Self Discovery Through Opportunity

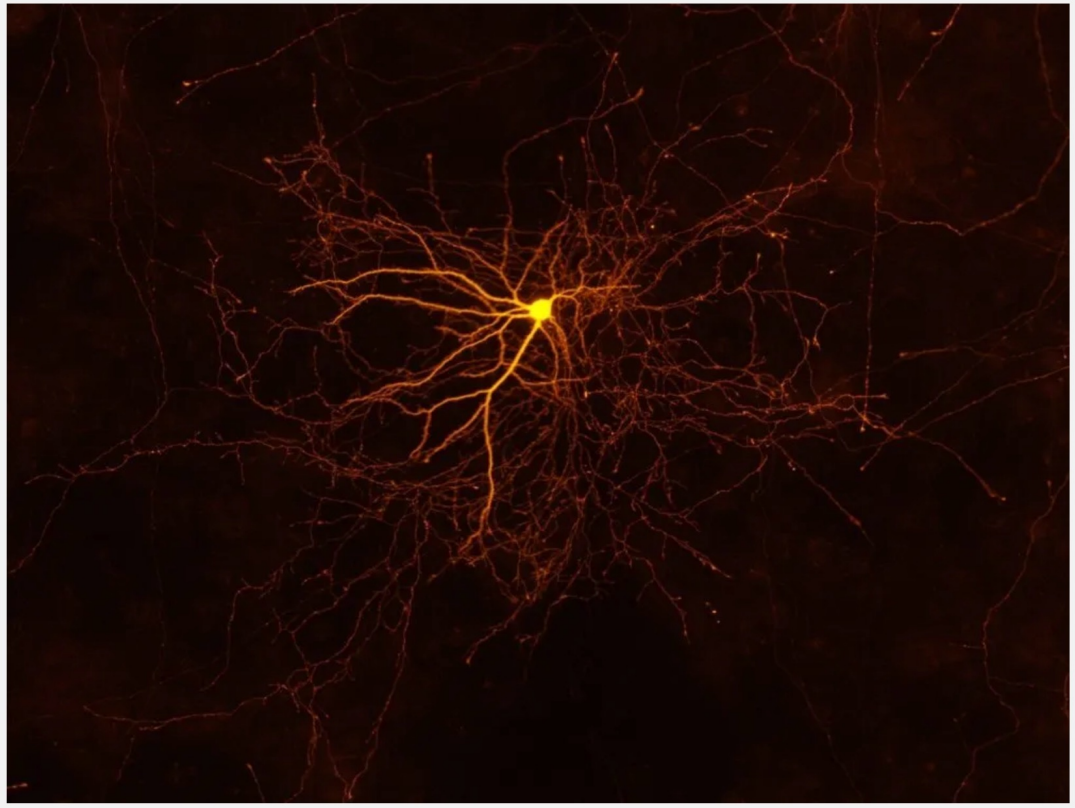
UC Davis GGIP Symposium

Cellas Hayes, PhD

Stanford University

My Start in Research

Why do we forget in old age?



Laboratory Assistant Inbox ×

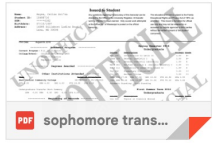
Cellas Hayes <cahayes3@go.olemiss.edu> Fri, Aug 12, 2016, 7:43 AM

to nmashpol

Good morning my name is Cellas Hayes. I am eligible for work-study, and I was interested in the position as a Laboratory Assistant. I am interested in this job because it would give me hands on research and an opportunity in a lab. I am currently a Sophomore Classics major with an emphasis in Latin along with being a Psychology(Pre-Med) major. I have attached a copy of my transcript and resume. I would be very grateful if we could have an interview. I am currently scheduled move back in on campus on the 17th.

2 Attachments • Scanned by Gmail

Cellas Hayes RES...



sophomore trans...

Nicole Ashpole <nmashpol@olemiss.edu> Sun, Aug 14, 2016, 6:14 PM

to me

Hello,

We will be holding interviews for the laboratory assistant position on Thursday, August 18th. If you are available to interview that day, please let me know what time is best.

Thanks

Tue, Aug 23, 2016, 9:41 AM

Hello Cellas,

I hope the first few classes are going well. When you get a chance, please stop by the Financial Aid office to get a sheet that needs to be filled out and signed by both you and I for the work-study position. I should be in the office this afternoon (except from 2-3), and most of the day tomorrow (except from 10:30-11:30).

Thanks

N. Ashpole

Detrimental Aspects of Aging

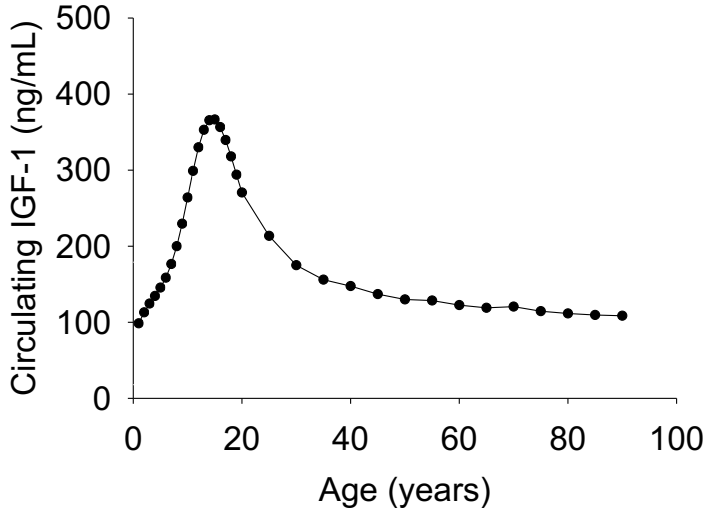
- Age-related cognitive decline is one of the fastest-growing health concerns
- Aging is associated with reductions in:
  - Processing speed
  - Inductive reasoning
  - Learning and memory
- Decreased synaptic structure; reduced neurotransmitter synthesis
- Altered receptor signaling; dysregulated neuronal gene and protein expression
- Comorbidities
  - Diabetes Mellitus, Hypertension, Cardiovascular Disease (i.e. Stroke), Dementia

As reviewed by Hedden and Gabrieli, Nat Rev Neurosci 2004; Rjell et al, Rev Neurosci 2010; VanGuilder et al, Front Aging 2011; Sonntag et al, Front Aging 2013

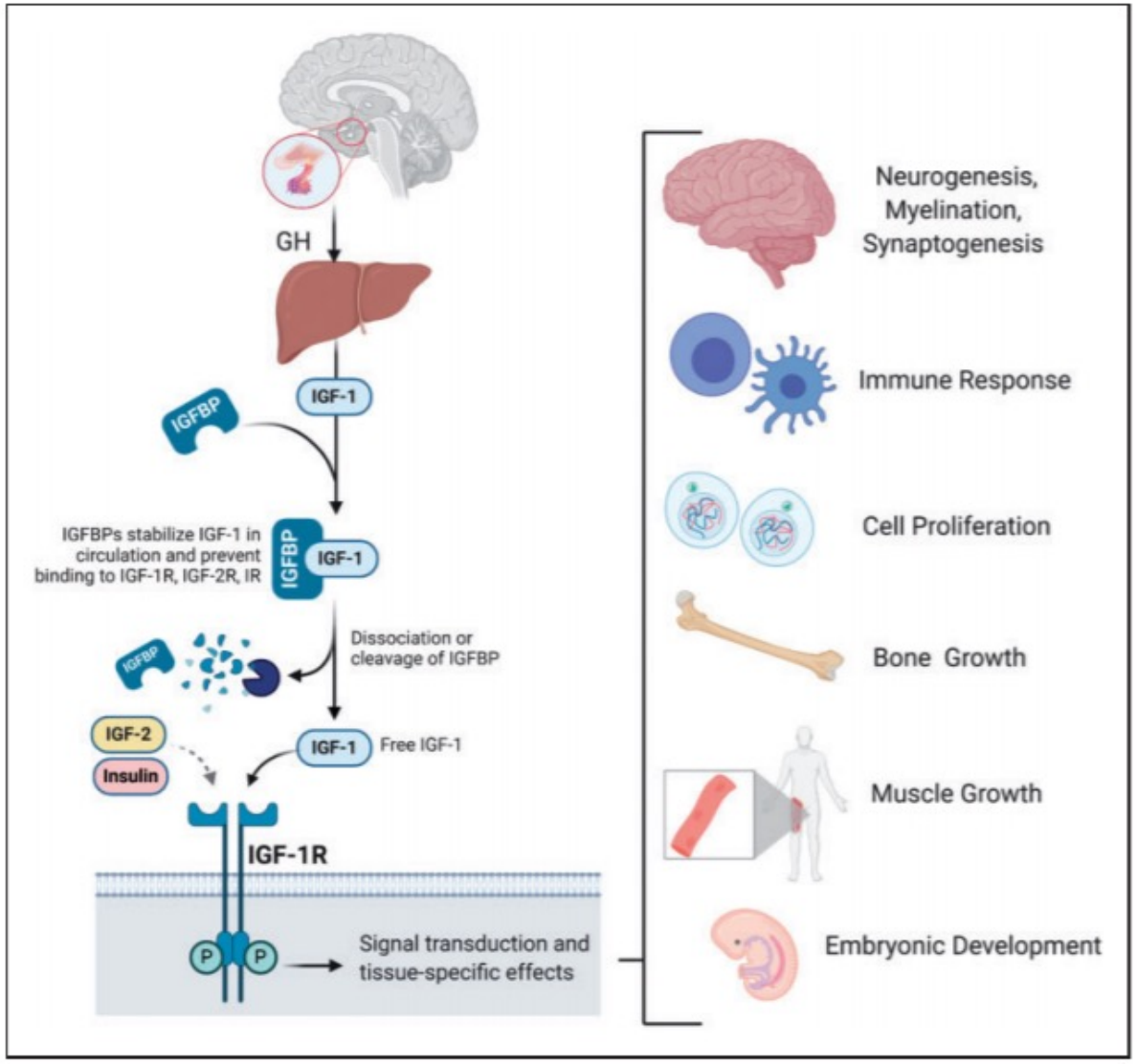
Insulin-like Growth Factor-1 (IGF-1)

- Pleiotropic hormone
- Regulated by pituitary and produced in liver
- Crosses the blood brain barrier
- Growth factor

Mayo Clinic Reference Guide

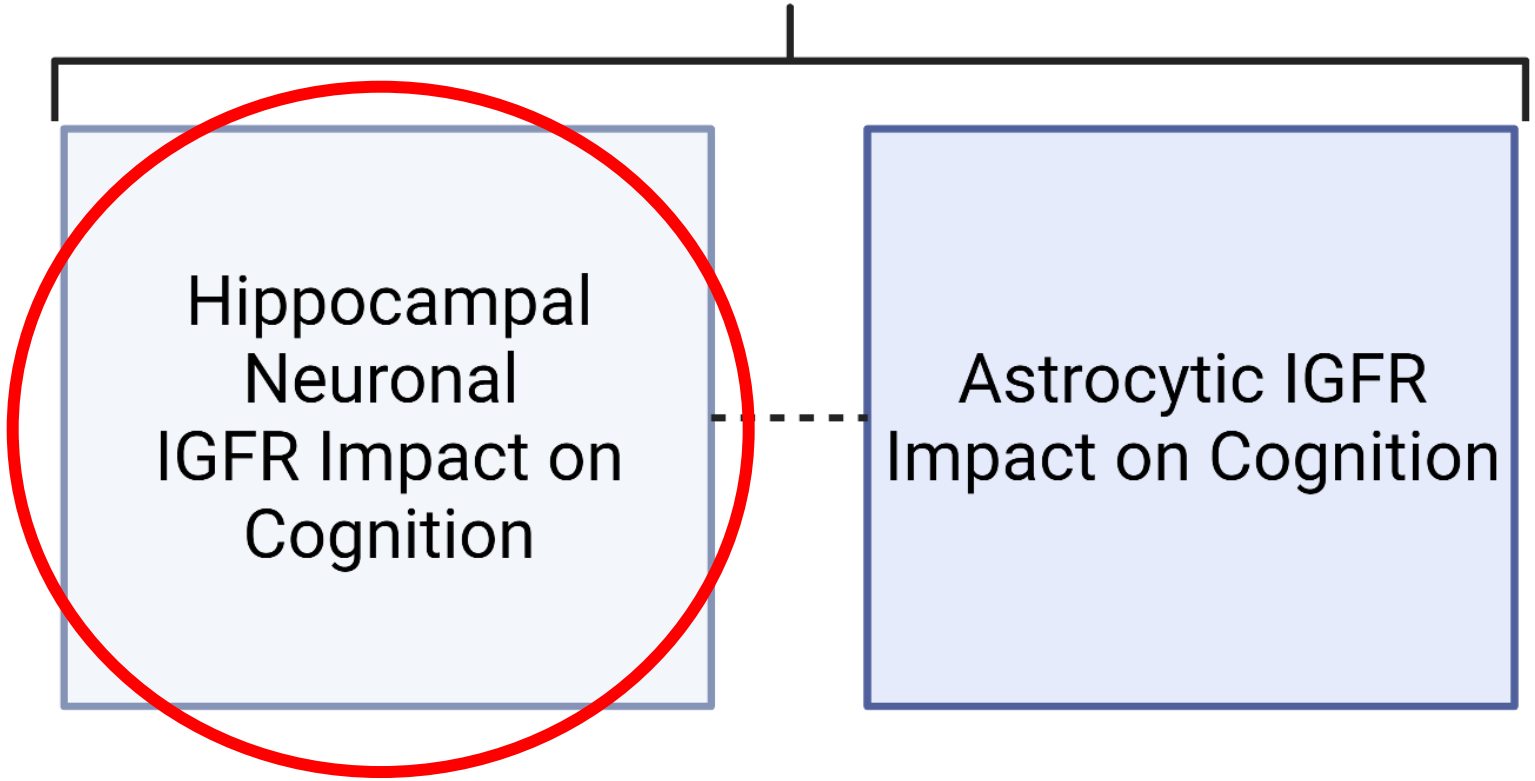


Adapted from www.mayomedicallaboratories.com



Hayes CA. et al (2021) *Journal of Cerebral Blood Flow and Metabolism* pp. 2475-2491

Does IGFR signaling impact learning and memory in a cell-specific manner?

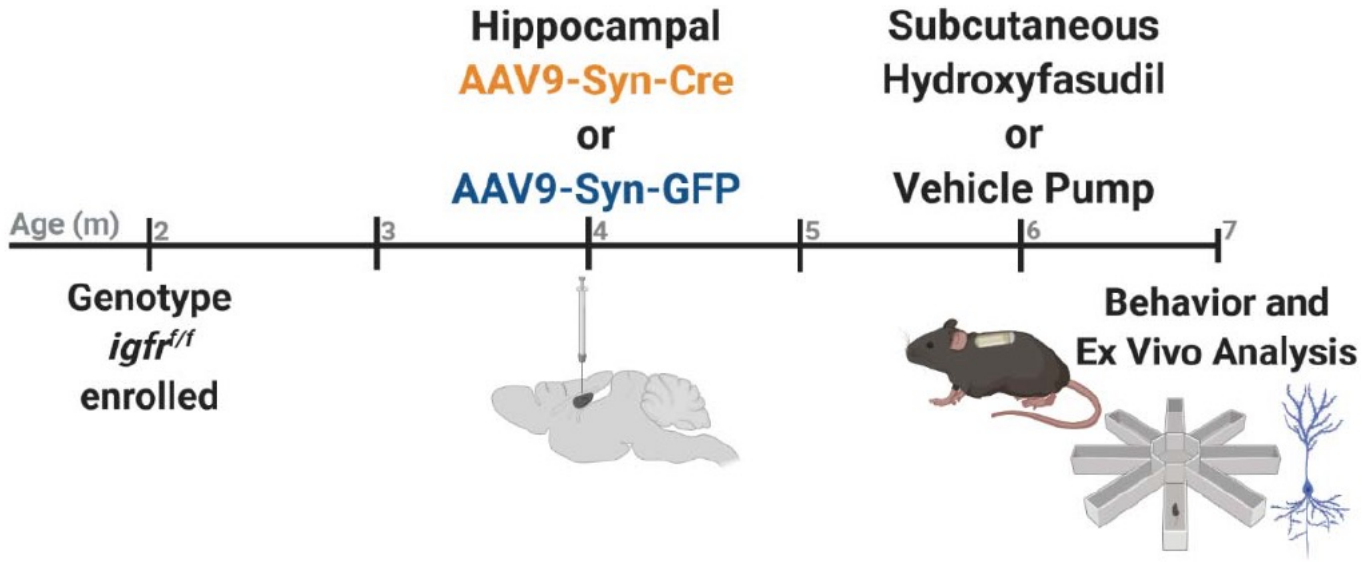


N. Ashpole

Hypothesis: Reductions in hippocampal neuronal IGFR in adulthood increases cognitive deficits.

