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Perspective

Human Leukocyte Antigen and Immune System Control in Posttraumatic Stress Disorder

Saleem Shah*

Department of Biochemistry, Government Medical College, Srinagar, India

DESCRIPTION

Posttraumatic Stress Disorder (PTSD) is a severe condition that harms both mental and physical health. Major Histocompatibility Complexes (MHC) contain genes called Human Leukocyte Antigens (HLA) that help code for proteins that differentiate between self and non-self. They are significant for immunity and illness prevention. The genes on chromosome 6 regulate the Human Leukocyte Antigen (HLA) system, also known as the Major Histocompatibility Complex (MHC) in humans, which is a crucial component of the immune system. It specifies cell surface molecules with the ability to deliver antigenic peptides to T cells' T-cell Receptors (TCR). While they may be harmful, they are useful for the immune system.

Class I MHC molecules that are all nucleated cells have transmembrane glycoproteins are Class I MHC molecules. An alpha heavy chain is joined to a beta-2 macroglobulin molecule to form intact class I molecules. Two peptide-binding domains, an immunoglobulin (Ig)-like domain and a transmembrane region with a cytoplasmic tail makes up the heavy chain. Genes in the HLA-A, HLA-B, and HLA-C loci encode the heavy chain of the class I molecule. Class I MHC molecules are responded with by T lymphocytes that express CD8 molecules. These cells frequently have a cytotoxic function, thus they must be able to identify any infected cell.

Class II MHC molecules are usually only seen on professional antigen-presenting cells (B cells, macrophages, dendritic cells, and Langerhans cells), the thymic epithelium, and activated T cells. Interferon (IFN)-gamma can promote the expression of class II MHC molecules in the majority of nucleated cells. Two polypeptide (alpha and beta chains make up Class II MHC molecules, and each chain has a peptide-binding domain, an Ig-like domain, and a trans membrane region with a cytoplasmic tail. MHC class III region of the genome encodes several molecules important in inflammation; they include complement components C2, C4, and factor B; Tumor Necrosis Factor (TNF)-alpha; lymph toxin; and three heat shock proteins.

According to previous research, immune system modifications that may result in an increase in inflammatory factors including IFN-, IL-6, TNF-, and IL-17 and a decrease in anti-inflammatory factors are related to PTSD. Posttraumatic Stress Disorder (PTSD) is a debilitating psychiatric disorder characterized by re-experiencing of trauma, avoidance of trauma reminders, and hyper arousal symptoms that cause negative alterations in cognition, mood, and physiologic health. Compared to other psychiatric diseases, PTSD is distinct in that it requires exposure to trauma to develop. Although more than 70% of people experience at least one stressful event in their lifetime, it is unclear why only some people proceed to acquire PTSD.

The peripheral immune system is activated as a result of these neuro hormonal processes as a natural defense mechanism. Healthy people experience a slow resolution of this initial inflammatory phase. But in some people with immune system dysregulation, the inflammatory response continues. New research has validated the possibility of an immune-related or inflammatory genesis for PTSD and suggested that inflammation may be a preexisting vulnerability factor for the onset of PTSD. The identified changes in PTSD-related inflammatory gene expression are compatible with changes in epigenetic profiles since changes in DNA methylation can control gene expression and are seen in PTSD. According to previous studies, PTSD is related to changes in the immune system, which may increase the level of inflammatory factors such as IFN-γ, IL-6, TNF-α, and IL-17 and a reduction in the level of anti-inflammatory factors.

Correspondence to: Saleem Shah, Department of Biochemistry, Government Medical College, Srinagar, India, E-mail: shah.saleem.lu.ck@email.com

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