

The 2008 Harvard / Paul F. Glenn

# Symposium on Aging

June 23, 2008



## Paul F. Glenn Laboratories for the Biological Mechanisms of Aging

Welcome to the 3rd Annual Harvard/Paul F. Glenn Symposium on Aging. Each year, the Paul F. Glenn Laboratories host the Harvard Symposium on Aging with a mission to educate the wider research community about advancements in this fast-paced field and to stimulate collaborative research in this area. We have been fortunate to have many of the leaders in the aging field speak at these symposia. As a result, attendees come not only from the Harvard research community but from across the nation and from overseas for this one day event. We are glad you could join us here today.

The reasons for accelerating research molecular biology of aging are clear. First and foremost, the number of aged individuals in developed countries is growing rapidly, which is going to place an unprecedented burden on the families and the economies of those nations. Because chronic illness in the elderly is a major medical cost, enormous savings would be achieved if mortality and morbidity could be compressed within a shorter duration of time at the end of life. A study by the RAND Corporation in 2006 concluded that advances in medicine arising from aging research would be 10-100 times more cost-effective than any other medical breakthrough.

Advances in aging research have shown that it is possible to extend the healthy lifespan of laboratory animals through genetic and pharmacological means. Many leaders in the aging field predict that significant strides will be made in understanding how human health and lifespan are regulated, leading to novel medicines to forestall and treat diseases of aging such as diabetes, cancer, Alzheimer's and heart disease. Thus, a major goal in the coming years is to understand how healthy lifespan is extended and to translate these discoveries into medicines that could not only postpone but treat diseases of aging.

The Paul F. Glenn Laboratories are located on two floors of the New Research Building at Harvard Medical School. To promote aging research at Harvard and foster collaboration, the Glenn Labs Steering Committee awards two \$100K pilot research grants each year to Medical School investigators with innovative approaches to applying their expertise to addressing critical questions in the aging field.

This past year included a number of initiatives at the Glenn Labs. The first graduate course on the biology of aging was launched with great success. In addition, the first "Boston Area Aging Data Club" kicked off in March this year, with attendees from entire New England area. On behalf of The Paul F. Glenn Laboratories and Harvard Medical School, we welcome you to the Harvard Symposium on Aging, 2008.

David Sinclair and Bruce Yankner  
Co-Directors, The Paul F. Glenn Laboratories

# Symposium on Aging Agenda

June 23, 2008  
9:00 - 5:00

- 9:00 - 9:15    **Welcome**
- 9:15 - 10:00    Thomas A. Rando, MD, PhD – Keynote Speaker  
                    **Stem Cells and Aging**
- 10:00 - 10:40    Scott Lowe, PhD  
                    **Senescence, Heterochromatin and Aging**
- 10:40 - 11:20    Toren Finkel, MD, PhD  
                    **Linking  $\beta$ -catenin and Aging**
- 11:20 - 12:00    T. Keith Blackwell, MD, PhD  
                    **Skn-1 in Longevity & Stress Resistance**
- 12:00 - 1:30    **Lunch**
- 1:30 - 2:00    **Panel Discussion:**  
                    “Is aging simply a collection of predictable disease?”  
                    Moderator: David A. Sinclair, PhD  
                    Panelist: Richard A. Miller, MD, PhD & Leonard Guarente, PhD
- 2:00 - 2:40    Jan Vijg, PhD  
                    **DNA Damage as a Driver of Aging**
- 2:40 - 3:20    Gary B. Ruvkun, PhD  
                    **Energy Metabolism and Lifespan**
- 3:20 - 4:00    Richard A. Miller, MD, PhD  
                    **Live Short and Prosper:  
                    Aging Secrets of the Snell Dwarf Mouse**
- 4:00 - 4:40    Heidi Tissenbaum, PhD  
                    **Fat and Lifespan Regulation in *C. elegans***
- 5:00 - 5:30    **Social** – Cheese & Crackers

