AND CORRESPOND TO REGIONS OF GREATEST ATROPHY IN PRIMARY PROGRESSIVE APHASIA

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Background: While deposition of abnormal proteins and other pathology in cortical gray matter in neurodegenerative disorders has received extensive experimental attention, little is known about the extent and nature of white matter abnormalities. Primary progressive aphasia (PPA) is a clinical dementia syndrome characterized by dissolution of language function and is associated with Alzheimer disease (AD) or frontotemporal lobar degeneration pathology. We have shown extensive activation of microglia in gray matter in PPA brains regardless of the underlying molecular pathology. Here we investigated activation of microglia in cortical white matter in PPA brains with AD or TDP-43 pathology, and its relationship with cortical atrophy.

Methods: Brains of PPA-AD (n=2) and PPA-TDP (n=3) participants were cut into whole hemisphere sections, and a 1/24 series of sections were processed with immunohistochemical or histopathological procedures to visualize plaques, tangles, TDP-43 inclusions, and HLA-DR-positive activated microglia. Atrophy was quantified using FreeSurfer software in two participants with structural MRI scans collected close to death, and assessed in three participants using clinical MRI scans or photographs of the postmortem brain.

Results: All cases displayed pronounced asymmetric atrophy restricted to the perisylvian language network. Whole hemispheric sections displayed substantial asymmetric densities of activated microglia throughout the white matter that surpassed the densities in adjacent gray matter, and allowed demarcation of the white/gray matter junction with the naked eye. The highest densities of activated microglia in white matter occurred asymmetrically in cortical areas affiliated with language function, and closely matched patterns of gray matter atrophy detected by MRI scans in each case.

Conclusions: Microglia display a pattern of activation in PPA characterized by substantial accumulation in cortical white matter with highest densities at sites of greatest atrophy. While the extent of activation of microglia in white matter in other neurodegenerative disorders is incompletely understood, our findings point to the possibility that activated microglia play an active role in neurodegenerative mechanisms in the white matter, and correspond to in vivo cortical gray matter atrophy.

POSTER 17

HYPERTROPHIC MICROGLIA ARE ASSOCIATED WITH MORE TANGLES AND LESS NEURONS IN PRIMARY PROGRESSIVE APHASIA WITH ALZHEIMER PATHOLOGY

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Alzheimer disease (AD) pathology (i.e., amyloid- β plaques [APs] and neurofibrillary tangles [NFTs]) can cause primary progressive aphasia (PPA), a clinical dementia syndrome characterized primarily by language deficits and asymmetric cortical atrophy concentrated in the left perisylvian language network. Activated microglia are associated with AD pathology, and the "hypertrophic" microglia (HM) subtype is more closely linked to pathologic insults than "ramified" microglia (RM). However, the presence of microglial subtypes and their relationships with APs, NFTs, and neurons have not been described in PPA patients with AD pathology (PPA-AD). The first goal of the current study was to determine the relative distribution of HM versus RM between hemispheres and between language versus non-language cortical regions. The second goal was to identify the relationships between HM and densities of APs, NFTs, and neurons. Unbiased stereology was performed on whole-hemisphere sections to quantify postmortem markers in five language regions of interest (ROIs) and two non-language ROIs, bilaterally. Wilcoxon signed rank tests revealed HM densities exceeded RM densities across all ROIs in both cases ($p < 0.0001$). In addition, HM were greater in left versus right ROIs ($p = 0.025$), and the left versus right difference in HM density was greater than the left versus right difference in RM density across all ROIs ($p = 0.017$). Spearman correlations combining all marker densities ($n = 28$ ROI densities per marker across the two participants) revealed a significant positive correlation between HM and NFT ($r = 0.41$, $p = 0.029$), and significant negative correlations between HM and AP ($r = -0.53$, $p = 0.004$) and neurons ($r = -0.44$, $p = 0.018$). Taken together, these preliminary findings suggest that HM may be a more sensitive correlate of NFT than AP accumulations, and an increase in HM and/or NFTs may contribute to the smaller neuronal densities potentially underlying cortical atrophy and cognitive decline in PPA-AD.

POSTER 18

RUN4PAPA: RUNNING MARATHONS AROUND THE WORLD TO FUND INNOVATIVE DEMENTIA RESEARCH

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Introduction:

In 2011, Jason Boschan contacted the Northwestern Medicine Cognitive Neurology and Alzheimer's Disease Center (CNADC) in hopes of partnering to raise \$10,000 in research funding for a rare dementia called Primary Progressive Aphasia (PPA). Jason's grandfather, Dr. Louis Heyman, was diagnosed with PPA in 2010, instilling in Jason a strong sense of urgency to act in honor of his grandfather. Dr. Heyman, a lifelong pediatrician, had devoted his life to helping children and imparted an attitude of service in Jason. Jason launched Run4Papa in 2011 with the thought of running marathons throughout the world to honor his grandfather and raise funds for dementia research.

Methods:

Run4Papa and the CNADC have used a multi-pronged approach to support this campaign. First, an inspiring narrative was created taking Jason to all 7 continents to complete marathons. Next, novel research projects were targeted for funding during different phases of the campaign. Each research project was matched up with a major marathon to promote interest. Multiple platforms were used to raise awareness including crowdfunding, community fundraisers, community partnerships, and social media. Run4Papa also piloted the first crowdfunding campaign in the history of the Feinberg School of Medicine on the Northwestern Catalyzer platform.

Results:

The Run4Papa campaign has raised over \$200,000 since its inception, engaging over 1,100 new donors to Northwestern. Jason has logged over 4,344 miles running races throughout the world including:

- 2012 Great Wall of China Marathon
- 2013 and 2014 Boston Marathon
- 2014 Big 5 Marathon in South Africa
- 2014 Rio de Janeiro Marathon
- 2015 Australian Outback Marathon
- 2015 Chicago Marathon
- 2016 Antarctica Marathon
- 2016 Athens Marathon

Run4Papa has helped provide funding for the following projects:

- Communication Bridge: Web based speech therapy for patients with aphasiac dementia
- Tau neuroimaging
- Capital project to build a new cutting-edge Alzheimer's disease research center at Northwestern

- Advanced scientific instruments to support research
- Funds to assist patient's travel to Northwestern for the latest treatments

Conclusions:

The model executed by Run4Papa and the CNADC has been extremely successful for this partnership. The CNADC has launched an innovative intervention in Communication Bridge, brought experimental cutting edge neuroimaging to Northwestern, contributed to a capital campaign to build a new research center, purchased new equipment to support research studies, and helped families travel to Chicago from all of the country to get the latest dementia treatments. Successful partnerships with motivated community advocates can make a tremendous impact in the fight against dementia. Stay tuned for what happens next in the Northwestern and Run4Papa partnership.

POSTER 19

STRUCTURE OF THE HUMAN CENTRAL OLFATORY SYSTEM

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There are profound interspecies variation in olfaction, with corresponding differences in the central olfactory apparatus. While various components of the central olfactory system are preserved when rodent species are compared to the primate, the layout of these components varies greatly. Most of our understanding of the anatomy of the central olfactory system in the primate is derived from the macaque brain. The purpose of this study was to investigate the detailed structure of this system in the human brain. Full hemispheric sections from three normal brains were processed to visualize cell bodies using the Cresyl violet Nissl stain. One brain was embedded in celloidin, and alternate thick sections were stained for Nissl, or for myelin using the Loyez method. Throughout most of its course, the olfactory tract (OTr) displays a triangular shape in cross section, with myelinated axons concentrated in the periphery. Once the tract reaches the most posterior aspects of the olfactory sulcus in the medial orbitofrontal cortex (OFC), the apex of the triangle elongates with two streams of axons directed dorsally towards OFC. More posteriorly, the bases of the triangle merge into a primary bundle of axons directed laterally, and a minor bundle directed medially, the latter contributing axons to medially streaming white matter. The lateral bundle further differentiates into two streams, with a minor component directed laterally towards the insula, and a major component directed ventrally towards the temporal pole, where it enters layer I of cortex. Another distinct target of the OTr is the anterior olfactory nucleus (AON), appearing first as a small collection of neurons within the posterior aspects of OTr, just before OTr enters the cortex. More posteriorly, the AON is located at the base of the olfactory sulcus as several relatively large collections of neurons surrounded by OTr axons. Just dorsal and posterior to AON lies the OFC component of piriform olfactory cortex (PCx). As in the macaque, the PCx trifurcates from a "root" near the limen insula (i.e., the junction where the orbitofrontal, temporal and insular cortices first join), contributing components to the OFC, the insula (Ins), and the temporal pole (TP). These three components occupy a large region, and the PCx protrudes in small but distinct gyri in the three cortical areas. In summary, the components of the central olfactory system described in the primate can be identified in the human brain. However, the extent of the area occupied by these components in the human is considerably expanded, underscoring the widespread anatomical diversity of afferent projections from the OTr that define the human olfactory system.

POSTER 20

ALTERED LANGUAGE NETWORK CONNECTIVITY IN PRIMARY PROGRESSIVE APHASIA

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Background: In primary progressive aphasia neurodegenerative processes (Alzheimer disease or frontotemporal lobar degeneration) cause a gradual loss of neurons. The distribution of neuronal loss and its progression have been mostly assessed by mapping focal cortical thinning (atrophy) or hypometabolism using structural magnetic resonance imaging (MRI) and positron emission tomography (PET) respectively. However, as shown in our previous study, the underlying mechanism of aphasia may be due to altered network connectivity rather than failure of specific nodes as measured by MRI and PET. The purpose of the current investigation was to look further into the anatomical organization of altered functional connectivity in patients with different forms of PPA. We also wanted to determine whether the anatomy of disrupted connectivity in these patients would add new insights into the organization of the language network.

