

Part IX

Top 100 Geroscience

Analytical Report 2017



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James Adjayes



Title: Director of the Institute for Stem Cell Research and Regenerative Medicine

Bio:

Prof. James Adjaye's was appointed as Director of the newly established Institute for Stem Cell Research and Regenerative Medicine (ISRM) in May 2012. In addition, he is a group leader of the Molecular Embryology and Aging Group at the Max Planck Institute for Molecular Genetics (Berlin). He is the co-ordinator of livSYSiPS - The systems biology of network stress based on data generated from in vitro differentiated hepatocytes derived from individual-specific human iPS cells (ERASysBio Plus).

This multi-national (Germany, UK, Italy USA and Austria) project which uses a systems biology approach to investigate the etiology of non-alcoholic fatty liver disease which comprises a broad spectrum of disease states ranging from manageable stress as in simple steatosis to excessive stress, as in steatohepatitis.

Bruce Ames



Title: Senior Scientist at Children Hospital Oakland Research Institute (CHORI)
Professor, University of California, Berkeley

Bio:

Dr. Ames is a Senior Scientist at Children Hospital Oakland Research Institute (CHORI), director of their Nutrition & Metabolism Center, and a Professor Emeritus of Biochemistry and Molecular Biology, University of California, Berkeley.

He is a member of the National Academy of Sciences and he was on their Commission on Life Sciences. He was a member of the board of directors of the National Cancer Institute, the National Cancer Advisory Board, from 1976 to 1982. He was the recipient of the General Motors Cancer Research Foundation Prize (1983), the Tyler Environmental Prize (1985), the Gold Medal Award of the American Institute of Chemists (1991), the Glenn Foundation Award of the Gerontological Society of America (1992), the Lovelace Institutes Award for Excellence in Environmental Health Research (1995), the Honda Prize of the Honda Foundation, Japan (1996), the Japan Prize, (1997), the Kehoe Award, American College of Occup. and Environ. Med. (1997), the Medal of the City of Paris (1998), the U.S. National Medal of Science (1998), The Linus Pauling Institute Prize for Health Research (2001), and the American Society for Microbiology Lifetime Achievement Award (2001).

His over 555 publications have resulted in his being among the few hundred most-cited scientists (in all fields).

Julie K. Andersen



Title: Professor at Buck Institute for Research on Aging

Bio:

As a renowned expert on age-related neurodegenerative disease, Dr. Andersen is pursuing a wide array of leads toward treatments for complex disorders including Alzheimer's and Parkinson's disease. Recently, the laboratory has joined efforts with the Lithgow laboratory at the Buck institute as part of a collaborative project aimed at identifying novel drugs that eliminate neurotoxic protein deposits in patients diagnosed with these devastating disorders. This would fill critical unmet need for drugs that can block disease progression in the brains of patients already impacted by these conditions.

Lysosomes are organelles that serve as the garbage disposal of the cell. Damaged proteins and other cellular components are broken down by lysosomes in a process known as autophagy. Autophagy has recently been found to be a crucial factor in the removal of damaged neurotoxic proteins associated with several age-related neurodegenerative diseases including Alzheimer's and Parkinson's. Joint research from the Andersen-Lithgow laboratory has recently identified a factor called TFEB as being critical to this process. A recent drug screen performed by their laboratories has identified a novel series of potent, structurally-related compounds that activate TFEB and prevent neurodegenerative phenotypes in *C. elegans* models of Alzheimer's and Parkinson's disease. Independent bioinformatic analysis suggests that these compounds have favorable characteristics for CNS-acting drugs in humans including high brain availability and low toxicity. They propose that these drugs have the wide-ranging potential to impact on all patients diagnosed with age-related neurodegenerative disease. Current efforts are towards pre-clinical studies in order to provide appropriate proof-of-principle to move forward into human phase I trials. A recent independent study from the Andersen laboratory has also identified lysosomal dysfunction as a prime driver of elevated toxic iron levels which occur in these disorders and suggests that these drugs may provide additional benefit by preventing associated brain metal toxicity.

In a recent collaborative effort with the Campisi lab, the Andersen lab has shown that a process known as cellular senescence, previously associated primarily with aging in peripheral tissues, may also play an important role in age-related brain pathologies. The laboratory is working to identify novel 'senolytics', compounds which prevent age-related brain senescence, as a novel potential cure.

The Andersen lab is also involved in identifying potential biomarkers for Parkinson's that may allow early interventional therapy.

Adam Antebi



MAX PLANCK INSTITUTE FOR
BIOLOGY OF AGEING



Title: Director at the Max Planck Institute for Biology of Ageing in Cologne

Bio:

Prof. Adam Antebi did his undergraduate studies at Swarthmore College (Swarthmore, PA) from 1979-83, where he received a Bachelors degree with distinction in Biochemistry. He performed his Ph.D. research at Massachusetts Institute of Technology (Cambridge, MA) from 1985-92 in yeast genetics, and went on with his post-doctoral studies at the Johns Hopkins University (Baltimore, MD) from 1992-97 in *C. elegans* (worm) development. He was an independent group leader at the Max Planck Institute for Molecular Genetics (Berlin) from 1997-2004, where he began studies on worm ageing. In 2004, Antebi returned to the USA where he was promoted to associate professor at the Huffington Center on Ageing, Baylor College of Medicine (Houston, TX). During this time, he also won the prestigious American Federation for Ageing Research Breakthrough in Gerontology Award and the Ellison Medical Foundation Senior Scholar Award in Ageing Research. Since 2008, he has served as a Director at the newly founded Max Planck Institute for Biology of Ageing (Cologne), continuing work on endocrine regulation of life span using worm as the model system.

William Bains



Massachusetts
Institute of
Technology

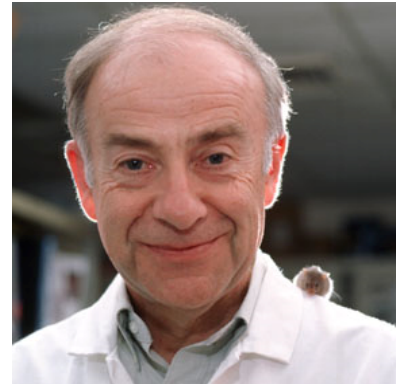


Title: CSO/Founder at Five Alarm Bio Ltd,
Researcher at MIT

Bio:

William is an academic and entrepreneur. After an academic career in the UK and the USA, he joined PA Consulting Group in 1988, and Merlin Ventures in 1996. In 1999 William founded Amedis Pharmaceuticals Ltd, (which was later acquired by Paradigm Therapeutics). He has since founded four other biotech companies, has helped create over 10 others, helping to raise over £60M in earlystage funding for UK biotech start-ups, as well as sitting on the Advisory Boards of the SULIS Fund, Icen Fund and Bath Ventures. William also runs Rufus Scientific, helping entrepreneurs, Universities and start-ups identify how to generate value from visionary science and technology. William continues to be engaged in research at MIT, where he is a visiting scientist researching astrobiology, and as founder and CSO of Five Alarm Bio Ltd.. He is author of over 100 papers on subjects as diverse as drug chemistry, company law and extraterrestrial life, and four books, including *Venture Capital* and *the European Biotechnology Industry* (2008), and teaches company creation and entrepreneurship in postgraduate courses at Cambridge University and the University of Warwick.

Andrzej Bartke]



Title: Distinguished Scholar and Professor of Internal Medicine and Physiology at Southern Illinois University (SIU)

Bio:

Andrzej Bartke, Ph.D., Distinguished Scholar and Professor of Internal Medicine and Physiology at Southern Illinois University (SIU) School of Medicine in Springfield, Illinois, USA. The focus of his research is on the genetic and hormonal control of aging in mammals. Current work is aimed at identifying mechanisms that link reduced growth hormone action with delayed aging and extended longevity. For this work, he is using mutant mice that live longer than normal mice and show various symptoms of delayed aging, including retention of cognitive function and protection from age-related disease.

His career includes work at the Jagiellonian University in Krakow, Poland, Worcester Foundation for Experimental Biology in Shrewsbury, Massachusetts, the University of Texas Health Science Center at San Antonio, and at SIU-Carbondale. He is a past president of the Society for the Study of Reproduction, the American Society of Andrology and the American Aging Association. Dr. Bartke has published more than 750 research papers, review articles and book chapters dealing with reproductive endocrinology, prolactin, growth hormone and aging, and has received numerous awards, in addition to 45 years of continuous NIH funding.

His laboratory was the first to show that mutation of a single gene can extend mammalian longevity and to suggest that the remarkable increase in the lifespan of Ames dwarf mice is due to growth hormone (GH) deficiency. During the last 20 years his lab has extensively characterized phenotypic characteristics of long-lived hypopituitary and GH-resistant mice. This included studies of behavioral, metabolic, molecular and morphological parameters. Contributions of his group to reproductive endocrinology and to the hormonal and genetic control of aging include nearly 600 peer reviewed publications and more than 150 book chapters and review articles. According to the Web of Science Cite Alert, his work has been cited an average of 490 times per year since 1975 and more than 774 times per year since 2004.

Currently Professor Emeritus and Director of Geriatric Medicine in the Departments of Internal Medicine and Physiology at Southern Illinois University School of Medicine in Springfield, IL, Bartke has been contributing to the field for more than five decades. After devoting his early career to the reproductive role of growth hormone and prolactin on testicular physiology, he has become a leader in the field of mammalian aging. His laboratory was the first to show that growth hormone and insulin-like growth factor 1 (IGF1) are major players in the regulation of lifespan in the mammal. He served as President of the American Society of Andrology and the Society for the Study of Reproduction. Bartke has written more than 700 publications and has had continuous National Institutes of Health funding for his research since 1972.

Nir Barzilai



Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY



Title: Professor, Department of Medicine (Endocrinology)
Professor, Department of Genetics
Ingeborg and Ira Leon Rennert Chair in Aging Research
Director, Institute for Aging Research

Bio:

Dr. Nir Barzilai is the director of the Institute for Aging Research at the Albert Einstein College of Medicine and the Director of the Paul F. Glenn Center for the Biology of Human Aging Research and of the National Institutes of Health's (NIH) Nathan Shock Centers of Excellence in the Basic Biology of Aging. He is the Ingeborg and Ira Leon Rennert Chair of Aging Research, professor in the Departments of Medicine and Genetics, and member of the Diabetes Research Center and of the Divisions of Endocrinology & Diabetes and Geriatrics.

Dr. Barzilai's research interests are in the biology and genetics of aging. One focuses on the genetic of exceptional longevity, where we hypothesize and demonstrated that centenarians have protective genes, which allows the delay of aging or for the protection against age-related diseases. In a Program he is leading we take full advantage of phenotypes, DNA, and cells from the Ashkenazi Jewish families with exceptional longevity and the appropriate controls and his group have established at Einstein (over 2600 samples of which ~670 are centenarians) and discovered underlying genomic differences associated with longevity. Longevity Genes Project (LGP) is a cross-sectional, on-going collection of blood and phenotype from families with centenarian proband. LonGenity is a longitudinal study of 1400 subjects, half offspring of parents with exceptional longevity, validating and following their aging in relationship to their genome. The second direction, for which Dr. Barzilai is holding an NIH Merit award that focuses on the metabolic decline of aging, and his team hypothesize that the brain leads this decline. His lab has identified several central pathways that specifically alter body fat distribution and insulin action and secretion by intraventricular or hypothalamic administration of several peptides that are modulated by aging including: Leptin, IGF-1, IGFBP3 and resveratrol.

He has received numerous grants, among them ones from the National Institute on Aging (NIA), American Federation for Aging Research, the Ellison Medical Foundation and The Glenn Medical foundation. He has published over 230 peer-reviewed papers, reviews, and textbook chapters. He is an advisor to the NIH on several projects and serves on several editorial boards and is a reviewer for numerous other journals. Dr. Barzilai is in the board of the American Federation for Aging Research, is its co-scientific director, and has served on several NIA study section. He is also a founder of CohBar Inc., a biotech that develops mitochondrial derived peptides as therapy for aging and its diseases. He is co-PI on the R24 Geroscience (Apollo) grant that is an effort to move the field of aging to translation. Dr. Barzilai has been the recipient of numerous prestigious awards, including the Beeson Fellow for Aging Research, the Ellison Medical Foundation Senior Scholar in Aging Award, the Paul F. Glenn Foundation Award, the NIA Nathan Shock Award, and the 2010 Irving S. Wright Award of Distinction in Aging Research.

He is currently leading an international effort to approve drugs that can target aging. Targeting Aging with METformin (TAME) is a specific study designed to prove the concept that multi-morbidities of

aging can be delayed by metformin, working with the FDA to approve this approach which will serve as a template for future efforts to delay aging and its diseases in humans.

Born in Israel, Dr. Barzilai served as chief medic and physician in the Israel Defense Forces. He graduated from The Ruth and Bruce Rappaport Faculty of Medicine at the Technion-Israel Institute of Technology in Haifa and completed his residency in internal medicine at Hadassah Medical Center in Jerusalem. He served in a refugee camp during the war in Cambodia (1979-1980) and built a nutritional village in the homeland of the Zulu (1983 – Kwazulu). He has completed 2 fellowships at Yale (metabolism) and Corenell (Endocrinology and molecular Medicine). He was an invited speaker to the 4th Israeli President Conference (2012) and a Vatican conference on efforts to enhance cures (2013, 2016). He has also taken part in Global initiatives and spoke at The Milken Global Institute, Asian Megatrends and is an advisor for the Prime Minister of Singapore on Aging. Dr. Barzilai has been on the 'Forward 50, top 50 influence Jews in the US (2011). His work has been profiled by major outlets, including the New York Times, the BBC and PBS' NOVA science now, TEDx talk Science and is the leading feature on the Ron Howard/Jonathan Silberberg/National Geographic film about the Age of Aging.

Chris Benz



Title: Professor of Cancer and Developmental Therapeutics at the Buck Institute for Research on Aging

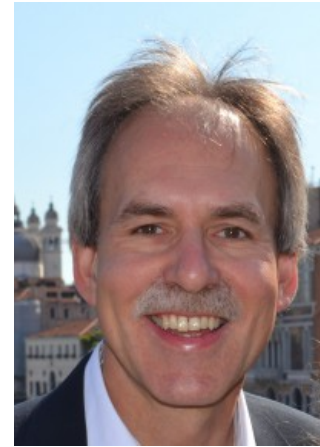
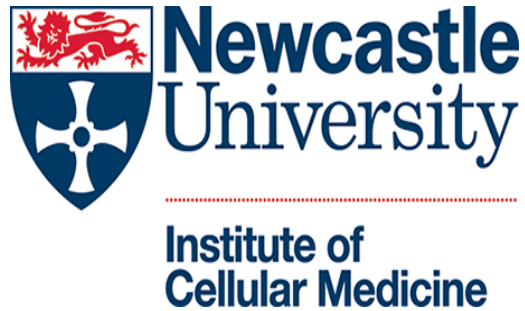
Bio:

Dr. Benz is a practicing oncologist and researcher seeking improved treatments for breast cancer. He obtained his MD from the University of Michigan at Ann Arbor, and took advanced postdoctoral training at Yale University School of Medicine after his internship and residency. He has held faculty positions at Yale and the University of California San Francisco. Dr. Benz joined the Buck Institute in 2000 as a founding faculty member. He continues his oncology practice at UCSF's Carol Franc Buck Breast Care Center, and is a senior member of the UCSF Cancer Center's Breast Oncology Program. Dr. Benz is also the co-principal investigator of the Buck Institute-UCSC Genome Data Analysis Center, one of seven national centers in The Cancer Genome Atlas (TCGA) program. In this effort he works closely with distinguished UC Professor of Biomolecular Engineering, Dr. David Haussler, deciphering TCGA cancer genomes in order to move us closer to personalized cancer care. Both men appear in this video.

Dr. Benz recently spoke at a TEDx conference in Madagascar. The conference was sponsored by the Akbaraly Foundation, which fights against breast and gynecological cancers among women in the African nation.

The Benz lab was among the first to study why age is such an important determinant for the onset and development of breast cancer. In a search for personalized treatments targeted to each patient's individual breast cancer subtype, he explores the genetic and structural differences among different breast cancers. Using model breast cancer cell systems, Dr. Benz looks for these individual variations primarily in the two major pathways linked to breast cancer. In the activated pathway most commonly causing breast cancers after age 50, the tumor cells make excess amounts of the estrogen receptor (ER) protein. Breast cancer can also develop in women at any age via another activated pathway, caused by a genetic abnormality that results in excess production of the ErbB2/HER2 receptor protein. Both the ER and ErbB2/HER2 receptor pathways in breast tumors can vary from one patient to another. Over a decade ago Dr. Benz designed and developed a novel treatment that specifically targets ErbB2/HER2 activated breast cancers, and that therapeutic is finally entering clinical trials this year (MM-302; Merrimack Pharmaceuticals). Dr. Benz predicts that newly discovered differences in the ER and ErbB2/HER2 will yield many more novel therapeutics, and a variety of new companion diagnostics.

Mark Birch-Machin



Quote: «There is now a possibility of finding anti-ageing treatments which can be tailored to differently aged and differently pigmented skin, and with the additional possibility to address the ageing process elsewhere in our bodies.» - Mark Birch-Machin, 2016 https://www.eurekalert.org/pub_releases/2016-02/nu-sms022516.php

Title: Professor of Molecular Dermatology and Associate Dean, Newcastle University Institute of Cellular Medicine

Bio:

Mark Birch-Machin, Ph.D., is Professor of Molecular Dermatology and Associate Dean (Business Development and Commercial Interface) at the Newcastle University Institute of Cellular Medicine. Prof. Birch-Machin pursued his Post-Doctoral career at the University of Oregon (USA), INSERM Paris, University of Toronto as well as Clinical Neuroscience and Biochemistry, Newcastle University (1986-1995) where he held several academic positions in the Department of Dermatology (1996-current). During this time, he was elected Treasurer of the British Society of Investigative Dermatology (BSID) and co-editor of *Experimental Dermatology*. He advanced to the position of Professor of Molecular Dermatology at Newcastle University in 2005 and Associate Dean in 2014.

Prof. Birch-Machin's research group focuses on the response of human skin to ultraviolet radiation particularly within the context of skin ageing and cancer, particularly involving the role of mitochondria and has published extensively including 3 different *Nature* journals. He also has interest in understanding the role of mitochondrial DNA in UV-induced oxidative stress, cancer and the relationship between oxidative stress, nutritional status and skin aging as well as the science and use of sunscreens.

Elizabeth Blackburn



Quote: “Telomeres listen to you, they listen to your behaviors, they listen to your state of mind.” - Blackburn suggesting that individuals have more control over their lives than may be perceived, 2017 <https://www.statnews.com/2017/01/03/aging-control-telomere-effect/>

Title: Nobel Laureate and Morris Herzstein Professor of Biology and Physiology, Department of Biochemistry and Biophysics, University of California San Francisco

Bio:

Born in Australia, Professor Blackburn earned her B.Sc. (1970) and M.Sc. (1972) degrees from the University of Melbourne, and her Ph.D. (1975) from the University of Cambridge in England. She did her postdoctoral work in Molecular and Cellular Biology at Yale University from 1975 to 1977.

In 1978, Professor Blackburn joined the faculty at the University of California Berkeley (UCB), in the Department of Molecular Biology. In 1990, she joined the Department of Microbiology and Immunology at UCSF, where she served as Department Chair from 1993 to 1999. Professor Blackburn is currently a faculty member in the Department of Biochemistry and Biophysics at UCSF. She is also a Non-Resident Fellow of the Salk Institute.

Professor Blackburn has won many prestigious awards throughout her career. She was elected Fellow of the American Academy of Arts and Sciences (1991) and the Royal Society of London (1992). She was elected Foreign Associate of the National Academy of Sciences (1993) and Member of the Institute of Medicine (2000). She served on the President’s Council on Bioethics from 2002 to 2004, and has been awarded honorary degrees by 11 Universities. She received the Albert Lasker Medical Research Award for Basic Medical Research in 2006, and in 2007 was named one of TIME Magazine’s 100 Most Influential People. In 2008 she was the North American Laureate for L’Oreal-UNESCO For Women in Science.

In 2009, Professor Blackburn was awarded the Nobel Prize in Physiology or Medicine.

Maria Blasco



Quote:

The next step that the CNIO Telomeres and Telomerase Group is already working on will be to «generate a new species of mice in which the telomeres of all the cells are twice as long as those in normal mice,» explain Blasco and Varela. «Then, we will be able to address some of the important questions that remain unanswered: would a mouse species with telomeres that are double in length live longer? Is this the mechanism that is used by nature to determine different longevity in genetically similar species? Would this new species present a higher or lower incidence of cancer?» - Maria Blasco 2016 <https://www.sciencedaily.com/releases/2016/06/160602083248.htm>

Title: Director, Centro Nacional de Investigaciones Oncológicas (Spanish National Cancer Research Centre)

Bio:

Maria A. Blasco (Alicante, 1965) obtained her PhD in 1993 for her research at the Centro de Biología Molecular «Severo Ochoa» under the supervision of M. Salas. That same year, Blasco joined the Cold Spring Harbor Laboratory in New York (USA) as a Postdoctoral Fellow under the leadership of C. W. Greider. In 1997 she returned to Spain to start her own research Group at the Centro Nacional de Biotecnología in Madrid. She joined the CNIO in 2003 as Director of the Molecular Oncology Programme and Leader of the Telomeres and Telomerase Group. In 2005 she was also assigned as Vice-Director of Basic Research and in 2011 she was appointed as CNIO Director.

David Botstein



Title: CSO, Google Calico

Bio:

David Botstein was educated at Harvard (A.B. 1963) and the University of Michigan (Ph.D. 1967). He joined the faculty of the Massachusetts Institute of Technology, rising through the ranks from instructor to professor of genetics. In 1987, he moved to Genentech, Inc. as vice president–science, and, in 1990, he joined Stanford University’s School of Medicine, where he was chairman of the Department of Genetics. In July, 2003 he became director of the Lewis-Sigler Institute for Integrative Genomics and the Anthony B. Evnin ’62 Professor of Genomics at Princeton University.

