2024-2025 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	Associated SENS strand
Amutha Boominathan	SENS Research Foundation	Mountain View	California	MitoSENS
Amit Sharma	SENS Research Foundation	Mountain View	California	ApoptoSENS and RepleniSENS

2024-25 Postbaccs only				
PI	Affiliation	City	State	Associated SENS strand
Amit Sharma	SENS Research Foundation	Mountain View	California	ApoptoSENS and RepleniSENS
Amutha Boominathan	SENS Research Foundation	Mountain View	California	MitoSENS

Amutha Boominathan (SENS Research Foundation, Mountain View, 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 as a consequence 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 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 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.
https://www.sens.org/engineering-new-mitochondrial-genes-to-restore-mitochondrial-function-mitosens/

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1 Cite Share
The Mitochondrial Genome in Aging and Disease and the Future of Mitochondrial Therapeutics.
Saravanan S, Lewis CJ, Dixit B, O'Connor MS, Stolzing A, **Boominathan A.**
Biomedicines. 2022 Feb 18;10(2):490. doi: 10.3390/biomedicines10020490.
PMID: 35203698 **Free PMC article.** Review.

2 Cite Share
Rapid enrichment of mitochondria from mammalian cell cultures using digitonin.
Dixit B, Vanhoozer S, Anti NA, O'Connor MS, **Boominathan A.**
MethodsX. 2020 Dec 23;8:101197. doi: 10.1016/j.mex.2020.101197. eCollection 2021.
PMID: 34434723 **Free PMC article.**

3 Cite Share
Stable nuclear expression of ATP8 and ATP6 genes rescues a mtDNA Complex V null mutant.
Boominathan A, Vanhoozer S, Basisty N, Powers K, Crampton AL, Wang X, Friedricks N, Schilling B, Brand MD, O'Connor MS.
Nucleic Acids Res. 2016 Nov 2;44(19):9342-9357. doi: 10.1093/nar/gkw756. Epub 2016 Sep 4.
PMID: 27596602 **Free PMC article.**

4 Cite Share
Codon optimization is an essential parameter for the efficient allotopic expression of mtDNA genes.
Lewis CJ, Dixit B, Batiuk E, Hall CJ, O'Connor MS, **Boominathan A.**
Redox Biol. 2020 Feb;30:101429. doi: 10.1016/j.redox.2020.101429. Epub 2020 Jan 11.
PMID: 31981894 **Free PMC article.**

Amit Sharma (SENS Research Foundation, Mountain View, CA):

The research goals of my laboratory are to **(A)** investigate the cellular mechanisms that underlie age-mediated tissue decline caused by cellular senescence and **(B)** investigate how this may be prevented with pharmacological or immune-based interventions that reduce or eliminate senescent cells. we are studying the cellular and molecular pathways involved in aging, inflammation, and the immune system's role in regulating cellular senescence. We intend to develop next generation senotheraputic interventions and biomarkers of aging and age-related diseases like lung fibrosis.

https://www.sens.org/catalyzing-degradation-of-tau-aggregates/
https://www.sens.org/enhancing-innate-immune-surveillance-of-senescent-cells/

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1 Cite Share
Aging of the Immune System: Focus on Natural Killer Cells Phenotype and Functions.
Brauning A, Rae M, Zhu G, Fulton E, Admasu TD, Stolzing A, **Sharma A.**
Cells. 2022 Mar 17;11(6):1017. doi: 10.3390/cells11061017.
PMID: 35326467 **Free PMC article.** Review.

2 Cite Share
Role of immune cells in the removal of deleterious senescent cells.
Kale A, **Sharma A**, Stolzing A, Desprez PY, Campisi J.
Immun Ageing. 2020 Jun 3;17:16. doi: 10.1186/s12979-020-00187-9. eCollection 2020.
PMID: 32518575 **Free PMC article.** Review.

3 Cite Share
Enhanced co-culture and enrichment of human natural killer cells for the selective clearance of senescent cells.
Kim K, Admasu TD, Stolzing A, **Sharma A.**
Aging (Albany NY). 2022 Mar 4;14(5):2131-2147. doi: 10.18632/aging.203931. Epub 2022 Mar 4.
PMID: 35245208 **Free PMC article.**

4 Cite Share
Musashi expression in intestinal stem cells attenuates radiation-induced decline in intestinal permeability and survival in Drosophila.
Sharma A, Akagi K, Pattavina B, Wilson KA, Nelson C, Watson M, Maksoud E, Harata A, Ortega M, Brem RB, Kapahi P.
Sci Rep. 2020 Nov 5;10(1):19080. doi: 10.1038/s41598-020-75867-z.
PMID: 33154387 **Free PMC article.**