

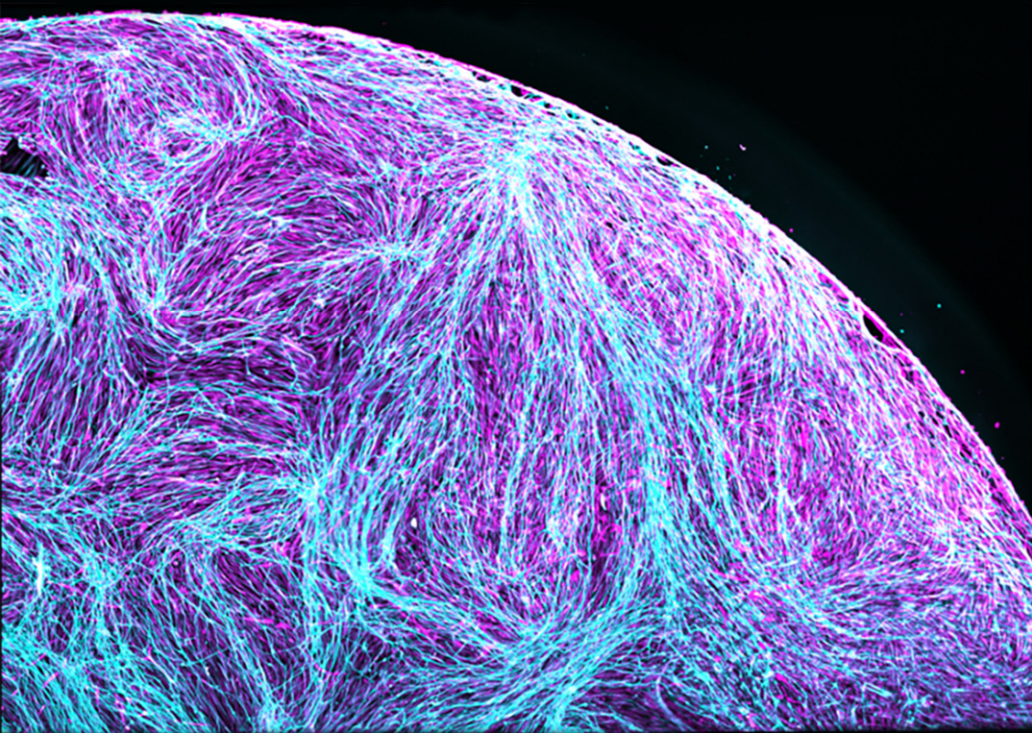
Abstracts of papers presented  
at the 2020 *virtual* meeting on

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# NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

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December 2–December 4, 2020



Cold Spring Harbor Laboratory  
MEETINGS & COURSES PROGRAM



Abstracts of papers presented  
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# NEURODEGENERATIVE DISEASES: BIOLOGY & THERAPEUTICS

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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

**Inhibition of  $\alpha$ -synuclein fibrillogenesis by diphenyl triazine hybrids—Design, synthesis and in-vitro efficacy studies**

Joshna Gadhavi, Mudasir Maqbool, Pravin Hivare, Sharad Gupta, Nasimul Hoda.

Presenter affiliation: Indian Institute of Technology Gandhinagar, Gandhinagar, India.

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**Intravascular delivery of neural precursors improves memory impairment in mouse models of Alzheimer's disease**

Nazaret Gamez Ruiz, Enrique Armijo Fuentes, Ruben Gomez Gutierrez, Claudio Soto, Ines Moreno Gonzalez.

Presenter affiliation: The University of Texas Health Science Center at Houston, Houston, Texas; Universidad de Malaga, Malaga, Spain.

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**ApoE4 disrupts insulin signaling in N2A neuroblastoma cells**

Chase Garcia, Alexey Tomilov, Gino Cortopassi.

Presenter affiliation: UC Davis School of Veterinary Medicine, Davis, California.

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**Human pluripotent stem cells as a research tool for elucidating the role of glial cells in Alzheimer's disease**

Juan Antonio García León, Laura Caceres Palomo, José Carlos Dávila, Javier Vitorica, Antonia Gutiérrez.

Presenter affiliation: University of Malaga/IBIMA/CIBERNED, Málaga, Spain.

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**Quantitative analysis of early endosome pathology in young and aged Ts65Dn mice following maternal choline supplementation (MCS) using 3D reconstructed z-stacks**

Megan K. Gautier, Christy M. Kelley, Elliott J. Mufson, Stephen D. Ginsberg.

Presenter affiliation: Nathan Kline Institute, Orangeburg, New York; NYU School of Medicine, New York, New York.

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**Can zebrafish (*Danio rerio*) produce amyloid beta peptide? Expressing tagged forms of Appa and Appb *in vivo***

Ewan Gerken, Morgan Newman, Michael Lardelli.

Presenter affiliation: Alzheimer's Disease Genetics Laboratory, Adelaide, Australia.

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# HUMAN PLURIPOTENT STEM CELLS AS A RESEARCH TOOL FOR ELUCIDATING THE ROLE OF GLIAL CELLS IN ALZHEIMER'S DISEASE

Juan Antonio García León<sup>1\*#</sup>, Laura Caceres Palomo<sup>1\*</sup>, José Carlos Dávila<sup>1</sup>, Javier Vitorica<sup>2</sup>, Antonia Gutiérrez<sup>1#</sup>

<sup>1</sup>University of Malaga/IBIMA/CIBERNED, Cell Biology, Genetics and Physiology, Málaga, Spain, <sup>2</sup>University of Seville/IBiS/University Hospital Virgen del Rocío/CSIC/CIBERNED, Biochemistry and Molecular Biology, Seville, Spain

\*First authors

#Corresponding authors

## **Background:**

Alzheimer's disease (AD) is characterized by presenting a complex pathology, not fully resolved yet. This fact, together with the lack of reliable models, has impeded the development of effective therapies. Recently, several studies have shown that functional glial cell defects have a key role in the pathology of AD. However, this glial dysfunction, currently, cannot be correctly modeled using the available animal models, so we hypothesized that cells derived from Alzheimer's patients can serve as a better platform for studying the disease. In this sense, human pluripotent stem cells (hPSC) allow the generation of different types of neural cells, which can be used for disease modeling, identification of new targets and drugs development.

## **Methods:**

We have a collection of hiPSCs derived from patients with sporadic forms of AD. We have differentiated these cells towards neural lineage to obtain neurons and astrocytes. For the generation of oligodendrocytes (OLs), we have developed a fast and robust protocol to generate mature OLs in just 22 days.

## **Results:**

We have generated neural precursors from all the lines tested. In the case of OLs, the cells generated resemble primary OLs and can myelinate neurons *in vivo* and *in vitro* using a screening compatible platform. This platform is being transferred for the generation of the other glial cells.

## **Conclusions:**

This methodology can be used to elucidate the pathogenic pathways associated with neurodegeneration and to identify new therapeutic targets susceptible to modulation, contributing to the development of new effective drugs against AD.

## **Acknowledgments:**

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