David’s research has centered on genetics, especially the use of genetic methods to understand biological functions. His early work in bacterial genetics contributed to the discovery of transposable elements in bacteria and an understanding of their physical structures and genetic properties. In the early 1970s, he turned to budding yeast (*Saccharomyces cerevisiae*) and devised novel genetic methods to study the functions of the actin and tubulin cytoskeletons. In 1980, he began his theoretical contributions on linkage mapping of the human genome by suggesting, with collaborators, that restriction fragment length polymorphisms (RFLPs) could be used to produce a linkage map of the human genome and to map the genes that cause disease in humans. Linkage mapping of human disease genes became one of the foundations of the Human Genome Project. David also participated in the sequencing the genome of *Saccharomyces cerevisiae*, the first eukaryotic genome to be sequenced.

In the 1990s, David’s research focused on the emerging science of genomics. With J. Michael Cherry, he founded the *Saccharomyces* Genome Database, which continues to be a major international resource that connects genomic sequences with biological functions; in this role he contributed to the founding of the Gene Ontology Consortium. With Patrick O. Brown, he contributed to the development of DNA microarray technology, notably analysis methods that connect gene expression data with the biological functions of genes. Together they adapted microarray technology to classify and study human tumors, resulting in discoveries of tumor subtypes with distinct biology and clinical consequences.

As director of the Lewis-Sigler Institute, David led a team of faculty to develop the innovative new Integrated Science Curriculum (ISC), where the basic ideas of physics, chemistry, computer science, and biology, along with the relevant mathematics, are taught together. David also directed one of the national Centers for Systems Biology established by the National Institute of General Medical Sciences (NIGMS). Under his leadership, a new graduate program, the Program in Quantitative and Computational Biology, was established, as well as the Lewis-Sigler Fellows program for early career scientists. David is now the chief scientific officer of Calico, a startup that aims to take innovative, interdisciplinary approaches toward anti-aging and increased lifespan.

Eric Le Bourg



Title: CNRS Researcher, University Paul-Sabatier (UT3, Toulouse, France),

Bio:

Eric Le Bourg is a French biogerontologist working mainly on *Drosophila melanogaster*. His research interests include learning, effects of mild stress, behavior, demography of human aging and so on. He obtained his “doctorat d’Etat” in 1990 and has published many papers in academic journals, edited books, newspapers, and so on. He has written four books on aging in the French language for the lay public (biology, demographic and social matters, and so on) and academics/students and edited three books, two of them with another editor.

Martin Bran



Title: Professor at the Buck Institute for Research on Aging

Bio:

Martin Brand is an authority on mitochondria, the energy-converting unit of cells and their influence on aging and disease. After receiving his PhD in Biochemistry at the University of Bristol in the UK, he was a postdoctoral fellow at Johns Hopkins University in Baltimore, Maryland, a faculty member at the University of Cambridge and then a group leader at the Medical Research Council. At Cambridge, he began collaborative studies with Buck faculty and joined the Buck Institute in 2008.

The Brand Lab is studying mitochondria, which extract energy from nutrients and distribute it to drive the machinery of life, in a process that also releases free radicals. Believed to be one of the primary actors in the aging process, free radicals are also implicated in numerous age-related diseases, including cancer, heart disease, stroke, and many neurological disorders. Dr. Brand's lab envisions treatments that would minimize the release of free radicals without inhibiting mitochondrial energy metabolism. His lab is collaborating with other Buck labs to evaluate the role of the mitochondria in aging and in such diseases of aging as cancer, diabetes, Parkinson's, Alzheimer's and Huntington's. This research has already opened up new potential drug targets for the control or treatment of these conditions.

Rachel Brem



Title: Ph.D., Associate Professor at the Buck Institute for Research on Aging

Bio:

Longevity, susceptibility to age-associated diseases, and many other attributes relevant for aging vary from one person to another. These differences are due in part to DNA sequence variants somewhere in our genomes—though exactly where is still a mystery in most cases. Worms, flies, and single-celled microbes can serve as powerful models for the study of the principles of genetic variation. Research in the Brem lab uses these model organisms to discover genetic changes that impact aging behaviors and other traits, as well as their evolutionary histories. The Brem lab approach uses large-scale analyses of thousands of genes at once, both computational and experimental, with ongoing work in the following areas:

The genetics of alternative polyadenylation. Most genes contain instructions for the production of a protein—a molecular machine that does work for the cell—including information that regulates the amount and timing of protein production. The regulatory sequence included at the end of a gene is defined by a process called polyadenylation. Cancer cells have dramatic changes in polyadenylation positions across the genomes, and in a few cases, other human diseases have been associated with naturally occurring genetic variants at gene ends. In general, how polyadenylation differs among genetically distinct individuals is almost completely unknown. The Brem lab is dissecting the genetic basis of naturally occurring variation in gene ends and its impact on protein production. The ultimate goal is to understand sequence signals that drive alternative polyadenylation in individual genes as well as the master regulatory factors that carry out processing at many gene ends in parallel. Current work involves cancer samples and wild single-celled brewers' yeast.

Natural variation in yeast and fly aging phenotypes. Longevity, and its dependence on diet, can vary dramatically between genetically distinct individuals and between species. In the vast majority of cases, the genetic basis of these differences is uncharacterized. The Brem lab is mapping genes that underlie naturally occurring differences in lifespan in two model organism systems: wild isolates of single-celled yeast cultured in different growth media, and wild-caught *Drosophila* flies reared on different food sources. The ultimate goal is to identify genes whose role in metabolism and aging is conserved between these simple organisms and humans.

Dr. Brem's Ph.D. thesis work was in the lab of Ken Dill at the University of California, San Francisco, where she developed computational methods to model the forces that drive protein molecules to assemble. As a postdoctoral fellow in the lab of Leonid Kruglyak at the Fred Hutchinson Cancer Research Center in Seattle, she pioneered large-scale experimental strategies to discover variants that underlie differences in gene regulation between genetically distinct individuals, using single-celled yeast as a model. She began her independent research career as an assistant professor at the University of California, Berkeley before joining the Buck Institute in 2014. She was a recipient of the Burroughs-Wellcome Career Award at the Scientific Interface and the Ellison Medical Foundation New Scholar Award in Aging.

William Burhans



ROSWELL
PARK
CANCER INSTITUTE



Title: Associate Member at Roswell Park Cancer Institute

Bio:

William Burhans is working as an Associate Member in the Department of Molecular and Cellular Biology at Roswell Park Cancer Institute. His research interest include: Oncogenic activation of RAS, AKT and other growth signaling pathways induces reactive oxygen species (ROS) and DNA replication stress, both of which cause DNA damage and senescence at early stages of cancer. In cultured human cells, high concentrations of glucose that mimic the effects of hyperglycemia also activate AKT signaling and induce ROS, DNA damage and senescence. Chronic hyperglycemia associated with diabetes and high carbohydrate diets is a risk factor for cancer and likely contributes to aging in humans.

David Burke



Title: Professor, University of Michigan

Bio:

The Burke Laboratory research effort is concentrated in three main areas: (1) quantitative trait analysis of complex, multigenic traits in synthetic populations of the laboratory mouse, (2) the development of engineering systems for microfluidic analysis, and (3) low cost technology systems for health care delivery.

The first research area is a collaborative effort with other investigators at the University of Michigan to identify regions of the mouse genome correlated with inter-individual variation in complex adult phenotypes. Several phenotypic measures are examined in parallel, including body mass, T-cell populations, circulating hormones, bone structure, late-life hearing loss, and cancer incidence. We have identified gene locations associated with several late-life phenotypes, using a reproducible, genetically heterogeneous laboratory mouse «synthetic» population. The second project is a collaborative effort with Dr. Mark Burns (University of Michigan, Department of Chemical Engineering), and is developing a high-throughput DNA genotype analysis systems that can be provided to researchers and clinicians at low cost. The microfluidic devices: a) require human interaction only for initial loading of samples, b) provide consistent experimental processing and quality control, c) decrease sample processing time and human labor, d) reduce reagent costs by reducing the genotyping biochemistry to nanoliter volumes, and e) be fully controlled by integrated circuitry. The third area of work is a newly established cross-disciplinary research effort that will attempt to bring low cost technologies to clinics and clinical researchers. The focus is on developing advanced technologies that are readily manufactured, robust, and distributed.

Dale Bredeesen



Title: CEO, Buck Institute for Research on Aging

Bio:

Dr. Bredeesen is internationally recognized as an expert in the mechanisms of neurodegenerative diseases such as Alzheimer's disease. He graduated from Caltech, then earned his MD from Duke University Medical Center in Durham, NC. He served as Chief Resident in Neurology at the University of California, San Francisco (UCSF) before joining Nobel laureate Stanley Prusiner's laboratory at UCSF as an NIH Postdoctoral Fellow. He held faculty positions at UCSF, UCLA and the University of California, San Diego. Dr. Bredeesen directed the Program on Aging at the Burnham Institute before coming to the Buck Institute in 1998 as its founding President and CEO.

The uniform failure of recent drug trials in Alzheimer's disease has highlighted the critical need for a more accurate understanding of the fundamental nature of Alzheimer's disease. Dr. Bredeesen's research has led to new insight that explains the erosion of memory seen in Alzheimer's disease, and has opened the door to a new therapeutic approach. He has found evidence that Alzheimer's disease stems from an imbalance in nerve cell signaling: in the normal brain, specific signals foster nerve connections and memory making, while balancing signals support memory breaking, allowing irrelevant information to be forgotten. But in Alzheimer's disease, the balance of these opposing signals is disturbed, nerve connections are suppressed, and memories are lost. This model is contrary to popular dogma that Alzheimer's is a disease of toxicity, caused by the accumulation of sticky plaques in the brain. Bredeesen believes the amyloid beta peptide, the source of the plaques, has a normal function in the brain — promoting signals that allow some of the nerve connections to lapse. Thus the increase in the peptide that occurs in Alzheimer's disease shifts the memory-making vs. memory-breaking balance in favor of memory loss. This work has led to the identification of several new therapeutic candidates that are currently in pre-clinical trials.

Dr. Bredeesen's novel insights into the fundamental nature of Alzheimer's disease recently attracted an investment of \$3.5 million toward a \$10 million goal for initial clinical trials of these new therapeutics. This generous support came from the private venture capitalist Douglas Rosenberg, who is helping to fund the Alzheimer's Drug Discovery Network, centered at the Buck Institute. The unit is screening drug candidates to find those that can preserve a healthy balance in the signaling pathways that support memory. Dr. Bredeesen's work on nerve cell signaling is also the focus of a collaboration between the Buck Institute and BioMarin Pharmaceuticals, Inc., which is seeking treatments for a rare form of Alzheimer's disease, early onset Familial Alzheimer's Disease (eFAD), which may develop in people as young as 30 years of age.

Anne Brunet



STANFORD
UNIVERSITY



Quote: ““The range of life spans seen in nature is truly astonishing, and really we have very little insight into how this has evolved or how this works... By having the genome of this fish (the killifish) and comparing it to other species, we start seeing differences that could underlie life span differences both between species and also within a species.” - Anne Brunet, 2015, <https://med.stanford.edu/news/all-news/2015/12/killifish-project-explores-the-genetic-foundation-of-longevity.html>

Title: Professor of Genetics, Stanford University

Bio:

Anne Brunet is a Professor of Genetics at Stanford University. At Stanford, she is also a member of Bio-X, the Cardiovascular Institute, the Cancer Institute, and the Neurosciences Institute. She has received numerous honors and awards throughout her career including the following: Glenn Award, The Glenn Foundation for Medical Research (2007); Alfred P. Sloan Fellow, Sloan Foundation (2006-2008); Klingenstein Fellow, The Esther A. and Joseph Klingenstein Fund (2005-2008); Innovation in Aging Research Award, Pfizer/American Association for Aging Research (2005-2007); Junior Investigator Award, California Institute for Regenerative Medicine (CIRM) (2008-2013); Senior Scholar Award, Ellison Medical Foundation (2009-2013). In 1992, Brunet received her B.Sc. from Ecole Normale Supérieure, Paris, in Molecular Biology. In 1997, she received her Ph.D. from University of Nice, France, in Cell Biology (1997). In 2013, Brunet was a Postdoctoral fellow, Harvard Medical School in Neuroscience.

Rafael de Cabo



National Institutes
of Health



Title: Head of Aging, Metabolism, and Nutrition Unit, Laboratory of Experimental Gerontology

Bio:

After receiving his B.S. and M.S. from the University of Cordoba, Spain, Dr. de Cabo earned his Ph.D. in 2000 from the Department of Foods and Nutrition at Purdue University. Upon completion of his graduate education, he received a postdoctoral position in the Laboratory of Neurosciences at the National Institute on Aging in Baltimore, Maryland. In 2004, he was appointed as a tenure track investigator in the Laboratory of Experimental Gerontology, where he now heads the Aging, Metabolism, and Nutrition Unit (AMNU). The AMNU applies both physiological and tissue-specific molecular approaches to investigate effects of nutritional interventions on basic mechanisms of aging and age-related diseases. Research within his unit strives to identify protective mechanisms invoked by caloric restriction and to evaluate the consequences of dietary interventions on lifespan, pathology, and behavioral function. The AMNU balances the exploration of in vivo rodent, as well as in vitro, paradigms of caloric restriction. Dr. de Cabo is an active member of the Board of the American Aging Association.

Judith Campisi



Quote: “The challenge of keeping older adults healthy throughout their lifespan is going to break the budgets of many countries, that’s why geroscience research is so important in extending healthspan.”

- Judith Campisi, 2015 <http://healthspancampaign.org/2015/10/22/dr-judith-campisi-on-the-changes-in-aging-research/>

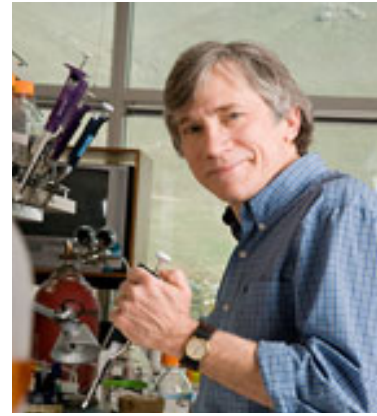
Title: Professor, Buck Institute for Research on Aging

Bio:

Judith Campisi has received international recognition for her contributions to understanding why age is the largest single risk factor for developing a panoply of diseases, ranging from neurodegeneration to cancer. Her highly acclaimed research integrates the genetic, environmental and evolutionary forces that result in aging and age-related diseases, and identifies pathways that can be modified to mitigate basic aging processes.

Dr. Campisi received a PhD in Biochemistry from the State University New York at Stony Brook and completed postdoctoral training at the Harvard Medical School. As an assistant professor at the Boston University Medical School, she became interested in the control of cellular senescence and its role in tumor suppression and aging. She joined the Lawrence Berkeley National Laboratory as a Senior Scientist in 1991. She established a second laboratory at the Buck Institute in 2002. At both institutions, she established a broad program to understand various aspects of aging, with an emphasis on the interface between cancer and aging. The Campisi laboratory has made several pioneering discoveries in these areas, and her research continues to challenge and alter existing paradigms. In recognition of the quality of her research and leadership in the field, she has received numerous awards, including two MERIT awards from the US National Institute on Aging, and awards from the AlliedSignal Corporation, Gerontological Society of America, American Federation for Aging Research, and, most recently, the Longevity prize from the IPSEN Foundation. She currently serves on numerous national and international editorial and advisory boards.

Richard Cawthon



Title: Research Associate Professor, University of Utah

Bio:

A nutraceutical supplement called TA-65, already on the market, is purported to activate telomerase in hopes of producing similar effects. Its active ingredient is a root extract of *Astragalus membranaceus*, a plant often used in traditional Chinese medicine. In a 2010 study, adults averaging age 63 who took the supplement had proportionally fewer short telomeres after a year. Despite lacking a control group, «it was a good paper,» says Richard Cawthon, MD, PhD, a research associate professor of human genetics at the University of Utah whose innovations in telomere testing have helped spur a flurry of recent research. «But whether taking a telomerase activator will help humans stay healthy or live longer is not yet known.» In fact, it may be dangerous. Though the study found no adverse side effects, «I would be certain to be very safe about cancer risks,» Dr. Cawthon says. «If telomerase activators raise the risk of cancer [a theoretical but unproven possibility], then, in principle, a therapeutic regimen that combines telomerase activators and interventions to lower cancer risk may prove optimal for health and longevity.»

Brian Charlesworth



THE UNIVERSITY
of EDINBURGH



Title: Professor at the University of Edinburgh'

Bio:

Brian Charlesworth has been at the forefront of evolutionary genetics research for the last four decades. Using theoretical ideas to design experiments and experimental data as a stimulant for the development of theory, Charlesworth investigates fundamental life processes. His work has contributed to improved understanding of molecular evolution and variation, the evolution of genetic and sexual systems, and the evolutionary genetics of life history traits. In 2010, Charlesworth retired from his faculty position as professor at the University of Edinburgh' Institute of Evolutionary Biology but actively continues to inspire students and conduct research.

George Church



HARVARD
UNIVERSITY



Quote: “One of our biggest economic disasters right now is our aging population. If all those gray hairs could go back to work and feel healthy and young, then we’ve averted one of the greatest economic disasters in history.”

“[Imagine] a scenario [in which] everyone takes gene therapy—not just curing rare diseases like cystic fibrosis, but diseases that everyone has, like aging.” - George Church, Washington Post 2015

Title: Professor of Genetics, Harvard

Bio:

George Church is Professor at Harvard and MIT, co-author of 425 papers, 95 patent publications and the book *Regenesi*s. He developed methods used for the first genome sequence (1994) and genome recoding and million-fold cost reductions since. He co-initiated the BRAIN Initiative (2011) and Genome Projects (1984, 2005) to provide and interpret the world’s only open-access personal precision medicine datasets.

George Church is Professor of Genetics at Harvard Medical School and Director of PersonalGenomes.org, which provides the world’s only open-access information on human Genomic, Environmental and Trait data (GET). His 1984 Harvard PhD included the first methods for direct genome sequencing, molecular multiplexing and barcoding. These led to the first genome sequence (pathogen, *Helicobacter pylori*) in 1994. His innovations have contributed to nearly all «next generation» DNA sequencing methods and companies (CGI-BGI, Life, Illumina, Nanopore). This plus his lab’s work on chip-DNA-synthesis, gene editing and stem cell engineering resulted in founding additional application-based companies spanning fields of medical diagnostics (Knome/PierianDx, Alacris, AbVitro/Juno, Genos, Veritas Genetics) and synthetic biology / therapeutics (Joule, Gen9, Editas, Egenesis, enEvolv, WarpDrive). He has also pioneered new privacy, biosafety, ELSI, environmental and biosecurity policies. He is director of an IARPA BRAIN Project and NIH Center for Excellence in Genomic Science. His honors include election to NAS and NAE and Franklin Bower Laureate for Achievement in Science. He has coauthored 425 papers, 95 patent publications and one book (*Regenesi*s).

Jose Cibelli



MICHIGAN STATE
UNIVERSITY



Title: Professor of Animal Biotechnology at Michigan State University

Bio:

Dr. Jose Cibelli is internationally recognized as one of the pioneers in the area of cellular reprogramming using oocyte-driven protocols. Dr. Cibelli together with his colleagues, were responsible for the generation of the world's first transgenic cloned calves, the first stem cells by nuclear transfer in bovine, the first embryonic stem cells by parthenogenesis in primates and the generation of the first line of iPSCs using oocyte factors alone. His work has been published in numerous scientific journals including Science, Nature Biotechnology, Nature Medicine, Nature Methods, PNAS and JAMA. He has testified about his work in public forums sponsored by the US Food and Drug Administration, the USA National Academy of Sciences, Canadian House of Commons, the USA Department of Agriculture, the United Nations Commission for Human Rights and the British Royal Society.

Irina Conboy



Berkeley
UNIVERSITY OF CALIFORNIA



Quote: «Aging is a synonym with diseases... When we are young, we don't have these diseases. But when we are old, it doesn't matter what background or gender or culture, we all have them. If we can better understand the aging process, then we don't need to have different hospitals, departments, and institutes that deal with each disease.» - Irina Conboy, 2015 https://motherboard.vice.com/en_us/article/engineering-the-end-of-aging

Title: Associate Professor in Bioengineering, University of California, Berkeley
Institution

Bio:

Irina Conboy's education includes a PhD from Stanford University in Molecular and Cellular Immunology (1998). Professional experience throughout her career includes the following: 2006-present Reviewer for CIRM Training Grant Program at UC Berkeley; 2006-2007 Faculty Mentor of CIRM training grant T1-00007; 2005-Present Faculty Mentor for UC Berkeley Chapter of Student Society for Stem Cell Research; 2004-present Assistant Professor of Bioengineering and QB3 Investigator, UC Berkeley; 1999-2004 Postdoc and Instructor, Neurology, Stanford University School of Medicine.

Antonei Csoka



Title: Director in Epigenetics Laboratory, Howard University, Washington DC, USA

Bio:

Antonei B. Csoka, Ph.D. is an Assistant Professor in the Department of Anatomy at Howard University, Washington DC, where he directs the Epigenetics Laboratory. Dr. Csoka received his B.S. in Genetics from the University of Newcastle, U.K., his M.S. in Molecular Pathology from the University of Leicester, U.K., and his Ph.D. in Cell and Molecular Biology from the University of Debrecen, Hungary. He performed postdoctoral research at the University of California, San Francisco, where he cloned the human hyaluronidase genes, which are involved in fertilization, embryonic development, and cancer. As a postdoctoral research associate at Brown University, Dr. Csoka was a member of team that identified the causative gene for Hutchinson-Gilford Progeria Syndrome (Progeria), a disease with many features of “accelerated aging.” The identification of the gene for Progeria is providing many insights into the mechanisms of normal aging. At Howard, Dr. Csoka is developing animal models of progeria, studying the role of cellular senescence in human aging, and investigating the potential of stem cells, cellular reprogramming and epigenetic rejuvenation for the treatment of age-related diseases

Ana Maria Cuervo



Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY



Title: Co-director of the Einstein Institute for Aging Research, Israel

Bio:

Dr. Cuervo is co-director of the Einstein Institute for Aging Research, and a member of the Einstein Liver Research Center and Cancer Center. In 2001 she started her laboratory at Einstein, where she studies the role of protein-degradation in aging and age-related disorders, with emphasis in neurodegeneration and metabolic disorders.

