

Figure 4. Impact of forced overexpression of ND5 gene on in vitro proliferation, invasion and superoxide production of lung cancer cells SW-900. A. The presence of the exogenous fusion protein in the mitochondria of the ND5 transfected cells was detected using anti-myc antibody by immunofluorescence (arrow). B. Number of proliferating cells was significantly higher ($P=0.0031$, indicated by asterisk) in the ND5 mutant (ND5-mt) transfected cells compared to the wild type (ND5-wt) transfected cells (Left panel). Representative photomicrograph from each experimental group is shown in the right panel. Magnification X 200. C. Number of invasive cells was also significantly higher ($P=0.0002$, indicated by asterisk) in the ND5 mutant (ND5-mt) transfected cells compared to the wild type (ND5-wt) transfected cells (Left panel). Representative photomicrograph from each experimental group is shown in the right panel. Magnification X 400 C. D. Number of superoxide producing cells was determined by the MitoSox superoxide assay. Number of positive cells was significantly higher ($P=0.0049$, indicated by arrow) in the ND5 mutant (ND5-mt) transfected group compared to the wild type (ND5-wt) transfected group (Left panel). Representative photomicrograph from each experimental group is shown in the right panel. Magnification X 200.

Supplementary Data

Supplementary Figure 1. Age distribution of mtDNA mutation among the never-smoker and current-smoker lung cancer patients. No significant association was observed ($P=0.631$) between the age groups and mtDNA mutation among the never-smoker or current-smoker lung cancer patients.

Supplementary Figure 2. Distribution of mtDNA mutation in gender groups among the never-smoker and current-smoker lung cancer patients. No significant association was observed ($P=0.253$) between the gender groups and mtDNA mutation among the never-smoker or current-smoker lung cancer patients.

Supplementary Figure 3. Age distribution of mtDNA content among the never-smoker and current-smoker lung cancer patients. No significant association was observed ($P=0.353$) between the age groups and mtDNA content among the never-smoker or current-smoker lung cancer patients.

Supplementary Figure 4. Distribution of mtDNA content in gender groups among the never-smoker and current-smoker lung cancer patients. No significant association was observed ($P=0.623$) between the gender groups and mtDNA content among the never-smoker or current-smoker lung cancer patients.

Supplementary Figure 5. Distribution of mtDNA content index in ethnic groups among the never-smoker and current-smoker lung cancer patients. No significant association was observed ($P=0.336$) between the two ethnic groups (Asia vs. Caucasian) and mtDNA content among the never-smoker or current-smoker lung cancer patients.

Supplementary Table 1. Pattern of somatic mtDNA mutations at different regions of the mitochondrial genome among the lung cancer patients.

Supplementary Table 2. *EGFR* mutations (exon 19 and 21) in the lung cancer patients.

Supplementary Table 3. *KRAS* mutations (exon 2) in the lung cancer patients