2025-2026 Placement Options:

Students who are selected for interviews, based on their application, will be contacted to rank their interest in the host lab placements. At this time, they will be able to express locational restrictions.

Graduate Students only			
PI	**Affiliation**	**City**	**State**
Amutha Boominathan	Lifespan Research Institute	Mountain View	California
Amit Sharma	Lifespan Research Institute	Mountain View	California

2025-2026 Summer Scholars Only			
PI	**Affiliation**	**City**	**State**
Amutha Boominathan	Lifespan Research Institute	Mountain View	California
Amit Sharma	Lifespan Research Institute	Mountain View	California
Cyclarity	Cyclarity	Novato	California
Evan Snyder	Sanford Burnham Prebys (SBP) Medical Discovery Institute	La Jolla	California
Chris Wiley	Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University	Boston	Massachusetts
Khalid Shah	Brigham and Women's Hospital (BWH), Harvard Medical School	Boston	Massachusetts
Michael Snyder	Stanford School of Medicine	Stanford	California
Sandra Liebel	University of California, San Diego	La Jolla	California
Gino Cortopassi	University of California, Davis	Davis	California

2025-2026 Post-baccalaureate Scholars Only			
PI	**Affiliation**	**City**	**State**
Amutha Boominathan	Lifespan Research Institute	Mountain View	California
Amit Sharma	Lifespan Research Institute	Mountain View	California
Cyclarity	Cyclarity	Novato	California
Evan Snyder	Sanford Burnham Prebys (SBP) Medical Discovery Institute	La Jolla	California
Chris Wiley	Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University	Boston	Massachusetts
Khalid Shah	Brigham and Women's Hospital (BWH), Harvard Medical School	Boston	Massachusetts
Sandra Liebel	University of California, San Diego	La Jolla	California
Gino Cortopassi	University of California, Davis	Davis	California

Amutha Boominathan (Lifespan Research Institute, Mountain, CA):

Mitochondria are power plants of the cell and are also the only cellular organelle that possess their own DNA in mammals. In humans, mitochondrial DNA (mtDNA) codes for 13 important proteins, all of which assemble into the oxidative phosphorylation relay. Mutations in mtDNA occur because of constant exposure to reactive oxygen species produced by the mitochondrial energy generation process as well as mistakes in mtDNA replication. These mutations accumulate over time due to inefficient repair mechanisms and compromise respiratory chain function. Inherited and acquired mutations in mtDNA result in impaired energy generation and are the cause for several pathologies such as Leber's hereditary optic neuropathy (LHON), Myoclonic Epilepsy with Ragged Red Fibers (MERRF), Kearns-Sayre syndrome and Leigh syndrome. Age-associated mitochondrial dysfunction has been implicated in several neuromuscular diseases including sarcopenia, Alzheimer's, and Parkinson's disease.

The Boominathan lab at SENS Research Foundation is utilizing gene therapy and small molecule-based approaches to develop translational avenues in treating inherited and acquired mutations in the mitochondrial DNA. Using the allotopic approach, we have identified specific targeting elements/ sequences that can improve the expression of these essential genes from the nuclear DNA and their transport to the correct location in mitochondria. The small molecule drug-based approach addresses an important inbuilt cellular mechanism in the selective removal of dysfunctional/ damaged mitochondria aka mitophagy. The summer scholar/ Postbaccalaureate Fellow selected will use a computational approach to design and test a library of constructs in model patient cell lines with specific mutations in mtDNA. The ability of re-engineered genes to rescue function will be evaluated through various techniques, such as protein gels, qPCR, and activity assays, with the potential of extending the studies to animal models.

Additional information: https://sens.org/advancing-mitochondrial-health-with-protective-gene-copies/

Amit Sharma (Lifespan Research Institute, Mountain View, CA):

The ApoptoSENS team investigates the interplay between the innate immune system and senescent cells. The innate immune system maintains tissue homeostasis and responds to cellular damage. Cellular senescence, a state of irreversible growth arrest, is promoted and suppressed by the innate immune system. The innate immune system activates the senescence-associated secretory phenotype (SASP), secreting pro-inflammatory cytokines, chemokines, and growth factors that reinforce senescence and contribute to age-related diseases.

On the other hand, the innate immune system (natural killer cells, macrophages, etc.) can suppress senescence by clearing senescent cells through immune surveillance. Understanding the innate immune system's role in senescence-related pathologies is crucial for developing strategies to prevent or treat age-related diseases. Manipulating the immune response to senescent cells may promote cell clearance and prevent aging-associated chronic inflammation and tissue damage. Targeting the SASP and other immune-mediated mechanisms of senescence may provide new therapeutic avenues. We use molecular and cellular biology assays in cell culture and mouse models.

