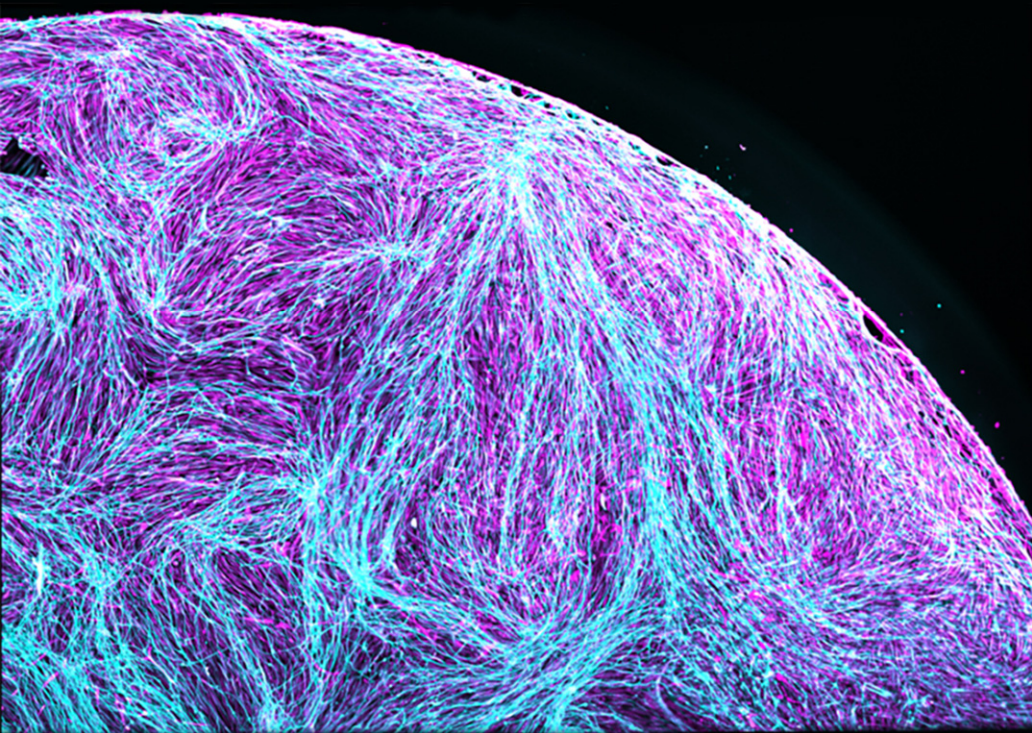


Abstracts of papers presented
at the 2020 *virtual* meeting on

NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

December 2–December 4, 2020



Cold Spring Harbor Laboratory
MEETINGS & COURSES PROGRAM

Abstracts of papers presented
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NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

December 2–December 4, 2020

Arranged by

Aaron Gitler, *Stanford University*

Richard Ransohoff, *Third Rock Ventures*

Scott Small, *Columbia University*

Li-Huei Tsai, *Massachusetts Institute of Technology*



Cold Spring Harbor Laboratory

MEETINGS & COURSES PROGRAM

Support for this meeting was provided in part by the **National Institute on Aging**, a branch of the **National Institutes of Health**; **Chan Zuckerberg Initiative (CZI)**, **IRBO/International Brain Research Organization**; **Regeneron**; and **Stem Cell Technologies**.

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NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

Virtual Meeting

Wednesday, December 2 – Friday, December 4, 2020

| | | |
|--------------------|-------------------|--|
| Wednesday | 10:00 am–11:00 am | Keynote Speaker: Virginia Lee |
| Wednesday | 11:00 am–1:00 pm | 1 Genetics, Genomics and Target Identification in Neurodegenerative Disease |
| Wednesday | 2:00 pm–5:00 pm | 2 Neuroinflammation and Glial Biology of Neurodegeneration |
| Thursday | 10:00 am–1:00 pm | 3 Therapeutic Initiatives in Neurodegenerative Disease |
| Thursday | 2:00 pm–5:00 pm | 4 ApoE and Lipid Metabolism |
| Friday | 10:00 am–12:45 pm | 5 Endolysosomal Dysfunction in Neurodegeneration |
| Friday | 1:30 pm–3:00 pm | Panel: Science, Society and COVID-19 |
| Friday | 3:00 pm–6:00 pm | 6 New Technologies to Study Neurodegeneration |
| Throughout Meeting | | Virtual Poster Session |

[Virtual Icebreaker](#), Wednesday, 5:30 pm

[StemCell Technologies Workshop](#): Thursday, 5:30 pm (*p. T-1*)

[Closing Social](#), Friday, 6:00 pm

All times shown are US EST: [Time Zone Converter](#)

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LONGITUDINAL ASSESSMENT OF TAU PET IMAGING AND ITS CORRELATION WITH NEUROPATHOLOGY AND CLINICAL SIGNS PROGRESSION.

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Alzheimer's disease (AD) and other associated dementias remain a consistent and unruly problem for the aging population and health. As the world's population increases, so does the prevalence of age-related dementias. The neuropathology of AD is characterized by the extracellular deposition of beta-amyloid protein (A β) and the formation of intraneuronal neurofibrillary tangles (NFT) composed of hyperphosphorylated tau (ptau), along with neuroinflammation and neuronal loss that ultimately induces to noticeable cognitive impairments. Abnormal ptau leads to the formation of insoluble, beta-sheet rich amyloid aggregates in tauopathies such as AD. Positron emission tomography (PET) imaging is a promising avenue that may identify tau aggregates in vivo cross-sectionally and longitudinally in various dementia conditions. The goal of this study is to characterize the longitudinal assessment of the tau tracer 18F-THK5351 by in vivo tau PET imaging concomitantly to behavior and tau pathology by histology and biochemistry from 6 to 12 months of age in tau transgenic P301S mice, a mouse model of tauopathies. Our results demonstrate an augmentation of overall gross brain tau pathology by in vivo PET imaging in P301S mice compared to age-matched wild-type (WT) animals accompanied by P301S-model associated pathological tau and phenotypic and behavioral deficits. This longitudinal study provides new insights on the relationship between imaging diagnostic tools, the in vivo neuropathological temporal pattern and the clinical signs observed in animal models of AD that could benefit early disease diagnosis.

This work was partially funded by Department of Defense Peer Reviewed Alzheimer's Research Program Convergence Science. Research Award grant AZ160106 and Alzheimer's Association New Investigator Research Grant NIRG-394284 to IMG.