

FULL SPEAKER BIOGRAPHY and ABSTRACT

Clive Svendsen, PhD
University of Wisconsin-Madison

Dr. Svendsen is a Professor of Neurology and Anatomy at the University of Wisconsin-Madison, Director of the NIH funded Stem Cell Training Program and Co-Director of the University of Wisconsin Stem Cell and Regenerative Medicine Center. He is also a consultant professor at Stanford University. He did his pre doctoral training at Harvard University and received his PhD from the University of Cambridge in England. He then established a research group at the University of Cambridge Center for Brain Repair before moving to the University of Wisconsin in 2000. Having pioneered some of the early work on human neural stem cell biology, the focus of his current research is modeling and treating neurological diseases using stem cells. On the basic science front, by deriving stem cells from patients with specific diseases it will be possible in the future to understand more about the mechanisms underlying the illness. On the translational side, working closely with neurosurgeons, neurologists and other scientists he is also developing novel ways to deliver cells engineered to release powerful growth factors to the brain and spinal cord of patients with serious neurological disease.

Modeling Neurodegeneration Using Human Neural Stem Cells

Stem cells may be important for future clinical trials, but also have great potential to increase our understanding of neurodegenerative mechanisms. Our central hypothesis is that the neurons generated in vitro from neural stem cells carrying disease specific gene defects will undergo specific degeneration related to that disease. Examples of disease specific neuronal degeneration will be given from Down Syndrome and Parkinson's disease. Our most important observation is direct proof of this hypothesis using disease specific induced pluripotent stem (iPS) cells from a patient with spinal muscular atrophy (SMA). In this condition, motor neurons selectively die in the spinal cord of young children. We have also observed that motor neurons derived from SMA-iPS cultures undergo similar degeneration. We are now examining some of the mechanisms underlying this phenomenon, and starting to screen drugs that might reverse or slow down this degeneration. The translational significance of this work lies in the fact that using these human cell models, drugs that target human biology can be screened for in a high throughput format.

What is the central hypothesis of my presentation?

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What is the most important observation I will discuss?

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