



2015

Autoimmunity and Homoeopathy

A Miasmatic approach

HCRI Publications

© DR. RAJNEESH KUMAR SHARMA MD (HOMOEOPATHY)

AUTOIMMUNITY AND HOMOEOPATHY

© Dr. Rajneesh Kumar Sharma MD (Homoeopathy)

Dr. Swati Vishnoi BHMS

Homoeo Cure Research Institute

NH 74- Moradabad Road

Kashipur (UTTARANCHAL) - INDIA

Ph- 09897618594

E. mail- dr Rajneeshhom@hotmail.com

www.homeopathictreatment.org.in, www.homeopathyworldcommunity.com

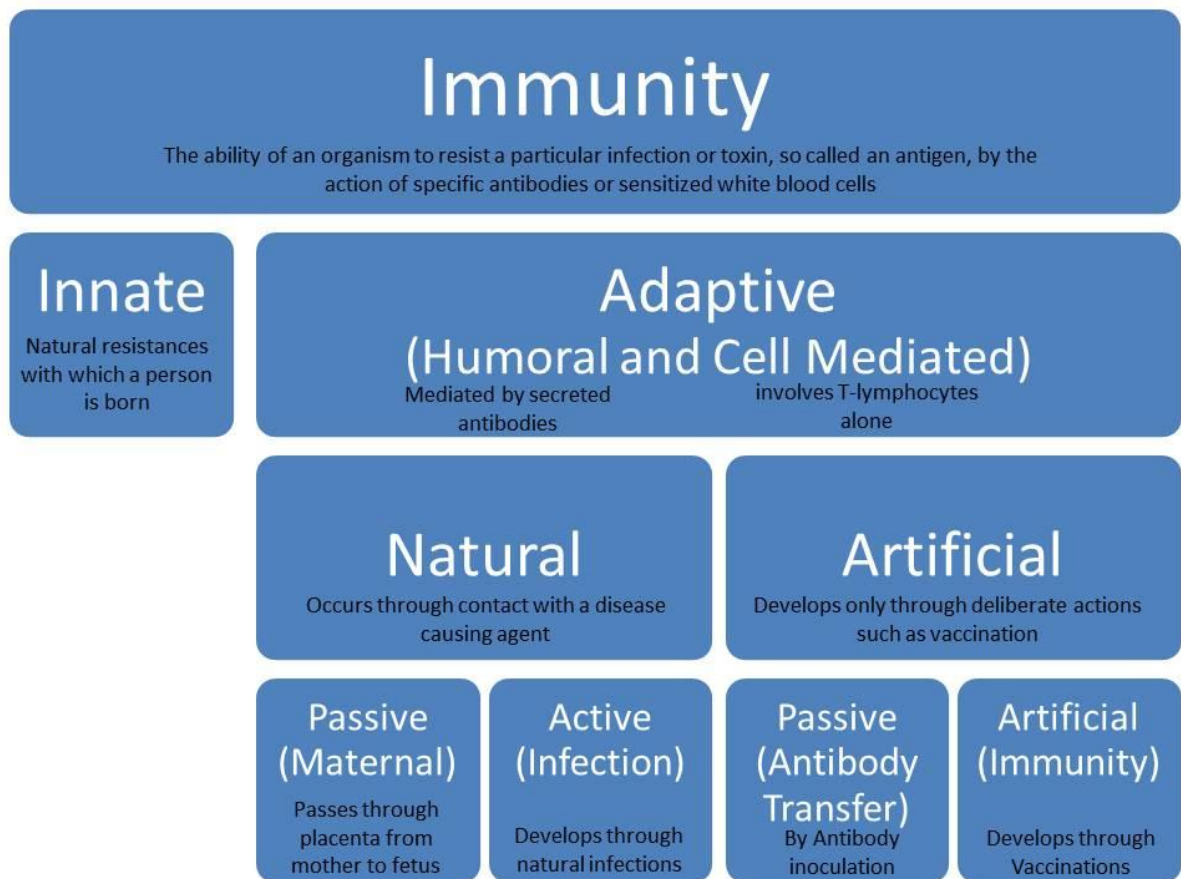


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INTRODUCTION TO IMMUNITY

The ability of an organism to resist a particular infection or toxin, so called an antigen, by the action of specific antibodies or sensitized white blood cells is called immunity. It is the balanced state of having sufficient defense mechanisms to fight infection, disease, or other unwanted invasion, called antigen, while having adequate tolerance to avoid allergy, and autoimmune diseases.



