Host Immunity

A disease results from the response of the host to infection with the parasite and can occur in either vertebrate or arthropod hosts. Immunity includes all properties of the host that confer resistance to infection and plays an important role in determining host suitability and the extent of disease or illness.

Some species or individuals within a population have natural (or innate) immunity and are refractory to infection. Natural immunity does not require that the host have previous contact with the parasite but may be age dependent. For example, humans do not become infected with avian malaria parasites such as *Plasmodium relictum*, even though infective *Culex* mosquito vectors feed frequently on humans. Conversely, mosquitoes do not become infected with measles or polio viruses that infect humans, even though these viruses undoubtedly are ingested by mosquitoes blood feeding on viremic human hosts.

Individuals may acquire immunity after becoming infected with parasites. This acquired immunity to the parasite ranges from transient to life-long and may provide partial to complete protection against future infections with the same or related parasites. Partial immunity may allow some parasite development or reproduction within the host and may reduce the severity of disease, whereas complete protection results in clearance of the initial infection and usually prevents immediate reinfection.

Acquired immunity may be humoral and result in the rapid formation of antibodies or may be cell-mediated and result in the activation of T cells and macrophages. Antibodies consist of five classes of proteins called immunoglobulins that have specific functions in host immunity. Immunoglobulin G (IgG) is most abundant, comprising more than 75% of the immunoglobulins present in the sera of normal individuals.

The IgGs are relatively small proteins, typically reaching high concentration several weeks after infection, and may persist at detectable and protective levels for years. Therefore, parasites such as many arboviruses that induce long-lasting immunity are good candidates for vaccine development. In contrast, immunoglobulin M (IgM) is a large macroglobulin that appears shortly after infection but decays rapidly relative to IgG.

For the laboratory diagnosis of many diseases, serum samples typically are tested during periods of acute illness and again during convalescence 2 to 4 weeks later. A 4-fold increase in parasite-specific IgG antibody concentration in these paired sera provides diagnostic evidence of infection. The presence of elevated concentrations of IgM presumptively implies current or recent infection. T cells and macrophages are cells that are responsible for the recognition and elimination of parasites.

In long-lived vertebrate hosts, acquired immunity may decline over time, eventually making the host susceptible to reinfection. Clinically, the host response to infection ranges from asymptomatic (i.e., inapparent) to severe. Disease may be acute, occurring during a relatively short interval that begins soon after infection, or chronic, extending over a long period of time after infection.

For most parasites, interactions with the vertebrate host are a delicate balance between the need for adequate virulence to amplify parasite concentrations in the host's blood to increase infectiousness to vectors and the need to avoid excessive virulence that could cause adverse effects on the host and result in illness or death that would reduce the host's infectious period. Generally, the susceptibility of the vector to infection dictates the concentration of parasites in the vertebrate host necessary to complete the transmission cycle.