José Antonio Enríquezgraduated in Biochemistry and Molecular Biology at the *Universidad Autónoma de Madrid* and obtained his PhD from the *Universidad de Zaragoza* in 1992. His thesis examined various aspects of mitochondrial DNA biogenesis.

From 1993 to 1997 he worked with Giuseppe Attardi at the California Institute of Technology, where he studied the pathogenic action of mutant mitochondrial tRNAs. His work in this period contributed to define the molecular mechanism underlying this phenomenon, and helped to establish the general methodologies for studying mitochondrial tRNAs. These methodologies havefound application in studies of mitochondrial biogenesis and in the analysis of mtDNA-linked diseases. José Antonio established his own laboratory on his return to the *Universidad de Zaragoza*, where he became a Full Professor in 2007. His group has made important contributions to the understanding of mitochondrial biogenesis and bioenergetics, the role of mitochondria in apoptosis, the structure, formation and regulation of the respiratory chain, and the pathological consequences of altered mitochondrial function in human disease. He recently established a possible explanation for the phenotypes associated with common mouse mtDNA variants affecting ROS production. He joined the CNIC in 2009, where his work focuses on the molecular processes underlying the involvement of mitochondrial dysfunction in cardiovascular disease and ischemic processes.