Methods:

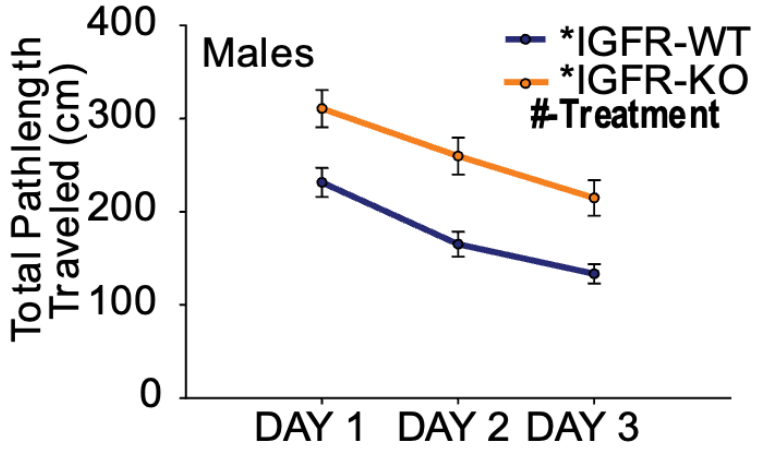
- Male and female transgenic mice
  - *igfr*$^{f/f}$
- Stereotaxic injections of AAV9-Syn-Cre or control AAV9-Syn-GFP (control) in the dentate gyrus and CA1 subregions of the hippocampus
- Behavioral assessments
  - RAWM
  - NOR
  - Golgi-Cox Stain
- Osmotic pump implantation (14 days)
- Primary neuronal cultures

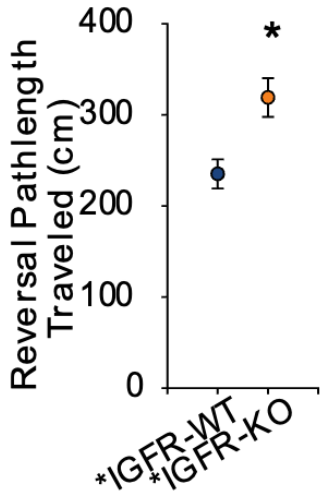


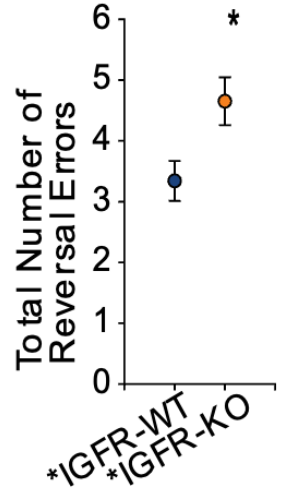
Hayes et al. 2021 bioRxiv 2021.08.08.455596

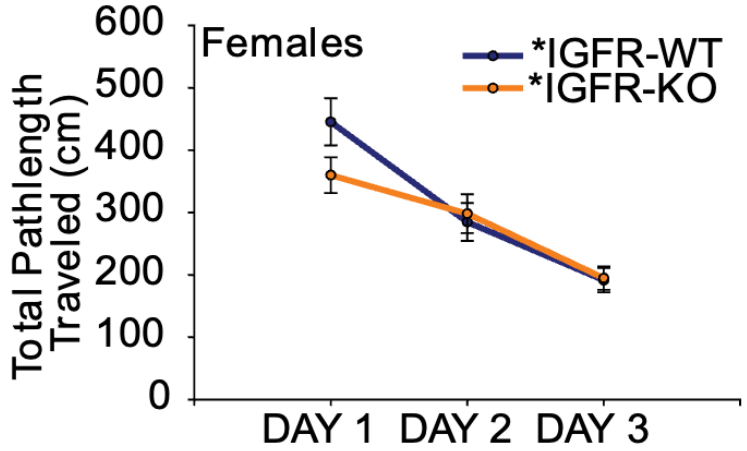
Behavioral Changes with Neuronal IGF-1 Reductions

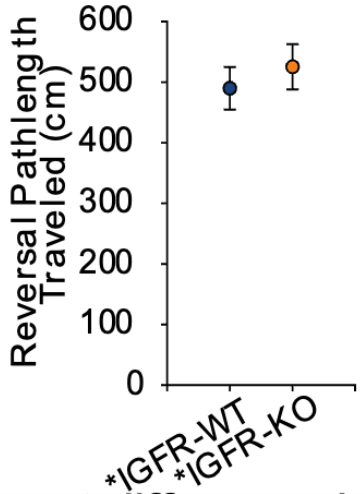
24 training sessions to swim to platform → Remove platform to test memory → Move platform to new location to test extinction

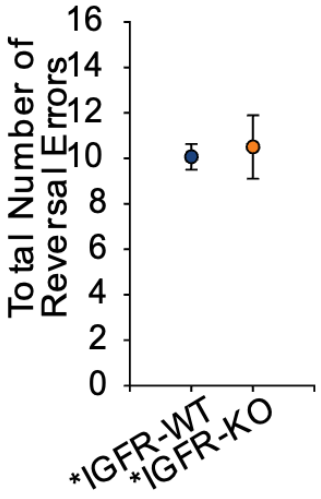






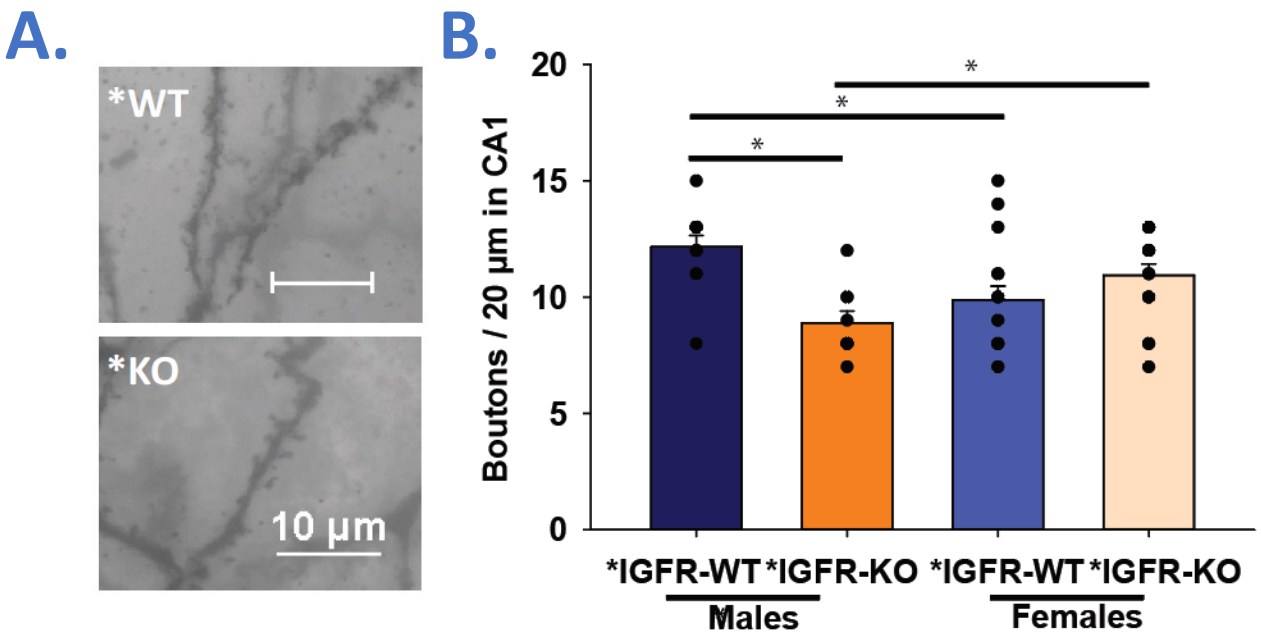


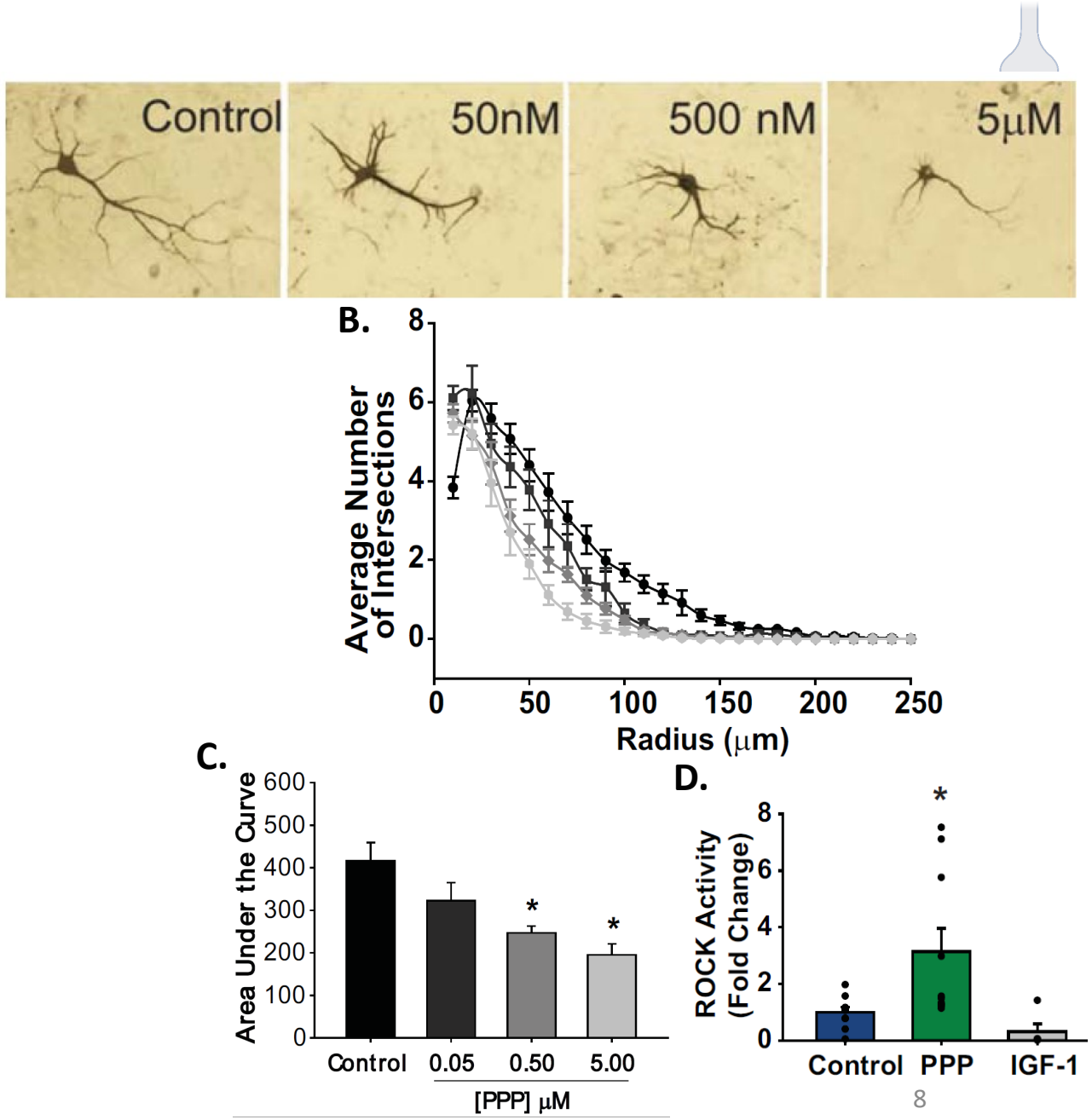




Reduced Synaptic Boutons

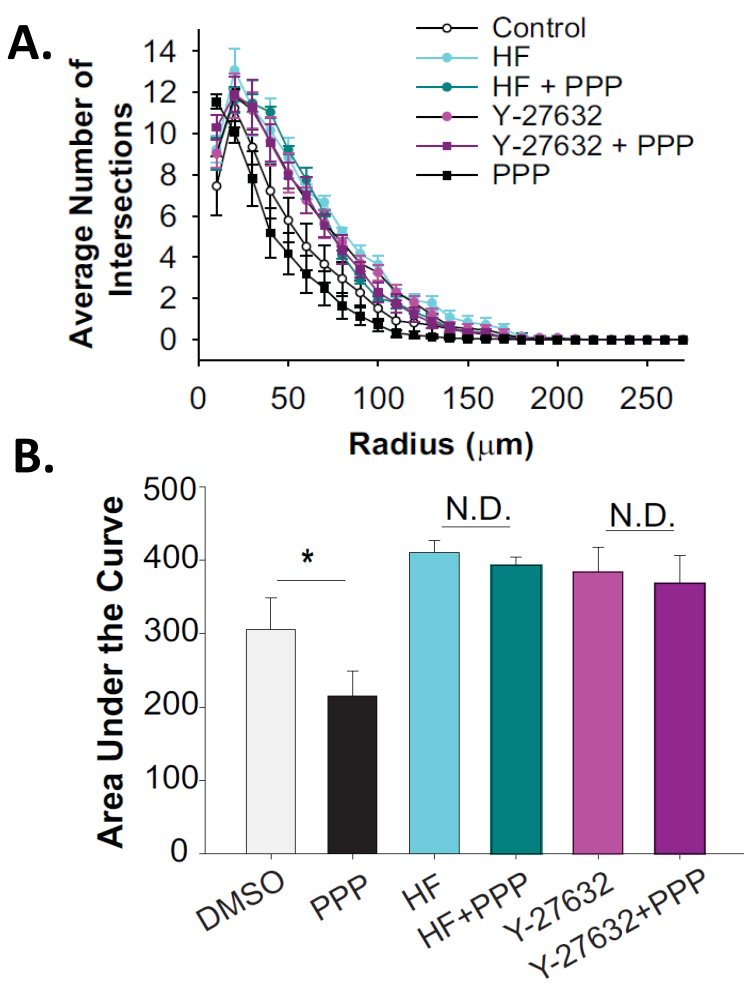
Method: 45µm brain slices were stained using Golgi's method and boutons were quantified in CA1 region of hippocampus.



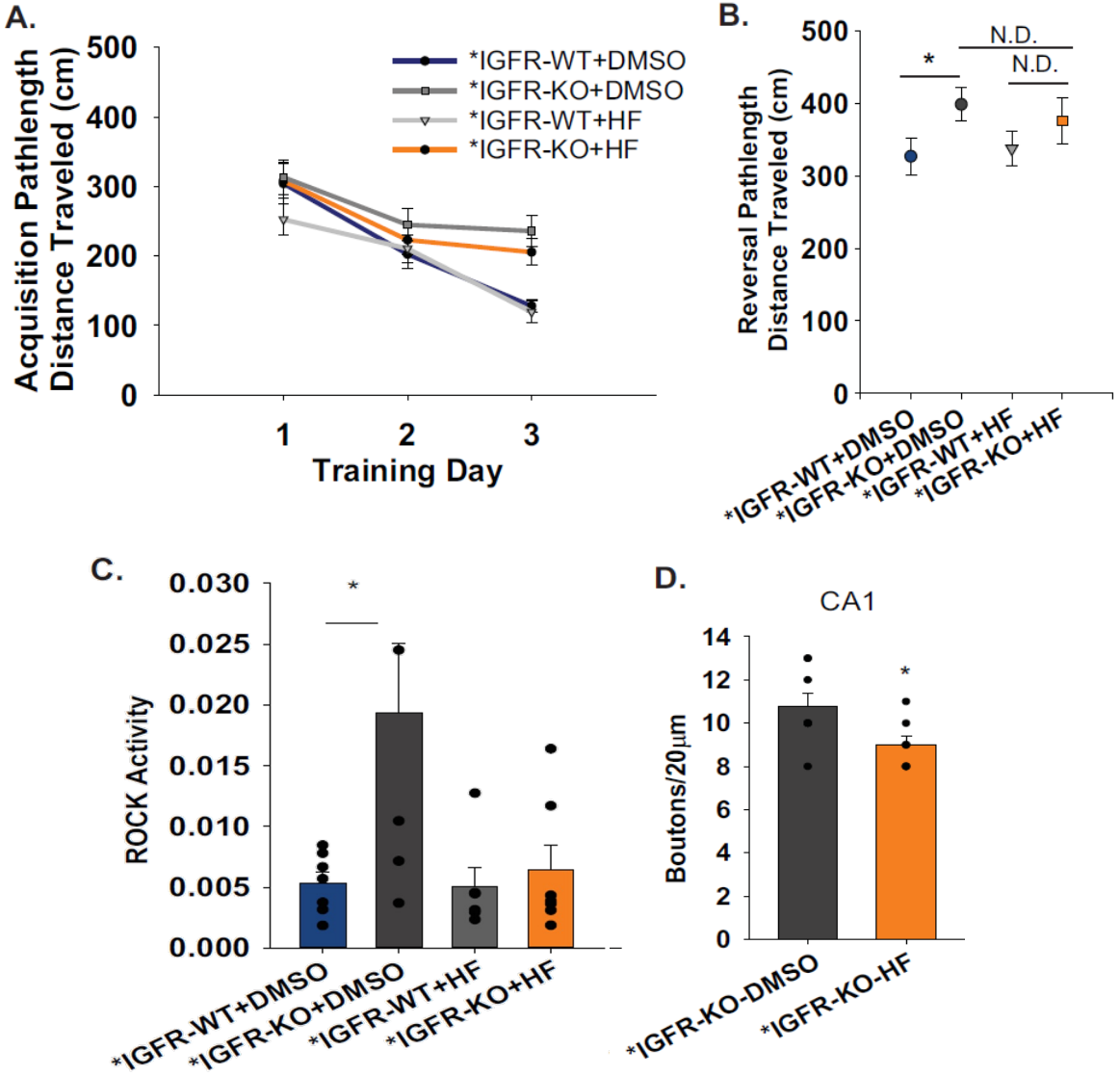


Hayes et al. 2021 bioRxiv 2021.08.08.455596

Neuronal Growth is Restored with ROCK Inhibition



Method: Primary cortical neurons treated with picropodophyllin toxin (IGFR Inhibitor) & ROCK activity assay.



Method: Male mice received a ROCK inhibitor (HydroxyFasudil) through implanted osmotic pumps and tested in RAWM. ROCK activity within the brain was quantified and boutons using Golgi's method.