## Thomas A. Rando, MD, PhD – Keynote Speaker



Thomas A. Rando is Associate Professor of Neurology and Neurological Sciences at the Stanford University School of Medicine where his lab focuses on the biology of adult skeletal muscle stem cells (satellite cells) in homeostasis, aging and disease. His laboratory described the critical roles of Notch and Wnt signaling in the lineage progression of satellite cells. He received a Paul Beeson Physician Faculty Scholar in Aging from the American Federation for Aging Research and an Ellison Medical Foundation Senior Scholar Award. In 2005 he received an NIH Director's Pioneer Award for his work at the interface between stem cell biology and the biology of aging. His laboratory continues to focus on adult muscle stem cells, with recent interests in epigenetic regulation and the role of microRNA's in quiescence and activation.

Scott W. Lowe is a Professor in the Watson School of Biological Sciences at Cold Spring Harbor Laboratory. He is also Deputy Director of the Cold Spring Harbor Laboratory Cancer Center and an Investigator for the Howard Hughes Medical Institute. Dr. Lowe received a Bachelor of Science Degree from the University of Wisconsin-Madison where he worked for several years studying the molecular basis of hypercholesterolemia. He performed his graduate studies at the Massachusetts Institute of Technology under the supervision of Dr. H. Earl Ruley, and received his Ph.D. for research on the role of the p53 tumor suppressor in oncogenic transformation, apoptosis, and chemosensitivity. After a brief postdoctoral position in the MIT Center for Cancer Research with Drs. David Housman and Tyler Jacks, Dr. Lowe initiated independent research at Cold Spring Harbor Laboratory as a Cold Spring Harbor Laboratory Fellow and then a member of the faculty. Dr. Lowe's research has made important contributions to our understanding of the p53 tumor suppressor pathway, as well as the processes of multi-step carcinogenesis, cellular senescence, and tumor-cell drug resistance. Dr. Lowe's current research focuses on control of apoptosis and senescence by cancer genes, and on the molecular genetics of drug sensitivity and resistance in spontaneous tumors. Most recently, he has been integrating mouse models, genomics, and RNA interference for studying cancer gene function in vivo. This work has been recognized by several awards, including a Sydney Kimmel Foundation Scholar Award, a Rita Allen Foundation Scholar Award, the AACR Outstanding Investigator Award, the AACR-NFRCR Professorship in Basic Cancer Research, the Paul Marks Prize for Cancer Research, and the AAAS Fellow Award.



## Heidi A. Tissenbaum, PhD



Heidi A. Tissenbaum is currently an Associate Professor at the University of Massachusetts Medical School in the Program in Gene Function and Expression and the Program in Molecular Medicine. Dr. Tissenbaum received her Ph.D. in Genetics in 1997 from Harvard Medical School where she worked in the laboratory of Gary Ruvkun. In a series of pioneering experiments she discovered that components of an insulin-like signaling pathway regulate life span in the roundworm *Caenorhabditis elegans*. She continued her work on the biology of aging as a postdoctoral fellow with Leonard Guarente at the Massachusetts Institute of Technology. Supported by the Helen Hay Whitney Foundation, she identified the SIR2 protein as a regulator of life span in *C. elegans*. She has received several prestigious awards for her work including a Career Award in the Biomedical Sciences from the Burroughs Wellcome Fund, the American Federation of Aging Research New Investigator Award, the Worcester Foundation for Biomedical Research Scholar Award and a William Randolph Hearst Young Investigator in Aging Award as well as funding from the National Institute on Aging, the William Randolph Hearst Foundation, the Concern Foundation, the W. M. Keck Foundation and the Glenn Medical Foundation.

Recent studies from her laboratory have included publications in Nature Genetics, Cell Metabolism, Proceedings of the National Academy of Sciences, Mechanisms of Aging and Development and EMBO Reports.