Immunity and its types

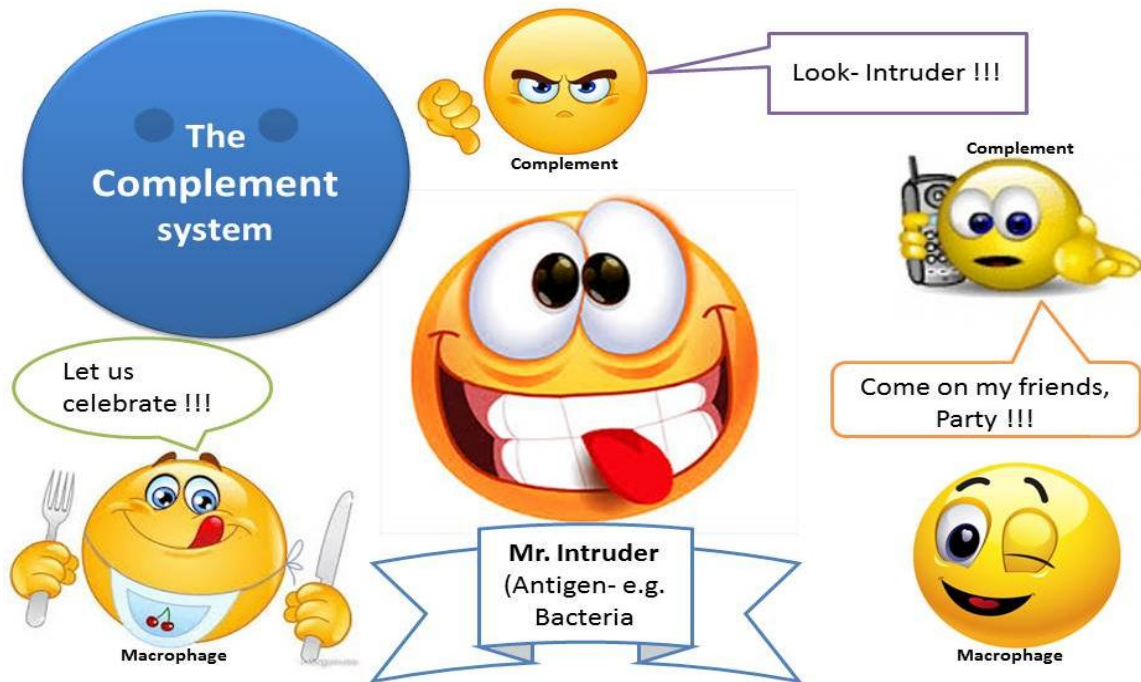
The reactivity of the immune system with itself is called autoimmunity (Psora++/ Syphilis+++/ Sycosis+). It is a composite process characterized and mediated by autoantibodies and auto-reactive cells. There are certain key points to remember in study of autoimmune diseases. The immunity system consists of the following main ingredients-

THE COMPLEMENT SYSTEM

The first part of the immune system that meets antigens is a group of proteins called the complement system. These proteins freely flow in the blood and are able to reach the site of an invasion quickly, where they react directly with antigens. This reaction is called activation of complement system with following functions-

- Inflammation (Psora/ Sycosis)

- Collection of macrophages (Psora)
- Coating of intruders so that macrophages are more likely to engulf them (Sycosis)
- Killing of invaders (Psora)



The complement system- complements recognizing the intruder, informing macrophages and inducing phagocytosis

PHAGOCYTES

This is a group of immune cells specialized in finding and engulfing antigens and dead or injured body cells. There are three main types-

GRANULOCYTE

The granulocytes work first during an infection by attacking any invaders in large numbers and continue to eat until they die (Psora/Syphilis). The pus in an infected wound contains mainly of departed granulocytes. Some granulocytes are dedicated in attacking larger parasites such as worms.

MACROPHAGE

The macrophages are big eaters and slower to respond. They are larger, live longer, and have far greater capacities. Macrophages also play an important part in warning the rest of the immune system of invaders. Monocytes that leave the blood stream turn into macrophages.

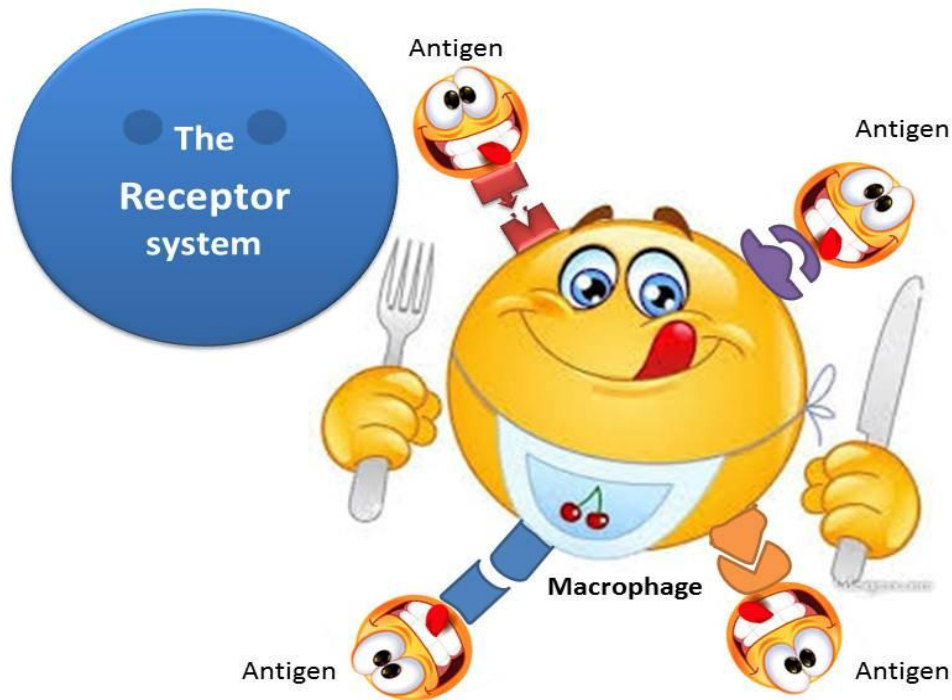
DENDRITIC CELL

The dendritic cells are also eater cells and engulf intruders. Like the macrophages, the dendritic cells also help with the activation of the rest of the immune system. They are capable of filtering body fluids to clear them of foreign organisms and particles.

