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Opinion Article

Genetic Variations of Alloantigen through Various Proteins and Enzymes

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DESCRIPTION

Alloantigen is genetic variations of the same antigen. Alloantigen includes various proteins and enzymes, as well as blood group substances on erythrocytes and histocompatibility antigens in grafted tissues that trigger an alloimmune response in the recipient who does not have them. Self-non-self-discrimination, or immune system recognition of genetically encoded polymorphisms among genetically distinct members of the same species, is referred to as alloantigen recognition. Alloantigen is recognized by secondary lymphoid organs after transplant.

Glycoproteins like Major Histocompatibility Complex (MHC) and MNS antigens make up the majority of alloantigen. Other types of alloantigen include sialoglycoproteins like CD43, oligosaccharides like ABO (H), Secretor, Lewis, Li, and P, sialo-oligosaccharides like Sialyl-Lewisx or Sialyl-Lewisa, and proteins like Rh. Although the majority of cell surface protein antigens are anchored by trans membrane hydrophobic interactions, an increasing number of structurally diverse cell surface antigens are anchored by a covalent linkage *via* GPI: Ly-6, Qa-2, RT6, Thy-1 (CDw90), CD14, CD55, CD59, and CD73 There are two types of some antigens, including CD58 (LFA-3) and probably CD55: One has a trans membrane anchor, and the other has a GPI anchor. Trans membrane signaling is facilitated by the GPI-anchored proteins; For instance, T cell activation *via* this molecule is dependent on the GPI anchor of Qa-2. In addition, the immunoglobulin supergene family includes a significant number of alloantigen: For instance, CD1, CD2, CD3, CD4, CD7, CD8, Ly-6, Qa-2, RT6, CD19, CD22, CD56, and CD58 (LFA-3), CD59, and CDw90's membrane-proximal domains.

Factors that are not alloantigen-independent can be categorized as donor- or recipient-related. The former may include the age of the donor, the nephron dose in relation to the metabolic demand of the recipient, and donor premorbid conditions like diabetes, hypertension, or dysfunction in the donor kidney. Age, ethnicity, the cause of native kidney disease, hypertension, diabetes, dyslipidaemia, Calcineurin Inhibitors (CNI) nephrotoxicity, the age of end-stage kidney disease, proteinuria, and concurrent infections are some of the recipient factors. BK nephropathy has emerged as a significant cause of allograft dysfunction and graft loss over the past ten years. The following section discusses a few alloantigen-independent factors.

Antibodies from members of the same species can identify alloantigen, which are phenotypically definable differences in a single protein that are frequently the result of inherited polymorphisms in amino acid residues. Plasma cells secrete alloantibodies, also known as alloantibodies. After alloantigen-driven B-cell activation in the presence of T-cell support, such as during rejection or a blood transfusion, alloantibodies are produced. Infections that result in heterologous immunity can also produce antibodies that cross-react with alloantigen. In the first case, the B cells themselves may serve as Antigen Presenting Cells (APCs) in addition to Dendritic cells (DCs). B cells are able to internalize the alloantigen and process it into peptides that are presented at the cell surface within MHC class II molecules because MHC class II molecules are presented to and bind Immunoglobulins (Igs) on the surface of B cells.

The primary step in the chain of all reactive T-cell activation and proliferation that results in rejection of the transplanted tissue or organ is alloantigen recognition. Two models have been proposed for how T cells mediate graft injury, though the exact mechanism is unknown: 1) Cell-mediated cytotoxicity of parenchymal (tubular and endothelial) cells, and 2) the effects of local cytokine release, which are similar to a delayed hypersensitivity reaction. Cytokines can affect parenchymal cells directly or indirectly by affecting the endothelium and blood supply.

Alloimmune response induced by foreign histocompatibility alloantigen is a complex phenomenon with innate and adoptive immune response-like mechanisms and characteristics. Numerous exocrine and autocrine immune regulating factors also alter it. In the beginning of the new era of functional genomics, understanding the structure of human genes necessitates a clearer understanding of not only the function and contribution of genes but also their historical background, origin, and significance in phylogenies. In order to comprehend the complexity of the immune and alloimmune responses, comparative immunology comes into focus. The fact that immune functions like phagocytosis and the production of cytokines like IL-1 and TNF first appeared in sponges and

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starfish 700 million years ago is almost unbelievable. During phylogenesis, functions such as the recruitment of coelomocytes, killing of microorganisms by lysosome-like enzyme activity, opsonization by complement-like proteins, and oxidative burst function remained unchanged. These functions can be found in representatives of innate immunity as well as in mammals.

The immune response to H-4 histocompatibility alloantigen is genetically controlled, and this is explained. An H-2-linked Immune Response (IR) gene controls the rejection of H-4.2-incompatible skin grafts. At the IrH-4.2 locus, a dominant allele determines rapid responsiveness. The fast response allele is shared by the H-2b, H-2d, and H-2s haplotypes; the slow response allele is found in H-2a. The IrH-4.2 locus is mapped to the I-B sub region of the H-2 complex using intra-H-2 recombinants; Fast responder haplotypes include the H-2h4, H-2i5, and H-2t4 haplotypes. The antigen-specific responsiveness of the recipient is ultimately what determines the strength of non-H-2 histocompatibility antigens.