Mitochondrial dysfunction is prominent feature of neurodegenerative diseases and aging. A recent study suggested that levels of mitochondrial fission protein, dynamin-related protein1 (DRP1), phosphorylation at Ser616 are increased in both mitochondrial and cytosolic fractions from AD brains compared with control brains. To identify the link between mitochondrial abnormalities and Alzheimer’s disease, SD rat cortical neurons were treated okadaic acid which induce tau phosphorylation and neuronal death like Alzheimer’s disease. The cells are stained with mitoTracker(red) to observe mitochondrial morphology’s change. As a result, mitochondrial fission is increased in OA treated neurons compared with control cells. These data correlate with EM image. In addition, we performed immunoblot and Immunocytochemistry with p-DRP1(ser616) or MitoSOX. Our data demonstrate the OA triggers DRP1 phosphorylation and mitochondrial ROS. Also mitochondrial fission colocalized with p-DRP1 in OA treated neurons. Our data indicates that Okadaic acid lead to neuron's mitochondrial fission by DRP1 phosphorylation and increase ROS at mitochondria, but decrease mitophagy which is mitochondrial autophagy. These data suggest that altered balance in mitochondrial fission and fusion lead to mitochondrial dysfunction in Alzheimer's disease.