Methods: Magnetic resonance imaging data were analyzed for cortical morphometry and resting state functional connectivity in 28 PPA patients and 33 controls. Thirteen of the patients had semantic PPA (PPA-S), where word comprehension and naming are impaired. The remaining 15 had non-semantic PPA (PPA-NS), where grammar, repetition and word retrieval are impaired. Strengths of pairwise connectivity in 5 cortical nodes of each hemisphere [pars triangularis and opercularis of the inferior frontal gyrus (IFGt and IFGo)], middle temporal gyrus (MTG), supramarginal gyrus (SM), and anterior temporal lobe (ATL)] were analyzed for group comparisons and correlation with measures of grammar, repetition, and comprehension. To avoid results that were biased by cortical atrophy, effect of cortical volume was factored out in all analyses.

Results: Both PPA groups had decreased strength of the IFGt-MTG connectivity. There were also group-specific connectivity impairments of the IFGo-SM pair in PPA-NS and the IFGt-ATL and MTG-ATL pairs in PPA-S. No significant alterations were seen in the connectivity of homologous parts of the right hemisphere. After factoring out the effects of atrophy, the IFGo-SM connection along the dorsal axis of the language network remained significantly decreased in PPA-NS and the MTG-ATL connection along the ventral axis in PPA-S. Performance on grammar and repetition correlated with strength of the IFGt-MTG and IFGo-SM connections, and performance on comprehension correlated with connectivity of the left IFGt-ATL node pair. The correlations of repetition impairment remained significant after correction for atrophy.

Conclusions: Our results show that the language impairments in PPA are mediated at least in part through perturbations of functional connectivity within concordant components of the language network. The impaired connectivity may reflect not only the loss of neurons (indexed by atrophy) but also the dysfunction of existing neurons. These findings have potential practical implications, especially since physiologic disturbances that precede neuronal death may be modulated by targeted neurostimulation and neuroprotective treatments.

POSTER 21

ASYMMETRIC INTRAHEMISPHERIC CONNECTIVITY BETWEEN LEFT DORSAL FRONTAL LOBE AND LANGUAGE NETWORK

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Background: Left hemisphere language network nodes (specifically, inferior frontal gyrus (IFG) and posterior middle temporal gyrus (MTG)) have been shown to demonstrate asymmetric (left>right) intra-hemispheric resting state functional connectivity in healthy populations. Reduction in connectivity between these nodes have been documented in non-fluent/agrammatic primary progressive aphasia. Task based fMRI studies and research in individuals with aphasia have shown that left dorsal frontal regions also take part in processing of grammar/phonology, and motor speech. However, the status of functional connectivity between these regions and perisylvian nodes has not been well investigated. In this study using resting state functional magnetic resonance imaging (rs-fMRI), we examined whether dorsal frontal nodes showed greater connectivity to perisylvian nodes in the left (versus right) hemisphere.

Methods: We analyzed resting state fMRI scans from two datasets from University of Texas at Austin (UTA), and Alzheimer Disease Neuroimaging Initiative (ADNI), which comprise of 40 and 44 right-handed healthy individuals respectively. Ten-millimeter spherical nodes included the IFG, MTG, superior frontal gyrus (SFG), and the premotor cortex (PM) in both hemispheres. RSFC between node pairs in each hemisphere were calculated and t-tests were used to compare the left and right intra-hemispheric connections. To demonstrate specificity, the functional connectivity between the frontal eye field (FEF) and intraparietal sulcus (IPS) nodes was also measured in both hemispheres.

Results: In both datasets, the IFG-MTG connectivity was stronger on the left side (UTA, $p=0.01$; ADNI, $p=0.01$). We also found additional connections that showed evidence of stronger functional connectivity on the left side: IFG-PM (UTA, $p=0.005$; ADNI, $p=0.001$), IFG-SFG (UTA, $p=0.005$; ADNI, $p=0.01$), MTG-PM (UTA, $p=0.0002$; ADNI, $p=0.01$), and MTG-SFG (UTA, $p=0.001$; ADNI, $p=0.001$). The FEF-IPS control connections showed no significant asymmetry (UTA, $p=0.79$; ADNI, $p=0.50$).

Conclusion: The concordant results from both datasets extend findings of enhanced intra-hemispheric RSFC for left hemisphere regions to include dorsal frontal regions, providing confirmatory evidence for their role in grammar/phonology, and motor speech. Future resting state fMRI studies should investigate the relation between these regions and speech/language regions of the brain in healthy and language impaired individuals. Advanced knowledge of language network connectivity will allow us to choose the right targets for neuromodulatory interventions and to better measure efficacy of therapeutic interventions.

POSTER 22

INVESTIGATING THE ROLE OF HGF ON MOTOR NEURON SURVIVAL AND FUNCTION IN ALS

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Hepatocyte growth factor (HGF) was first discovered as a hepatocyte mitogen and its role in angiogenesis, fibrosis, muscle regeneration, apoptosis and neurodegeneration has been studied. Recent evidence suggests that HGF has a supportive role for motor neuron survival. ALS is a progressive neurodegenerative motor neuron disease involving both the upper and lower motor neurons. Therefore, HGF treatment has been considered as a potential option for ALS disease. VM 202 is a plasmid DNA containing HGF-x7, which encodes two isoforms of HGF, and allows production of HGF in the introduced cells and neurons.

We initially investigated the expression patterns of HGF-receptor (c-Met) and HGF in the neuromuscular junction, spinal cord and the motor cortex of hSOD1^{G93A}-UeGFP mice, in which both the upper and the spinal motor neurons are genetically labeled with eGFP expression that is stable and long-lasting, allowing visualization and cellular analysis of different components of the motor-neuron circuitry.

We detect HGFR (HGF receptor) at the neuromuscular junction, spinal cord and the motor cortex. Interestingly, CSMN displayed selectively high levels of HGFR expression at P80 and P120. Our initial studies suggest that astrocytes are the primary source of HGF in the spinal cord, and that spinal cord has higher levels of HGF than that of the cortex. Interestingly, especially towards end-stage, HGFR levels were increased in diseased mice. These results suggest that both upper and lower motor neurons would respond to HGF treatment.

Our goal is to investigate whether constant supply of HGF would have an impact on degenerating motor neurons. Current experiments involve biweekly intramuscular injections of the VM202 plasmid into 4 different muscles in the leg of hSOD1^{G93A}-UeGFP mice, at P60, – a time of disease onset–, and investigate the correlation between motor function improvements and motor neuron survival with respect to disease progression. Upon completion, we will have a better understanding for the correlation between HGF treatment and improved motor neuron health and motor function, an important preclinical assessment in ALS therapies.

POSTER 23

ALTERATIONS IN SOLUBLE APP- β AND SOLUBLE APP- α KINETICS IN THE HUMAN CENTRAL NERVOUS SYSTEM IN ALZHEIMER'S DISEASE: A PILOT STUDY

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The amyloid hypothesis proposes that increased production or decreased clearance of amyloid-beta ($A\beta$) leads to higher order amyloid structures that initiate a cascade of events, culminating in neuronal death manifesting as Alzheimer's disease (AD). Sequential cleavage of Amyloid Precursor Protein (APP) by β - and γ -secretase generates $A\beta$. APP is processed in one of at least two pathways, initially being cleaved by either α -secretase or β -secretase (BACE1). α -secretase cleavage of APP precludes the formation of $A\beta$ and produces non-toxic soluble APP- α (sAPP α). Alternatively, APP is initially cleaved by BACE1 releasing soluble APP- β (sAPP β) and subsequently cleaved by γ -secretase, producing $A\beta$. In some studies BACE1 protein and sAPP β are increased in cerebrospinal fluid (CSF) and post-mortem AD brain. Our previous data demonstrate an increase in CSF sAPP β : sAPP α ratio in AD subjects versus age-matched controls, indicating a shift toward BACE1 processing of APP under pathophysiological conditions. Further, a high positive correlation exists between sAPP β and $A\beta$ concentrations in human CSF, and a stable isotope labeling kinetics (SILK) study suggests about 50% of AD patients may overproduce $A\beta$. Together these findings suggest increased BACE1 activity may cause increased $A\beta$ in at least a subpopulation of AD patients. However, this has not been previously directly assessed.

Using highly sensitive SILK/immunoprecipitation/liquid chromatography-mass spectrometry methods, in our pilot study we have quantified sAPP β and sAPP α kinetics in CSF from six human AD subjects and controls, in order to determine β - and α -secretase activity in human CNS. We report a higher absolute production rate of sAPP β , when controlled by sAPP α , in those subjects with detectable amyloid deposition in the brain. We also will describe fractional synthetic rates (FSRs) of both sAPP species as well as compare these to $A\beta$ FSRs, which have been previously measured. In our next step, we plan to expand this proof-of-concept study to include a total of 100 human subjects. We hypothesize that approximately half of AD patients overproduce $A\beta$ due to increased BACE1 activity as measured by increased absolute production rates of sAPP β . By directly measuring production rates of sAPP β and sAPP α in vivo, we are determining if, and by how much, BACE1 activity is increased in AD subjects. These results would allow for characterization of AD subpopulations most likely to benefit from BACE1 inhibitor treatment. Outcomes of this study will elucidate human CNS APP physiology and AD pathophysiology, and also prove useful for measuring pharmacodynamic effects of candidate therapeutics. BACE1 is currently a high priority target for AD, thus results of altered BACE1 activity in AD are critical for understanding AD pathophysiology and development of disease modifying therapeutics.

POSTER 24

ANALYSIS OF PROTEOME DEGRADATION KINETICS WITH IN VITRO AND IN VIVO MODELS OF ALZHEIMER'S DISEASE

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Alzheimer's Disease (AD) is characterized molecularly by the misfolding and aggregation of proteins, particularly amyloid beta peptides ($A\beta$). However, how accumulating $A\beta$ confers toxicity to cells remains poorly understood. One hypothesis is that amyloid beta impairs or siphons off protein degradation machinery causing certain proteins to stick around the cell longer, thus disrupting protein homeostasis. My project aims to identify proteins with decreased degradation dynamics, as these proteins may represent linchpins that drive disease dysfunction.

