



# In Autoimmune Disease the Immune System Attacks the Body in which it is Located

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## Abstract

Autoimmune diseases are diseases in which the immune system mistakenly attacks the body in which it is located. The function of the immune system is extremely important because it protects against bacteria, parasites, viruses and cancerous mutations. Unfortunately, there are also situations in which the mentioned system can turn against the person himself. There is no clear reason why this happens, which is why autoimmune diseases are very often the subject of many studies today, and there are over a hundred known autoimmune diseases that are mostly accompanied by certain mutations and changes in antibody states. Depending on how the immunity turns against the person, autoimmune diseases develop that attack tissues or organs. This is precisely why the number and character of symptoms can be very colorful and diverse, which significantly complicates the diagnostic process even for skilled specialists.

**Keywords:** Human Body; Immunity; Autoimmune Diseases; Health

## Introduction

The human immune system utilizes a number of instruments for keeping up adjust between resistant actuation and concealment [1]. Disease, cancers, and injury trigger inflammatory processes that have to be “checked” in arrange to avoid distorted reactions. This complex prepare involves multiple cellular components that must work in concert to attain an fitting level of aggravation. Beneath homeostatic conditions, safe cells are planned so that they are not constitutively dynamic which might lead to harm of solid tissues or infection. One of the ways in which the resistant framework moderates this adjust is through the official of ligand on antigen presenting cells (APCs) to comparing negative safe checkpoint receptors (NCRs) on T cells. Particularly, these resistant checkpoints are proteins that act as T cell receptor (TCR) co-signaling accomplices

that provide either positive or negative signals to T lymphocytes. Ligand official comes about in down direction of T cell enactment and concealment of effector capacities. In any case, in numerous malady states, this framework is captured, subsequently driving to T cell brokenness and incapable reactions. Unremitting disease and cancers have been appeared to do fair that.

Much exertion has been made towards superior understanding these NCRs and the part they play in health and disease. In addition, recognizing ways in which these NCRs can be blocked to reestablish effector work and superior position T cells to battle cancerous tumors and contaminations has been a major center of investigate. Tackling this framework in arrange to improve T cell work has been groundbreaking for the field of immunology, so much so that the 2018 Nobel prize in physiology and medication went to the two men who

found these important immune molecules, James Allison and Tasuku Honjo. The thought of mobilizing our claim safe frameworks to battle malady isn't a modern thought. In any case, the revelation of these safe modulatory receptors has made this tantalizing plausibility attainable as a promising clinical treatment. Among the foremost unmistakable safe checkpoint receptor-ligand combinations are CTLA-4/CD80, PD-1/PD-L1, TIGIT/PVR and Tim-3/Galectin 9.

### Congenital Defects

Clinical examinations of different intrinsic surrenders have made a difference characterize numerous angles of ordinary safe physiology [2]. The exceptionally nature of a imperfection in have safe reactions places the helpless person at tall hazard for a assortment of irresistible, harmful, and immune system infections and disarranges. The nature of the particular useful deformity will essentially impact the sort of contamination that influences the have. Any immunopathogenic instrument that impedes T-lymphocyte work, or cell-mediated resistance, inclines the have to the improvement of genuine unremitting and possibly lifethreatening deft contaminations with infections, mycobacteria, parasites, and protozoa including any or all organ frameworks. Additionally, immunopathogenic dysfunction of B lymphocytes coming about in counter acting agent insufficiency will predispose the have to pyogenic sinopulmonary and mucosal contaminations. As the atomic bases of numerous essential immunodeficiency clutters are being found, it has gotten to be clear that diverse atomic absconds can result in common clinical phenotypes.

The T lymphocyte plays a central part in actuating and planning safereactions, and dysfunction can be related with an expanded frequency of immune system phenomena. These incorporate diseases clinically comparative to rheumatoid joint pain, systemic lupus erythematosus, and safe hematologic cytopenias. Patients with impeded safe reactions are moreover at more noteworthy hazard for certain malignancies than the common populace. The event of cancer may be related to an fundamental impedance of tumor reconnaissance, dysregulation of cellular expansion and separation, chromosomal translocations amid imperfect antigen receptor quality modification, or the nearness of irresistible operators inclining to or causing cellular change. Non-Hodgkin lymphoma or B-cell lymphoproliferative illness, skin carcinomas, and gastric carcinomas are the foremost regularly happening tumors in patients with immunodeficiency.