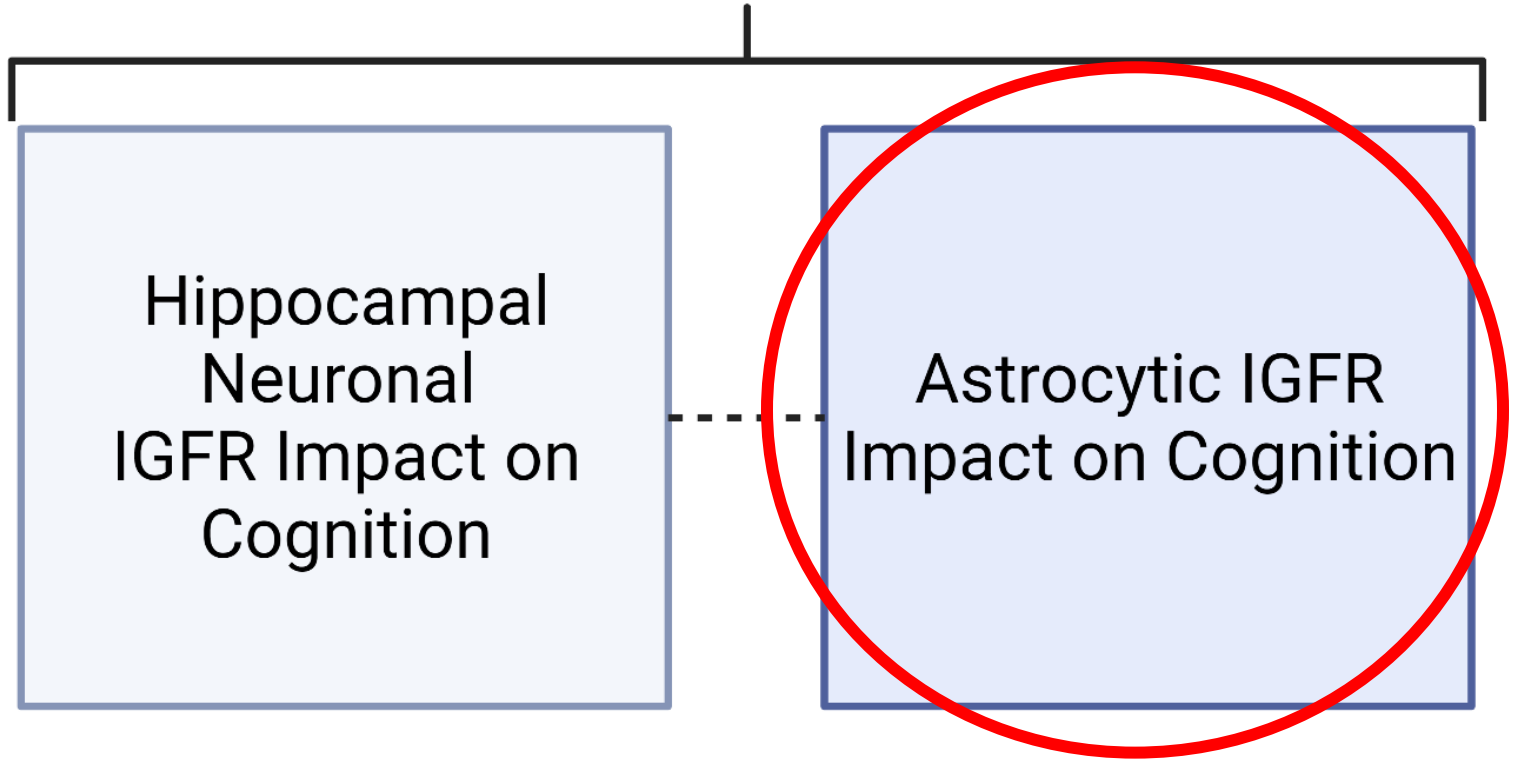
Hayes et al. 2021 bioRxiv 2021.08.08.455596

Conclusions of Neuronal IGFR Study

- Male IGFR-KOs exhibit spatial learning and memory impairments in RAWM
- IGFR reductions results in upregulated ROCK activity both in vitro and in vivo
- ROCK inhibitor in vitro restores neurite outgrowth but does NOT restore observed spatial learning and memory impairments in RAWM

Hayes et al. 2021 bioRxiv 2021.08.08.455596

Does IGFR signaling impact learning and memory in a cell-specific manner?

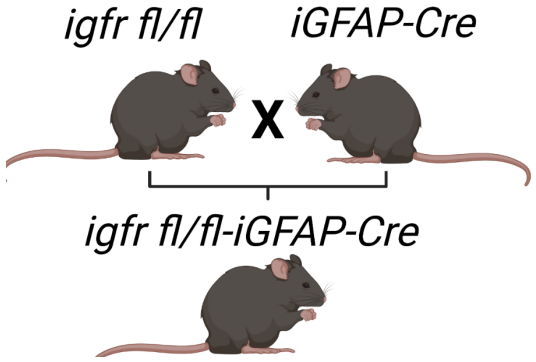


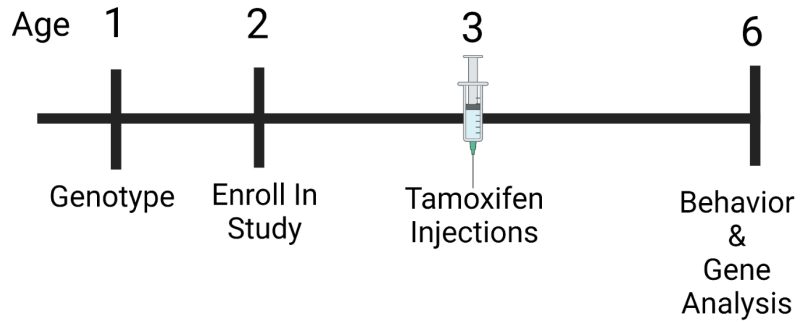
N. Ashpole

Hypothesis: Astrocytic IGF-1 is an essential component in maintaining proper cognition throughout adulthood.

Methods:

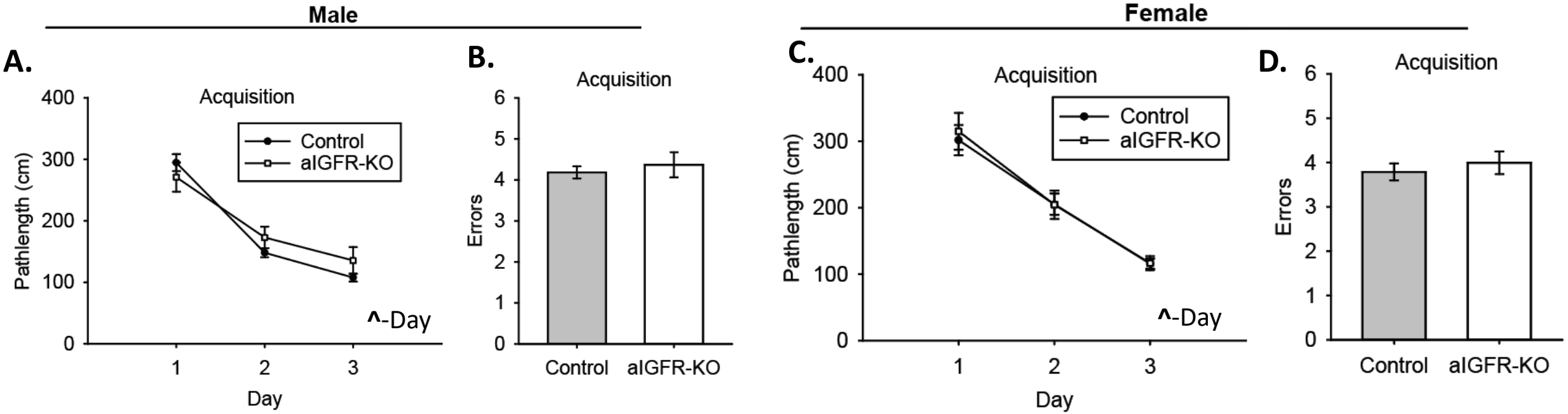
- Male and female transgenic mice
  - GFAP-Cre/ERT+ *igfr*$^{f/f}$ and *igfr*$^{f/f}$ (Control)
- Tamoxifen injections to induce KO
- Behavioral assessments:
  - RAWM
  - Barnes Maze





Astro-KO does not alter cognition

Method: Two months post-knockdown, male and female 6-month-old mice were tested in RAWM.

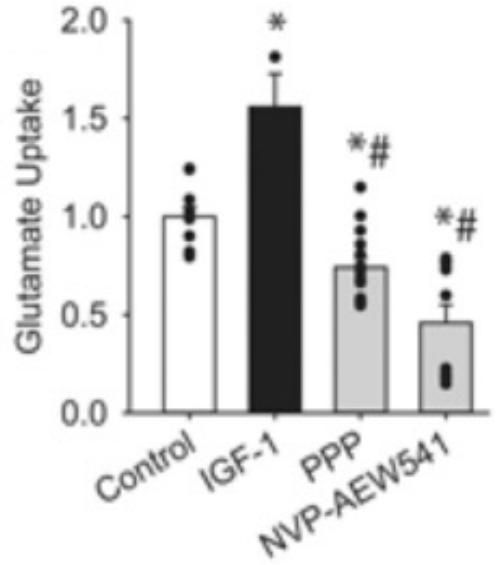


Findings: IGF-1R Inhibition disrupts glutamate uptake in vitro and in vivo

In Vitro

In Vivo

A.



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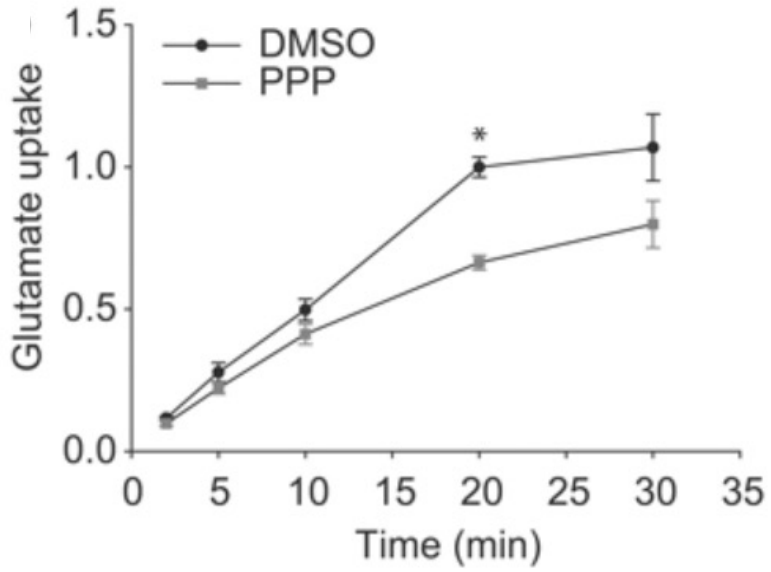
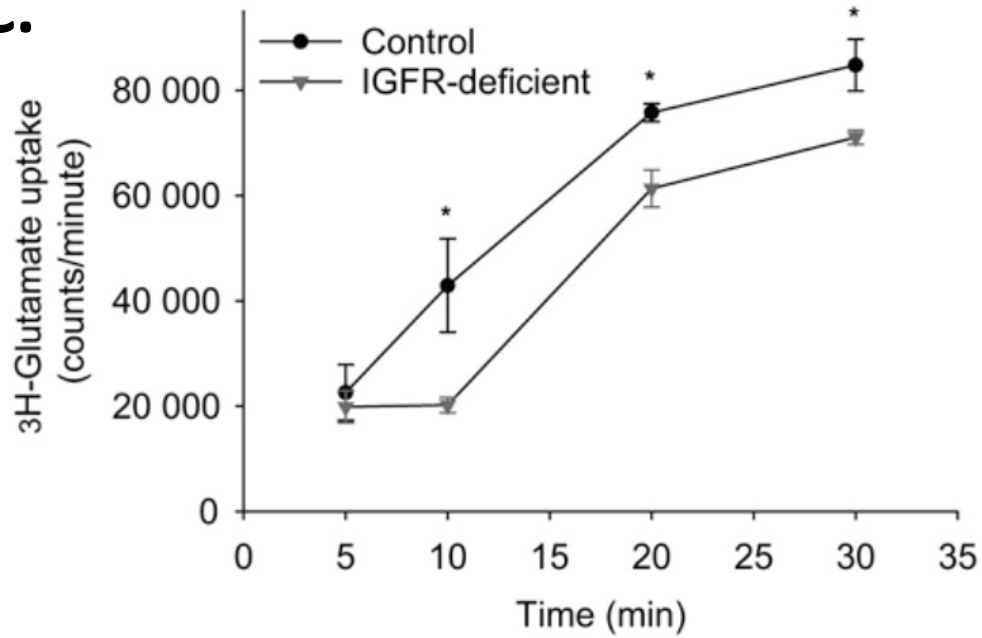


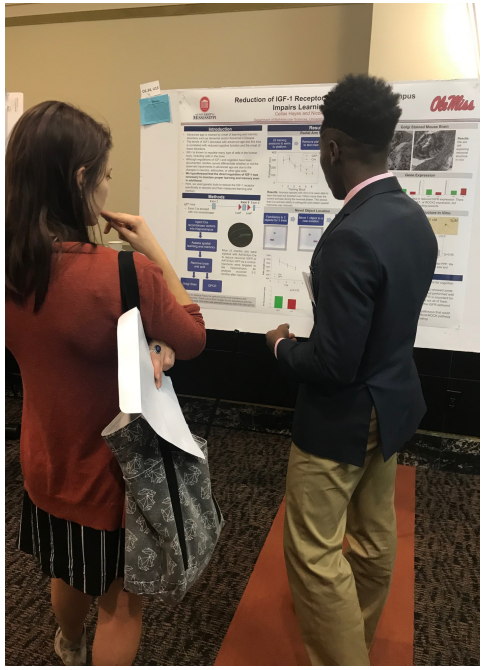
C.

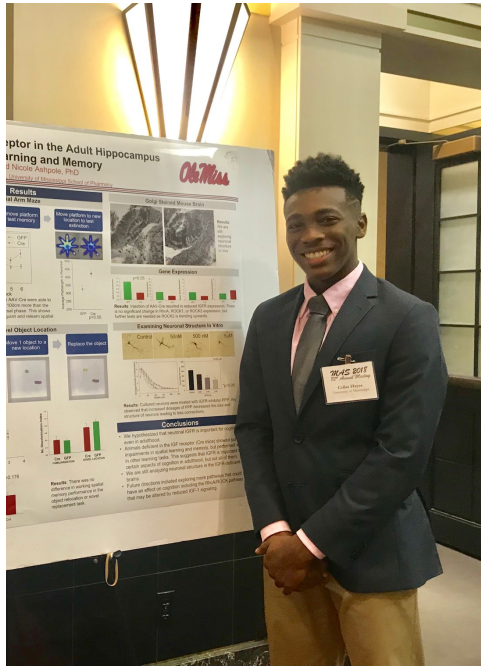


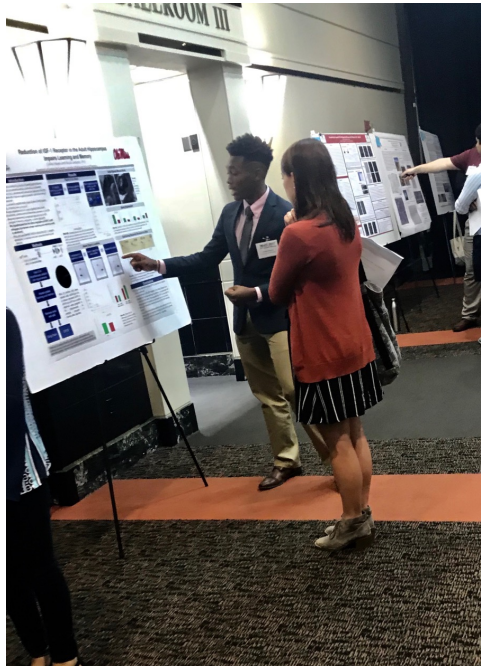
Prabhu, D et al (2019). *Journal of neurochemistry*, *151*(6), 689-702.

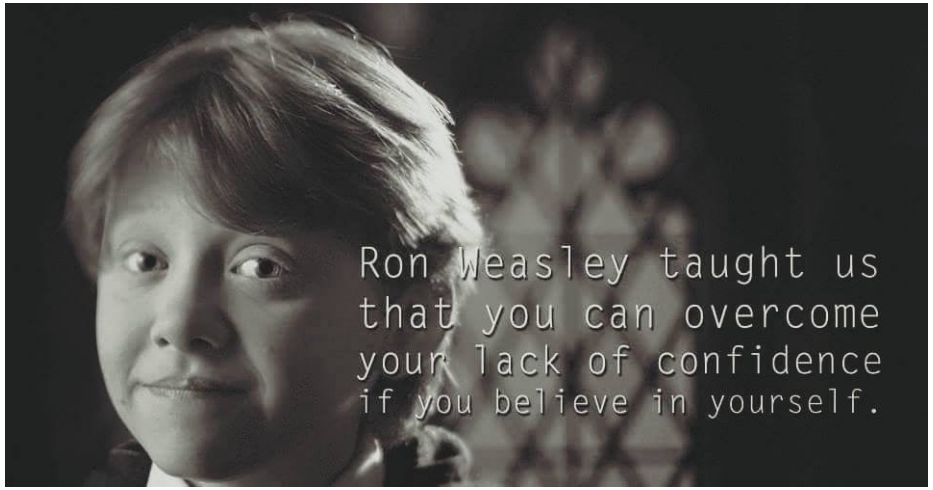
Astro-IGFR Consensus

- Neither males or females KO have cognitive impairments in either RAWM or Barnes Maze
- Reduced glutamate uptake when IGFR is inhibited in vitro and in vivo
- Glutamate machinery is altered in male and female KOs





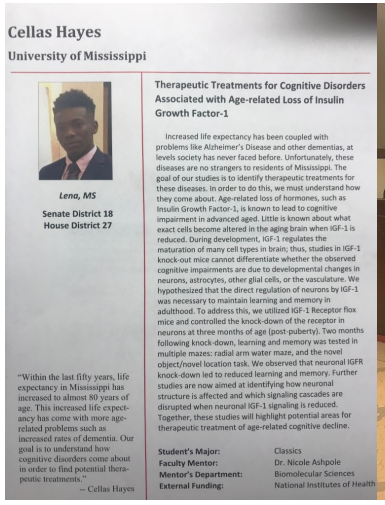




Sci-Comm

Dr. Celtas A. Hayes

Sci-Comm


Cellas Hayes

University of Mississippi

Lena, MS

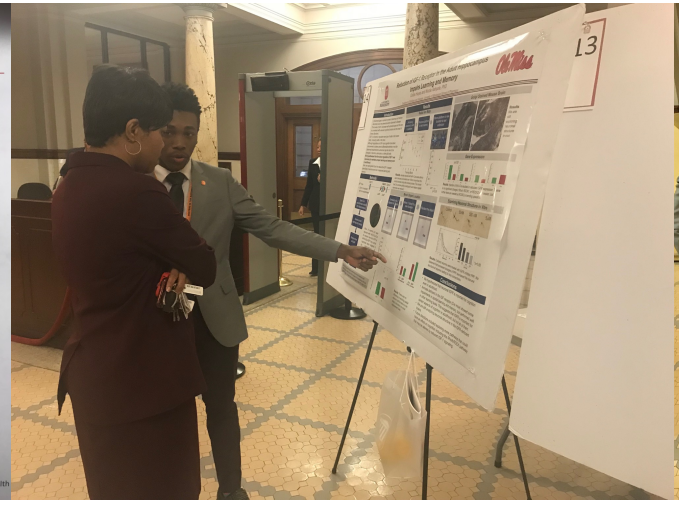
Senate District 18
House District 27

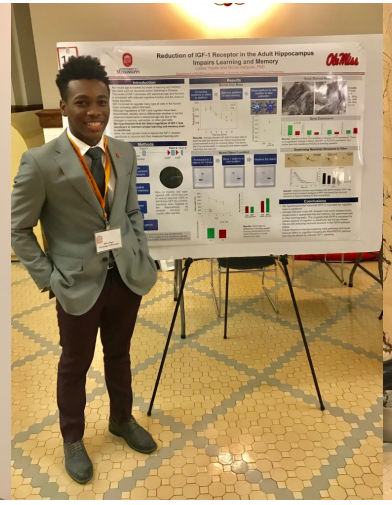
"Within the last fifty years, life expectancy in Mississippi has increased to almost 80 years of age. This increased life expectancy has come with more age-related problems such as increased rates of dementia. Our goal is to understand how cognitive disorders come about in order to find potential therapeutic treatments."
-- Cellas Hayes

Therapeutic Treatments for Cognitive Disorders Associated with Age-related Loss of Insulin Growth Factor-1

Increased life expectancy has been coupled with problems like Alzheimer's Disease and other dementias, at levels society has never faced before. Unfortunately, these diseases are no strangers to residents of Mississippi. The goal of our studies is to identify therapeutic treatments for these diseases. In order to do this, we must understand how they come about. Age-related loss of hormones, such as Insulin Growth Factor-1, is known to lead to cognitive impairment in advanced aged. Little is known about what exact cells become altered in the aging brain when IGF-1 is reduced. During development, IGF-1 regulates the maturation of many cell types in brain; thus, studies in IGF-1 knock-out mice cannot differentiate whether the observed cognitive impairments are due to developmental changes in neurons, astrocytes, other glial cells, or the vasculature. We hypothesized that the direct regulation of neurons by IGF-1 was necessary to maintain learning and memory in adulthood. To address this, we utilized IGF-1 Receptor flox mice and controlled the knock-down of the receptor in neurons at three months of age (post-puberty). Two months following knock-down, learning and memory was tested in multiple mazes: radial arm water maze, and the novel object/novel location task. We observed that neuronal IGFR knock-down led to reduced learning and memory. Further studies are now aimed at identifying how neuronal structure is affected and which signaling cascades are disrupted when neuronal IGF-1 signaling is reduced. Together, these studies will highlight potential areas for therapeutic treatment of age-related cognitive decline.