Dr. Cuervo's group is interested in understanding how altered proteins can be eliminated from the cells and their components recycled. Her group has linked alterations in lysosomal protein degradation (autophagy) with different neurodegenerative diseases including Parkinson's, Alzheimer's and Huntington's disease. They have also proven that restoration of normal lysosomal function prevents accumulation of damaged proteins with age, demonstrating this way that removal of these toxic products is possible. Her lab has also pioneered studies demonstrating a tight link between autophagy and cellular metabolism. They described how autophagy coordinates glucose and lipid metabolism and how failure of different autophagic pathways with age contribute to important metabolic disorders such as diabetes or obesity.

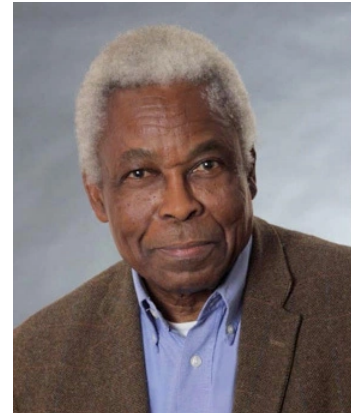
Dr. Cuervo is considered a leader in the field of protein degradation in relation to biology of aging and has been invited to present her work in numerous national and international institutions, including name lectures as the Robert R. Konh Memorial Lecture, the NIH Director's, the Roy Walford, the Feodor Lynen, the Margaret Pittman, the IUBMB Award, the David H. Murdoxk, the Gerry Aurbach and the Harvey Society Lecture. She has organized and chaired international conferences on protein degradation and on aging, belongs to the editorial board of scientific journals in this topic, and is currently co-editor-in-chief of *Aging Cell*.

Dr. Cuervo has served in NIH advisory panels, special emphasis panels, and study sections, the NIA Scientific Council and the NIH Council of Councils and has been recently elected member of the NIA Board of Scientific Counselors. She has been elected member to the Royal Academy of Sciences of Spain and has received numerous awards for the pioneerign work of her team such as the 2005 P. Benson Award in Cell Biology, the 2005/8 Keith Porter Fellow in Cell Biology, the 2006 Nathan Shock Memorial Lecture Award, the 2008 Vincent Cristofalo Rising Start in Aging Award, the 2010 Bennett J. Cohen Award in Aging Biology, the 2012 Marshall S. Horwitz, MD Faculty Prize for Research Excellence and the 2015 Saul Korey Prize in Translational Medicine Science.

Lloyd Demetrius



HARVARD
UNIVERSITY



Title: Scientist at Department of Organismic and Evolutionary biology, Harvard University, USA and Max Planck Institute for Molecular Genetics at Berlin, Germany

Bio:

Lloyd A. Demetrius is a mathematician and theoretical biologist at the Max Planck Institute for Molecular Genetics at Berlin, Germany, and the Department of Organismic and Evolutionary biology, Harvard University. He is best known for the discovery of the concept, evolutionary entropy, a statistical parameter that characterizes Darwinian fitness in models of evolutionary processes at various levels of biological organization - molecular, organismic and cultural. Evolutionary entropy, an analogue of the Gibbs entropy in statistical physics, is the cornerstone of directionality theory, an analytical study of evolution by variation and selection. The theory has applications to: a) the development of aging and the evolution of longevity; b) the origin and progression of age related diseases such as cancer, and neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease; c) the evolution of cooperation and the spread of inequality

Jan van Deursen



Title: Lab Director, Department of Pediatric and Adolescent Medicine at Mayo Clinic

Bio:

Jan van Deursen, Ph.D., received his Bachelor of Science in biology from the University of Nijmegen in the Netherlands, and continued at that institution to earn his master's degree and doctorate in molecular and cellular biology.

In 1994, Dr. van Deursen started his independent research career as a faculty member at the St. Jude Children's Research Hospital in Memphis, Tennessee. During the next five years, he conducted research in his laboratory, elucidating the mechanisms that underlie nuclear-cytoplasmic transport and how regulation of this process contributes to leukemia. He also developed a mouse gene knockout core facility.

In 1999, Dr. van Deursen was recruited to the Department of Pediatric and Adolescent Medicine at Mayo Clinic, where he again developed and continues to direct an effective mouse gene knockout core facility. His research program investigates far-ranging topics such as the role of aneuploidy in development of cancer and the molecular mechanisms that contribute to aging. His work has been published in top journals, including *Nature*, *Science* and *Cell*, and is widely cited.

One of Dr. van Deursen's notable discoveries originated from work published in 2004, when his group generated a new mouse strain defective in chromosome replication due to reduced cellular levels of the spindle assembly checkpoint protein BubR1. Dr. van Deursen's lab found that these mice aged much more quickly than normal, dying of old age before they were one year old. Normal mice live 2.5 to three years.

In studying these mice, Dr. van Deursen's lab discovered that the animals showed rapid development of prematurely senescent cells — that is, cells that had turned on the p16 cell cycle inhibitor gene (INK4A), which made them stop dividing and begin to secrete inflammatory cytokines. To determine whether the senescent cells were not just indicators that the mice were aging rapidly but might actually be causing the rapid aging, fast-aging mice were crossed to mice that lacked the p16 gene.

The lab found that the cross-bred mice no longer suffered from the rapid development of age-associated syndromes, including poor vision from cataracts and loss of muscle and fat. This finding verified the hypothesis that the relatively rare senescent cells in tissues were somehow causing the surrounding normal cells to decay and develop problems associated with old age.

Next, in collaboration with the research group of James L. Kirkland, M.D., Ph.D., Dr. van Deursen's lab developed a different strategy to specifically remove the problematic senescent cells from mice. They generated a special transgenic strain of mice that would delete senescent cells if they were exposed to a specific drug. Dr. van Deursen's and Dr. Kirkland's labs found that this treatment not only removed the senescent cells but also prevented the rapid aging in these mice.

The team's work was published in Nature in November 2011 and captured the imagination of many in the scientific community as well as the public. Here, for the first time, was a clear demonstration of an intervention that dramatically delayed the deleterious effects of aging on multiple organs in a mouse model, in a way that, at least conceptually, could eventually be applied to humans.

In addition to generating a large number of exciting new projects in Dr. van Deursen's laboratory, this discovery has also prompted other scientists to begin studying the role of p16 and cellular senescence in mouse and human aging. As a concrete demonstration of the importance of this discovery, the journal Science selected the lab's Nature paper as one of the top 10 discoveries of the year in 2011. In addition to Dr. van Deursen's scientific accomplishments, he is very active as an educator, having mentored more than 60 students, residents, postdoctoral fellows and junior faculty members. He has a strong commitment to supporting the successful careers of young upcoming scientists.

Dr. van Deursen has been recognized by numerous awards. He is the Vita Valley Professor of Cellular Senescence and was Mayo Clinic Investigator of the Year for 2012. He hopes that one day centenarians will be qualifying for the Boston Marathon.

Andrew Dillin



Berkeley
UNIVERSITY OF CALIFORNIA



Quote: «It is ... looking increasingly likely that pharmacological manipulation of these ... pathways could form the basis of new preventative medicines for diseases aging, and aging itself.” - Andrew Dillin, 2010 <http://www.reuters.com/article/us-ageing-disease-idUSTRE64I6HV20100520>

Title:

University of California, Berkeley
Thomas and Stacey Siebel Distinguished Chair in Stem Cell Research
Howard Hughes Medical Investigator
Professor, Department of Molecular and Cell Biology

Bio:

Andrew Dillin is a Howard Hughes Medical Investigator and the Thomas and Stacey Siebel Distinguished Chair in Stem Cell Research at the Department of Molecular and Cell Biology at Berkeley . His lab studies the loss of protein homeostasis in aging, particularly in *C. elegans*.

His lab specifically looks at the manipulation of stress response pathways within a single cell type or subcomponents within specific cell types. In particular, his lab found a cell non-autonomous mitochondrial stress response that can be transmitted to very distal cells.

Larry Donehower



Title: Professor, Baylor College of Medicine

Bio:

Lawrence A. Donehower, Ph.D., received the award for his research on the function of a gene, p53, that is critical for protecting humans from early cancers. Dr. Donehower generated mice with an altered p53 tumor suppressor gene that confers cancer resistance and accelerated aging. This p53 mutant mouse suggests that not only is the p53 gene important in preventing cancers, but that it may also play a major role in influencing the aging process. Thus, cancer protection can be augmented but perhaps at the risk of accelerating various aspects of aging. Dr. Donehower is now studying these p53 mutant mice to better understand the molecular, cellular, and hormonal bases of normal aging processes.

Monica Driscoll



RUTGERS



Title: Professor, Dept.of Molecular Biology and Biochemistry. Rutgers University

Bio:

Professor Monica Driscoll, is interested in developmental neurogenetics, molecular genetics of neuronal cell death, mechanosensory transduction in touch and feeling, molecular mechanisms of aging. One of the looming mysteries in signal transduction is the question of how mechanical signals such as pressure or force delivered to a cell are interpreted to direct biological responses. A long-standing problem in the mechanotransduction field has been that genes encoding mechanically-gated channels eluded cloning efforts resulting in a large gap in our understanding of their function. A new family of ion channels (the degenerin channels) are hypothesized to function as the central mediators of touch transduction and proprioception (how the body maintains coordinated movement) in *C. elegans*. Her lab combines genetic molecular and electrophysiological approaches to determine and compare the composition/regulation of mechanosensitive complexes in an effort to contribute to the understanding of the function of this newly discovered channel class.

Lisa Ellerby



Title: PhD, Professor at the Buck Institute for Research on Aging

Bio:

Dr. Ellerby is an expert on cell death in neurodegenerative disorders such as Huntington's disease, a progressive inherited disorder that attacks both motor coordination and thinking ability. Dr. Ellerby and her Buck colleague Dr. Robert Hughes recently discovered a new lead to potential drug therapies for Huntington's disease by focusing on a mysterious protein linked to the illness, the huntingtin protein (Htt). Huntington's disease stems from a gene mutation that produces an abnormal form of the huntingtin protein, which breaks down into toxic fragments. These fragments accumulate in neurons, which malfunction and eventually die. Dr. Ellerby and her team identified a set of enzymes that help split up Htt into fragments, and whose activity contributed to nerve toxicity. In a novel discovery, the lab found this harmful activity in a class of enzymes already implicated in stroke, cancer, and other disorders. Drug researchers have already developed experimental compounds to inhibit these enzymes, called the matrix metalloproteinases (MMPs). Dr. Ellerby's work suggests that inhibiting the MMPs may lessen symptoms of Huntington's disease and prevent nerve cell death. In a 2010 article about this research, Dr. Ellerby was one of the lead authors of the cover story in the prestigious scientific journal *Neuron*.

The Ellerby lab is also exploring possible methods to stimulate the growth of new nerve cells to replace those lost in Huntington's disease sufferers.

Dr. Ellerby received her PhD in Chemistry from the University of California, Santa Cruz. She took postdoctoral training in the Department of Biochemistry and Chemistry at the University of California, Los Angeles. She was a Senior Research Associate in Neurodegenerative Disease and Apoptosis and a Co-Investigator with the Program on Aging at the Burnham Institute in La Jolla, CA before she joined the Buck Institute in 2000.

Preston Estep

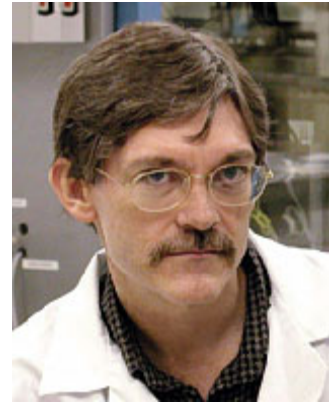


Title: Director of Gerontology at Harvard Medical School
Co-founder and Chief Scientific Officer of Veritas Genetics

Bio:

Preston Estep, Ph.D. is Director of Gerontology and manages genome sequencing as part of the senior management team of the PGP at Harvard Medical School. He is also a co-founder and Chief Scientific Officer of Veritas Genetics, the company that first broke the \$1000 barrier for whole genome sequencing and interpretation. He is a co-founder and adviser to multiple startup companies and non-profit organizations. Dr. Estep is Chairman of the Mind First Foundation, which he co-founded with thought leaders at Harvard Medical School and in the healthcare industry. The foundation is dedicated to scientific mind research and the development of better diagnoses and therapies for brain disorders. In 2016 Dr. Estep published *The Mindspan Diet*, a comprehensive synthesis of scientific evidence on the causes of Alzheimer's disease and cognitive decline.

Gregory Fahy



Title: Vice President and Chief Scientific Officer at Twenty-First Century Medicine
Editor-in-Chief of «*The Future of Aging: Pathways to Human Life Extension*»

Bio:

Greg earned his B.S. from the University of California at Irvine in 1972 and his Ph.D. from the Medical College of Georgia in 1977 for work on basic aspects of cryobiology. He spent the next 18 years developing methods for preserving whole organs at cryogenic temperatures at the American Red Cross in Maryland. In 1980, he conceived of preserving organs by vitrification. He published the first proof of principle of this concept in *Nature* in 1985 using mouse embryos as a model system, an event that led to the wide use of vitrification in academic and commercial animal husbandry as well as in human assisted reproduction

In 1995, he won the Grand Prize for Medicine from INPEX for his invention of the first effective computer-operated equipment for perfusing organs with cryoprotective agents. The same year, he left the Red Cross to become Chief Scientist of two biotechnology companies and the Head of the Tissue Cryopreservation Section of the Transfusion and Cryopreservation Research Program at the Naval Medical Research Institute in Bethesda, Maryland. In 1998 he became the Chief Scientific Officer and Vice President of 21st Century Medicine, where he invented several new principles in cryopreservation that have been extraordinarily effective in practical applications ranging from tissues to whole organs.

Greg's efforts have recently raised the question of whether human suspended animation might be an attainable goal that might allow the human species to survive in deep time as a result of enabling migration from the earth to other habitats in the cosmos.

Greg is a sought-after speaker and problem-solver. He is on the Board of Directors of several organizations concerned with cryopreservation or aging, serves on the Editorial Board of *Cell Preservation Technology* and *Rejuvenation Research*, and has served as a reviewer for numerous journals and granting bodies. He has over 20 patents in fields related to cryopreservation, aging, transplantation, metabolic protection, and the reversal of autoimmunity and immunosenescence, and has many publications in the fields of cryobiology, aging, and nanotechnology.

Richard Faragher



University of Brighton



Title: Professor of Biogerontology at the University of Brighton

Bio:

Richard Faragher is Professor of Biogerontology at the University of Brighton and is past Chair of both the British Society for Research on Ageing and the International Association of Biomedical Gerontology. He is the first British citizen to be elected to the Board of Directors of the American Federation for Aging Research (AFAR), the leading US non-profit organisation supporting and advancing healthy aging through biomedical research.

His primary research interest is in uncovering the causal mechanisms driving the human ageing process and in translating that knowledge into effective interventions which will improve the wellbeing of older people. His particular interest is the phenotype of “senescent” cells. These are cells which can no longer divide, frequently as a result of tissue turnover through life, and which accumulate in mammalian tissue. It has been shown that the deletion of these cells in animal models improves multiple markers of health, opening radical prospects for the improvement of human health in the future.

In July 2016, Richard received the highest honour of the British Society for Research on Ageing (BSRA) - the Lord Cohen of Birkenhead Medal for services to gerontology. The BSRA is the oldest scientific society in the world devoted to researching the biology of ageing.

Miguel Ferreira

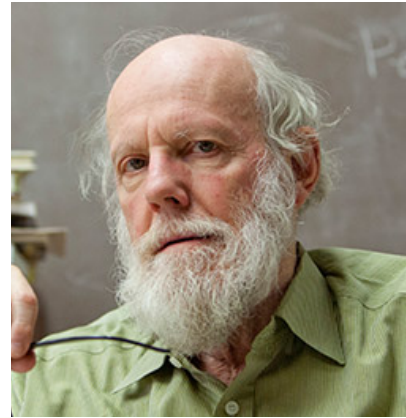


Title: Professor at Instituto Gulbenkian de Ciência

Bio:

Miguel Ferreira have expertise in telomere biology, DNA repair both in fission yeast and zebrafish. His research focused on the molecular mechanisms of cell division and how damaged DNA (stemming DNA replication and telomeres) signals to block cell division in normal cells and how this process is deregulated in cancer cells. This work resulted in a series of studies in chromosome biology and DNA repair (Ferreira et al. *Mol Cell Biol* 1999; Ferreira & Cooper *Mol Cell*, 2001; Ferreira & Cooper *Genes & Dev*, 2004). Since 2006, as an independent group leader, he focused on the molecular mechanisms underlying genome protection and their consequences (Carneiro et al. *Nature* 2010; Reis et al. *EMBO J* 2012; Avelar et al. *Nat Comm* 2013; Hentges et al. *CellReports* 2014). ~ In 2009, he decided to incorporate cancer research in our laboratory. Two events impelled him to take on this challenge using zebrafish as a model system: 1- the advent of zebrafish as a cancer model and 2- they showed that zebrafish telomerase mutants anticipate spontaneous cancer to early age similar to further old age phenotypes (Henriques et al. *PLoS Genetics* 2013; Carneiro et al. *PLoS Genetics* 2016). His goal is to use the knowledge acquired on the molecular nature of telomere protection to understand the consequences of its failure at the organism level. Their base hypothesis implies that telomere dysfunction signals a cascade of events that triggers cellular senescence and organism aging (described in Henriques & Ferreira *Curr Opin in Cell Biology* 2012). He plans to test this idea by manipulating telomere dysfunction (in a time- and tissue-specific manner), using transgenic zebrafish. His vision is that enabling timely telomere protection in a few key tissues will enhance organism tissue regeneration and, as a consequence, reduce the frequency of age-associated diseases, namely, cancer.

Caleb Finch



Quote: "We are born dying." - Caleb Finch, 2016 <https://www.sciencenews.org/article/brain-blueprint-aging-set-early-life?mode=magazineandcontext=192078>

Title: ARCO/William F. Kieschnick Professor in the Neurobiology of Aging and University Professor, USC Leonard Davis School of Gerontology

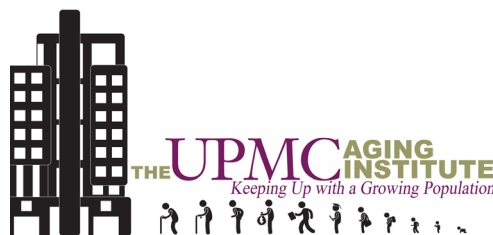
Bio:

Caleb Ellicott Finch (born July 4, 1939) is a professor at the University of Southern California's Leonard Davis School of Gerontology who studies aging in humans, with expertise in cell biology and Alzheimer's disease.

He was the founding Director of USC's NIH funded Alzheimer Disease Research Center in 1984, and is currently co-Director. In 1989, the university made him one of its twelve «University Distinguished Professors». He is a full professor in Gerontology and Biological Sciences, and an adjunct professor in departments of Anthropology, Psychology, Physiology, and Neurology. He was the Chair of the National Research Council Committee on Biodemography of Aging. He is co-author of 520 scientific papers and four books, most recently *The Biology of Human Longevity* (Academic Press, 2007). He currently serves on the Scientific Advisory Board for Cure Alzheimer's Fund.

Finch and his colleague at the USC Davis School of Gerontology Eileen Crimmins have developed a unique interdisciplinary upper division course (Health, Stress, and Aging), which combines biomedical, demographic, and psychosocial perspectives of the human lifespan.

Toren Finkel



Title: Director of the University of Pittsburgh Aging Institute

Bio:

Toren Finkel, MD, PhD, a physician-scientist renowned for his research on the basic science of aging, has been named Director of the UPMC-University of Pittsburgh Aging Institute and a Professor of Medicine in the Pitt Department of Medicine's Division of Cardiology.

Currently, Finkel is Chief of the Center for Molecular Medicine in the National Heart, Lung, and Blood Institute (NHLBI). He will begin his position in Pittsburgh later this summer.

After earning a bachelor's degree in physics from the University of Maryland, Finkel obtained his MD and PhD degrees from Harvard Medical School in 1986. He completed residency training in internal medicine at Massachusetts General Hospital in Boston, and followed it with a fellowship in cardiology at Johns Hopkins Medical School.

Finkel began his 25-year career with the National Institutes of Health in 1992 when he joined the National Heart, Lung, and Blood Institute as an investigator in its Cardiology Branch. He eventually became Chief of that branch, and later, chief of the NHLBI's Translational Medicine Branch.

His lab's research focuses on the role of cellular metabolism and oxidative stress in aging and age-related diseases. In his new role at the Aging Institute, Finkel will direct and support research to identify signaling pathways and therapies that target the process of aging.

He is the author or coauthor of more than 200 publications. According to Google Scholar, Finkel ranks as the 12th most highly cited author in aging and the 11th most cited in cardiovascular disease. Among his many recognitions is his 2013 induction as a Fellow of the American Association for the Advancement of Science.

Michael Fossel



Quote: “«Ageing is dynamic, not static. Never mind the low-hanging fruit. [...] Go for the important one! The reason to [reverse aging] is not to double somebody’s lifespan. The reason to do this is because people out there are hurting. They are frightened. They are terrified by the things that happen to them when they get disease. The reason to do this is because we are human and we should be working at this. It’s not playing God, it is working at being human. It’s compassion. It’s not a matter of living longer, it is a matter of making people healthy again.» - Michael Fossel, 2014 <https://www.fightaging.org/archives/2014/11/an-interview-with-michael-fossel/>

Title: Founder/President, Telocyte

Bio:

Michael Fossel graduated cum laude from Phillips Exeter Academy, received a joint BA and MA in psychology in four years from Wesleyan University in Connecticut, and, after completing a PhD in neurobiology at Stanford University in 1978, went on to finish his MD at Stanford Medical School in two and a half years. He was awarded a National Science Foundation Fellowship and taught at Stanford University, where he began studying aging, emphasizing premature aging syndromes. Dr. Fossel was a Clinical Professor of Medicine at Michigan State University for almost three decades and taught the Biology of Aging at Grand Valley State University.