Dr. Blackwell received a BS in Chemistry from Duke University in 1978, and the MD and PhD degrees from Columbia University in 1987 and 1988, respectively. He performed his graduate and initial postdoctoral work with Dr. Frederick W. Alt, with whom he studied the mechanism and regulation of B- and T- cell receptor gene assembly. In 1989 he joined the lab of Dr. Harold Weintraub (Fred Hutchinson Cancer Research Center) as a postdoctoral fellow of the Life Sciences Research Foundation. He then developed in vitro selection systems for analyzing protein-nucleic acid interactions, and studied how various transcription factors recognize DNA sequences. In 1993 he became a Junior Investigator at the Center for Blood Research (now IDI), and an Assistant Professor of Pathology at Harvard Medical School. He was named a Searle Scholar in 1995. He became an Associate Professor in 2001, and in 2004 moved to the Joslin Diabetes Center, where he is Head of the Section on Developmental and Stem Cell Biology, a Staff Member of the Board of Trustees, and a principal faculty member of the Harvard Stem Cell Institute. His lab uses the *C. elegans* model to investigate how SKN-1 and other transcription regulatory networks defend against free radicals and environmental stresses, and influence longevity. They also study mechanisms that regulate gene expression programs during germ cell and early embryo development.



## Jan Vijg, PhD



Jan Vijg, Ph.D., is a Professor at the Buck Institute for Age Research in Novato, California, since January, 2006. He received his Ph.D. at the University of Leiden, The Netherlands, in 1987. From 1987 to 1990 he served as Head of the Department of Molecular Biology of the TNO Institute for Experimental Gerontology in Rijswijk, The Netherlands. From 1990 to 1993 he was founder and Scientific Director of Ingeny B.V., a Dutch Biotechnology company. From 1987 to 1990 he was founder and Chairman of the Board of the Biomedical Study Group on the Etiology of Aging of the Netherlands Foundation of Medical and Health Research and the organizer of the Molecular Biology Group within EURAGE, the EU Concerted Action on Aging Research. In 1993 he moved to Boston, to take up a position as Associate Professor of Medicine at Harvard Medical School and Director of the Molecular Genetics Section of the Gerontology Division at the Beth Israel Deaconess Medical Center. In 1998 he accepted an offer from the University of Texas Health Science Center in San Antonio, Texas, to become a full professor in the Department of Physiology and Director of the Human Genetics Program of the Sam and Ann Barshop Center for Longevity and Aging Studies. With his research team he was the first to develop transgenic mouse models for studying mutagenesis *in vivo* (in 1989) and used these models ever since in studying the possible relationship between damage to the genome and aging. He has published more than 200 scientific articles and is inventor or co-inventor on 8 patents.



Gary B. Ruvkun is a professor of genetics at Harvard Medical School. Dr. Ruvkun is a graduate of UC Berkeley (AB, Biophysics, 1973) and Harvard (PhD Biophysics, 1982). During a three year interlude between undergraduate and graduate school, Dr. Ruvkun worked in reforestation in the Pacific Northwest and travelled extensively in Latin America. His PhD thesis with Fred Ausubel explored the symbiotic nitrogen fixation genes of *Rhizobium*. Dr. Ruvkun began to work on developmental control genes in the nematode *C. elegans* in 1982 as a Harvard Junior Fellow working with Bob Horvitz at MIT and Walter Gilbert at Harvard.



Dr. Ruvkun's research has explored two major themes: regulation of developmental control genes by microRNA genes and other small RNAs, and control of longevity and metabolism. Dr. Ruvkun's laboratory discovered that an insulin-like signaling pathway controls *C. elegans* metabolism and longevity and identified the *daf-16* gene product as a highly conserved Forkhead transcription factor and the signal transduction components from the DAF-2 insulin receptor to the DAF-16 transcription factor. The Ruvkun lab also showed that insulin-like signaling in neurons alone is sufficient to specify wild type lifespan. These findings point to the nervous system as a central regulator of animal longevity. Recently, full genome RNAi libraries were used to reveal a comprehensive set of 90 genes that regulate aging and metabolism. Finally, functional genomic studies from the Ruvkun lab have also revealed a few hundred regulators of fat storage. Like the insulin-signaling pathway genes, many of these genes involved in fat storage are broadly conserved in animal phylogeny and are likely to reveal the neuroendocrine system that assesses and regulates energy stores and assigns metabolic pathways based on that status.