To investigate protein degradation dynamics, I utilize metabolic labeling via Stable Isotope Labeling in Cell Culture or in Mammals (SILAC and SILAM), followed by quantitative mass spectrometry (MS). In SILAC, primary neurons are labeled with "heavy" isotopes of arginine and lysine, and in SILAM, mice are labeled with "heavy" nitrogen (N^{15}). Following chase periods with natural ("light") amino acids and nitrogen, proteins that have not been degraded remain labeled with the heavy isotopes, while newly synthesized proteins incorporate the "light" isotopes. MS separates the light and heavy peptide signals, allowing measurement of degradation dynamics.

In the SILAC experiments, I have established efficient labeling of primary rat neuronal cultures. After 15 days, nearly 65% of identified peptides were over 90% heavy labeled. Furthermore, I have established efficient SILAC chase. After growing in heavy media for over 12 days, I changed the media to natural media, and grew the cells for several additional days. As expected, the number of peptides identified as fully light increased with days spent in light media and the number of peptides identified as fully heavy decreased accordingly. Adding synthetic $A\beta$ peptides in either monomer, oligomer, or fibril form shifts protein degradation rates causing more proteins to be identified as a greater percentage of heavy labeled. Further analysis is ongoing to investigate pathways and cellular systems that are particularly vulnerable to $A\beta$ exposure. In the SILAM experiments, I am labeling a recently developed APP knock-in mouse model for AD with N^{15} , which will allow me to investigate how accumulating $A\beta$ affects protein degradation in a physiological system related to human pathology.

Proteomic investigations into protein degradation dynamics in response to $A\beta$ accumulation has never been investigated, especially in APP knock-in mice. These experiments may reveal novel protein pathways that are critical to Alzheimer's pathology, and thus may serve as a new target to help prevent or delay AD progression.

POSTER 25

SUPERAGING STUDY: CORRELATES OF ACTIVE ENGAGEMENT IN LIFE IN THE ELDERLY

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Many individuals have come to expect that as they grow older, their memory and other cognitive abilities will begin to deteriorate. Though such decline is common, the SuperAging study at the Northwestern University Cognitive Neurology and Alzheimer's Disease Center (CNADC) has found that some individuals are able to maintain high levels of cognitive function as they age. The Northwestern SuperAging Project, has identified a group of individuals over the age of 80 with exceptional episodic memory ability that is at least as good as that of individuals 20-30 years their junior. The study seeks to identify factors that help an individual avoid age-related cognitive decline and memory loss.

To qualify as a SuperAger, individuals must perform at or above average normative values for individuals in their 50s and 60s on tests of episodic memory and at least within the average range for their age and education on non-memory cognitive domains according to published normative values.

Participants visit our center every 2 years for a comprehensive cognitive evaluation, structural and functional MRI scans, and blood collection for genetic testing. SuperAgers also complete questionnaires investigating personality, family history, and daily health habits. Some participants have undergone a guided "Life Story" interview detailing their life experiences, which allows researchers to evaluate the correlation between psychological well-being and superior cognitive aging. All participants are invited to take part in a brain donation program, providing researchers the opportunity to further investigate the biological mechanisms behind SuperAging.

Previous neuroimaging results have shown that SuperAgers have thicker brains compared to their cognitively average, same-age peers and do not demonstrate cortical atrophy compared to middle-age adults. Further, SuperAgers have thicker cortex in a region of the anterior cingulate compared to even the healthy individuals in their 50s and 60s year olds. Over a period of 18-months, the rate of cortical brain atrophy was approximately two times faster in Elderly Controls compared to SuperAgers. Over this same 18-month interval, SuperAgers tend to maintain outstanding cognitive performance on tests of episodic memory.

Since its inception, the SuperAging Project has used a multidisciplinary approach to study successful cognitive aging. The study has wide ranging implications and may ultimately provide clues on how to slow or avoid age-related cognitive decline. Moving forward, the study will continue to use cognitive, structural, genetic, and histopathologic markers to identify factors that promote resistance to age-related changes in the brain and allow individuals to maintain high memory capacity in old age.

POSTER 26

IMPLICATIONS FOR LRRK2 AND AUXILIN IN PARKINSON'S DISEASE PATHOGENESIS

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Parkinson's disease (PD) is the second most common neurodegenerative disorder, characterized by the dramatic loss of dopaminergic (DA) neurons in the substantia nigra pars compacta. The majority of patient cases described arise sporadically. However, several monogenic forms of the disease have been identified within the past two decades. Through functional studies, genetic implications for PD pathogenesis have been linked to lysosomal, mitochondrial, and more recently synaptic dysfunction. Mutations in clathrin-mediated synaptic vesicle recycling genes leading to early-onset Parkinsonism, such as the recently identified synaptic PD gene DNAJC6 (auxilin), are rare. However, there is growing interest to study the regulation of synaptic function by more common PD genes such as LRRK2, the most commonly mutated gene in PD. In this study, we investigate the cellular consequences resulting from LRRK2 regulation of auxilin in clathrin-mediated synaptic vesicle recycling using PD patient-derived human DA induced pluripotent stem cells (iPSCs). Our results show that LRRK2 is able to interact with and phosphorylate auxilin at novel sites. As mutations in LRRK2 have been shown to increase its kinase activity, our data further suggests that misregulated phosphorylation of auxilin by LRRK2 results in deficient synaptic vesicle recycling. Taken together, our results propose a new role for LRRK2 at the synapse through modulation of auxilin and a potential mechanism by which dopaminergic neurodegeneration is mediated by synaptic dysfunction. The work from this project expands the range of proteins for therapeutic intervention to those located at the synapse, potentially leading to the development of targeted treatments for patients with Parkinson's' disease.

POSTER 27

THE ROLE OF THE HIPPOCAMPUS IN RECOLLECTION PRECISION: EVIDENCE FROM HEALTHY AGING AND UNILATERAL MESIAL TEMPORAL LOBE RESECTION

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Normal aging is associated with episodic recollection memory decline, a cognitive function that depends critically on the hippocampus. Recollection is often measured with source memory tests in which, for example, participants are asked to recall the background context in which an object was studied. However, even when recollection is successful (e.g., “I parked my car at the museum”), the quality of information and details recollected vary (e.g., “I parked my car at the museum in the left parking garage on the fourth floor”). Most studies explore how often memory success occurs but do not consider the varying amounts of detail (memory precision) successfully recalled. The underlying neural processes and the role of the hippocampus in recollection precision relative to success are unclear. Because diminished hippocampal integrity has been associated with recollection impairments, we assessed recollection precision in healthy aging and following unilateral mesial temporal lobe resection. 18 young adults (age range: 18-35), 18 older adults (age range: 65-80) and 9 adults (age-range: 31-51) with unilateral mesial temporal lobe resection (including hippocampus) participated in an associative object-location memory study, designed to segregate memory success from memory precision. Objects were randomly assigned and studied at distinct locations on a specific context. After a brief delay, participants were cued with an object and had to recall its associated location on a new context. Distance error was modeled for each group, resulting in a group-specific recollection success and precision parameters. Success was defined as proportion of trials successfully recalled within the same study quadrant, while precision was defined as the mean distance error of those successful trials. Recollection success was matched among groups ($p > 0.1$). However, relative to young adults recollection precision was significantly impaired for older adults ($p < 0.01$) and resection patients ($p < 0.001$). Reductions in recollection precision for older adults and hippocampal resection patients provide evidence that the hippocampus necessarily contributes to memory precision. Findings suggest recollection precision is a sensitive measure of hippocampal function and can capture subtle impairments evident in aging.

POSTER 28

GLUTAMATERGIC INPUT FROM THE SUBTHALAMIC NUCLEUS (STN) TO EXTERNAL GLOBUS PALLIDUS (GPe) NEURON CLASSES

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Basal ganglia are a highly interconnected group of subcortical nuclei that are involved in motor control. The external globus pallidus (GPe) and the subthalamic nucleus (STN) form a bidirectionally connected loop that is implicated in basal ganglia dysfunction, such as Parkinson's disease (PD). One of the hallmarks of PD is the synchronized bursting of GPe and STN neurons and this pathology is correlated with the hypokinetic symptoms of the disease. The GPe is recently recognized to have a heterogeneous cellular makeup. PV-expressing GPe neurons and Npas1-expression GPe neurons have distinct firing properties, projections targets, and responsiveness to chronic dopamine depletion. Whether inputs to these two principal GPe neuron classes are different and whether the inputs to the GPe are altered in PD is not known.

We found that STN preferentially targets PV+ GPe neurons over Npas1 GPe neurons. Furthermore input from the STN to the PV+ GPe neurons is selectively reduced in a chronic Dopamine-depletion model of PD, indicating that this synapse could be specifically involved in the pathological synchrony observed in PD.

In summary, we have investigated the anatomical, physiological, and functional properties of the STN-GPe subcircuits to understand their roles in movement outcomes in health and Dopamine-depleted conditions.

POSTER 29

THREE-DIMENSIONAL VISUALIZATION OF CELLULAR AND DISEASE MARKERS IN HUMAN BRAIN USING TISSUE CLEARING METHODS

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Recent advances in methods for optical tissue clearing have enabled 3D visualization of long-range neuronal circuitry and detailed cellular structures in a diverse range mammalian tissues. These procedures remove and/or homogenize lipid bilayers while preserving the structure of proteins, ultimately producing a transparent sample that can be fluorescently-labeled, and imaged. To date, few studies have specifically investigated the applications of these methods to specimens of human nervous tissue. The Cognitive Neurology and Alzheimer's Disease Center (CNADC) at Northwestern University has worked to optimize clearing protocols for application to postmortem human tissue with the aim of conducting large scale 3D analysis of anatomic and pathologic features of neurodegenerative diseases. We have successfully applied the "CLARITY" and "iDISCO/+" protocols, and our trials have enabled us to fine-tune these procedures for relatively large human sections. We have validated labeling for neuronal, glial, and pathologic markers, including amyloid plaques, neurofibrillary tangles and TDP-43 inclusions. While both methods produced "cleared" cortical and cerebellar tissue, the solvent based iDISCO+ protocol has proved easy, reliable, cheap, and fast when compared with the relatively complicated and variable CLARITY protocol. We describe prototyped pipelines for study which generate high-resolution, high signal-to-noise 3-dimensional models (z-stacks) that can exceed 2mm, all while remaining comparable to classic IHC studies in terms of resources used. In sum, we present here methodologies which enable us to observe fine- scale anatomic distributions of pathology and directly inform systems-level understanding of intact and impaired cognitive functions.

