

The 2007 Harvard / Paul F. Glenn

Symposium on Aging

May 21, 2007



Paul F. Glenn Laboratories for the Biological Mechanisms of Aging

Welcome to the 2nd 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 also from across the nation and from overseas for this one-day event. We are glad you could join us here today.

Advances in aging research have shown that it is possible to extend the healthy lifespan of laboratory animals through genetic and pharmacological approaches. 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 more fully understand how healthy lifespan may be expanded and to translate these discoveries into medicines that could not only postpone but also 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 research grants each year to Medical School investigators with innovative approaches to critical questions in the aging field. The Labs also seek new technologies to speed aging research and, to this end; a state-of-the-art mass spectrometer was purchased in 2005, which has greatly accelerated biochemical and proteomic studies.

We are also pleased to announce the recruitment of Dr. Marcia Haigis to the Paul F. Glenn Labs. Marcia has advanced our understanding of links between metabolism and aging in mammals, and will speak at today's symposium. Thank you for joining us for this special event. On behalf of The Paul F. Glenn Laboratories and Harvard Medical School, we welcome you to the Harvard Symposium on Aging, 2007.

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

Symposium on Aging Agenda

May 21, 2007

9:00 - 5:00

- 9:00 - 9:15 Introductions by:
Mark R. Collins, President, Glenn Foundation Medical Research
Paul F. Glenn, Founder, Paul F. Glenn Laboratories
- 9:15 - 10:00 Elizabeth H. Blackburn, Ph.D.- Keynote Speaker
Telomeres, Aging, and Cancer
- 10:00 - 10:40 Douglas C. Wallace, Ph.D.
Mitochondria, Disease, and Aging
- 10:40 - 11:20 Marcia Haigis, Ph.D.
Sirtuins and Mitochondrial Function
- 11:20 - 12:00 Nir Barzilai, M.D.
Novel Human Longevity Genes
- 12:00 - 1:00 Lunch Break
- 1:00 - 1:40 Pere Puigserver , Ph.D.
Energy Metabolism and Lifespan
- 1:40 - 2:20 Tomas Prolla, Ph.D.
Mouse Models of Premature Aging
- 2:20 - 3:00 Marc Tatar, Ph.D.
Insulin and Aging in the Fly
- 3:00 - 3:40 Richard Weindruch, Ph.D.
Calorie Restriction in Primates
- 3:40 - 4:20 David A. Sinclair, Ph.D.
Small Molecules that Delay Aging
- 4:30 - 5:00 Social - Cheese and Crackers

Elizabeth H. Blackburn, Ph.D. - Keynote Speaker



Professor Blackburn is a leader in the area of telomere and telomerase research. She has broad experience in the different aspects of telomere function and biology. She discovered the ribonucleoprotein enzyme, telomerase. Hers is a leading laboratory in manipulating telomerase activity in cells, and she has amassed considerable knowledge and experience in the effects this has on cells.

Prof. Blackburn earned her B.Sc. (1970) and M.Sc. (1972) degrees from the University of Melbourne in Australia, and her Ph.D. (1975) from the University of Cambridge in England. She did her postdoctoral work in Molecular and Cellular Biology from 1975 to 1977 at Yale. Dr. Blackburn is currently a faculty member in Department of Biochemistry and Biophysics at UCSF. She is also a Non-Resident Fellow of the Salk Institute.

Throughout her career, Prof. Blackburn has been honored by her peers as the recipient of many prestigious awards. Most recently, she was awarded The Kirk A. Landon-AACR prize for Basic cancer Research (2005) and The Albert Lasker Medical Research Award in Basic Medical Research (2006). She was elected Foreign Associate of the National Academy of Sciences in 1993, and was elected as a Member of the Institute of Medicine in 2000. Dr. Blackburn was recently voted one of the “100 most influential people in the world” according to TIME Magazine.

