Alzheimer’s disease (AD) is a chronic disorder that slowly destroys neurons and causes serious cognitive disability. AD is associated with senile plaques and neurofibrillary tangles (NFTs). Amyloid-beta, a major component of senile plaques, has various pathological effects on cell and organelle function. Intracellular amyloid-beta may contribute to toxicity by facilitating the hyperphosphorylation of tau, disrupting mitochondrial function and triggering calcium dysregulation. To date, genetic studies have revealed four genes that may be linked to autosomal dominant or familial early onset AD (FAD). These four genes include: amyloid precursor protein (APP), presenilin 1 (PS1), presenilin 2 (PS2) and apolipoprotein E (ApoE). All mutations associated with APP and PS proteins can lead to an increase in the production of amyloid-beta peptides, specifically the more amyloidogenic form, amyloid-beta 42. FAD-linked PS1 mutation downregulates the unfolded protein response and leads to vulnerability to ER stress.