POSTER 30

PRIMARY PROGRESSIVE APHASIA RESEARCH PROGRAM AT NORTHWESTERN UNIVERSITY'S CNADC

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Primary progressive aphasia (PPA) is a neurodegenerative dementia syndrome characterized by a progressive loss of language function. PPA has a low prevalence in clinical practice compared to Alzheimer's dementia. Currently, there is no cure for PPA. The Cognitive Neurology and Alzheimer's Disease Center (CNADC) seeks to advance PPA research through a collaborative program aimed at studying, educating, and improving treatment for patients with PPA.

Over the past decade, more than 175 participants from 35 states, Canada, and Singapore have enrolled in PPA studies at the CNADC. Participants visit Chicago to complete assessments that precisely measure their language, memory, and thinking abilities. Participants also undergo multiple brain imaging examinations with MRI and PET scanners in the state-of-the-art Center for Translational Imaging. Researchers combine cognitive testing with these advanced neuroimaging techniques to better understand the underlying mechanisms of language decline in the brain. Some participants choose to take part in the CNADC's speech therapy and educational research programs. These life-enrichment interventions use innovative technology to improve access to care. In addition to several multi-day visits to our center, most CNADC research participants agree to take part in our brain donation program. These studies allow us to improve the diagnosis, prognosis, and quality of life for individuals living with PPA, as well as understand the biological basis of language in the brain.

Funding from the National Institute of Health, Illinois Department of Public Health, Run4Papa campaign, and generous personal donations, have provided the opportunity for the CNADC to research novel diagnostic and therapeutic initiatives in PPA. In order to leverage these resources, the CNADC has maintained a centralized website to facilitate international collaboration in PPA research. Currently, there are over 245 registered researchers representing 156 institutions across 32 different countries. By engaging in these partnerships, as well as employing a multidisciplinary approach for both patients and their families, the Northwestern CNADC remains one of the top referral centers in the world for PPA.

POSTER 31

THE OLDEST-OLD WITH PRESERVED COGNITION AND THE FULL RANGE OF ALZHEIMER PATHOLOGY

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Recent reports indicate presence of amyloid plaques (AP) and neurofibrillary tangles (NFT) in brains of cognitively normal elderly in sufficient density and distribution to satisfy criteria for pathological diagnosis of Alzheimer's disease (AD). The purpose of this study was to investigate whether full AD pathology is present in the University of California Irvine 90+ study participants with preserved cognitive performance. Eight 90+ participants (95-100 years) were selected based on higher than average performance on tests of memory and preserved performance in all other cognitive domains. The Thioflavin-S stain, and antibodies to phosphorylated tau (AT8) and amyloid- β (6E10) were used to assess the presence, density and morphology of AP and NFT. The Cresyl violet Nissl stain and antibodies to non-phosphorylated neurofilaments (SMI-32) and microtubule associated protein 2 (MAP-2) were employed to assess neuronal density. The hippocampus, parahippocampal gyrus, prefrontal cortex (Brodmann area 9) and inferior parietal cortex (area 39-40) were examined. Despite similarly preserved cognitive abilities, the eight cases displayed divergent patterns of AD pathology. One brain displayed very sparse NFT / Pre-NFT (Braak Stage [BS] I) and diffuse AP. Another showed slightly higher densities of NFT / Pre-NFT (BS II) and a moderate density of diffuse AP. Three cases presented with greater density of NFT / Pre-NFT (BS III) and variable AP density. The remaining three cases were characterized by progressively increased densities and widespread distributions of NFT / Pre-NFT, cored / neuritic AP and neuropil threads (BS IV, V and VI, respectively), with two satisfying pathological diagnosis of AD. Apparent density of neurons in hippocampus and neocortex did not distinguish brains with divergent AD pathology. However, clinically confirmed AD brains with comparable density and distribution of AP and NFT to 90+ brains with the most severe pathology displayed clear neuronal loss in the hippocampus and neocortex. These results indicate presence of pathologically confirmed AD in the absence of cognitive abnormalities. It is likely that as yet unidentified factors mitigate the deleterious effects of AD pathology on neurons. Additionally, in some individuals, the brain is protected from the processes that lead to AP / NFT formation.

POSTER 32

NEUROTOXICITY RELATED TO TETRACYCLINE TRANSACTIVATOR EXPRESSION IN TDP-43 MOUSE MODEL OF FRONTOTEMPORAL LOBAR DEGENERATION

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Frontotemporal lobar degeneration (FTLD) constitutes the third most prevalent dementia after those caused by Alzheimer's disease and Lewy bodies, and is among the most prevalent dementias of early-onset. We report on a mouse model for FTLD that overexpresses wild-type TAR DNA-binding protein 43 (TDP-43) gene using tetracycline transactivator (tTA), a system widely used to create transgenic models of neurological disorders. Transgenic expression of tTA is used to activate a second transgene of interest, e.g. the wild-type TDP-43, which is placed downstream of the tetracycline response element (TRE). Exposure to doxycycline, a more stable analog of tetracycline in mouse diet, can cause a conformational change in tTA that inhibits binding to TRE to stop the expression of the TRE-controlled transgene. This allows us to control when the transgene of interest turns on and off. A recent study found that the genetic strain background on which tTA is expressed dramatically influences neurodegeneration caused by the tTA protein. Granule neurons of the dentate gyrus (DG) appeared most sensitive in this process. Our TDP-43 transgenic mice are bred on FVB and 129/SVE backgrounds, which are among the few mouse strains susceptible to neurodegeneration from tTA. Here, we report on the effects of tTA alone in our 3 month- and 7 month old tTA and TDP-43 biallelic transgenic mice. Approximately 40% of the area in DG was lost when tTA protein was expressed by 7 months of age. This effect was smaller at the earlier age. Thin coronal sections showed that the worst loss of granule cells in DG was in the mid-hippocampal region. Interestingly, the CA fields were spared from neurodegeneration. Other unaffected regions included cingulate, motor and somatosensory cortices. The only region affected outside of the hippocampus was the piriform cortex, with 25% reduction in its area. Neuronal loss by tTA is likely caused by off-target interactions involving extraneous proteins or promoters. Similar to an earlier report, we found that doxycycline treatment prevented tTA neurotoxicity. Behavioral tests in our bigenic mice expressing tTA and TDP-43 revealed no hippocampally mediated memory impairment. However, they did display deficits of working memory, novel object recognition and social interaction that are likely related to TDP-43 transgene expression. Although this study adds a note of caution for using the tTA system to generate transgenic mice, our mice remain useful for providing critical insight into the presence of TDP-43 pathology co-occurring with neuron loss and executive function deficits in FTLD.

POSTER 33

NEUROIMAGING AT THE NORTHWESTERN ALZHEIMER'S DISEASE CENTER

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The Neuroimaging Core at the Cognitive Neurology and Alzheimer's Disease Center (CNADC) was created to enhance research activities on aging and dementia within and outside of Northwestern University. Neuroimaging is focused on the spectrum of extraordinary cognitive aging to dementia, including the FTLD-spectrum of disorders. The Neuroimaging Core contains data from scans that provide optimal quantitative information on brain structure (MPRAGE), white matter properties (FLAIR), axonal pathways (DTI), resting state hemodynamic fluctuations for establishing functional connectivity (rsfMRI), and amyloid (Amyvid – PET) or tau (¹⁸F-AV-1451 – PET) binding. Neuroimaging data are available to enrich projects of our collaborators. This poster will highlight the neuroimaging data available and some of the recent findings from studies using neuroimaging data from Clinical Core participants in our Center.

POSTER 34

PROGRANULIN AND CATHEPSIN D- A POTENTIAL MECHANISM FOR NEURODEGENERATION

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Frontotemporal Dementia (FTD) encompasses a group of neurodegenerative disorders characterized by cognitive and behavioral impairments as a result of progressive degeneration of the frontal and temporal lobes. Heterozygous mutations in the gene encoding progranulin (PGRN) account for up to 25 percent of familial FTD and result in decreased PGRN expression. PGRN is normally expressed in neurons and microglia but the function of PGRN and the mechanism by which its decreased expression leads to disease is still unknown. While PGRN has been implicated in a wide array of biological functions, including inflammation and neurite outgrowth, recent literature has shown that complete loss of PGRN due to a homozygous mutation leads to neuronal ceroid lipofuscinosis (NCL), a group of neurodegenerative lysosomal storage disorders. The discovery that patients with homozygous PGRN mutations present with a lysosomal storage disorder suggests that the pathogenesis caused by PGRN deficiency could be dose-dependent and that PGRN mutations which lead to FTD may also cause partial lysosomal dysfunction. Our current research has demonstrated that decreased PGRN levels significantly impaired lysosomal proteolysis. To elucidate the mechanism of this impaired lysosomal proteolysis, we examined the relationship between PGRN and the lysosomal enzyme cathepsin D as previous studies have shown that CTSD mutations, which result in decreased levels of the lysosomal enzyme cathepsin D, also cause NCL. Our results demonstrated that PGRN expression altered levels of cathepsin D. Furthermore, we demonstrated that PGRN interacts with cathepsin D and that individual granulins can increase its activity in vitro. Taken together, these experiments suggest that PGRN, or individual granulins, may act as an activator of cathepsin D. Reduced cathepsin D activity due to decreased PGRN levels may provide a potential mechanism by which PGRN haploinsufficiency leads to lysosomal dysfunction. This project will determine if PGRN and/or granulins are activators of cathepsin D by examining if decreased PGRN levels result in reduced cathepsin D activity in vitro and if PGRN and/or granulins are able to increase the activity of cathepsin D in a dose-dependent manner. Additionally, we will examine the specificity of the PGRN-cathepsin D interaction by determining if PGRN and/or granulins interact with and alter activity of other lysosomal enzymes. Importantly, this project will also examine the relationship between lysosomal and neuronal dysfunction by examining the PGRN-cathepsin D interaction in human induced pluripotent stem cell (iPSC)-derived cortical neurons harboring PGRN mutations. This analysis will examine if lysosomal dysfunction due to decreased cathepsin D activity contributes to the pathogenesis of human neurons with PGRN mutations. Ultimately, this study may provide insight into the mechanism by which PGRN mutations cause FTD and contribute to our understanding of how lysosomal dysfunction contributes to neurodegeneration. Furthermore, validation of PGRN-cathepsin D interaction as a mechanism of neurodegeneration would identify a potential therapeutic target to combat FTD.

