

# International Journal of Immunology Research



ISSN Print: 2664-8865  
ISSN Online: 2664-8873  
Impact Factor: RJIF 5.48  
IJIR 2023; 5(1): 14-18  
[www.immunologyjournal.in](http://www.immunologyjournal.in)  
Received: 20-01-2023  
Accepted: 22-03-2023

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## Antibodies protect the body from various pathogens

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**DOI:** <https://doi.org/10.33545/26648865.2023.v5.i1.a.13>

### Abstract

Antibodies are large Y-shaped proteins produced by plasma cells that use the immune system to identify and neutralize foreign objects and substances, such as bacteria and viruses. Antibodies recognize a unique part of a foreign target called an antigen. Antibodies are proteins from the group of globulins that are created in mammals as a reaction to foreign substances that have penetrated the body. These foreign substances are neutralized by antibodies, which are defensive substances that create a stimulated immune system.

**Keywords:** Antibodies, immunoglobulins, viruses, immune system, health

### Introduction

Antibodies are antigen-binding proteins created by terminally separated effector B cells called plasma cells<sup>[1]</sup>. Antibodies that tie antigens derived from the host organism (i.e., self-antigens) are alluded to as autoantibodies. Autoantibodies are considered one of the hallmarks of certain immune system diseases; be that as it may, the nearness of autoantibodies does not relate impeccably with disease. Autoantibodies have been identified in solid people as well as those with immune system diseases. The components whereby autoantibodies apply their impacts within the infection prepare are the same utilized by antibodies against remote antigens (i.e., non-self-antigens). These incorporate, but are not restricted to, opsonization, neutralization, complement actuation, expansion, and engagement of constant region (Fc) receptors.

Neutralization of an antigen or pathogen communicating the target antigen is one effector instrument credited to antibodies. For example, antibodies against flu infection hemagglutinin neutralize the infection by blocking the interaction of the infection with the receptor on the target cell, in this manner avoiding contamination. In expansion, whereas not avoiding flu contamination, antibodies against flu neuraminidase limit replication of the infection by anticipating discharge of virus from tainted cells. Typically one of the ways antibodies, which actuate pathogen-specific antibodies, inspire security from illnesses. In any case, neutralization of self-antigens by autoantibodies can moreover contribute to the pathogenesis of a few immune system diseases. For illustration, neutralizing autoantibodies against the cytokine granulocyte/macrophage colony-stimulating figure (GM-CSF) are found in immune system pneumonic alveolar proteinosis, which is characterized by broken alveolar macrophages and practically disabled neutrophils. Autoantibodies against GM-CSF square interaction of the cytokine with receptors on macrophages, repressing their development, and on neutrophils, driving to impedance of phagocytosis, adhesion, bacterial killing, and oxidative burst.

### Immunoglobulins

The World Health Organization (WHO) in 1964 coined the term immunoglobulin (Ig) for the term counter acting agent<sup>[2]</sup>. The immunoglobulins integrated essentially by plasma cells and lymphocytes are structural and chemical concepts. The immunoglobulin incorporates counter acting agent globulins and the cryoglobulins, macroglobulins, and irregular myeloma proteins. In this way, all antibodies, which are natural and utilitarian models, are immunoglobulins, but not all immunoglobulins may be antibodies.

Immunoglobulins or antibodies constitute 20–25% of the whole serum protein.

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Depending on physicochemical and antigenic contrasts, they are partitioned into five classes of immunoglobulins: (1) immunoglobulin G (IgG), (2) immunoglobulin M (IgM), (3) immunoglobulin A (IgA), (4) immunoglobulin E (IgE) and (5) immunoglobulin D (IgD).

Immunoglobulins are glycoproteins. A single counter acting agent particle has two indistinguishable overwhelming chains and two indistinguishable light chains, H2L2, or a different (H2L2) n of this essential four-chain structure. This gives immunoglobulin an in general “Y” or “T” shape, which is the foremost broadly perceived include of immunoglobulin structure.