He has personal experience with a number of biotech companies, both as an inside or angel investor (Geron, Sierra Sciences), an advisor (Geron, Sierra Sciences, Phoenix Biomolecular, Betterhumans, Androcyte, PhysioAge, BioViva), and an executive (Cerner, Double Helix). He is the medical advisor for the Dementia Society of America, and has been a member of numerous scientific organizations including the American Association for the Advancement of Science, the American Aging Association, the American Gerontological Society, the American Society on Aging, the American Geriatrics Society, and the Alzheimer’s Association ISTAART, among others.

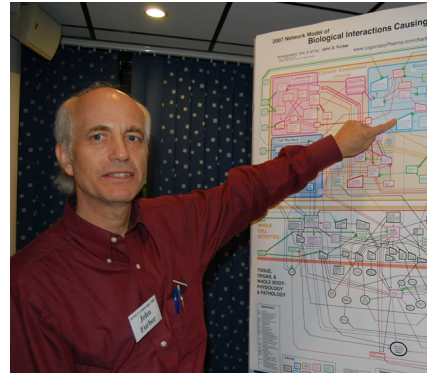
He has lectured at the National Institute for Health and the Smithsonian Institute, and still lectures at universities, institutes, and conferences internationally. He is frequently interviewed regarding aging by major media in the US and worldwide. He was founding editor of Rejuvenation Research.

John D. Furber



LEGENDARY
PHARMACEUTICALS

www.LegendaryPharma.com



Title: CEO/Founder of Legendary Pharmaceuticals

Bio:

John D. Furber is the CEO and founder of Legendary Pharmaceuticals. He is an entrepreneur and scientist who has been studying the biology of aging and regeneration for more than 20 years. He earned a Bachelor of Arts degree in Physics and Mathematics from the University of California at Santa Cruz in 1975, and a Master of Science degree in Biological Sciences from the University of California at Irvine in 1990. Between degrees, he served the United States Congress as a Technology Policy Analyst in the Congressional Office of Technology Assessment.

Mr. Furber was a principal in starting five companies during the 1980s and 90s. Currently, he is running a biotechnology company, Legendary Pharmaceuticals, which is engaged in the discovery of pharmaceutical drugs and gene therapies able to repair and reverse accumulating molecular damage to subcellular mitochondria, lysosomes, nuclei, and extracellular proteins in order to prevent and treat serious, late-onset diseases commonly associated with aging. Legendary Pharmaceuticals is a privately-held small business.

Teaching: During the 1990's, he taught Human Biology at Skyline College, near San Francisco, and taught Management Decision Making at Menlo College, near Stanford. During the 1980's he taught Biology laboratory courses at the University of Kentucky and the University of California. He also started and ran a successful private tutoring service during the 1990's, which helped many students to learn Physics, Mathematics, Statistics, Chemistry, Biology, and Computers. Currently, he frequently lectures on topics related to analyzing the Systems Biology of Aging to find effective therapeutic targets to prevent or treat diseases of aging.

Early career: Mr. Furber served four years on Capitol Hill at the Office of Technology Assessment of the United States Congress. In 1979, he joined Solar Electric International, which set up photovoltaic-powered irrigation systems for World Bank projects in developing countries. He also started Pleasant Valley Software Corporation (1984) with a consortium of investors from Europe, India, Canada, and the US.

Laboratory training: In the mid-1980's he changed careers and began graduate school to study the biology of aging and regeneration, first at the University of Kentucky, and then at UC Irvine. During graduate school, his studies emphasized Developmental and Molecular Biology.

Currently, at Legendary Pharmaceuticals, he is engaged in pharmacological research involving therapeutic target identification, screening and optimizing candidate therapies, organic synthesis, purification, and testing, as well as ensuring regulatory compliance.

International: Mr. Furber has made multiple trips to Europe, China, India, the Middle East, Southeast Asia, Africa, and the Caribbean to consult, lecture, and study. These have given him an appreciation of other cultures, and advanced possibilities for foreign collaborations.

Professional affiliation: His professional memberships include:

- American Aging Association (former Vice President & Board of Directors)
- Gerontological Society of America
- International Aging Research Portfolio (IARP) (Scientific Advisory Board)
- Aging Intervention Foundation (Scientific Advisory Board)
- Alzheimer Research Forum
- Federation of American Scientists
- The Lifeboat Foundation (Advisory Board)
- Mitochondria Interest Group at the U.S. National Institutes of Health.
- Oxygen Club of California

He is a frequent contributor at meetings in the fields of aging, mitochondria, autophagy, and oxidative stress. He served on the Board of Directors of the American Aging Association from 2000 to 2011, and was Vice President in 2008-2009.

John has created several web pages which provide useful links for researchers and the lay public interested in Aging, Nutrition, Bioinformatics, Genomics, and Molecular Cell Biology. For further details, please see <http://www.LegendaryPharma.com/jdf/>.

Vadim Fraifeld



Title: Senior Researcher, Ben-Gurion University of the Negev, Israel.

Bio:

Vadim Fraifeld, Ph.D., M.D. is Senior Researcher, Department of Microbiology and Immunology, Center for Multidisciplinary Research in Aging, Ben-Gurion University of the Negev, Beer-Sheva, Israel.

Vadim's research interests are biology of aging and longevity and thermoregulation. His research projects are genomic and proteomic analyses of exceptional longevity, analysis of protein profile in aging muscle using MALDI-TOF mass spectrometry: implications in sarcopenia, mechanisms of anti-tumor activity of polyphenolic compounds, and alternative pathways of arachidonate metabolism, fever, and aging.

He coauthored Evidence supporting involvement of leukotrienes in LPS-induced hypothermia in mice, p66ShcA and aging: modulation by longevity-promoting agent aurintricarboxylic acid, Superoxide dismutase, catalase and glutathione peroxidase activities in the liver of young and old mice: linear regression and correlation, Dietary restriction modifies fever response in aging rats, Glutathione S-transferase hGSTM3 and ageing-associated neurodegeneration: relationship to Alzheimer's disease, Mitochondrial Genome Anatomy and Species-Specific Lifespan, and From Disease-Oriented to Aging/Longevity-Oriented Studies.

Vadim earned his M.D. at the Lvov Medical Institute, Lvov, Ukraine in 1974 and his Ph.D. at the Institute of Gerontology, Kiev, Ukraine, in 1989. Watch his SENS 3 presentation Do mitochondrial DNA and metabolic rate complement each other in determination of the mammalian maximal life span?

Claudio Franceschi



Quote: “Within this perspective, healthy aging and longevity are likely the result not only of a lower propensity to mount inflammatory responses but also of efficient anti-inflammatory networks, which in normal aging fail to fully neutralize the inflammatory processes consequent to the lifelong antigenic burden and exposure to damaging agents. Such a global imbalance can be a major driving force for frailty and common age-related pathologies, and should be addressed and studied within an evolutionary-based systems biology perspective.” - Claudio Franceschi, 2007 <http://www.sciencedirect.com/science/article/pii/S0047637406002491>

Title: Full Professor of Immunology, School of Medicine, University of Bologna

Bio:

Prof. Claudio Franceschi (M) is Full Professor of Immunology at the School of Medicine where he leads the Lol. He is the founder and Director of Interdepartmental Center “L. Galvani” for studies on Bioinformatics and Biocomplexity and former Scientific Director of the Italian National Research Center on Aging, INRCA, Ancona, Italy, the largest Italian Institution of the Ministry of Health on research and care of the elderly (1,500 employees, 300 researchers and MD). He has published more than 550 papers in peer-reviewed journals (h-index = 80, 26,413 citations; h-index in the last 10 years = 48, 11,015 citations). He coordinates the large EU projects GEHA (GEnetics of Healthy Ageing, 204-2010) and NU-AGE (Nutrition and Ageing, 2011-2016) and is a partner in (MARK-AGE, biomarkers of ageing, 2008-2013; MYOAGE, sarcopenia, 2009-2013; IDEAL, epigenetics and ageing, 2010-2015; MISSION T2D immune system model of type 2 diabetes, 2013-2016). He is a member of the editorial board of several journals (Aging Res Review, Aging, Exp Gerontology, Mechanisms of Ageing and Development). He pioneered the field of the biological basis behind human longevity by proposing centenarians as models of successful aging and by conceptualising theories on human aging (“remodelling”; “inflamm-ageing”).

Robert Freitas



Title: Senior Research Fellow, Institute for Molecular Manufacturing

Bio:

Robert A. Freitas Jr., J.D., published the first detailed technical design study of a medical nanorobot ever published in a peer-reviewed mainstream biomedical journal and is the author of *Nanomedicine*, the first book-length technical discussion of the medical applications of nanotechnology and medical nanorobotics. Volume I was published in October 1999 by Landes Bioscience while Freitas was a Research Fellow at the Institute for Molecular Manufacturing (IMM) in Palo Alto, California. Freitas published Volume IIA in October 2003 with Landes Bioscience while serving as a Research Scientist at Zyvex Corp., a nanotechnology company headquartered in Richardson, Texas during 2000-2004. Freitas is now completing *Nanomedicine* Volumes IIB and III and is also consulting on diamond mechanosynthesis, molecular assembler design, and nanofactory implementation as Senior Research Fellow at IMM. He won the 2009 Feynman Prize in nanotechnology for theory, the 2007 Foresight Prize in Communication, and the 2006 Guardian Award from Lifeboat Foundation, and was awarded the first patent on diamond mechanosynthesis on 30 March 2010.

Steven Garan



Title: Director of Bioinformatics at CREA, UC Berkeley, USA

Bio:

Steven A. Garan is the Director of Bioinformatics at CREA and serves on its Advisory Board, he is also a researcher at the Lawrence Berkeley National Laboratory. While at the University of California, Berkeley, he played a major role in the invention and the development of the Automated Imaging Microscope System (AIMS). While at UC Berkeley, Garan collaborated for many years with a group from Paola S. Timiras's lab, on the role that caloric restriction plays in maintaining estrogen receptor-alpha and IGH-1 receptor immunoreactivity in various nuclei of the mouse hypothalamus. Garan was also the director of the Aging Research Centre, and is a leading scientist in the field of aging research. His numerous publications, include articles on systems biology, the effects of caloric restriction on the mouse hypothalamus and on the Automated Imaging Microscope System (AIMS). He is best known for the coining of word «Phenomics», which was defined in an abstract titled: «Phenomics: a new direction for the study of neuroendocrine aging», that was published in the journal *Experimental Gerontology*. Steven A. Garan, was the lead scientists that developed the AIMS system along with Warren Freitag, Jason Neudorf and members of the UC Berkeley lab where AIMS was developed and utilized. Many journals articles have been published about the system and the results that it produced. Since the completion of the first version in 1998, newer versions were developed, with the final version being completed in 2007. Empowering investigators to accurately count specific cell populations is essential to all fields of neurobiology. While computer assisted counting technology has been in use for over a decade, advances in an Automated Imaging Microscope System (AIMS), now insure 97% accuracy when comparing computer counts to human counts for both nuclear and cytoplasmic stained tissue. More importantly, regional analysis can now be customized so that only cell populations within specified anatomic regions will be targeted for counting, thus reducing the background noise of non-immunoreactive cells when characterizing specific cell populations. This application was recently used to successfully map the density and distribution of both nuclear expressed estrogen receptor-alpha and cytoplasmically expressed IGF-1 receptor in specific hypothalamic nuclei. Furthermore, AIMS can now detect intra-hypothalamic differences in receptor expression and measure phenomenon such as lateralization. By using this technology, the evaluation of tissue-level biology can be used to establish neuroendocrine biomarkers of aging, and analyze the neuroendocrine effects of caloric restriction and gene knockout models that extend the lifespan.

Steven Garan



Title: Professor of Biobehavior Health at the Genetics Center, Penn State University, USA

Bio:

Dr. Roger McCarter is Professor of Biobehavior Health at the Genetics Center, Penn State University, University Park, Pennsylvania. He is currently President of the International Biogerontology Resources Institute (Cividale, Italy) and co-chair-elect of the Gordon Conference on the Biology of Aging. He is past Chair of Biological Sciences and a Fellow of the Gerontological Society of America (2002), past President of the American Aging Association (1997) and past President of the Pan American Congress on Gerontology and Geriatrics (1995-1999). He serves on the Advisory Council of the American Federation for Aging Research and is Executive Editor of the journal *Aging Clinical and Experimental Research*.

Dr. McCarter has directed courses in physiology and aging for Medical, Dental, Graduate, Nursing and Physical Therapy students as well as serving as a mentor for numerous post-doctoral fellows, doctoral and masters degree students over a 30-year period. His teaching activities have been recognized by many awards, including a Presidential Award for Excellence in Teaching (1986) from the University of Texas Health Science Center at San Antonio where he was Deputy Chair of the Department of Physiology and Associate Director of the Aging Research and Education Center. His research activities are focused on skeletal muscle function, metabolism and theories of aging. These activities have been continuously funded by grants from the National Institutes of Health and private agencies for the past 25 years, have been published in major peer reviewed journals and have been recognized by awards such as the Associate Editor's Award for the Best Paper Published in 1992/1993 in the *Journal of Gerontology: Basic Sciences*. His research has also been presented in numerous invited presentations in the USA, Europe and South America. Dr. McCarter's many community activities have included being President of the Oak Hills, San Antonio Rotary Club (1990), Chair of the Advisory Board of the Warms Springs Rehabilitation Hospital (2000-2002) and Coach of the Health Science Center Rugby Club, 1980.

George Garinis



UNIVERSITY
OF CRETE



Title: Professor, University of Crete, Greece

Bio:

Research in his laboratory is focused on:

- a. The impact of genome instability on pathways associated with longevity, aging and age-related pathology: We aim to identify common genome-wide expression patterns between a number of TCR-defective, progeroid mice and mice that carry (tissue-specific) constitutive defects in transcription. We are using a number of advanced molecular, genomics and imaging approaches to investigate the role of transcriptional instability in progeria and age-related pathology.
- b. The impact of progeroid mutations on tumour development: we aim at investigating the fine balance between cancer protection and accelerated aging. This approach might provide us both with mechanistic insight into the complex process of tumorigenesis as well as gene targets for its experimental modulation in the future.
- c. Genome instability and tissue-specific progeria: we are focusing at investigating age-related processes driven by genome instability in a tissue-specific manner using the loxP/ Cre recombinase technology to restrict DNA repair deficiency in a single type of tissue while leaving the remaining part of the organism intact.
- d. Development of advanced bioinformatics approaches to integrate functional genomics data with and biological endpoints in cancer and aging.

Jennifer L. Garrison



Title: Ph.D., Assistant Professor at the Buck Institute for Research on Aging

Bio:

Dr. Jennifer Garrison is interested in understanding how neuropeptides control behavior at both the cell biological and neural circuit level. Neuropeptides comprise a large class of signaling molecules which are secreted from neurons and transmit messages within the brain and across the nervous system.

Dr. Garrison received her PhD from the University of California San Francisco in the laboratory of Dr. Jack Taunton, where she discovered the molecular target of a natural product and elucidated a novel mechanism by which small molecules can regulate protein biogenesis. As a postdoctoral fellow in Dr. Cori Bargmann's lab at the Rockefeller University, she showed that the nematode *C. elegans* produces a neuropeptide that is an evolutionary precursor of the mammalian peptides vasopressin and oxytocin, and mapped a neural circuit by which this molecule, nematocin, modulates mating behavior.

Dr. Garrison was named an Alfred P. Sloan Research Fellow and received a Glenn Foundation Award for Research in Biological Mechanisms of Aging in 2014, and a Next Generation Leader at the Allen Institute for Brain Science in 2015. Her work is funded by the NIH National Institute of General Medical Sciences, the Glenn Foundation for Medical Research, the Alfred P. Sloan Foundation, and the Larry L. Hillblom Foundation.

Leonid Gavrilov



at the UNIVERSITY of CHICAGO



Title: Principal Investigator, Center on Aging, NORC at the University of Chicago

Bio:

Leonid A. Gavrilov is a Senior Research Scientist for the Center on the Demography and Economics of Aging with NORC at the University of Chicago. He also is a faculty member in the Center for East European and Russian/Eurasian Studies at the University of Chicago.

Gavrilov specializes in the biodemography of aging and longevity, mathematical modeling of aging and mortality, population aging and the demography of the Former Soviet Union.

Gavrilov is currently the principal investigator for Biodemography of Exceptional Longevity in the United States, a study funded by the National Institute on Ageing (NIA) in which he is investigating biological and social correlates for people that live over 100. Other studies funded by the NIA include Middle-Life Physical Markers, Socioeconomic Status and Exceptional Longevity: An Exploratory Study of a New Data Resource, and the Biodemography of Human Longevity - Training Program. Both projects focused on aging and acted as pre-cursors to Gavrilov's current work with Biodemography of Exceptional Longevity in the United States.

Before coming to the United States, Gavrilov held senior research positions in Moscow (Russia) at the A.N. Belozersky Institute, Moscow State University and at the Institute for Systems Analysis, Russian Academy of Sciences.

In demand as a speaker at conferences, meetings, and workshops internationally, Gavrilov's work has been featured in a variety of publications, such as *Population Research and Policy Review*, *North American Actuarial Journal*, *Social Biology*, *Journal of Theoretical Biology*, *Population*, *Demographic Research*, *Perspectives in Biology and Medicine*, *Human Biology*, *Handbook of the Biology of Aging* (Sixth Edition), and many more. He serves on several editorial boards, including *Experimental Gerontology* (Elsevier Science, Inc.), *Gerontology* (Karger), *Rejuvenation Research* (Mary Ann Libert Inc. Publishers), *Advanced Science Letters* (American Scientific Publishers) and *Theoretical Biology and Medical Modelling* (BioMed Central).

The Moscow Society of Naturalists, the International Science Foundation, the European Union and others have honored and recognized Gavrilov for his ongoing contributions to research in aging. He is a member of the Population Association of America and a Fellow of the Gerontological Society of America, where he has served on the Executive Committee on Biological Sciences and the Task Force on Organizational Technology and Computers. He is currently a Convener for the Gerontological Society of America Interest Group «Societal Implications of Delayed Aging.»

David Gems



Title: Assistant Director of the Institute of Healthy Ageing

Bio:

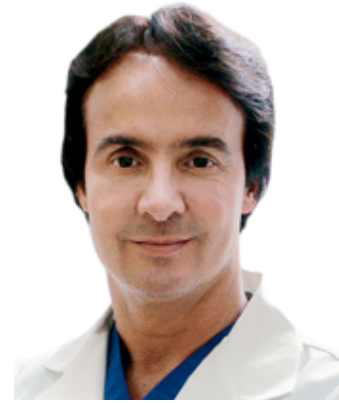
Professor David Gems is the Assistant Director of the Institute of Healthy Ageing within which his own laboratory studies ageing using the model organism *c.elegans*.

Professor David Gems is the Assistant Director of the Institute of Healthy Ageing within which his own laboratory studies ageing using the model organism *c.elegans*.

Background:

- 2012- Professor of Biogerontology, Institute of Healthy Ageing, UCL
- 2005-2011 Reader in the Biology of Ageing, Department of Biology, UCL
- 1997-2004 Royal Society University Research Fellow, Department of Biology, University College London, U.K. Genetics of aging in *C. elegans* and other model organisms
- 1993-1996 Postdoctoral fellow, Molecular Biology Program, University of Missouri, with Prof. Don Riddle. Genetics of aging in *Caenorhabditis elegans*
- 1991-1993 Postdoc, Department of Biology, Imperial College, London, with Prof. Rick Maizels. Biology of infective larvae of the ascarid nematode parasite *Toxocara canis*
- 1987-1990 Ph.D., Institute of Genetics, University of Glasgow, U.K. *Aspergillus nidulans* genetics. With A.J. Clutterbuck
- 1984-1986 Various work in Costa Rica, Nicaragua (Sandinista regime), Mexico, USA
- 1980-1983 School of Biological Sciences, University of Sussex, U.K. B.Sc. Biochemistry
- 1974-1978 Dartington Hall School

Vincent C. Giampapa



Quote: "The key to longevity and a quality life span is to address the factors which impact aging in their early stages." - Vincent C. Giampapa, N/A http://www.newliving.com/issues/apr_2004/articles/aging%20code.html

Title: Assistant Clinical Professor of Plastic and Reconstructive Surgery, University of Medicine and Dentistry of New Jersey

Bio:

Dr. Giampapa is Assistant Clinical Professor of Plastic and Reconstructive Surgery at the University of Medicine and Dentistry of New Jersey. He was one of the first board-certified anti aging physicians. Dr. Giampapa was founder and Chief Science Officer of Optigenex Inc., a publicly traded company focusing on development of naturally sourced compounds for DNA repair in humans. He was also the founder and Chief Science Officer of Suracell, Inc., a company focusing on genetic testing to create the world's first personalized genetic health program to mimic calorie restriction in an intracellular capacity.

Dr. Giampapa has filed over 20 U.S. patents and is presently focusing his research on stem cell therapy (VSEL) technology for regenerative medicine and age-management protocols, as well as developing the Echelon Biomatrix™ - an advanced AI software program to analyze multiple biomarkers of aging for his Executiv Health Program Patients. This software may also be used as a means of evaluating age management protocols and individual product effectiveness on human aging. Dr. Giampapa has published his most recent theory of aging - the MULTIPLE DNA DAMAGE THEORY - asserting that changes in DNA damage levels cause decline in gene expression, as well as stem cell function, as humans' age. Dr. Giampapa recently received a nomination for the Nobel Prize for his groundbreaking stem cell research related to the new science of Epigenetics, which studies science's ability to help optimize human cell function so that people age better

Dr. Giampapa received his MD and general surgery training from Mount Sinai Medical School and Hospital. He completed his postdoctoral training in Microsurgery and Transplantation Surgery at NYU Medical Center and Columbia Presbyterian Medical Center. He received his BA in Molecular Biology, Physiology and Psychology at Butler University in Indianapolis, Indiana.