Dr. Wallace has been a pioneer in the study of human mitochondrial genetics and the role of mitochondrial DNA (mtDNA) variation in human evolution, disease, cancer, and aging. In the 1970s Dr. Wallace defined the basic principles of human mtDNA genetics, demonstrating that the human mtDNA encodes heritable traits, is maternally transmitted, has a high mutation rate, that intracellular mixtures of mutant and normal mtDNAs (heteroplasmy) is common and can segregate randomly during both mitotic and meiotic cell division, and that the clinical phenotype of a mutation depends on the severity of the mitochondrial defect and the reliance of each individual tissue on mitochondrial energy production. Once Dr. Wallace had defined the basic principles of mtDNA genetics, he applied these principles to the investigation of human origins and disease. By analyzing the mtDNA variation found in aboriginal populations from around the world, Dr. Wallace was able to define the human mtDNA mutational tree, reconstruct the ancient migration patterns of women, demonstrate that indigenous mtDNA variation was highly region-specific, and ultimately discover that the regional specificity of mtDNA variation was a product of environmental selection acting on differences in mitochondrial energy utilization determined by ancient mtDNA polymorphisms. Dr. Wallace also identified the first maternally inherited mtDNA diseases and has subsequently shown that deleterious mtDNA mutations are common and result in a plethora of complex multi-system diseases which encompasses all of the clinical phenotypes associated with aging including neurological problems such as deafness, blindness, movement disorders, and dementias; cardiovascular disease; muscle degeneration and pain; renal dysfunction; endocrine disorders including diabetes; cancer, etc. Dr. Wallace also showed that mtDNA mutations accumulate with age in post-mitotic tissues resulting in the age-related decline in mitochondrial function. This age-related decline can exacerbate other inherited deficiencies and thus may account for the delayed-onset and progressive course of degenerative diseases and cancer and also provide a molecular explanation for the aging clock.





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.

Dr. Barzilai is the Director of the Institute for Aging Research at the Albert Einstein College of Medicine. He is a Professor of Medicine and Molecular Genetics and a member of the Diabetes Research Center, the Divisions of Endocrinology and Geriatrics. He is also the co-Director of the Montefiore Hospital Diabetes Clinic.



Dr. Barzilai's interests focus on several basic mechanisms in the biology of aging, including the biological effects of nutrients on extending life and the genetic determinants of life span. Indeed, he has discovered the first longevity gene in humans, and is further characterizing the phenotype and genotype of humans with exceptional longevity through an NIH supported Program Project. He also is leading a Program Project to investigate the metabolic decline with aging and its impact on longevity. He received numerous grants, among them ones from the National Institute of Aging (NIA), American Federation of Aging Research, and the Ellison Medical Foundation. Dr. Barzilai has published over 120 peer-reviewed papers, reviews and chapters in textbooks. He is an advisor to the National Institutes of Health on several projects and initiatives and is a member of the Aging Systems and Geriatrics. He serves on the editorial boards of the American Journal of Physiology, Diabetes, Journal of Gerontology and the Science of Aging Knowledge Environment (SAGE KE), and is a reviewer for numerous other journals. Dr. Barzilai was a recipient of numerous prestigious awards, including the Beeson Fellow for Aging Research, the Senior Ellison Foundation award, and the NIA-Nathan Shock Award for his contributions in elucidating metabolic and genetic mechanisms of aging. In his capacity as the Director of the Institute for Aging research at Einstein he coordinates 3 large programmatic approaches to biology of aging, a training grant and additional individual grants.

Pere Puigserver, Ph.D.



Dr. Puigserver joined the faculty of Harvard Medical School as an Assistant Professor in 2006. He received his Ph.D. in Biochemistry from the University of Illes Balears, Spain in 1992. His graduate work was followed by postdoctoral research at the Dana-Farber Cancer Institute and Harvard Medical School. In 2002, he was appointed Assistant Professor at Johns Hopkins University School of Medicine.

The research interests of Dr. Puigserver's lab are on the genetic and biochemical mechanisms underlying the control of intermediary metabolism by nutrients and hormonal signals in mammals. The precise organization and regulation in time and space of these metabolic pathways are essential to maintain nutrient and energy homeostasis in development, survival and health of an organism. Defects in this control will lead to important metabolic abnormalities detected in cancer, aging, diabetes and obesity. The ultimate goal of their research is to understand the coordination, activities and assembly of these regulatory biochemical processes and to lay the foundation for new therapies for metabolic diseases and cancer.