### Function

The foremost pivotal function of antibodies is to confer protection against microbial pathogens <sup>[2]</sup>. Antibodies confer protection in numerous ways: (1) they avoid connection of organisms to mucosal surfaces of the have, (2) they diminish the destructiveness of organisms by killing poisons and infections, (3) they encourage phagocytosis by opsonisation of microbes and (4) they actuate complement, driving to complement-mediated exercises against organisms.

They are a complex structure of four polypeptide chains: two indistinguishable overwhelming (regularly 55 kDa each) chains and two indistinguishable light chains (25 kDa each). The terms “heavy” and “light” allude to the atomic weights of the chains, though light chains have a atomic weight of 25,000 Da. The overwhelming chains are longer, and the light chains are shorter. Sub-isotypes exist for  $\alpha$  and  $\gamma$  chains, and this leads to the presence of subclasses of the individual immunoglobulins.

### Production

It is the most include of humoral resistant reactions <sup>[2]</sup>. The counter acting agent generation includes three steps: (1) the afferent appendage that comprises of the antigen section, its location and outcome within the tissues together with its contact with appropriate safe cells; (2) the central work that includes antigen handling and control of counter acting agent generation; and (3) the efferent appendage that consists of counter acting agent discharge, its dispersion in tissues, blood and other body liquids and its appearances in combating antigen of a pathogen.

The generation of antibodies takes after a characteristic design as takes after:

- 1. Lag phase:** Usually the prompt stage taking after introduction to antigen. During this phase, no antibodies are recognized in circulation.
- 2. Log phase:** This is often the following stage characterised by a relentless rise in counter acting agent titres within the circulation.
- 3. Plateau:** This is often a stage of harmony between counter acting agent union and catabolism.
- 4. Phase of decline:** This stage is characterised by an increment in counter acting agent catabolism compared to counter acting agent generation, driving to a drop in counter acting agent titre within the circulation.

### Engineering

The pressing need for non-immunogenic therapeutic mAbs (monoclonal antibodies) provided a strong stimulus for the development of a set of new technologies collectively referred to as antibody engineering <sup>[3]</sup>. The most commonly used approach was taking advantage of the ease of

immunizing rodents and making hybridoma. Parts of the rodent mAbs thus generated were then stepwise replaced with homologous human immunoglobulin (Ig) sequences. Naturally, these manipulations could not have been performed on the protein itself, but they were readily doable at the DNA (Deoxyribonucleic Acid) level, on rearranged immunoglobulin genes.

The first generation of engineered antibodies, termed ‘chimeric’, was constructed by replacing the heavy (H) and light (L) chain constant (C) regions of rodent mAbs with the corresponding human domains. As expected on the basis of model experiments, chimerization did reduce immunogenicity substantially, but the rodent variable regions were still sufficiently foreign to induce a HAMA (human anti-murine antibodies) response. Another useful aspect of the chimerization technology was that it permitted to exchange Ig isotypes at will, and thereby control the effector function of the antibody. For example, the human  $\gamma 1$  CH region was chosen, when the antibody was required to induce the complement cascade and cell-mediated killing. Conversely, the relatively inactive  $\gamma 4$  isotype could be used in blocking antibodies and in mAbs for diagnostic imaging.

The next step in reducing immunogenicity was the exchange of framework residues in the variable (V) region of rodent mAbs, referred to as ‘humanization’. The procedure itself consisted of transferring the complementary determining regions (CDR) of an active rodent antibody onto a human VH/VL region framework (of course, at the DNA level). The method had several variants, and was also referred to under different names, e.g., ‘CDR grafting’, ‘reshaping’, ‘hyperchimerization’, etc. Because the framework of V domains is a rigid  $\beta$ -sheet structure with the antigen-binding site formed by the six CDRs sitting on its top, the replacement of CDRs with different ones could be done without much concern about non-permissive conformational changes. Nevertheless, in some instances, conformational problems did occur, and a few additional changes in framework residues were necessary to correct them. Altogether, the humanization procedure was considered successful in reducing the immunogenicity of therapeutic mAbs to a manageable minimum. The industry was encouraged by these results, so that by the mid-1990s, humanized mAbs were already in the R&D pipeline of different pharma and biotech companies.