Bradford W. Gibson



Title: PhD, Professor, Director of the Buck Institute Chemistry and Mass Spectrometry Core

Bio:

Dr. Gibson's work is focused on understanding the biological and chemical processes that are common to both age-related diseases and aging. The Gibson laboratory employs mass spectrometry, chemistry, and structural biology techniques to define the molecular details of processes that are important to aging and age-related diseases. In the fall of 2000, Dr. Gibson established the Chemistry and Mass Spectrometry Core at the Buck Institute to support research in his laboratory as well as those of his colleagues. He deploys a wide array of specialized chemistry and structural techniques to delve into the functions of mitochondria, subcellular organelles which provide the major energy needs of cells as well as critical metabolic and regulatory roles. He tracks the changing structures of mitochondrial proteins and lipids in aging cells, and in age-related conditions, such as diabetes, cancer and Parkinson's disease. The Gibson lab developed methods that are now being used to examine the role of mitochondrial dysfunction in a broader range of illnesses with the ultimate goals of finding opportunities for novel drug intervention strategies. He was interviewed for the January 2013 issue of Nature Methods for an article focusing on «Method of the Year -- Targeted Proteomics.»

Dr. Gibson supports the work of other Buck faculty through his expertise in chemistry and mass spectrometry, which can be used together to identify structure and functional changes of biological molecules such as proteins, glycoconjugates and lipids. Proteins and other biomolecules have complex chemical structures that determine their overall function, shape, location, and interacting partners. Dr. Gibson developed chemical and mass spectrometry-based methods to capture these events with precise and quantitative measurements.

The Gibson lab is also part of a national consortium that is identifying early protein biomarkers of cancer in human plasma using mass spectrometry and other technologies. These "cancer biomarkers" may yield early diagnostic tests for specific cancers. Ultimately, it is Dr. Gibson's goal to leverage these studies to develop the tools needed to identify biomarkers of aging that could be used to assess drugs and other therapies designed to influence the aging process itself.

Dr. Gibson received his PhD in Analytical Chemistry from the Massachusetts Institute of Technology in 1983, and then took a postdoctoral fellowship in Chemistry at Cambridge University in England. He was a professor at the University of California, San Francisco (UCSF) before joining the Buck Institute in 2000, and currently holds a joint appointment as an Adjunct Professor of Chemistry and Pharmaceutical Chemistry at UCSF.

Vadim Gladyshev



HARVARD
MEDICAL SCHOOL



Title: Professor, Medicine, Harvard Medical School
Director of Redox Medicine Lab, Brigham And Women's Hospital

Bio:

Gladyshev Laboratory - Brigham & Women's Hospital

The Gladyshev lab research interests focus on redox biology and trace elements as applied to cancer, aging and male reproduction. They are trying to understand the mechanisms of redox regulation of cellular processes by studying reactive oxygen species (ROS) and thiol oxidoreductase functions of cellular components. Little is known of what the specific targets of ROS are and how oxidant and antioxidant signals are transmitted in the cell. To understand mechanisms of redox control, the Gladyshev lab needs to know the identities and functions of most of the participants in the redox process. Thus, they are developing and employing various bioinformatics approaches and carrying out genome sequencing, proteomics and functional genomics studies, which are followed with in vitro and in vivo tests of the identified targets. They are particularly interested in the redox control that involves specific and stochastic oxidation of cysteine and methionine residues in proteins.

In mammals, major redox systems are dependent on the trace element selenium, which is an essential component of various redox enzymes in thioredoxin, glutathione and methionine sulfoxide reduction pathways. Selenium is present in proteins in the form of the 21st amino acid, selenocysteine, encoded by UGA codon. Selenocysteine can be viewed as redox super-cysteine because it is only used as the catalytic residue in oxidoreductases. Because UGA is also a stop signal, selenoprotein genes are typically misannotated in sequence databases. To overcome this problem, they identify these genes by genome-wide searches for structural and thermodynamic properties of specific RNA structures and independently by searches for selenocysteine/cysteine pairs in homologous sequences. Subsequently, the Gladyshev lab characterizes functions, regulation and specific targets of selenoproteins and other oxidoreductases to gain a system-wide view on selenium metabolism and redox regulation of cellular processes.

The Gladyshev lab is expanding their research on the basic mechanisms of aging, which they characterize using methods of biochemistry and bioinformatics and utilizing model organisms, primarily yeast and fruit flies. They also characterize the methionine sulfoxide reductase system, which is a protein and metabolite repair system. More generally, they think aging is the consequence of accumulation of damaged biomolecules in cells and organisms. Therefore, understanding the mechanisms by which organisms deal with damage accumulation and how these processes themselves deteriorate is crucial to an understanding of the aging process.

The lab also studies the 15 kDa selenoprotein (Sep15), which is involved in the quality control of protein folding in the endoplasmic reticulum. They identified Sep15 as a candidate protein that mediates the cancer chemopreventive effect of selenium. The lab is characterizing its function and role in cancer prevention to identify a mechanism by which dietary selenium decreases cancer incidence. Another project involves functional characterization of animal thioredoxin reductases. Mammals have three

(cytosolic, mitochondrial and spermatid-specific) thioredoxin reductases; each of them occurs in multiple forms generated by alternative first exon splicing. The Gladyshev lab identified one of them as thioredoxin-glutathione reductase, which is involved in male reproduction.

The Gladyshev lab hopes that their studies will provide a better understanding of the role of redox processes in physiological and pathophysiological states, particularly with regard to aging, cancer, and male reproduction, and will lead to new therapeutic and disease-preventive agents.

Allan Goldstein

School of Medicine & Health Sciences

THE GEORGE WASHINGTON UNIVERSITY



Title: Professor & Chairman, The George Washington University School of Medicine and Health Sciences

Bio:

Allan L. Goldstein is professor and Catharine B. & William McCormick Chair of the department of Biochemistry and Molecular Biology at The George Washington University School of Medicine and Health Sciences, where he has served since 1978. He is a world-renowned authority on the thymus gland and the workings of the immune system, and co-discoverer of the thymosins.

Dr. Goldstein is the author of over 400 scientific articles in professional journals, the inventor on more than 15 U.S. Patents, and the editor of several books in the fields of biochemistry, biomedicine, immunology and neuroscience.

He is on the editorial boards of numerous scientific and medical journals and has been a consultant to many research organizations in industry and government; co-founder of The Institute for Advanced Studies in Aging and Geriatric Medicine, a non-profit research and educational institute; a member of the Board of Trustees of the Albert Sabin Vaccine Institute; and serves as the Chairman of the Board of RegeneRx Biopharmaceuticals.

Dr. Goldstein received his B.S. from Wagner College in 1959 and his M.S. and Ph.D. from Rutgers University in 1964. He served as a faculty member of the Albert Einstein College of Medicine from 1964 to 1972, and moved to the University of Texas Medical Branch in Galveston in 1972 as professor and director of the division of Biochemistry.

Aubrey de Grey



Quote: Aubrey de Grey 'argues that aging can be «cured» if it's approached as an «engineering problem.»' 2015 <http://www.npr.org/2015/05/22/408025154/can-aging-be-cured>

Title: Chief Science Officer, SENS Research Foundation

Bio:

Dr. de Grey is the biomedical gerontologist who researched the idea for and founded SENS Research Foundation. He received his BA in Computer Science and Ph.D. in Biology from the University of Cambridge in 1985 and 2000, respectively. Dr. de Grey is Editor-in-Chief of Rejuvenation Research, is a Fellow of both the Gerontological Society of America and the American Aging Association, and sits on the editorial and scientific advisory boards of numerous journals and organizations.

Leonard Guarente

ELYSIUM



Quote: "Aging is one of the great, unsolved mysteries in life sciences. I was captivated by the importance of the problem, both in terms of the complexity of the science and also the importance to society." - Leonard Guarente, 2015 <https://www.elysiumhealth.com/blog/qa-with-elysium-co-founder-and-chief-scientist-leonard-guarente>

Title: Chief Scientist, Elysium Health, Inc.

Bio:

Dr. Leonard Guarente, Ph.D. served as Co-Chair of Scientific Advisory Board at Sirtris Pharmaceuticals, Inc. Dr. Guarente serves as the Novartis Professor of Biology at the Massachusetts Institute of Technology. Dr. Guarente's lab identified SIR2 as the key gene regulating life span in yeast and *C. elegans* an extra copy of SIR2 significantly increases the life span of both organisms. Notably, his lab first discovered the novel biochemical activity of the SIR2 gene product NAD-dependent deacetylase. Dr. Guarente's lab also studies the mammalian ortholog of SIR2 -- SIRT1. He trained as a postdoctoral fellow at Harvard with Mark Ptashne and has been on the faculty of MIT since 1981. He serves as a Member of Advisory Board of Segterra Inc. His book *Ageless Quest* (Cold Spring Harbor Press, 2003) describes the pathway of discovery of SIR2 as a key regulator of life span in response to diet. Dr. Guarente received his B.S. from the Massachusetts Institute of Technology and his Ph. D. at Harvard, under the supervision of Jon Beckwith

Pejmun Haghighi



Title: Ph.D., Professor at the Buck Institute for Research on Aging

Bio:

A growing consensus suggests that stability and homeostasis in synaptic growth and function may be key in maintaining the health of neural circuits, and as such, disruption in regulatory mechanisms that control synaptic homeostasis may lead to developmental and neurodegenerative nervous system diseases. His research program investigates the molecular mechanisms that underlie synaptic homeostasis. In particular, they are interested in learning how retrograde signaling cascades participate in this process.

His laboratory has been identifying and characterizing genes and mechanisms that participate in this regulation by exploiting the power of *Drosophila* genetics in combination with imaging and electrophysiology. In particular, his group's success in understanding the basic biology of synaptic function have led us to the identification of the target of rapamycin (TOR) as a critical regulator of synaptic homeostasis. This finding is of particular interest, since TOR-dependent pathways play a highly conserved role in the regulation of life span in a wide range of organisms from yeast to mice. They believe that our work will generate important insight into how nervous system function and life span regulation may be mutually modulated.

Aging and synaptic function:

Based on their findings, they have hypothesized that abnormal synaptic function negatively influences life span in *Drosophila*. They are addressing this hypothesis by establishing that interference with synaptic transmission can influence life span, while restoring normal synaptic function in mutant combinations with shorter life span can restore normal life span. They are also investigating the role of human disease-related genes in this process.

Postsynaptic translation:

They are interested in identifying mRNAs that are under the control of TOR-dependent postsynaptic translational cascades. They have been using genetic screens as well as biochemical approaches for these studies.

The role of miRNAs:

Their recent findings suggest that miRNAs may be acting as negative regulators of synaptic strength by limiting the amount of presynaptic neurotransmitter release. They are pursuing this idea by combining genetic and optogenetic approaches to monitor miRNA activity in neurons while manipulating synaptic activity and/or signaling.

Dr. Haghighi received his PhD in Physiology from McGill University in Montreal, Canada, where he also served as an assistant, then associate professor. In 2010 he received the Ann Wechsler Award for Excellence in Teaching Physiology. Dr. Haghighi's research was funded in large part, by the Canadian Institute for Health Research. He was a postdoctoral fellow in the lab of Corey Goodman, PhD, at the University of California Berkeley.

Leonard Hayflick



Quote: “My interest in understanding the biology of aging is not only based on curiosity but on why the aging process makes old cells more vulnerable to age-associated diseases. The critical question is, ‘What is the molecular constitution of old cells that makes them more vulnerable than young cells?’” - Leonard Hayflick, 2012 <http://agedmed.org/AMMGejournal/September2012/MorrisHayflickEtiologyOfAgingSept2012/tabid/753/language/en-US/Default.aspx>

Title: Professor of Anatomy, University of California, San Francisco

Bio:

Leonard Hayflick received his [Ph.D.](#) at the University of Pennsylvania in 1956. After receiving a post-doctoral Fellowship for study at the University of Texas Medical Branch in Galveston, under the tutelage of the renowned cell culturist Prof. Charles M. Pomerat, he returned to Philadelphia, where he spent ten years as an Associate Member of the Wistar Institute and two years as an Assistant Professor of Research Medicine at the University of Pennsylvania.

In 1968 Hayflick was appointed Professor of Medical Microbiology at the Stanford University School of Medicine, Stanford, California. In 1982 he moved to the University of Florida, Gainesville, where he became Director of the Center for Gerontological Studies and Professor of Zoology in the College of Liberal Arts and Sciences and Professor of Microbiology and Immunology in the College of Medicine. In 1988 Hayflick joined the faculty of the University of California, San Francisco, where he is currently Professor of Anatomy. Hayflick was Editor-in-Chief of the international journal “Experimental Gerontology” for 13 years.

He was a founding member of the Council of the National Institute on Aging, NIH and Chairman of its Executive Committee. He was a consultant to the National Cancer Institute and the World Health Organization, and is now a member of several scientific advisory boards. He was Chairman of the Scientific Review Board of the American Federation for Aging Research where he was also a vice president and a Member of the Board of Directors. He was also recruited by Michael D. West, founder of Geron (NASDAQ: GERN) and current CEO of BioTime, to join the company’s Scientific and Clinical Advisory Board, on which he served from 1991-2000.

Heinrich Jasper



Title: PhD, Professor at the Buck Institute for Research on Aging

Bio:

Dr. Jasper is interested in regulatory mechanisms that control stress tolerance, metabolism and aging in multi-cellular organisms. In particular, he has been recognized for making seminal discoveries about the effects of aging on stem cell behavior, and about the role of stress signaling in regulating stem cell function. Current projects in his lab focus on the control of tissue regeneration, metabolic homeostasis, and cell death by insulin and stress signaling pathways. Most of these studies are being performed using *Drosophila melanogaster*, taking advantage of the wide range of genetic, molecular, and genomic techniques available for this model organism. Current and future work is extending this research to stem cell systems in the mouse. It focuses on signaling mechanisms that influence critical physiological processes with relevance to aging:

Stem Cells and Regeneration

Regenerative processes are important for long-term tissue homeostasis in metazoans. Pluripotent stem cells are central to such regenerative processes. The Jasper lab is studying stem cells in the *Drosophila* midgut epithelium to ask how stress and aging influences their ability to self-renew, and whether optimizing stem cell activity can influence the aging process in metazoans. Current studies are extending this work to mouse tracheal stem cells, a mammalian stem cell population with striking functional and regulatory similarities to *Drosophila* intestinal stem cells.

Signaling networks controlling metabolic homeostasis and lifespan

Metabolic homeostasis is regulated by endocrine interactions between insulin producing and insulin target tissues. Stress signaling, in particular the Jun-N-terminal Kinase (JNK) pathway, antagonizes insulin signaling through various cell autonomous and endocrine mechanisms. Studies in the lab aim at establishing how these interactions control metabolic homeostasis and influence lifespan.

Stress-induced cell death

The Jasper lab uses the developing *Drosophila* retina as a model system in which to assess the regulation of cellular survival vs. death decisions, and to identify molecular and cellular mechanisms governing tissue recovery after genotoxic stress.

Dr. Jasper received his PhD from the University of Heidelberg and the European Molecular Biology Laboratory, where he studied transcriptional regulation of developmental processes in *Drosophila*. He became a Research Assistant Professor at the University of Rochester Medical Center in 2003, and an Assistant Professor of Biology at the University of Rochester in 2005. Dr. Jasper received a Senior Fellow Award of the Ellison Medical Foundation in 2008 and a Glenn Foundation Award for Research in Biological Mechanisms of Aging in 2010. His work was and is funded by the American Federation for Aging Research, National Institute of Aging, National Eye Institute, National Institute of General Medical Sciences, New York Stem Cell Initiative, and the Ellison Medical Foundation.

Matt Kaeberlein

UW Medicine
UNIVERSITY OF WASHINGTON
MEDICAL CENTER

Kaeberlein Lab



Title: Lab Hed, University of Washington Medical Center, Seattle

Bio:

The Kaeberlein Lab is located in the University of Washington Medical Center in Seattle. The lab has been in operation as part of the UW Pathology Department since March 1, 2006. Dr. Matt Kaeberlein is the primary investigator.

Matt Kaeberlein, Ph.D., is recognized globally for his research on the basic biology of aging. The premise of his research is that understanding the molecular mechanisms of aging will lead to interventions that slow the onset and progression of age-related chronic conditions, such as cancer, diabetes, kidney disease, heart disease, Alzheimer's and others. Dr. Kaeberlein received his Ph.D. from the Massachusetts Institute of Technology in 2002 and performed his post-doctoral research in the Department of Genome Sciences at the University of Washington. Dr. Kaeberlein was appointed as an Assistant Professor of Pathology in 2006 and was promoted to Associate Professor in 2011. Dr. Kaeberlein has authored more than 140 publications in top scientific journals, including 19 published in *Nature* and *Science*, his work has also been featured in the popular press. Dr. Kaeberlein has been recognized with several awards, including a Breakthroughs in Gerontology Award from the Glenn Foundation, an Alzheimer's Association Young Investigator Award, an Ellison Medical Foundation New Scholar in Aging Award, an Undergraduate Research Mentor of the Year Award, and a Murdock Trust Award. In 2011, he was named the Vincent Cristofalo Rising Star in Aging Research by the American Federation for Aging Research and appointed as a Fellow of the Gerontological Society of America, and in 2012 he joined the Board of Directors of the American Aging Association. Dr. Kaeberlein currently serves on the editorial boards for *Science*, *Aging Cell*, *Cell Cycle*, *PLoS One*, *Frontiers in Genetics of Aging*, *BMC Longevity and Healthspan*, *F1000 Research*, *Ageing Research Reviews*, and *BioEssays*.

In addition to his primary appointment, Dr. Kaeberlein is the Director of the Dog Aging Project, co-Director of the University of Washington Nathan Shock Center of Excellence in the Basic Biology of Aging, the founding Director of the Healthy Aging and Longevity Research Institute, and the current President of the American Aging Association.

Pankaj Kapahi



Title: Ph.D., Professor at the Buck Institute for Research on Aging

Bio:

Overall Goals. The overall goal of the Kapahi laboratory is to understand how an organism responds to nutrient status to influence health and disease. They utilize worms, flies and mice as model systems to understand how nutrients influence age-related changes in physiology and disease processes. Using interdisciplinary approaches they are examining the mechanisms by which various organs and the microbiome communicate with each other to influence various physiological processes. They study how various physiological and molecular processes including physical activity, fat metabolism, circadian clocks, advanced glycation end products, calcification and intestinal permeability are influenced by nutrients to impact organismal health and survival. Their work has relevance to a number of age-related human diseases including diabetes, kidney stone formation, intestinal diseases, cancer and obesity. Some of the projects currently being pursued in the lab are described below.

Lab philosophy. 'Creativity and Collaboration' they aim to utilize creative approaches to understand the mechanisms by which nutrients influence cellular homeostasis and disease processes. They also strive to develop models for various human diseases that are influenced by nutrient status using invertebrates.

Brian Kennedy



Quote: ““We are going to look back on this century. Will it be a time when we didn’t address the problem of chronic disease and all of the consequences that went with it? Or did we decide to target chronic diseases of aging to ensure these people were healthy instead? I hope we chose healthspan.”
- Brian Kennedy, 2015 <http://healthspancampaign.org/2015/02/23/buckinstitute/>

Title: Professor, Buck Institute for Research on Aging

Bio:

Dr. Kennedy’s innovative work in the biology of aging began when he was a doctoral student at MIT. Under the guidance of MIT Professor Leonard Guarente, he contributed to the first studies to show that a class of proteins called Sirtuins influence aging. Currently, he studies the pathways that modulate longevity in life forms ranging from yeast to mice. A major focus of his current research is to study the target of rapamycin (TOR) pathway. TOR generated excitement in the age research field when it was shown recently that the drug rapamycin can extend mouse lifespan. One of the goals of his research is to determine whether pathways like TOR can be regulated to treat the diseases of aging. Specifically, Dr. Kennedy’s lab focuses on cardiovascular disease and metabolic syndromes like type II diabetes. Dr. Kennedy also studies the genetic mutations underlying diseases such as dilated cardiomyopathy, muscular dystrophy and Hutchinson-Gilford Progeria Syndrome, which resembles premature aging. The mutations being studied affect a class of molecules called A-type nuclear lamins, and the lab is exploring their roles in health and disease.

Dr. Kennedy earned his PhD in Biology at Massachusetts Institute of Technology, where he took part in groundbreaking studies on aging. He completed postdoctoral training at the Massachusetts General Hospital Cancer Center in Charlestown, Massachusetts. Dr. Kennedy was an associate professor in the biochemistry department at the University of Washington in Seattle when he was appointed president and chief executive officer of the Buck Institute in 2010.

Cynthia Kenyon



Quote: “«There are long- and short-lived insects, birds and mammals. During evolution, there must have been a first insect, a first bird, a first mammal, and probably all of them had a short life span... It's not like life span changed just once and that's it. It can happen again.» - Cynthia Kenyon, 2007 <https://www.ucsf.edu/news/2007/01/3785/kenyon2>

Title: Vice President, Aging Research, Calico

Bio:

Kenyon graduated valedictorian in chemistry from the University of Georgia in 1976. She received her Ph.D. from MIT in 1981 and was a postdoctoral fellow with Nobel laureate Sydney Brenner in Cambridge, England. In 1986 she joined the faculty of the University of California, San Francisco, where she became the Herb Boyer Distinguished Professor and an American Cancer Society Professor, before joining Calico in 2014. Kenyon is a member of the U.S. National Academy of Sciences, the National Academy of Medicine and the American Academy of Arts and Sciences, and she is a former president of the Genetics Society of America. She has received many scientific honors and awards.

James Kirkland



Quote: “Although considerable advances have been made in understanding the basic biology of aging, especially during the past five to 10 years, not enough attention has been paid to translating this research into practical solutions that address end-of-life issues and the wider societal implications of biomedical research.” - James Kirkland, N/A <http://www.mayo.edu/research/centers-programs/robert-arlene-kogod-center-aging/about/message-from-director>

Title: Director, Robert and Arlene Kogod Center on Aging, Mayo Clinic

Bio:

James Kirkland received both his MD and PhD from the University of Toronto. The major research focus of Dr. Kirkland is the impact of cellular aging (senescence) on age-related dysfunction and chronic diseases, especially developing methods for removing these cells and alleviating their effects. Senescent cells accumulate with aging and in such diseases as dementias, atherosclerosis, cancers, diabetes and arthritis.