POSTER 35

NONINVASIVE BRAIN STIMULATION TARGETING ASSOCIATIVE MEMORY NETWORKS IN OLDER ADULTS

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Noninvasive brain stimulation can change the specific location that is targeted as well as the large-scale brain networks that include the stimulation site. Here we examined whether stimulation of a parietal cortex location that is part of the hippocampal network responsible for episodic memory, would influence the function of this network. Older adults aged 64-81 years were given repetitive transcranial magnetic stimulation (rTMS) for five consecutive days and were tested the day before and after stimulation using a recognition and source memory task. For this task participants were asked to learn pairings of objects and scenes while being scanned using fMRI. Participants were tested by being shown previously studied and new objects and asked to indicate whether they recognized the object as old or new and how confident they were in that judgement. Following this recognition judgement, participants were asked to indicate the associated scene for old items. A wealth of previous findings indicates that memory for the object-scene locations should be dependent on the hippocampal network to a larger degree than memory for the individual objects. We found that stimulation resulted in an improvement in the ability to remember object-scene pairings, but not in object memory. These findings suggest that noninvasive stimulation can selectively influence the function of hippocampal memory networks in older adults. The hippocampus is one of the earliest brain regions affected during the progression of Alzheimer's Disease, and these findings are therefore relevant to the development of non-invasive stimulation interventions for individuals with clinical memory impairments.

POSTER 36

COGNITIVE NEUROLOGY & ALZHEIMER'S DISEASE CENTER (CNADC) CLINICAL TRIALS 2017

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The treatment of Alzheimer's disease (AD) is a central aim for the Cognitive Neurology and Alzheimer's Disease Center (CNADC). In response to promising new treatments and efforts to design biomarkers for AD and other forms of dementia, the CNADC has joined forces with the Alzheimer's Therapeutic Research Institute (ATRI), a consortium supported by the National Institutes of Aging and industry, to sponsor clinical trials for individuals with AD and other forms of dementia.

Emerging clinical trials and research studies are reviewed and approved by the Executive Committee of the CNADC. Recruitment of eligible individuals from the Clinical Core of the CNADC, advertising in the Chicago area community, and aging registries throughout Northwestern Medical Group are aided by the Outreach, Recruitment, and Education (ORE) Core. This cross-core collaboration emphasizes the inclusion of participants from all minority groups and otherwise underserved communities.

Current trials are as follows: **1) Anti-Amyloid Treatment in Asymptomatic Alzheimer's Disease (A4)**: A4 is a secondary prevention trial aimed at treating amyloid-positive but otherwise healthy individuals (aged 65-85) at risk for developing Alzheimer's disease (AD). Individuals with normal cognitive test scores will be screened with PET amyloid imaging. Those with positive amyloid PET scans will be enrolled into the study and will be treated for 3 years with an anti-amyloid drug or placebo. **2) Longitudinal Evaluation of Amyloid Risk and Neurodegeneration (LEARN)**: The goal of this trial is to evaluate the rate of cognitive change in amyloid-negative participants. Therefore, participants who do not show evidence of elevated amyloid will be enrolled in this 3-year study that will run in parallel to A4. Participants will be receive another PET scan at the end of the study. **3) CONNECT**: This Phase IIa study will evaluate an investigational medicine called AZD0530 (saracatinib) to treat early AD. In this study, 152 participants will be randomly assigned to receive either an active dose of AZD0530 or a dose of a placebo for the 52-week treatment period. **4) Study of Nasal Insulin to Fight Forgetfulness (SNIFF)**: This study is designed to determine whether insulin administered as a nasal spray improves memory in patients diagnosed with amnesic Mild Cognitive Impairment (aMCI) or Alzheimer's disease (AD). Previous studies have shown that insulin is responsible for multiple functions in the brain, and poor regulation of insulin may contribute to the development of AD. All participants will be randomly assigned to receive insulin or placebo for 12 months. **5) Alzheimer's Disease Neuroimaging Initiative – 3 (ADNI3)**: This study is designed to identify biomarkers that may be useful in the diagnosis of early AD, by examining annual MRIs, PET scans, lumbar punctures, blood tests and cognitive assessments in AD, Mild Cognitive Impairment (MCI), and healthy control subjects. **6) Memory Improvement through Nicotine Dosing (MIND)**: The purpose of the study is to see if use of a daily transdermal nicotine patch is able to produce a significant cognitive, clinical and functional improvement in participants with MCI. Neuronal nicotinic receptors have long been known to play a critical role in memory function in preclinical studies, with nicotine improving attention, learning, and memory function. The study will enroll 300 participants for a 2 year period.

POSTER 37

IS UNUSUALLY HIGH WORKING MEMORY PERFORMANCE ASSOCIATED WITH SUPERAGER'S SUPERIOR EPISODIC MEMORY PERFORMANCE?

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Objective: SuperAgers are a unique cohort of adults over age 80 who perform at least as well as average middle-age adults on episodic memory tests and at least average-for-age on tests in other cognitive domains. Age-related working memory decline is hypothesized to contribute to age-related impairment in episodic memory and the transition from Mild Cognitive Impairment to Alzheimer's dementia. The present study examined whether SuperAgers who have superior episodic memory ability also have better working memory than their cognitively average-for-age peers.

Participants & Method: 60 SuperAgers and 25 cognitively average elderly adults completed the Wechsler Adult Intelligence Scale (WAIS)-III and Rey Auditory Verbal Learning Test (RAVLT). WAIS-III Working Memory Index, Letter-Number Sequencing, and Longest Digit Span backward span were analyzed as tests of working memory. WAIS-III Processing Speed Index was examined to determine if it influenced performance on timed working memory tests. Associations between episodic and working memory were examined with Pearson's correlations. Between-group differences were examined with independent t-tests. Mean \pm SD are reported.

Results: There were no group differences in demographics or estimated premorbid intelligence. Working memory was positively correlated with RAVLT delayed recall across groups (p 's <0.05). SuperAgers outperformed their cognitively average peers on the WAIS-III Working Memory Index (121.1 \pm 12.9 vs. 108.5 \pm 15.1), Letter-Number Sequencing raw score (10.5 \pm 2.0 vs. 8.5 \pm 2.1), and longest Digit Span backward span (5.2 \pm 1.0 vs. 4.5 \pm 1.2; p 's <0.01). There was no group difference in processing speed.

Conclusion: SuperAgers outperformed their cognitively average-for-age peers on measures of working memory. This difference cannot be attributed to differences in processing speed or demographic factors, including premorbid intelligence. Results suggest that better working memory is associated with better episodic memory even in cognitively average and above-average elderly adults.

POSTER 38

RE-EVALUATING THE OLD AND RECLAIMING THE NEW: RESULTS FROM A DYADIC STORYTELLING INTERVENTION

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INTRODUCTION: Dementia affects both the person with the diagnosis (PWD) and their family. As the disease progresses, the family often takes on many caregiving duties and the relationship can evolve into “patient and caregiver”. Dyadic approaches have produced positive outcomes such as identifying and building the couple’s strengths, improved communication between caregiver and the PWD, and improvements in caregiver well-being (Scherrer, Ingersoll-Dayton & Spencer, 2013). Meeting with participants of the CNADC support groups for early-stage dementia [one for PWD and one for care partners] several common concerns emerged. 1) More programs that supported people with early stages dementia were needed, noting stigma that accompanied the diagnosis and feelings of isolation, 2) their early-stage experience was not reflected in media representation, which predominantly focuses on later-stages and 3) care partners expressed being referred to as “caregiver” invalidated the complexity of their relationship. These concerns inspired the development of the storytelling workshop, which was to create a meaningful shared activity for PWD and their partners, offering a platform to share their lived experience, advocating for resources and improving understanding around early-stage dementia.

METHODS: Two 8-week storytelling workshops for 1.5 hours each week included a “2 Good Thing” check-in, feedback on stories, discussion of the writing process and an 8-week curriculum building on storytelling techniques, writing prompts and homework assignments. The groups consisted of 5 dyads [n=10, 7 females & 3 males] with a median age of 57 and an age range of 34 – 71. Dyads were from Chicago and neighboring suburbs, Indiana and Michigan with mild cognitive impairment and Alzheimer’s disease diagnoses. Qualitative methods consisted of pre-post focus groups, which were audio-taped and transcribed and observational session field notes, Inductive content analysis was used to identify emerging themes.

RESULTS: Thematic analysis revealed there was commonality in the motivation to participate in the workshop, which included altruistic intention and shared time spent with their family member. Families benefitted from the intervention in several ways. 1) The couples were able to safely embrace and adjust to unambiguous loss as they navigated the progressive changes of the disease. This included shifting identities and roles, as well as shifting expectations, 2) The dyads experienced a strengthening of relational connectivity; i.e., within the core relationship, among the other group members, as well as a feeling of leaving a legacy, which was the story itself.

CONCLUSION: This storytelling workshop is a feasible dyadic intervention and provides opportunity for adaptation to change and loss associated with dementia and improvement in communication and relational connectivity.

POSTER 39

OBJECTIVE COGNITIVE AND FUNCTIONAL LOSS AND DEMENTIA RISK IN SUBJECTIVE COGNITIVE DECLINE

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Objective

Subjective cognitive decline (SCD) may represent a very early stage in the clinical development of dementia of the Alzheimer type. The present study sought to determine if cognitively normal older adults with SCD had different rates of cognitive change compared to those without SCD in a large, longitudinal dataset.

