# Oxidative stress-induced depolymerization of microtubules and alteration of mitochondrial mass in human cells

謝榮鴻
Lee CF;Liu CY;Hsieh RH;Wei YH

## Abstract

Mitochondrial biogenesis is a biological process that has been intensively studied over the past few years. However, the detailed molecular mechanism underlying this increase in mitochondria remains unclear. To investigate the mechanism of such a mitochondrial proliferation, we examined alterations in mitochondria of human osteosarcoma 143B cells that had been treated with 100 to 500 microM hydrogen peroxide (H2O2) for 48 h. The results showed that mitochondrial mass of the cell was increased with the increase of the concentration of H2O2. On the other hand, by using real-time PCR techniques, we observed the changes of mitochondrial DNA (mtDNA) content in the cells exposed to oxidative stress. The copy number of mtDNA was increased by treatment with a low dose of H2O2 but was drastically decreased after treatment with H2O2 higher than 300 microM. Transmission electron microscopic images revealed that mitochondria were abnormally proliferated in cells exposed to oxidative stress. Moreover, we found that the percentage of 143B cells arrested at the G2/M phase increased upon treatment with H2O2. Immunostaining and microtubule fractionation assay revealed that microtubules were depolymerized in the cells that had been treated with H2O2. To understand the effect of microtubules depolymerization on the mitochondrial mass, we treated the cells with several kinds of microtubule-active drugs, which arrest cultured cells at the G2/M phase. The results showed that mitochondrial mass and mtDNA copy number all were increased after such treatments. Taking these findings together, we suggest that oxidative stress-induced microtubule derangement is one of the molecular events involved in the increase of mitochondrial mass upon treatment of human cells with H2O2.