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Effects of NF-κB inhibitor on the regulation function of cigarette smoke extract to human β defensins in the immortalized human oral mucosal epithelial cell line

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Cigarette smoke increases the susceptibility to oral mucosal infection and is a risk factor for malignant transformation. Cigarette smoke is a mixture of thousands of toxic components generated upon the burning or heating of tobacco leaves. The toxic components first interface with the immune system at the oral mucosal surfaces. NF-κB is a new member of the family of transcription factors, which positively regulate the expression of many genes involved in inflammatory and other responses, including human β defensins (hBDs). The hBD family is one type of cationic antimicrobial peptides that can be secreted by epithelial cells. Among hBD family, hBD-1, -2 and -3 are critical members of the defense system of oral mucosal epithelium. We treated immortalized human oral mucosal epithelial (Leuk-1) cells with various concentrations of cigarette smoke extract (CSE) for 24 h. Western blotting and immunofluorescence assays were performed to study CSE-induced alteration of NF-κB, and P-NF-κB protein expression. The change of hBDs expression were tested using qPCR and ELISA. And then we adopted BAY 11-7082, a specific inhibitors of NF-kB, to inhibit the activation of NF-kB signalling. Different concentrations of NF-κB inhibitor NF BAY 11-7082 were treated to Leuk-1. Then 10 μM BAY 11-7082 was added to the cell culture 24 h before the addition of 4% CSE for 24 h. Leuk-1 cells were treated with 0.5% DMSO as a mock-treated control. Real-time PCR and ELISA were performed to detect the mRNA levels and secretion of hBD-1, -2, and -3, respectively. In this research, we found CSE treatment suppressed NF-κB expression and activated P-NF-κB expression in Leuk-1 cells. The mRNA and secretory levels of hBD-1 and -3 were down-regulated by CSE, while the mRNA and secretory level of hBD-2 were up-regulated by CSE. The BAY 11-7082 treatment significantly abrogated the inhibitory effect of CSE on hBD-1 mRNA expression and release, BAY 11-7082 treatment remarkably reversed the induced effect of CSE on hBD-2 mRNA level and release, while BAY 11-7082 treatment clearly removed the inhibitory effect of CSE on hBD-3 mRNA level and release. The present study indicated that CSE regulated the expression levels of hBDs via down-regulated NF-κB in oral mucosal epithelial cells.

Biography

Qian Yajie completed her Master's degree from Nanjing University. She is now a Clinical Doctor at Nanjing Stomatological Hospital Medical School of Nanjing University. Since the beginning of the graduate student stage, she has been engaged in Oral Medicine research. So far, she has published a paper as the first author, in *Cellular Physiology and Biochemistry*. She contributed in the other three papers, which were all included in SCI. In recent years, she has participated in several research projects, and won a number of awards, including "New Technological Introduction Award" from the Health Department of Jiangsu Province in 2014.

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