The goal of Dr. Kirkland’s current work is to develop methods to remove these cells to delay, prevent, alleviate or partially reverse age-related chronic diseases as a group and extend health span, the period of life free of disability, pain, dependence and chronic disease.

Don Kleinsek



Title: CEO & Co-Founder, Cellagen Technology LLC.

Bio:

Dr. Kleinsek is a veteran of over 30 years in the field of gerontology, delineating the mechanism(s) of the human aging process and focusing on translational medicine – the development of aging research discoveries into clinical products that reach the commercial sector. Notably his fundamental breakthrough in cholesterol metabolism was an important advancement in medicine and into the \$20 billion marketplace for the number one drug in sales today, the statins. Currently his patented cell therapy products are cures for a variety of age-related conditions and is being readied for commercialization. He is a pioneer in personalized medicine and cellular therapeutics. His expertise includes the field of cell culture and bioengineering.

Dr. Kleinsek is the CEO and co-founder of Cellagen, LLC. Seeking expedited and effective medical solutions for the marketplace, he was one of the very early entrepreneurs to enter into the dawn of the biotechnology era in the 1980s and founded GeriGene Medical Corporation. Prior to this approach, Dr. Kleinsek was a faculty member at Baylor College of Medicine and the University of Wisconsin as well as Director/President of the Bjorksten Research Foundation, a 40 plus year old non-profit organization.

Makoto Kuro-O



Title: Associate Professor of Pathology, University of Texas Southwestern Medical Center.

Bio:

Mr. Makoto Kuro-O, M.D., Ph.D. is an Associate Professor of Pathology from University of Texas Southwestern Medical Center. Since 1998, Mr. Kuro-O was an Assistant Professor of Pathology at the University of Texas Southwestern Medical Center at Dallas. Mr. Kuro-O serves as a Member of Anti-Aging Scientific Advisory Board at Nu Skin Enterprises, Inc. His laboratory focuses on understanding the molecular mechanism by which the klotho protein suppresses aging. Dr. Kuro-o received an M.D. in 1985 from the University of Tokyo, Japan. He completed his residency training at Tokyo Metropolitan Geriatric Hospital in 1988, after which he returned to the University of Tokyo as a clinical fellow in cardiology until 1998. He received a Ph.D. in 1991 from the University of Tokyo, following which he pursued postdoctoral training at the National Institute of Neuroscience in Japan. During his postdoctoral work, he identified the klotho gene—an aging suppressor gene in mammals.

Marios Kyriazis



Title: Co-director of the Einstein Institute for Aging Research, Israel

Bio:

Marios Kyriazis qualified as a medical doctor (MD) from the University of Rome, Italy, and after preclinical work in the USA he worked as a clinician in acute medicine in Cyprus, and the UK. He subsequently qualified as a Gerontologist with interest in the biology of aging and became a Chartered Member of the academic organisation 'Society of Biology' in the UK. He also has a post-graduate qualification in Geriatric Medicine from the Royal College of Physicians of London.

Other appointments include Member of the Board of Trustees at the Mediterranean Graduate School of Applied Social Cognition, and affiliate researcher at the Evolution, Complexity and Cognition Group, University of Brussels.

Currently, he works with the ELPs Foundation for Indefinite Lifespans, a serious endeavour to study the elimination of age-related degeneration. The research is focused on transdisciplinary models and explores common principles between biology, complexity sciences, evolution, cybernetics, neurosciences, and techno-cultural elements. Areas of interest include robustness and degeneracy in organic systems, fragility and redundancy, repair processes (including self-repair) and immortalisation of somatic cells.

One particular project involves the concept that agents which are useful in the evolution and adaptation of any system, are retained by that system. This concept can be applied in the specific case of humans who are actors within a highly technological and hyper-connected society, forming part of a Global Brain. The rationale is that these humans are valuable in the evolution of the global society and are thus more likely to live and function for longer. Biological mechanisms involved in this process could include microRNA and epigenetic modifications, phase transitions in metabolic and repair signaling, and other, hitherto poorly studied processes

Deepak A. Lamba



Title: M.B.B.S., PhD, Associate Professor at the Buck Institute for Research on Aging

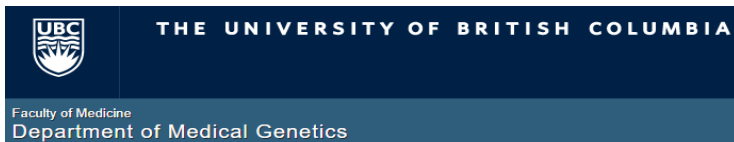
Bio:

Dr. Lamba's research is focused on identifying new methods to treat degenerative vision disorders, including macular degeneration and retinitis pigmentosa, using stem cell technology. He began this work as a graduate student developing methods of generating retinal cells from human embryonic stem cells. He has differentiated retinal neurons including photoreceptors, the cells in the eye that respond to light. His work is considered pioneering amongst those focused on developing efficient methods of making retinal cells in a laboratory dish. He has shown that these cells can be transplanted into the eyes of mice and rats. After testing vision in blind mice, the treated eyes are now responding to light. His lab is currently concentrating on long-term efficacy and safety studies which are essential before this form of therapy becomes available to patients.

Dr. Lamba is also interested in finding newer approaches of deriving patient-specific stem cells. Newer technologies are allowing scientists to reprogram skin cells into an embryonic stem cell counterpart called induced pluripotent stem cells (iPS) and then converting them to retinal cells. This will result in a better understanding of disease mechanism which in turn will provide an opportunity to discover treatments and drugs to halt photoreceptor cell death and either prevent or, at least, delay the degenerative process.

Dr. Lamba earned his medical degree from University of Mumbai, India and practiced as a physician in Mumbai, India before moving to the US to pursue full-time research. He received his Masters in Bioengineering from University of Illinois, Chicago where he worked on a chemically-stimulating retinal prosthesis device. He then moved to the University of Washington in Seattle where did his doctoral thesis and post-doctoral work on generating and transplanting retinal cells derived from human embryonic stem cells and iPS cells in the lab of Dr. Thomas Reh.

Peter Lansdorp



Title: Professor, Medical Genetics, University of British Columbia (UBC)

Bio:

Peter Lansdorp obtained his MD from the Erasmus University in Rotterdam before receiving a PhD in Experimental Hematology from the University of Amsterdam in 1985. During his graduate studies he discovered that selected monoclonal antibodies can be crosslinked into stable, bispecific tetrameric antibody complexes. These reagents have found numerous applications. In Amsterdam he became increasingly interested in growth factors such as IL-6 and the role of stem cells in blood cell formation. In 1985 he moved to the Terry Fox Laboratory at the BC Cancer Agency in Vancouver, where his work on the purification and culture of human and murine hematopoietic stem cells led him to studies of telomere biology. He developed quantitative fluorescence in situ hybridization (Q-FISH) techniques using peptide nucleic acid probes to measure the length of telomere repeats in chromosomes and cells. Most laboratories involved in telomere research have adopted these methods. Other current interests are related to the possibility that gene expression and cell fate is regulated in part by chromatin differences between sister chromatids (the “silent sister” hypothesis; Cell 129:1244, 2007) and to the role of telomere length and genome instability in cells of various tissues in ageing. For the latter his laboratory developed novel single cell DNA template strand sequencing techniques (Nature Methods, 2012). Peter Lansdorp is a Distinguished Scientist at the Terry Fox Laboratory and an affiliated Professor at the University of British Columbia. In 2010 he has been appointed to an Endowed Chair at the University Medical Center Groningen (UMCG). He was the first Scientific Director of the European Research Institute for the Biology of Ageing of the University of Groningen and the UMCG. In 2011 Peter Lansdorp received a €2.5 million Advanced Grant from the European Research Council for a research project on the role of telomeres and stem cells in ageing.

Robert Lanza



Title: Head of Astellas Global Regenerative Medicine
Chief Scientific Officer of the Astellas Institute for Regenerative Medicine
Adjunct Professor at Wake Forest University School of Medicine

Bio:

Dr. Lanza has hundreds of publications and inventions, and over 30 scientific books, including “Principles of Tissue Engineering” and “Essentials of Stem Cell Biology,” which are considered the definitive references in the field. He received his BA and MD degrees from the University of Pennsylvania, where he was both a University Scholar and Benjamin Franklin Scholar. He was also a Fulbright Scholar, and was part of the team that cloned the world’s first human embryo, as well as the first to successfully generate stem cells from adults using somatic-cell nuclear transfer (therapeutic cloning).

Lanza’s work has been crucial to our understanding nuclear transfer and stem cell biology. In 2001 he was also the first to clone an endangered species (a Gaur), and in 2003, he cloned an endangered wild ox (a Banteng) from the frozen skin cells of an animal that had died at the San Diego Zoo nearly a quarter-of-a-century earlier. Lanza and his colleagues were also the first to demonstrate that nuclear transplantation could be used to reverse the aging process and to generate immune-compatible tissues, including the first organ tissue-engineered from cloned cells. One of his early achievements came from his demonstration that techniques used in preimplantation genetic diagnosis could be used to generate human embryonic stem cells (hESCs) without embryonic destruction.

He and colleagues have also succeeded in differentiating human pluripotent stem cells into retinal (RPE) cells, and has shown that they provide long-term benefit in animal models of vision loss. Using this technology some forms of blindness may be treatable, including macular degeneration and Stargardt disease, a currently untreatable form eye disease that causes blindness in teenagers and young adults. Lanza’s company just completed two clinical trials in the United States using them to treat degenerative eye diseases. ACT carried out the only pluripotent stem cell trial in Europe.

In October 2014, Dr. Lanza and his colleagues published a paper in the journal *The Lancet*, providing the first evidence of the long-term safety and possible biologic activity of pluripotent stem cell progeny into humans with any disease. “For a nice two decades scientists have dreamt about using human embryonic stem cells to treat diseases,” said Gautam Naik, Science Reporter at the *Wall Street Journal* “that day has finally come...scientists have used human embryonic stem cells to successfully treat patients suffering from severe vision loss.” RPE cells derived from embryonic stem cells were injected into the eyes of 18 patients with either Stargardt’s disease or dry-AMD. The patients were followed for more than three years, and half of them were able to read three more lines on the eye chart, which translated to critical improvements in their daily lives as well.

Lanza and his colleagues in South Korea also recently published the first report of the safety and potential efficacy of pluripotent stem cells in Asian patients. hESC-derived RPE were transplanted in

four Asian patients: two with dry age-related macular degeneration and two with Stargardt's macular dystrophy. There were no safety issues related to the transplanted cells. Visual acuity improved 9-19 letters in three patients, and remained stable (+1 letter) in one patient. The results confirm that hESC-derived cells could serve as a potentially safe new source of tissue for regenerative medicine.

Lanza has been a major player in the scientific revolution that has led to the documentation that nuclear transfer and reprogramming factors can restore developmental potential in a differentiated cell. One of his successes was showing that it is feasible to generate functional oxygen-carrying red blood cells from human pluripotent stem cells. The blood cells were comparable to normal transfusable blood and could serve as a potentially inexhaustible source of "universal" blood. His team also discovered how to generate functional hemangioblasts — a population of "ambulance" cells — from hESCs. In animals, these cells quickly repaired vascular damage, cutting the death rate after a heart attack in half and restoring the blood flow to ischemic limbs that might otherwise have to be amputated. He has recently published similar pre-clinical work showing hESC-derived cells can be used to treat a range of other diseases, including multiple sclerosis and lupus, among others.

In 2009, Lanza and a team lead by Kwang-Soo Kim at Harvard University reported a safe method for generating induced pluripotent stem (iPS) cells. Human iPS cells were created from skin cells by direct delivery of proteins, thus eliminating the harmful risks associated with genetic manipulation. This new method provides a potentially safe and non-controversial source of patient-specific stem cells for translation into the clinic. The Editors of the *Nature* selected Lanza and Kim's paper on protein reprogramming as one of five "Research Highlights" of the year. *Discover* magazine stated, "Lanza's single-minded quest to usher in this new age has paid dividends in scientific insights and groundbreaking discoveries."

Dr. Lanza has received numerous awards, including *TIME* Magazine's 2014 *TIME* 100 list of the "100 Most Influential People in the World," the Top 50 "World Thinkers" (2015), the 2013 Il Leone di San Marco award in Medicine; an NIH Director's Award (2010) for "Translating Basic Science Discoveries into New and Better Treatments"; the 2013 "TOP 50 Global Stem Cell Influencers" (voted Top 4 "Most Influential People on Stem Cells" along with James Thomson and Nobel laureate Shinya Yamanaka); the 2010 "Movers and Shakers" Who Will Shape Biotech Over the Next 20 Years (*BioWorld*, along with Craig Venter and President Barack Obama); the 2007 100 Most Inspiring People in the Life-Sciences Industry (*PharmaVOICE*, "For his discoveries 'behind the medicines making a significant impact on the pipelines of today and of the future'"; the 2007 Outstanding Contribution in Contemporary Biology Award (Brown University, "For his groundbreaking research and contributions in stem cell science and biology"; the 2006 All-Star Award for Biotechnology (MA High Tech, for "pushing stem cells' future"); the 2005 Rave Award for Medicine (*Wired* magazine, "For eye-opening work on embryonic stem cells"); Massachusetts Medical Society award; and The Boston Globe's William O. Taylor award, among others.

Dr. Lanza and his research have been featured in almost every media outlet in the world, including CNN, *TIME*, *Newsweek*, *People*, as well as the front pages of the *New York Times*, *Wall Street Journal*, *Washington Post*, among others. Lanza studied with some of the greatest thinkers of our time, including Nobel laureates Gerald Edelman and Rodney Porter, renowned Harvard psychologist B.F. Skinner (the "Father of modern behaviorism"), Jonas Salk (discoverer of the Polio vaccine), and heart transplant pioneer Christiaan Barnard.

In 2007, Lanza published a feature article, "A New Theory of the Universe" in *The American Scholar*, a leading intellectual journal which has previously published works by Albert Einstein, Margaret Mead, and Carl Sagan, among others. His theory places biology above the other sciences in an attempt to solve one of nature's biggest puzzles, the theory of everything that other disciplines have been pursuing for the last century. This new view has become known as Biocentrism. In 2009, he co-authored a book "Biocentrism: How Life and Consciousness are the Keys to Understanding the True Nature of the Universe" with leading astronomer Bob Berman. In biocentrism, space and time are

forms of animal sense perception, rather than external physical objects. Understanding this more fully yields answers to several major puzzles of mainstream science, and offers a new way of understanding everything from the microworld (for instance, the reason for Heisenberg's uncertainty principle and the double-slit experiment) to the forces, constants, and laws that shape the universe. Nobel laureate E. Donnall Thomas stated "Any short statement does not do justice to such a scholarly work. The work is a scholarly consideration of science and philosophy that brings biology into the central role in unifying the whole."

"Robert Lanza is the living embodiment of the character played by Matt Damon in the movie "Good Will Hunting." Growing up underprivileged in Stoughton, Mass., south of Boston, the young preteen caught the attention of Harvard Medical School researchers when he showed up on the university steps having successfully altered the genetics of chickens in his basement. Over the next decade, he was "discovered" and taken under the wing of scientific giants such as psychologist B.F. Skinner, immunologist Jonas Salk, and heart transplant pioneer Christiaan Barnard. His mentors described him as a "genius," a "renegade thinker," even likening him to Einstein." – U.S. News & World Report, cover story

Pamela Larsen



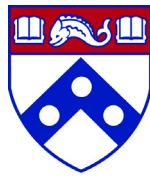
Title: Associate Professor, Department of Cell Systems and Anatomy, University of Texas Health Science Center at San Antonio

Bio:

What biological mechanisms govern adult health and life span? Genetic and environment components contribute to a long healthy life. The longevity manipulations we use in *C. elegans* are: 1) mutation of the *daf-2* gene, which is homologous to insulin/IGF1 signaling pathways, and 2) cultivation at a cool temperature. Both cooler core body temperatures and reduction of the *daf-2*/insulin/IGF-1 signaling pathway are pro-survival in multiple species including humans. Pamela Larsen and her team are defining environment by genotype interactions that alter gene transcription (epigenetics) and then testing the functional contribution to adult health and life span. For this their studies include different genotypes at different non-stress-inducing temperatures. They have found that health can be uncoupled from long life. Environmental changes trigger beneficial and detrimental responses. By associating molecular signatures with phenotypes, they can better predict adult health and life span outcomes in different genotypes and environments.

The figure shows results from a neurological exam we did on individuals each day from when they were healthy middle-aged adults. In wild type, we find locomotion slows with age and a small number become paralyzed when old. When they inhibit our candidate adaptive response, we see nearly all show progressive paralysis and yet the life span is not significantly shorter

Virginia Lee



Perelman
School of Medicine
UNIVERSITY of PENNSYLVANIA



Title: Endowed Professor, Pathology and Laboratory Medicine, University Of Pennsylvania

Bio:

Dr. Lee's research focuses on disease proteins that form pathological inclusions in hereditary and sporadic Alzheimer's disease (AD), Parkinson's disease (PD), frontotemporal lobar degeneration (FTLD), amyotrophic lateral sclerosis (ALS) and related neurodegenerative disorders of aging. Her work demonstrated that tau, alpha-synuclein and TDP-43 proteins form unique brain aggregates in neurodegenerative diseases and provided critical evidence that aggregation of brain proteins is a common mechanistic theme in diverse neurodegenerative diseases including AD, PD, FTLD, ALS and related disorders. Significantly, Dr. Lee's studies implicated the abnormal aggregation of tau, alpha-synuclein and TDP-43 in mechanisms that compromise neuronal viability. Most importantly, this research has opened up new avenues of research to identify targets for drug discovery to develop better treatments for these disorders.

Research Techniques

Protein biochemistry; cell and molecular biology; monoclonal antibody production; immunochemical and immunocytochemical techniques; tissue culture; transgenic mouse models; and electron microscopy.

Christiaan Leeuwenburgh



Title: Professor and Chief, Division of Biology of Aging, Institute on Aging, USA

Bio:

Christiaan Leeuwenburgh's research interests are aging and mitochondrial biology. Aging is thought to result from increased formations of reactive oxygen species and free radicals, mainly produced by the mitochondria, which can result in oxidative stress.

Mitochondria are organelles within cells that, when damaged, can have devastating effects on the body. When the mitochondria fail and become dysfunctional, they produce increasing amounts of reactive oxygen species (some are free radicals) and cells and tissue can be damaged and die. Reactive oxygen species are highly reactive molecules that cause damage to plasma membranes, enzymes, and DNA. Free radicals, unstable and potentially damaging molecules generated by the body's normal chemical reactions, may contribute to mitochondrial dysfunction and cell death, thereby accelerating diseases such as cardiovascular disease, diabetes, cancer, and Parkinson's disease. Free radicals are unstable molecules because they possess one or more unpaired highly reactive electrons. These molecules can steal electrons from other molecules, thereby altering the structure. The molecule that loses an electron becomes oxidized. Oxidation is thought to damage tissues, including neurons, myocytes, and muscle cells. Normally, free radical damage is kept under control by antioxidants, compounds that protect cells from this damage.

Dr. Leeuwenburgh's research focuses on finding interventions that prevent the mitochondria from becoming dysfunctional. His focus is on genetic, pharmacological and natural (specific diet and exercise) interventions. For example, restricting the amount of calories older animals consume (or periodic fasting) has shown that these animals live 30 to 40% longer than animals not on a restricted diet and they produce less free radicals.

Leeuwenburgh says, for humans, studies have shown that people who participate in life-long exercise will, on average, live two years longer than those who do not exercise. Plus, there is substantially less disease among those who exercise regularly

Rodney Levine



National Heart, Lung,
and Blood Institute



Title: Senior Investigator, Protein Function in Disease Section, NHLBI

Bio:

Rodney Levine graduated from Stanford University with a B.S. in chemistry and an A.B. in biology in 1968 and earned his M.D. and Ph.D. in physiology from Stanford in 1973. He completed his residency in pediatrics at the University of Colorado Medical Center in 1975 and his fellowship in perinatal medicine at the University of Colorado Medical Center and Denver Children's Hospital in 1977. He joined the NHLBI in 1977 as a research associate and rose to become Chief of the Laboratory of Biochemistry in 2008. He is board certified in pediatrics and in neonatal-perinatal medicine and is professor emeritus at the Uniformed Services University of the Health Sciences. Dr. Levine has authored or coauthored more than 200 papers. Currently, he serves on the editorial and scientific review boards of *Free Radical Biology and Medicine*, *Journal of Gerontology*, and *Mechanisms of Ageing and Development*, among others. He holds memberships with the American Association for the Advancement of Science, American Pediatric Society, American Society for Biochemistry and Molecular Biology, and Society for Free Radical Biology which has named him a Fellow of the Society.

Research Interests

Because eukaryotic cells depend on molecular oxygen for normal metabolism, they generate reactive oxygen species (ROS) that can cause multiple forms of cellular stress and damage. For several years, Dr. Levine has focused his research on the identification of oxidative modifications of proteins. He is interested in the conditions that give rise to modifications in which amino acids are modified, and the impact those modifications have on enzymatic function or structural integrity.

Several years ago, in collaboration with the late Earl Stadtman, Dr. Levine discovered a novel form of protein modification—carbonylation—that is now a widely accepted measure of oxidative modification in cells ranging from bacteria to humans. Carbonylation is typically associated with protein dysfunction. In a variety of species, carbonyl content rises dramatically in the last third of an organism's life and may be causally related to the phenotypic changes of aging. Although reactive oxygen species are usually promiscuous in their reactivity, Dr. Levine and his colleagues found that this modification was localized to the metal binding sites of proteins that contained them. Such binding sites normally recruit Mg^{2+} , Mn^{2+} , or Ca^{2+} , but upon binding of Fe^{3+} , the protein was rendered vulnerable to ROS modifications and loss of the metal binding site.