Traditionally, the essential immunodeficiencies are classified concurring to which component of the resistant reaction is mainly compromised: the humoral reaction, cellmediated insusceptibility, complement, or

phagocytic cell work. Unmistakable formative stages characterize the development and separation of the cellular components of the resistant framework. The fundamental pathophysiologic variations from the norm driving to essential immunodeficiency are assorted and incorporate the taking after: (1) early formative defects in cellular development, (2) particular enzyme defects, (3) anomalies in cellular multiplication and functional differentiation, (4) abnormalities in cellular regulation, and (5) abnormal responses to cytokines.

### Immunity

Immunity has two distinct but complementary branches to combat disease: innate and acquired immunity [3]. The trademark of obtained resistance is particular acknowledgment and end of nonself. The pathogen is recognized as a particular substance, not fair as nonself, and a record is held for more quick reaction to future experiences, a wonder known as immunologic memory. Antigen acknowledgment is interceded by lymphocytes, T cells, and B cells. T cells ensure the cells of the body against changes by change or viral contamination (cellular insusceptibility) and bind peptide antigens that have been prepared by the body's cells. B cells give assurance against extracellular irresistible living beings and foreign material (humoral immunity) and recognize antigens in their local natural state. Parenchymal cells express lesson I MHC atoms. Course I particles show peptides from inside, e.g., peptides from ordinary cellular forms or from viral replication, which are bound by T cells communicating an grip particle with uncommon partiality to course I, the CD8 particle. Hematopoietic cells moreover express course II MHC atoms. These particles show peptides that have been phagocytized from encompassing extracellular spaces and bind to T cells complemented by an grip atom with partiality to course II, the CD4 particle. B cells tie dissolvable antigens and emit solvent shapes of their receptor, known as antibodies, to tie these remote particles. Material that's bound by an counter acting agent is opsonized (flagged) for destruction by cells of the natural arm of insusceptibility — phagocytic cells missing the capacity to recognize self from nonself — fundamentally macrophages, monocytes, and polymorphonuclear leukocytes (PMNs). Antibody-bound surfaces actuate a dangerous enzymatic cascade known as the complement framework. This leads to pulverization of the layer to which the complement is bound and further opsonization. The whole resistant handle is encouraged by a implies of increasing the reaction of one cell to one antigen. Cytokines [known as interleukins (ILs)] are polypeptides that are discharged by numerous cell sorts and enact or stifle adjoining safe cells. The safe reaction to an allograft is the result of incongruence between the recipient's receptor collection and the donor's MHC polymorphisms. Effector components that have advanced to check viral,

parasitic, and bacterial diseases, as well as those in put to anticipate harm and autoimmunity, all come into play after transplantation. Dismissal, like physiologic resistance, can be partitioned into humoral and cellular components. Humoral dismissal of a unite can be the result of antibodies existing in circulation earlier to presentation or antibodies procured after presentation. Cellular dismissal is the result of T-cell inconsistency between the benefactor and beneficiary.

### Immune System

The immune system is designated to guard the body from assaults that begin exterior (contaminations) or interior (tumors) [4]. When reacting to assaults from non-self, the resistant framework employments both non-specific (intrinsic insusceptibility) and exceedingly particular frameworks (obtained insusceptibility). Natural resistance is enacted quickly, whereas the procured insusceptibility takes longer.

