Light chain (AL) amyloidosis is an incurable disease characterized by the misfolding, aggregation, and systemic deposition of amyloid composed of immunoglobulin light chains causing organ failure and death. Our laboratory has conducted extensive biophysical and biochemical studies of the properties of amyloidogenic light chains in order to understand the role of low thermodynamic and kinetic stability in the process of misfolding and aggregation. We have confirmed that amyloid formation occurs within a thermodynamic range and that kinetic stability could play a role populating partially folded species that promote aggregation.

We have found that both soluble and aggregated light chains internalize into human cardiomyocytes in a size dependent manner. External amyloid aggregates rapidly surround the cells and act as a recruitment point for soluble protein, triggering amyloid fibril elongation. Overall, our studies emphasize the complex interactions between immunoglobulin light chains and cells that result in fibril internalization, protein recruitment, and cytotoxicity that may occur in AL amyloidosis.

Refreshments will be available prior to seminar at 10:45 a.m. in the Shain Atrium

Graduate Students can meet with the speaker in Room 8305F at 1:00 pm