

Desmoglein 3 Vaccination causes Mice to Produce Non-Pathogenic Autoantibodies

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Abstract

A type I trans membrane protein known as the polymeric immunoglobulin receptor (pIgR) is primarily made up of an intracellular area, a trans membrane region, and an extracellular region. Additionally, the repeating immunoglobulin-like (Ig-like) domains in the extracellular domain of pIgR increase in number with vertebrate evolution, from four in birds, six in mammals, and eight in humans. The Ig-like domains consist of a variable region (V), a transmembrane region (T), and a constant region (C). The C region contains the binding sites for IgG molecules. The V region is involved in the recognition of specific epitopes on antigens. The T region is involved in the transport of the protein across the membrane.

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Desmoglein 3 (DSG3) is a transmembrane protein that is expressed on the apical surface of epithelial cells. It is involved in the formation of tight junctions and plays a role in the regulation of ion transport across the apical membrane. DSG3 has been implicated in various diseases, including pemphigus vulgaris, a autoimmune disorder characterized by the formation of blisters on the skin and mucous membranes. In this study, we investigated the immune response to DSG3 in mice. We found that mice immunized with DSG3 produced non-pathogenic autoantibodies against DSG3. These antibodies did not bind to normal DSG3-expressing cells, but did bind to cells expressing mutated DSG3. This suggests that the antibodies recognize conformational epitopes on the protein. Our results provide new insights into the mechanisms of autoimmunity and the potential therapeutic applications of DSG3 vaccination.