Participants and Methods

This retrospective analysis utilized data from the National Alzheimer Coordinating Center on 3,915 cognitively normal older adults who had received neuropsychological testing on an annual basis. SCD was determined by the participants' report of memory decline at baseline [yes = SCD+ (n=3152), no = SCD- (n=763)]. On 8 cognitive tests, the rate of cognitive change was measured using best linear unbiased predictors (BLUPs), which incorporate all available longitudinal data. An ANOVA with Tukey-Kramer post-hoc comparisons identified differences in BLUPs as a function of SCD.

Results

Performance improved in both groups on tests of episodic memory (Logical Memory Immediate and Delayed Recall) with significantly more improvement in the SCD- than SCD+ group (both $p < 0.005$). Performance declined in both groups on tests of executive attention (Trails B), processing speed (Coding, Trails A), and object naming (Boston Naming Test) with significantly worse decline the SCD+ than SCD- group (all $p < 0.005$).

Conclusions

Practice effects on episodic memory tests and slowed processing speed may represent sensitive metrics for identifying objective cognitive changes in SCD. More sensitive neuropsychological instruments may be needed detect these differences at the individual level.

POSTER 40

UNDERSTANDING AND COMBINING WORDS DURING SENTENCE COMPREHENSION IN PRIMARY PROGRESSIVE APHASIA

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Purpose. Sentence comprehension involves accessing individual words and integrating them into syntactic and semantic units (e.g., integration of a verb (eat) with its argument (the cake)). Sentence comprehension deficits have been observed across the major clinical subtypes of primary progressive aphasia (agrammatic (PPA-G), logopenic (PPA-L), and semantic (PPA-S)). Impaired single-word comprehension is one of the defining features of PPA-S and contributes to impaired sentence comprehension (Gorno-Tempini et al., 2011). However, little work has addressed whether integration of word meanings in sentence contexts is impaired in PPA-S (as in, for example, Wernicke's aphasia; Piñango and Zurif (2001)). In contrast, PPA-G and PPA-L are associated with relatively preserved single-word comprehension, and sentence-level impairments have been attributed, respectively, to impaired grammatical processing and working memory (Thompson & Mack, 2014; Wilson, Galantucci, Tartaglia, & Gorno-Tempini, 2012). However, it is not known whether deficits in higher-level word integration processes (e.g., prediction of verb-arguments) are present in these subtypes of PPA (as in, e.g., stroke-induced agrammatism; Mack, Ji, and Thompson (2013)). Thus, the purpose of the present study was to use eyetracking to examine the processes supporting online verb-argument integration and prediction in PPA.

Methods. 28 individuals with PPA (12 PPA-G, 10 PPA-L, 6 PPA-S) and 15 cognitively healthy older adults participated. In a non-linguistic control experiment, participants viewed an array of four object pictures. A circle appeared around one picture and latencies of eye movements to that picture were measured. In a lexical access experiment, participants viewed an array of four object pictures, heard a noun corresponding to one picture (e.g., cake), and clicked on the matching picture. In a verb-based integration experiment, participants heard sentences with restrictive verbs that were semantically compatible with only one object in a four-picture array (e.g., Susan will eat the cake, when the array included a cake and three non-edible objects) and unrestrictive verbs (e.g., move) that were compatible with all four objects. After sentence end, participants viewed a screen containing the words YES and NO and clicked a word to indicate whether the visual array had contained a picture of the final word of the sentence. In a verb-based prediction experiment, participants heard sentence fragments with semantically restrictive verbs (e.g., Susan will eat the ...) and clicked on the picture that best completed the sentence. Mixed-effects regression was used to analyze the accuracy and eye movement data; eye movement analyses were restricted to trials in which participants had responded correctly.

Results. Non-linguistic experiment. Accuracy was at ceiling in all participant groups (Table 1) and eye movement latencies did not differ significantly between healthy controls and any subtype of PPA (Fig. A). Lexical access. Accuracy was significantly greater in all groups compared to PPA-S, and in controls compared to PPA-G, but not PPA-L (Table 1). Eye movement data (Fig. B) indicated lexical access delays in all PPA groups, which were more severe in PPA-S as compared to PPA-G and PPA-L. Verb-based integration.

Accuracy was significantly higher in all groups compared to PPA-S in the restrictive condition, and in controls compared to PPA-S and PPA-G (but not PPA-L) in the unrestrictive condition (Table 1). Eye movement data (Fig. C) revealed overall delays in lexical access in PPA-G and PPA-S, which were more severe in PPA-S. In addition, listeners with PPA-S showed a rapidly-decaying effect of verb meaning, which emerged in the Noun region but disappeared in the PostNoun region (in contrast with other groups). Verb-based prediction. Accuracy was significantly higher in controls compared to PPA-G (Table 1). Impaired predictive eye movements were also observed in PPA-G, but not in the other PPA groups (reflected by a reduced proportion of fixations on pictures (vs. other areas of the array); Fig. D).

Discussion. Eye movements in the non-linguistic task did not differ between the PPA groups and controls, indicating that impaired performance on other tasks cannot be attributed to impairments in eye movement control. Consistent with previous research, PPA-S was associated with reduced accuracy and speed of lexical access (cf. Seckin et al. (2016)). In addition, the PPA-S group showed deficits in verb-argument integration. Online effects of verb meaning disappeared more rapidly than in other participant groups, and reduced verb meaning effects were evident in offline comprehension patterns as well. The results suggest that PPA-S is associated with a slow rise and rapid decay of lexical information, which may contribute to impairments in combining word meanings. The PPA-G group showed relatively mild impairments in lexical access accuracy and speed, which were evident both in single-word and sentence contexts. However, this group evinced a marked impairment in verb-based prediction, consistent with previous findings from stroke-induced agrammatism (Mack et al., 2013). The PPA-L group also showed a mild impairment in lexical access latency (but not accuracy). These effects were evident only in the single-word task, consistent with the view that sentence comprehension impairments in PPA-L are not lexically-based (e.g., working memory accounts).

Implications. The present results contribute to our understanding of the nature of word and sentence processing impairments in PPA, which may guide the development of novel language treatment protocols.

POSTER 41

NORTHWESTERN ALZHEIMER'S DISEASE CENTER OUTREACH AND RECRUITMENT CORE

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INTRODUCTION: OR Core specific aims are to: 1) Provide outreach and educational programs for the recruitment of underrepresented groups to the Northwestern ADC; 2) Optimize the recruitment of subjects into the Clinical Core and their retention through novel non-pharmacological interventions; and 3) Initiate and coordinate public education programs in conjunction with city, state and national entities.

METHODS/RESULTS: Social workers assess patient and family coping, determine needed resources, provide education and counseling and tailor recommendations to needs and symptoms, as part our clinical services and to enhance our recruitment and retention mission of the ADC. OR Core offers innovative Quality of Life Enrichment research programs and support groups. The Buddy Program (see separate abstract), persons with Alzheimer's disease or other related illnesses mentor first year medical students, is in its 20th year. A total of 220 buddy pairs have been matched. African American and Latino teaching artists have been trained to conduct the Memory Ensemble (an improvisational theatre experience and collaboration of the Northwestern CNADC and Lookingglass Theatre) made possible through a Civic Practice Lab project grant to Lookingglass Theatre. OR Core continues to collaborate with the African American community via partnerships with the Endealeo Institute and 6 faith-based organizations in, REACH to Faith: Research and Education for African American Caregiver Health (see separate abstract). We continue to partner with the Atlas Regional Senior Center for research recruitment and retention of African American research participants. A Retirement Research Foundation grant with Hana Center expands the work of KARE: Korean American Alzheimer Research and Education (see separate abstract). OR Core is a founding member of LA CARE: Latina/o Alzheimer's Coalition for Advocacy, Research and Education in the creation of an Alzheimer's resource directory.

The ORE Core continues to be responsive to collaborative efforts articulated by the National Alzheimer's Project Act (NAPA). ORE Core leader completed work on the HRSA (Health Resources & Services Administration) Dementia Curriculum for Healthcare Professionals; in addition to the state-wide collaboration Geriatrics Workforce Enhancement Program, CATCH-ON: Collaborative Action Team Training for Community Health-Older Adult Network. Via the Illinois Cognitive Resources Network (ICRN). OR Core is involved in the development of Dementia Friendly America-Illinois. A statewide workshop was held to bring together over 75 stakeholders from various sectors that share in the vision of a dementia friendly Illinois (see separate abstract). OR Core is pivotal to dementia-related education of trainees, fellows and the community. Eighteen CME-accredited AD seminar series were attended by over 850 researchers and clinicians and 26 education programs were delivered to over 3,100 individuals. The Storytelling Program (see separate abstract) has supplemented the education of the lived experience of dementia for over 1000 individuals of multiple disciplines.

CONCLUSION: The CNADC OR Core continues to increase public awareness of dementia and treatment using community outreach, the training of scientists and clinicians, the provision of programs and support services for diagnosed persons and families and engagement in community-based research.

POSTER 42

ON YOUR SIDE: ONLINE SUPPORT AND EDUCATION FOR INDIVIDUALS WITH PRIMARY PROGRESSIVE APHASIA

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Primary Progressive Aphasia (PPA) is a clinical dementia syndrome characterized by progressive loss of language affecting other cognitive domains over time. There are no approved drug treatments for PPA; however, psychoeducational support programs have been shown to improve quality of life and well-being for persons with neurodegenerative cognitive decline and their families. Persons with PPA and their families rarely have the opportunity to benefit from this type of intervention, largely due to difficulty recruiting enough members with PPA in one geographic region.