Dr. Levine has focused on oxidative modifications that are specific to methionine residues. This amino acid is particularly vulnerable to oxidation. Virtually every aerobic organism has at least one enzyme (methionine sulfoxide reductase) that functions to reduce oxidized methionine back to its original form. To study these modifications, Dr. Levine's laboratory is using a variety of techniques from biochemistry and mass spectrometry to transgenic mouse models with altered enzymatic activity of the reductases. Based on his research, Dr. Levine proposed the concept that methionines serve an important role as an antioxidant, both for the proteins in which they reside and the cell itself. Counter to the commonly held notion that methionines (which are hydrophobic residues) are typically buried within proteins, many methionine residues lie on the surfaces of proteins. Much like well-known antioxidant molecules

such as vitamins E and C, these exposed methionines intercept ROS and become oxidized. In this context, methionine sulfoxide reductase operates to bring the antioxidant system full circle. More recently, Dr. Levine and his colleagues have found that methionine sulfoxide reductase catalyzes oxidation and reduction of methionine residues bidirectionally. This finding opens the door to a role for oxidative modification of methionine as a cellular signaling mechanism. Whereas ROS production has been viewed as uniformly deleterious to cellular function, it is now clear that cells actually require a certain level of oxidants for optimal function. Dr. Levine is pursuing the hypothesis that oxidative modifications of proteins are not always a negative effect of stress, but also participate in normal cellular signaling.

Gordon Lithgow



Title: Professor at The Buck Institute`

Bio:

Dr. Lithgow sheds light on the mechanisms of aging by identifying agents that extend lifespan or prevent age-related disease. He has discovered a range of factors that can lengthen life in the microscopic worm *Caenorhabditis elegans*, and he applies these findings to studies in human cell cultures. Much evidence points to stress contributing to a breakdown in the ability to maintain optimal molecular stability resulting in aging and disease. Certain life-extending agents help *C. elegans* respond to lifelong stress by remodeling the natural stress fighting cellular mechanisms, the Lithgow lab has found. For example, long-lived mutants of *C. elegans* are very stress resistant as a result of elevated levels of “heat shock proteins.” Heat shock proteins promote longevity probably by preventing a loss of protein balance. Long-lived mutant strains are also resistant to heavy metals, so the Lithgow lab is now studying the relationship between longevity and “metallostasis”

The Lithgow lab has discovered that certain cell proteins capable of extending life can also be closely involved in disease prevention. But when proteins play such dual roles, they may sometimes make tradeoffs that affect the fate of the organism. Dr. Lithgow is studying genetic variations in “checkpoint proteins” that may create a trade-off between the rate of aging and incidence of cancer.

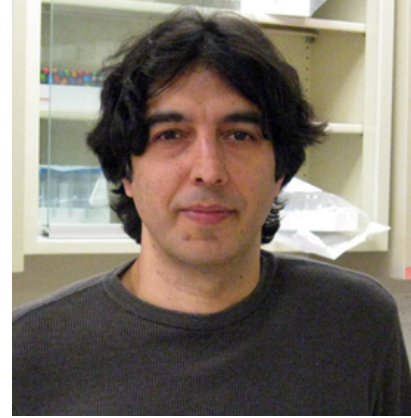
The Lithgow lab has made seminal discoveries in the use of pharmacological agents to intervene in aging processes, such as antioxidants that protect cells against damage from unstable chemicals called free radicals. More recently, his lab have uncovered compounds that act as “stress response mimetics” that maintain protein balance and stability. These compounds suppress pathology associated with Alzheimer’s disease. The lab continues to undertake screens for chemical compounds that slow aging and extend healthspan.

Dr. Lithgow received his PhD in Genetics from the University of Glasgow, Scotland. He completed postdoctoral training at the Institute for Behavioral Genetics at the University of Colorado, Boulder. Dr. Lithgow was a Senior Lecturer in Molecular Gerontology at the School of Biological Sciences at the University of Manchester in England before coming to the Buck Institute in 2001. He is the Principal Investigator and Director of the Buck Institute’s Interdisciplinary Research Consortium on Geroscience. He is also the Principal Investigator of the Larry L. Hillblom Network on the Chemical Biology of Aging, and is the Coordinator of the Hillblom Center for the Biology of Aging Support Award.

Valter Longo



USC University of
Southern California



Title: Director of the USC Longevity Institute

Bio:

Dr. Longo is the Edna Jones Professor in Gerontology and Professor in Biological Science. He is also the Director of the USC Longevity Institute. He is interested in understanding the fundamental mechanisms of aging in yeast, mice and humans by using genetics and biochemistry techniques. He is also interested in identifying the molecular pathways conserved from simple organisms to humans that can be modulated to protect against multiple stresses and treat or prevent cancer, Alzheimer's Disease and other diseases of aging. The focus is on the signal transduction pathways that regulate resistance to oxidative damage in yeast and mice.

Carlos Lopez-Otin



Universidad de Oviedo



Title: Lab Director, Department of Biochemistry and Molecular Biology from the Universidad de Oviedo

Bio:

The Lopez-Otin laboratory belongs to the Department of Biochemistry and Molecular Biology from the Universidad de Oviedo (Spain), and to the Instituto Universitario de Oncología del Principado de Asturias (IUOPA). Carlos Lopez-Otin is a Professor of Biochemistry and Molecular Biology at this Department, where he combines his teaching responsibilities with his research lines on Cancer and Aging Biology, as well as on the Functional Analysis of Genomes. His works have been collected in more than 350 articles in international journals and have been cited about 40,000 times to date, with an aggregate Hirsch index of $h=96$. He is a member of numerous journal editorial boards, committees, and scientific societies, including the Spanish Royal Academy of Sciences and the European Academy, among others. Throughout his scientific career, he has received different national and international awards and distinctions, such as the Doctorate “Honoris Causa” from the International University Menendez Pelayo, University of Zaragoza, and the Autonomous University of Chile, the “Rey Jaime I” Award in Research, the European “25th FEBS Jubilee” Award in Biochemistry, the Mexico Award in Science and Technology, and the “Santiago Ramón y Cajal” National Research Award. In 2017, he has been awarded an “ERC Advanced Grant” from the European Union.

Janet M. Lord



UNIVERSITY OF
BIRMINGHAM



Title: Director of the Institute of Inflammation and Ageing

Bio:

Janet's research focuses on the innate immune system, the body's front line defense against infection, and how the efficiency of this system is affected by ageing and stress, the latter including physical trauma and emotional stress such as bereavement. She is also interested in how the ageing of the immune system predisposes adults to chronic inflammatory diseases such as Rheumatoid Arthritis and COPD and the muscle wasting associated with age and these conditions. In all of her work she aims to translate research findings into interventions, whether lifestyle (exercise, diet) or pharmacological, to improve immunity and health in old age.

Professor Lord is also a leading member of the NIHR SRMRC, researching the impact of major trauma on the immune system and how this differs with age. Find out more about the work of the research centre on the SRMRC website.

Janet has published over 175 research papers and reviews in the fields of immunosenescence, chronic inflammatory disease and neuroendocrine-immune biology. She was elected a Fellow of the Academy of Medical Sciences in 2015 and awarded the Lord Cohen Medal by the British Society for Research into Ageing in 2013. Her research is currently funded by grants from MRC, Arthritis Research UK, NIHR, The Healing Foundation, the European Commission and the Glenn Foundation.

Alvaro Macieira-Coelho

Instituts
thématiques

Inserm

Institut national
de la santé et de la recherche médicale



Title: Research Director at the French National Institute of Health

Bio:

Dr. Alvaro Macieira-Coelho is a Research Director at the French National Institute of Health. He received an MD from the University of Lisbon, Portugal, and a PhD from the University of Uppsala Sweden. He made an internship at the University Hospital in Lisbon and was a research associate at the Wistar Institute in Philadelphia (USA) and at the Department of Cell Biology of the University of Uppsala (Sweden). He became Head of the Department of Cell Pathology at the Cancer Institute in Villejuif (France) and was a visiting Professor at the University of Linkoping (Sweden). He published 150 papers in professional Journals and 9 books on cancer and aging. He received the following awards: Fritz Verzar Prize (University of Vienna, Austria), "Seeds of Science", Career Prize (Lisbon, Portugal), Dr. Honoris Causa (University of Linkoping, Sweden), Johananof International Visiting Professor (Institute Mario Negri, Milano, Italy).

Joao Pedro de Magalhaes



UNIVERSITY OF
LIVERPOOL



Quote: “I generally make the analogy of the Wright brothers, who built and flew the world’s first successful aircraft. They used to look at birds and say these birds are heavier than air and they can fly. If birds can fly, we can make aeroplanes. There is no law of nature that says ageing is immutable. On the contrary we know now that ageing is surprisingly plastic in the sense that it can be manipulated by genes, it can be manipulated by evolution and it can be manipulated by diet.” - Joao Pedro de Magalhaes, 2014 <http://theconversation.com/ageing-isnt-fixed-we-can-manipulate-it-to-live-longer-31808>

Title: Senior Lecturer, University of Liverpool
Principal Investigator, Integrative Genomics of Ageing Group

Bio:

Joao Pedro de Magalhaes graduated in Microbiology from the Escola Superior de Biotecnologia in 1999. As a doctoral fellow, he joined the Ageing and Stress Group at the University of Namur in Belgium. With Olivier Toussaint as his advisor, Magalhaes’ work from 1999 to 2004 spanned molecular mechanisms of cellular senescence and responses to oxidative stress, evolutionary models of ageing, and analyses of gene networks.

He then did a postdoc from 2004 to 2008 with George Church at Harvard Medical School. In this role with Church, Magalhaes helped develop high-throughput approaches for studying ageing, including computational tools and databases, statistical models of mortality, methods for cell-based RNAi screens, and comparative genomics methods for investigating the evolution of longevity.

In 2008, Magalhaes joined the University of Liverpool to develop his own group on genomic approaches to ageing. The group was initially in the School of Biological Sciences (which later became the Institute of Integrative Biology), and is now in the Institute of Ageing and Chronic Disease. Joao Pedro de Magalhaes is also an affiliate Principal Investigator in the Neuroendocrinology and Aging Group at the University of Coimbra in Portugal.

Mark Mattson



National Heart, Lung,
and Blood Institute



Title: Senior Investigator, Laboratory of Neurosciences, NHLBI

Bio:

Dr. Mattson received his Ph.D. in Biology from the University of Iowa in 1986. After 3 years of postdoctoral studies in Developmental Neuroscience at Colorado State University, Dr. Mattson took a faculty position at the Sanders-Brown Research Center on Aging at the University of Kentucky Medical Center where he was promoted to Full Professor in 1997. Dr. Mattson is currently Chief of the Laboratory of Neurosciences at the National Institute on Aging, and Professor of Neuroscience at Johns Hopkins University. He is Editor-in-Chief of Ageing Research Reviews and NeuroMolecular Medicine, a Section Editor for Neurobiology of Aging, and an Associate Editor for Trends in Neurosciences. In addition, he has edited 10 volumes in the areas of mechanisms of brain function, stress responses, aging and age-related neurodegenerative disorders. Dr. Mattson is a Fellow of the American Association for the Advancement of Science, and has received numerous awards including the Metropolitan Life Foundation Medical Research Award and the Alzheimer's Association Zenith Award. He is considered a leader in the area of cellular and molecular mechanisms underlying neuronal plasticity and neurodegenerative disorders, and has made major contributions to understanding of the pathogenesis of Alzheimer's disease, and to its prevention and treatment. Dr. Mattson has published more than 400 original research articles and more than 200 review articles and commentaries.

Anne McArdle



UNIVERSITY OF
LIVERPOOL



Title: Head of the Department of Musculoskeletal Biology II, University of Liverpool

Bio:

Professor McArdle graduated with a BSc (Hons) in Biochemistry from the University of Liverpool in 1988 and completed a PhD in the Department of Medicine in 1993. Anne undertook postdoctoral training at the Institute of Gerontology at the University of Michigan and was awarded a Research into Ageing Queen Elizabeth the Queen Mother Fellowship in 1998 to examine the mechanisms by which the age-related failure of muscle to adapt to contractions resulted in sarcopenia. Anne was appointed as Lecturer at the University of Liverpool in 2001 and as Professor in the Faculty of Health and Life Sciences at the University of Liverpool in 2007. She is currently acting Head of the Department of Musculoskeletal Biology II. Anne is past Chair of the British Society for Research on Ageing and the British Council for Ageing. She is an active member of the American Physiological Society and the UK Physiological Society and Biochemical Society. Professor McArdle is Associate Editor for the American Journal of Physiology, International Advisor on the Environmental & Exercise Physiology Committee of the American Physiological Society and a core member of BBSRC Grant Committee A. Professor McArdle's work on frailty has received considerable public interest with press releases and presentation of our applied work to the general public at several events. As School Director of Postgraduate Research, Anne led a complete overhaul of student monitoring and support procedures within the School which has led to substantial improvements in the student experience.

Professor McArdle's research interests include the basic processes by which cells respond and adapt to stress and damage and in particular, the role that the age-related failure in the stress response plays in the development of age-related skeletal muscle dysfunction and has made key observations in this area of research. Her research group has demonstrated the importance of rapid induction of responses to the increased ROS generated by contractions in maintaining muscle viability and the role that attenuation of these ROS signals and responses play in muscle ageing. Anne has considerable experience of cell and molecular biological studies at the sub-cellular level through to physiological analysis of muscle function in a number of model systems including cell culture, animal models and in humans. This work is funded by the National Institutes of Health (USA), MRC, BBSRC and AgeUK.

Simon Melov



Title: Ph.D., Assistant Professor at the Buck Institute for Research on Aging

Bio:

Simon Melov is one of the founding faculty of the Institute, and has been at the Buck since its doors opened in 1999. He has broad expertise in multiple domains and model systems of aging, including *C. elegans* biology, functional decline with age in mice, the role of endogenous oxidative stress in the mitochondria, exercise physiology and age-related disease.

Over the last few years, a key focus of the Melov lab has been to define what “aging» means in the context of different organ systems in aging mice, and to use non-invasive techniques to quantitate and enumerate such functional changes. The end goal is to be able to relate age-related functional decline in mice to human aging. Other research interests include the development of molecular techniques to better understand how single cells change with age, and then to use that understanding to elucidate how such changes impact tissue function.

Dr. Melov has always placed a high value on collaborative studies, believing that in the current research environment, the best science is done by synergizing expertise. Based on multiple collaborations, he has published with most of the faculty at the Institute, and has maintained collaborations with researchers at other locations as well. This approach has consistently resulted in multiple discoveries being made in conjunction with other laboratories within the institute, as well as those outside.

Dr. Melov received his PhD in Biochemistry from the University of London in the UK. He held positions at Emory University in Atlanta and at the University of Colorado in Boulder before joining the faculty of the Buck Institute as an associate professor in 1999.

S. Jay Olshansky

UIC UNIVERSITY OF ILLINOIS
AT CHICAGO



Quote: “My research suggests that slowing down aging will be the next great public health advance in this century because it targets multiple age-related chronic diseases. Importantly, this approach to public health can save far more health care dollars than treating one disease at a time. The time has arrived to take a new approach to chronic fatal and disabling diseases.” - S. Jay Olshansky, 2016 https://www.reddit.com/r/science/comments/4eqnyf/im_s_jay_olshansky_an_epidemiologist_at_the/

Title: Professor, School of Public Health, University of Illinois at Chicago

Bio:

S. Jay Olshansky received his Ph.D. in Sociology at the University of Chicago in 1984. He is currently a Professor in the School of Public Health at the University of Illinois at Chicago, Research [Associate](#) at the Center on [Aging](#) at the University of Chicago and at the London School of Hygiene and Tropical Medicine, and Chief Scientist at Lapetus Solutions, Inc. The focus of his research to date has been on estimates of the upper limits to human longevity, exploring the health and public policy implications associated with individual and population aging, forecasts of the size, survival, and age structure of the population, pursuit of the scientific means to slow aging in people (The Longevity Dividend), and global implications of the re-emergence of infectious and parasitic diseases. Dr. Olshansky is on the Board of Directors of the American Federation of Aging Research; he is the first author of *The Quest for Immortality: Science at the Frontiers of Aging* (Norton, 2001) and *A Measured Breath of Life* (2013); and co-edited *Aging: The Longevity Dividend* (Cold Spring Harbor Laboratory Press, 2015). In 2016, Dr. Olshansky was honored with the Donald P. Kent Award from the Gerontological Society of America, the Irving S. Wright Award from the American Federation for Aging Research, and he was named one of Next Street’s Influencer in Aging.

Alexandre Quintanilha



Title: Director of the Institute for Molecular and Cell Biology (IBMC) in Porto, Portugal

Bio:

Alexandre Tiedtke Quintanilha is the director of the Institute for Molecular and Cell Biology (IBMC) in Porto, Portugal and professor of biophysics. Born in Mozambique he completed his Ph.D. in solid state physics at Witswaterstrand University, South Africa in 1972 and at Porto University, Portugal. From 1972 to 1991 he was professor at University of California, Berkeley and Lawrence Berkeley National Laboratory, California, USA. He became Assistant Director of the Energy and Environment Division and later Director of the Center for Environmental Studies. Back in Portugal since 1991 he has served as the Dean of Science at the University of Porto and as Director of Institute for Molecular and Cell Biology (IBMC). In 2006 he was also appointed as Director of Institute for Biomedical Engineering (INEB) and as Secretary-General of the Council of Associated Laboratories.

His main areas of scientific interest concern physiological stress in animals, microorganisms and ecosystems. Alexandre Quintanilha has published close to 100 articles in peer-reviewed international journals. He is the editor/author of 6 Volumes in areas of Stress and Environment, an editorial consultant of the Encyclopedia of Applied Physics and is on the editorial boards of several international journals. Currently he serves on the committees and councils of a number of scientific organizations, such as the committees for Science and Society and Women in Science of the Federation of the European Biochemical Societies (FEBS), the Council on Research and Exploration of the National Geographic Society, USA, the Council on Biology and Biotechnology of the Foundation for Science and Technology (FST, UK) and a member of the advisory council for the Fundação da Juventude in Portugal. He is the chair of National Agency for Food Safety Scientific Council (Portugal) and the Standing Committee of Life and Environmental Sciences of the European Science Foundation (ESF).

Arvind Ramanathan



Title: PhD, Assistant Professor at the Buck Institute for Research on Aging

Bio:

Dr. Ramanathan is taking an integrative approach to answer fundamental questions in aging and musculoskeletal regeneration. His research career has spanned single DNA molecule microscopy, genomics, chemical biology, mTOR biology, skeletal muscle differentiation, cancer metabolism and metabolomics. He has recently used metabolomics and chemical biology to discover metabolic dependencies induced by oncogenes including ras, he has also identified metabolic signals that mediate mTOR signaling and skeletal muscle differentiation. Using mass-spectrometric and imaging based approaches his laboratory addresses the following questions: What are the molecular signals that integrate nutrients, and organismal and cellular physiology with tissue regeneration? And by what mechanisms does aging affect these molecular signals? How do oncogenic mutations remodel cellular metabolism?

Dr. Ramanathan was born in Pondicherry, India. He earned a doctorate in chemistry from New York University. He carried out his graduate work at New York University and University of Wisconsin Biotechnology Center. His post-doctoral work was performed at Harvard University under the mentorship of Prof. Stuart L. Schreiber, and at the Chemical Biology Program at the Broad Institute of Harvard and MIT as a research fellow. He joined the Buck Institute for Research on Aging in 2011 as assistant professor.

Thomas Rando



STANFORD
UNIVERSITY



Quote: "If we could somehow figure out the mechanisms of aging and are able to intervene, it would potentially offer therapy to a wide variety of diseases — not just cancer, heart disease or Alzheimer's, but all of them." - Thomas Rando, 2011 <http://randolab.stanford.edu/2011/02/new-center-for-research-on-aging-established-with-grant-from-glenn-foundation/>

Title: Professor of Neurology, Stanford University

Bio:

Thomas Rando is a Professor of Neurology at Stanford University. At Stanford, he is also a member of Bio-X, the Cardiovascular Institute, and the Neurosciences Institute. Rando's administrative appointments throughout his career include the following: Founding Director, Muscular Dystrophy Association Clinic, Stanford Medical Center (1999 - 2003); Director, Geriatric Research, Education, and Clinical Center (GRECC), Palo Alto VA Medical Center (2000 - 2007); Chief, Neurology Service, Palo Alto VA Medical Center (1996 - Present); Deputy Director, Stanford Center on Longevity, Stanford University (2006 - Present); Director, Rehabilitation Research and Development Center of Excellence, Palo Alto VA Medical Center (2009 - Present); Director, The Glenn Laboratories for the Biology of Aging, Stanford University School of Medicine (2011 - Present). Honors and awards that Rando has received throughout his career include the following: Frederick E. Terman Fellowship, Stanford University (1996); Paul Beeson Physician Faculty Scholar in Aging, American Federation for Aging Research (1999); Ellison Medical Foundation Senior Scholar Award in Aging, The Ellison Medical Foundation (2004); NIH Director's Pioneer Award, NIH (2005); NIH Transformative R01 (coPI with Dr. Tony Wyss-Coray); NIH (2013). Rando's professional education include a AB from Harvard College in Biochemistry in 1979, an MD from Harvard Medical School in 1987 in Medicine and a PhD from Harvard University in Cell and Developmental Biology in 1987.

Suresh Rattan



Title: Editor-in-Chief of the international peer reviewed journal BIOGERONTOLOGY, published by Springer Publishers.

Bio:

Specialist in the biology of aging and anti-aging.

Discoverer of kinetin and zeatin for their anti-aging properties for human cells.

Proponent of the idea of hormesis - mild stress-induced beneficial effects - in aging research and interventions.

Based on the ideas about hormesis, now screening for natural and synthetic compounds termed hormetins which bring about their health beneficial and anti-aging effects by challenging the systems through stress response pathways.

Very much interested in public communication of science - especially the science of healthy aging - excellent speaker and writer.

Specialties: Skin care, healthy aging, extension of health span, discoverer of kinetin and zeatin for their use as anti-aging molecules and hormetins in cosmetics and nutraceuticals.

