S1-1

IMMUNE RESPONSE TO TSUTSUGAMUSHI DISEASE

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Tsutsugamushi disease is widespread in Asian-Pacific regions and it is assumed that about 50,000 new patients occur every year in Korea. Cellular and humoral immunity are involved in protection to *Orientia tsutsugamushi* infection and as the immunity to *O. tsutsugamushi* infection is strain-specific, 56-kDa protein, a strain-specific molecule, has been believed to be important in inducing protective immunity. To confirm this, we immunized C3H mice with the recombinant Bor56, a recombinant protein of 56-kDa protein of *O. tsutsugamushi* Boryong that contains 83rd to 534th amino acids, and could induce protective immunity. Antibody could be demonstrated by IFA assay in the sera of mice and also IL2 and r-IFN production could be detected by stimulating splenic mononuclear cells with *O. tsutsugamushi.* Although protective level induced by recombinant Bor56 is relatively low when compared with that induced by live *O. tsutsugamushi,* it can be expected that immunization with the recombinant antigen could prevent or modify the severity of the disease.

S1-2

GASTRIC EPITHELIAL IMMUNE RESPONSES TO *HELICOBACTER PYLORI* INFECTION: THE PROINFLAMMATORY GENE PROGRAM AND APOPTOSIS

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Gastric epithelial cells (GEC) that line the gastric mucosa are an initial site of interaction between *H. pylori* and the host. Early after bacterial infection, GEC rapidly activate NF-κB which is a central regulator of the epithelial cell immune response. Following activation of NF-κB, GEC upregulate the expression of a proinflammatory host gene program consisting of cytokines which can signal the initiation of the gastric mucosal inflammatory response. A second component of the GEC response to *H. pylori* infection includes the upregulated expression of COX-2, a key enzyme in prostaglandin synthesis and a regulator of epithelial cell growth, and the upregulated expression of iNOS. The upregulated expression of the adhesion molecule ICAM-1 may control epithelial cell-neutrophil interactions. Apoptosis is a later occurring GEC response to *H. pylori* infection. The apoptotic GEC response and proinflammatory neutrophil response are be linked, since immune mediators, including soluble form of Fas ligand and TNF α, produced by neutrophils can regulate the apoptotic response of GEC. The latter may function to cause mucosal cell loss manifesting acute gastric ulcer or chronic atrophic gastritis.