

Faculty

Dr. Sibdas Ghosh, PhD

Dr. Ghosh earned his PhD at the University of Waterloo, Canada and holds the MSc from University of Reading, United Kingdom; BSc from University of Lancaster, United Kingdom; BSc from University of Calcutta, India. Dr. Ghosh's research interests are the effects of environmental stress on gene expression and on the dynamics of protein synthesis in the photosynthetic apparatus; the molecular aspects of membrane turnover; plant microbial interactions; and methods development in bioanalytical and separations chemistry.

Dr. Maggie Louie, PhD

Dr. Louie earned her PhD in Biochemistry and Molecular Biology, with an emphasis on Cancer Biology from University of California, Davis in 2004. Her current research is focused on understanding the development and progression of hormone refractory breast cancer. Dr. Louie's research is focused on how cadmium, a toxic metal found in contaminated food and water and cigarette smoke contributes to the development and progression of breast cancer. Hormone responsive cancer is typically treated with hormone ablation therapy or endocrine therapy to block the ER.

Dr. Vania Coelho, PhD

Dr. Coelho completed most of her doctorate research while she was working as a visiting scientist at the National Museum of Natural History, Smithsonian Institution. After completing her doctorate, Dr. Coelho held a post-doctoral research scientist position initially and later an associate research scientist position, at Columbia University. Dr. Coelho's research focuses on ecology and evolutionary biology of marine invertebrates. Her research interests include benthic community ecology, population biology, behavior, systematics of crustaceans, and coral reef ecology.

Dr. Mohammed El Majdoubi, PhD

Dr. El Majdoubi holds a BS in Physiology (1991), an MS (1992), and a PhD in Neuroscience & Pharmacology (1996) from the University of Bordeaux, France. Dr. Majdoubi worked at the University of Pittsburgh as a Research Associate; in 2000 he joined the University of California San Francisco (UCSF) as a Visiting Scholar, then as an Assistant Research Endocrinologist and director of the Morphology and Cell Imaging Core in the Center for Reproductive Sciences. His current research is focused on mouse embryonic stem cells as an in vitro model of the differentiation and development of hormone-secreting neurons.

Dr. Diara Spain, PhD

Dr. Spain joined the faculty as an assistant professor in the fall of 2002. She holds a Bachelor of Science degree in biology education from North Carolina Agricultural and Technical State University in Greensboro, North Carolina. Her PhD is in biology, with an emphasis in marine invertebrates, from the University of North Carolina at Chapel Hill. Currently, her research focuses on functional morphology and locomotion in echinoderms. Other research interests are skeletal support systems, locomotion in soft-bodied animals.

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Dr. James Cunningham, PhD

Dr. Cunningham joined the department in 1991 and served as the chair of the department from 1998 through 2001. Dr. Cunningham holds a position as research associate in the Department of Mammalogy and Ornithology at the California Academy of Sciences, San Francisco, California. Prior to joining the Dominican faculty Dr. Cunningham taught biology at San Francisco City College, College of San Mateo, College of Marin, College of Notre Dame, Cosumnes River College, and Golden Gate University. Dr. Cunningham's research interests include studies of the vocal behavior and social organization of birds. He is also interested in the growth rates of birds and how this relates to energy allocation during breeding.

Dr. Julie K. Andersen, PhD

Dr. Julie Andersen studies the molecular and cellular mechanisms that give rise to Parkinson's disease (PD). Andersen, among others, has shown that the neuronal death associated with Parkinson's may be caused by an increase in oxidative stress within nerve cells, and that the development of Parkinson's could involve environmental factors as well as genetic predisposition to the disease. Internationally renowned for her work on Parkinson's disease, Andersen continuing work includes examining the regulation of iron levels in newborns and their subsequent susceptibility to Parkinson's, environmental exposure to the herbicide paraquat as a risk factor for Parkinsonian neurodegeneration, the effects of decreases in antioxidant levels (specifically glutathione) associated with the onset of Parkinson's, and the consequences of the age-related increase in an enzyme involved in dopamine metabolism (monoamine oxidase B).

Dr. Chris Benz, MD

A primary goal of the Benz lab is to understand the link between aging and breast cancer-- why the incidence of breast cancer increases with age, how the biology of breast cancer is impacted by normal aging, and how to use this information to improve breast cancer prevention and treatment. The Benz lab focuses on several clinical types of breast cancer including those that overexpress the estrogen receptor (ER), an age-associated mechanism that drives the most rapidly increasing form of breast cancer worldwide, and a proven target for breast cancer prevention and treatment with effective agents like antiestrogens and aromatase inhibitors. Dr. Benz and colleagues also continue to build on their two decades of effort to understand and design treatments against another more clinically aggressive form of breast cancer that overexpresses the ERBB2/HER2 oncogene.

