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Rheumatoid Arthritis in Animals

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Editorial Note

The immune system has been considered as a set of structures (molecules, cells, and specialized tissues) and biological processes responsible for the defense against aggression by a variety of infectious agents, chemicals, and tumor cells. One of the fundamental characteristics of the immune system is its ability to discriminate foreign antigens. Autoimmunity is a multifactorial condition in which the host organizes an immune response against its own antigens. Autoimmunity is associated with genetic, immunological, hormonal, and environmental factors, with it being classified as organ-specific and systemic, of which RA is one of the most representative.

RA has an incidence of 5 per 100,000 adults and occurs in 0.5-1% of the population in industrialized countries. The RA can manifest as pain, stiffness, swelling, and loss of mobility. There are different strategies for the study of RA, including experimental animal models that help elucidate different aspects of the disease, as well as evaluate compounds that can reduce the inflammation that triggers the disease. An ideal animal model for RA should reproduce as close as possible the complex pathogenesis and symptoms that underlie the disease, including the presence of chronic inflammatory infiltrates, the development of destructive arthropathy, bone erosion, and degradation of the articular cartilage and subchondral bone. Current RA animal models are highly reproducible and of short duration, having similar patterns to those occurring in human disease although they present some differences, such as: faster progression of the disease, characterized by an acute inflammatory response and rodents have a tendency to marked resorption and bone formation (especially of the periosteum/endosteum) in response to joint inflammation. The use of animal models has contributed significantly to the knowledge of processes and mediators that generate inflammation and bone and cartilage damage and, in this sense, can be used as an intermediary to provide knowledge and for the evaluation of therapeutic molecules to correct these disorders. There are numerous therapeutic alternatives for RA; however, the duration of these therapies and the side effects associated with some of these drugs mean that until now, there is no effective therapy for this disease.

The immune system is responsible for protecting the human body from external and potentially pathogenic organisms. It is made up of a series of cells, tissues, and organs distributed widely throughout the body. From the point of view of its structural characteristics, there are organs such as the thymus, spleen, and lymph nodes and tubular structures such as the lymphatic vessels that are intercommunicated. If the functions performed are taken into account, they can be classified into primary and secondary lymphoid organs. The primary lymphoid organs (thymus and bone marrow) produce T and B lymphocytes, while secondary lymphoid organs include lymph nodes, spleen, peyer's patches, and mucosal tissues, nasal-associated lymphoid tissue, adenoids, and tonsils, which also harbor perifollicular areas.

The immune system has two lines of defense, specific and nonspecific (adaptive and innate), which are responsible for keeping the body free from pathogens or, if present, can eliminate them as well as their residue. The innate, nonspecific antigen response destroys microorganisms and triggers an inflammatory process that blocks the spread of infection. If microorganisms get past this first barrier, antigen-specific adaptive immunity composed of T and B lymphocytes can produce antibodies and killer cells that destroy infected cells. The innate immunity is constituted of external barriers such as mucous and skin; the inflammatory process; cells such as macrophages, natural killer (NK) cells, and phagocytic cells; and chemicals. In relation to adaptive immunity, this is generated only after exposure of inducing agents, and two distinct responses are generated, the cellular response, in which T lymphocytes are responsible for generating this reaction and the humoral response, carried out by B lymphocytes, which in turn are responsible for producing antibodies against the agents that cause damage.

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