LYMPHOCYTES - T CELLS AND B CELLS

White blood cells or lymphocytes develop in bone marrow and travel to different parts of the lymphatic system such as the lymph nodes, spleen, and thymus. The lymphatic system feeds cells into the body and filters out dead cells and invading organisms.

On the surface of lymphatic cells, there are receptors enabling them to identify foreign substances or antigens. These receptors are very specialized - each can match only one specific antigen. There are two main types - T cells and B cells.



The receptors match only one specific antigen

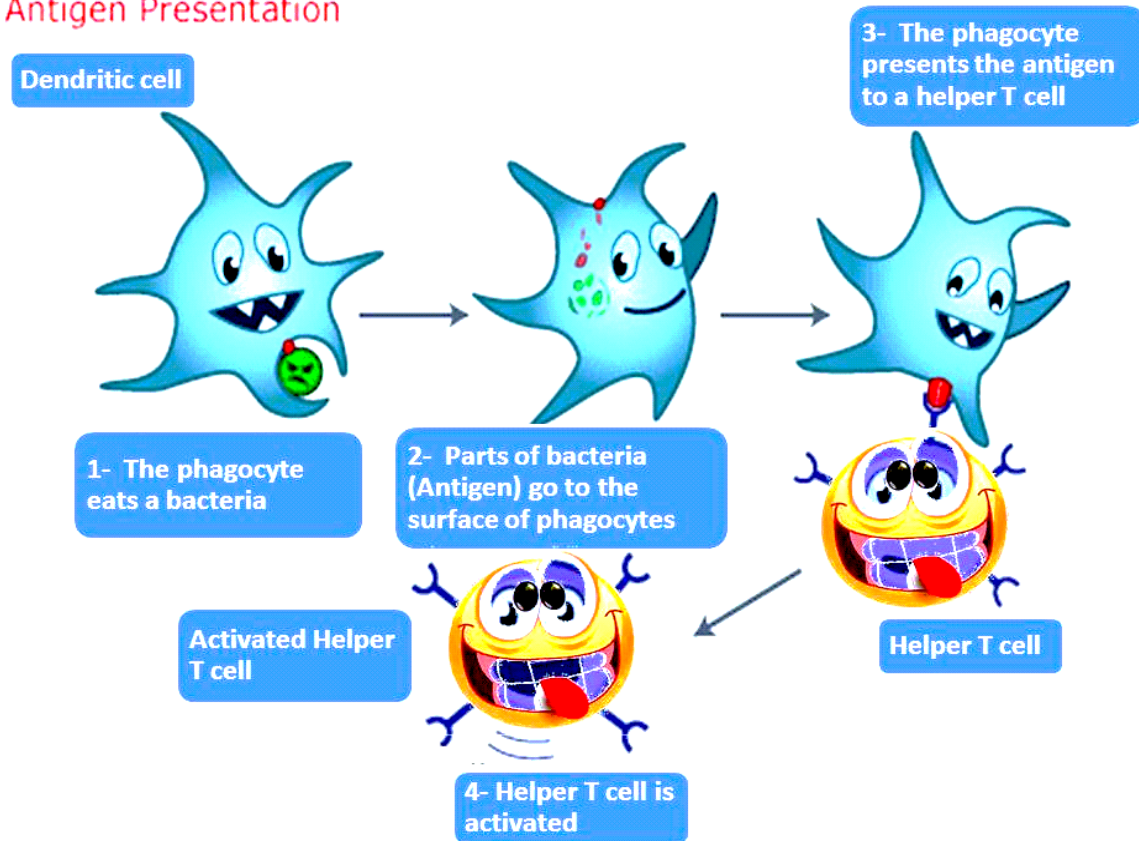
T CELLS

The lymphocytes maturing in thymus are termed as T cells. T cells are of two types, helper cells and killer cells.

HELPER T CELLS

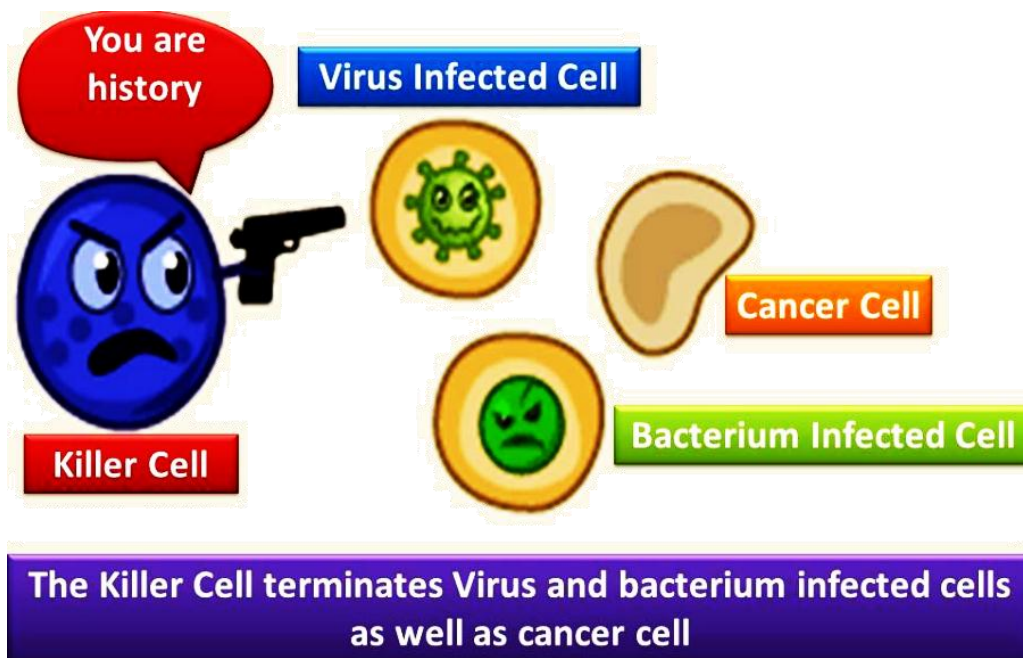
These are the main regulators of the immune defense. They primarily activate B cells and killer T cells. Whenever a macrophage or dendritic cell, which has eaten an antigen, passes to the adjacent lymph node the information about the captured pathogen is presented (Psora). The phagocytes display an antigen fragment from the invader on its own surface, the process called antigen presentation (Sycosis). When the receptor of a helper T cell recognizes the antigen, the T cell is activated (Psora). Once activated, helper T cells start to divide and to produce proteins that activate B and T cells as well as other immune cells (Sycosis). The failure of lymphocytes to respond to an antigen after encountering that antigen is called tolerance and is the opposite of activation.

