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Effects of apigenin and wogonin on NF-kB Signaling involved in mucin production from human mucoepidermoid carcinoma cells

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In this study, we investigated whether the flavonoids, wogonin and apigenin, significantly affect MUC5AC mucin gene expression and production in human mucoepidermoid carcinoma cells. Confluent NCI-H292 cells were pretreated with wogonin or apigenin for 30 min and then stimulated with tumor necrosis factor-α (TNF-α) for 24 h or the indicated periods. The MUC5AC mucin gene expression and mucin protein production were measured by reverse transcription - polymerase chain reaction (RT-PCR) and enzyme-linked immunosorbent assay (ELISA), respectively. We found that incubation of NCI-H292 cells with wogonin or apigenin significantly inhibited mucin production and down-regulated MUC5AC gene expression induced by TNF- α, in a dose-dependent fashion. To elucidate the action mechanism of wogonin or apigenin, effect of each compound on TNF-a-induced NF-kB signaling pathway was investigated by western blot analysis. Wogonin inhibited NF-kB activation induced by TNF-a. Inhibition of IKK by wogonin led to the suppression of IkB phosphorylation and degradation, p65 nuclear translocation and NF-kB-regulated gene expression. This, in turn, led to the down-regulation of MUC5AC protein production in NCI-H292 cells. Wogonin also inhibited the gene products involved in cell survival (Bcl-2) and proliferation (cyclooxygenase-2). Apigenin inhibited NF-κB activation induced by TNF-α. Inhibition of inhibitory kappa B kinase (IKK) by apigenin led to the suppression of inhibitory kappa B alpha (IκBα) phosphorylation and degradation, p65 nuclear translocation. This, in turn, led to the down-regulation of MUC5AC protein production in NCI-H292 cells. Apigenin also has an influence on upstream signaling of IKK because it inhibited the expression of adaptor protein, receptor interacting protein 1 (RIP1). These results suggest that wogonin and apigenin inhibit the NF-kB signaling pathway, which may explain their roles in the inhibition of MUC5AC mucin gene expression and production from human mucoepidermoid carcinoma cells.

Biography:

Dr. Choong Jae Lee is a Full Professor of Medical Pharmacology at the School of Medicine, Chungnam National University in Daejeon, South Korea. He obtained his PhD in Cellular and Molecular Pharmacology from the College of Pharmacy, Seoul National University in 1997. Dr. Lee did a postdoctoral training at Seoul National University where he carried out studies in pulmonary and anticancer pharmacology focusing the development of candidates for novel therapeutics. He worked as a research fellow at the School of Pharmacy, University of Maryland at Baltimore under advice of Dr. K. Chul Kim, in 1995. He joined Chungnam National University in 2002 as an Assistant Professor and is continuing his research in anticancer pharmacology with emphasis on elucidation of mechanism and action of potential chemopreventive natural products. He spent his sabbatical year at the University of California Santa Barbara studying cancer biology with the renowned professor Dr. Leslie Wilson, in 2008. Dr. Lee published over 40 research publications, majorly dealing with bioactive natural products, in top peer-reviewed scientific journals.