A pilot online/videoconference psychoeducational support program was launched in Spring/Summer 2016 specifically for persons living with PPA to circumvent geographic limitations and improve healthcare access. Five couple dyads from the United States and Canada were recruited into the study and included persons with PPA with mild (n=4) to moderate (n=1) expressive and receptive language impairments and their spouse care-partners. Dyads met by videoconference for 8-weekly 2-hour sessions. Sessions included dyad education on PPA, communication strategies, and psychosocial interventions followed by separate consecutive support groups for diagnosed persons and care-partners. Pre-post questionnaires measured confidence for communication abilities, life participation, mood, well-being and perceived stress. Pre-post interviews with dyads and care-partners were documented and all videoconference sessions were audio-recorded. Audio recordings and field notes were reviewed and coded for emerging themes using inductive content analysis.

Results suggest that an online videoconference psychoeducational support program for persons with mild PPA and their care partners is feasible and represents a novel solution to connect individuals with rare diseases such as PPA. Participants appreciated the opportunity to meet each other; however, the variability among symptoms, age and disease knowledge impacted satisfaction, particularly for care partners. Further research is needed to identify the ideal group composition and content.

POSTER 43

REACH TO FAITH: RESEARCH AND EDUCATION FOR AFRICAN AMERICAN CAREGIVER HEALTH

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Introduction: The African American (AA) population represents roughly 39% of the local Chicago population aged 65 and older. African Americans are two to three times more likely to be diagnosed with dementia than white Americans and disproportionately receive less dementia care and education. There is a prominent role of religion and religious organizations in the AA community. An inherent sense of duty coupled with religious beliefs about the importance of helping behaviors have a strong influence on why AAs assume the caregiver role. It has also been shown that AAs are more likely to use religious methods such as prayer to cope with burden experienced from caregiving. Despite the importance of religion to AA caregivers and widespread interest in developing interventions for caregivers of dependent adults, few researchers have involved faith communities in caregiver intervention research.

Methods: Beginning Summer 2013, with the support of Northwestern University's Alliance for Research in Chicagoland Communities Seed Grant, Endeleo Institute-Health and Northwestern University (NU) Cognitive Neurology and Alzheimer's Disease Center (CNADC) began their community-academic partnership for the purpose of identifying a means to alleviate the burden experienced by AAs caring for persons with dementia. Our partnership building focused on forming relationships among the CNADC, Endeleo, and seven faith-based organizations located on the Southside of Chicago in predominately AA neighborhoods. Our partnership building eventually expanded to include Rush College of Nursing. Two focus groups were held in March and June 2015 with three and nine participants respectively. These focus groups were audio recorded, transcribed, and qualitatively analyzed. Surveys were also completed to collect preferences about needed community services.

Results: Work to date has included an exploration of attitudes about Alzheimer's disease, caregiving, and barriers to seeking help among the AA community in addition to an opportunity for the collaborating churches to educate the NU CNADC on cultural issues surrounding dementia and caregiving from the African American perspective. Monthly meetings provided opportunities for the NU CNADC to help the partners develop research capacity through discussions on the basics of research, results of a literature review of caregiving in the AA community, barriers to seeking help, and human subjects training by NU Institutional Review Board staff. With this foundation two focus groups with AA caregivers were held. Preliminary data analysis showed that AA caregivers are experiencing significant amounts of stressors related to caregiving but rarely describe this as burdensome. Many would like greater access to resources for education and support, but mostly desire this support to come from within their own families. This project has also inspired the development of a larger consortium that will encompass both faith and secular communities.

Conclusion: This community engaged research project has been successful in establishing a partnership among Northwestern, Endeleo Institute and south side Chicago faith based communities for the purposes of exploring and better understanding the needs and experience of African Americans caring for persons with Alzheimer's disease. Results from the focus groups are consistent with current literature and will guide further collaboration in developing helpful interventions.

POSTER 44

KARE: KOREAN AMERICAN ALZHEIMER'S RESEARCH AND EDUCATION

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Introduction: The barrier to adequate dementia care in the Asian and sub-Asian Korean American community includes a general non-acknowledgement of Alzheimer's disease and related dementia (ADRD) and a prevalent stigma against the field of mental health. Memory loss, difficulty with daily tasks, and other common symptoms of Alzheimer's disease and dementia are interpreted as "insanity or senility" that naturally comes with aging. ADRD prevalence in both the Asian and Korean American community is underreported and unknown. Korean Americans are one of the least studied groups among other Asian subgroups. Misunderstanding of ADRD and underrepresentation of Korean Americans in research may prevent persons with ADRD and their families from receiving adequate care that would allow them to remain independent and maintain meaningful relationships.

Methods: Building on previous community-academic partnership building in addition to results of interviews and focus groups among Korean American older adults and health care professionals, the KARE team presented translated National Institute on Aging/Association for Community Living brain health workshops to a total of 414 Korean older adults (318 who attend an education program for 'younger seniors' and 96 'older seniors' who live in senior housing). Younger seniors are defined as 65 to 75 years old who have worked in the U.S. and have a higher level of acculturation. Their interest in brain health focuses on knowledge gain and prevention measures of dementia and Alzheimer's disease. Older seniors are at least 75 years old and have not worked in the U.S.; therefore, they have lower acculturation levels, meaning that they do not speak English well and rely on a specific public benefit package and housing assistance.

Results: On pre/post ADRD knowledge tests, the younger seniors (n=318 over 5 lectures had a pre-mean (sem) = 9.71 (0.24) and a post-mean (sem) = 10.94 (0.24) (p<0.0001). Older seniors (n=96 over 3 lectures) demonstrated a pre-mean (sem) = 5.47 (0.42) and a post-mean (sem) = 5.78 (0.42) (p=0.31). Two focus groups were conducted, one for younger and one for older seniors. The younger seniors focused on brain health measures that they could incorporate into their lives for the long term while the older seniors were more focused on what immediate actions they could take to better their situation.

Conclusion: KARE provided a successful education program on brain health for younger seniors in the Korean American community, with an expressed interest in learning more. There was a statistically significant improvement in the younger seniors' knowledge but not the older seniors. The older seniors need a specifically tailored and culturally relevant education program and evaluation. Additional education is requested by younger seniors. Future efforts will be devoted to these goals.

POSTER 45

SONGS BY HEART: A MUSIC INTERVENTION FOR PERSONS WITH DEMENTIA

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INTRODUCTION: Research demonstrates that musical aptitude and music appreciation are two of the last remaining abilities in patients with Alzheimer's disease. Musical involvement by persons with dementia has shown positive psychological outcomes, such as decreased levels of depression and agitation along with an increase in social interactions. This study explored the efficacy of a 10-week; 5 day per week music intervention for persons with early and moderate dementia in an assisted living community in the Chicagoland area and impact on residents' quality of life.

METHODS: Sixteen participants were recruited from a Silverado Assisted Living facility in the Chicagoland area: 12 women and 4 men. The participants ranged in ages from 72 to 98. Mean age was 87. Thirteen participants had a clinical diagnosis of Alzheimer's disease. The remaining three had a diagnosis of behavioral variant frontotemporal dementia, Lewy body dementia and primary progressive aphasia respectively. All were in the advanced stage of dementia. Participants' family member and the certified nursing assistant assigned to them (CNAs) were interviewed separately regarding patients' mood, well-being, and overall quality of life prior to the treatment. The intervention included a group session where a trained vocal performer sang from a repertoire chosen from caregiver reports of musical preferences 5 times per week, 45 minutes per day with piano accompaniment. During the performance of familiar songs, the vocalist circled the room while making eye contact with participants or holding their hands. She regularly invited them to join in the singing or clapping. Detailed observational field notes were documented on the Monday and Friday of each week. After the 10-week intervention participants' family members and CNA were re-interviewed. All interviews and observational fieldnotes were coded for emerging themes.

RESULTS: A sample of observational field notes from the 10 week intervention (weeks 1, 3 and 10) identified an overall general theme of: 1) Active participant engagement during the music intervention. The following sub-themes illustrate what facilitated this engagement: 1a) Participants who demonstrated high levels of engagement (singing robustly, clapping, dancing) on the first day of the intervention continued to do so throughout the intervention and appeared to be the most physically functional; 1b) Participants with greater cognitive or physical challenges, such as those using walkers or sitting in wheelchairs, demonstrated their engagement in more subtle ways, such as tapping feet while resting with closed eyes or making eye contact with or holding the hand of the singer; 1c) Therapeutic tools introduced during several sessions, such as hand bells and hand scarves, demonstrated increased participation among members; 1d) Song selection impacted participation, with patriotic songs resulting in the highest observable engagement compared to songs of other genres; 1e) Increased levels of engagement were observed with larger

“audience” size or if a CNA or family member sat next to a participant during the program; 1f) Participation during sessions increased as the singer engaged participants one on one, utilizing therapeutic techniques such as holding or rubbing participant’s hands and singing at participant eye level. Post-interviews with CNAs revealed the SBH intervention had among the highest rate of people remaining in the sessions as compared to other Silverado activities. Interviews with family members and CNAs regarding effects of the intervention outside of the session were mixed. Some participants were more engaged after the sessions with other residents and activities. Others demonstrated no observable differences outside the session.

CONCLUSION: The Songs By Heart program is a feasible musical intervention that promotes social engagement in persons with advanced dementia. The multiple components in this intervention (active and personalized approach of the singer, attention to song selection, use of therapeutic tools) positively influence program outcomes.

POSTER 46

COMMUNICATION BRIDGE: AN INTERNET-BASED PERSON CENTERED INTERVENTION FOR IMPROVING ACCESS TO CARE AND QUALITY OF LIFE FOR INDIVIDUALS WITH DEMENTIA

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Individuals with aphasia symptoms due to neurodegenerative dementia [(e.g., primary progressive aphasia)] are under-referred for speech-language therapy (SLT) services. The goal of this study was to bridge geographic limitations and improve access to care for individuals with dementia by providing person centered SLT via the Internet using a custom web-application. Participants received an Initial Evaluation, 8 person centered web-based SLT sessions, followed by 2- and 6-month Evaluations. Outcome measures assessed the feasibility of providing web-based SLT, strategy use and compliance, functional gains, and the duration of benefit using data from therapist reports, participant questionnaires and post-therapy interviews. Over forty participants from 21 states and Canada have been enrolled thus far, suggesting web-based therapy is feasible and improves access to care. Functional gains were identified at 2-months and maintained at 6-months. Participants reported significantly higher levels of confidence in communication at the 2-month Evaluation and no significant decline at 6-months. Therapist reports suggested care-partner involvement is a critical and essential element for SLT. Internet-based speech-language therapy using a person centered intervention approach appears to provide an effective method for providing care to individuals with dementia and mild/moderate aphasia symptoms who have an engaged care-partner and prior familiarity with a computer.