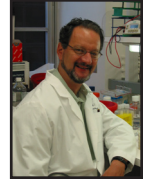
Dr. Ruvkun's honors and awards include a Harvey Lecture at Rockefeller University, the Rosenstiel Award from Brandeis University (with Victor Ambros, Andy Fire, and Craig Mello), the Warren Triennial Prize from Massachusetts General Hospital (with Victor Ambros), the Benjamin Franklin Medal from the Franklin Institute (with Victor Ambros and David Baulcombe), the Gairdner International Award from the Gairdner Foundation of Canada (with Victor Ambros), and the United States National Academy of Sciences.

## Richard A. Miller, MD, PhD



Richard A. Miller, M.D., Ph.D., is a Professor of Pathology and Associate Director of the Geriatrics Center at the University of Michigan; he is also a Research Scientist at the Ann Arbor DVA Medical Center. He received the BA degree in 1971 from Haverford College, and MD and PhD degrees from Yale University in 1976-1977. After postdoctoral studies at Harvard and Sloan-Kettering, he moved to Boston University in 1982 and then to his current position at Michigan in 1990. Dr. Miller has served in a variety of editorial and advisory positions on behalf of the American Federation for Aging Research and the National Institute on Aging, and is currently one of four Editors-in-Chief of Aging Cell. He is the recipient of the Nathan Shock Award, the AlliedSignal Award, the Kleemeier Award for aging research, and the Irving Wright Award. His main research interests all relate to the control of aging rate in mice, and include ongoing studies of mutations that slow aging, the relation of cellular stress resistance to longevity, mapping of genes that influence lifespan and age-sensitive traits, screens for drugs that extend lifespan in mice, and methods to improve function of T lymphocytes from old donors.

Toren Finkel received his undergraduate degree in Physics and his MD and PhD degree from Harvard Medical School in 1986. Following a residency in Internal Medicine at the Massachusetts General Hospital he completed a fellowship in Cardiology at Johns Hopkins. In 1992, he accepted a position within the Intramural Research Program of the National Heart, Lung and Blood Institute (NHLBI) at the National Institutes of Health in Bethesda, Maryland. In 2001 he became the Chief of the Cardiology Branch and in 2007 he became Chief of the newly formed Translational Medicine Branch within the NHLBI. His current research interests include the role of reactive oxygen species in aging and stem/progenitor cell dysfunction in age-related diseases.





# **The Paul F. Glenn Laboratories Steering Committee**

## Lewis C. Cantley, Ph.D.



Dr. Cantley joined the faculty of Harvard Medical School in 1992, when he was also appointed Chief of the Division of Signal Transduction in the Department of Medicine at Beth Israel Hospital. He graduated summa cum laude from Wesleyan College and earned his Ph.D. from Cornell University in 1975. His postdoctoral research and first faculty appointment were in the Department of Biochemistry and Molecular Biology at Harvard University.

Although Dr. Cantley has made major contributions to numerous fields, he is best known for his discovery of the phosphoinositide 3-kinase (PI3K) pathway, which opened a window on the way biochemical signaling pathways control normal cell growth and how they can trigger the development of cancer when they are defective. Dr. Cantley's discoveries formed the foundation for the elucidation of longevity pathways in the worm *C. elegans*. His work has also enabled researchers to use the genetic blueprints of signaling proteins to predict their cellular targets, with a view to finding new cancer therapies.

Dr. Cantley has received numerous awards in recognition of these achievements. He received the Caledonian Prize of the Royal Society of Edinburgh in 2002. He has well over 300 publications to his name, including research papers, reviews, and book chapters. Cantley was elected to the National Academy of Sciences in 2001.

