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Host Immune Response to Gastric *Cryptosporidium*

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Genus *Cryptosporidium* are intracellular protozoan parasites that infect primarily epithelial cells of gastrointestinal tract and cause diarrheal disease worldwide. Molecular mechanism of host immune response to the parasites has not been well understood, especially, knowledge of the mechanism to gastric *Cryptosporidium* is extremely less. This study analyzed changes of cytokine expression levels in gastric local immune response to gastric *Cryptosporidium* by using mice with different immune properties.

Here, we used *Cryptosporidium andersoni* Kawatabi types to conduct the experimental infection. Firstly, we orally inoculated 1.0×10^6 oocysts into BALB/c mice, which is immunocompetent, and counted oocysts shed in feces in certain intervals. According to the observed oocyst shedding shifts, three periods were determined as follows: period 1 was high oocyst shedding, period 2 was repressing of shedding, period 3 was no oocyst. In the next experiment, the same number of oocysts was orally inoculated into BALB/c mice (normal immune state) and SCID mice (immunological incompetence state). Their stomachs were collected in each period, and their total RNA was extracted. Then, expressions of four cytokines (IFN- γ , IL-4, IL-10, IL-12) were compared by quantitative RT-PCR.

Oocyst shedding peaked in 8 to 17 days post inoculation (DPI) in BALB/c mice, and this term was determined as period 1. Then, oocyst shedding gradually decreased and was not detectable after 41 DPI. Therefore, period 2 was from 18 to 40 DPI, and period 3 was after 41 DPI. In the comparison of IL-4 expression, significant difference was not observed in any analyzed periods nor in any species. By contrast, IFN- γ highly expressed in period 2 of BALB/c mice, while its increase was not shown in SCID mice during the period. Therefore, it was suggested that Th1 immune response including IFN- γ might eliminate gastric *Cryptosporidium*.