Dr. Tom Prolla's laboratory at the University of Wisconsin, Madison is focused on understanding the molecular basis of the aging process in mammals through the use of large-scale gene expression analysis, and mouse models of accelerated or retarded aging.



They have characterized the gene expression profile of thousands of genes during the aging process of skeletal muscle and brain, and its retardation using DNA microarrays. These studies have allowed them to develop a method to measure the aging process at the molecular level through the use of hundreds of biomarkers. They complement these studies with the generation of transgenic and “knock-out” mice that overexpress or are deficient in specific genes, and analyze the influence of these interventions on the aging process. As an example of this work, they have developed mice that display elevated mitochondrial mutation rates due to a mutation in the POLG gene. These animals have proved very valuable in testing the hypothesis that mtDNA mutations contribute to aging.

Marc Tatar, Ph.D.



Dr. Marc Tatar is Associate Professor in The Division of Biology and Medicine at Brown University. Dr. Tatar has studied the demography, evolution and genetics of aging working with a variety of insect systems to explore the regulation and basic mechanisms of life history traits and senescence. Current work in the Tatar laboratory focuses on genetic analysis of *Drosophila* to understand how insulin/IGF signals and lipid hormones regulate aging, and how these endocrine signals interact with nutrition and reproduction. Dr. Tatar received his Ph.D. from UC Davis in the laboratory of James Carey and completed post-doctoral training at the University of Minnesota with James Curtsinger. Dr. Tatar is an Ellison Senior Scholar, founding Past Joint Editor-in-Chief of *Aging Cell*, and a member of the Board of Review Editors for *Science*.

Dr. Richard Weindruch is a Professor in the University of Wisconsin Department of Medicine's Section of Geriatrics and Gerontology, an investigator with the VA Geriatric Research, Education and Clinical Center (GRECC), and director of the UW-VA Shared Aging Rodent Facility (SARF). He has been the Principle Investigator on a Program Project Grant at the Wisconsin National Primate Research Center since 1994, "Dietary Restriction and Aging in Rhesus Monkeys".



Dr. Weindruch earned his B.S. and M.S. in Biology at the University of Illinois (Urbana) and his Ph.D. (1978) in Experimental Pathology at UCLA under the direction of Dr. Roy L. Walford. He is the author and co-author of more than 170 publications, including *The Retardation of Aging and Disease by Dietary Restriction*, written with Dr. Walford and published in 1988. His scientific awards include the Kleemeier Award in Aging Research, GSA (1998); Nathan W. Shock Award, NIA (2000); Harman Research Award, American Aging Association (2000) and the Glenn Award, GSA (2000).

Dr. Weindruch's research career has focused on the biology of aging and age-related diseases, studying caloric restriction (CR), which slows the aging process and retards the appearance of a broad spectrum of diseases (including most cancers) in diverse animal models. Dr. Weindruch has a long history of investigative interest on the efficacy of CR started at or beyond middle age. In 2001, he and Dr. Tomas Prolla founded LifeGen Technologies, LLC a company focused on nutritional genomics, including the impact of nutrients and caloric restriction on the aging process.

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.

His lab was the first to identify small molecules called STACs that can activate the SIRT pathway and extend lifespan of a diverse species. They also discovered key components of the aging regulatory pathway in yeast and is now focused on finding genes and STACs that extend the healthy lifespan of mice.

Dr. Sinclair obtained a BS with first-class honors at the University of New South Wales, Sydney. In 1995, he received a Ph.D. in Molecular Genetics. 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 and coordinated with Lenny Guarente the article, "Unlocking the Secrets of Longevity Genes" in *The Scientific American*, March 2006 issue.

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.

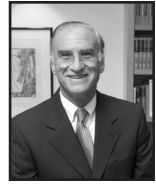
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Peter M. Howley, M.D.



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.



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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.

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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

