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Dr. Ole Isacson is Professor of Neurology (Neuroscience) at Harvard Medical School. He is the Director of the Center for Neuroregeneration Research/Neuroregeneration Laboratories at McLean Hospital and an NIH Udall Parkinson's Disease Research Center of Excellence grant awardee. Dr. Isacson is also a member of the Scientific Advisory Board of the Harvard NeuroDiscovery Center and Principal Faculty of Harvard Stem Cell Institute. Dr. Isacson received his Medical Bachelor (1984) and Doctor of Medicine (a research doctoral degree in Medical Neurobiology, 1987) from the University of Lund in Sweden. In 1989, after a 2 year postdoctoral position at Cambridge University, England, Dr. Isacson was recruited to Harvard as an Assistant Professor of Neuroscience and there established a small independent research laboratory for his work on neuroregeneration. Over the last decade his original laboratory has grown to an internationally recognized academic research center for Parkinson's disease and related disorders, funded by the NIH, DOD and private foundations. Dr. Isacson's scientific models and studies of conceptually new therapies for neurodegenerative diseases have resulted in many new findings and clinical trials for Parkinson's and Huntington's disease. Dr. Isacson is Receiving Editor of the European Journal of Neuroscience and on the board of numerous scientific journals. He is a founding member and past President of the American Society for Neural Transplantation and Repair, and is the current President of the international Cell Transplant Society, CTS (branch of The Transplantation Society, TTS). He serves as a scientific reviewer and advisor to the NIH, DOD and many Parkinson community groups. Dr. Isacson has received several international prizes, research awards and lectureships. He is author or co-author of over 250 scientific research publications in neuroscience and neurology, and 3 books in his field.

The Center for Neuroregeneration Research has had impact on several neurodegenerative disease problems. Studies indicated in the early 90s that cells vulnerable to the disease processes in Parkinson's or Huntington's disease could be protected from various toxins, including lesions and energetic metabolic failures (Schumacher et al. 1991, Frim et al. 1994). Work also showed that neurons in the striatum could be replaced by implanted fetal GABAergic neurons with functional effects (Hantraye et al. 1992) and that these cells could also grow in Huntington patients in an appropriate way (Freeman et al. 2000). Dr. Isacson's lab was the first to demonstrate that normal midbrain dopaminergic neurons could develop from uninduced embryonic stem cells in 1998 (Deacon et al 1998). This work led to the first demonstration of functional dopamine neurons transplanted to animal models of Parkinson's disease by Dr. Isacson's research group in 2002 (Bjorklund et al. 2002). Parkinson's disease cell transfer work revealed that many different kinds of deficits could be repaired by fetal neurons, even when donor cells were from different donor species. For example, using porcine cells, we demonstrated both in animal models and later in the clinic that in principle, these cells can repair the dopamine system (Isacson et al. 1995, Deacon et al. 1997, Mendez et al. 2005, 2008). These studies demonstrated that functional repair is possible using cell therapies, either for trophic preservation or by restituting the neurotransmission. Dr. Isacson's lab was the first to transplant embryonic stem cell derived gamma-aminobutyric acid (GABA) expressing neurons in animal models in 1995 (Dinsmore et al. 1996). Recently, scientists at the Center for Neuroregeneration Research demonstrated that embryonic stem cells also can generate the dopamine neurons (A9

and A10) that are involved in the degeneration that creates the syndrome of Parkinson's disease (S. Chung et al. 2002, 2005), and that defining the molecular profile of vulnerable neurons in neurodegenerative disease can lead to neuroprotective treatments (C.Y. Chung et al. 2005, 2007). These discoveries show that ES or induced pluripotent cell derived neurons, including human cells, can reverse functional deficits in animal models and patients and create neurotransmission that involves basal ganglia, motor circuitry and behavioral recovery. Innovative axon and synapse regeneration approaches (Lin et al. 2006, Inoue et al. 2007, Wernig et al. 2008), novel neural cell sorting methods (Pruszak et al. 2007, Wernig et al. 2008), genetic engineering and gene therapy (Seo et al. 2007, C.Y. Chung et al. 2007, Hemming et al. 2007) complement the in vivo studies to achieve realistic goals in regenerative medicine for patients with neurological diseases. The accomplishments of this research center include providing novel technology and biological insights beyond currently available drug therapies.