CONCLUSION
The present study has identified systematically the various forms of mitochondrial dysfunctions following in vitro oxidative stress which include loss of cardiolipin, membrane depolarization and a marginal decrease in complex IV activity. Interestingly, a strong parallelism has been noticed between mitochondrial dysfunction existing in aged brain and those induced by in vitro oxidative stress, especially in the context of mitochondrial depolarization and cardiolipin loss. Further a very conspicuous accumulation of oxidative damage products of lipid and protein takes place in aged brain mitochondria along with enhanced production of ROS. It is likely that such age-related mitochondrial dysfunction in brain is the result of enhanced oxidative stress.

Further, in vitro oxidative stress leads to strand breakage in rat brain genomic DNA and also results in gene-specific damage which can be detected by PCR-amplification inhibition of the affected genes in general and gene-specific DNA damages similar have been observed in brain DNA from aged rats indicating oxidative DNA damage during brain aging.

Despite having several forms of mitochondrial dysfunctions and increased ROS production by mitochondria during brain aging, no evidence of apoptosis is available in aged brain. Further, the expression of anti-apoptotic protein bcl-2 is not affected in aged rat brain. The absence of apoptosis in aged rat brain is in agreement with recent studies indicating very little neuronal loss during brain aging.

In totality the study has confirmed in certain measures that oxidative stress plays a central role in brain aging in the context of mitochondrial dysfunction and DNA damage. It will be interesting to study further how antioxidant rich dietary regimen can retard such changes during brain aging. Moreover, it is necessary to
prove further whether mitochondrial dysfunction can be related to cognitive decline which is characteristic of brain aging.