Mitochondrial dysfunction and oxidative stress are associated with the pathogenesis of Parkinson's disease (PD) and dementia with Lewy body disease (DLB). Superoxide dismutase 2 (SOD2) is a key enzyme in mitochondrial defences as it inactivates the superoxide radical produced during oxidative phosphorylation.

Human brain proteins from PD, DLB and control tissue were separated and the molecular forms of SOD2 were determined using Western blotting. The cellular distribution of SOD2 was examined using light immunohistochemistry and confocal immunofluorescence with antibodies to specific cellular markers and with α-synuclein as a marker of Lewy body (Lb) pathology.

SOD2 was an abundant enzyme in all brain regions examined with a molecular weight of 22kD. Light immunohistochemistry indicated that SOD2 was present in most cells with a granular staining and with a network of processes consistent with mitochondrial staining in cells and axons. Control brain tissue stained more prominently than DLB or PD tissue. Confocal immunohistochemistry with cellular markers indicated that neurones and glial cells were all positive for SOD2 consistent with this enzyme being essential in all cells. Co-localising of α-synuclein and SOD2 in PD and DLB showed that mitochondria were sequested into Lb in a progressive manner and in advanced Lb mitochondrial integrity was lost.

In conclusion, these results show that SOD2 is an abundant antioxidant enzyme in human brain and is an excellent marker for mitochondria. The finding that mitochondria are sequested into Lb and the resultant loss of cellular energy may be the mechanism of cell death in these diseases.