A few diseases can influence the immune system. Disarranges related with a diminished reaction against nonself-antigens are called immunodeficiencies. The foremost common immunodeficiencies are secondary to (1) contaminations, such as HIV, (2) chemotherapy or (3) systemic infections such as diabetes. On the other hand, the essential immunodeficiencies (PIDs) are in most cases uncommon illnesses related with particular hereditary changes. An over the top reaction against exogenous antigens is the cause of unfavorably susceptible clutters and extreme touchiness responses; these are exceptionally common disarranges. In any case, a few uncommon conditions related with lack of atoms included in natural resistance, such as innate angioedema due to C1 esterase inhibitor insufficiency, have a clinical introduction that will mirror a few unfavorably susceptible appearances. At last, there's a wide range of diseases characterised by an irregular reaction against self-antigens. Because of the part of natural insusceptibility, most of the autoinflammatory diseases contains a systemic association. On the other hand, autoimmune infections may influence a single organ or tissue or the complete body (systemic autoimmune diseases).

Regularly there's an affiliation between diverse infections of the immune system. For case, immunodeficiencies are frequently related with autoimmune diseases, such as autoimmune hemolytic weakness and thrombocytopenia, bowel provocative infections, or systemic sarcoidosis-like signs; Job's syndrome could be a primitive immunodeficiency showing with an expanded serum level of IgE, defenselessness to bacterial and contagious contaminations, and signs of atopic dermatitis unclear from a hyperreactivity response; Churg-Strauss syndrome is an immune system, ANCA-associated dispersed necrotizing

vasculitis with extravascular granulomas happening nearly solely among patients with asthma and tissue eosinophilia; at long last numerous patients with autoimmune diseases have secondary cellular or humoral immunodeficiencies due to the illness or to immunosuppressive treatment.

### Innate Immunity

Innate immunity is speedy to reply but as it were to the foreordained cluster of atomic designs related with pathogens or those particles characteristic of harmed cells [5]. The innate resistant reaction is constrained to particular challenges that have habitually happened amid human advancement.

Not at all like us, many pathogens have the capacity to rapidly advance to counter our have protections. Indeed amid the course of an contamination, life forms may create instruments to upset our innate guards components. In expansion, the development of populaces and changes within the environment always uncovered us to modern challenges with which innate immunity may be incapable to manage.

To begin with found in lower vertebrates with jaws (cartilaginous angle such as sharks), adaptive immunity— comprised of cell-mediated resistance and humoral immunity— gives us with the capacity to identify and adaptively react basically to anymolecular structure outside to us (indeed one never some time recently experienced). The perspectives of versatile acknowledgment and reaction to outside structures and resistant memory characterize versatile insusceptibility and separate it from natural resistance. Natural insusceptibility can as it were adjust to modern challenges with advancement, though adaptive insusceptibility can adapt with already unrecognized specialists about in “real time.” Shockingly, the adaptive resistant framework may fail, recognizing the self as outside and coming about in autoimmune disease.

### Tolerance

The method of self-recognition (immunological tolerance) is central to the appropriately coordinated work of have defense [5]. Failure comes about in self-reactivity and autoimmune disease. Positive and negative determination of self-reactive T cells and B cells within the thymus and bone marrow is vital for self-recognition. For this to operate properly, the self-antigen must be communicated in those areas during resistant advancement. Typically not all around the case. Self-antigens from ineffectively vascularized tissue or basic proteins such as collagen or cartilage may not create self-tolerance, and a moo level of autoreactivity to these proteins (not related with illness) is now and then recognized. Antibodies coordinated toward the proteolytic

items of Ig breakdown are all around show in typical people and may play a physiological part in evacuating these items from the circulation. A more striking case is that of sperm antigens, which are not displayed in fetal advancement and are not displayed to creating T cells within the thymus. Traumatic or surgical harm to the testicles or spermatic conduits in an grown-up can uncover sperm-related antigens to the resistant framework and result in resistant reactivity toward have sperm. Sperm autoantibodies once in a while result in male sterility after such intercessions.

The development of autoimmune disease in 3 percent of the populace recommends that an continuous prepare must happen which failure comes about in self-recognition. Certain groups of people are more inclined to creating autoimmunity. Women are up to eight times more vulnerable to certain immune system conditions than men, which may be related to endocrine hormone generation. (There are a few special cases to this. The over said HLA B27-related disease ankylosing spondylitis presents distant more commonly in men.) There's a clear genetic component to the advancement of autoimmune disease, and these diseases run in families. In a few cases, the qualities of the MHC (HLA) framework are ensnared, but in numerous cases the nature of the genetic components dependable is unclear.

