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Commentary

Ontogeny of Host Defense Mechanisms

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DESCRIPTION

The human immune system is composed of cellular and molecular components designed to prevent infection while avoiding potentially harmful inflammation and autoimmunity. Immunity varies with age and reflects unique age-related challenges such as fetal pregnancy, neonatal period, and infancy. Plasma, the liquid phase of blood, regulates pro-inflammatory danger signals that initiate host defense during infection. In addition to high expression of basal anti-inflammatory cytokines (IL4, IL10, IL13, TGFβ) in cord blood pairs. Adult plasma expresses various immunosuppressive plasma factors at higher levels earlier in human life. These plasma-derived factors, such as proteins, lipids, purines, and sugars, may help maintain fetal and maternal resistance in the womb. It enables postnatal microbial colonization and acts as an anti-inflammatory/pro-degradation function during infection. New-born hosts show quantitative and qualitative differences from adults in almost every aspect of immunity. This at least partially explains the increased susceptibility to infection. Here, we describe how differences in susceptibility to infection do not result from immaturity, but reflect adaptation to the specific requirements of the immune system in early childhood. We will explore the underlying mechanisms of host defense at a very young age and discuss how specific developmental requirements increase the risk of specific infections. The ability to adapt to childhood demands also offers the potential to take advantage of protecting young people from infectious diseases and illnesses through a variety of interventions. The immune system distinguishes between self and non-self and eliminates potentially harmful non-self-molecules and cells from the body. The immune system also has the ability to recognize and destroy abnormal cells from host tissues. Any molecule that the immune system can recognize is considered an antigen. The respiratory, gastrointestinal, and genitourinary skin, cornea, and mucous membranes form the physical barrier, which is the body's first line of defense. Some of these barriers also have active immune function. Innate immunity does not require prior exposure to antigen (i.e., immunological memory) to be fully effective. Therefore, it may react immediately to an intruder. Innate immunity recognizes primarily a wide range of molecular patterns, rather than organism-or cell-specific antigens. Scavenger cells (neutrophils in blood and tissues, monocytes in blood, macrophages in tissues) take up and destroy invading antigens. Phagocyte attack is facilitated when the antigen is coated with an antibody produced as part of adaptive immunity, or when the complement protein opsonizes the antigen.

Acquired immunity requires prior exposure to the antigen to be fully effective and takes time to develop after the first encounter with a new invader. Then there is a quick reaction. The system remembers previous exposures and is antigen-specific. The cells of the immune system are activated when foreign antigens are recognized by cell surface receptors. These cell surface receptors can be broadly specific. Highly specific antibodies expressed on B cells or T cell receptors expressed on T cells. Broadly specific receptors recognize common microbial pathogen associated molecular patterns such as gram-negative lipopolysaccharide; gram-positive peptidoglycans, bacterial flagellin, unmethylated cytosineguanosine dinucleotide and viral double stranded RNA. These receptors can also recognize molecules that are produced by stressed or infected human cells (called damage associated molecular patterns). Activation may also occur when antibody antigen and complement microorganism complexes bind to surface receptors for the crystallisable fragment region of IgG and for C3b and iC3b. Once recognized, an antigen, antigen antibody complex, or complement microorganism complex is internalized. Most microorganisms are killed after they are phagocytosed, but others inhibit the phagocyte`s intracellular killing ability (eg, mycobacteria that have been engulfed by a macrophage inhibit that cell's killing ability). In such cases, T cell-derived cytokines, particularly interferon gamma, stimulate the phagocyte to produce more lytic enzymes and other microbicidal products and thus enhance its ability to kill or sequester the microorganism.

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