

The 18th Leading Special Lecture

Protective
immunity
to helminths



The Jikei University School of Medicine
Prof. NAOHIRO WATANABE

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Lecture Hall, Graduate School of Veterinary Medicine,
Hokkaido University, JAPAN

One Health

Hokkaido University Leading Graduate School
Veterinary Science for One Health



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1969 B.S. Tokyo University of Education

1971 Research associate, Department of Parasitology, Jikei University

1974 Research scientist, Department of Pathology, New York University

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Protective immunity to helminths

The helminth infection induces Th2 cells in the host which result in IgE production, eosinophilia and mastocytosis through cytokines. These Th2 responses are considered to be responsible for the protection to helminth. We evaluated protective role of IgE by comparing between IgE-deficient and IgE-producing mice with helminth infection. IgE antibody dependent protection was found in some helminth infections. In addition, we reported an IgE level regulatory gene, which controls IgE production both antigen-nonspecifically and IgE-isotype-specifically. This gene functions as a helminth protective gene in mice infected with *Trichinella spiralis*. Mast cells are also responsible for protection with IgE antibody in the expulsion of *Vampirolepis nana* adult worms from the intestine and in acquired immunity against ticks *Haemaphysalis longicornis* in the skin. Basophils, having some similar characteristics to mast cells, are involved together with IgE antibody in acquired immunity to *Nippostrongylus brasiliensis* and ticks in the skin. In the case of innate immunity, oral infection with *Vampirolepis nana* eggs induces strong protection to reinfection with eggs within 2 days in mice. CD4+ $\alpha\beta$ T cells and CD4+ $\gamma\delta$ T cells are participated in this innate immune response through costimulatory signals and IL-4 and/or IL-13.