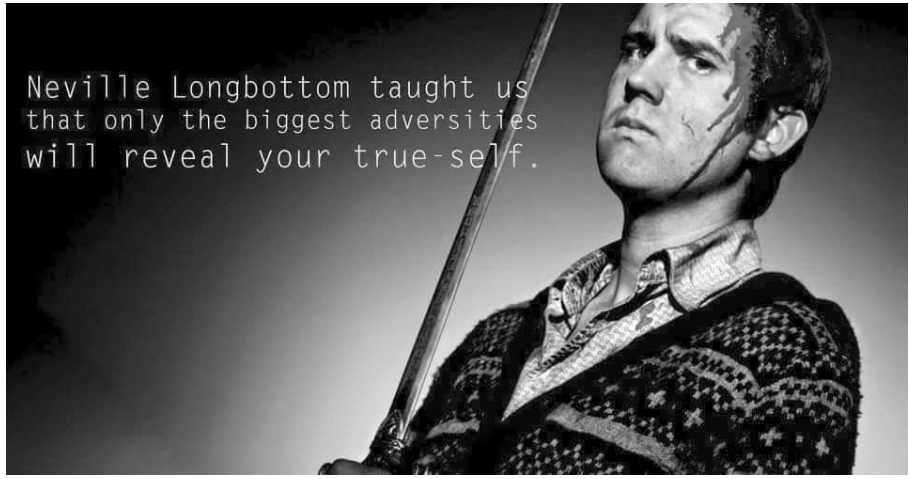
Student's Major: Classics
Faculty Mentor: Dr. Nicole Ashpole
Mentor's Department: Biomolecular Sciences
External Funding: National Institutes of Health





Dr. Cellas A. Hayes

The Graduate School Snap Shot



Required Course Work

Fall: Bisc 504, Phcl 661, BMS 601
Spring: Psy 511, Phcl 662, Bisc 579
Summer: Phcl 797

Fall: BMS 797, BMS 643, Phcl 675, Grad 600, Bms 602
Spring: BMS 641, BMS 797
Summer: Phcl 797

Advance to Candidacy

MBL SPINES

Fall: BMS 605, Phcl 797
Spring: Phcl 797, Phcl 612

Research Prospectus

Fall: Phcl 797, Phcl 611

Secure post-doc

Year 1 | Year 2 | Year 3 | Year 4 | Year 5

AIM 1

AIM 2

Southern Regional Educational Board Scholar

Smith Scholar & NSP Associate

Completed Neuro-IGFR Project

Published IGF-1/Stroke Lit. Review

Published Editorial

Complete hs-CRP/Stroke JHS Project

Submit Modulation of IGFR & Astro-IGFR Manuscripts

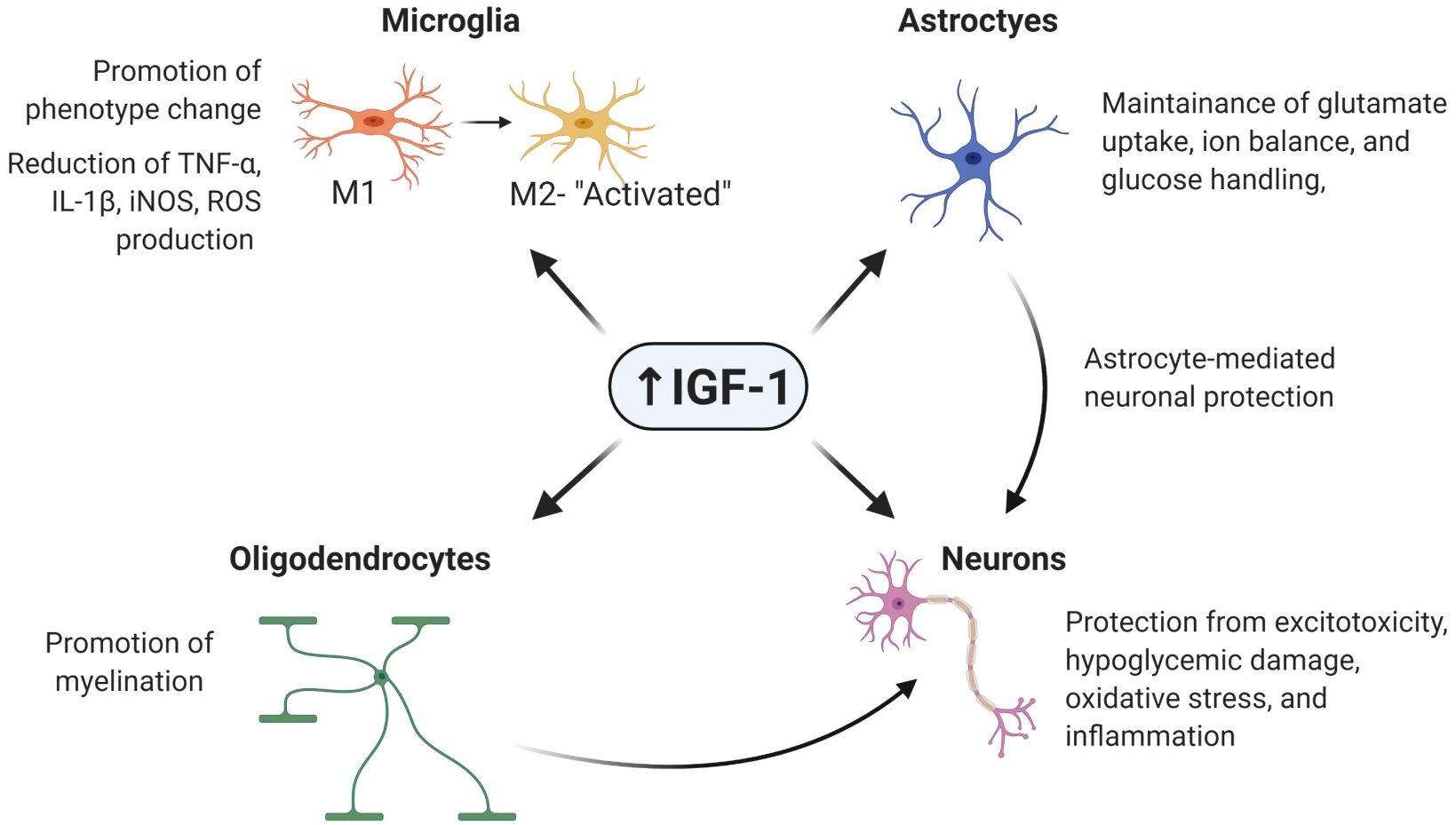
Submit Aim 1 Manuscript

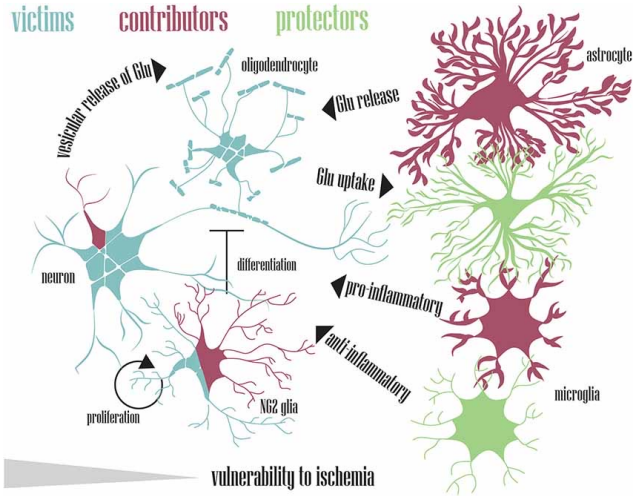
Complete AGE/RAGE Project

Submit Aim 2 Manuscript

Write&Defend Dissertation

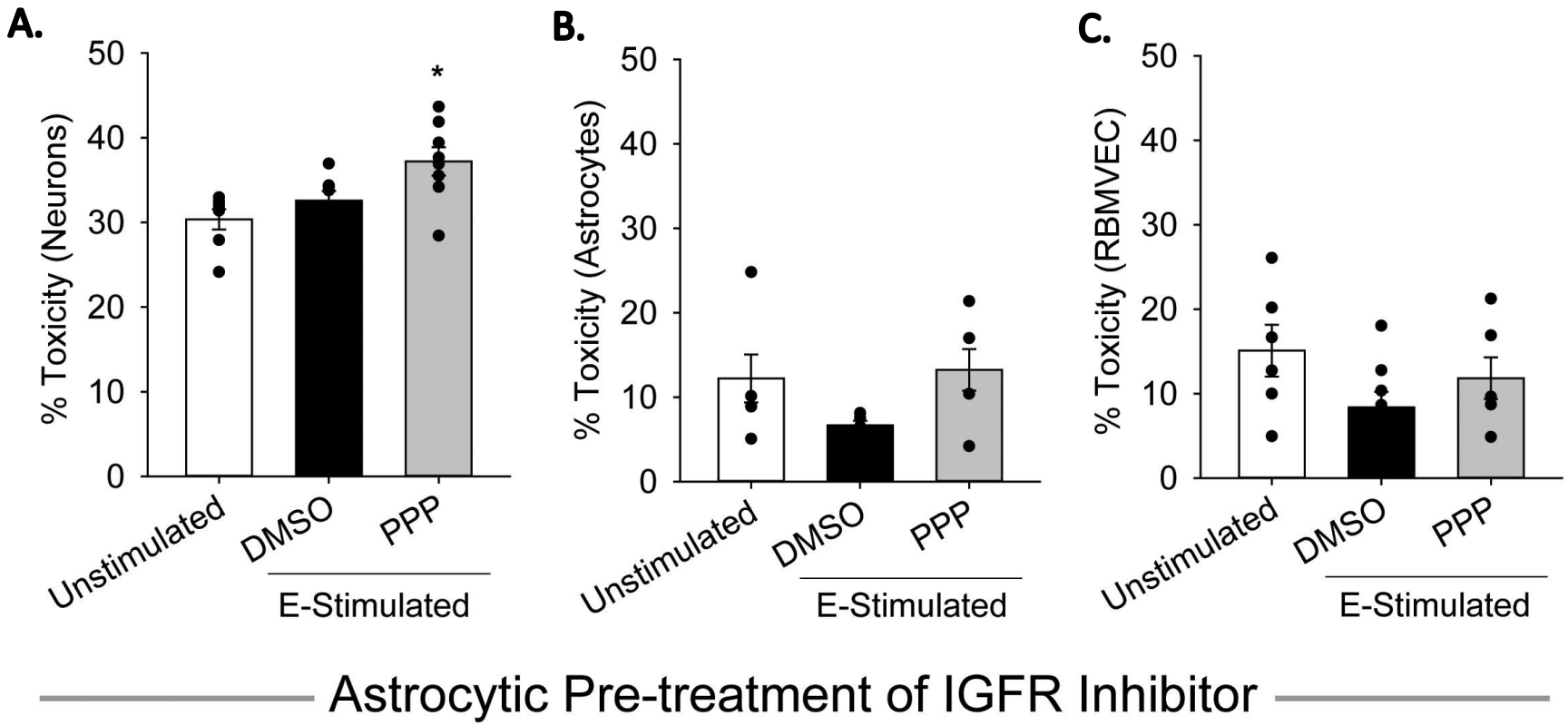
IGF-1 Signaling Within the Brain





Hayes CA. et al (2021) *Journal of Cerebral Blood Flow and Metabolism* pp. 2475-2491
https://www.frontiersin.org/articles/10.3389/fncel.2020.00051/full

Results: Astrocytes fail to protect against excitotoxicity when IGFR is inhibited.

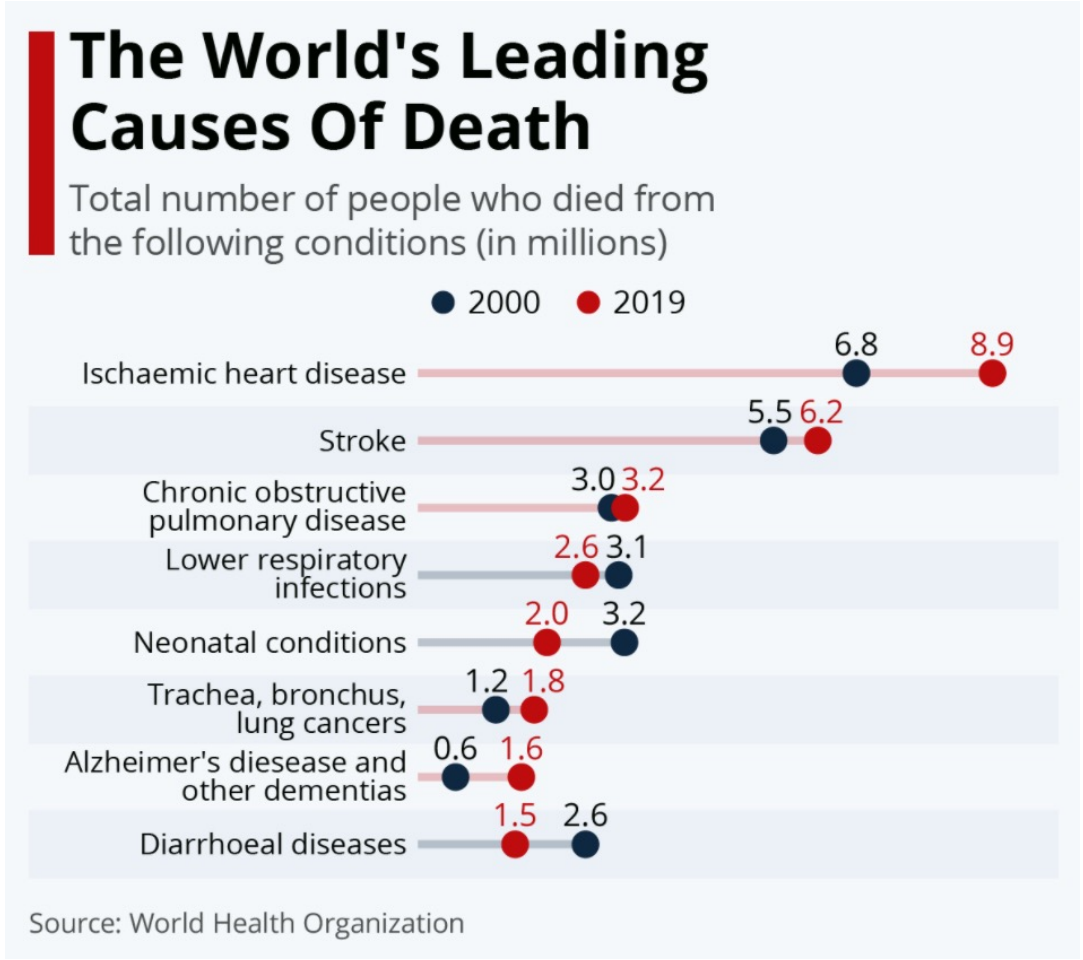


Method: Triple cell culture; Live-dead assay using fluorescence microscopy
Statistics: $p<0.05$ via one-way ANOVA
Equation: % Toxicity = (dead/total cells)*100
Abbreviations: PPP-picropodophyllin toxin; E-stim-100µM Glutamate