Antigen Presentation



THE KILLER T CELLS

These are specialized in attacking body cells infected by viruses and sometimes also by bacteria. They may attack cancer cells too. The killer T cell has receptors, used to search each cell that it meets. If a cell is infected, it is quickly killed (Syphilis). Infected cells are recognized because tiny traces of the invader or antigen, are often found on their surface.



B CELLS

The B cells have receptors on their surface, specific to certain antigens. B lymphocyte cell searches for antigen matching its receptors. If such an antigen is found, it connects to it, and a triggering signal is set off inside the B cell (Psora). This B cell now needs proteins produced by helper T cells to become fully activated. After activation, the B cell starts to divide and produces clones of itself (Sycosis). During this process, two new cell types are formed-

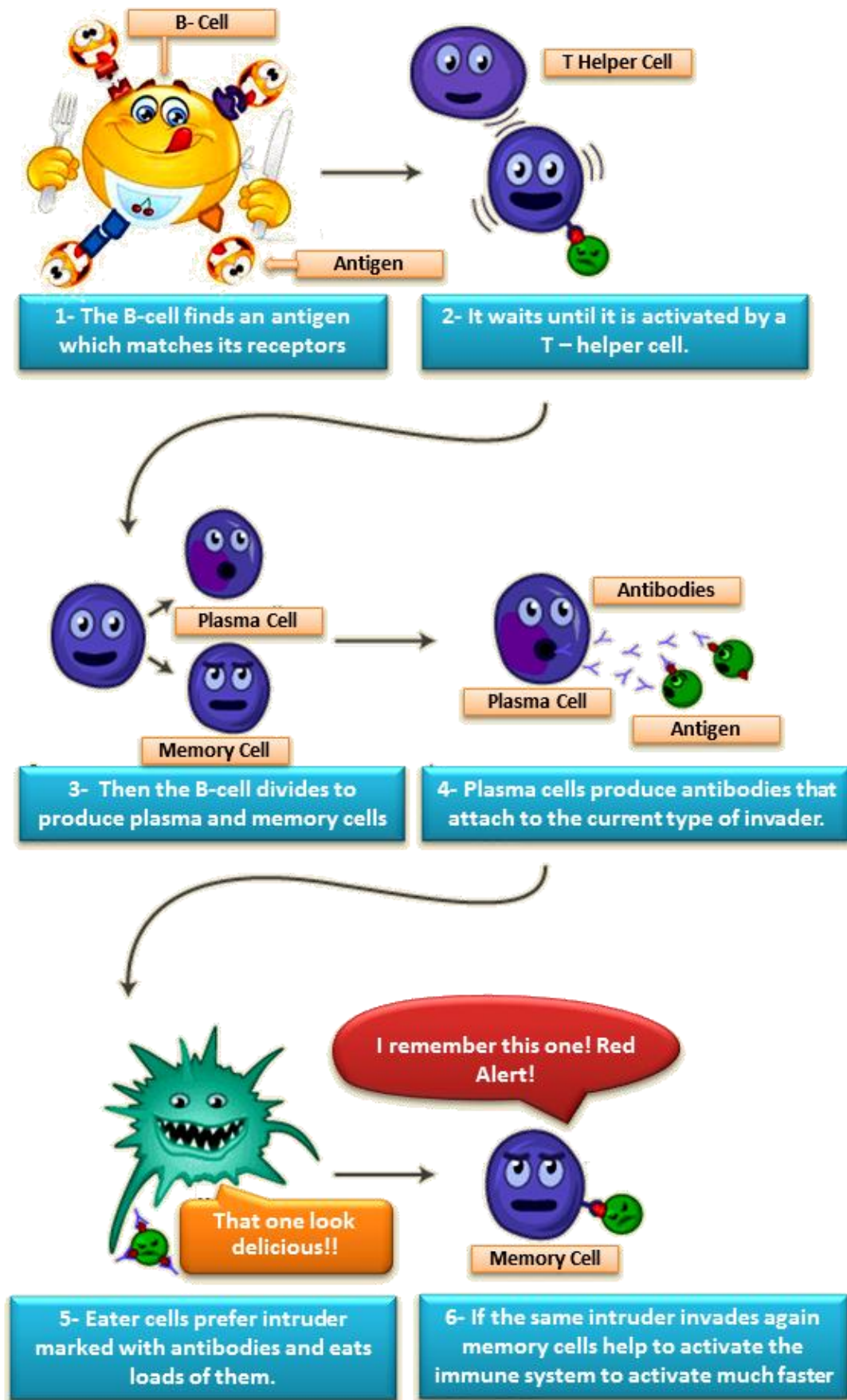
PLASMA CELLS

The plasma cells produce a specific protein, the antibody which responds to the same antigen that matched the B cell receptor. These antibodies can seek out intruders and help in their destruction. (Syphilis)