Stephen J. Elledge, Ph.D. is The Gregor Mendel Professor of Genetics and Professor of Medicine at Harvard Medical School and Senior Geneticist at Brigham and Women's Hospital. He is also an Investigator of the Howard Hughes Medical Institute, and is a Pew Scholar in the biomedical sciences. He is a recipient of the G.H.A. Clowes Memorial Award, the Paul Marks Prize in cancer research, the Michael E. DeBakey Award for research excellence, and the 2002 National Academy of Sciences Award in molecular biology. Dr. Elledge was elected to the National Academy of Sciences in 2003.



The Elledge lab uses model organisms to characterize cell cycle control and the cellular response to DNA damage. They have identified and characterized a signal transduction pathway that senses and responds to DNA damage. The lab is interested in technology development to aid in gene and drug discovery, and most recently applied a screen to identify transcriptional regulators of telomerase, an important player in human cancer. Currently they are initiating genome wide siRNA screens for regulators in cell cycle, checkpoint signaling tumorigenesis and cell proliferation. Until recently large-scale genetic screens have not been possible in mammals. The Elledge Lab initiated a genetic screen to find transcriptional regulators of human telomerase, an important player in human cancer. Using a technique that places endogenous genes under the control of a regulated promoter, we uncovered multiple negative regulators of this pathway.

## Judith A. Glaven, Ph.D.



Judith A. Glaven, Ph.D. – Dr. Glaven is the Director of Basic Science Programs at Harvard Medical School. Dr. Glaven came to HMS with over four years of experience as a Senior Scientific Editor at the journal *Cell*, where she evaluated and recruited scientific papers and worked closely with the scientific community in the areas of basic Cell Biology, Immunology, Cancer Biology and Aging. Previous to being at *Cell* her own research focused on basic molecular mechanisms of cell proliferation and migration. She brings a broad perspective of basic biomedical science to the steering committee and represents the involvement and commitment of the Medical School to this initiative.



Dr. Haigis is the most recent recruit to the Paul F. Glenn Laboratories, having joined in October 2006. Dr. Haigis's lab focuses on understanding the role that mitochondria play in mammalian aging and disease. Mitochondria are dynamic organelles that provide cells with energy even during dramatic changes in diet, stress and development. Mitochondria are also a major site for reactive oxygen species production, ion homeostasis, and apoptosis. Not surprisingly, mitochondrial dysfunction has been implicated in aging, neurodegeneration and metabolic diseases, such as diabetes.



The regulation of aging is highly conserved. For example, an extra copy of SIR2 (silent information regulator; sirtuins) significantly increases the lifespan of yeast, worms and flies. Mammals have seven homologs of SIR2, three of which are found in mitochondria. Recent studies have shown that sirtuins affect mitochondrial biogenesis and energy production. Our lab is interested in understanding how sirtuins mediate the interplay between mitochondrial activity and aging.

The main goals of the lab's research are: 1) to identify signals generated by mitochondria that contribute to aging and to identify those regulated by mammalian sirtuins, 2) to determine molecular mechanisms for these signals, and 3) to understand how these pathways regulate biological functions that decline during normal aging. To accomplish these goals, the lab's research integrates biochemistry, proteomics, cell biology and mouse genetics. These studies have the potential to lead to novel therapies that could treat a spectrum of human diseases.

## Peter M. Howley, M.D.



Peter M. Howley, M.D., is the George Fabyan Professor of Comparative Pathology Head of the Department of Pathology at Harvard Medical School. Research in the Howley laboratory is focused on the molecular biology of cancer and the role of viruses in its formation. The lab studies “high risk” HPV types such as HPV16 and HPV18 encode two oncoproteins, E6 and E7, which target the important cellular growth regulatory proteins p53 and pRb, respectively. They have previously shown that E6 promotes the ubiquitination and degradation of p53, and are now interested in the general question of how proteins are recognized within cell by the ubiquitin proteolytic machinery. The E6 promoted ubiquitylation of p53 is mediated by a cellular protein, , called the E6 Associated Protein (E6AP), that binds to E6 and participates directly in its ubiquitination. We are interested in how E6AP is regulated and the identification of additional cellular proteins that E6AP targets in cells, either in the presence of or absence of the viral E6 protein.

