AB001. Low mitochondrial DNA copy number is associated with low mitochondrial DNA integrity and advanced T-status in non-small cell lung cancer

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Background: The aim of this study is to appraise the role of mitochondrial DNA (mtDNA) alterations, including the mtDNA copy number and mtDNA integrity (percentage of mtDNA templates without 8-OHdG formation, a marker for oxidative damage to DNA), in non-small cell lung cancer (NSCLC).

Methods: Specimens from a total of 21 NSCLC patients who had received pulmonary resection in Feng-Yuan Hospital were retrospectively collected. The tumor part and the corresponding non-tumor part of the resected lung tissues were subjected to DNA extraction. The copy number and integrity of mtDNA were analyzed by quantitative real-time polymerase chain reaction (Q-PCR).

Results: Active/ex-smoking (P=0.023), advanced T-status (T1/T2/T3/T4, P=0.028), and advanced N-status (N0/N1/N2, P=0.039) were poor prognostic variables related to shorter survivals. Among the non-tumor parts, patients with active or previous smoking tended to associate with a lower mtDNA integrity (i.e., higher oxidative mtDNA damage, P=0.088) but a higher mtDNA copy number (P=0.066) in lung tissues. Among the tumor parts, a lower mtDNA integrity (i.e., higher mtDNA oxidative damage) was associated with a lower mtDNA copy number (P=0.035). Besides, advanced T-status (T3/T4 *vs.* T2/T1, P=0.017; P=0.007), longer tumor diameter (P=0.024; P=0.025) and smaller body mass index (BMI) (P=0.029; P=0.046) were related to a lower copy number and a lower copy ratio of mtDNA.

Conclusions: The non-tumor parts harbored the ability to increase total mtDNA copy number to compensate for the oxidatively damaged mtDNA during cigarette smoking. However, the tumor parts seemed to lose such a compensatory ability during tumor progression. We suggest that alterations of mtDNA may play an important role in the carcinogenesis and progression of human NSCLC.

Keywords: Mitochondrial DNA (mtDNA); oxidative damage; non-small lung cancer (NSCLC)

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