At a few level, autoimmune disease is likely to be related to a failure in continuous fringe T and B cell resistance, which shows up to be an progressing dynamic handle during life. Anergy is the proceeded nonresponsiveness of T cells to a have (or outside antigen) that happens when the antigen is displayed to the T cell without vital costimulatory signals (B7 cell receptor) from an antigen showing cell. APC express B7 in restricted areas, essentially lymph nodes, but not in tissue. A T cell experiencing a self-antigen in tissue (outside the lymph node) will gotten to be anergic and be incapable to reply to the self-antigen. During thymic advancement, a populace of long-lived administrative T cells create that are particular for self-antigens. These cells square actuation of autoreactive T cells, maybe by creating inhibitory cytokines or conceivably by capturing IL-2 essential for T cell actuation. An unprecedented genetic disease that removes the advancement of this administrative cell populace comes about in IPEX, which is an autoimmune disease coordinated toward numerous endocrine organs. Introduction of T cells to self-antigen may trigger apoptotic cell passing in spite of the fact that the mechanism remains unclear. In spite of the fact that diverse in detail, the instruments for peripheral B cell resistance have a comparable work.

## Failure

The reasons an person shapes an autoantibody to his or her own cells or tissue components are not well caught on

[5]. Unfortunately, after an person creates an autoimmune disease, it more often than not "doesn't go away." In spite of the fact that the affected person encounters periods when the illness is in abatement or the signs are controlled by treatment, the disease holds on and frequently advances. A few components have been hypothesized to clarify the pathogenesis of autoimmune diseases. As already famous, numerous immune system infections have a hereditary component (which is frequently complex). Localized contaminations may incite an immune system infection by permitting fiery antigen displaying cells to show fundamental costimulatory signals regularly not show at the tissue location. Tentatively it is conceivable to actuate selfreactive antibodies in creatures by showing either a self-antigen or a closely related antigen along side strong provocative chemicals. Molecular mimicry can happen when a outside antigen is adequately comparative to a self-antigen (cross responsive to a self-antigen) that autoimmunity can happen. The group A streptococcal M protein is likely to result within the autoimmune reaction dependable for rheumatic fever. Cross receptive viral proteins may play a part in type 1 diabetes, which is clearly an immune system infection. Certain drugs may actuate immune system annihilation of cells by changing their surface properties to advance safe acknowledgment.

## Autoimmunity

It is accepted that autoimmunity is the failure of an life form to recognize its claim constituent parts as self, which permits an safe reaction against its possess cells and tissues and hence to disease(s) [6]. Nearly any tissue/organ/system of the body might be the target of such an immunological assault in spite of the fact that certain tissues/organs are more commonly included. The foremost common autoimmune diseases are: celiac disease, type 1 diabetes mellitus, rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), Sjogren's, Hashimoto's thyroiditis, Graves' disease, idiopathic thrombocytopenic purpura (ITP), etc. Paul Ehrlich, at the starting of the twentieth century, was the primary to propose the concept of frightfulness autotoxicus, wherein a "normal" body does not mount an resistant reaction against its claim tissues. In this way, any autoimmune response was seen to be unusual and hypothesized to be associated with human disease. At show, immune system reactions are considered an integral part of vertebrate resistant frameworks, normally prevented from causing disease by the marvel of immunological resistance to self-antigens.

In common, it is considered that certain sum of low level of autoimmunity does exist within the solid body and is, in fact, useful. Such low-level autoimmunity might help within the acknowledgment of neoplastic cells by CD8+ T



cells, and in this way diminish the rate of cancer. Low-level of autoimmunity may permit a quick safe reaction within the early stages of an contamination when the accessibility of remote antigens limits the reaction (i.e., when there are few pathogens present).

