รายงานการวิจัยฉบับสมบูรณ์

ชื่อโครงการ (ไทย) ผลผล interleukin-18 ด้วยการคำนวณของวงจรเซลล์ และการขั้นนำการตายของเซลล์แบบ apoptosis ในเซลล์ KB
(อังกฤษ) Effect of interleukin-18 (IL-18) on cell cycle progression and induction of apoptosis in KB cell line.

ชื่อหัวหน้าโครงการ คร.อธิป นิตาภัสร์
ภาควิชาชุษึ่งวิทยา คณะวิทยาศาสตร์ มหาวิทยาลัยสงขลานครินทร์
แหล่งทุน เงินรายได้คณะวิทยาศาสตร์ ประเภททั่วไป ปีงบประมาณ 2546
Abstract

Objectives: Interleukin-18 (IL-18), a pro-inflammatory cytokine that is produced by both lymphoid and non-lymphoid cells, has a critical role in modulation of innate and adaptive immunity. Its primary function in stimulation of IFN-γ production, stimulation of NK cell cytotoxic activities makes this cytokine a candidate for cancer immunotherapy. In oral cavity, this cytokine is produced by oral epithelia and carcinoma cells and is related to tumor regression in nude mice bearing salivary adenocarcinoma. However, direct effects of this cytokine on oral cancer cells have not been elucidated.

Methods: In this project, we investigated IL-18R expression in an oral carcinoma (KB) cell line using RT-PCR. Effect of this cytokine on cell proliferation, toxicity, and induction of apoptotic cell death were studied using MTT assay, LDH assay and apoptosis assays, respectively.

Results: KB cell line was found to express IL-18 receptors (IL-18Rα and IL-18Rβ), indicating that this oral carcinoma line can be used as a target for IL-18 study. We showed that recombinant human IL-18 inhibited KB cell proliferation by 17 percent at concentration of 100 ng/mL (p<0.05), whereas LDH release by these cells in treatment group and control groups was comparable, indicating that IL-18 suppression of cell proliferation was not mediated by the induction of cell death. To further address this hypothesis, we found that IL-18 treatment did not induce apoptotic cell death as studied by DNA laddering and TUNEL assays. However, analysis on cell cycle progression of these cells revealed that IL-18 treatment led to cell cycle delay.

Conclusions: Findings in these studies indicated that suppression of KB cell proliferation was attributed to control of cell cycle, growth delay or induction of cell differentiation. The data presented in this project could provide an insight of how oral carcinoma cells directly respond to IL-18 as this cytokine is an important regulator of anti-cancer mechanisms.