Synthesis of immune dysregulation: Emerging therapies mechanism and its clinical application.

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Abstract

Increasing evidence suggests that inflammation and immune dysregulation play an important role in the pathogenesis of bipolar disorder. Because the brain can be affected by various autoimmune processes, it is possible that some psychiatric disorders may have an autoimmune basis. The link between immune dysregulation, autoimmunity, and bipolar disorder may be closer than previously thought. Psychiatrists should be vigilant for autoimmunity in presentations of bipolar disorder due to its high morbidity and therapeutic implications. Advances in neuroimaging and biomarker identification related to immune dysregulation and neuro inflammation will contribute to our knowledge of the pathophysiology of bipolar disorder.

Keywords: Anti-NMDA encephalitis, Autoimmunity, Bipolar disorder, Cytokines, Hashimoto's encephalopathy, Immune dysregulation.

Introduction

Immune dysregulation is any proposed or confirmed breakdown or maladaptive change in molecular control of immune system processes. For example, dysregulation is a component in the pathogenesis of autoimmune diseases and some cancers [1]. Immune system dysfunction, as seen in IPEX syndrome leads to immune dysfunction, poly endocrinopathy, enteropathy, X-linked (IPEX). IPEX typically presents during the first few months of life with diabetes mellitus, intractable diarrhea, failure to thrive, eczema, and haemolytic anaemia. Unrestrained or unregulated immune response.

IPEX (Immune dysregulation, poly endocrinopathy, enteropathy, X-linked syndrome) is a syndrome caused by a genetic mutation in the FOXP3 gene, which encodes a major transcription factor of regulatory T cells (Tregs). Such a mutation leads to dysfunctional Tregs and, as a result, autoimmune diseases. The classic clinical manifestations are enteropathy, type I diabetes mellitus and eczema [2]. Various other autoimmune diseases or hypersensitivity are common in other individuals with IPEX syndrome. In addition to autoimmune diseases, individuals experience higher immune reactivity (e.g. chronic dermatitis) and susceptibility to infections. Individuals also develop autoimmune diseases at a young age.

Dysregulation of the immune system is also associated with Immuno senescence, which arises due to aging. Immuno senescence is manifested by a decrease in reactivity to vaccination or infection, an impaired ability of T and B lymphocytes to activate and proliferate, or a lower ability of antigen presentation by dendritic cells. In Immuno senescence, memory and effector T cells accumulate at the expense of naïve T cells [3]. The lack of naïve T lymphocytes is the cause of low plasticity of the immune system in the elderly. In aging of the immune system are also a decrease in central tolerance and an increase in the number of auto reactive T cells. B cells also have a decreased repertoire of naïve cells and an increase in memory B cells. They also have reduced the production of antibodies against antigens [4]. In Immuno senescence, here is a change in the individual subtypes of Immuno globulins. IgM and IgD levels decrease while IgG1, IgG2, and IgG3 levels increase. IgA is higher in the form of monomers in serum but lower as a dimer on the mucosal surface. The overall accumulation of both effector T and B cells is due to the presence of chronic inflammation due to long-term exposure to antigens. In Immuno senescence is also a reduced ability to apoptosis, which promotes the survival of memory cells. In old age, innate immunity cells are also affected, when activated cells have a lower ability to return to a quiescent state, only effector functions decrease [5]. Elderly people show poor NK cell reactivity and impaired ability of antigen presentation by dendritic cells. In macrophages, the ability of phagocytosis is reduced and the M2 phenotype of macrophages (alternatively activated) is promoted. Immuno senescence also results in increased production of some immune mediators, such as proinflammatory IL-6 or IL-1. There may also be higher production of anti-inflammatory IL-10 or IL-4. In old age, the ability to heal wounds also decreases, leading to a susceptibility to further infections at the site of injury. The aging of the immune system is also supported by chronic infections, oxidative stress, or the production and accumulation of reactive oxygen species (ROS). The increase in the proportion of memory cells is also affected by cytomegalovirus infection. A chronic pro-

inflammatory condition in an aging organism is also referred to as inflammation. It is a long-term, low-grade systemic inflammation present without the presence of infection.

Allergic reactions are misdirected reactions of the immune system to substances commonly found in the environment. Allergens elicit a Th2 immune response, including the involvement of IgE, mast cells, Innate lymphoid cells 2 (ILC2), eosinophil, and basophils. Allergy symptoms are often related to the body's efforts to expel the allergen from the body and to protect it from further exposure to the allergen. Allergic reactions increase the production of mucus by goblet cells on the mucosa. The production of mucus is promoted by IL-13 from ILC2 and Th2 cells. Higher mucus production then creates stronger barrier protection and supports runny nose, coughing, or sneezing. Removal of the allergen from the body by sneezing, coughing, vomiting, or diarrhea is enabled by the activation of peristalsis and contractions of the smooth muscles of the digestive and respiratory systems. Activation of smooth muscles occurs after the action of histamine, which is released by mast cells. Manifestations of allergies generally aim to eliminate the body's allergen [6]. This is also related to hearing the flushing of antigens in the eyes or to attempts to achieve mechanical removal of the surface of the organism.

Allergies can be caused by genetic and environmental factors. Some theories support the view that allergies enter as protection against environmental substances that can disrupt the body, such as insect venom. Another possibility of activating an allergic reaction is the similarity of some allergens to the molecular patterns of parasites against which the immune system also uses a type 2 immune response. The hygiene hypothesis then relates to changes in lifetime exposure to pathogens in developed countries. In the case of insufficient exposure to pathogens and insufficient stimulation of the Th1 response during an individual's development, the balance between Th1 and Th2 type responses may predominate to pro allergic Th2. The theory is supported by the more frequent occurrence of allergies in developed countries compared to developing countries, but also by the higher incidence of allergies in cities compared to villages, where individuals can meet with pathogens of farm animals. Children from small families are also more likely to have allergies than children from families with more children, where there is more frequent contact with pathogens from siblings. Another environmental factor that may promote the predisposition to allergies is a reduction in the diversity of the micro biome - this affects the diet of individuals, but also the diet of the mother during pregnancy, method of delivery, breastfeeding, antibiotics, and the presence of domestic or farm animals in the normal life of individuals.

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