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Alveolar type II (ATII) cells make and secrete pulmonary surfactant, and they proliferate to restore the epithelium after damage to the more sensitive alveolar type I cells. Emphysema is caused by the destruction of alveolar wall septa. The pathogenic mechanisms of this disease are still not fully understood and effective therapy is limited. Cigarette smoking induces oxidative stress and is the most common cause of pulmonary emphysema. Control ATII cells obtained from non-smoker and smoker organ donors, whose lungs were not suitable for transplantation and donated for medical research. We also isolated ATII cells from excess tissue obtained from lung volume reduction surgery in patients with emphysema. DJ-1 protects cells from oxidative stress and induces Nrf2 expression, which activates the antioxidant defense system. We analyzed the cell injury obtained from non-smokers, moderate smokers and heavy smokers. In ATII cells isolated from moderate smokers, we found high DJ-1 expression. However, we observed lower DJ-1 expression and high oxidative stress in heavy smokers. Moreover, we found high inflammation and apoptosis in ATII cells obtained from heavy smokers. Furthermore, we found the highest ROS generation in ATII cells obtained from patients with emphysema. We cultured ATII cells obtained from heavy smokers in vitro and overexpressed DJ-1 by adenovirus construct. We found that this induced NRF2 and decreased ATII cell apoptosis caused by cigarette smoke. Our results indicate that DJ-1 activates the Nrf2-mediated antioxidant defense system. In addition, DJ-1 overexpression can restore the impaired Nrf2 pathway leading to ATII cell protection in heavy smokers. This suggests a potential therapeutic strategy for targeting DJ-1 in cigarette smoke-related lung diseases. An impaired antioxidant defense system regulated by DJ-1 and the contribution of ROS to cell injury in ATII cells isolated from patients with emphysema indicated in our results.

“Oxidative stress-induced alveolar type II cell injury”

Host: S. Zacchigna

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