### Effector Function

In addition to different classes of antibodies, some antibodies like IgG and IgA have a few subclasses that are slightly different in their structure and also in their function <sup>[4]</sup>. In humans, IgG has 4 subclasses of IgG1 to IgG4, and IgA has IgA1 and IgA2 subclasses. Several cells of the immune system express Fc receptors, which enable them to capture immune complexes.

**Neutralization:** One of the main functions of antibodies is to recognize the microbial antigen or toxin and neutralize them in order to prevent them from binding or entering the host cells and causing infection. This is the function of the variable segment of antibodies. IgG is mainly neutralizing the antigens in the blood, while IgA does this function on mucosal surfaces.

**Opsonization and phagocytosis:** Some classes of antibodies (mostly IgG1 and IgG3) recognize the antigen

through the variable segment and facilitate phagocytosis of antigen through Fc-Fc receptor interaction on phagocytic cells. When antibody molecules cover an antigen, this is called opsonization. Macrophages and neutrophils will recognize the opsonized antigen, and they ingest and kill it intracellularly. Some complement system products such as C3b can also opsonize and facilitate the uptake of antigens.

#### **Antibody-dependent cell-mediated cytotoxicity (ADCC)**

This is another important function of antibodies. NK cells express the Fc receptor for IgG, and they can recognize antibody-coated cells, which results in NK cell degranulation and killing of the target cells.

IgG antibodies can pass through the placenta to the fetus. Therefore, maternal antibodies are the major defense mechanism of neonates. Other classes of antibodies like IgM and IgG1, IgG2, and IgG3 have the ability to activate the so-called classic pathway of the complement system.

Mast cells and eosinophils express Fc receptor for IgE and it can trigger mast cell degranulation and eosinophil mediated defense against helminths. IgA antibodies are crucial for mucosal immunity. IgA antibodies are secreted to the lumen of respiratory and GI tracts and neutralize the microbial antigens in the first encounter location.

#### **Viruses**

Viruses are the littlest irresistible agent that cause irresistible disease with tall dismalness and mortality on a worldwide scale [5]. Infections are classified on the premise of the genomic sort as either DNA or RNA (Ribonucleic Acid) infections. There's advance classification of the viral nucleic acid as either single-stranded or double-stranded. Infections are able to cause contamination in have cells with fitting particular have cell receptors that encourage viral connection, section, and intrusion of the have cell. Infections require have cells to duplicate and survive. Pulverization of the have cell by viral replication, along side safe reaction to the virus, results in clinical appearances of viral contamination. DNA infections more often than not experience replication within the core of the have cell while majority of infections with an RNA genome reproduces within the cytoplasm of the have cell. Viral replication within the host cell comes about in disturbance of ordinary cellular work, coming full circle in harm and passing of the contaminated cell. When infection disease causes quick cell passing, typically named lytic disease, wherein the contaminated cell is lysed as the virions develop. Be that as it may, numerous infections are moreover known to cause determined viral disease, which may take the shape of a inactive or determined beneficial disease. In inactive contamination, viral nucleic corrosive endures interior the have cell without experiencing replication or murdering the cell. Persistent profitable disease, or immortalization, is related with have cell senescence since of dynamic viral replication. This happens generally with RNA infections such as HTLV-1, during which the contaminated have cell gets to be immortalized due to obstructions with typical cell cycle, this comes about in have cell change. These shapes of viral way of life are related with the improvement of infection particular CD8+ T cells, which gotten to be fortified amid viral reactivation.

