

Schwartz Laboratory

Research Interests: Apoptosis, Muscle Development, Neurodegeneration

One of the counter-intuitive aspects of biology is that all of our cells carry genetic programs that allow them to commit suicide. This process, known as Programmed Cell Death (PCD) or apoptosis, removes surplus or deleterious cells from our bodies. For example, the formation of fingers during embryogenesis requires the death of parallel rows of cells in the differentiating hand. Unfortunately defects in the regulation of cell death serve as the basis for most human diseases, most notably neurodegeneration, auto-immunity, and most cancers.

To define the molecular mechanisms that mediate PCD, we initially examined the death of giant muscles during insect metamorphosis. Using a variety of molecular techniques we demonstrated that PCD requires new gene expression and then cloned a number of these death-associated genes. Several of these genes encode previously characterized proteins involved in protein degradation, like ubiquitin and proteasome subunits. Others encoded novel proteins of unknown function.

Building on these results, we cloned the human homologs of these genes and have been examining their roles in normal development and pathogenesis. One of our main efforts has focused on Acheron, an evolutionarily conserved protein that we have found is required for muscle development in vertebrates. Besides providing insight into the development of muscle, we have found that targeting Acheron function can facilitate the survival of transplanted muscle stem cells.

In a separate series of studies, we have been examining the role of the ubiquitin-proteasome pathway in neurodegeneration. It has been shown that defects in this pathway contribute to neuron loss in disorders like Parkinson's disease. The mechanisms that allow some neurons to survive in these individuals while others are endangered are poorly understood.

Selected Publications

Schwartz, L.M. and Truman, J.W. (1982) Peptide and Steroid Regulations of Muscle Degeneration in an Insect. *Science*, 215: 1420-1421.

Schwartz, L.M. and Ashwell, J. editors (2001) Methods in Cell Biology Series, Apoptosis. Academic Press, volume 66, pp533.

Valavanis, C., Wang, Z., Sun, D. Vaine, M., and **Schwartz, L.M.** (2007) Acheron, a novel member of the Lupus Antigen family, is induced during the programmed cell death of skeletal muscles in the moth *Manduca sexta*. *Gene* 393:101-109

Bouchentouf, M., Benabdallah, B.F., Rousseau, J., **Schwartz, L.M.** and Tremblay, J.P. (2007) Induction of Anoikis following myoblast transplantation into SCID mouse muscles requires the Bit1 and FADD pathways. *American Journal of Transplantation*, 7:1491-1505

Gao, Z., Deblis, R., Glenn, H., and **Schwartz, L.M.** (2007) Differential Role Of Hic-5 Isoforms On The Regulation Of Cell Death And Myotube Formation During Myogenesis *Experimental Cell Research* in press.

Professional Highlights

NIH Panel on Mechanisms of Parkinson's Disease, 2005
Chancellor's Medal, UMass Amherst, 1999
Distinguished Faculty Lecturer, UMass 1998-99
Samuel Conti Faculty Fellowship Award, UMass Amherst 1999
Reviewer for 44 different journals and 14 funding agencies



Lawrence Schwartz, PhD

Science Director, PVLSI
Director, Center of Excellence in Apoptosis Research (CEAR)
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Education

B.A., Biology, Northwestern University
Ph.D., Zoology, University of Washington

Postdoctoral

Membrane Biophysics, University of Washington, Seattle, 1982-1984
Molecular Development, University of Washington, Seattle, 1984-1987
Mouse Transgenics, Whitehead Institute for Biomedical Sciences, MIT, 1995-1996

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