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Infection with CagA+ *Helicobacter pylori* induces epithelial to mesenchymal transition in human cholangiocytes

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Recent reports suggest that the East Asian liver fluke *Opisthorchis viverrin*i serves as a reservoir of *Helicobacter pylori*, which is implicated in pathogenesis of Opisthorchiasis-associated cholangiocarcinoma(CCA). The affected cholangiocyte lining intrahepatic biliary tract might be the origin of CCA. Here we investigated interaction of CagA+ve *Helicobacter pylori* and *Helicobacter bili*s with H69 cells, an immortalized form of human cholangiocyte. Exposure of H69 cells to increasing numbers of H. pylori at 0, 1, 10,100 bacteria per H69 cell for 24 hours induced morphological changes in cholangiocytes including the appearance of mesenchymal phenotype, profusion of thread-like filopodia and loss of cell-cell contact, in dose-dependent fashion. In parallel, changes in mRNA expression followed exposure to *H. pylori*, with increased expression of Epithelial to Mesenchymal Transition (EMT) associated-factors including snail, slug, vimentin, matrix metalloprotease, zinc finger E-box-binding homeobox, and cancer stem cell marker CD44. Transcription levels encoding cell adhesion marker CD24 decreased. Analysis in real time using the xCELLigence approach revealed that exposure to 10 to 50 of *H. pylori* stimulated migration of H69 cells and CCLP1 cells, a derived form of human cholangiocarcinoma, and invasion through Matrigel extracellular matrix. Similarly, 10 bacteria of CagA+ve *H. pylori* but not *H. bilis* stimulated contact-independent colony establishment in soft agar. These findings support the hypothesis that infection with H. pylori contributes to the fibrogenesis and malignant transformation of the biliary epithelium.

Keyword: EMT, *Helicobacter pylori*, cholangiocarcinoma.

Biography

Prissadee Thanaphongdecha has done Doctoral of philosophy in Pathobiology and Bachelor degree in Medical science. He is currently working in Faculty of medicine, Khonkaen university, Thailand

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