Antibody attaches to matching antigen. The attached antibodies serve as an appetizing coating for eater cells such as the macrophage. Antibodies also neutralize toxins and debilitate viruses, preventing them from infecting new cells. Each branch of the Y-shaped antibody can bind to a different antigen, so while one branch binds to an antigen on one cell, the other branch could bind to another cell - in this way pathogens are gathered into large groups that are easier for phagocyte cells to demolish. Bacteria and other pathogens covered with antibodies are also more likely to be attacked by the proteins from the complement system. (Psora)

MEMORY CELLS

These cells have a prolonged life span and can thereby remember specific invaders. T cells can also produce memory cells with an even longer life span than B memory cells. The second time an intruder tries to attack the body, both B and T memory cells help the immune system to activate much faster. The invaders are rubbed out before the infected body feels any symptoms or signs. This is the immunity against that intruder or antigen. (Psora)



Mechanism of Immunity

AUTOIMMUNE DISEASE

Autoimmune disease or autoimmunopathy is dysfunction (Psora), damage or destruction of its own tissues (Syphilis) caused by autoimmunity.

The susceptibility to autoimmune diseases can be either inherited (Syphilis) or acquired (Psora) or in many diseases, both. These diseases result from a complex interplay of pathways and events which initially allow autoreactivity (Psora) to manifest, and then, after an initiating event, allow development of self-sustaining (Sycosis) tissue damage (Syphilis).

TYPES OF AUTOIMMUNITY

Autoimmunopathies are chronic inflammatory diseases (Psora/Sycosis) which can be subdivided into several specificities on the ground of the clinical picture as well as serological findings and involvement of organ systems.

A- ORGAN SPECIFIC AUTOIMMUNITY

It affects a specific organ and is due to certain organ or tissue specific antibodies or lymphocytes. Organ-specific autoimmune diseases run in families, but within a family, they run variably e.g. one member may have type one diabetes, other autoimmune thyroid disease, while another multiple sclerosis. The inheritance of susceptibility to organ-specific autoimmunity is extraordinarily complex. Particular haplotypes of the major histocompatibility complex, such as HLA-DR3-DQ2 are strongly associated with human susceptibility to multiple organ-specific autoimmune disorders. HLA DR3-DQ2 is double serotype that specifically recognizes cells from individuals who carry a multigene HLA DR, DQ haplotype. Certain HLA DR and DQ genes have known involvement in autoimmune diseases. (Psora/Syphilis/Sycosis)

B- SYSTEMIC AUTOIMMUNITY

Systemic diseases involve multiple organs or organ systems and are caused by a variety of specificities or circulating immune complexes. The systemic autoimmune diseases are a complex group of disorders which include systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), autoimmune myopathies, Sjögren's syndrome (SS), vasculitis and scleroderma. The diseases manifest in a wide range of clinical phenotypes, often united by chronic inflammation and involvement of multiple organ systems. A dominant feature of these diseases is the elaboration of high titer, high affinity autoantibodies, which are strongly associated with phenotype (Sycosis/Syphilis). Autoimmune myopathies occur in myositis, SLE and scleroderma, or the sicca complex (dry eyes/mouth) in many of the systemic autoimmune diseases. Autoantigens in systemic autoimmune diseases appear to be ubiquitously expressed molecules and may be essential to the disease process.

CAUSES OF AUTOIMMUNITY

Factors that trigger the commencement include abnormalities in –

- Tolerance induction (Psora)
- Regulatory T-cell (Treg) development (Sycosis)
- Immune signaling thresholds (Psora)

The propagation phase of autoimmunity has two striking features-

- Self-sustaining nature (Sycosis)
- Capacity for auto-amplification, exemplified by disease flares (Psora/ Sycosis)

These features suggest the presence of a feed-forward cycle in disease propagation (Sycosis). This feed-forward cycle of autoimmunity is accompanied by tissue damage (Syphilis). In this cycle, immune effector pathways cause damage and provide antigen to get-up-and-go the ongoing immune response, which in turn fuel the immune response (Syphilis/Sycosis).

Overall, autoimmunity is governed by multiple factors-

- Genes and the environment contribute to the development of autoimmune diseases. (Psora/Syphilis/Causa occasionalis)
- Nearly 80% of autoimmune diseases occur in women.
- Sex hormones regulate inflammation and alter autoimmune disease in animal models.
- Estrogens increase auto-antibodies, which are key diagnostic criteria for most autoimmune diseases.
- Many theories exist for how infections and chemicals could cause autoimmune diseases.
- Microbial and/or chemical activation of innate immunity must occur at the same time as damage to self-tissues in order for autoimmune disease to develop.
- Dysregulation of peripheral immunoregulatory mechanisms is a key characteristic of autoimmune diseases.
- Successful treatment of autoimmune diseases might require the targeting of multiple effector pathways.

