Crucial to the task of preventing or potentially reducing neuronal injury during Alzheimer disease (AD) is the ability to elucidate the cellular mechanisms that precipitate neuronal degeneration. There is overwhelming evidence implicating impaired metabolism and oxidative damage in AD. The fact that mitochondria are the major sources of energy and reactive oxygen species in cells placed mitochondria in the center of interest, and much evidence has accumulated implying that mitochondrial defects play a key role in the pathogenesis of AD.

This special issue of the Journal of Alzheimer’s Disease reviews and discusses the most relevant data concerning the relationship between mitochondria and AD. As Guest Editors of this special issue we would like to thank all the participants for their excellent contributions, giving all the readers the opportunity to have an update on the field.