POSTER 47

A POSITIVE EMOTION REGULATION INTERVENTION FOR FAMILY CAREGIVERS OF PEOPLE WITH DEMENTIA: THE LEAF STUDY

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Background: In the United States, 80% of caregiving is provided by family members (Etters, Goodall et al. 2008). Family caregivers for individuals with dementia face a unique set of stressors and are highly susceptible to burnout, physical health issues, and depression (Epstein-Lubow et al., 2012; Fisher et al., 2011; Pinquart & Sörensen, 2003). Caregivers can be particularly hard to reach for psychosocial interventions as they are often unable to leave home due to their caregiving duties. Research suggests that interventions focused specifically on increasing positive emotion may decrease the deleterious consequences of stress (Danner, Snowdon et al. 2001; Moskowitz 2003; Blazer and Hybels 2004; Pressman and Cohen 2005), but this work has yet to be applied to caregivers. The purpose of the LEAF study was to conduct a randomized controlled trial testing a video-conference delivered positive emotion skills intervention for caregivers of people with dementia. Here we present data on recruitment, retention and acceptability of the intervention.

Methods: The LEAF (Life Enhancing Activities for Family caregivers) Study is a randomized wait-list control trial that evaluates a 6-session skills-building program aimed at increasing positive emotion and reducing stress amongst family caregivers. Intervention sessions are delivered by trained facilitators via videoconference on study-supplied tablets with WebEx software. Participants were recruited via brochures in clinic waiting rooms, by word of mouth in online and in-person caregiver support groups, by internet postings on online bulletin boards, clinical trial matching sites, and Facebook. All participants are assessed at baseline, after intervention, and at three follow up time points: 1 month, 3 months and 6 months post intervention.

Results: Recruitment took place between August 2014 and November 2016 (28 months). One hundred and seventy (n=170) family caregivers (143 female, 27 male, mean age = 63 years, range 34-87 years) participated in the intervention (86 Intervention, 84 Control). Participants came broadly from Urban (50%), Suburban (33%) and Rural (17%) areas. Overall, 79% of all participants (n=175) were retained through the post-intervention period: 3% became ineligible before randomization and 18% dropped out during or after the intervention. On a scale of 0-10, participants rated this program very highly, saying they would be likely to recommend it to a friend (Mean = 9.28, n=139) or another caregiver (Mean = 9.51, n=139).

Practical Implications: The LEAF study provides promising support for the use of web-delivered interventions targeting stress among caregivers. Study retention was similar to or higher than other interventions with older adult caregivers (Waelde 2017, Czaja 2013, Tremont 2008, Wilz 2011). LEAF participants found the intervention highly acceptable. The online delivery of the intervention has the potential to be cost-effective and allows for broad dissemination.

POSTER 48

THE COMPREHENSION OF SENTENCES WITH STRUCTURALLY DEFINED GAPS IN AGRAMMATIC PRIMARY PROGRESSIVE APHASIA: EVIDENCE FROM EYE-TRACKING

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Introduction. Primary progressive aphasia (PPA) is a degenerative disease affecting language while leaving other cognitive facilities relatively unscathed (Mesulam, Wieneke et al. 2012). Here we focus on agrammatic PPA (PPA-G), which is characterized by agrammatic language production with impaired comprehension of non-canonical syntactic structures, but spared single word comprehension (Gorno-Tempini, Hillis et al. 2011).

Non-canonically ordered sentences subvert the dominant agent-verb-theme order of arguments in English, such that the theme precedes the verb in these structures. For example, in an object relative clause (The boy that the girl saw [gap] is ...), the theme argument (boy) has been extracted from the [gap] position, and so precedes its verb (saw) and the agent argument (girl). In a subject relative clause (The girl that [gap] saw the boy is ...), the dominant agent- verb-theme order is preserved.

Individuals with agrammatic aphasia following stroke also have particularly impaired comprehension of non-canonical syntactic structures (Grodzinsky 1986). Empirical accounts of this deficit vary – here we focus on two: One posits a deficit at thematic integration of a verb with its arguments (Thompson and Choy 2009), another that structure building is intact but delayed (slow syntax) (Burkhardt, Avrutin et al. 2008). Distinguishing between these hypotheses requires a method with a fine-grained temporal resolution.

Method. We used an eye-tracking method to examine these hypotheses of agrammatic comprehension in 12 participants with the agrammatic subtype of primary progressive aphasia and 15 healthy controls matched for age and education. Eye-movement patterns in healthy controls were expected to be consistent with a structural link between the extracted argument and structural gap, which should take the form of increased looks to the extracted element immediately following the gap position in the sentence. Deficient thematic integration predicts on-time but abnormal eye-movements to structural gaps in object-relative sentences. Slow syntax predicts delayed but not otherwise abnormal eye-movements to structural gaps in both sentence types.

During the experiment, participants listened to 32 four-sentence stories (1). In each story, the fourth sentence was created in two versions, one with a subject-relative clause (1a) and one with an object relative clause (1b).

- (1) One day a bride and groom were walking in the mall.
The bride was feeling playful, so the bride tickled the groom.
A clerk was amused.
- a) Point to the one that _[gap] was tickling the groom in the mall.
 - b) Point to the one that the bride was tickling _[gap] in the mall

For each story, we also created a 3x3 array with pictures of the sentence elements (e.g., bride, groom, mall, clerk) in the four corners. Participants responded to each story by clicking on one of the four pictures via computer mouse. The correct answer was the picture corresponding to the antecedent of the gap (e.g., bride in 1a; groom in 1b). We measured eye-movements during the auditory story.

Results. For healthy controls, looks to the extracted argument increased in both sentence types following the gap. For participants with PPA, looks to the extracted argument showed a similar increase to that of the controls after the gap in subject-relative clause sentences. However, eye-movement patterns in the PPA participants were abnormal for the non-canonical object relative clause sentences, where looks to the extracted argument decreased immediately following the gap. These results are consistent with the hypothesis that agrammatic comprehension deficits reflect impaired thematic integration, but are inconsistent with a slow syntax hypothesis.

Implications. The findings from this study help to more accurately delineate the nature of the language deficit in PPA, which may lead to more effective treatment interventions and a deeper understanding of the neurological basis of language.

POSTER 49

NORTHWESTERN ALZHEIMER'S DISEASE CENTER (NADC) CLINICAL CORE

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INTRODUCTION: The Northwestern Alzheimer's Disease Center (NADC) is entering its 22nd year of funding from the National Institute on Aging (NIA). The goals are to:

- 1) provide state-of-the-art care to patients with Alzheimer's disease and other causes of dementia and mild cognitive impairment, and
- 2) support clinical and basic research on memory and aging through the collection, storage and dissemination of clinical data and brain tissue from research participants.

Resources support local, national and international collaborations. The NADC is comprised of five Cores: Clinical, Administrative, Neuropathology, Data Management and Biostatistics, and Outreach, Recruitment and Education. Over the past year, the Clinical Core has worked closely with the Outreach, Recruitment and Education Core, Data Management and Biostatistics Core, and the Neuropathology Core to recruit and enroll subjects, facilitate brain donations, support research on dementia and aging, and educate the public on effectively coping with these illnesses.

METHODS: The Clinical Core recruits cognitively healthy individuals and patients with different forms of dementia (e.g. AD, PPA, FTD) and cognitive impairment. Participants are followed annually according to the methods of the Uniform Data Set (UDS) of the NIA Alzheimer's Disease Center (ADC) program, many for the remainder of their lifetime, after which brain donation provides tissue for investigators studying Alzheimer's and related disorders. The Clinical Core works with the Data Management and Biostatistics Core to compile and electronically store all data obtained to make it available for approved collaborative studies, and the National Alzheimer's Coordinating Center (NACC) database.

RESULTS: From 1996-2017 the Clinical Core has enrolled more than 2,053 participants, and the current active cohort is 537 (See Figure 1). In the past year, the Clinical Core supported over 17 different investigators and 27 studies being conducted in the areas of cognitive neuroscience, clinical trials, neuroimaging and neuropsychology (See Table 1). A total of 34 original publications, 45 abstracts and 31 publications based on collaborative NIH studies, including the Alzheimer's Disease Genetics Consortium (ADGC), the Alzheimer's Disease Neuroimaging Initiative (ADNI), the International Genomics of Alzheimer's Project (IGAP) and the Women's Health Initiative Memory Study (WHIMS).

CONCLUSIONS: The Clinical and Data Management/Biostatistics Cores of the NADC together have facilitated research on Alzheimer's disease, frontotemporal dementia, primary progressive aphasia and age-related cognitive change and have promoted collaborative efforts nationally and internationally.



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Both Janna Dutton and Kathryn Casey are Certified Elder Law Attorneys (CELA). The Certified Elder Law Attorney is the only American Bar Association approved designation for elder law.

Helen Mesoloras is President of the National Academy of Elder Law Attorneys, Illinois Chapter.

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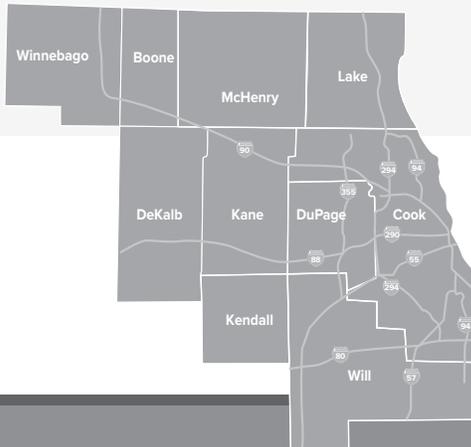
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