

Helicobacter pylori Changes Microprocess on the Surface of Cultured AGS and Hep-G2 Cells

Chang-Sub Uhm, Hun Jae Chun¹, So Hyun Ahn, Jeong Whan Lee²,

Eun Kyung Park, Dong Gyu Park¹ and Jin Hae Hyun¹

Department of Anatomy, ¹Department of Internal Medicine and Institute of Digestive Disease and Nutrition, Korea University College of Medicine, ²Department of Internal Medicine, College of Medicine, Inje University

H. pylori adhere gastric epithelial cells to induce various upper gastrointestinal diseases. Surface microprocesses such as filopodia and lamellipodia are tools of the cell to respond to the signals in the environment. Recently it was reported that the adhesion of H. pylori to cultured AGS cells activates rho GTPases. In this study, the authors tried to elucidate whether there H. pylori could induce the surface microprocess changes by electron microscopy.

AGS, and Hep-G2 cell lines were purchased from ATCC and cultured on 8-chamber slides for 3 days using RPMI-1640 with appropriate additives (10% FBS, penicillin-streptomycin, Fungizone). H. pylori (ATCC 43504) was a gift from Dr. Kim Kyung Hyun (Graduate School of Biotechnology, Korea University) and cultured for 3 days in Brucella broth with 10% FBS. The cultured H. pylori were added onto the cultured cells for 30 min., 1 hr., 2 hrs., and 4 hrs. The cocultures were then, processed for transmission and scanning electron microscopy and observed with Hitachi H-600 and S-450 electron microscopes.

Scanning electron microscopy revealed a interweaving network of thin processes on the control Hep-G2 cell surface, and short microvilli on the control AGS cell surface. These processes disappeared with the contacts by H. pylori at first, except for the direct contact area, which was most prominent at 2 hr on AGS cells. After 2 hr coculture, new microvillous processes reappear on the surface of Hep-G2 cells. At the area where H. pylori directly attached, the microprocesses of both Hep-G2 and AGS cells were mostly lamellipodial membrane ruffle, which had very intimate relation with H. pylori.

Transmission electron microscopy showed the changing density of microprocesses on the Hep-G2 and AGS cells by the adhesion of H. pylori. However, no apparent changes of

actin filaments were observed at the cortical cytoplasm.

These results suggest that the changing form and number of the surface microprocesses on Hep-G2 and AGS cells may be caused by the rearrangement of the preexisting actin filaments, not by the formation of new actin filaments. Also, it is suggested that the changing form may be the results of selective activation of Rho GTPase subtypes by *H. pylori* adhesion. The significance of these findings to the pathophysiology of *H. pylori* needs to be explored.

(This study was supported in part by the 1999 Research Grant of Medical Science Research Center of Korea University to HJ Chun.)

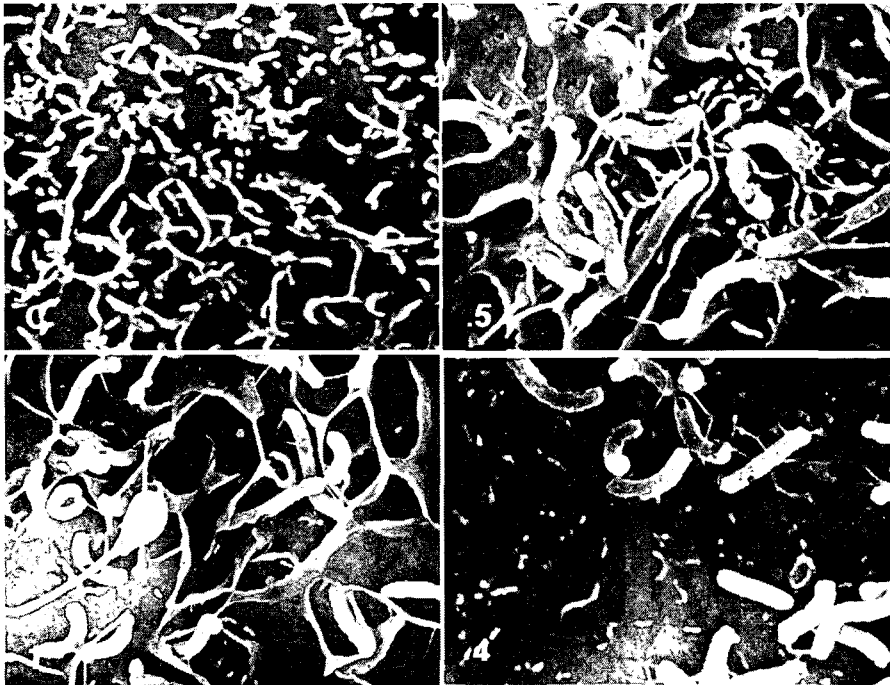


Figure. Surface microprocess changes of AGS cells induced by *H. pylori*. In control (C), surface of AGS cells is covered mostly by microvillous projections of various length. *H. pylori* induces the formation of lamellipodial projections, which is most prominent at 1 hr coculture (I). At 4 hr coculture (4), the density of microprocesses is decreased and most of the surface processes are short microvilli.