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Editorial

The Study of Host-Specific Interactions

Trevor Hancock*

Department of Microbiology, Carleton University, Ottawa, Canada

EDITORIAL NOTE

Parasitism is a kind of symbiotic relationship between two organisms: a parasite, mostly the smaller of the two, and a host, upon which the parasite is physiologically dependent. The host in a host-parasite interaction is the animal that maintains the parasite. There are two significant types of parasites namely endoparasites, and ectoparasites. Endoparasites live within the body of the host at sites, such as urinary bladder, alimentary tract, liver, and lungs, whereas ectoparasites are attached to the outer surface of the host or are superficially embedded in the body surface.

The host may be classified as: A definitive host: if the parasite attains sexual maturity there in; An intermediate host: if it serves as a temporary, but essential, environment for the development of the parasite and its transformation short of sexual maturity; A transfer or paratenic host: if it is not necessary for the completion of the parasite's life cycle but is used as a temporary refuge and a vehicle for reaching an obligatory, usually the definitive, host in the cycle. The host and parasite are in dynamic interaction, the result of which depends on the properties of the parasite and of the host. The parasite has its determinants of virulence that allow it to invade and destroy the host and to resist the defenses of the host. The host has different degrees of resistance to the parasite in the form of the host defenses.

A healthy animal can defend itself against pathogens at various stages in the infectious disease process. The host defenses may be of such an extent that infection may be prevented completely. Or on the other hand, if an infection occurs, the defenses may stop the process before the disease is apparent. At different occasions, the defenses that are necessary to defend a pathogen may not be effective until an infectious disease is well into progress. Immune defenses against pathogenic organisms are customized to meet the wide scope of their extracellular and intracellular life cycles within the host environment. A defense against bacterial agents primarily utilizes antibodies, antibodies and complement, and direct cytotoxic mechanisms to control infection. Defenses against mycobacteria requires T-cell–mediated DTH responses that results in granuloma formation. Antifungal defenses also use similar mechanisms to control organisms. Defenses against viral agents require antibody neutralization upon initial infection, and cytotoxic mechanisms controlled by NK cells and CTLs while expanding within cellular compartments. Defenses against protozoal agent incorporate DTH and antibody to limit growth. Defenses against helminths and larger multicellular organisms utilize atopic and ADCC-dependent reactions, as well as granulomatous responses, to sequester and destroyed stored eggs. Organisms have evolved multiple mechanisms to evade host responses, ranging from antigenic modulation of surface proteins to coordinate immunosuppressive activity on specific cellular subsets.

Parasites are able to produce as they possess certain structural or biochemical or genetic traits that render them pathogenic or virulent. The sum of the characteristics that allow a given bacterium to produce disease are the pathogen's determinants of virulence. Some pathogens may rely on a single determinant of virulence, such as toxin production while others maintain an enormous amount of virulence determinants and thus are able to produce a more complete range of diseases that affect various tissues in their host. They may change their surface antigens (antigenic modulation), hide within cells, and produce factors that hinder the immune response (immunosuppression) or fool the immune system into responding with an ineffective effector mechanism (immune deviation). Also, bacteria have evolved to evade different aspects of phagocyte-mediated killing. For example, they may secrete toxins to inhibit chemotaxis; contain outer capsules that block attachment; block intracellular fusion with lysosomal compartments; escape from the phagosome to multiply in the cytoplasm. Viral entities also subvert immune responses, usually through the presence of virally encoded proteins.

THE RESULT OF INTERACTION

In case the host defenses are of an effective degree, it can overcome the parasite and thus the infection can be prevented entirely. On the other hand, if an infection does occur, the defenses may stop the process before the disease is apparent. At different occasions, the defenses that are necessary to defeat a pathogen may not be effective until an infectious disease is well into progress. However, the ultimate endpoint of evolution of the human host and its infectious organisms results in an

Correspondence to: Trevor Hancock, Department of Microbiology, Carleton University, Ottawa, Canada, Tel: +1 691-554-2400; E-mail: thancock@oic.ca

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eventual mutual coexistence with most environmental organisms. In many human infections, the infectious agent is never fully destroyed and the disease enters an inactive state, possibly to be reactivated when immune surveillance wanes. In

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