Our main questions are:

1. Senescent cells contribute to systemic inflammation by affecting innate immune regulators, especially NK cells. Age-related declines in NK cell numbers and cytotoxicity have been observed. Inflammatory factors, such as inhibitory cytokines like IL-10 and TGF-β, partly cause this decline. SASP factors induce inhibitory receptors on NK cells and upregulate inhibitory receptor ligands on senescent cells, further inhibiting NK cell activity. A sub-set of senescent cells can escape NKCC. Senescent human dermal fibroblasts exhibit heterogeneity with distinct gene expression profiles. We investigate if these subpopulations are resistant to immune clearance.

2. While SA β Galactosidase, p16INK4A, p21cip1, etc., are commonly used, they are non-specific. We've identified surface biomarkers of senescence through proteomics and phage display analysis and are validating them. We also developing chimeric antigen receptors-NK cells to target senescent cells in aging and age-associated disease models.

3. Understanding senescence propagation mechanisms is crucial for targeting senescent cells and preventing age-related diseases. Senescent cells secrete the SASP, which propagates senescence autocratically and paracrinally. Our unpublished data suggests that increased protein aggregate secretion might be involved. We're testing whether engineered catalytic antibodies can target protein aggregates like tau oligomers. We also investigate how these proteins drive systemic inflammation and a declining innate immune system.

4. Senescent cell death resistance mechanisms include upregulated anti-apoptotic proteins, activated survival pathways, and decreased senolytic target expression. We've identified a subset of SASP-secreting senescent cells resistant to ferroptosis (iron-dependent apoptosis) and senolytic drugs targeting anti-apoptotic BCL family proteins. We've also developed a novel senolysis approach based on senescent cells' accumulation of labile ferrous iron. Additionally, unpublished data shows another vulnerability of senescent cells that could lead to safer therapeutic interventions. These research projects will advance our understanding of aging and inform novel therapeutic interventions for aging-related diseases.

Additional Information: https://sens.org/new-breakthroughs-in-clearing-out-brain-cell-plaques-from-the-inside/

Amelia Anderson (Cyclarity Therapeutics, Novato, CA):

Cyclarity Therapeutics, a startup-level pharmaceutical company spun out from the SENS Research Foundation, is looking for a motivated summer intern to join our team. Cyclarity is engineering drugs that can bind and reverse the pathological effects of certain oxidized forms of cholesterol implicated in atherosclerosis and several other diseases of aging. The laboratory techniques involved include growing macrophages; flow cytometry to characterize macrophages in various states of differentiation, polarization, and disease; semi-automated (robotic liquid handler) biochemical binding and toxicity assays; ELISA; and other common molecular biology and biochemistry techniques. We may expand to other cell types to investigate other diseases as well.

The choice of a specific project will depend on the skillset and preferences of the trainee. Interested applicants do not need to know all the techniques required to run these assays but should be familiar with routine lab protocols and should not be uncomfortable working with human or animal blood and tissues. Cyclarity also specializes in computational chemistry, so the student may choose to focus on performing molecular dynamics simulations to characterize host-guest complexes. If promising hits are identified from computational experiments, they may be synthesized and tested in the lab.

Additional Information: https://cyclaritytx.com/our-science/

CYCLARITY THERAPEUTICS

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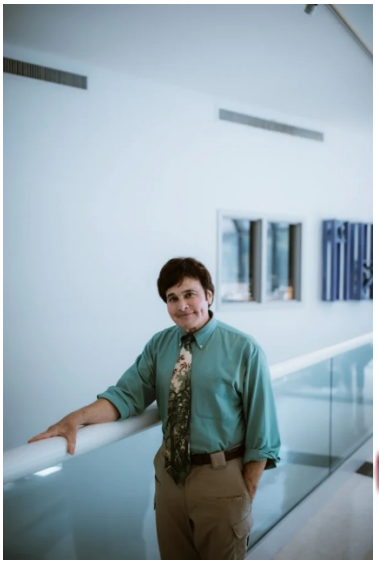
Evan Snyder (Sanford Burnham Prebys (SBP) Medical Discovery Institute, La Jolla, CA):

Our lab heavily focuses on the translation of science to medicine and the clinic to better serve humanity from childhood and into old age. To achieve our goals, we pursue projects that utilize patient derived iPSCs for directed differentiations into organoids modeling the brain and respiratory systems. We utilize the brain organoids to model and investigate the deeper workings of neurodegenerative (Alzheimer's, Parkinson's, etc.) and neuropsychiatric disorders (schizophrenia, bipolar, etc.) in hopes to improve methods of diagnosis and treatment. While lung organoids are utilized for the modeling of respiratory deficiencies (reduced production of mucins due to genetic mutations) and viral agonists (RSV, SARS, etc.) of the respiratory system to better prevent the long-term effects of the correlating conditions. Lastly, we are a primarily cell culture lab, but we occasionally utilize mouse and rat models for ALS and hypoxic ischemic injury respectively and how hNSC treatments can mitigate symptoms.