PATHOGENESIS OF AUTOIMMUNITY

Sometimes, immune tolerance is lost for body's own tissues (Psora/Syphilis). It usually occurs after destruction of some of the body's own tissues (Syphilis), which releases significant amounts of self-antigens that circulate in the body and apparently cause acquired immunity in the form of either activated T cells or antibodies (Psora/ Sycosis). Autoimmunity arises when innate immune responses within the host are focused against self-components. Autoimmune diseases may be mediated in two ways-

IMMUNE COMPLEX MEDIATED-

- Deposition in tissues (Sycosis)
- Inflammatory response (Psora)

AUTOANTIBODY MEDIATED

- Complement activation (Psora)
- Cell mediated (Psora/Sycosis/Syphilis)
- Functional- Receptor stimulation or blockade (Psora)

MECHANISM OF AUTOIMMUNITY

Autoimmunity is mediated by a variety of molecular and cellular events, and responses. The progress of an autoimmune disease is very complex and is due to altered tolerance (Psora). There is recognition of self-antigens by lymphocytes, centrally involved in pathologic organ damage (Psora). Autoimmune disease is inherited as a complex trait, with multiple loci, governing various aspects of disease susceptibility (Psora/Sycosis/Syphilis).

TOLERANCE

Unresponsiveness to an antigen induced by prior exposure to that antigen is called tolerance. Self-tolerance is a fundamental property of the immune system and is induced when self-reactive lymphocytes encounter self-antigens. Its failure leads to autoimmunity (Psora/Syphilis). The principal mechanisms are divided into central tolerance and peripheral tolerance. At times, the immune system may fail to recognize or react against some self-antigens; this phenomenon is called ignorance (Psora/Syphilis/Sycosis). Tolerance may be-

CENTRAL TOLERANCE

In this case, many of the immature lymphocytes that see self-antigens in the central (generative) lymphoid organs (thymus, bone marrow) are killed (Syphilis). Immature lymphocytes recognize the antigen in generative lymphoid organs with high affinity. This results in death of the lymphocytes by apoptosis (Syphilis), also called negative selection (Syphilis). The thymus expresses many self-antigens thought to be restricted to peripheral tissues, thus providing an immunological representation of self (Psora/Syphilis). The expression of some of these tissue antigens in the thymus is mediated by a transcription factor called AIRE. Other mechanisms of central tolerance like receptor editing (B cells), generation of regulatory and T cells (CD4⁺ T cells) render self-reactive lymphocytes harmless. (Sycosis)

PERIPHERAL TOLERANCE

In this case, lymphocytes that recognize self-antigens in peripheral tissues are shut off, suppressed by regulatory T lymphocytes, or killed (Psora/Syphilis). It can be seen in-

IN T CELLS

- Anergy (functional unresponsiveness)- results from antigen recognition without co-stimulation, or engagement of inhibitory receptors of lymphocytes (e.g. CTLA-4) (Psora)
- Suppression (by regulatory T cells) (Syphilis)
- Deletion (apoptosis) (Syphilis)

IN B CELLS

- Anergy- exclusion from lymphoid follicles and death (Psora/Syphilis)

Understanding the mechanisms of peripheral tolerance is leading to new strategies for shutting off harmful immune responses and restoring the normal balance of lymphocyte activation and tolerance. Development of autoimmunity is a consequence of the failure of self-tolerance; usually results from a combination of genetic susceptibility and environmental triggers e.g. infections (Psora/Syphilis/Sycosis/Causa occasionalis).

GENETIC SUSCEPTIBILITY

Multiple genes influence susceptibility to autoimmune disease; MHC (major histocompatibility complex) is the most important. Many non-MHC genes are known to be involved. A few polymorphic genes are also associated with autoimmune diseases. (Psora/Syphilis)

ENVIRONMENTAL TRIGGERS

Infections usually trigger and/or exacerbate autoimmunity. The mechanisms include induction of co-stimulators on APCs (Antigen-presenting cells), and "molecular mimicry" of foreign antigens with self. (Psora/Causa occasionalis)

Astonishingly, some infections seem to guard individuals from certain autoimmune diseases. Their mechanisms are not known.

PROPAGATION OF AUTOIMMUNITY

Autoimmunity propagates through following steps-

1- MOLECULAR OR EPITOPE MIMICRY

An epitope, also known as antigenic determinant, is the part of an antigen that is recognized by the immune system, specifically by antibodies, B cells, or T cells i.e. the epitope is the specific piece of an antigen that an antibody binds to. In autoimmune disorders, similarity between foreign and self-epitope develops (Psora).

2- EPITOPE SPREADING

Epitope spreading is the development of immune responses to endogenous epitopes secondary to the release of self-antigens during a chronic autoimmune or inflammatory response. (Psora/Sycosis)

3- FOREIGN T CELL HELP

Helper T cells help the activity of other immune cells by releasing T cell cytokines (Psora). These cells help suppress or regulate immune responses (Psora/Syphilis). They are essential in B cell antibody class switching, in the activation and growth of cytotoxic T cells, and in maximizing bactericidal activity of phagocytes such as macrophages (Psora/Sycosis).