The human body is ensured from viral disease by physical and chemical boundaries. Be that as it may, when these boundaries are breached, diverse arms of the resistant

framework come into activity. The safe reaction includes the intrinsic resistant framework intervened by Dendritic cells (DCs), Natural Killer (NK) cells, monocytes, and sort I interferons (IFNs), and the versatile resistant reaction carried out by neutralizing antibodies and T cells. A complex transaction between diverse sorts of resistant cellular and dissolvable components of the natural and versatile resistant frameworks exists in anticipating and controlling constant infection contaminations. NK cells, complement proteins and cytokines are particular components of the antiviral natural resistant reactions, with DCs operating as immunosurveillance and immunostimulatory cells that handle and show viral antigen to T cells. DCs are partitioned into plas-macytoid and routine DCs. Plasmacytoid DCs respond to viral DNA and RNA to deliver sort I IFN that hinder viral replication in both tainted and noninfected cells. Ordinary DCs are one of the primary safe cells to come across infections at their harbour of passage into the have. The three subsets of routine dendritic cells, imperative in viral contamination, incorporate tissue-derived transient DC, lymphoid-resident DC, and monocyte-derived DC. Tissue-derived transient DCs incorporate Langerhans' cells and dermal/interstitial DCs that reside and survey the skin and mucoepithelial tissue. Lymphoid-resident DCs exist as juvenile DCs within the lymph nodes, spleen, and thymus. Monocyte determined or inflammatory DCs are those that are created from monocytes beneath incendiary conditions.

Dendritic cells are found in key parts of the body to guarantee ideal execution of immunosurveillance and immunostimulatory capacities with the expectation of activating both intrinsic and adaptive safe reaction to the nearness of infections that breach the anatomical and chemical defense obstruction of the have. Siglec-1 (CD169), DC-SIGN, mannose receptor, Langerin, safe dendritic cell receptor (DCIR), heparan sulfate proteoglycan, FC gamma receptors, and syndecan-3 are surface connection receptors on DCs that encourage take-up of viruses by DCs. Infection interaction with DCs can cause debasement of the infection inside the cell to permit for antigen-MHC complex arrangement for introduction to T cells. This trigger the T cell interceded antiviral resistant reaction. Viral antigens are either created amid intracellular replication in infection tainted cells or created from recognition of viral components from other tainted cells. A few infections can tie to and duplicate inside DCs, with DCs acting as lenient cells that encourage viral spread to other cells or tissues within the have, coming about in trans-infection of lymphocytes in territorial lymph hubs. This sort of disease is especially seen when viral antigens are not complexed to MHC particles as another way to alarm the resistant framework and enact T cells.

#### **Immune System**

The immune system contains a momentous task [6]. Within the least complex terms, it must secure the have from cancers and irresistible malady whereas carefully directing reactions so as not to deliver any long-term harm of have tissues. These resistant reactions are not culminate: cancers do create indeed within the confront of an introductory resistant reaction; irresistible infections do overpower the resistant system and claim lives; and immune system infections are a cause of noteworthy pathology in those beset. In spite of these flaws, a huge sum of information

shows tackling the useful reactions of the resistant framework gives imaginative conceivable outcomes to treat patients enduring from cancers, constant diseases, and autoimmune diseases. This field, known as immunotherapy, has quickly created over the past 2 decades and delivered a number of compelling medicines primarily within the cancer field. Immunotherapies are based on either counter acting agent intervened (humoral insusceptibility) or cell interceded resistance by enacting T cell resistant reaction.