Dr. Martin D. Brand, PhD

Mitochondria oxidize nutrients to release energy, and capture that energy to make ATP. Dr. Brand is particularly fascinated by variations in the efficiency of this process. This leads naturally into study of the efficiency, of how its regulation and its effects on cells and organisms can be described quantitatively, of its mechanism and its functions, and of how we might be able to alter it to affect conditions such as obesity, degenerative diseases and normal ageing. The inefficiency is caused by leaks of protons across the mitochondrial inner membrane. He is investigating basal proton leak catalysed by non-specific processes and inducible proton leak catalysed by specific uncoupling proteins (UCP1, UCP2 and UCP3). Dr. Brand and colleagues are interested in the mechanism of proton transport by these proteins, and how they are regulated by nucleotides, fatty acids, free radicals and other molecules to produce relevant responses to physiological signals.

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Dr. Dale Bredeesen, MD

The Bredeesen laboratory focuses on the molecular processes that control intrinsic cell death pathways. Cell suicide is a vital mechanism for development and maintenance in metazoans, and the loss of the tight regulation of cell death can result in a wide variety of diseases. Cancer develops when cells fail to commit suicide and continue to grow and survive, but too much cell death in particular tissues can also be deleterious, such as occurs with neurodegenerative diseases like Alzheimer's disease. It was long believed by many that a single program—apoptosis—was responsible for programmed cell death. However, alternative pathways have recently been described, including one that the Bredeesen lab dubbed paraptosis. These programs display complementarity and may therefore act as fail-safe mechanisms to ensure that cell death occurs when and where required. Proteomic, genomic, and computational analyses of this novel cell death program suggest that it is a response to hypertrophic cellular stimulation, and thus may be important in preventing autocrine loop-induced neoplasia.

Dr. Judith Campisi, PhD

The Campisi lab strives to understand several fundamental aspects of the aging process. The Campisi laboratory works primarily with human and other mammalian cell cultures and mouse models to study the evolutionary, cellular and molecular relationships between aging, tumor suppressor mechanisms and the development of cancer. The laboratory also studies nuclear structures such as telomeres, and nuclear processes such as DNA repair and transcription, to understand how genetic and epigenetic damage leads to aging and cancer phenotypes. A recent focus of the Campisi lab is to identify links between mitochondrial function and cellular responses that can affect the development of aging phenotypes and age-related diseases in tissues and organisms.

Dr. Lisa M. Ellerby, PhD

Dr. Lisa Ellerby studies cell death mechanisms and polyglutamine expansion disorders, such as Huntington's disease (HD), Kennedy's disease and Machado-Joseph disease. The Ellerby lab is particularly interested in the relationship between protease action and cell death, and has shown, through their characterization of transgenic animal and cellular models, that seven of the eight polyglutamine expansion disease proteins are cleaved by caspases (specialised proteases that trigger cell death). At the forefront of Huntington's research, Dr. Ellerby is dedicated to finding effective treatments that can be applied to all polyglutamine expansion diseases. By examining the sequential cleavage of the huntingtin protein, potential drug targets may be uncovered that could eventually lead to a cure for this debilitating illness. Dr. Ellerby is also involved in the development of methods that attempt to stimulate nerve cell growth to replace those that have been lost in Huntington's sufferers.

Dr. Bradford Gibson, PhD

Dr. Gibson's research focuses on discovering the molecular details of biological processes associated with aging and age-related diseases. Combining biology, chemistry, and technology, Gibson specializes in using mass spectrometry to characterize protein and carbohydrate structures, along with their expression changes and interacting networks. In recent years, Gibson and his team have focused primarily on identifying and characterizing proteins in the mitochondria, a sub-cellular organelle involved in energy production and other cellular functions. They are responsible for one of the most comprehensive databases of mitochondrial proteins that are being examined in relation to aging and disease. In addition, they are developing new proteomic methods for identifying sites of a protein modification that plays key roles in the regulation of protein function in normal and pathological states.

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Dr. Matthew Gill, PhD

Dr. Matthew Gill studies the endocrinology of aging in the nematode *Caenorhabditis elegans*. Dr. Gill's work aims to capitalize on the discoveries that have been made in the genetic analysis of aging in the worm by using a chemical and biochemical approach to study the endocrinology of lifespan determination with particular focus on hormones, small molecules and the process of dauer formation. During development the nematode can enter an alternate larval stage, the dauer larva, which in its natural habitat allows it to survive harsh environmental conditions. Genetic analysis of dauer formation has identified a complex network of genes that regulate this process and a number of these genes define an insulin-like signalling pathway that also acts to determine adult lifespan.

Dr. David A. Greenberg, MD, PhD

Dr. David Greenberg studies mechanisms that the brain uses for protection and self-repair in stroke, and in neurodegenerative diseases like Alzheimer's disease. Two general mechanisms are being investigated in the Greenberg lab: increased expression of neuroprotective proteins and the production of new nerve cells (neurogenesis). The underlying hypothesis is that evolution has selected for biological strategies that promote neuronal survival, and that these can be adapted for therapy. Examples of protective proteins under study include vascular endothelial growth factor (VEGF), which stimulates the growth of new blood vessels in the brain after stroke, but also protects neurons directly, and neuroglobin, an oxygen-binding protein that confers relative resistance to reductions in oxygen or blood supply. Both of these proteins are up-regulated during stroke, and help to reduce the extent of stroke-induced brain damage.