Hayes CA. et al (2021) *Frontiers in Aging Neuroscience* Volume 13

Stroke-A World Leading Cause of Death

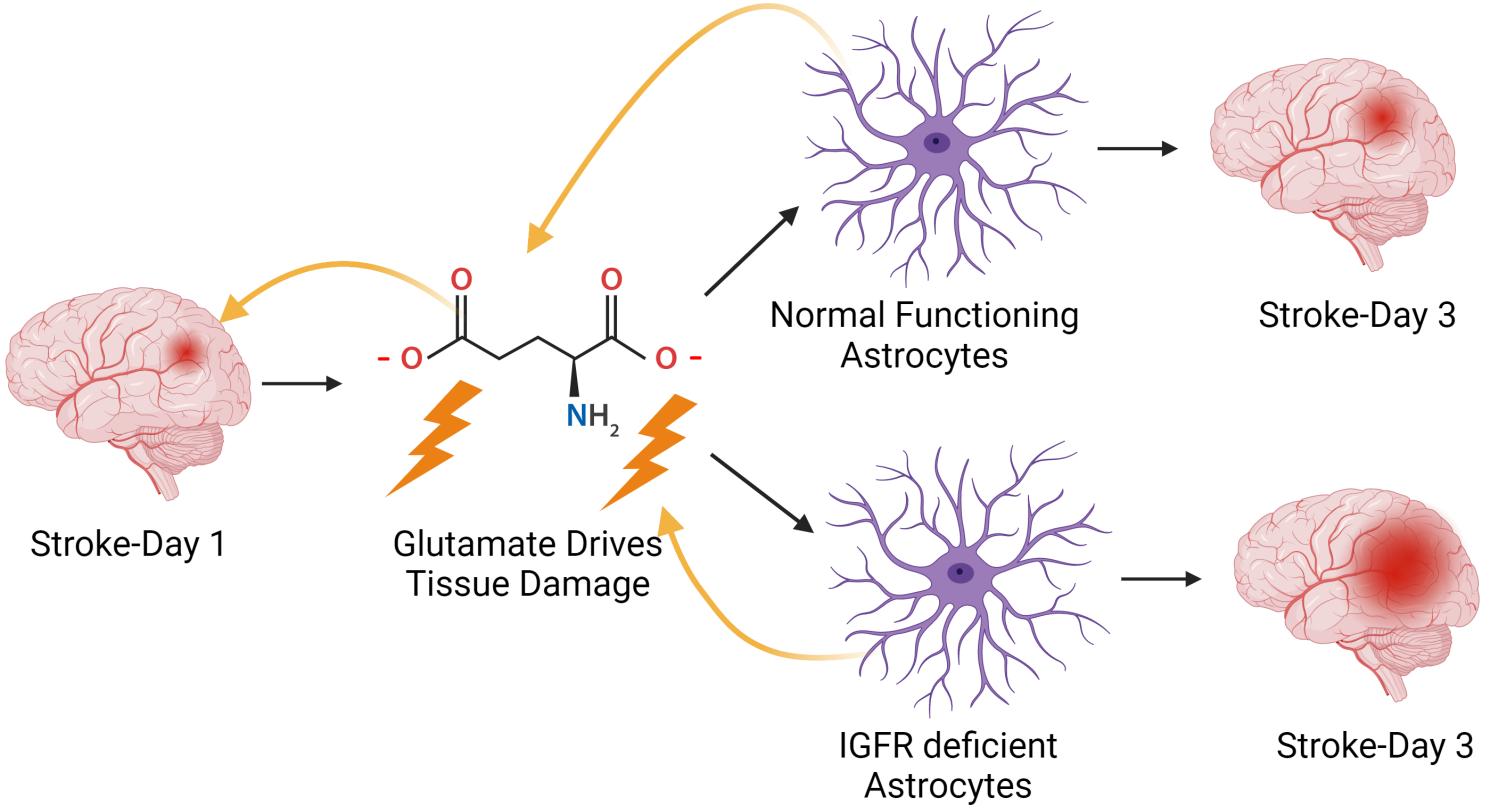
- 2020-**1 in 6 deaths**
- Every **40 seconds** someone in the United States has a stroke
- Every **3.5 minutes**, someone dies of stroke
- 2017-2018-**$53 billion** dollars in care



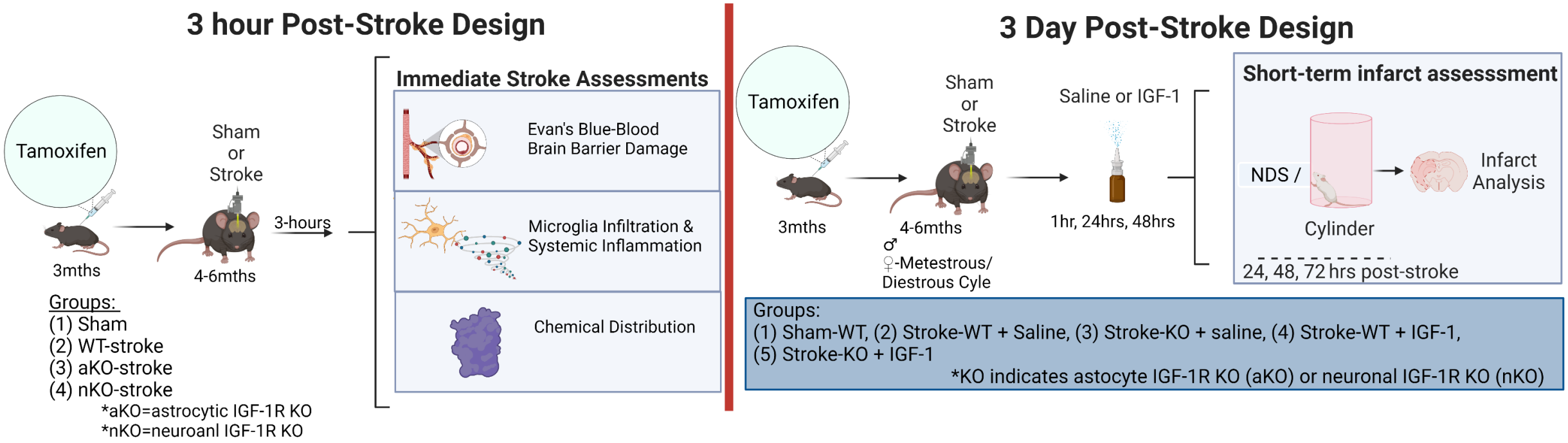
N. Ashpole

Centers for Disease Control and Prevention. Underlying Cause of Death, 1999–2018.
CW, Aday AW et al. Heart Disease and Stroke Statistics—2022 Update:
A Report From the American Heart Association external icon. *Circulation*. 2022;145(8):e153–e639.

<u>Hypothesis:</u> Reductions in IGF-1R in astrocytes attenuate neuroprotective functions of IGF-1 in stroke.



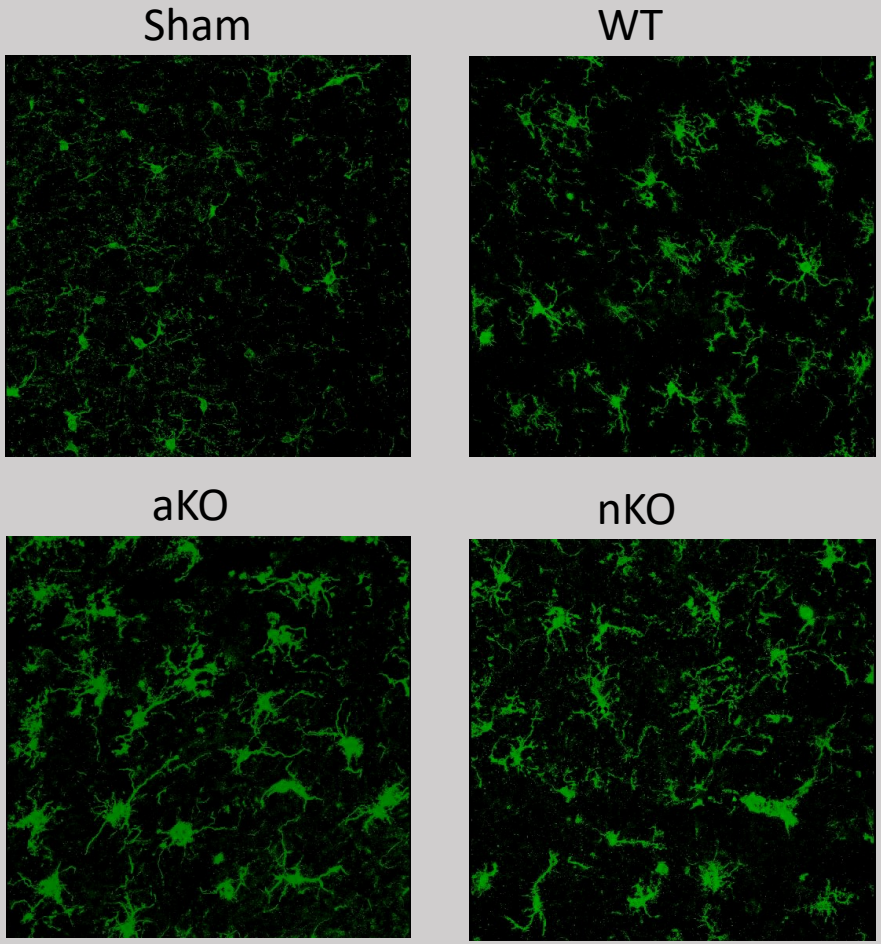
Dissertation Research Design

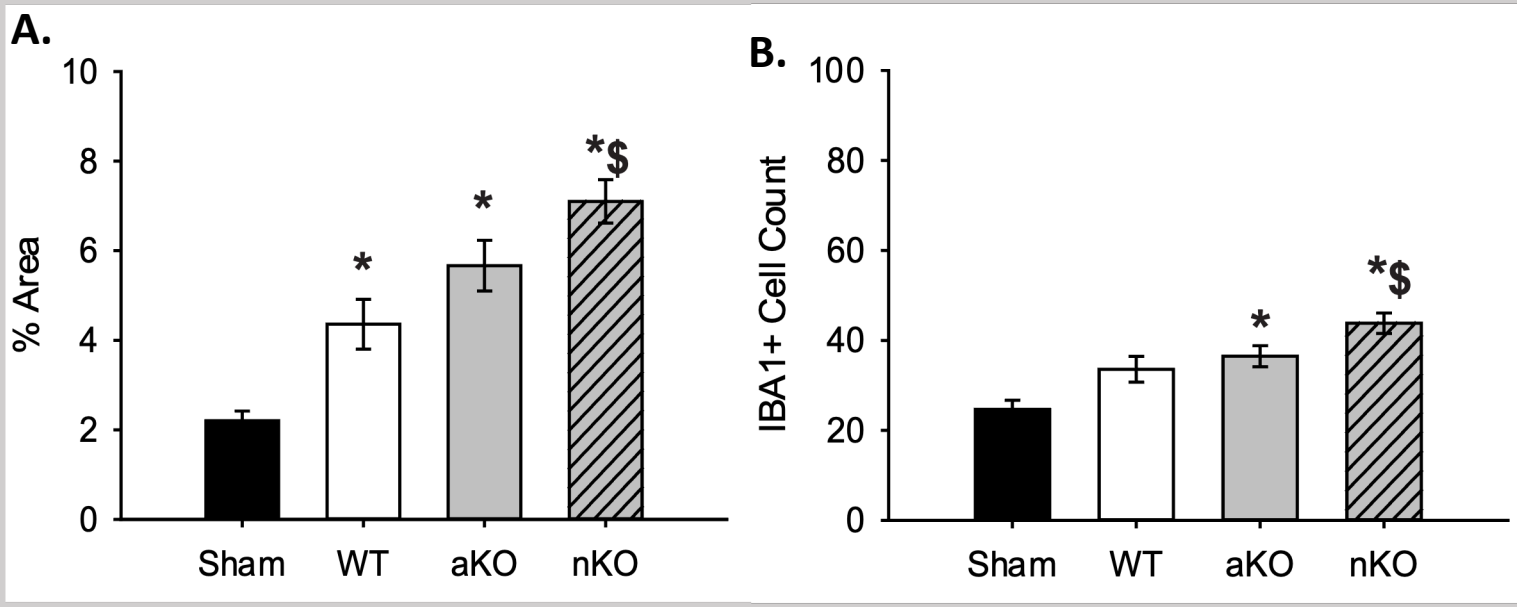


Methods:

- Transgenic Models; Photothrombosis
- Behavior: Neurological Deficit Score and Cylinder
- Ex Vivo: Blood Brain Barrier Damage (Evan's Blue/ Fluorescence Microscopy); Microglia Infiltration (IHC IBA1+/Confocal Microscopy; Inflammation (Cytokine/Chemokine Multiplex/FM3D); Chemical Shifts (Fourier Transform Infrared Spectroscopy (FTIR))

Results: Microglia Infiltration Into the Infarct Core

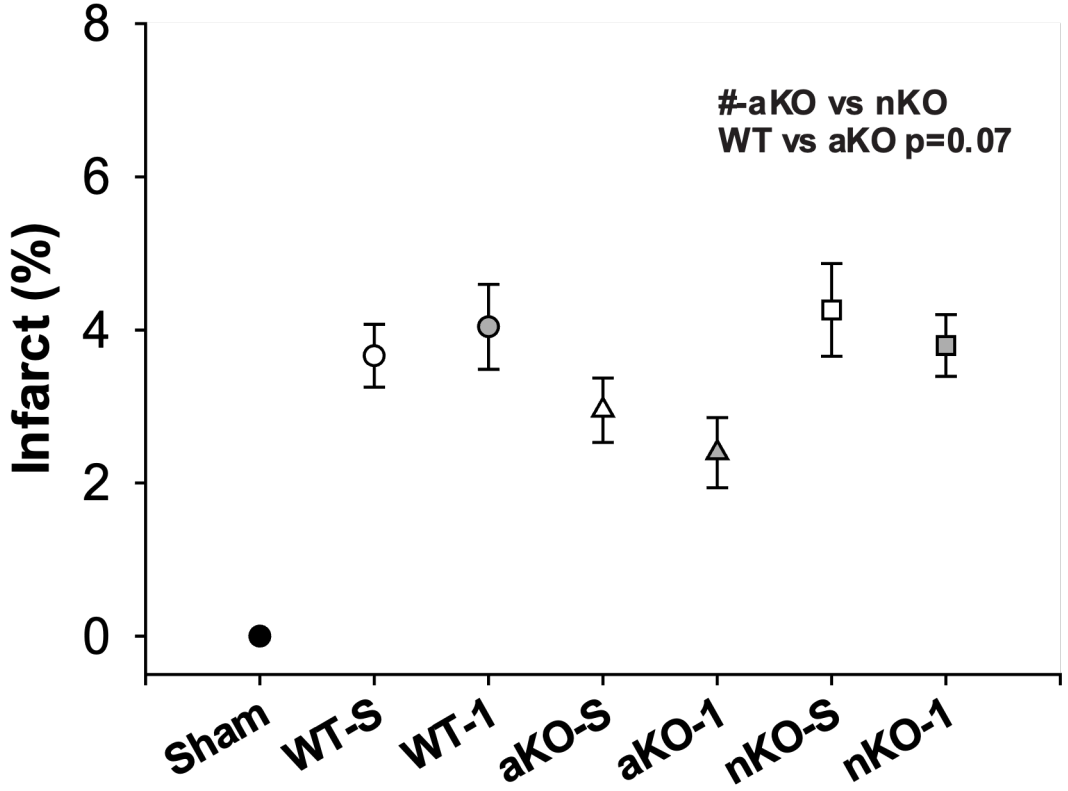




Method: IHC IBA1+; confocal microscopy; Image J/Fiji
Statistics: * $p<0.05$ compared to surgical sham; $ $p<0.05$ compared to WT ischemic stroke via one-way ANOVA
Abbreviations: *WT*-wildtype; *aKO*-astrocytic IGF-1R knockout; *nKO*-neuronal IGF-1R knockout

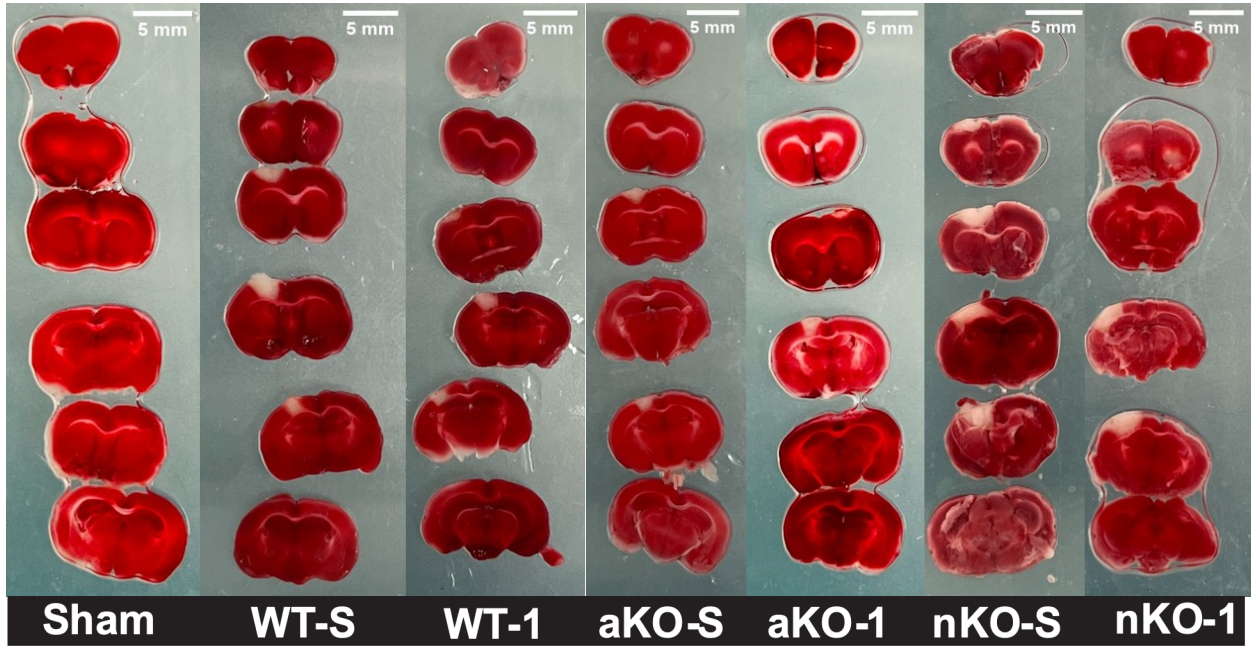
Results: Astrocytic IGF-1R Reduction Exerts Neuroprotection

A.



Method: TTC & Image J
Statistics: #-p<0.05 group differences via two-way ANOVA

B.