Cell mediated immunity, driven by CD4+ and/or CD8+ T lymphocytes, plays a basic part in in protecting the have against cancers and irresistible diseases. These reactions start in auxiliary lymphoid organs (i.e. lymph nodes or spleen) when dendritic cells and/or macrophages display parts of protein antigens, named peptide epitopes, to the T cells. These peptide epitopes are created through a number of antigenic preparing pathways. Antigens endocytosed from the extracellular environment are broken down within the endosomal/lysosomal system and stacked onto major histocompatibility course (MHC)-II atoms for introduction to CD4+ T cells. In differentiate, antigens biosynthesized interior of the showing cell are broken down by the proteasome, peptide parts transported into the endoplasmic reticulum, trimmed encourage, and stacked onto MHC-I atoms for introduction to CD8+ T cells. Vitaly, there's a awesome bargain of cover between these pathways; endocytosed antigenic parts can be handled by the proteasome and stack onto MHC-I molecules while biosynthesized antigens can be prepared within the endosomal/lysosomal system and stacked onto MHC-II particles. This "cross-talk" without a doubt broadens the number of targets vital for an effective cell intervened reaction. In typical, solid cells, self-proteins go through these pathways and an cluster of peptides is displayed on the MHC particles. These peptides are recognized as 'self' and thus don't provoke a T cell reaction. In any case, changes in this MHC signature of cells alarm T cells to changes within the have that will be associated with disease, dangerous change, or other anomalous cellular forms, coming about in a cascade of occasions that initiate a cell intervened resistant reaction. In this case, when the proper "match" is found, the appropriately coordinated T cell clone is actuated, extends, and moves to the tumor or location of contamination to intervene effector capacities.

### Immune Response

Antigen presentation starts an cluster of responses including the safe system, counting the actuation of white blood cells called lymphocytes that battle contamination [1]. After antigen introduction, two sorts of lymphocytes, B cells and T cells, separate into effector (e.g., antibody-producing B cells and cytotoxic and partner T cells) and memory cells. For both B and T cells in a normal resistant reaction to an antigen presentation, the inactivity between the primary (essential) introduction and improvement of the essential reaction is characterized by a slack stage, logarithmic stage, and level stage. The slack stage is characterized by the introductory actuation of B and T cells upon experience with the antigen for which they are particular, and this triggers the cells' separation into effector and memory cells. The slack stage between essential presentation to an antigen and the logarithmic stage is classically thought to be 4 to 7 days, but it changes depending on course of introduction and the antigen itself. For B cells, the logarithmic stage is

characterized by an increment in serum counter acting agent levels that classically is logarithmic. The level stage is characterized by the support of top counter acting agent levels for a length of time that's taken afterby a decrease within the serum counter acting agent levels. For numerous antigens the idleness (slack stage) between essential introduction and improvement of the essential counter acting agent reaction is 7 to 10 days. Due to the improvement of memory B and T cells amid the essential resistant reaction, the idleness between ensuing presentation to the antigen and advancement of the resistant reaction will ordinarily be shorter. The slack stage is by and large 1 to 3 days; the logarithmic stage of the auxiliary counter acting agent reaction happens over the another 3 to 5 days. As said for the essential safe reaction, these time periods will change depending on the course of introduction, the timing of the ensuing presentation, the antigen itself, and the antigen dose. While this dialog isn't particular to a specific antigen, it can be utilized as a reference point for the idleness between antigen presentation and the start of a few of the immune-mediated mechanisms described underneath.

Contributing to the actuation of B and T cells and the start of the adaptive resistant reaction are cells classically related with the natural safe system (e.g., macrophages and dendritic cells). These cells play parts at each of the stages said over and are as a rule the primary cells of the safe framework to be uncovered to antigen. Upon antigen experience, macrophages and dendritic cells engulf the antigen, a handle that moreover enacts these intrinsic resistant cells to gotten to be antigen-presenting cells. Antigen-presenting cells, as their title proposes, display the antigen to T cells and discharge inflammatory mediators (e.g., cytokines and chemokines) that contribute to the enlistment, actuation, and multiplication of B and T cells. Enacted B and T cells in turn discharge fiery arbiters driving to the enrollment and enactment of extra resistant cells that encourage open up the resistant reaction through the discharge of provocative arbiters. Administrative cells and dissolvable immunoregulatory arbiters (not examined in this report) play parts in stifling the resistant reaction.