C. Ronald Kahn, B.A., M.D., M.S., D.Sc. is President and Director of the Joslin Diabetes Center in Boston. Dr. Kahn received his M.D. from The University of Louisville School of Medicine (with highest honors) and holds honorary doctorates from University of Louisville (Honoris Causa) (1984), Universite de Paris Pierre et Marie Curie (1994) and The University of Geneva (2000). He has received over 60 awards and honors and is the author of over 170 publications. The Kahn lab seeks to understand the link between metabolism and aging. Recent studies from the lab showed that a reduction of fat mass without caloric restriction can be associated with increased longevity in mice, possibly through effects on insulin signaling. Work in the lab is also aimed at defining the roles of each of the IRS-proteins and isoforms of PI 3-kinase in insulin signaling and insulin resistance, the same pathway that regulates aging in *C. elegans* and *Drosophila*. The Kahn lab has also found interesting links between SIRT1-3 and the control of metabolism and longevity. Ronald Kahn has also been recognized for his pioneering work in the field of diabetes, including seminal discoveries that have defined the molecular and cellular mechanisms of insulin action and have yielded critical information about insulin resistance in obesity and diabetes. His recent work has made important contributions to the aging field by demonstrating that a mutation of the insulin receptor gene in adipocytes and that the hormone Klotho, which down-regulates insulin and IGF-1 signaling, extends mouse lifespan.



## Gary B. Ruvkun, Ph.D.



Gary B. Ruvkun, Ph.D., is a Professor in the Department of Genetics at Harvard Medical School. The Ruvkun lab identified some of the first ever longevity genes using the nematode *C. elegans* as a model and have been instrumental in deciphering the insulin/IGF-1 pathway of longevity regulation. The lab showed that longevity is regulated by insulin signaling within the nervous system, suggesting that it is the metabolism within particular neurons that are key to regulation of lifespan. They study how these neuroendocrine pathways are coupled to sensory inputs. For example, the insulin pathway is coupled to a thermosensory pathway, allowing metabolism to be coupled to temperature. We are now exploring the neural signaling pathways that couple these systems. They have used powerful genetic selections to identify signaling molecules downstream of insulin-like receptors, as well as a novel insulin reception pathway that may act more broadly in animals. Another Glenn Lab Associate, Dr Ron Kahn, showed that disruption of the same insulin-signaling pathway can extend the lifespan of mice.

Pamela A. Silver, Ph.D. is a Professor in the Department of Systems Biology at Harvard Medical School and a member of the Department of Cancer Biology at the Dana Farber Cancer Institute. She studied translocation of proteins across membranes as a graduate student with Bill Wickner at the University of California and nuclear transport as an ACS Postdoctoral Fellow with Mark Ptashne at Harvard University. First as a faculty member at Princeton University, and later at Harvard Medical School, she developed novel genetic and cell biological approaches to study movement of macromolecules in eukaryotic cells. Her current interests range from the systems biology of the nucleus to the use of genomics, chemical genetics, cell-based screening and proteomics in the study of diseases and the brain. She has been the recipient of an NSF Presidential Young Investigator Award, an Established Investigatorship from the American Heart Association, the BBS Mentoring Award from Harvard Medical School and an NIH MERIT award. The Silver group focuses on several major areas in biology from a systems-wide point of view. They model and design biological circuits and parts with well-defined properties for engineering biological systems with an emphasis on intracellular spatial programming. Their recent work has identified new aging regulatory genes in yeast and has led to “cellular oscillator” technology for analyzing cellular aging. The system allows researchers to determine the lifespan of individual yeast cells based on nuclear/cytoplasmic localization.



