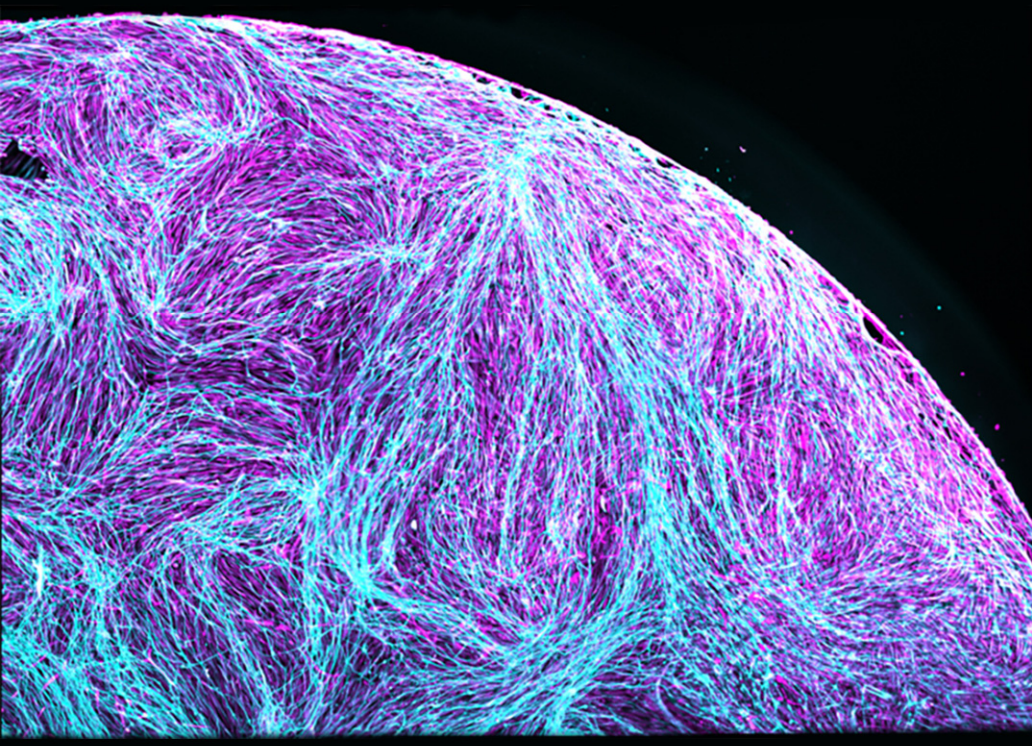
Abstracts of papers presented
at the 2020 *virtual* meeting on

NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

December 2–December 4, 2020



Cold Spring Harbor Laboratory
MEETINGS & COURSES PROGRAM

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NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

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Arranged by

Aaron Gitler, *Stanford University*
Richard Ransohoff, *Third Rock Ventures*
Scott Small, *Columbia University*
Li-Huei Tsai, *Massachusetts Institute of Technology*

CSH Cold Spring Harbor Laboratory
MEETINGS & COURSES PROGRAM

ApoE4 inhibits mitochondrial lipid oxidation in neural cells
Alexey A. Tomilov, Chase A. Garcia, Robert Mahley, Gino Cortopassi.
Presenter affiliation: UC Davis, Davis, California.
View Poster 306

Assessment of neurodegenerative microgliosis induced by oral pathogenic bacteria
Van Thi Ai Tran, Youjung Kang, Hyung-Ryong Kim, Hansang Cho.
Presenter affiliation: Sungkyunkwan University, Suwon, South Korea.
View Poster 307

The poly ADP-ribose response is affected directly by the Huntingtin protein in Huntington's disease
Tamara Maiuri, Carlos Barba, Tae-in Kam, Lauren M. Byrne, Rachel J. Harding, Cheryl H. Arrowsmith, Edward J. Wild, Ted M. Dawson, Valina L. Dawson, Ray Truant.
Presenter affiliation: McMaster University, Hamilton, Canada.
308

Amyloid-β seeding and propagation processes in a hAβ-KI model of Alzheimer's disease
Laura Trujillo-Estrada, Kelly Do Huynh, Marie Minh Thu Nguyen, Alwin Cheung, Janine Pham Tran, Cristina Nunez-Diaz, Stefania Forner, Alessandra C. Martinie, Celia da Cunha, Mohammad Shahnawaz, Claudio Soto, Ines Moreno-Gonzalez, Antonia Gutierrez, Frank M. LaFerla, David Baglietto-Vargas.
Presenter affiliation: University of Malaga/CIBERNED/IBIMA, Malaga, Spain.
View Poster 309

A distinct tau code controlled by HDAC6 is linked to tau pathogenesis
Jui-Heng Tseng, Todd Cohen.
Presenter affiliation: University of North Carolina at Chapel Hill, Chapel Hill, North Carolina.
View Poster 310

C9ORF72 dipeptide repeat proteins disrupt the formation of GEM bodies and result in aberrant localization of survival of motor neuron protein
Yuma Kato, Minnie Yokokawa, Kazunari Onodera, Aaron Gitler, Haruhisa Inoue, Mitsuharu Hattori, Yohei Okada, Hitomi Tsuiji.
Presenter affiliation: Nagoya City University, Graduate School of Pharmaceutical Science, Nagoya, Japan.
View Poster 311

AMYLOID-β SEEDING AND PROPAGATION PROCESSES IN A hAβ-KI MODEL OF ALZHEIMER'S DISEASE

Laura Trujillo-Estrada[1], Kelly Do Huynh[2], Marie Minh Thu Nguyen[2], Alwin Cheung[2], Janine Pham Tran[2], Cristina Nunez-Diaz[1], Stefania Forner[2], Alessandra C Martinie[2], Celia da Cunha[2], Mohammad Shahnawaz[3], Claudio Soto[3], Ines Moreno-Gonzalez[1], Antonia Gutierrez[1], Frank M LaFerla[2], David Baglietto-Vargas[1]

[1]University of Malaga/CIBERNED/IBIMA, Cell Biology, Malaga, Spain, [2]University of California Irvine, Institute for Memory Impariments and Neurological disorders, Irvine, CA, [3]University of Texas Health Science Center at Houston, The Mitchell Center for Alzheimer's Disease and Related Brain Disorders, Department of Neurology, Houston, TX

Recent evidence indicates that Aβ can misfold and aggregate into seeds that structurally corrupt native proteins, mimicking a prion-like process. Several studies using FAD animal models have demonstrated that intracerebral infusion of brain extracts from APP-transgenic mice or AD patients induce Aβ deposition and cerebral amyloid angiopathy. To carry out most of these Aβ-seeding studies, APP-transgenic animal have been used. Nevertheless, it remains to be elucidated whether Aβ deposition can be induced by Aβ-seeds in a sporadic AD model that does not overexpress APP and produces wild type human Aβ.
We used an innovative model to better understand the amyloidogenic events that occur in sporadic AD. This hAβ-KI model, expresses wild-type human Aβ under the control of the endogenous mouse APP gene. Aβ-seeds from AD patients (stage C) from the AD Research Center (UCI) were administered into 7-8-month-old hAβ-KI and as positive controls 3xTg-AD mice were employed.
We demonstrated that amyloid seeds can stimulate Aβ aggregations in 3xTg-AD and hAβ-KI models. We found that Aβ aggregates occur earlier in the 3xTg-AD vs hAβ-KI and that a longer term of treatment is necessary to accelerate diffusible Aβ pathology in the hAβ-KI mice. Thereferoe, this hAβ-KI model represents an important step towards the development of next-generation animal models that will provide better predictive outcomes for human patients.

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