Neuronal growth, maintenance and regeneration are all critically dependent on axonal transport and an increasing number of neuropathologies are associated with disruptions in transport processes. Movement of organelles by fast axonal transport is essential for neuronal functions that include synaptic transmission, conduction of action potentials and supply of trophic factors. Discovery that translocation of membrane bounded organelles (MBOs) in fast transport was mediated by a new class of motor proteins, the kinesins, was a critical step toward providing a molecular basis for neuronal dynamics. In recent years, advances in our understanding of kinesin function and regulation established a foundation for studying the cellular and molecular biology of kinesin-based fast axonal transport. These studies are now providing novel insights into pathogenesis associated with defects in fast axonal transport seen in models for Huntington’s, Alzheimer’s, and Parkinson’s diseases as well as other adult-onset neurodegenerative diseases that proceed as a dying-back neuropathy. We propose the term “dysferopathy” (from the Greek “fero” meaning to "carry" or "transport") to describe pathologies associated with compromises in FAT that lead to a late-onset, dying-back neuropathy.