WT-wildtype; *aKO*-astrocytic IGF-1R knockout; *nKO*-neuronal IGF-1R knockout; *S*-intranasal saline treatment; *1*-intranasal IGF-1 treatment

Overall Findings

- aKOs and nKOs have neuroprotective phenotypes at 3 hours post-stroke
  - BBB
  - Lipid/Protein distribution
  - Microglia
  - Systemic Inflammation
- aKOs and nKOs have neuroprotective phenotypes at 3 days post-stroke
  - Infarct size

SREB AND SMITH SCHOLARS

SREB-State Doctoral Scholars Program

Increasing Faculty Diversity

More than one-third of America's college students are people of color. But racial and ethnic minorities make up only small fractions of college faculty. Nationwide, 6 percent of faculty are African-American or Black, just over 5 percent are Hispanic and about 1 percent are Native American. *The SREB-State Doctoral Scholars Program is working to change that.*

The goal: more minority Ph.D. students who seek careers as faculty on college campuses

CREATING A HEALTHIER MISSISSIPPI THROUGH RESEARCH

Welcome

2020 Robert Smith, M.D. Graduate Scholars

Alexcia Shanise Carr
Pharmacy
University of Mississippi

Torrye R. Evans II
Medicine
University of Mississippi Medical Center

Cellas Ari'ka Hayes
Pharmaceutical Sciences/ Pharmacology
University of Mississippi

Maria A. Jones-Muhammad
Neuroscience
University of Mississippi Medical Center

Rashun Jamal Miles
Social Welfare
University of Mississippi

Nicole Kaitlyn Reeder
Food Science, Nutrition, and Health Promotion
Mississippi State University

Jamarius Paul Waller
Pharmacology
University of Mississippi Medical Center

Roxanne Alecia Watts
Counseling Psychology
University of Southern Mississippi

2020 Robert Smith, MD, Graduate Scholars

The Jackson Heart Study Graduate Training and Education Center at UMMC is a two-year research mentoring and training program funded by the National Heart. Lung and Blood Institute. Designed for doctoral and health professional students who are considering careers in cardiovascular health sciences, the program allows students to observe and participate in the research process alongside mentors from leading research institutions. Download a composite photo of the 2020 scholars here.

The Jackson Heart Study

- "The **JHS** is the largest single-site, community-based epidemiologic investigation of environmental and genetic factors associated with cardiovascular disease among African Americans ever"

-The JHS

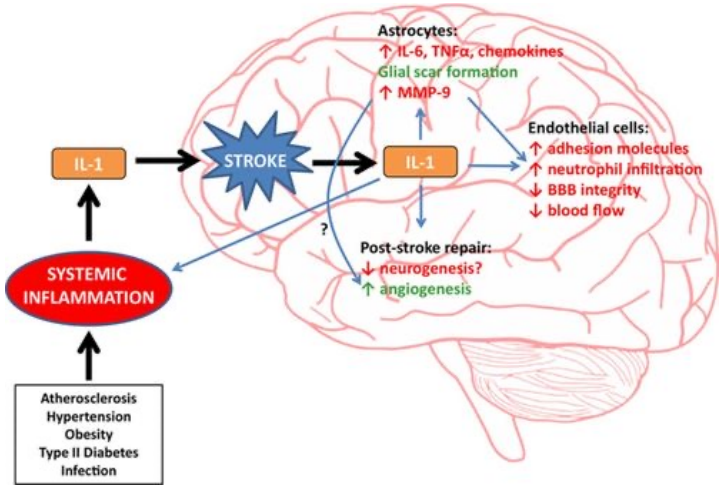
In addition, the JHS conducts community education and outreach activities to promote healthy lifestyles and reduce disease risk burden, undergraduate- and graduate-level research training programs, and high school science and math enrichment programs to prepare and encourage underrepresented minority students to pursue biomedical careers.

Measuring Inflammation in the Clinic

High Sensitivity C-Reactive Protein

- Inflammation (hs-CRP) is arguably the major contributor to ongoing stroke damage.
- Elevated hs-CRP levels correlate with the risk of ischemic stroke and stroke incidence (Rost et al 2001),(Dawood et al 2016), (Ford et al 2000).
- African Americans have a higher hs-CRP levels compared to Caucasians, Hispanic and Japanese individuals (Kelley-Hedgepeth et al 2008), (Veeranna et al 2013), (Cushman et al 2009).

Hypothesis: Increased levels of inflammation as measured by high sensitivity-C reactive protein are positively associated with stroke incidence among African Americans.



J. Renneker R. Thorpe Jr.

Sobowale, O.A., et al., *Interleukin-1 in Stroke: From Bench to Bedside.* Stroke, 2016. **47**(8): p. 2160-7

Results: hs-CRP and Stroke Incidence Association

Methodology

- Analytical sample included:
  - All 3 visits (2000-2013)
  - Hs-CRP level (independent variable) divided into quintiles
  - Stroke incidence (dependent variable)
  - Covariates
    - Demographics (age and sex)
    - Anthropometrics (body mass index (BMI) and obesity status)
    - Health conditions (high cholesterol, hypertension, and diabetes statuses)
    - Behavioral risk factors (smoking status and alcohol intake status)
    - Cardiovascular History
- Exclusion criteria:
  - Missing hs-CRP or stroke at visit 1
  - Missing stroke data at visits 1-3
  - Medical record refusals at visits 1-3

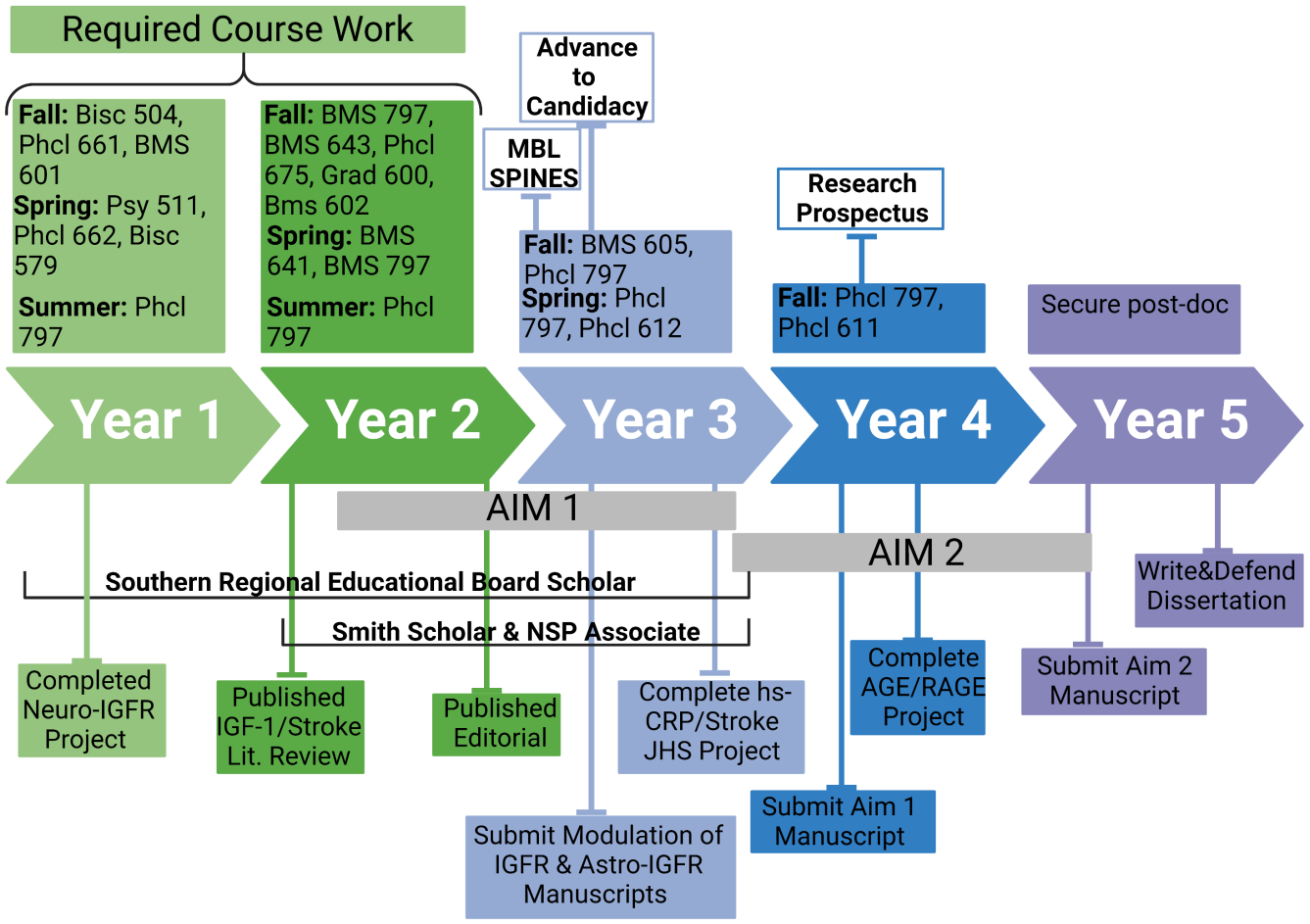
Table. Hazard ratios and 95% confidence intervals of stroke incidence among hs-CRP quintiles in the unadjusted and fully adjusted model controlling for known stroke risk factors

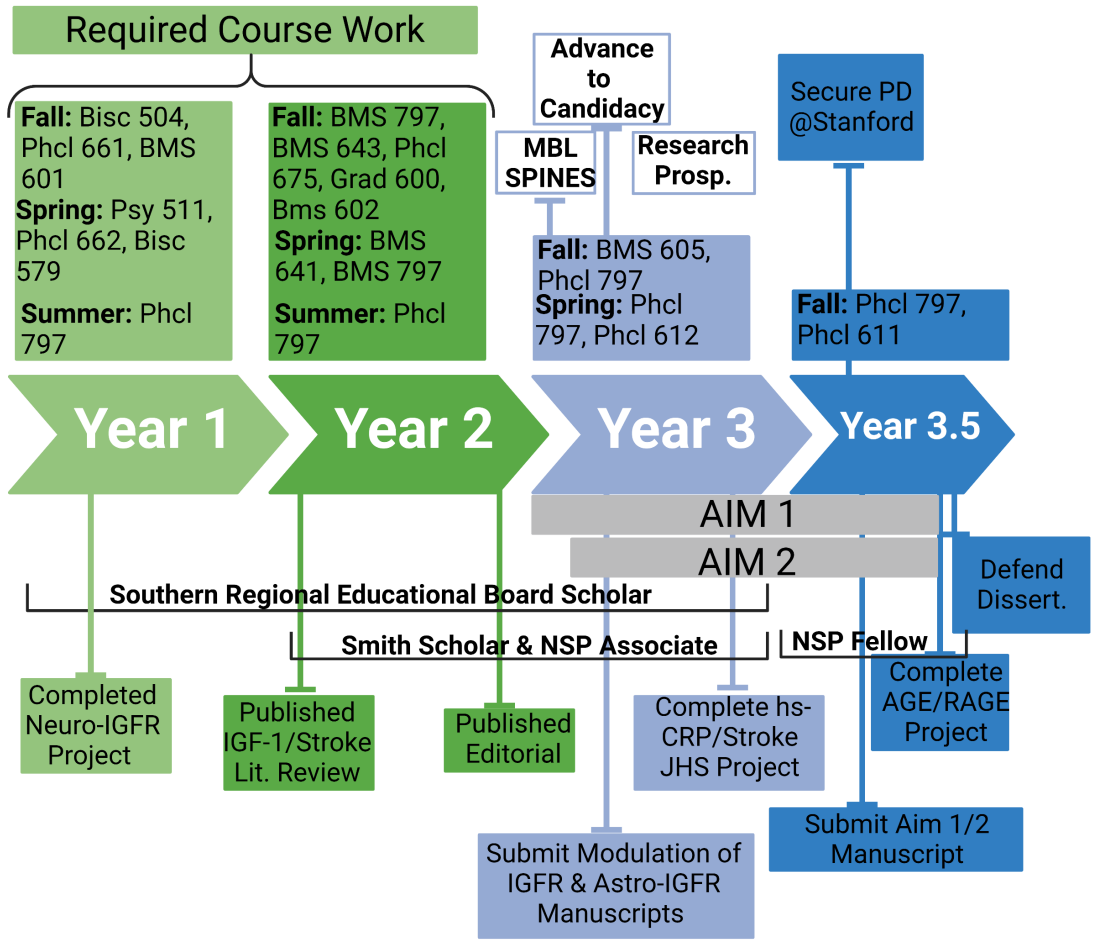
	Sequential Models	
hs-CRP quintile (mg/L)	**Unadjusted Model**	**Adjusted Model**
Quintile 1 **< 0.084 mg/L** **n=923**	1.0 (ref)	1.0 (ref)
Quintile 2 **0.085-0.189 mg/L** **n=919**	1.48 (0.96-2.29)	1.27 (0.79-2.03)
Quintile 3 **0.19-0.36 mg/L** **n=922**	1.44 (0.93-2.24)	1.18 (0.73-1.91)
Quintile 4 **0.361-0.675 mg/L** **n=918**	1.09 (0.68-1.74)	0.91 (0.55-1.52)
Quintile 5 **> 0.676 mg/L** **n=919**	**1.78 (1.17-2.72)**	**1.87 (1.17-2.98)**

Abbreviations: Highly sensitive C reactive protein - hs-CRP

Unadjusted model only included hs-CRP.
Adjusted Model hs-CRP + age, sex, obesity category, total cholesterol, hypertension status, diabetes status, alcohol status, current smoking status, ever smoking status, cardiovascular history

So… what did it actually look like for me ?





KEY ISSUES OF GRADUATE SCHOOL

Greatest lesson was time management and self-preservation through self-investment

- Pre-pandemic work hours
- Pandemic work hours
- Post-pandemic work hours
- Graduate school hours
- Postdoc hours

Theoretical Expectations? Realistic Expectations? Self Expectations

You Learn More In Failure Than You Ever Do In Success – Jay Z

Successful Grants

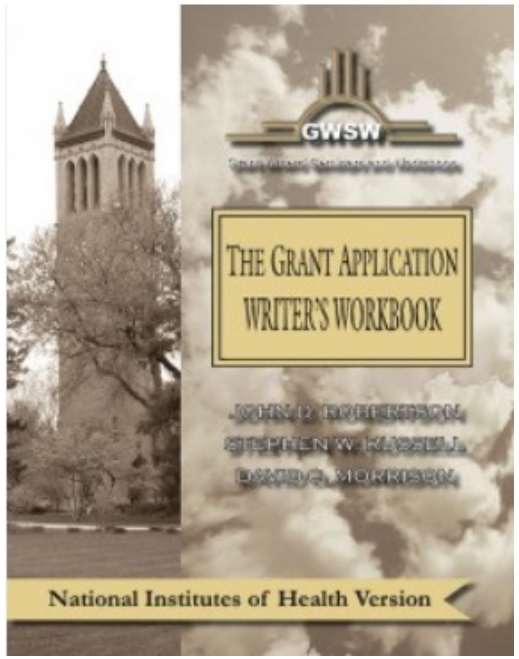
- UM Grant
- NIH NRSA F31
- Propel Stanford

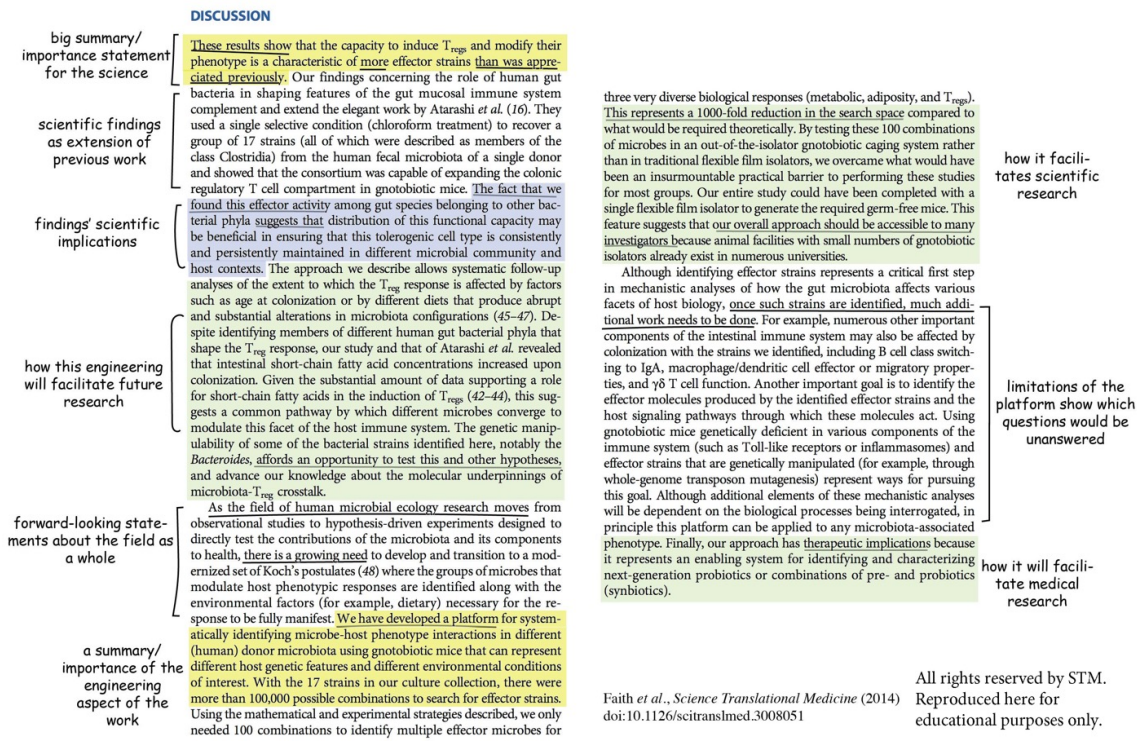
Failed Grants

- NSF GRFP
- NSF GRFP
- Ford Predoctoral Fellowship
- American Heart Association Predoctoral Fellowship
- NIH NRSA F31

2019 Strength: Science Communication
2019 Weakness: Scientific Writing

- Scientific reading takes more strategy than it does skill.
- To develop a strategy, you need practice and time.





