A framework of host immune responses against four types of parasitic infections

Running title: Immunity against parasites

Wan-Chung Hu*
*Correspondence: Wanchung.Hu09@gmail.com
Department of Clinical Pathology, Taipei Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation
No. 289, Jianguo Road, Xindian District, New Taipei City 231, Taiwan

Abstract
Host immunity against parasitic infections are complicated. It will be categorized into four immunological pathways against four types of parasitic infections. For intracellular protozoa, the host immunological pathway is TH1 immunity with macrophages, IFNg CD4 T cells, CD8 T cells, and IgG3 B cells. For free-living extracellular protozoa, the host immunological pathway is TH22 immunity with neutrophils, IL-22/IL-17 CD4 T cells, and IgG2 B cells. For helminths (endoparasites), the host immunological pathway is TH2a immunity with eosinophils, IL-5/IL-4 CD4 T cells, and IgG4 B cells. For insects (ectoparasites), the host immunological pathway is TH2b immunity with basophils, mast cells, IL-3/IL-4 CD4 T cells, and IgE B cells. Thus, the framework of the whole immunological pathways against four types of parasitic infection is given.

Key words: TH1, TH2, TH17, eosinophils, basophils, protozoa, helminths, insects

Introduction

Host immunological pathways against parasitic infections are complicated. Parasites include protozoa, helminths, and insects. Previously, I have proposed a framework of all discovered host immunological pathways and their roles for four specific types of pathogens and hypersensitivities[1]. Here, I am extending the previous framework and proposing a new framework of host immunological pathways for the four types of parasitic infections.

Intracellular protozoa and TH1 immunity

For intracellular protozoa, the host immunological pathway is TH1 immunity with macrophages, IFNg CD4 T cells, CD8 T cells, and IgG3 B cells. TH1 immunity is the host immune response against intracellular pathogens. It is important that intracellular location is more important than the pathogen types. Thus, TH1 immunity can be triggered to defend intracellular bacteria, fungi, and protozoa. Activated macrophages are the key effector cells to digest intracellular bacteria, fungi, and protozoa. Intracellular protozoa are categorized into the parasite groups. Intracellular protozoa including malaria, leishmania, toxoplasma, babesia, and cryptosporidium can all trigger TH1 host immune response against these pathogens.[2-6] Intracellular bacteria such as chlamydia and intracellular fungi such as histoplasma can also trigger TH1 immunity.

Extracellular protozoa and TH22 immunity

For free living extracellular protozoa, the host immunological pathway is TH22 immunity with neutrophils, IL-22/IL-17 CD4 T cells, and IgG2 B cells. Neutrophils are the major effector cells of TH22 host immunological pathway. TH22 immunity is the host immune response against extracellular protozoa, bacteria, and fungi. It is worth noting that extracellular location decides the host immunological pathway which is more important than whether the pathogen is bacteria, fungi, or protozoa. Neutrophils can use neutrophil extracellular trap and kill these extracellular free-living pathogens. These extracellular free-living protozoa include trypanosoma, ameba, giardia, and trichomonas[7-11]. These pathogens can all induce TH17/TH22 host immunity. Extracellular bacteria such as E. coli. and extracellular fungi such as aspergillus can also trigger TH17/TH22 host immune reaction.

Helminths (endoparasites) and TH2a immunity

For helminths (endoparasites), the host immunological pathway is TH2a immunity with eosinophils, IL-5/IL-4 CD4 T cells, and IgG4 B cells. Eosinophils are the major effector cells and they use IgG4 mediated ADCC to attack helminths teguments. This TH2a pathway belongs to TH2 immunity and is a subtype. The letter “a” means “acid” which is derived from the name of eosinophils. Helminths (endoparasites) which can induce TH2a immunity with eosinophilia include ascaris, hook worm, tape worm, pin worm, toxocara, and strongylosis[12-17]. However, several helminths can also induce IgE antibody, so this immune response is only a subtype of traditional TH2 immunity.

Insects (ectoparasites) and TH2b immunity

For insects (ectoparasites), the host immunological pathway is TH2b immunity with basophils, mast cells, IL-3/IL-4 CD4 T cells, and IgE B cells. The major effector cells of TH2b immunity are basophils and mast cells. Circulating basophils and resident mast cells have the same characteristics. Resident mast cells can also serve as antigen presenting cells. The letter “b” means “base” which is derived from the name of basophils. IgE can cause physical expel of insects (ectoparasites) via skin itchiness, skin wheal with toxin dilution, rhinorrhea, mucus formation and secretion, nausea/ vomiting, bronchoconstriction, and increased bowel movement. Basophil accumulation is usually noted in the site of insect bite or dwelling. However, these IgE mediated mechanism can also expel helminths in lung or intestine. Thus, this immune response (TH2b) is only a subtype of TH2 immunity. Insects or insect bite including ticks, flea, bee, wasp, and mosquitos can induce this TH2b immune reaction[18-22].

Conclusion

Thus, the framework of the whole immunological pathways against four types of parasitic infection is given. Intracellular protozoa induce TH1 immunity, and extracellular protozoa induce TH22 immunity. Helminths(endoparasites) induce TH2a immunity, and insects(ectoparasites) induce TH2b immunity.

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