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Research Article

Cure of Immunotoxicity and Significance of Immunotoxicity

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Abstract

The immune system defends the body against certain tumour cells and against foregin agents such as fungi, viruses, bacteria and many parasites. One of its main role is to distinguish endogenous component from non self component.

Keywords: Immunotoxicity; Microbiota; Hypersensitivity; Allergies; Immune System; Immunosuppressive; Stressors; Cigarette; Chemicals

Introduction

	T-Helper 1 Cell	T-Helper 2 Cell	T-Helper 17 Cell	T-Regulatory
Secretes	IFN-γ, IL-2	IL-4, IL-5, IL-6, IL-10, IL-13	IL-17, IL-21, IL-22	TGF-β, IL-10, IL-35
Function	Activates macrophages and cytotoxic T cells to kill phagocytosed microbes	Activates eosinophils and promotes production of IgE for parasite defense	Immunity against extracellular microbes, through induction of neutrophilic inflammation	Prevents autoimmunity by maintaining tolerance to self-antigens
Induced by	IFN-γ, IL-12	IL-2, IL-4	TGF-β,IL-1, IL-6	TGF-β, IL-2
Inhibited by	IL-4, IL-10 (from T-helper 2 cell)	IFN-γ (from T-helper 1 cell)	IFN-γ, IL-4	IL-6

Table 1: Function of T helper cells [1].

IFN-γ: interferon-gamma, IL: interleukin, Ig: immunoglobulin, TGF-β: transformation growth factor-beta.

This table describe about the function of t helper cells and how they prevent autoimmunity and Hypersensitivity.

Autoimmune diseases

Autoimmunity is seen patient which are treated by the cytokines [2]. This shows the leading role of cytokines in immunopathology not the single cytokine is responsible for the autoantigen development their are pleotropic cytokines. Autoantigens on different cells are targeted by different lymphocytes. Many autoimmune diseases are driven by TH1 except the ulcerative cloitis [3] and systemic lump erythematous [4].

Stressors vs immunotoxicity

Differentiating between stress and immunotoxicity [5].

Figure 1 shows the difference between the immunotoxicity and the stress.

Molecular immunotoxilogy of environment stressors

Physical stressors

Basically comes uv rays that is uv b and uv c which cause up regulation of b and t cells and process of suppression of immunity starts

	Peripheral Blood	Serum Chemistry	Supportive Data
Stress leukogram: corticosteroid mediated	↓Lymphocytes, eosinophils ↑Neutrophils, monocytes Neutrophils are mature, may be hypersegmented	Hyperglycemia Lymphoid depletion in thymus Adrenal cortical hypertrophy	Overt organ toxicity Weight loss, anorexia Deaths in other animals in dose group. Findings only seen at doses at or higher than the maximum tolerated dose
Immunotoxicity: inflammation and infection	↑Neutrophils, monocytes ↓Lymphocytes (nonrodents) Immature neutrophils, toxic change	Mild nonregenerative anemia Inflammation in tissues Organisms Hypercellular bone marrow, increased M/E ratio	Clinical signs of infection, fever, anorexia, weight loss

Figure 1

from skin chromophores and leading to supression of memory b cells and activation of t cells and leading to the immunotoxicity.

Chemical stressors

Arsenic alters B and T lymphocyte functions as well as macrophage function, affecting both innate and humoral immunity [6]. Exposure to this metal induces oxidative stress, inflammation, and apoptosis, which render the host immunocompromised and susceptible to infections, cancers, and lung diseases. The skin is a primary target organ for chronic arsenic toxicity. In fact, its effects range from lesions known as arsenical keratosis to squamous cell carcinoma in situ of the skin known as Bowen disease. Chronic exposure can also cause a spectrum of liver pathologies such as hepatocellular carcinoma, angiosarcoma, cirrhosis, and hepatoportal sclerosis [7].

Beryllium (Be) and nickel (Ni) are the perfect examples of metals that cause hypersensitivities. Individuals that are exposed occupationally or non-occupationally to beryllium dust or fumes are at high risk of developing a non-caseating granulomatous inflammation that leads to chronic beryllium disease, which principally affects the lungs, lymphatics, and skin. This resulting disease, which is caused by CD4+ T cells, occurs more in genetically susceptible persons whose adaptive immune responses, are mainly mediated through single nucleotide polymorphisms in HLA-DP and, to a lesser extent, HLA-DR. On the other hand, high levels of nickel can inhibit the development of immune organs by extensively inducing apoptosis and inflammation [10]. These mechanisms are activated through toll like TL4-mediated nuclear factor-κB (NF-κB) and signal transduction cascades mitogen-activated protein kinase (MAPK) pathways [8]. And many other types like pscychological stressors like any kind of mental stress or depression can lead to mediate t cells and NK cells leafing to immunotoxicity [9].

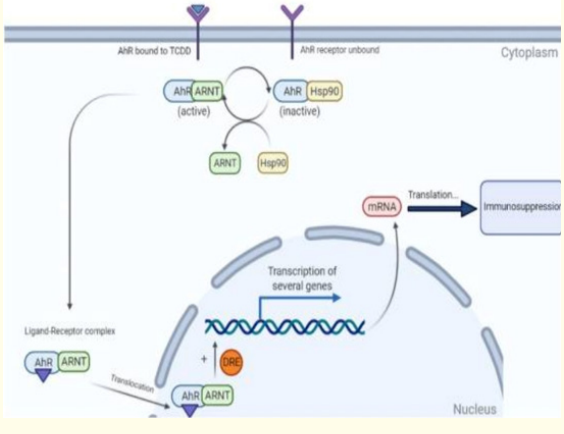

Figure 2: Shows the aryl hydrocarbon receptor function [10].

Treatment of immunotoxicity

- Genetic related issues can be corrected by giving immunosuppressive genes during antinatal period
- Acquired mutated issues can be corrected by giving the immunosuppressive monoclonal antibodies.
- Doses of monoclonal antibody should be maintained between 2 times per day to 1 times per day

Discussion

- Autoimmune diseases
- Immunotoxicity
- Stressors

Conclusion

Mechanism of immunotoxicity and its treatment found.

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