DISCUSSION

big summary/ importance statement for the science

These results show that the capacity to induce T_{regs} and modify their phenotype is a characteristic of more effector strains than was appreciated previously. Our findings concerning the role of human gut bacteria in shaping features of the gut mucosal immune system complement and extend the elegant work by Atarashi *et al.* (*16*). They used a single selective condition (chloroform treatment) to recover a group of 17 strains (all of which were described as members of the class Clostridia) from the human fecal microbiota of a single donor and showed that the consortium was capable of expanding the colonic regulatory T cell compartment in gnotobiotic mice.

scientific findings as extension of previous work

findings' scientific implications

The fact that we found this effector activity among gut species belonging to other bacterial phyla suggests that distribution of this functional capacity may be beneficial in ensuring that this tolerogenic cell type is consistently and persistently maintained in different microbial community and host contexts. The approach we describe allows systematic follow-up analyses of the extent to which the T_{reg} response is affected by factors such as age at colonization or by different diets that produce abrupt and substantial alterations in microbiota configurations (*45*–*47*). Despite identifying members of different human gut bacterial phyla that shape the T_{reg} response, our study and that of Atarashi *et al.* revealed that intestinal short-chain fatty acid concentrations increased upon colonization. Given the substantial amount of data supporting a role for short-chain fatty acids in the induction of T_{regs} (*42*–*44*), this suggests a common pathway by which different microbes converge to modulate this facet of the host immune system. The genetic manipulability of some of the bacterial strains identified here, notably the *Bacteroides*, affords an opportunity to test this and other hypotheses, and advance our knowledge about the molecular underpinnings of microbiota-T_{reg} crosstalk.

how this engineering will facilitate future research

forward-looking statements about the field as a whole

As the field of human microbial ecology research moves from observational studies to hypothesis-driven experiments designed to directly test the contributions of the microbiota and its components to health, there is a growing need to develop and transition to a modernized set of Koch's postulates (*48*) where the groups of microbes that modulate host phenotypic responses are identified along with the environmental factors (for example, dietary) necessary for the response to be fully manifest.

a summary/ importance of the engineering aspect of the work

We have developed a platform for systematically identifying microbe-host phenotype interactions in different (human) donor microbiota using gnotobiotic mice that can represent different host genetic features and different environmental conditions of interest. With the 17 strains in our culture collection, there were more than 100,000 possible combinations to search for effector strains. Using the mathematical and experimental strategies described, we only needed 100 combinations to identify multiple effector microbes for three very diverse biological responses (metabolic, adiposity, and T_{regs}). This represents a 1000-fold reduction in the search space compared to what would be required theoretically. By testing these 100 combinations of microbes in an out-of-the-isolator gnotobiotic caging system rather than in traditional flexible film isolators, we overcame what would have been an insurmountable practical barrier to performing these studies for most groups. Our entire study could have been completed with a single flexible film isolator to generate the required germ-free mice. This feature suggests that our overall approach should be accessible to many investigators because animal facilities with small numbers of gnotobiotic isolators already exist in numerous universities.

how it facilitates scientific research

Although identifying effector strains represents a critical first step in mechanistic analyses of how the gut microbiota affects various facets of host biology, once such strains are identified, much additional work needs to be done. For example, numerous other important components of the intestinal immune system may also be affected by colonization with the strains we identified, including B cell class switching to IgA, macrophage/dendritic cell effector or migratory properties, and γδ T cell function. Another important goal is to identify the effector molecules produced by the identified effector strains and the host signaling pathways through which these molecules act. Using gnotobiotic mice genetically deficient in various components of the immune system (such as Toll-like receptors or inflammasomes) and effector strains that are genetically manipulated (for example, through whole-genome transposon mutagenesis) represent ways for pursuing this goal. Although additional elements of these mechanistic analyses will be dependent on the biological processes being interrogated, in principle this platform can be applied to any microbiota-associated phenotype. Finally, our approach has therapeutic implications because it represents an enabling system for identifying and characterizing next-generation probiotics or combinations of pre- and probiotics (synbiotics).

limitations of the platform show which questions would be unanswered

how it will facilitate medical research

Faith *et al.*, *Science Translational Medicine* (2014) doi:10.1126/scitranslmed.3008051

Then & Now

- Paper Tracking

Time Started Reading: Time Finished Reading:

Article Analysis-Title of Article: _____

Abstract summary:

Hypothesis of article:

Figures analysis:

Summary of Article and Analysis of Results:

Thomas 2021

Title: Thomas 2021
Date: 2022-12-28
Time: 18:13
NEUROPATH

Association of TDP-43 proteinopathy, cerebral amyloid angiopathy, and Lewy bodies with cognitive impairment individuals with or without Alzheimer's disease neuropathology

Authors:

David X. Thomas1,3*, Sumali Bajaj2,3, Kevin McRae-McKee2 , Christoforos Hadjichrysanthou M.Anderson2 & John Collinge1

Year: 2021

tags/ key terms

#tdp43 #caa #lbd #AD

Introduction

Objective: Compare the association of common neuropathologies with pre-mortem cognitiv decline in the presence vs absence of concomitant Alzheimer's disease.

Methods

- NACC 2005-2018 #NACC #np
- CDR-SOB & MMSE
- Bayesian Hierarchical regression models to estimate the association of Alzheimer's disea neuropathology, TDP-43 proteinopathy, cerebral amyloid angiopathy (CAA), and Lewy b with cognitive trajectories afer accounting for the covariates consisting of demographic features, and other neuropathologies
- *SIMILIAR VARIABLES AS CELLAS MANUSCRIPT PLAN AND DATA PROCESSING*
- Binary categories were created for Alzheimer's disease neuropathological change, Lewy bodies, CAA, and TDP-43 proteinopathy using cut-of values which led to reasonably balanced groups.

Statistics

#bayesianhierarchicalregression
linear mixed effect models in a Bayesian framework
#linearmixedeffectregression #chisquare

Results

In ADNC+individuals, we assessed if the rate of cognitive decline was the same in the presence absence of a co-pathology, using one model each for TDP-43, CAA and Lewy Bodies. We estimated the two way interactions of TDP-43 proteinopathy and time (βMMSE=−0.34, 95% BC (−0.64, −0.04); βCDR-SB =0.33, 95% BCI (0.13, 1.47)), CAA and time (βMMSE=−0.04, 95% BCI (−0.42, 0.35); βCDR-SB =0.09, 95% BCI (−0.11, 0.29)) and Lewy bodies and time (βMMSE=−0.29 95% BCI (−0.57, −0.01); βCDR-SB =0.19, 95% BCI (−0.01, 0.39)) (Fig. 2). These results suggest th ADNC+individuals with these pathologies have a steeper rate of cognitive decline compared to those without co-morbid pathologies.

However, our data showed no association of Lewy bodies with cognitive impairment over time ADNC- individuals.

Main Findings

ADNC+individuals with these TDP-43, CAA, and lewy bodies individually have a steeper rate of cognitive decline compared to those without co-morbid pathologies. No association of Lewy bodies with cognitive impairment over time in ADNC- individuals.

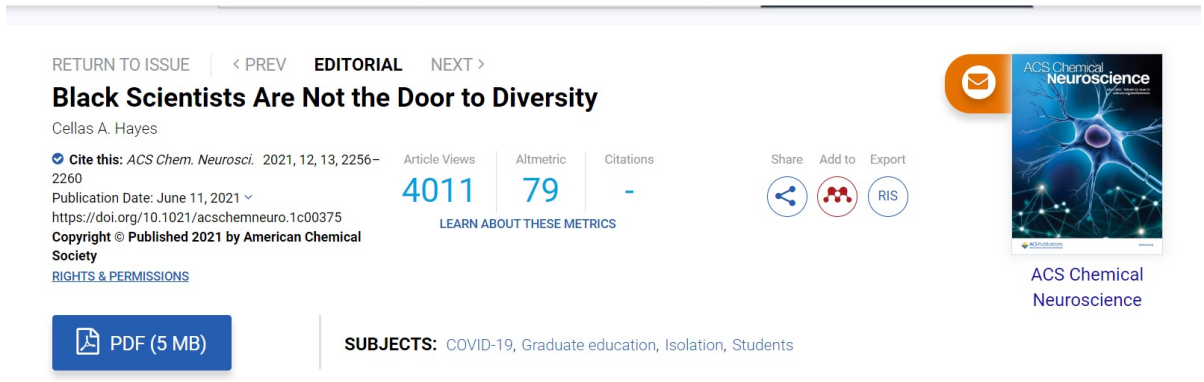
IDEAS

non-binary dichotomizing variables to capture complexity of neuropathologies.

PDF

Dr. Cellas A. Hayes

A Black Academic Journey



RETURN TO ISSUE | < PREV **EDITORIAL** NEXT >

Black Scientists Are Not the Door to Diversity

Cellas A. Hayes

Cite this: *ACS Chem. Neurosci.* 2021, 12, 13, 2256–2260

Publication Date: June 11, 2021

https://doi.org/10.1021/acschemneuro.1c00375

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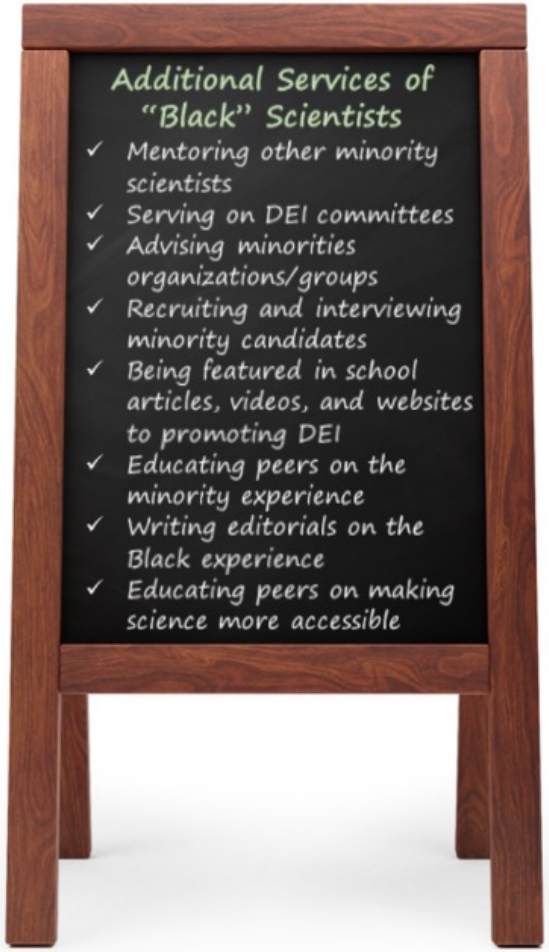
ACS Chemical Neuroscience

PDF (5 MB)

SUBJECTS: COVID-19, Graduate education, Isolation, Students

- THE UPROAR OF 2020, AN INTRODUCTION
  - A GRADUATE STUDENT PERSPECTIVE
- THE ADDITIONS OF BEING A MINORITY GRADUATE STUDENT
  - ARE BLACK STUDENTS THE BENEFICIARIES TO DEI WORK?
- THE FEELING OF OBLIGATION AND PROVING SELF-WORTH
  - BEING A SCIENTIST AND BEING A "BLACK SCIENTIST"
- BLACKNESS AS A STEPPING STONE AND THE REAL-WORLD PERSPECTIVE
  - BLACK STORIES AS TOKENS AND SPOTLIGHTS
- MAKING THE SYSTEM BETTER: A CONCLUSION AND GUIDE FOR THE FUTURE

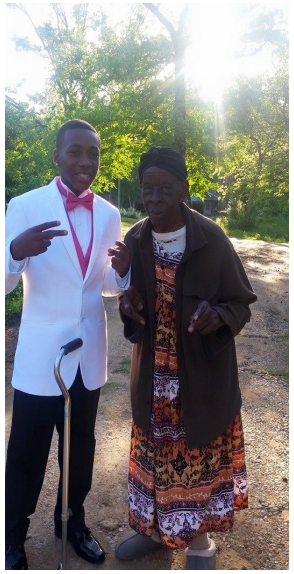


Hayes CA; *ACS Chemical Neuroscience* **2021**

Dr. Cellas A. Hayes

Postdoctoral Appointment ~ Stanford University (2022-)

Interest



The Realization

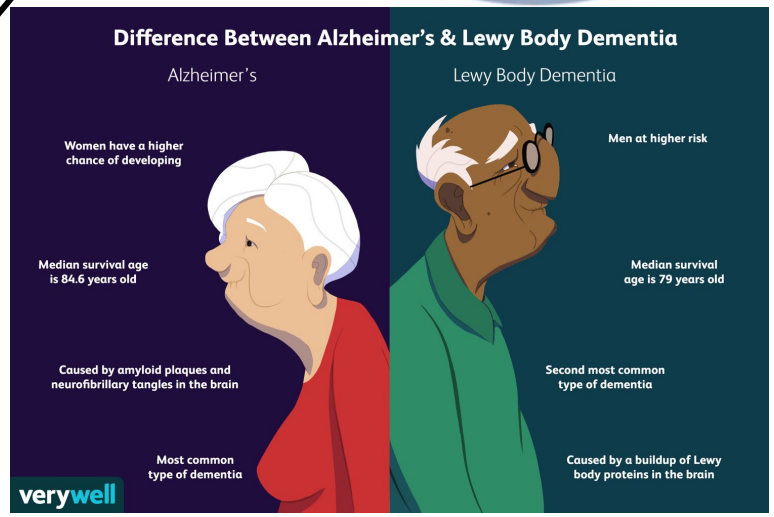
Postdoctoral Recruitment Initiative in Science and Medicine
Stanford PRISM

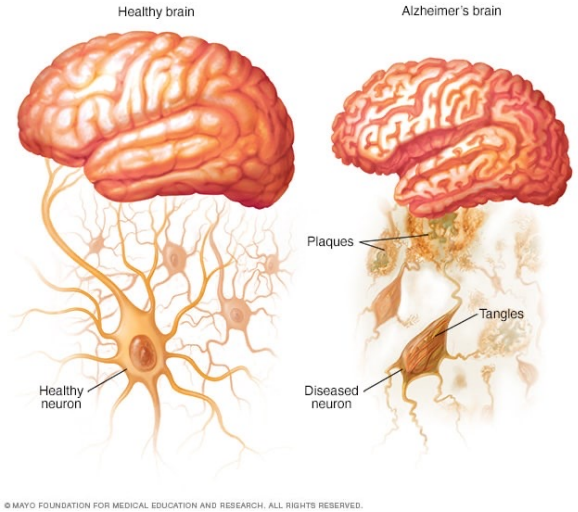
Propel.
Postdoctoral Scholars Program
https://propel.stanford.edu/

Stanford MEDICINE

Research

NACC

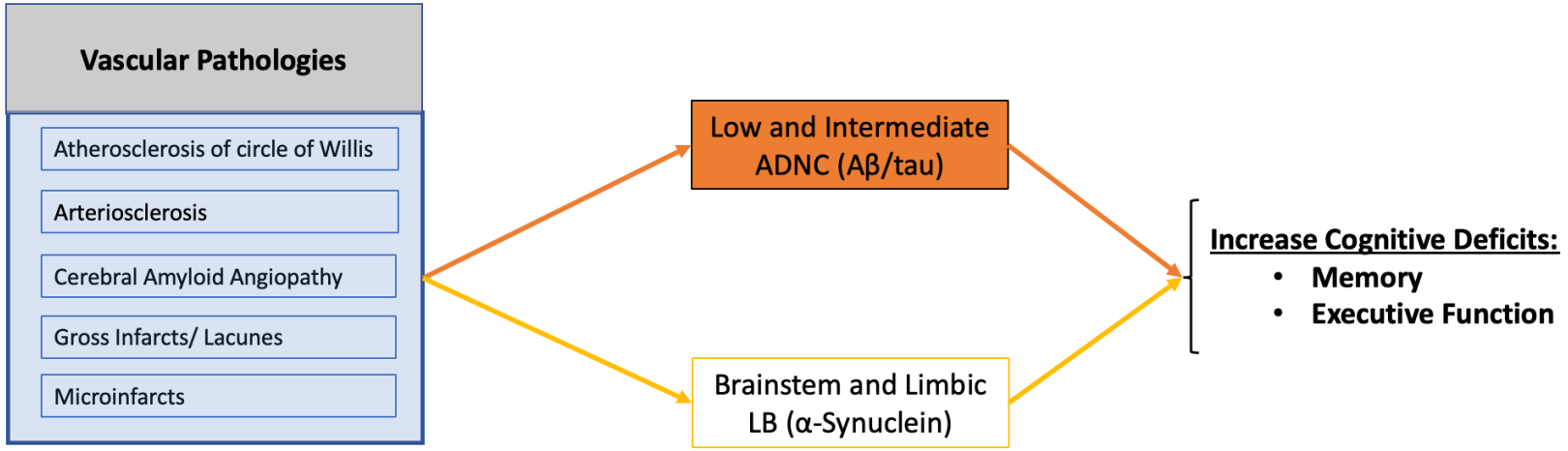




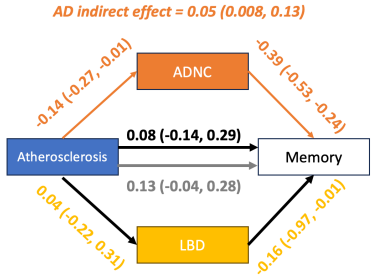
A·D·R·C
for healthy brain aging

Dr. Celias A. Hayes

National Alzheimer's Disease Coordinating Center Dataset

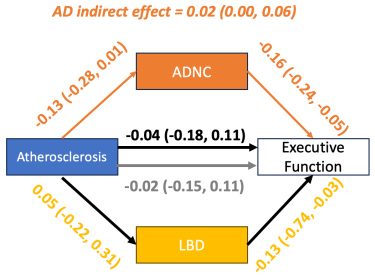


Atherosclerosis



Total indirect effects = 0.05 (-0.15, 0.23)

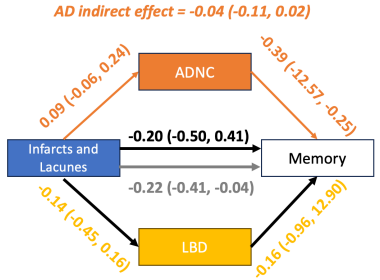
AD mediates effects on memory for ACW.



Total indirect effects = 0.01 (-0.07, 0.08)

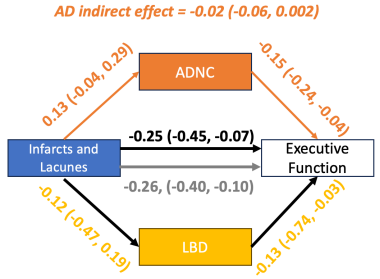
Neither AD or LBD mediates effects on executive function for ACW.