Additional Information: https://sbpdiscovery.org/labs/snyder-lab/



Sanford Burnham Prebys

Christopher Wiley (Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University, Boston, MA):

Cellular senescence is a process by which a cell undergoes a permanent proliferative arrest - coupled to the secretion of a myriad of inflammatory cytokines, growth factors, proteases, and oxylipins collectively known as the senescence-associated secretory phenotype (SASP). Molecules that allow elimination of senescent cells, known as senolytics, prevent several age-related diseases. In our lab, the SENS Scholars will study the metabolic signatures of senescent cells, potentially working to identify therapies that target these signatures. These will include genetic and pharmacological manipulation of metabolism, and outcomes will include cellular senescence, the SASP, and cell survival. Overall, the scholar will contribute to the identification of new therapies for aging and age-related diseases.

Additional Information: https://gsbs.tufts.edu/faculty-research/chris-wiley-lab#research

JEAN MAYER USDA HUMAN NUTRITION RESEARCH CENTER ON AGING HNRCA

Tufts UNIVERSITY

Khalid Shah (Brigham and Women's Hospital (BWH), Harvard Medical School, Boston, Massachusetts):

The overall mission of our lab (CSTI) is to develop and test novel targeted cell-based immunotherapies for cancer. CSTI capitalizes on the unique expertise in interdisciplinary research spanning the biology of different cell types, gene editing/engineering and cell signaling, as well as our proven abilities to synthesize new insights from disparate fields, to lead interdisciplinary teams into new territory.

Additional Information: https://csti.bwh.harvard.edu/

CSTI Center for Stem Cell and Translational Immunotherapy
Director: Khalid Shah, MS, Ph.D.

Brigham and Women's Hospital
Founding Member, Mass General Brigham

HARVARD MEDICAL SCHOOL

Michael Snyder (Stanford School of Medicine, Stanford, CA):

We are presently in an omics revolution in which genomes and other omes can be readily characterized, and new omics technologies can be applied to understand fundamental biology and improve human health. Our laboratory has invented many technologies to analyze genomes, transcriptomes and other omes.

We have applied these to:

1) Study fundamental principles of regulatory networks

2) Analyze human variation i.e. what makes people different from one another

3) Perform deep omics profiling/big data collection on individuals over time to understand what keeps them healthy and what happens when they become ill or undergo other sorts of changes (e.g. diet, etc)

4) Solve mystery diseases and disease prognosis.

**The lab description is from Dr. Michael Snyder's laboratory website. Additional information can be found here: https://med.stanford.edu/snyderlab/research.html*

Stanford MEDICINE | School of Medicine

Sandra Liebel (University of California, San Diego, La Jolla, CA):

More severe cases of RSV are seen in older patient populations, often characterized by hyperactive inflammatory responses and infectivity. This project aims to elucidate the mechanisms driving this heightened pathogenesis and to explore therapeutic approaches that may expedite recovery. Past research indicates that senescent cells release Senescence-Associated Secretory Phenotype (SASPs), which contribute to chronic low-level inflammation, impair wound healing, and compromise airway barrier integrity. Given that older patients exhibit higher levels of epithelial senescence in their lungs and may not mount an adequate response to viral infections, we hypothesize that cellular senescence in aged lungs leads to an amplified inflammatory response, weakened airway barrier integrity, and increased susceptibility to RSV infection. We will use our novel human lung organoid model to test this hypothesis and identify mechanisms that could be targeted therapeutically to improve outcomes for this at-risk population.

Additional Information: https://www.leibellab.com/

Sanford Consortium
FOR REGENERATIVE MEDICINE

Gino Cortopassi (University of California, Davis, Davis, CA):

We work on anti-aging therapeutics. One set of therapeutics is directed against mTORC1. Another set of therapeutics is directed against Shc. The novel mTORC1 inhibitors extend longevity in mice and yeast and reduce inflammation in innate immune cells. The novel Shc inhibitors reduce inflammation in Amyloid-inflamed innate immune cells in vitro, and rescue memory in two mouse models of Alzheimer's disease in vivo.

Additional Information: https://www.cortopassilab.com/home.html

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