### Autoimmune Disease

With an autoimmune disease, the safe framework erroneously assaults the body's healthy organs, tissues, and cells as in spite of the fact that they were antigens [7]. At the center of the resistant framework is the capacity to tell the distinction between what's you and what's outside (or, as talked about prior, self and non-self). In some cases, the body is incapable to tell the contrast, and when this happens, the body makes autoantibodies that assault ordinary cells by mistake. At the same time, T-cells fall flat to do their work of keeping the safe framework sound. The result may be a misinformed assault on the body. Solid organs, tissues, and cells are devastated, which clears out the body incapable to perform critical day by day capacities.

Autoimmune diseases are genuine and persistent, and affect nearly all the parts of the body. There are more than 80 known sorts, and a few have comparable side effects. Although each illness is special, numerous share the primary notices of ailment, such as fatigue, dizziness, muscle aches, and fever. This makes it difficult for suppliers to analyze these infections, which can be frustrating and upsetting for patients.

Autoimmune diseases are common, influencing more than 23.5 million Americans, and are a driving cause of passing and inability. It is obscure what causes an autoimmune disease, but numerous variables are included, counting the taking after:

**Gender** - More women than men have autoimmune diseases, which frequently begin during their childbearing years.

**Family history** - A few autoimmune diseases run in families, so acquiring certain qualities makes it more likely to induce an autoimmune disease.

**Environmental exposure** - Certain natural exposures may cause or compound a few autoimmune diseases. Daylight, chemicals, and diseases are connected to numerous autoimmune diseases.

**Race or ethnic background** - A few autoimmune diseases are more common and more serious in certain racial and ethnic groups than in others.

For numerous autoimmune diseases, side effects can

come and go and can change in seriousness at distinctive times. When indications go absent for a whereas or disappear, the understanding is said to be in remission. The diseases may also have flares, or sudden and serious side effects. Autoimmune diseases are inveterate and don't more often than not go absent, but side effects can be treated. A few autoimmune diseases are depicted somewhere else in this book, but an overview of the taking after common autoimmune diseases is in this area:

- Alopecia areata
- Celiac disease
- Type 1 diabetes
- Guillain-Barre syndrome
- Multiple sclerosis (MS)
- Myasthenia gravis (MG)
- Rheumatoid arthritis (RA)
- Juvenile rheumatoid arthritis (JRA)
- Scleroderma
- Lupus

### Etiology

What triggers autoimmune disease isn't known, but this subject has been goal explored since the depiction of the primary autoimmune disease (thyroiditis driving to annihilation of the thyroid organ and in this way to hypothyroidism) in 1956 [8]. It is presently clear that resistant responses regularly include self-recognition (through the necessity for Mhc acknowledgment in antigen introduction), so the issue is likely one of control instead of absolutely a disappointment to recognize between self and non-self. Evidence clearly appears that a few hereditary factors are related with a better frequency of autoimmune disease (e.g., certain Mhc alleles, female sex) which natural variables moreover play a role.

A few autoimmune diseases have been hypothesized to result from irresistible living beings whose antigens are exceptionally comparative to typical have antigens. Hence the resistant framework may be "tricked" by an contamination into responding against typical have antigens. this instrument of start of autoimmune disease is called molecular mimicry.

### Conclusion

An autoimmune disease is a condition in which the immune system mistakenly attacks the body because it recognizes it as a foreign body. Some autoimmune diseases attack only one organ, while others attack the whole body. There is no cure for these diseases, but the symptoms are treated to ensure an easier daily life. Under normal conditions, the immune system guards the body and protects it from bacteria and viruses that it recognizes as external factors,

i.e. foreign bodies dangerous to the body. In the case of an autoimmune disease, the immune system recognizes body parts such as joints or skin as a foreign body. It is not known exactly why this phenomenon occurs. Autoimmune diseases are more common in women. Some autoimmune diseases are transmitted genetically and can even skip a generation. Most often, they arise due to a combination of genetics and external factors, and stress can have a big impact on the occurrence of autoimmune diseases.

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