Infarcts/Lacunes



Total indirect effects = -0.01 (-0.47, 0.26)

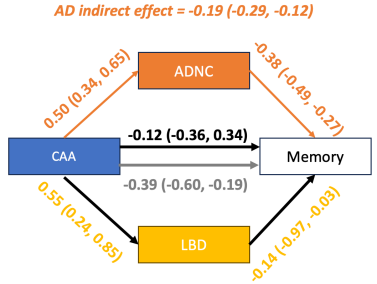
Neither AD nor LBD mediates an effect on memory for INF.



Total indirect effects = -0.003 (-0.06, 0.22)

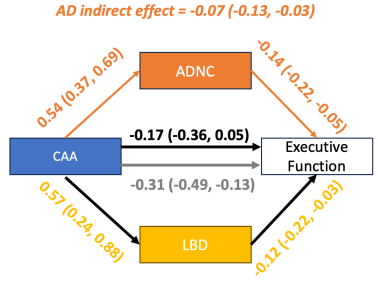
Neither AD or LBD mediates effects on executive function for INF.

Cerebral Amyloid Angiopathy



Total indirect effects = -0.27 (-0.74, -0.17)

AD and LBD mediates a lower effect of CAA on memory performance.



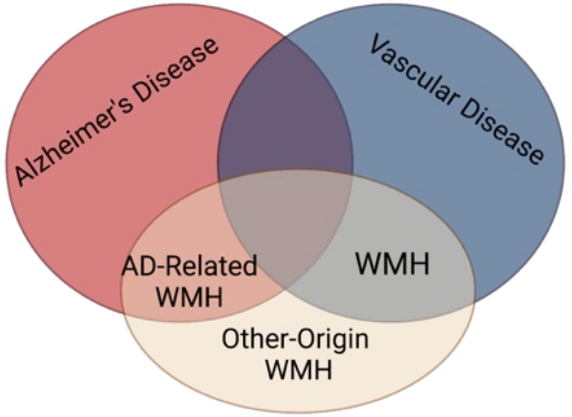
Total indirect effects = -0.14 (-0.26, -0.07)

AD and LBD mediates a lower effect of CAA on executive function.

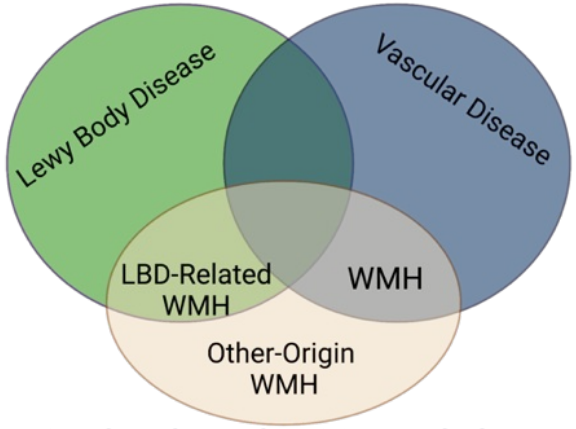
B. Mormino K. Poston K. Younes

POSTDOC RESEARCH TRAJECTORY

Aim 1a



Aim 1b

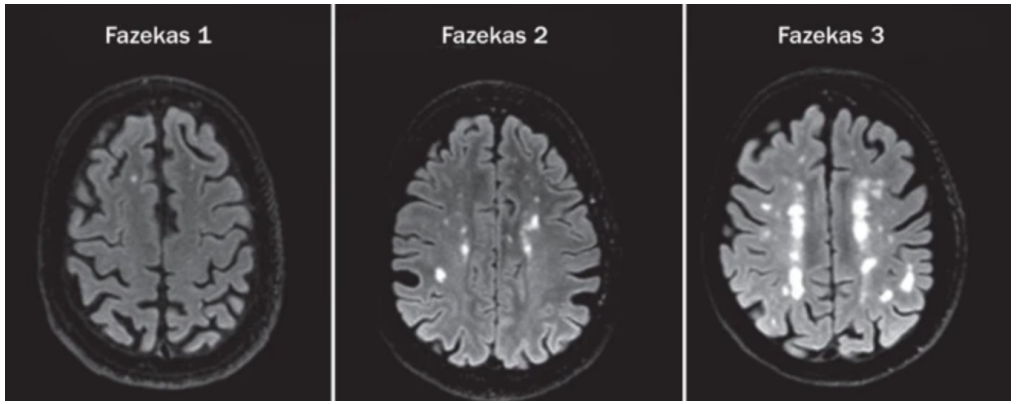


TRADITIONAL HYPOTHESIS: WMH originate and potentiate through vascular origins/ cerebral small vessel disease.

AD ALTERNATIVE HYPOTHESIS: Some WMH is moderated by AD pathology and progression.

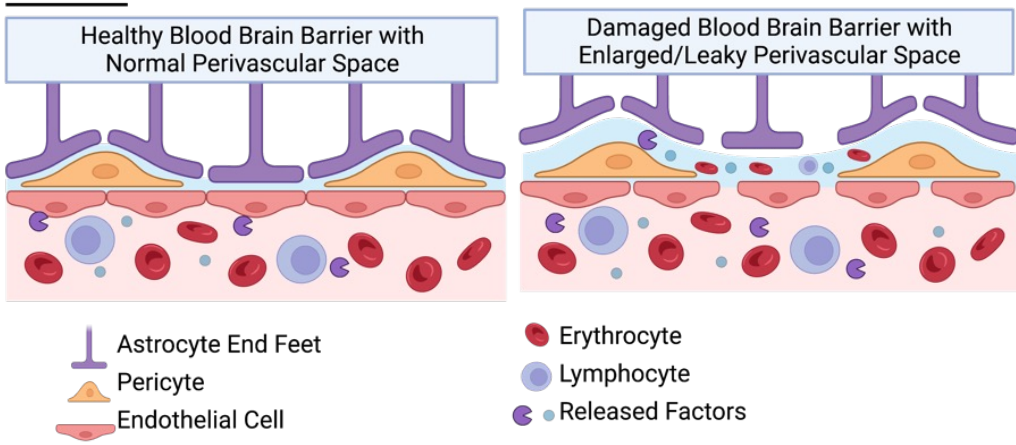
LBD ALTERNATIVE HYPOTHESIS: Some WMH is moderated by LBD pathology and progression.

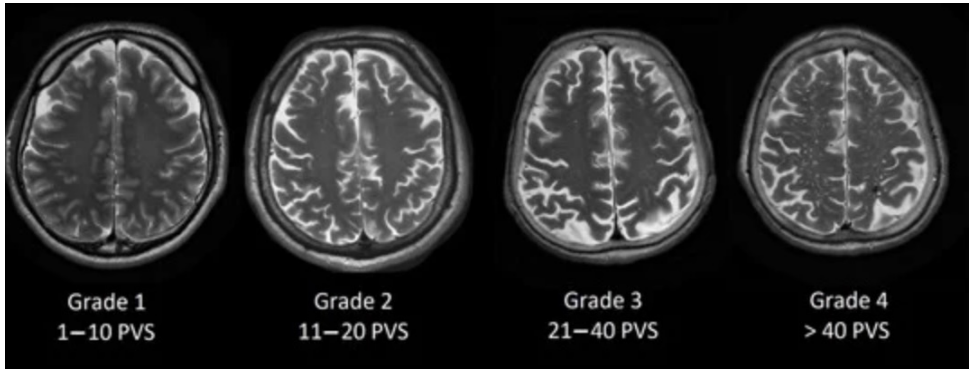
Question: Are there proteomic signatures that can be used to identify significant changes in WMH that are of vascular origin, AD-related, or LBD-related?



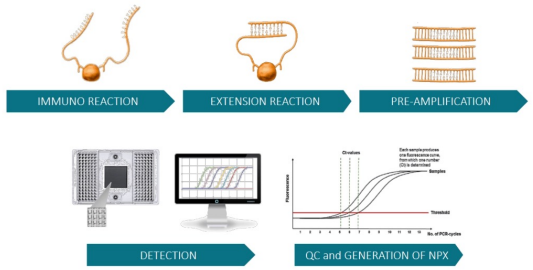
Prins, N. et al, *Nat Rev Neurol* **11**, 157–165 (2015).

Aim 2

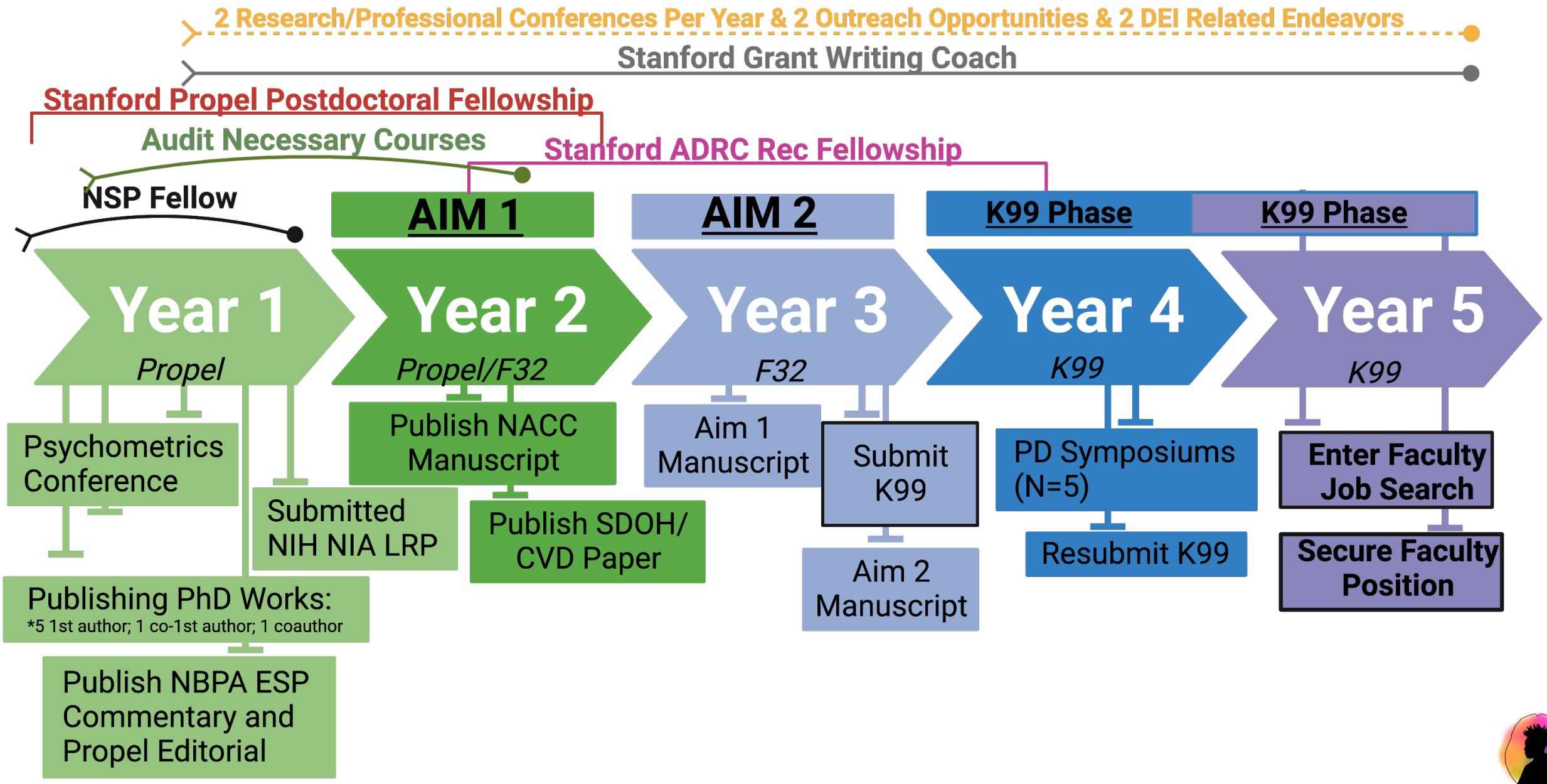




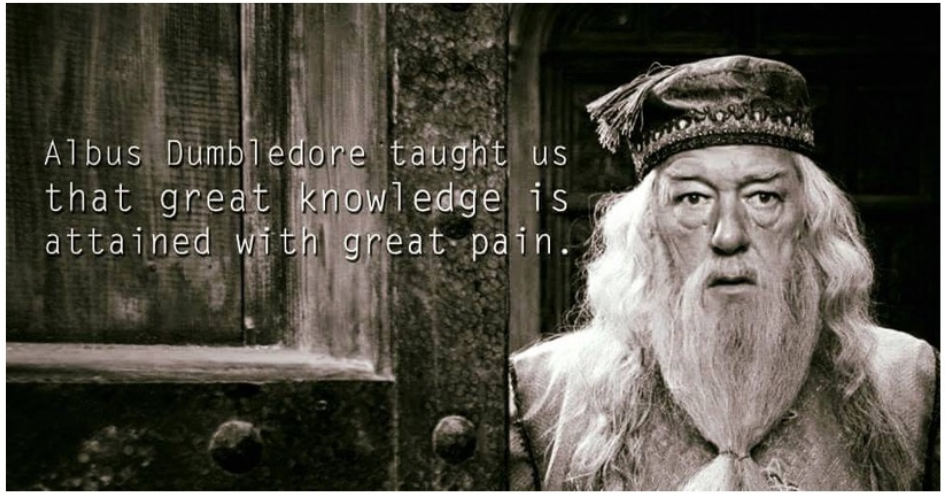
Zdanovskis, N. et al *Medicina* **2022**, *58*, 887.

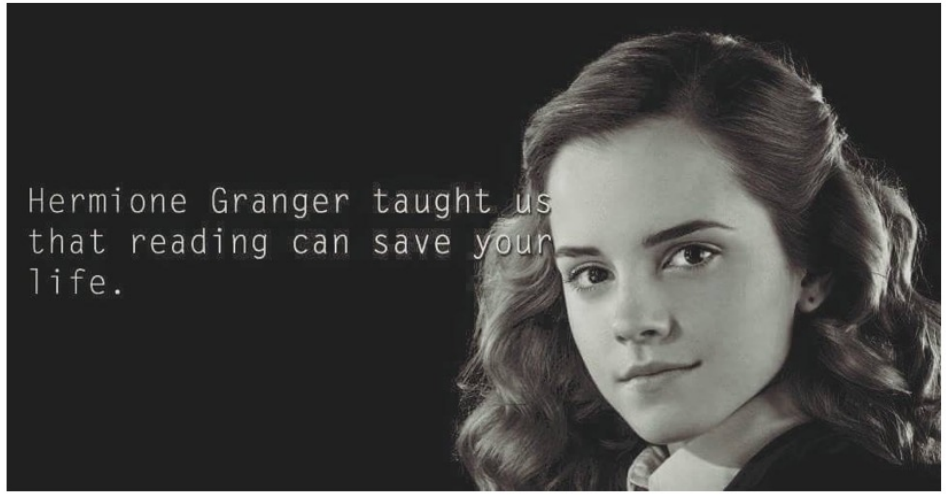


POSTDOC TRAINING PLAN



“Obstacles” = “Opportunities”





Reading, Consolidating, Asking Questions, Opportunities

BMBH Emerging Scholars Program

hhmi
Gilliam Fellowships
for Advanced Study
51 Adviser-Student pairs in 2022
401 Fellows in the community to date
$53k Per year per award
Building a community committed to advancing science through diversity and inclusion

D-SPAN
NIH Blueprint for Neuroscience Research

Apply for an oSTEM Scholarship
Applications Close Friday, June 10th, 2022
$70k in Scholarships available

NSF Graduate Research Fellowship Program

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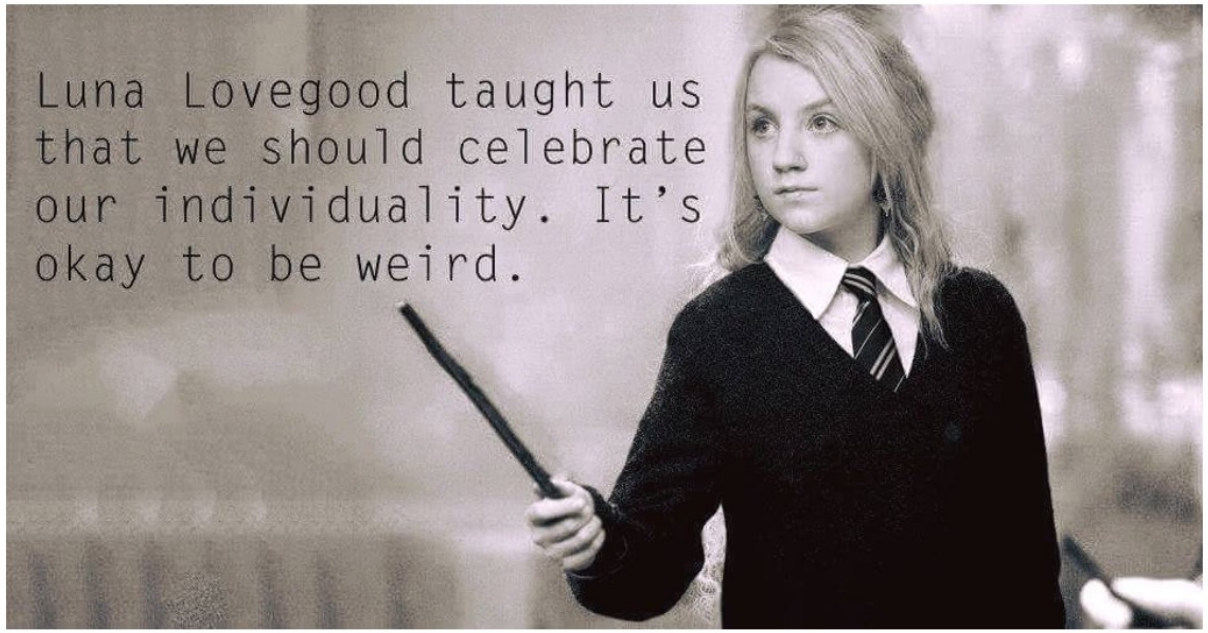
Blinq Me



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