Dr. Robert E. Hughes, PhD

Dr. Robert Hughes utilizes yeast cell-based molecular methods to examine protein interactions that are important in neurodegenerative disorders, such as Huntington's disease. The ultimate goal of the Hughes lab is to use the biochemical tools available in yeast to identify and develop novel therapeutic compounds that will modulate key protein interactions involved with neurodegeneration. With a background in protein biochemistry, Hughes is interested in how proteins fold into the correct shape and the disastrous consequences of when this folding goes awry. Incorrect folding leads to an inability of the protein to perform its specific job, and often results in the aggregation (clumping) of mis-folded proteins.

Dr. Pankaj Kapahi, PhD

The Kapahi lab strives to understand the basic biological mechanisms involved in the aging process. Using an interdisciplinary approach, which combines fly (*Drosophila melanogaster*) and worm (*Caenorhabditis elegans*) genetics, genomics, biochemistry and physiology, Kapahi and his team address how gene-nutrient interactions shape lifespan. Dietary restriction (DR) represents one of the few interventions known to extend lifespan in a variety of species, from yeast to mammals. DR also slows down the progression of number of age related diseases including cancer, neurodegeneration and diabetes in rodents. DR entails a reduction in nutritional level without causing malnutrition that is thought to cause a metabolic shift from a state of reproduction and growth to one of repair and maintenance within the organism, resulting in increased longevity.

Dr. Gordon J. Lithgow, PhD

Dr. Gordon J. Lithgow is dedicated to understanding the basic biological mechanisms involved in the aging process. By using the microscopic nematode worm, *Caenorhabditis elegans*, Lithgow and his team address the molecular, metabolic and evolutionary aspects of lifespan determination and aging rate. Many molecular processes are conserved between simple animals and more complex organisms, thus the Lithgow lab's work yields information that may be applicable to understanding human aging and age-related diseases. Known worldwide for his work establishing links between stress responses and longevity, Lithgow's current research interests include the roles of insulin signalling and molecular chaperones, the regulation of the heat shock response, cell cycle checkpoint pathways and the evolutionary cost of long life.

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Dr. Simon Melov, PhD

Dr. Melov studies the damaging effects of reactive oxygen species (free radicals) generated from the mitochondria, the powerhouses of the cell. Damage caused by these reactive molecules is believed to be a key contributor to the pathobiology of aging, and is implicated in a number of age-related conditions, such as cancer and Parkinson's disease. A variety of interdisciplinary approaches are used to gain insights into mitochondrial dysfunction and aging which is carried out via collaborations with the Nicholls, Gibson, Lithgow, Campisi, and Vijg laboratories within the Institute. The Melov lab is also using microarray technologies to generate gene expression profiles. This methodology simultaneously measures the levels of thousands of genes and is being used to generate profiles of normal aging in multiple species, and to investigate whether antioxidant treatment can retard aging rate, as well as illuminate how the transcriptome of aging varies between species.

Dr. David G. Nicholls, PhD, FRSE

Dr. David Nicholls studies the biochemistry and physiology of the mitochondrion, the 'powerhouse' of the cell. Most of the knowledge available on these sub-cellular structures has been gained by the study of mitochondria isolated from cells, but the Nicholls lab now focuses on developing novel techniques that allow the precise examination of mitochondrial bioenergetics in intact cells. The motto is 'leave the mitochondrion in the cell'. This is now possible because of improved methods for monitoring mitochondrial membrane potential changes and a novel 'cell respirometer' allowing respiration of intact cells attached to coverslips to be monitored continuously.

Dr. Ram Rao, PhD

Dr Rao collaborates with Dr. Dale Bredezen on two areas of research: Endoplasmic Reticulum stress and mechanisms of age-associated neurodegenerative diseases. Neurodegenerative disorders such as Alzheimer's disease, Parkinson's disease, Huntington's disease, Amyotrophic Lateral Sclerosis (ALS) and prion protein diseases all feature misfolded proteins and their aggregates that appear to play a role in disease pathogenesis. Prolonged stress leads to organelle damage and dysfunction and ultimately leads to cell death. We have been investigating the biochemical pathways that couple misfolded proteins to the cell death programs. These studies have led to the identification of several new proteins that function in this link for example, valosin-containing protein, apoptosis-linked gene 2 (ALG-2), and p23 that therefore represent potential therapeutic targets.

Dr. Xianmin Zeng, PhD

Dr. Zeng chose human embryonic stem cells (hESCs) as her major research interest because of the great potential of hESCs in regenerative medicine and developmental biology. One of her laboratory's focuses is the use of hESCs as a potential treatment for Parkinson's disease (PD). This requires an understanding of the molecular and cellular mechanisms that regulate dopaminergic differentiation of hESCs. The Zeng lab is particularly interested in understanding the relationship between transcription factors and neuronal fate, and developing methods for isolating dopaminergic precursors from human embryonic stem cells for transplantation therapy of PD.

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