Down-regulation of NADH dehydrogenase 1 beta subcomplex 6 implicates mitochondrial dysfunction in MPP+ treated SH-SY5Y cells

도진환*, 조세빈
동양대학교
(jinhwando@dyu.ac.kr*)

Parkinson's disease (PD) is a slowly progressing neurodegenerative disorder. The 1-methyl-4phenyl-1,2,3,6-tetrahydropyridine (MPTP) and its metabolite, 1-methyl-4phenyl-pyridinium (MPP+) have been used to model PD neurodegeneration experimentally in nonhuman primates and mice. The neurotoxic MPTP metabolite, MPP+, is actively transported into dopaminergic neurons by dopamine transporters and is concentrated in the mitochondria. There, it inhibits complex I of the electron transport chain (ETC), which results in mitochondrial dysfunction. We examined gene expression of all subunits in complex I of ETC in the MPP+ treated human neuroblastoma SH-SY5Y cells. Among submits of complex I, only the NADH dehydrogenase 1 beta subcomplex 6 (NDUFB6) showed down-regulation. Since complex I functions in the transfer of electrons from NADH to the respiratory chain, the down-regulation of NDUFB6 might lead to malfunction of the complex I which results in mitochondrial dysfunction. This indicates that lack of NDUFB6 might affect the function of complex I in mitochondria.