

Introduction

The most basic requirement of any immune system is distinguishing the cells, tissues and organs that are a legitimate part of the host body from foreign things, called “non-self,” that might be present. The second job is to eliminate those non-self invaders (*Goldsby et al., 2003*).

Immunity is defined as resistance to disease, specifically infectious disease. The collection of cells, tissues, and molecules is the immune system, and the coordinated reaction of these cells and molecules to infectious microbes is the immune response. The physiological function of the immune system is to prevent infections and to eradicate established infections (*Abbas and Lichtman 2009*).

The mammalian immune system has innate and adaptive components, which cooperate to protect the host against microbial infections. The innate immune system consists of functionally distinct 'modules' that evolved to provide different forms of protection against pathogens. It senses pathogens through pattern-recognition receptors, which trigger the activation of antimicrobial defenses and stimulate the adaptive immune response. The adaptive immune system, in turn, activates innate effector mechanisms in an antigen-specific manner. The connections between the various immune components are not fully understood, but recent progress brings us closer to an integrated view of the immune system and its function in host defense (*Palm et al., 2009*).

The immunopathogenesis of many human diseases is characterized at the molecular level. Therefore, a basic understanding of immune function is often useful (*Pamela, 2010*).

Apoptosis is an active process that leads to the ordered destruction of cells, avoiding the release of intracellular contents into the extracellular microenvironment, where they have a powerful inflammatory effect. Apoptotic cells undergo a series of distinct physical changes, including alteration of the surface lipid membrane, cytoskeletal disruption, cell shrinkage and a characteristic pattern of DNA fragmentation (*Coornaert et al., 2009*).

A properly functioning immune system mounts immune responses to foreign molecules while remaining tolerant to molecules produced by the host. The primary responsibility of the immune system is to protect the host from foreign materials. Immune tolerance is selective in that the immune system disregards molecules native to the host and responds aggressively to remove foreign molecules. Autoimmune diseases are the result of breakdowns in immune tolerance (*Kee and Murre 2001*).

Immune system disorders occur when it does not fight tumors or harmful substances as it should. That response may be underactive in the form of immune deficiency disorders or overactive as autoimmunity and hypersensitivity (*Bonilla, 2008*).

Systemic autoimmune diseases as ; rheumatoid arthritis (RA) and juvenile RA, systemic lupus Erythematosus (SLE), scleroderma Sjögren's, syndrome (SS), Goodpasture's syndrome, Wegener's granulomatosis (WG), polymyalgia Rheumatica (PMR) (*Gorska, 2008*).

Localized autoimmune diseases such as; Type-I diabetes mellitus, Hashimoto's thyroiditis, Graves' disease, celiac disease, Crohn's disease, ulcerative colitis , multiple sclerosis , Addison's disease, primary biliary cirrhosis, sclerosing cholangitis, autoimmune hepatitis and temporal arteritis / giant cell arteritis (*Saunders et al., 2007*).

In some cases, a person may have more than one autoimmune disease; for example, persons with Addison's disease often have type-I diabetes, while persons with sclerosing cholangitis often have ulcerative colitis (*Hyman, 2008*).

The antibodies may not be directed at a specific tissue or organ; for example, antiphospholipid antibodies can react with clotting proteins in the blood, leading to formation of blood clots within the blood vessels (thrombosis) (*Meroni et al., 2009*).

Anti-nuclear antibodies are directed against nuclear antigens. They are one of the hallmarks of an autoimmune disease, they may be considered as an intrinsic to the diagnostic criteria, useful for monitoring or prognosis, not useful in diagnosis or may present in normal persons (*Tozzoli et al., 2002*).

Screening for disease-specific autoantibodies may be useful in asymptomatic ANA-positive individuals as a means of evaluating the risk of developing a systemic autoimmune disease such as systemic lupus erythematosus, polymyositis/dermatomyositis, scleroderma, Sjögren's syndrome , rheumatoid arthritis , or primary biliary cirrhosis in the future. New technology enabling screening for multiple autoantibodies may further enhance the clinical usefulness of autoantibody testing, making it

possible to diagnose autoimmune disease in its earliest stages and to intervene before serious end organ damage occurs (*Lyons et al., 2005*).