4- INDUCTION OF AUTOIMMUNITY

The main factors involved in autoimmunity induction are-

- Presence of autoreactive lymphocytes (Psora)
- Induction of reactive as opposed to tolerant state (Sycosis/Syphilis)

HOMOEOPATHY AND AUTOIMMUNITY

Autoimmunity is Syphilitic disorder triggered by Psora and maintained by Sycosis. The chief remedies for autoimmune diseases are-

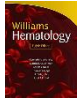
GENERALS – AUTOIMMUNITY- am-c. anac. Ang. Ant-c. Ant-t. Apis arg-met. ARG-N. arn. Ars. Asaf. asar. AUR-M. AUR. berb-a. berb. Carb-an. Carb-v. Caust. Cinnb. clem. Colch. Con. euph. Ferr. Fl-ac. flav. Graph. guaj. Hep. hydr. Iod. Kali-bi. Kali-c. KALI-I. Kreos. Lac-c. Lach. Lyc. MERC-C. MERC. Mez. mill. NIT-AC. nux-v. Petr. Ph-ac. phos. plat. PSOR. Sabad. Sars. Sec. Sel. SIL. STAPH. SULPH. THUJ. Thyr.

SOME AUTOIMMUNE DISEASES

Organ-Specific Autoimmune Diseases			
Organ	Disease(s)	Self-Antigen	Major Autoimmune Mechanism
Adrenal cells	Addison's disease	Cytochrome P-450 antigens	Autoantibodies
Red blood cells	Autoimmune hemolytic anemia	Red blood cell membrane proteins	Autoantibodies
Platelets	Idiopathic thrombocytopenic purpura	Platelet antigens (GP IIb/IIIa)	Autoantibodies
Stomach	Pernicious anemia	Gastric parietal cell antigens (H+/ATPase, intrinsic factor)	Autoantibodies/T cells
Small bowel	Celiac sprue (gluten enteropathy)	Transglutaminase	Autoantibodies/T cells
Thyroid	Hashimoto's thyroiditis	Thyroid cell antigens (e.g.,	T cells/autoantibodies
	Graves' disease	Thyroid-stimulating hormone	Autoantibodies
Muscle	Myasthenia gravis	Acetylcholine receptors	Autoantibodies
Pancreatic islets	Type 1 diabetes	Beta cell antigens (glutamic acid decarboxylase, insulin)	T cells (autoantibodies present)
Hepatocytes	Autoimmune hepatitis	Hepatocyte antigens (cytochrome P450 2D6)	T cells/antibodies
Bile duct cells	Primary biliary cirrhosis	Intrahepatic bile duct (pyruvate dehydrogenase complex protein)	Autoantibodies/ T cells
Heart	Rheumatic heart disease	Myocardial antigens	Autoantibodies
Kidney/lung	Goodpasture's syndrome	Basement membrane antigens (type IV collagen α3 chain)	Autoantibodies

Systemic Autoimmune Diseases		
Disease(s)	Self- antigen	Major Autoimmune Mechanism
Ankylosing sponkylitis	Vertebrae	Immune complexes
Multiple sclerosis	Brain or white matter	TH ₁ cells and T _c cells, auto-antibodies
Rheumatoid arthritis	Connective tissue, IgG	Auto-antibodies, immune complexes
Systemic lupus erythematosus	DNA, nuclear protein, RBC and platelet membranes	Auto-antibodies, immune complexes
Scleroderma	Nuclei, heart, lungs, gastrointestinal tract, kidney	Auto-antibodies
Sjogren's syndrome	Salivary gland, liver, kidney, thyroid	Auto-antibodies

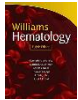
BIBLIOGRAPHY



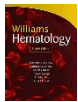
Chapter 119. Thrombocytopenia > Thrombocytopenia in Patients with Systemic Lupus Erythematosus and Other Autoimmune Conditions Williams Hematology, 8e ... Systemic lupus erythematosus (SLE) is a complex autoimmune disease that primarily afflicts women...



Chapter 120. Rheumatoid Arthritis and Other Autoimmune Diseases > Rheumatoid Arthritis and Other Autoimmune Diseases: Introduction Hazzard's Geriatric Medicine and Gerontology, 6e ... Our understanding of the pathogenesis and approach to treatment of autoimmune diseases continue...



Chapter 132. The Antiphospholipid Syndrome > Systemic Lupus Erythematosus and Other Autoimmune Conditions Williams Hematology, 8e ... APS patients frequently present with other autoimmune conditions. A significant proportion...



Chapter 133. Antibody-Mediated Thrombotic Disorders: Thrombotic Thrombocytopenic Purpura and Heparin-Induced Thrombocytopenia > Autoimmune Disorders Williams Hematology, 8e ... Autoimmune thrombocytopenia may be confused with TTP if other causes of microangiopathic...



Chapter 154. Mechanisms of Autoimmune Disease > Drugs and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e ... Certain drugs can induce autoimmune diseases, with potentially protean clinical manifestations...



Chapter 154. Mechanisms of Autoimmune Disease > Environmental Factors and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 154. Mechanisms of Autoimmune Disease > Infections and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e ... Infection has been suggested as a possible causative factor for autoimmunity. Postulated...



Chapter 154. Mechanisms of Autoimmune Disease > Innate Immunity and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 154. Mechanisms of Autoimmune Disease > Pathogenesis of Tissue Damage in Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e ... The mechanisms involved in tissue damages in autoimmune diseases are similar to those...