## David A. Sinclair, Ph.D.



David A. Sinclair, Ph.D. is Co-director of the Paul F. Glenn Laboratories for the Biological Mechanisms of Aging, an Associate Professor of Pathology at Harvard Medical School, Associate Member of the Broad Institute for Systems Biology, and co-founder of Sirtris Pharmaceuticals, Cambridge, MA. Dr. Sinclair's research aims to identify conserved longevity control pathways and devise small molecules that activate them, with a view to preventing and treating diseases caused by aging.

Dr. Sinclair obtained a BS with first-class honors at the University of New South Wales, Sydney, and received the Commonwealth Prize for his research. In 1995, he received a Ph.D. in Molecular Genetics and was awarded the Thompson Prize for best thesis work. He worked as a postdoctoral researcher with Dr. Leonard Guarente at M.I.T. before being recruited to Harvard Medical School. Dr. Sinclair has received several additional awards including a Helen Hay Whitney Postdoctoral Award, a Special Fellowship from the Leukemia Society, a Ludwig Scholarship, a Harvard-Armenise Fellowship, an American Association for Aging Research Fellowship, and was a New Scholar of the Ellison Medical Foundation. He won the Genzyme Outstanding Achievement in Biomedical Science Award for 2004.

Bruce A. Yankner, M.D., Ph.D is Professor of Pathology and Neurology at Harvard Medical School and Co-Director of the Paul F. Glenn Laboratories for the Biological Mechanisms of Aging. Dr. Yankner graduated from Princeton University, received his M.D. and Ph.D. from Stanford, and did his residency at the Massachusetts General Hospital. His early work was seminal to the understanding of the pathology of Alzheimer's disease, and included the discovery of beta amyloid neurotoxicity, the elucidation of cell death mechanisms in Down's syndrome, and the biology of the presenilin genes. His more recent work has defined molecular features of the aging process in the brain, and has demonstrated a genetic signature of brain aging characterized by changes in genes that are critical for learning and memory. His laboratory has also demonstrated that gene damage may contribute to the aging of the brain, and may start in middle age. He has received many awards, including the Major Award for Medical Research from the Metropolitan Life Foundation, the Derek Denny-Brown Award from the American Neurological Association, the Zenith Award from the Alzheimer's Association, and the Ellison Medical Foundation Senior Scholar Award in Aging.



## Junying Yuan, Ph.D.



Junying Yuan, Ph.D. is a Professor in the Department of Cell Biology at Harvard Medical School. Dr. Yuan received a Ph.D. in Neuroscience from Harvard University in 1989. Dr. Yuan carried out her postdoctoral research at the Massachusetts Institute of Technology where she made seminal discoveries about apoptosis that formed the basis of a Nobel Prize for Medicine in that field. She was first appointed as Assistant Professor at Harvard Medical School in 1992, when she became a Principal Investigator of the Cardiovascular Research Center at Massachusetts General Hospital. She joined the Department of Cell Biology in 1996 and was appointed a Professor of Cell Biology at Harvard Medical School in 2000. The Yuan lab aims to understand the basic mechanisms of cell death as well as their implications in neurodegenerative diseases using cellular, genetic, molecular and chemical biological approaches. The lab has developed a high throughput assay for ER stress and identified a small molecule inhibitor of ER stress induced cell death, which they named salubrinal (sal). They have identified a new form of cell death called “necroptosis” and identified a small molecule inhibitor named necrostatin-1 (Nec-1). Nec-1 reduced ischemic brain injury with an extended time window for treatment and thus may be very important for mediating acute neurological injury.



## Notes

---



## Notes

---



Nearby locations for lunch:

1. Elements Café  
located at Harvard Medical School, New Research Building
2. Bertucci's  
(at Children's), 1 Blackfan Circle (Exit rear of Harvard Medical School)
3. Galleria Longwood Food Court  
342 Longwood Avenue