Holly Van Remmen



Title: Scientist, Oklahoma Medical Research Foundation

Bio:

Oklahoma Medical Research Foundation scientist Holly Van Remmen, Ph.D., has received the Denham Harman Award from the American Aging Association. The honor, the highest bestowed by the organization, was presented to Van Remmen at the Aging Association's annual meeting in Seattle earlier this month.

Established in 1978, the prize is a lifetime achievement award that recognizes scientists who have made significant contributions to the field of research in aging.

Van Remmen joined OMRF in 2013 and heads the foundation's Aging & Metabolism Research Program. Prior to that, she spent more than two decades as an aging researcher at the University of Texas Health Sciences Center in San Antonio, where she earned her Ph.D. in 1991.

Her work has focused on age-related muscle loss and amyotrophic lateral sclerosis, also known as Lou Gehrig's disease. During her career, she has made a series of important insights on muscle degeneration, and this past year she led study that found new links between traumatic brain injuries and neurodegenerative conditions.

Michael R. Rose



Quote: «Aging is not a relentless process that leads to death, «It's a transitional phase of life between being amazingly healthy and stabilizing.” - Michael R. Rose, 2016, <http://www.businessinsider.com/forget-everything-you-know-about-aging-2016-4>

Title: Professor of Biology, University of California, Irvine

Bio:

On receiving a British Commonwealth Scholarship in 1976, Michael Rose proceeded to the University of Sussex for his doctoral studies under the supervision of Brian Charlesworth and John Maynard Smith. The subject of his doctoral research was the quantitative genetics of aging in *Drosophila melanogaster*.

A federally-funded research faculty position took Rose to Canada in 1981, where he became an Assistant Professor in the Department of Biology, Dalhousie University, Halifax, Nova Scotia. While at Dalhousie, Rose pursued postponed aging, beginning with the organismal physiology involved, particularly energetic metabolism. This work extended aging research in an influential new direction, combining physiology with evolution. In particular, Rose and his students showed that resistance to various stresses was a key factor in postponed aging; flies with the genetic capacity to live longer are better able to resist stress at every adult age.

In 1987, Rose returned to the United States to become an Associate Professor at the University of California, Irvine. In 1990, Rose was promoted to Professor. In 1991, his *Evolutionary Biology of Aging* appeared, a book that ranged from mathematical genetics to cell biology. This work offered a view of aging that was a complete departure from the views that had dominated the aging field since 1960. The journal *Evolution* described the field of gerontology as having become «after Rose». In 1997, Rose received the Busse Prize of the World Congress of Gerontology. In 1998, his book *Darwin's Spectre* was published, a popular introduction to the history and significance of evolutionary biology.

David Sinclair



HARVARD
MEDICAL SCHOOL



Quote: “Unfortunately, because aging is so common and natural, we tend to think of it as destiny or something we should accept. But over the last 300 years, we’ve been fighting diseases that cause us to suffer.” - David Sinclair, 2015 https://www.washingtonpost.com/national/health-science/this-serious-scientist-is-working-on-an-anti-aging-pill--and-taking-it-himself/2015/08/17/07628214-3179-11e5-8f36-18d1d501920d_story.html?utm_term=.6055b4a57fe2

Title: Professor of Genetics, Harvard Medical School

Bio:

David A. Sinclair, Ph.D. is a Professor in the Department of Genetics at Harvard Medical School and co-Director of the Paul F. Glenn Center for the Biological Mechanisms of Aging. He is best known for his work on understanding why we age and how to slow its effects. He obtained his Ph.D. in Molecular Genetics at the University of New South Wales, Sydney in 1995. He worked as a postdoctoral researcher at M.I.T. with Dr. Leonard Guarente where he co discovered a cause of aging for yeast as well as the role of Sir2 in epigenetic changes driven by genome instability. In 1999 he was recruited to Harvard Medical School where his laboratory’s research has focused primarily on understanding the role of sirtuins in disease and aging, with associated interests in chromatin, energy metabolism, mitochondria, learning and memory, neurodegeneration, and cancer. Dr. Sinclair is co-founder of several biotechnology companies (Sirtris, Ovascience, Genocoea, Cohbar, MetroBiotech, ArcBio, Liberty Biosecurity) and is on the boards of several others. He is an inventor on 35 patents and has received more than 25 awards and honors.

Olivier Toussaint

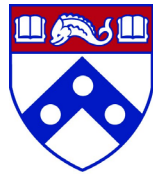


Title: Director of the Laboratory of Cellular Biology of the University of Namur

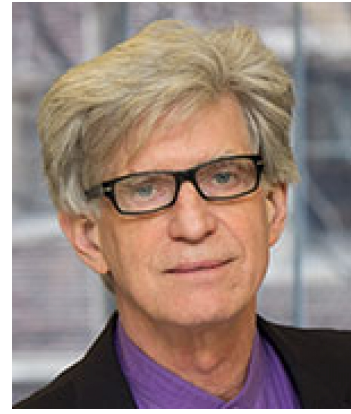
Bio:

Senior Research Associate of the Belgian National Fund for Scientific Research (FNRS), he established his research group twenty years ago in the Laboratory of Cellular Biology (URBC) of the University of Namur. He always loved to share his passion for research with students, through the course on ageing and senescence he delivered at the university, but also by guiding them through their doctoral training. His research activities were first focused on the interactions between stress and cellular senescence, and he was a pioneer in the concept of SIPS or Stress Induced Premature Senescence. He then turned his attention to the toxicity of nanoparticles and created a new group on this subject. Author of more than 100 scientific publications, he was renowned for his expertise beyond Belgium. Readily, he developed European and international collaborations. He was a partner and coordinator of several EU projects in his two areas of research. In particular, he was responsible for two European coordination actions, "Linkage" and "WhyWeAge". These actions resulted in a series of thematic workshops allowing people in the ageing field to connect and collaborate but also to raise awareness of European policymakers on the importance of ageing and the need to support ageing research. By virtue of his visionary and entrepreneurial leadership, Olivier developed several lines of innovative research and was one of the founding pillars of the Straticell company and of the Namur Nanosafety Center. Although he rarely spoke of his illness, he was conscious of its progression and lived his life and his career in the fast lane. The number of projects he brought is impressive, as was his ability to create research networks

John Trojanowski



Perelman
School of Medicine
UNIVERSITY of PENNSYLVANIA



Title: Director of a National Institute of Aging (NIA) Alzheimer's Disease Center
Director of a Institute on Aging

Bio:

Dr. Trojanowski obtained his M.D./Ph.D. in 1976 from Tufts University in Boston. After a medicine internship at Mt. Auburn Hospital and Harvard Medical School, he began pathology/neuropathology training at Massachusetts General Hospital and Harvard Medical School (1977-1979), and completed training at the University of Pennsylvania School of Medicine in 1980 where he was appointed assistant professor of Pathology and Laboratory Medicine (1/1/1981) and rose to tenured full professor in 1990. Dr. Trojanowski holds major leadership positions at the University of Pennsylvania including: Director of a National Institute of Aging (NIA) Alzheimer's Disease Center (1991-present), Principal Investigator of a NIA Program Project Grant on Alzheimer's (AD) and Parkinson's (PD) disease (1990-present), Director of Medical Pathology (1988-2002), Interim Director (2001-2002) and Director (2002-present) of the Institute on Aging, Co-Director (1992-present) of the Center for Neurodegenerative Disease Research, named the first William Maul Measey -Truman G. Schnabel, Jr., M.D., Professor of Geriatric Medicine and Gerontology in 2003 and Co-director of the Marian S. Ware Alzheimer Drug Discovery Program.

For >15 years, Dr. Trojanowski has conducted research on AD, PD, motor neuron disease, dementia with Lewy bodies (DLB), frontotemporal dementias (FTDs) and related disorders. Most of his >500 publications focus on the pathobiology of neurodegenerative disorders, especially the role of abnormal filamentous protein aggregates in these diseases. Dr. Trojanowski received awards for his research including: a MERIT Award (1986-1994) from the National Institutes of Health (NIH), the Metropolitan Life Foundation Promising Investigator Award For Alzheimer's Disease Research (1991), membership in the American Society of Clinical Investigation (1991), an Established Investigator Award from the National Alliance for Research on Schizophrenia and Depression (1994), the Metropolitan Life Foundation Award For Alzheimer's Disease Research (1996), the Potamkin Prize For Research In Pick's, Alzheimer's And Related Diseases (1998), the first Pioneer Award from the Alzheimer's Association (1998), ISI Highly Cited Researcher 2000 (most highly cited neuroscientists for 1981-1999), the Stanley Cohen Biomedical Research Award of the University of Pennsylvania (2000), membership in the Association of American Physicians (2000), the 2004 Irving Wright Award of Distinction of the American Federation for Aging Research, and the 2005 Rous-Whipple Award of the American Society for Investigative Pathology. He was elected President of the American Association of Neuropathologists (1997-1998), and is on the editorial board of several neuroscience and pathology journals.

Dr. Trojanowski was elected to the Institute of Medicine (2002) and he has served and continues to serve on local and national aging research committees including the NIA Neuroscience, Behavior and Sociology of Aging Study Section (1987-1991), the National Advisory Council on Aging (NACA) of the NIA (1994-1998), the NACA Working Group Chair (1996-1998), the Medical and Scientific Advisory Board of the National Alzheimer's Association (1994-1997) as well as of the Southeastern Pennsylvania Chapter of the Alzheimer's Association (1992- present), the NIA Board of Scientific

Counselors (1998-present), the Scientific Advisory Boards of the Paul Beeson Physician Faculty Scholars In Aging Award (1998-present), the Alliance for Aging Research (2002-present) and the Association of Frontotemporal Dementia (2003-present), the Program Committee of the World Alzheimer Congress 2000 (1998-2000), Chair of the «Biology of Synuclein and Cortical Lewy Bodies Associated with Dementia in AD, LBD, and PD» (July, 2001) and «Genetics of Alzheimer's Disease (March, 2002) workshops organized by NIA and the National Institute on Neurological Diseases and Stroke in Bethesda, Maryland, and the Organizing Committee of the 6th (Seville, Spain, 2003) and 7th (Sorrento, Italy, 2005) International Conferences On Progress In Alzheimer's And Parkinson's Disease (2001-2005).

James Vaupel



MAX-PLANCK-INSTITUT
FÜR DEMOGRAFISCHE
FORSCHUNG



Title: Founding Director of the MPIDR

Head of the Laboratories of Survival and Longevity and of Evolutionary Demography

Bio:

James W. Vaupel is Founding Director of the MPIDR and Head of the Laboratories of Survival and Longevity and of Evolutionary Demography. Since January 2013 he has also been heading the newly founded Max-Planck Odense Center on the Biodemography of Aging. James W. Vaupel studied mathematical statistics and received his PhD in public policy at Harvard University. After serving as a professor at Duke University, the University of Minnesota, and the University of Southern Denmark, he became Founding Director of the MPIDR in 1996. James W. Vaupel is a leading scientist in the field of aging research, and has been instrumental in developing and advancing the idea of the plasticity of longevity. He pioneered research on the heterogeneity of mortality risks, and on the deceleration of death rates at the highest ages.

J. Craig Venter

J. Craig Venter[®]
I N S T I T U T E



Quote: ““Your age is your No. 1 risk factor for almost every disease” - Craig Venter, 2014 <https://www.nytimes.com/2014/03/05/business/in-pursuit-of-longevity-a-plan-to-harness-dna-sequencing.html>

Title: Founder, Chairman, and Chief Executive Officer, J. Craig Venter Institute
Institution

Bio:

J. Craig Venter, Ph.D., is founder, chairman, and CEO of the J. Craig Venter Institute (JCVI), a not-for-profit, research organization dedicated to human, microbial, plant, synthetic and environmental genomic research, and the exploration of social and ethical issues in genomics.

Dr. Venter is co-founder, executive chairman and co-chief scientist of Synthetic Genomics, Inc. (SGI) and is also a co-founder and executive chairman of Human Longevity, Inc. (HLI).

Dr. Venter began his formal education after a tour of duty as a Navy Corpsman in Vietnam from 1967 to 1968. After earning both a Bachelor's degree in Biochemistry and a Ph.D. in Physiology and Pharmacology from the University of California at San Diego, he was appointed professor at the State University of New York at Buffalo and the Roswell Park Cancer Institute. In 1984, he moved to the National Institutes of Health campus where he developed Expressed Sequence Tags or ESTs, a revolutionary new strategy for rapid gene discovery. In 1992 Dr. Venter founded The Institute for Genomic Research (TIGR, now part of JCVI), a not-for-profit research institute, where in 1995 he and his team decoded the genome of the first free-living organism, the bacterium *Haemophilus influenzae*, using his new whole genome shotgun technique.

In 1998, Dr. Venter founded Celera Genomics to sequence the human genome using new tools and techniques he and his team developed. This research culminated with the February 2001 publication of the human genome in the journal, *Science*. He and his team at Celera also sequenced the fruit fly, mouse and rat genomes.

Dr. Venter is one of the most frequently cited scientists, and the author of more than 280 research articles.

Jan Vijg



Title: Professor at the Buck Institute for Age Research

Bio:

Jan Vijg, Ph.D. is Professor at the Buck Institute for Age Research, Novato, California. He is the author of more than 200 scientific publications and holds eight patents in research processes and methodologies. In 1989 he helped develop the MutaMouse™, the first transgenic animal engineered to detect gene mutations in a living organism. This allowed scientists to monitor the effects of toxic agents on mouse DNA in any of its tissues or organs. Since that time, he has developed new versions of this mouse model, which make it easy for researchers to monitor ongoing changes in DNA in different tissues or during various developmental stages of the mouse lifespan.

Research at the Vijg Lab is focused on genome instability and the mechanisms through which this may cause human disease and aging. Genome instability is generally considered as a cause of cancer since Theodor Boveri proposed, over 100 years ago, that cancer is based on aberrant chromosome combinations. When in the late 1940s it was discovered that low, daily doses of radiation accelerated symptoms of normal aging in rodents (including non-cancer, degenerative symptoms), the possibility was considered — by such eminent scientists as Leo Szilard, Frank Macfarlane Burnet and Howard Curtis — that genome instability could play a general role in the etiology of human aging and disease.

This possible connection between damage to the genome and aging found strong support in the discovery that heritable defects in genome maintenance are often associated with premature aging, as for example in Werner Syndrome and Hutchinson Gilford Progeria Syndrome. The DNA repair defects present in these conditions, and other defects as well, have been engineered in mice and shown to also cause premature aging in these animals. Interestingly, while such defects sometimes increase both cancer and non-cancer, degenerative symptoms, they often greatly reduce spontaneous tumor formation. This antagonism between cancer and aging is still incompletely understood. In general, research in this area has now begun to attract increased attention and is the main topic of his NIH program project “DNA repair, mutations and cellular senescence” which began in 1999.

Bryant Villeponteau

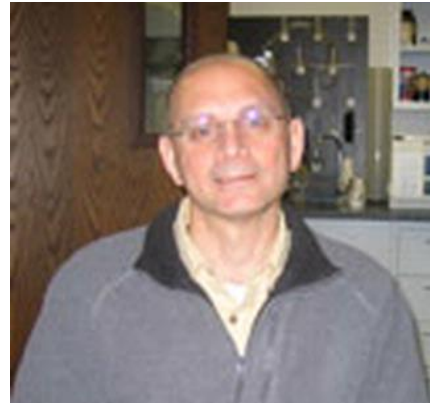


Title: VP of R&D with Genescent Corporation

Bio:

He has 30 years of scientific leadership experience and about 60 journal and patent publications. Dr. Villeponteau holds a B.A. in Economics, a M.A. in Biostatistics, and a Ph.D. in Molecular Biology from UCLA. Bryant was Assistant Research Chemist in the Department of Chemistry and Biochemistry at UCLA for 4 years and Assistant Professor of Biological Chemistry at the University of Michigan Medical School for 6 years. Bryant then led a research group at Geron Corporation for 4.5 years, where he was the lead inventor in cloning human telomerase, thereby winning the Distinguished Inventor Award for the 2nd best US patent of 1997. As the Champion of Telomerase Therapeutics at Geron, he also worked on human stem cells, which were pioneered by Geron in the 90s. Bryant then joined HealthSpan Sciences, Inc. as CSO and later served as CEO for three years. For the next 8 years, Dr. Villeponteau served as a VP/consultant for Sierra Sciences. From September of 2008 to present, Dr. Villeponteau has been the VP of R & D with Genescent Corporation. Bryant also cofounded Centagen, Inc. in January of 2009, and Life Code LLC in October of 2010 to market Stem Cell 100TM.

Stephen Welle

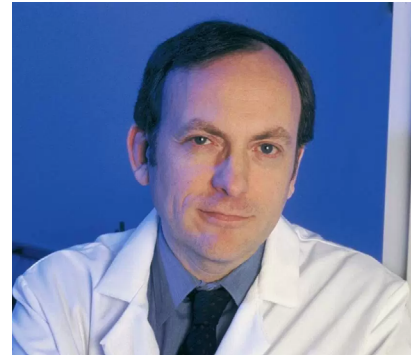


Title: Director of the Functional Genomics Center, University of Rochester

Bio:

After receiving his B.S. in Psychology from the University of Illinois, Dr. Welle obtained a Ph.D. in Neurobiology from Northern Illinois University. Dr. Welle's research deals with regulation of muscle protein metabolism and gene expression, and how these are affected by aging and by endogenous growth factors. He also collaborates with faculty in the neuromuscular research group (neurology department) to study the effects of muscular dystrophies on gene expression. Finally, Dr. Welle plays a critical role in running many of the core facilities at the University of Rochester, serving as Director of the Functional Genomics Center and the CRC Core laboratory.

Michael West



Quote: “My experiences have made me more certain than ever that science can uncover mysteries of nature that we couldn’t have imagined – and unlock their power. And we’re not finished yet – I believe there are still a lot of surprises to come.” - Michael West, 2016

<https://thetranslationalscientist.com/issues/0816/lessons-ive-learned-with-michael-west/>

Title: Chief Executive Officer, BioTime

Bio:

Dr. Michael West is the Chief Executive Officer of BioTime, Inc. He received his Ph.D. from Baylor College of Medicine in 1989 concentrating on the biology of cellular aging. He has focused his academic and business career on the application of developmental biology to the age-related degenerative disease.

He was the founder and first CEO of Geron Corporation and from 1992 to 1998 he was a Director, and Vice President, where he initiated and managed programs in telomerase diagnostics, oligonucleotide-based telomerase inhibition as anti-tumor therapy, and the cloning and use of telomerase in telomerase-mediated therapy wherein telomerase is utilized to immortalize human cells.

From 1998 to 2007 he was President and Chief Scientific Officer at Advanced Cell Technology, Inc. now Ocata Therapeutics where he managed programs in nuclear transfer, retinal differentiation, and PureStem™, a technology for the multiplex derivation and characterization of diverse clonal human embryonic progenitor cell lines.

In 2013 he led BioTime’s efforts to acquire Geron’s stem cell assets through the subsidiary Asterias Biotherapeutics.

Xianmin Zeng



Title: PhD, Associate Professor at the Buck Institute for Research on Aging

Bio:

Dr. Zeng is exploring one of the most hopeful prospects of current biomedical research – that versatile stem cells may some day be a source of replacement cells for damaged tissues of the brain and other organs. She is working toward a treatment for Parkinson's disease, which causes the death of nerve cells in the brain that are needed for agile and controlled muscle movement. Symptoms of the disabling malady include hand tremors and an inability to walk.

Dr. Zeng has developed methods to induce stem cells to transform into the type of nerve cells that are depleted in Parkinson's disease. These nerve cells produce dopamine, a chemical signal that helps deliver the brain's orders to the muscles. Her team has been able to derive such nerve cells from embryonic stem cells, and also from the modified adult cells called induced pluripotent stem cells. These induced pluripotent stem cells can mimic the versatility of embryonic stem cells.

The Zeng lab has tailored a process to make and purify its nerve cell preparations to improve their safety as potential treatments in humans. These nerve cells could also be used for the testing of potential new drugs, and for basic research on the mechanisms of disease.

Dr. Zeng earned a PhD in Molecular Biology at the Technical University of Denmark, and then began research on human embryonic stem cells during her postdoctoral training at the National Institutes of Health. She joined the Buck Institute in 2005. The California Institute for Regenerative Medicine, the state's stem cell research funding agency, has awarded Zeng grant funds to establish a shared research laboratory and to develop a stem cell course as well as to lead an effort to get a stem cell treatment for Parkinson's disease ready for clinical trials.

Alexander Zhavoronkov



Biogerontology
Research Foundation
Prevent. Restore. Preserve.



Quote: “I think that even people past their 70s, who are in good health, have a fighting chance to live past 150.” - Alexander Zhavoronkov, 2015 <http://www.telegraph.co.uk/news/science/science-news/11562492/Meet-the-doctor-who-is-convinced-he-will-live-to-150.html>

Title: Director and Trustee, Biogerontology Research Foundation

Bio:

Alex Zhavoronkov, PhD, is the director and a trustee of the Biogerontology Research Foundation, a UK-based think tank supporting aging research worldwide and is the founder of the International Aging Research Portfolio, a curated knowledge management system for aging research. He heads the laboratory of regenerative medicine at the Clinical Research Center for Pediatric Hematology, Oncology and Immunology where his research interests include Hutchinson-Gilford Syndrome, new methods of cellular reprogramming, molecular mechanisms of skin and cartilage regeneration and personalized medicine in oncology. He is also the international adjunct professor at the Moscow Institute of Physics and Technology.

Together with scientists from Canada, Russia and the US, he co-founded the First Oncology Research and Advisory Center, a personalized medicine organization providing contract research services to oncologists interested in gene expression and activated signalling pathway analysis and predicted effectiveness of targeted drugs to improve clinical decision making.

He is also the head of research at NeuroG Neuroinformatics, a neuroinformatics company developing algorithms for cost-effective EEG devices to recognize imagined visual images and delay the onset of age-related neurodegenerative diseases.

He holds two bachelor degrees from Queen's University, a masters in biotechnology from Johns Hopkins University and a PhD in physics and mathematics from the Moscow State University.