Chapter 154. Mechanisms of Autoimmune Disease > Tolerance and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 154. Mechanisms of Autoimmune Disease > Toxins and Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e ... Environmental chemicals and toxins also have been reported as a potential cause for autoimmune...



Chapter 164. Systemic Necrotizing Arteritis > Autoimmune Serologies Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 164. Systemic Necrotizing Arteritis > Vasculitis Associated with Other Autoimmune Diseases Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 166. Adamantiades–Behçet Disease > Autoimmune Mechanisms Fitzpatrick's Dermatology in General Medicine, 8e



Chapter 18. Innate Immunity > The General Strategy of Innate Immune Responses and the Concept of Forward Feedback Loops in Autoimmunity Williams Hematology, 8e ... Although the term autoimmunity is reserved for inappropriate adaptive immune responses...



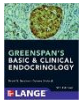
Chapter 2. Endocrine Autoimmunity > Autoimmune Aspects of Thyroid Disease Greenspan's Basic & Clinical Endocrinology, 9e ... Autoimmune thyroid disease can present in a polarized fashion with Graves disease (thyroid...



Chapter 2. Endocrine Autoimmunity > Autoimmunity Is Multifactorial Greenspan's Basic & Clinical Endocrinology, 9e ... of autoimmune diseases, autoimmunity is a multifactorial event. Specifically, defects in apoptosis-related...



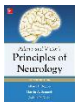
Chapter 2. Endocrine Autoimmunity > Environmental Factors in Autoimmunity Greenspan's Basic & Clinical Endocrinology, 9e ... Environmental factors also play a critical role in the pathogenesis of autoimmune disease...



Chapter 2. Endocrine Autoimmunity > Genetic Factors in Autoimmunity Greenspan's Basic & Clinical Endocrinology, 9e ... Epidemiologic studies demonstrate that susceptibility to most autoimmune diseases has...



Chapter 234. Immunobiologicals, Cytokines, and Growth Factors in Dermatology > Autoimmunity Fitzpatrick's Dermatology in General Medicine, 8e



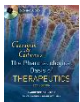
Chapter 36. Multiple Sclerosis and Other Inflammatory Demyelinating Diseases > White Matter Lesions Associated with Systemic Autoimmune and Inflammatory Diseases Adams & Victor's Principles of Neurology, 10e ... In systemic lupus erythematosus and less often in other autoimmune diseases (mixed connective...



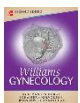
Chapter 38. Urticaria and Angioedema > Autoimmunity and Chronic Urticaria Fitzpatrick's Dermatology in General Medicine, 8e ... suggestion that patients with chronic urticaria and angioedema might have an autoimmune diathesis...



Chapter 41. Hematologic Emergencies > Autoimmune Drug-Related AIHA CURRENT Diagnosis & Treatment Emergency Medicine, 7e ... Autoimmune drug-related AIHA results when the offending drug triggers formation of autoantibodies...



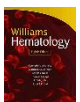
Chapter 42. ACTH, Adrenal Steroids, and Pharmacology of the Adrenal Cortex > Autoimmune Destruction of Erythrocytes Goodman & Gilman's The Pharmacological Basis of Therapeutics, 12e ... Patients with autoimmune destruction of erythrocytes (i.e., hemolytic anemia with a positive...



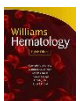
Chapter 6. First-Trimester Abortion > Autoimmune Factors Williams Gynecology, 2e



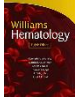
Chapter 6. Intestinal Mucosal Immunology and Ecology > Autoimmunity Gastrointestinal Physiology, 2e



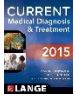
Chapter 94. Chronic Lymphocytic Leukemia and Related Diseases > Autoimmunity Williams Hematology, 8e ... Patients with CLL are prone to developing systemic autoimmune disease. The most common...



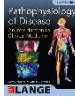
Chapter 94. Chronic Lymphocytic Leukemia and Related Diseases > Systemic Autoimmune Disease Williams Hematology, 8e ... CLL patients have an increased risk of autoimmune disease, in particular autoimmune hemolytic...



Chapter 97. General Considerations of Lymphoma: Epidemiology, Etiology, Heterogeneity, and Primary Extranodal Disease > Autoimmunity Williams Hematology, 8e ... Some autoimmune disorders are risk factors for lymphoma. An increased incidence of malignant...



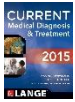
Diabetes Mellitus & Hypoglycemia > 5. Autoimmune Hypoglycemia Current Medical Diagnosis & Treatment 2015 ... Patients with autoimmune hypoglycemia have early postprandial hyperglycemia followed...



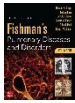
Disorders of the Adrenal Cortex > Autoimmune Adrenocortical Insufficiency— Pathophysiology of Disease: An Introduction to Clinical Medicine, 7e ... Autoimmune destruction of the adrenal glands is thought to be related to generation...



Encyclopedia Homoeopathica



HIV Infection & AIDS > B. Autoimmunity/Allergic and Hypersensitivity Reactions Current Medical Diagnosis & Treatment 2015 ... Autoimmunity can occur as a result of disordered cellular immune function or B lymphocyte...



Pulmonary Alveolar Proteinosis Syndrome > Autoimmune PAP Fishman's Pulmonary Diseases and Disorders ... (autoimmune PAP). (Reproduced with permission from Uchida K, Nakata K, Suzuki T, et al. GM-CSF autoantibodies...



Radar 10