### Adaptive Immunity

On a very basic level, intrinsic resistant system is all almost the fight of self-versus non-self-antigens [7]. Wouldn't it be pleasant on the off chance that our body won all the fights? Unnecessary to say, life doesn't work that way. In some cases, the natural safe framework can't very handle the stack. Possibly, for a few obsessive or natural reason your framework is compromised ("immunocompromised") or debilitated or smothered ("immunosuppression"). Maybe the antigen isn't being expelled (determined cause), or it keeps reoccurring (re-exposure) as the natural system tries to eliminate it. Or possibly the antigen is as well inexhaustible or as well pernicious (virulent) for the intrinsic resistant reaction to overcome it. In such conditions, after a number of days to a week of feeling "not so great," the quality of the human resistant framework starts to sdemonstrate a more forceful "adaptive safe response." All in all, this adaptive resistant framework could be a capable guard and defender to a point.

Adaptive immunity is more energetic than the natural shape and control of its concentrated and term is ineffectively caught on with hereditary inclination and three particular components accepted to be interrelated. To begin with is



“feedback inhibition” where expulsion of the antigen diminishes jolt and in this way diminished generation of antibodies and cytokines, successfully diminishing and turning around the reaction. Moment is when T-suppressor cells diminish T-helper cells and hence a commensurate decrease of B-cell movement (controlled by T-helper cells). And third, the exceptionally complex framework of idiotype antigen-specific B-cells expanding through hereditary cloning and making their possess immunogenic boosts which actuates anti-idiotype-specific antibodies (“antibodies 1, 2”) that build up an counter acting agent idiotype-specific administrative circuit.

Failure to evacuate the irritating antigen in a opportune way, or breakdown of any one of the three components portrayed over, can lead to an uncontrolled reaction coming about in intense aggravation (talked about underneath), unremitting aggravation, or a condition alluded to as a “cytokine storm” which has possibly annihilating results (as demonstrated in SARS-CoV-2 contaminations). These wonders and the endpoints of versatile insusceptibility speak to the moment half of our prior expressed conundrum around the resistant framework. It can undoubtedly be our most noticeably awful foe.

### COVID-19 Test

Counter acting agent tests check a person’s blood by searching for antibodies, which may (or may not) tell in case the individual had a past disease with the coronavirus [7]. Antibodies are proteins that offer assistance battle off contaminations and hence, can give insusceptibility and security against getting the disease once more. Neutralizing antibodies are particular to an antigen (The infection) and in this way, give security as it were against the particular infection related with the antigen (Within the case of coronavirus as the antigen, the illness being COVID-19). In the event that the individual is uncovered to the antigen (Coronavirus) once more, the antibodies create “memory” (Anamnestic security) toward the infection. In any case, there are expanding reports of reinfection with the novel coronavirus recommending that a few coronavirus antibodies may not be neutralizing nor endure for amplified periods.

But in occurrences in which viral testing is deferred, counter acting agent tests ought to not be utilized to diagnose a current COVID-19 disease. An counter acting agent test may not appear on the off chance that you have got a current COVID19 contamination since it can take one to three weeks after disease for your body to create antibodies. To see in the event that you're as of now tainted, you would like a viral test. Viral (antigen) tests portrayed over recognize the infection in tests from your respiratory framework, such as a swab from the interior of your nose. It is conceivable to confine the coronavirus from respiratory emissions, blood, pee, and fecal tests for symptomatic testing. Clinically, contaminations can be analyzed with respiratory viral boards that are broadly commercially accessible.

### Conclusion

Antibodies are defensive proteins that are created when antigens enter the body, i.e. substances that under certain conditions trigger a specific immune response and stimulate the formation of antibodies with which they then react. The function of antibodies is to protect the body from bacteria, viruses, fungi, tumors and other disease-causing agents.

Sometimes the immune system does not recognize the body's products and rejects them. This is how various autoimmune diseases arise. Elevated antibodies in the blood, but also lowered ones, point to a disproportion that often leads to certain diseases.

### Conflict of Interest

Not available

### Financial Support

Not available

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#### How to Cite This Article

F Sinisa. Antibodies protect the body from various pathogens. International Journal of Immunology Research. 2023;5(1):14-18.

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