Mitochondria lie at the heart of cellular metabolism and energy production. Mitochondrial dysfunction is known to afflict every organ in the human body and has been implicated in numerous diseases including inborn errors of metabolism, brain encephalopathies, muscular disorders, cardiomyopathies, diabetes, and age-related neurodegenerative diseases. Despite its importance however, we lack a comprehensive understanding of mitochondrial biology. A central challenge arises from the fact that they are semiautonomous organelles with their own genome. Defects in the mitochondrial genome constitute a large fraction of the mitochondrial disorders. The Patel Lab studies biology of the mitochondrial genome. We employ the tiny but mighty nematode C. elegans for our research. There are currently four major research efforts underway in the lab:

I. Understand the selection forces that determine the evolution and spread of mutant mitochondrial genomes in populations.
II. Determine the mechanisms that lead to different levels of mutant mitochondrial genomes between cell types.
III. Understand how cells control mitochondrial genome copy number.
IV. Characterize cellular response to defects in mitochondrial RNA processing.

While these four areas of research are connected via a common thread, they span the gamut of scales from evolutionary to mechanistic. In the long-term, taking this broad approach will allow us to gain deeper insights into mitochondrial genome biology than would not be possible otherwise.

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